Volume 1 Issue 2 April 2019 ISSN 1106-7237

Hellenic Society of Vascular and Endovascular Surgery

Hellenic Journal of Vascular and Endovascular Surgery

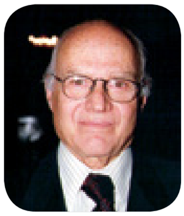
HOT TOPICS

PUBLICATIONS ROTONDA

www.heljves.com

PAST EDITORS

First period: Hellenic Vascular Surgery (2007-2014)



Prof. Michael Sechas
Founding Editor
Editor in Chief 2007-2010

Prof. Chris Liapis
Editor in Chief 2011-2014

Second period: Hellenic Journal of Vascular and Endovascular Surgery (2019-)

Prof. Miltos Matsagkas
Founding Editor
Editor in Chief 2019-

Prof. John Kakisis
Senior Editor 2019-

Hellenic Society of Vascular and Endovascular Surgery
Feidipidou 8, Athens, Greece
Tel: +30 211 409 46 43
e-mail: info@vascularsociety.gr

Rotonda Publications
8 Kamvounion Str, 54621 Thessaloniki, Greece
Tel: +30 210 212 212

Hellenic Journal of Vascular and Endovascular Surgery

Instructions for authors

Submission

All manuscripts are submitted through the official site of the Journal:

heljves.com
https://heljves.com/Submit-Paper/

Submission list:

- Cover letter (word format)
- Manuscript [word format; includes: abstract, main manuscript, table legends, tables, figure legends and figures (optional)]
- Figures (Tiff format; more details in figure files section)

Peer Review

All manuscripts are reviewed initially by the Chief Editor, Executive Editor, and/or their representatives. A submission is rejected outright if the majority opinion finds that (1) the material does not have sufficient merit to warrant further review or (2) the subject matter is outside the scope of the Journal. The submission may also be returned for inadequate presentation or failure to comply with the HJVES's submission instructions.

Authors should read the HJVES Editorial Policies before constructing the manuscript; it contains detailed information about originality, authorship, primacy, research and animal experimentation, patient consent, conflict of interest disclosure, funding, permissions, scientific misconduct, peer review, contributor publishing agreement, and publication.

Instructions for authors in detail

Overview

Manuscripts considered for publication must be written in English and structured as a basic or clinical research, editorial, case report, vascular images, letter to the editors, debate with pros and cons, technical description on 'how I did it', review, systematic review and meta-analysis. Type of articles and their word count, tables', figures' and references' number are demonstrated in table 1.

Manuscript type	Word count of abstract	Word count of body of the manuscript	Number of figures/ tables	Number of references
Vascular image	NA	200	2	NA
Case report	150	1000	4	10
Letter to the editor	NA	500	1	5
How I did it	150	1500	4	10
Editorial	NA	1000	1	10
Debate: Pros vs. Cons	NA	1000 (each)	1 (each)	10
Basic research	200-300	3500	10	50
Clinical research	200-300	3500	10	50
Review, Systematic review, Meta- analysis	250-350	3500-5000	15	100

Cover Letter

Manuscripts (except correspondence) must be accompanied by a cover letter signed by all authors stating (1) there has been no duplicate publication or submission of any part of the work; (2) all authors have read and approved the manuscript; and (3) there is no financial arrangement or other relationship that could be construed as a conflict of interest. If a potential conflict exists, its nature should be stated in the letter and on the title page for each author involved (see Editorial Policies for a complete explanation).

Title Page File

- Construct a title that does not exceed 50 words.
- List first and last names, highest academic degree(s), affiliations, and email addresses for all authors.
- Give the total word count.
- Acknowledge all sources of financial support (grants, fellowships, equipment, or remuneration of any kind) and any relationships that may be considered a conflict of interest (ie, employment, stock holdings, retainers, paid or unpaid consultancies, patents or patent licensing arrangements, or honoraria) that may pertain to the manuscript (see Editorial Policies, Conflict of Interest).
- Give details of any prior presentation, including meeting name, location, and date.
- List acknowledgments, any shared first authorship, and other author notes.
- Give the name, mailing address, and email address of the corresponding author.

Abstract

- Give a substantive summary of a basic or clinical research article in 300 words or less, and up to 350 words for systematic reviews and meta-analysis, separating the abstract according to Introduction, Methods, Results, and Conclusion. For case reports and 'how I did it' the abstract should be no longer than 150 words and divided into Purpose, Case Report/ Technique and Conclusion.
- Provide up to 5 keywords.

Text

- Text material must be submitted as a single Word document (not a PDF) named the "Main document."
- Organize the text for clinical or basic experimental investigations and systematic review and meta-analysis into sections entitled Introduction, Methods, Results, Discussion, and Conclu-

sion. Case reports and ‘How I did it’ require only Introduction, Case Report/Technique, Discussion, and Conclusion. Editorials, letter to the editor, vascular images, and debate with pros and cons may be structured as appropriate for the material.

- Avoid naming any institution(s) in the work or otherwise identifying the author(s). • Use SI measurements; generic drug names should be used.
- Define abbreviations and acronyms when they first appear in the text; do not list them at the beginning of the manuscript file.
- Identify tables and figures using Arabic numerals in parentheses (eg, Table 1, Figure 1).

References

- Follow the guidelines in the AMA Manual of Style (10th ed., 2007). Do NOT use endnotes or other bibliographic style function for reference lists.
- Limit to 10 the number of references for a case report/ ‘how I did it’/ editorial and debate with pros and cons. Letters may have no more than 5 references. Basic and clinical articles may have up to 50 references, while systematic review and meta-analysis up to 100 references.
- Number references in the order of appearance in the text. Identify references in the text, tables, and legends as superscript Arabic numerals.
- List the first 6 authors (last name and initials separated by a comma); use “et al” for 6 or more authors. Abbreviate journal titles according to the style of Index Medicus; formats for the common types of journal citations are: Journal article: authors' names and initials, article title, journal name, year, volume, and inclusive page numbers.

Examples:

Matsagkas MI, Kouvelos G, Spanos K, Athanasoulas A, Giannoukas A. Double fixation for abdominal aortic aneurysm repair using AFX body and Endurant proximal aortic cuff: mid-term results. Interact Cardiovasc Thorac Surg. 2017;25:1-5.

Giannoukas AD, Chabok M, Spanos K, Nicolaides A. Screening for Asymptomatic Carotid Plaques with Ultrasound. Eur J Vasc Endovasc Surg. 2016;52:309-12.

- Cite entire books by giving the author/editor(s), title, edition, city/state of publication, publisher, and year. Example: Ahn SS, Moore WS, eds. Endovascular Surgery, 2nd ed. Philadelphia, PA: WB Saunders Co.; 1991.
- Cite chapters in books giving the author(s), chapter title, editor(s), book title, city/state of publication, publisher, year, and inclusive page numbers. Example: Towne JB. Postintervention surveillance. In: White RA, Fogarty TJ, eds. Peripheral Endovascular Interventions. St. Louis, MO: Mosby-Year Book, Inc.; 1996:109-115.

Legends

- Type all figure and table legends on a separate page of the manuscript file, explaining abbreviations and symbols used in the figure. Previously published figures must be acknowledged and accompanied by written permission from the publisher to reproduce the material if it is copyrighted.
- Do not use Word’s caption function for figure legends or include the actual figures in the manuscript file.

Table Files

- Use tables to supplement the text, not duplicate it.
- Insert tables in the text or create/save tables as an image.
- Format tables using the table formatting function in Word; elaborate formatting (shading, color) should not be used.
- Define any abbreviations as the first footnote under the table; list the abbreviations alphabetically.
- Use footnotes for explanatory material, labeling each with a superscript lower case letter (a-z) in alphabetical order.

Figure Files

- Number any pictures, charts, graphs, or line art sequentially as figures.
- Use color judiciously in pictures and graphics. Figures will be printed in grayscale unless color charges are paid. There is no fees for color figures for print/online display.
- Add arrows and symbols to digitally created images using functions supplied with the imaging program.
- Do not import images into the text document but transmit each image file separately.
- Supply all figures in a digital format of suitable quality for printing: TIF for pictures or EPS for graphs and line drawings (to preserve quality when enlarged/zoomed). Image resolution should be at least 300 ppi for color or grayscale images and 600 ppi (preferably higher) for black and white line drawings or graphs. Image size at these resolutions should be no less than 3 inches wide for vertical images and 5 inches wide for horizontally oriented figures. Use a lossless compression algorithm (eg, LZW) that does not degrade the resolution.
- Convert PowerPoint slides to individual TIF files for upload.

Publication

Accepted manuscripts will be scheduled for publication generally in the order in which they are received after no further author revisions are required and the Journal Contributor’s Publishing Agreement has been signed by the corresponding author. The HJVES reserves the right to edit accepted manuscripts to comply with the journal’s format, to correct grammatical faults, to remove redundancies, and to improve readability without altering the meaning. Several weeks before the scheduled publication of an article, the Editorial Office will send via email an edited version of the manuscript to the corresponding author for approval. After the author has approved the edited version, the publisher will send a PDF of the page proof by email. At this stage, only correction of typographical errors or mistakes in the presentation of data can be made. Approval/changes to the proof must be returned within 2 days. The HelJVES value their relationships with their authors and appreciate author compliance with these instructions.

Scientific Misconduct

The HJVES takes issues of copyright infringement, plagiarism, or other breaches of best practice in publication very seriously. Every effort is made to protect the rights of authors, and claims of plagiarism or misuse of articles published in the journal are always investigated. Equally, the reputation of the journal is protected against malpractice. Submitted articles may be checked using duplication-checking software. Where an article is found to have plagiarized other work or included third party copyright material without permission or with insufficient acknowledgment, or where authorship of the article is contested, the publisher reserves the right to take action including but not limited to publishing an erratum or corrigendum (correction); retracting the article (removing it from the journal); taking up the matter with the head of department or dean of the author’s institution and/or relevant academic bodies or societies; banning the author from publication in the HJVES or all journals, or appropriate legal action.

Contents

CASE REPORTS

VASCULAR IMAGES


Cilos®
cilostazol
Qualitative and Quantitative Composition: cilostazol 50 mg και 100 mg

Xarelto®
rivaroxaban
PP-XAR-GR-0038-1
5 Dec 2018
Bayer
Κάτοχος της άδειας κυκλοφορίας:
Bayer AG, 51368 Leverkusen, Γερμανία
Τοπικός αντιπρόσωπος του κατόχου άδειας κυκλοφορίας στην Ελλάδα: Bayer Ελλάς ΑΒΕΕ,
Σωρού 18-20, 151 25 Μαρούσι,
Τοπικός αντιπρόσωπος του κατόχου άδειας κυκλοφορίας στην Κύπρο: Novagem Ltd,
Τηλ:00357 22483858
Τμήμα Επιστημονικής Ενημέρωσης
Τηλ: +30 210 6187742, Fax: +30 210 6187522
Email: medinfo.gr.cy@bayer.com
Εταιρεία συμπροώθησης
ELPEN
ELPEN Α.Ε. ΦΑΡΜΑΚΕΥΤΙΚΗ ΒΙΟΜΗΧΑΝΙΑ
Λεωφ. Μαραθώνος 95, 190 09 Πικέρμι Αττικής,
Τηλ: 210 6039326 - 9, Fax: 210 6039300
ΓΡΑΦΕΙΑ ΕΠΙΣΤΗΜΟΝΙΚΗΣ ΕΝΗΜΕΡΩΣΗΣ
Σεβαστείας 11, 115 28 Αθήνα,
Τηλ: 210 7488711, Fax: 210 7488731
Εθν. Αντιστάσεως 114, 551 34 Θεσσαλονίκη,
Τηλ: 2310 459920 - 1, Fax: 2310 459269
Βοηθήστε να γίνουν τα φάρμακα πιο ασφαλή και Αναφέρετε
ΟΛΕΣ τις ανεπιθύμητες ενέργειες για ΟΛΑ τα φάρμακα
Συμπληρώνοντας την «ΚΙΤΡΙΝΗ ΚΑΡΤΑ»
Πριν τη συνταγογράφηση για κάθε ένδειξη συμβουλευθείτε την αντίστοιχη Περίληψη των Χαρακτηριστικών του Προϊόντος.

EDITORIAL

Treatment of Thoracoabdominal Aortic Aneurysms in 2019: Endovascular or open repair as first line treatment?

Athanasios Katsargyris, Eric LG Verhoeven

Department of Vascular and Endovascular Surgery, Paracelsus Medical University, Nuremberg, Germany

In the last decade, three options have emerged to treat thoracoabdominal aortic aneurysms (TAAA): (i) open repair, (ii) endovascular repair with the use of fenestrated/branched stent-grafts (F/BEVAR), and (iii) hybrid repair consisting of visceral debranching in conjunction with endovascular aneurysm exclusion with standard stent-grafts. Open repair has been considered the first line treatment for appropriate-risk patients and has benefitted with the development of adjunctive techniques such as left-left heart bypass and neuromonitoring.[1,2] Endovascular approach has expanded the treatment options, especially in patients "unfit for open repair".[3-5] Hybrid repair enjoyed great interest about 10 years ago, but results were disappointing and now seems to play a marginal role in some patients unfit for open repair and with unsuitable anatomy for endovascular repair.[6,7] Marc Schermerhorn in our opinion correctly summarized the current status of all techniques in following quote: *"The described hierarchy of treatment preferences is mostly artificial, based on the historical reliability of open repair and the investigational nature of endovascular techniques."*[8]

Ideally, a randomized controlled trial (RCT) would provide a higher degree of evidence to answer the question whether open or endovascular repair should nowadays be considered as the 1st line treatment for most TAAAs. Such an RCT is currently not available, and will unfortunately probably never become available given the inherent difficulties regarding timing (quick evolution of endovascular techniques, different level of operative skills, experience, logistics in participating centres) and inclusion criteria (only patients deemed suitable for both techniques). Despite the lack of RCTs, there are contemporary data originating mainly from observational studies that are useful to compare open with endovascular treatment of TAAA.

In a propensity matched comparison with 341 patients, two Italian groups demonstrated an early benefit of endovascular repair with reduced perioperative respiratory morbidity compared to open repair. Mid-term survival and reintervention rates between the two methods were equal.[9] A recent analysis of the nationwide German DRG data with a total of 2607 patients showed that endovascular repair was increasingly used over time (from 6% in 2005 to 76% in 2014) and is currently the 1st line treatment for TAAA in Germany.[10] Endovascular TAAA repair with F/BEVAR was associated with a significant reduction of in-hospital mortality [risk reduction (RR) 0.35, 0.24-0.51, p<0.001]. A meta-analysis of eight studies comparing endovascular (831 patients) vs open repair (2231 patients) of TAAA showed significantly lower 30-day mortality (RR: 0.63; p < 0.01), spinal cord ischemia (RR: 0.65; p = 0.05), incidence of dialysis (RR: 0.44; p = 0.01) and length of hospital stay (mean difference, 4.4 days; p < 0.01) for endovascular repair.[11] Similarly, Locham et al. in a comparative study of 879 patients (481 endovascular repair vs 398 open repair) showed a significant reduction of 30-day mortality (5% vs 15%, p<0.001) and morbidity (2-3 fold reduction for all major complications) for endovascular repair.[12] One could therefore cautiously conclude that endovascular TAAA repair provides a significant early advantage over open TAAA repair.

Searching for long-term results reveals that durability of open TAAA repair is not well-documented.[1] The long-term patency of branch grafts to the visceral and renal arteries after open TAAA repair is actually unknown. The largest available series of patients with long-term angiographic follow-up of branch grafts after open TAAA repair was published only recently, in 2017 by Kouchoukos et al. including a total of 33 patients with a follow-up of more than 5 years.[13]

Durability of branches after endovascular TAAA repair has been documented in multiple series[14-16], with the longer term outcomes being reported by the group of the Cleveland Clinic. Mastracci et al. showed in a cohort of 650 patients with follow-up duration up to 9 years that branches in F/BEVAR are durable and rarely the cause of patient death (0.46% during the whole follow-up period).[16] Most importantly, the same group showed excellent overall efficacy for F/BEVAR in the long-term, as reflected by a freedom from aortic related mortality of 98% at 8 years.[15] Similar outcomes are also seen in our series, with aortic related mortality <1% at 5 years in our updated experience with more than 350 patients treated up to now. Reinterventions mainly for branches are required in almost 20% of the patients after 5 years, but most of these reinterventions can be completed successfully by endovascular means with minimal morbidity and zero mortality. Nev-

Author for correspondence:

Athanasios Katsargyris, MD, PhD

Klinikum Nürnberg, Vascular and Endovascular Surgery, Germany
E-mail: kthanassos@yahoo.com
ISSN 1106-7237/ 2019 Hellenic Society of Vascular and Endovascular Surgery Published by Rotonda Publications

ertheless, we also concluded that one should be critical with patient selection, especially those at highest risk (ASA IV), as in these patients both early and late (unrelated) mortality are significantly higher.[17]

Financial costs also merit evaluation. Clearly, material costs for F/BEVAR significantly exceed those of open repair. But if one calculates the total costs and resources (materials, hospitalization including intensive care unit costs, blood transfusion costs), F/BEVAR may in the end be more cost effective than open repair. As Locham et al. recently demonstrated, F/BEVAR was almost 8,000 US dollars cheaper than open repair (mean total cost 36.612 US dollars for endovascular vs 44,355 US dollars for open, p=0.004).[12] This difference was driven by higher morbidity and longer hospitalization after open repair.

Putting all of the above together, it appears that endovascular repair of TAAA is associated with reduced perioperative mortality and morbidity and lower overall costs compared to open repair, while mid-term durability remains good provided adequate individualized surveillance. If one adds the patient's preference, which is most commonly in favor of a minimal invasive treatment, it seems reasonable to consider endovascular repair as the first line treatment for most patients with TAAAs. This statement should exclude patients with connective tissue disease and maybe some other younger patients. This treatment shift has not been officially adopted by the current guidelines yet, but F/BEVAR to treat TAAA is clearly the real-life "first choice" in many countries nowadays.

Such a shift towards "endovascular first" has resulted in growing endovascular experience in many centers worldwide. But at the same time this leads unavoidably to decreasing experience in open TAAA repair. Certainly, the number of surgeons who can perform the traditional open TAAA operation with good results is decreasing and will decrease further in the future. And this may not be without consequences especially for cases that open repair is needed to correct failures of endovascular repair.

REFERENCES

1 Latz CA, Cambria RP, Patel VI, et al. Durability of open surgical repair of type I-III thoracoabdominal aortic aneurysm. J Vasc Surg 2019

2 Moulakakis KG, Karaolanis G, Antonopoulos CN, et al. Open repair of thoracoabdominal aortic aneurysms in experienced centers. J Vasc Surg 2018;68:634-45 e12

3 Law Y, Tsilimparis N, Rohlffs F, et al. Fenestrated or branched endovascular aortic repair for postdissection thoracoabdominal aortic aneurysm. J Vasc Surg 2019

4 Maurel B, Delclaux N, Sobocinski J, et al. The impact of early pelvic and lower limb reperfusion and attentive peri-operative management on the incidence of spinal cord ischemia during thoracoabdominal aortic aneurysm endovascular repair. Eur J Vasc Endovasc Surg 2015;49:248-54

5 Katsargyris A, Oikonomou K, Kouvelos G, Renner H, Ritter W, Verhoeven EL. Spinal cord ischemia after endovascular repair of thoracoabdominal aortic aneurysms with fenestrated and branched stent grafts. J Vasc Surg 2015;62:1450-6

6 Di Marco L, Murana G, Leone A, et al. Hybrid repair of thoracoabdominal aneurysm: An alternative strategy for preventing major complications in high risk patients. Int J Cardiol 2018;271:31-5

7 Hawkins RB, Mehaffey JH, Narahari AK, et al. Improved outcomes and value in staged hybrid extent II thoracoabdominal aortic aneurysm repair. J Vasc Surg 2017;66:1357-63

8 Schermerhorn ML, Jones DW. Management of Descending Thoracic Aorta Disease: Evolving Treatment Paradigms in the TEVAR Era. Eur J Vasc Endovasc Surg 2017;53:1-3

9 Ferrer C, Cao P, De Rango P, et al. A propensity-matched comparison for endovascular and open repair of thoracoabdominal aortic aneurysms. J Vasc Surg 2016;63:1201-7

10 Geisbusch S, Kuehnl A, Salvermoser M, Reutersberg B, Trenner M, Eckstein HH. Hospital Incidence, Treatment, and In Hospital Mortality Following Open and Endovascular Surgery for Thoraco-abdominal Aortic Aneurysms in Germany from 2005 to 2014: Secondary Data Analysis of the Nationwide German DRG Microdata. Eur J Vasc Endovasc Surg 2019

11 Rocha RV, Friedrich JO, Elbatarny M, et al. A systematic review and meta-analysis of early outcomes after endovascular versus open repair of thoracoabdominal aortic aneurysms. J Vasc Surg 2018;68:1936-45 e5

12 Locham S, Dakour-Aridi H, Nejim B, Dhaliwal J, Alshwaily W, Malas M. Outcomes and cost of open versus endovascular repair of intact thoracoabdominal aortic aneurysm. J Vasc Surg 2018;68:948-55 e1

13 Kouchoukos NT, Kulik A, Castner C. Branch graft patency after open repair of thoracoabdominal aortic aneurysms. J Thorac Cardiovasc Surg 2017;153:S14-S9

14 Walker J, Kaushik S, Hoffman M, et al. Long-term durability of multibranched endovascular repair of thoracoabdominal and pararenal aortic aneurysms. J Vasc Surg 2019;69:341-7

15 Mastracci TM, Eagleton MJ, Kuramochi Y, Bathurst S, Wolski K. Twelve-year results of fenestrated endografts for juxtarenal and group IV thoracoabdominal aneurysms. J Vasc Surg 2015;61:355-64

16 Mastracci TM, Greenberg RK, Eagleton MJ, Hernandez AV. Durability of branches in branched and fenestrated endografts. J Vasc Surg 2013;57:926-33; discussion 33

17 Verhoeven EL, Katsargyris A, Bekkema F, et al. Editor's Choice - Ten-year Experience with Endovascular Repair of Thoracoabdominal Aortic Aneurysms: Results from 166 Consecutive Patients. Eur J Vasc Endovasc Surg 2015;49:524-31

Outcome of open Repair of Ruptured Thoracoabdominal Aortic Aneurysms. A Systematic Review and Meta-Analysis

Konstantinos G. Moulakakis[1], Constantine N. Antonopoulos[2], Georgios Karaolanis[3], John Kakisis[1], Christos Klonaris[3], Andreas Lazaris[2], George Sfyroeras[1], George Theocharopoulos[1], Spyridon N. Vasdekis[1], George Geroulakos[1]

[1]Department of Vascular Surgery, Medical School, National and Kapodistrian University of Athens, Attikon University Hospital, Athens, Greece

[2]Department of Cardiothoracic and Vascular Surgery, Evangelismos General Hospital of Athens, Athens, Greece

[3]Vascular Unit, First Department of Surgery, "Laiko" General Hospital, Medical School, National and Kapodistrian University of Athens, Athens, Greece

Abstract:

Introduction-Aim: Rupture is the most lethal complication in untreated cases of thoracoabdominal aortic aneurysms (TAAAs). We performed a systematic review and meta-analysis attempting to identify all published reports on ruptured thoracoabdominal aortic aneurysms (rTAAs) treated with open repair with the aim to assess the mortality rate and common complications.

Methods: We used the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. A thorough search of the English-language literature published until January 2019 was performed to identify studies relative to ruptured TAAAs. Data extracted from the eligible studies, included first author's name, study year, type of study, total number of patients with rTAA, number of deaths, mean age and mean follow-up period.

Results: Overall, 16 articles were included in the meta-analysis after application of inclusion and exclusion criteria. A total of 1,036 patients with ruptured aneurysms were included in the present analysis. Pooled mortality rate after open repair of rTAAs from all 16 studies was 33.40% (95%CI=22.60-45.07; I2=92.5%, p<0.001). When we pooled data only from four studies reporting data exclusively on rTAA, the pooled mortality rate was 47.77% (95% CI=32.71-63.04, I2=84.4%, p<0.001). Furthermore, we found a statistically significant decreasing trend in mortality rates over time (Q=4.13, p<0.04) Cardiac event rate was 8.79% (95% CI=1.55-20.20, I2=85.81%, p<0.001), pooled permanent paraplegia was estimated at 5.56% (95%CI=1.76-10.96) pooled stroke rate was 1.61% (95%CI=0.01-6.67; I2=43.29%, p=0.17) and re-intervention rate was 8.33% (95%CI=2.86-15.82).

Conclusions: Rupture is a lethal complication in untreated cases of TAAAs and it is associated with an approximately 50% mortality rate. Young and fit patients with contained rupture who are not shocked on presentation might have a better outcome, especially if transferred in experienced centers.

INTRODUCTION

Open repair of thoracoabdominal aortic aneurysms (TAAs) remains a technically demanding operation. Adjunctive strategies especially used in the era of organ protection, have improved the outcome of these patients and have reduced the occurrence of paraplegia, renal failure, and mesenteric ischemia. These strategies include distal aortic perfusion with left heart bypass, cerebrospinal fluid drainage, and intercostal artery reimplantation. However, despite the advances in open surgical techniques, the morbidity and mortality rates for the treatment of TAAs continue to remain considerably high. In a recent metanalysis, the pooled mortality rate among patients with TAAAs treated electively was 11.3%.[1] This metanalysis also estimated a pooled spinal cord ischemia rate of 8.3%, a stroke rate of 3.1%, whereas the need for permanent dialysis rate was 7.9%.[1]

Rupture is the most lethal complication in untreated cases of TAAAs. Nationwide clinical data derived from the inpatient sample on patients who underwent repair of a ruptured TAAA from 1988 to 1998 in the USA showed an overall surgical mortality of 53.8%.2 The renal failure (28%) and the cardiac complications (18%) were the most common complications. In this context, we performed a systematic review and meta-analysis attempting to identify all published reports on ruptured TAAs (rTAAs) treated with open repair with the aim to assess the mortality rate and common complications.

Author for correspondence:

Konstantinos G. Moulakakis

Attikon University Hospital, Athens, Greece,
Rimini 1 Str, Haidari, Athens, 12462 Greece
Tel: +30 6937357508, Fax: +30 2105831484,
E-mail: konmoulakakis@yahoo.gr
ISSN 1106-7237/ 2019 Hellenic Society of Vascular and Endovascular Surgery Published by Rotonda Publications

Authors	Journal / Year	Study period	TOTAL Patients	Mean age (years)	Number of r- TAAA (n)/%	Technique	Organ protection
Murana et al	European Journal of Cardio-Thoracic Surgery, 2015	1994-2014	542	65	48/8.9%	LSHB(87.3%) / CPB+DHCA(occasionally)	Sequential aortic clamping/ CSFD/SSEP&MEP/ Reattachment of intercostal arteries
Coselli et al	The Journal of Thoracic and Cardiovascular Surgery,2015	1986-2014	3309	67	170/ (5.1%)	LSHB/MHCA/ DHCA(Rarely)	Cold crystalloid perfusion/ selective visceral perfusion/ Reattachment of intercostal arteries/CSFD
Zanetti et al	Kardiochirurgia I Torakochirurgia Polska, 2015	1994-2014	51	62	51/100%	LSHB (50%) / CPB+FFB (15.6%)/ CS (13.7%)/ CPB+DHCA (19.6%)	DHCA/CSFD//Reattachment of intercostal arteries
Youssef et al.	J Vasc Surgery,2015	1998- 2012	62	66	15/24.2%	CPB/PCPB/ LSHB	CSFD/SSEP&MEP Cold crystalloid perfusion/ selective visceral perfusion
Fukui et al	Ann Vasc Surg, 2015	2009-2015	44	65	6/13.6%	CPB (100%)	MHCA/DHCA /Sequential aortic clamping/Reattachment of intercostal arteries/ cold crystalloid perfusion/ selective visceral perfusion/ CSFD
Gaudino et al	The Journal of Thoracic and Cardiovascular Surgery, 2015	1997-2014	57	67.2	57/100%	CS (82.4%)/ PCB(15.7%)/ CPB+DHCA (1.7%)	Cold crystalloid perfusion/ selective visceral perfusion/ Reattachment of intercostal arteries/CSFD
Conrad et al.	Ann Thorac Surg,2007	1997-2005	455	71.1	52/ (11.4%)	CS+DAP (92%)	CSFD/ Reattachment of intercostal arteries
Rigberg et al	J Vasc Surgery,2006	1991-2002	1010	72	213/ (21%)	NR	NR
Cowan et al	J Vasc Surgery,2003	1988-1998	321	71.5	321/ (100%)	NR	NR
Dardik et al.	J Vasc Surgery,2002	1992-2001	257	66	17(6.6%)	LSHB / MHCA	/CSFD/sequential clamping/ reattachment of intercostal arteries
Cina' et al	Ann Vasc Surg,2002	1990-2001	121	69	22/ (18.1%)	CS+ Gott shunt/LSHB	CSFD/sequential clamping/ reattachment of intercostal arteries
Bradbury et al	Eur J Vasc Endovasc Surg,1999	1983-1996	23	71.3	23/ (100%)	NR	NR
Ross et al	The Journal of Thoracic and Cardiovascular Surgery, 1998	1987-1997	132	NR	21/15.9%	CS (100%)	Reattachment of intercostal arteries
Grabitz	J Vasc Surgery,1996		260	63	38(14.6%)	CS (100%)	Reattachment of intercostal arteries, SSEP&MEP
Svensson et al (Safi H.J)	J Vasc Surgery,1993	1960-1991	1679	66	61/(4.0%)	CS(83%)/ LSHB(17%)	Reattachment of intercostal arteries
Cox et al.	J Vasc Surgery,1992	1966-1991	129	66	24(75%)	CS	CSFD /Cold crystalloid perfusion/selective visceral perfusion/Reattachment of intercostal arteries

Table 1: Overall, 16 studies were included in the meta-analysis after application of inclusion and exclusion criteria.

CPB: cardio-pulmonary bypass, PCPB: Partial cardio-pulmonary bypass,LSHB: left-side heart bypass, CS: clamp and sew, PLSFFB: partial left sided femoro-femoro bypass,DHCA: Deep hypotermic cardiocirculatory arrest, MHCA: Moderate hypotermic cardiocircylatory arrest, CSFD: Cerebro-spinal fluid drainage, ICAs: Intercostal arteries,MEP/SSEP: motor-evocated potentials/somato-sensory evocated potentials.

MATERIALS AND METHODS

We used the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. Medical databases included Medline, Scopus, EMBASE, Google Scholar, Ovid and the Cochrane Library were investigated, while we also searched manually the reference lists of the eligible articles for additional articles. Keyword algorithm included "thoracoabdominal" AND "thoraco-abdominal" OR "aortic aneurysms" OR "open repair" OR "ruptured." Our main interest focused in studies, published in English, reporting mortality rates after open repair of ruptured thoracoabdominal aneurysms.

A thorough search of the English-language literature published until January 2019 was performed to identify studies relative to ruptured TAAAs.

Studies were included in the review if they:

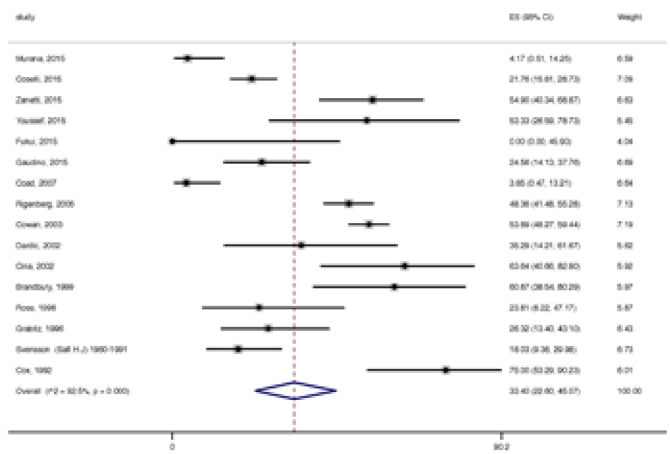
- Provided detailed data on open repair of TAAAs;
- Presented data of the in-hospital mortality
- Described >20 patients
- Were published in the English language
- Articles were excluded if they:
- Reported non ruptured TAAAs;
- Reported data for hybrid or endovascular reconstructions;
- Referred to treatment of infected TAAAs;

Two authors (GK, CNA) independently extracted and analyzed data and the final decision was reached by consensus. Data extracted from the eligible studies, included first author's name, study year, type of study, total number of patients with rTAA, mean age, number of deaths, complications (if described). We thereafter calculated mortality rates in patients with rTAA (number of deaths / total number of patients) for each eligible study. We expressed the rates as proportions and 95% confidence intervals (95% CIs). We transformed the values into quantities according to the Freeman-Tukey variant of the arcsine square root transformed proportion and the pooled mortality was calculated as the back-transformation of the weighted mean of the transformed proportion, using DerSimonian-Laird weights of random effects model and expressed as % proportion.

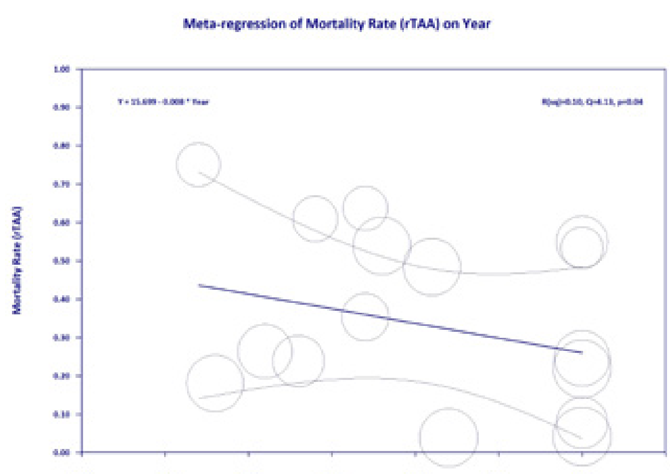
We also performed a second analysis deriving pooled mortality rates after excluding three studies with very low mortality rates (<15%; sensitivity analysis). A methodological quality assessment of the selected studies and analysis of heterogeneity and publication bias was performed. A meta-regression analysis with mortality rates over time as a covariate was performed in order to explore potential time trend upon mortality rates.

RESULTS

Our literature review identified a total of 16 articles 2-17 to be included in the meta-analysis after application of inclusion and exclusion criteria (Table 1). A total of 1,036 patients with ruptured aneurysms were included in the present analysis. An Egger's regression analysis did not reveal significant publication bias (int = 3.36, p=0.07) among the eligible studies. Pooled mortality rate after open repair of rTAAs from all 16 studies was 33.40% (95%CI=22.60-45.07; I2=92.5%, p<0.001; Figure 1).



Furthermore, we found a statistically significant decreasing trend in mortality rates over time (Q=4.13, p<0.04; Figure 2).



We performed a meta-regression analysis with total number of ruptured only cases per study as a covariate, which did not reveal significant association with mortality rate. However, a meta-regression analysis with total number of patients per study (elective and ruptured) revealed a significant reverse association between mortality rate and caseload (Q=4.13, p=0.04). This is a potential indicator that busiest departments dealing with larger number of elective and ruptured TAAs presented with better results in terms of mortality after rTAA repair.

Sensitivity analysis evidenced a pooled mortality rate of 41.73% (95%CI=31.68 – 52.13, I^2=88.8%, p<0.001), after exclusion of three studies reporting mortality rates <15%.[3,7,9] A second sensitivity analysis after exclusion of the study by Cowan et.al. showed a pooled mortality rate of 31.83% (95%CI=20.68-44.06, I^2=90.9%, p<0.001) from the remaining 15 studies.

When we pooled data only from four studies reporting data exclusively on rTAA, the pooled mortality rate was 47.77% (95% CI=32.71-63.04, I^2=84.4%, p<0.001).[2,5,8,13] Additionally, when we considered morbidity data from only these four studies, pooled cardiac event rate was 8.79% (95% CI=1.55-20.20, I^2=85.81%, p<0.001; data from all four studies), pooled permanent paraplegia was estimated at 5.56% (95%CI=1.76-10.96)[5,8] pooled stroke rate was 1.61% (95%CI=0.01-6.67; I^2=43.29%, p=0.17)[5,8] and reintervention rate was 8.33% (95%CI=2.86-15.82) [5,8] In the paper by Cowan et al. acute renal failure (28.0%) and cardiac-related events (18.1%) were the most common complications. More than half (51.2%) of all deaths occurred within 24 hours after surgery.

DISCUSSION

We reviewed 1,036 patients, with ruptured aneurysms, and we found an overall in-hospital mortality rate of 33.4% independently of the type of aneurysm. Interestingly, when we analyzed the data from four studies reporting exclusively the outcomes on ruptured TAAs, the pooled mortality rate was 47.77%.[2,5,8,13] The study of Cowan et al using national data from the Nationwide Inpatient Sample, reported the largest number of patients with 321 ruptured aneurysms and found an overall "real-world" mortality rate of 53.8%.[2] Regarding the morbidity associated with the technique, data from the four studies reporting the outcome exclusively for ruptured aneurysms, showed that the cardiac event rate was 8.8%, the permanent paraplegia was estimated at 5.6%, pooled stroke rate was 1.6% and re-intervention rate was 8.3% [2,5,8,13]

We found a relatively high heterogeneity between the analyzed studies. This can be explained by whether the rupture was contained or free and by the presence of haemodynamic instability. Although it is known that contained ruptures have a better outcome compared to free ruptures,[17] unfortunately, no specific data categorizing the patients according to the haemodynamic status were provided by the eligible studies. In addition, the heterogeneity can also be explained by the diversity in surgeon's team case load and familiarity with the procedure among the studies but it was also related to the institution's experience in managing the perioperative care of these challenging cases.[2] A trend of lower mortality in high-volume centers was shown in our study. Coselli et al reported a 21.5% mortality rate while Svensson et al reported a 18% mortality occurrence.[4,16]

A recent metanalysis showed that the pooled mortality rate among patients with unruptured TAAAs treated electively was 11.3%.[1] Our study provides evidence that the mortality for open reconstruction in rTAAs is 3 to 5-fold higher than the mortality in unruptured aneurysms. Previous studies have questioned whether repair is worthwhile in all ruptured cases. It has been suggested that patients in shock with a Crawford type II aneurysm have a poor prognosis and that intervention should be offered only in the most favorable of these cases.[13] Patients with types III or IV contained rupture who are not shocked on presentation might have a better outcome, especially if transferred in experienced centers. Validation of prognostic scoring systems for ruptured TAAs is lacking. However, clinical experience has shown that free rupture, patient's overall deteriorated functional status, advanced age > 76 years, preoperative cardiac arrest and loss of consciousness are independent predictors of high risk of death.[2,5,8,13] It should also considered that patients presenting with impending rupture, represent a high-risk subgroup, having probably denied elective repair because of associated comorbidities.[18] In the paper by Cox et al. none of the patients with free rupture or with preoperative hypotension or cardiac arrest survived.[17] Other studies showed that age > 77 years, female gender and preoperative renal dysfunction were also independent risk factors for increased mortality.[2,8]

Regarding the complications rate in patients with rTAAAs, more than half (51.7%) presented with at least one postoperative complication.[2] Acute renal failure of 28.0% and cardiac-related events of 18.1% were the most common complications in patients who survived from open surgery.[2] More than half (51.2%) of all deaths occurred within 24 hours after surgery. Uncontrolled bleeding from dilutional coagulopathy caused by massive blood loss and multiple organ failure were the most common causes of death. [2,5,8,13]

Interestingly, we found a statistically significant decreasing trend in mortality rates over time. This finding can be explained by the evolution and improvements in surgical technique over time and the use of adjunct measures for organ protection such as distal aortic perfusion with left heart bypass, cerebrospinal fluid drainage, and intercostal artery reimplantation. This study has limitations. Only a few studies reporting outcomes exclusively for rTAAAs have been reported in the literature. No detailed data for the type of aneurysm and the outcome were described in the majority of the studies. In addition, only a few studies specified complications for ruptured TAAs.Selection bias or referral patterns of ruptures may have skewed these results.

In conclusion, rupture is a lethal complication in untreated cases of TAAAs. It is associated with an approximately 50% mortality rate. Young and fit patients with contained rupture who are not shocked on presentation might have a better outcome, especially if transferred in experienced centers.

No conflict of interest.

REFERENCES

1 Moulakakis KG, Karaolanis G, Antonopoulos CN, Kakisis J, Klonaris C, Preventza O, et al. Open repair of thoracoabdominal aortic aneurysms in experienced centers. J Vasc Surg. 2018;68:634-645

2 Cowan JA Jr, Dimick JB, Wainess RM, Henke PK, Stanley JC, Upchurch GR Jr. Ruptured thoracoabdominal aortic aneurysm treatment in the United States: 1988 to 1998. J Vasc Surg. 2003;38:319-22

3 Murana G, Castrovinci S, Kloppenburg G, Yousif A, Kelder H, Schepens M, et al. Open thoracoabdominal aortic aneurysm repair in the modern era: results from a 20-year singlecentre experience. Eur J Cardiothorac Surg 2016;49:1374-81

4 Coselli JS, LeMaire SA, Preventza O, de la Cruz KI, Cooley DA, Price MD, et al. Outcomes of 3309 thoracoabdominal aortic aneurysm repairs. J Thorac Cardiovasc Surg 2016;151: 1323-37

5 Zanetti PP, Krasoń M, Walas R, Cebotaru T, Popa C, Vintila B, et al. "Open" repair of ruptured thoracoabdominal aortic aneurysm (experience of 51 cases). Kardiochir Torakochirurgia Pol. 2015;12:119-25

6 Youssef M, Neufang A, Jungmann F, Vahl CF, Dorweiler B. Patency of renal and visceral vessels after open thoracoabdominal aortic replacement. J Vasc Surg 2015;62: 594-9.

7 Fukui T, Takanashi S. Moderate to Deep Hypothermia in Patients Undergoing Thoracoabdominal Aortic Repair. Ann Vasc Surg. 2016;31:39-45

8 Gaudino M, Lau C, Munjal M, Girardi LN. Open repair of ruptured descending thoracic and thoracoabdominal aortic aneurysms. J Thorac Cardiovasc Surg. 2015;150:814-21

9 Conrad MF, Crawford RS, Davison JK, Cambria RP. Thoracoabdominal aneurysm repair: a 20-year perspective. Ann Thorac Surg 2007;83:S856-61

10 Rigberg DA, McGory ML, Zingmond DS, Maggard MA, Agustin M, Lawrence PF, et al. Thirty-day mortality statistics underestimate the risk of repair of thoracoabdominal aortic aneurysms: a statewide experience. J Vasc Surg. 2006;43:217-22

11 Dardik A, Krosnick T, Perler BA, Roseborough GS, Williams GM. Durability of thoracoabdominal aortic aneurysm repair in patients with connective tissue disorders. J Vasc Surg 2002;36:696-703

12 Cinà CS, Laganà A, Bruin G, Ricci C, Doobay B, Tittley J, et al. Thoracoabdominal aortic aneurysm repair: a prospective cohort study of 121 cases. Ann Vasc Surg 2002;16:631-8

13 Bradbury AW, Bulstrode NW, Gilling-Smith G, Stansby G, Mansfield AO, Wolfe JH. Repair of ruptured thoracoabdominal aortic aneurysm is worthwhile in selected cases. Eur J Vasc Endovasc Surg. 1999;17:160-5

14 Ross SD, Kron IL, Parrino PE, Shockey KS, Kern JA, Tribble CG. Preservation of intercostal arteries during thoracoabdominal aortic aneurysm surgery: a retrospective study. J Thorac Cardiovasc Surg 1999;118:17-25

15 Grabitz K, Sandmann W, Stühmeier K, Mainzer B, Godehardt E, Ohle B, et al. The risk of ischemic spinal cord injury in patients undergoing graft replacement for thoracoabdominal aortic aneurysms. J Vasc Surg 1996;23:230-40.

16 Svensson LG, Crawford ES, Hess KR, Coselli JS, Safi HJ. Experience with 1509 patients undergoing thoracoabdominal aortic operations. J Vasc Surg 1993;17:357-68

17 Cox GS, O'Hara PJ, Hertzer NR, Piedmonte MR, Krajewski LP, Beven EG. Thoracoabdominal aneurysm repair: a representative experience. J Vasc Surg. 1992 ;15:780-7

18 Cambria RP, Clouse WD, Davison JK, Dunn PF, Corey M, Dorer D. Thoracoabdominal aneurysm repair: results with 337 operations performed over a 15-year interval. Ann Surg. 2002;236:471-9

INVITED COMMENTARY

Open repair of thoraco-abdominal aneurysms

Igor Koncar[1,2], Lazar Davidovic[1,2]

[1]1Clinic for Vascular and Endovascular Surgery, Serbian Clinical Centre, Belgrade Serbia
[2]Belgrade Medical School, University of Belgrade, Serbia

In this issue of the HJVES Moulakakis et al.[1] reported systematic review and meta-analysis on open repair of ruptured thoraco-abdominal aortic aneurysms (RTHAA). They searched English-language literature published until January 2019 and 16 articles with 1036 treated patients with RTHAA were identified and included in analysis. They found that mortality rate is improving over decades and it was substantially lower in papers reporting results of treatment of all TAAA including elective and ruptured comparing to papers reporting only RTHAA (33% versus 47%).

The former group of papers had results that are comparable with mortality rate of open repair of ruptured abdominal aortic aneurysm (RAAA) which is, no matter how difficult still, far easier clinical condition in comparison to RTHAA[2].

Results are improving over the decades. It might be explained with centralization of care of patients with THAA. Cowan et al.[3] reported higher incidence of mortality in low volume centers and proposed centralization of THAA repair which ten years later probably proved to have half reduced mortality. In all twelve studies, from this systematic review, that are reporting data for both intact an ruptured THAA, rate of ruptured aneurysms was less than 10%.[3,4] Among others reporting only RTHAA, results from the literature are demonstrating that they are high volume centers as well with 660 patients in 20 years in Zanetti group and 675 in Gaudino group. Another reason for better results might be higher turndown rate. In 1999 report from Bradbury et all suggested that patients in shock and type II RTHAA have very poor prognosis so all treatment efforts are questionable while others should be transferred to high volume centers[5]. We might speculate that such a strategy has led to selection of those patients with better prognosis. Even the high volume centers are not reporting turndown rate in this group of patients. From experience in RAAA we know that turndown rate can be even 50%.[6]

Finally and most importantly surgical techniques changed and this is what has significantly improved results. Improved intraoperative organ perfusion, neurological monitoring and methods of preventions of spinal cord and kidney ischemia are nowadays milestones of successful open repair of THAA even more important in ruptured cases. Such a complex techniques and expensive technologies performed by well-educated and experienced physicians in multidisciplinary teams are possible only in centers of excellence.

Author for correspondence:

Lazar B. Davidovic

Clinic for Vascular and Endovascular Surgery, Clinical Center of Serbia, Koste Todorovića Street 8, 11000 Belgrade, Serbia
Tel: +38 1113065176, Fax: +38 1113065177
ISSN 1106-7237/ 2019 Hellenic Society of Vascular and Endovascular Surgery Published by Rotonda Publications

REFERENCE:

1 Moulakakis K, Antonopoulos C, Karaolanis G, , Kakisis J, Klonaris C, Lazaris A, et al Outcome of open Repair of Ruptured Thoracoabdominal Aortic Aneurysms. A Systematic Review and Meta-Analysis.HJVES 2019;2: 49-53

2 Marković M, Tomić I, Ilić N, Dragaš M, Končar I, Bukumirić Z, et al. The Rationale for Continuing Open Repair of Ruptured Abdominal Aortic Aneurysm. Ann Vasc Surg. 2016 Oct;36:64-73

3 Cowan JA Jr1, Dimick JB, Henke PK, Huber TS, Stanley JC, Upchurch GR Jr.Surgical treatment of intact thoracoabdominal aortic aneurysms in the United States: hospital and surgeon volume-related outcomes. J Vasc Surg. 2003 Jun;37(6):1169-74

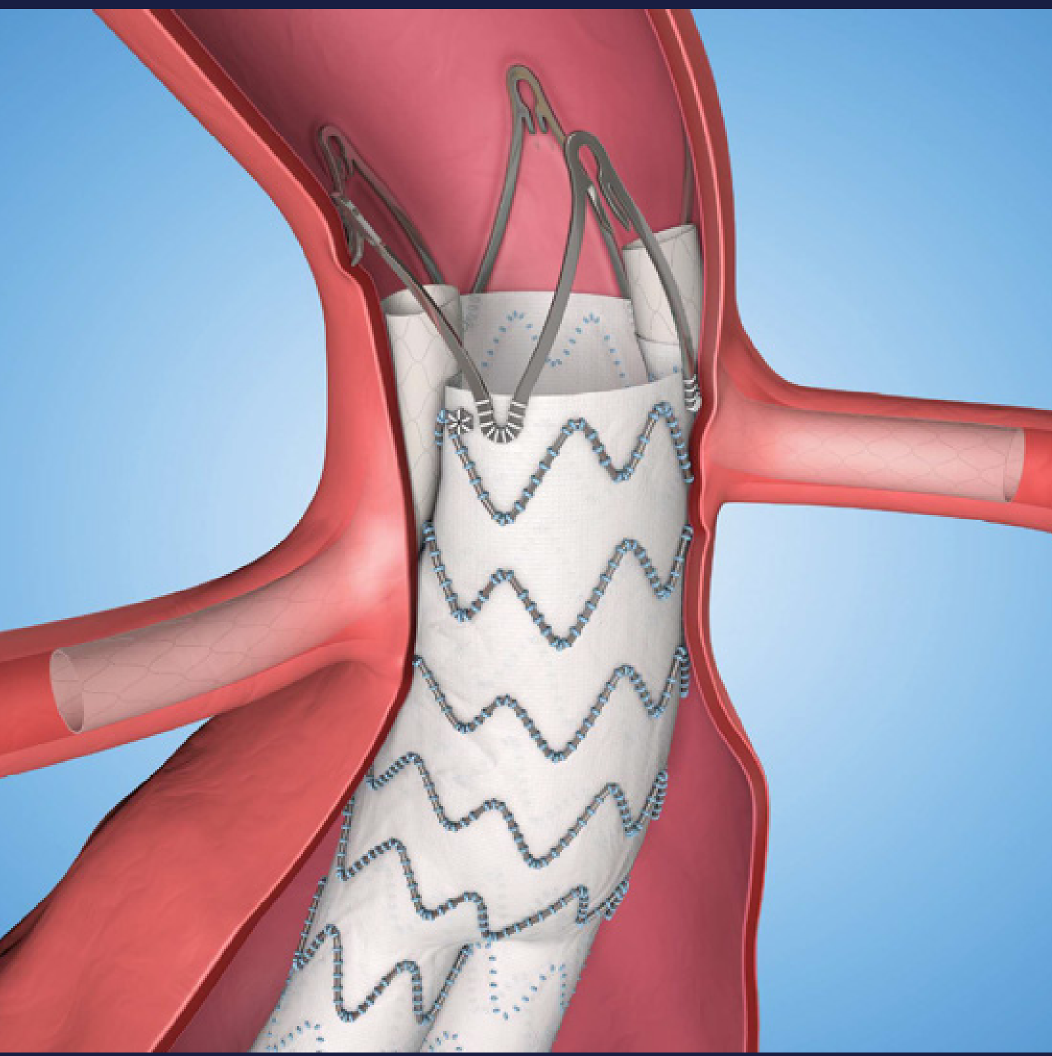
4 Bensley RP1, Curran T, Hurks R, Lo RC, Wyers MC, Hamdan AD, et al..Open repair of intact thoracoabdominal aortic aneurysms in the American College of Surgeons National Surgical Quality Improvement Program. J Vasc Surg. 2013 Oct;58(4):894-900

5 Bradbury AW, Bulstrode NW, Gilling-Smith G, Stansby G, Mansfield AO, Wolfe JH. Repair of Ruptured Thoracoabdominal Aortic Aneurysm is Worthwhile in Selected Cases Eur J Vasc Endovasc Surg 1999;17:160–5.

6 Park BD, Azefor N, Huang CC, Ricotta JJ. Trends in treatment of ruptured abdominal aortic aneurysm: impact of endovascular repair and implications for future care. J Am Coll Surg 2013;216:745-54

LEADING BY EXAMPLE

The first stent graft approved for use in ChEVAR



Endurant™ II/IIs

AAA Stent Graft System

Medtronic is the first and only company to provide a standardized, off-the-shelf alternative for juxtarenal with short neck aneurysm patients.

- **Indicated for patients with ≥2 mm neck length and ≤60° infrarenal angle**

medtronic.com/aortic

ChEVAR Indication not approved globally. Check local regulatory status.
For complete product information visit www.medtronic.com/manuals. Consult instructions for use at this website. Manuals can be viewed using a current version of any major internet browser. For best results, use Adobe Acrobat® Reader with the browser.

Medtronic
Further, Together

[1] Torsello et al. Safety and effectiveness of the INCRAFT® AAA Stent Graft for endovascular repair of Abdominal aortic aneurysms. JOURNAL OF VASCULAR SURGERY; January 2015, Volume 61, Number 1. Pages 1-8.

Ruptured abdominal aortic aneurysm repair: introducing a step-by-step protocol

Miltiadis Matsagkas[1], George Kouvelos[1], Konstantinos Spanos[1], Aikaterini Bouzia[2], Eleni Arnaoutoglou[2], Athanasios Giannoukas[1]

[1]Department of Vascular Surgery, University of Thessaly, Medical School, Larissa, Greece
[2]Department of Anesthesiology, University of Thessaly, Medical School, Larissa, Greece

INTRODUCTION

A controversy still exists around the world on the best strategy to treat ruptured abdominal aortic aneurysms (rAAA). For decades open repair has been considered the treatment of choice, though characterized by high and constant 30-day mortality despite improvements in intensive care unit (ICU)

Author for correspondence:
Miltiadis Matsagkas, MD, PhD, FEBVS
Department of Vascular Surgery, Medical School,
University of Thessaly, Larissa University Hospital,
Mezourlo, Larissa, Greece
Tel: +30 6945585876, E-mail: milmats@gmail.com
ISSN 1106-7237/ 2019 Hellenic Society of Vascular and Endovascular Surgery Published by Rotonda Publications

and anesthesiology care.[1] Nowadays EVAR is becoming increasingly popular in rAAA treatment. Both Society of Vascular Surgery (SVS) and European Society of Vascular Surgery guidelines recommend endovascular repair (EVAR) over open repair for a rAAA if anatomically suitable,.[2,3] In the largest meta- analysis so far, Van Beek et al by including 32 studies investigated the short term survival after EVAR or open repair for rAAA.[4] Based on the randomized controlled trials (RCTs) in defined populations 30-day or in-hospital survival were equal between EVAR and open repair, while based on observational studies with probably more selection of patients EVAR performed better than open repair.[4]

Hereby, based on the current literature and personal experience, we try to propose a step-by-step detailed protocol regarding patients with rAAA that arrive at dedicated Hospitals having the ability to perform both treatment modalities. The protocol algorithm is summarized in figure 1 and analyzed below.

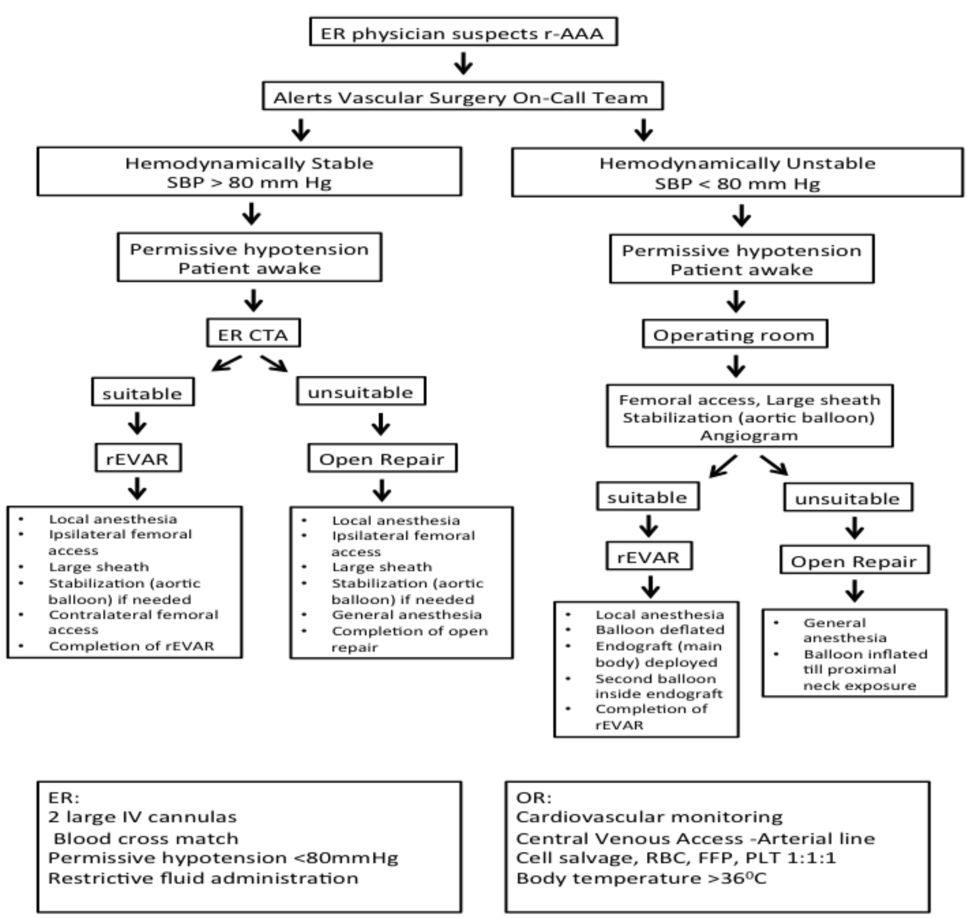


Figure 1. Summary of the protocol algorithm.

PROTOCOL

Emergency room

Information of a rAAA inside or outside the hospital should trigger an immediate alert to the vascular on-call team. This team should include at least two vascular surgeons, an anesthesiologist, OR staff and a radiology technician if available. Patients in the emergency room presenting with a rAAA should be evaluated according to hemodynamic stability (Systolic blood pressure>80 mmHg). The preoperative management of the patient should include securing intravenous (IV) access, two peripheral large bore cannulas, blood cross match and restricted resuscitation (permissive hypotension).[5-9] Central venous access and arterial line are not necessary at this stage and there should be no delay in emergency room (ER) in order to secure them. Intravenous fluids and vasoactive medication should be avoided as long as possible. The concept of permissive hypotension in which low systolic blood pressure (50-70 mm Hg) is accepted, to the extent that oxygen delivery to the brain (conscious patient) and the myocardium is maintained (no arrhythmias or chest pain), until hemorrhage control is achieved should be followed. This concept limits internal bleeding and associated loss of platelets and clotting factors. Systolic pressures of 50-70 mmHg are acceptable for a short period of time.

Stable patients should rapidly undergo a computed tomography angiography (CTA) to assess anatomical suitability for EVAR, while unstable patients should be transferred immediately to the operating room without a CTA. Conventional exclusion criteria for EVAR are used and include: (1) aortic neck diameter greater than 32 mm, neck length shorter than 10 mm, significant calcification (circumferential), thrombus (**>**40%), or aortic neck angulation greater than 60 degrees; and (2) iliac vessels less than 6 mm in diameter for delivery of the main body graft or with significant tortuosity.[10] Although the planning phase for cases of ruptured aneurysms is often under significant time restrictions, and perhaps with limited imaging quality available, it is important to make the most of the imaging and to spend a few extra minutes reviewing the possible difficulties of each case. A dedicated three dimension (3D) reconstruction software could aid in performing accurate length and diameter measurements and design properly the whole procedure. In any case, the experience of the whole team and the logistics of the hospital should be taken into consideration when the suitability for EVAR is decided. We should also keep in mind that the haemodynamic condition of the patient on presentation may influence CTA image of the proximal neck and, to avoid an intraoperative or late Type Ia endoleak, 30% oversizing maybe preferable when treating a rAAA assessed by CTA performed during permissive hypotension.[3]

To initiate and achieve a proper rEVAR programme it is very important to retain a wide stock of available endografts. It is logical that due to logistic reasons it is not possible to have all types of endografts, however an endograft with suprarenal fixation (28-36 mm diameter of main body) and several limbs can really cover nearly all rAAAs. It is important that the devices used for rAAAs should be the ones that the operator routinely uses for elective EVAR and with which the operating team has significant experience.[2,3]

Operating room

<u>*Stable patient with suitable anatomy*</u>: Stable patients with a suitable anatomy determined by CTA should undergo EVAR. Permissive hypotension is continued, and the patient is carefully monitored by the anesthesiology team (Monitor Anesthesia Care, MAC). The procedure should be performed under local anesthesia. Use of local anesthesia helps to avoid the hemodynamic changes associated with muscle relaxation and general anesthesia. Emergency EVAR may be performed under local anesthesia alone, but the anesthesiology team should be prepared to emergency conversion to general anesthesia if necessary. The EVAR procedure can be accomplished by the standard way. Systematic heparin administration could be deferred until the rupture is sealed. The uni-iliac configuration offers a fast and reliable solution, especially in inexperienced hands. However, bifurcated stent-grafts, as also stated in the recent ESVS guidelines[3], offer a more physiological solution with a shorter overall operation time in experienced hands, and obviate the need for a femoro-femoral bypass. By appropriately using occlusion balloons (described later), the hemodynamic stability of the patient can mostly be ensured during placement of a bifurcated stent graft without the need to convert to a uni-iliac repair. The main indication for using a uni-iliac system is in the setting of unilateral iliac occlusion or a severely compromised access that cannot be overcome.

<u>*Stable patient with unsuitable anatomy*</u>: In patients anatomically unsuitable for EVAR as determined by CTA, open repair should be performed. An aortic occlusion balloon could be also used in a similar manner (as described later), as an extra asset to preserve or restore intraoperative instability before exposing the proximal aortic neck. Induction of general anesthesia should be commenced after draping of the patient, in order not to affect patient hemodynamics during preparation. Central venous pressure and arterial line monitoring are suggested for all patients undergoing rAAA open repair. The lines should be secured before induction to general anesthesia, but without causing unnecessary delay.

<u>*Unstable patient*</u>: Unstable patients should immediately be transferred to the operating room without a CTA. To stabilize patients, which seems necessary in about 10-20% of them, aortic balloon occlusion can be used effectively. This technique was also used in open repair in the earlier years, when clamping proved difficult. The technique of balloon-occlusion in rEVAR involves the use of a large compliant balloon over a stiff guidewire, and inside a long (45cm in length), 12-14F sheath to retain correct position. With proper training, this technique can be applied in every unstable patient at the beginning of the procedure.

After femoral access under local anesthesia is achieved, we recommend placement of a large sheath (12-14 F, 45 cm) in the aorta over a stiff guidewire. The sheath is placed in the suprarenal position (at the level of 12T to L1 vertebra), to provide adequate passage and support for the occlusion balloon. Within this sheath in place, a large balloon for aortic occlusion is inserted and deployed above the renal arteries. After inflation of the aortic occlusion balloon, an angiogram can easily be performed through the same large sheath to assess for anatomic suitability for EVAR. This should allow to determine the eligibility for EVAR and make a definite choice between rEVAR under local anesthesia or OR under general anesthesia. The patient should be prepared for both types of anesthesia immediately after arriving in the OR.

During intraoperative angiography to determine EVAR-eligibility the vascular surgeon should take into account the angulation, shape and length of the proximal aortic neck. The length can be estimated with the use of a metric catheter. However we must not forget that the angiogram shows only the lumen and certain properties as the proximal aortic neck diameter, the presence of thrombus or a tapered neck cannot precisely be estimated. In every way in these cases the decision on the proper endograft relies on the selection and experience of the vascular surgeon.

In *unstable patients with anatomic suitability* for EVAR the endovascular procedure is continued under local anesthesia. The main body of the endograft is inserted from the contralateral of the balloon side and deployed just below the renal arteries. At this point, a repositioning of the occlusion balloon to a more proximal position may be needed in order to advance the proximal tip of the endograft's delivery system. The 12-14F sheath's position must be maintained well above the renal arteries until the main stent graft has been deployed, in order not only to secure the position of the inflated balloon, but also to facilitate the withdrawal of it after deflation, especially when endografts with suprarenal active fixation are used. When the main endograft has been deployed in the desired position, it's delivery system is withdrawn and a second aortic balloon is advanced inside the endograft and inflated just below the renal arteries and inside the main body of the endograft. The first balloon is then deflated, and removed through the 12-14F sheath, which then should also withdrawn in the aneurysm sac. Alternatively, if a second balloon is not available, the first balloon can be used in the main graft's ipsilateral side after withdrawal. Since an infrarenal occlusion has been achieved inside the main body of the endograft, catheterization of the contralateral gate and completion of the EVAR procedure could be performed, without time strain and additional blood loss.

In *unstable patients anatomically unsuitable for EVAR*, open repair should be attempted. Similar (as described above), the aortic occlusion balloon could act as an initial endo-clamping to preserve intraoperative stability during the induction of anesthesia and laparotomy. Such a maneuver also allows the proper dissection and exposure of the proximal aortic neck to achieve a safe proximal control. Afterwards the balloon could easily withdrawn to the level of the iliac artery (usually at the level of L4 vertebra) and used for an iliac endoclamping.

If the intraoperative hemoglobin level is <10 g/dL and blood loss is ongoing, transfusion of packed blood cells along with fresh frozen plasma and platelets in a ratio of 1:1:1 is recommended. Fibrinogen and prothrombin complex concentrates administration are recommended during massive transfusion, whereas rFVIIa should be reserved until all means have failed. In the operating room the patients' body temperature should be maintained above 36^{0}C using forced air warming blankets in order to avoid blood clotting dysfunction and metabolic acidosis.[11]

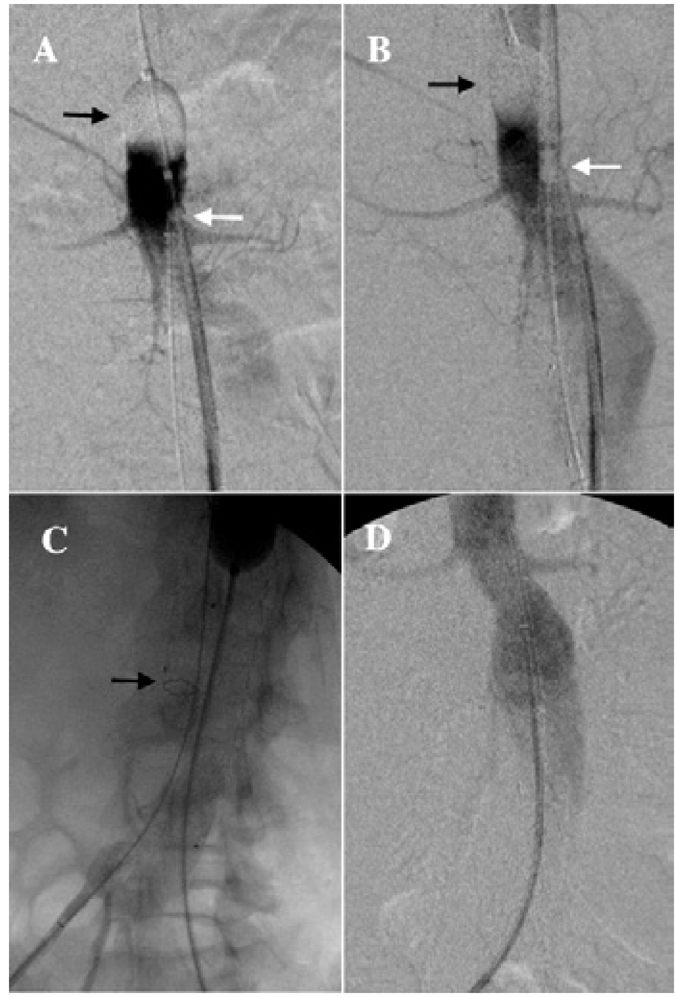


Figure 2. Fluroscopy image showing A. The deployment of the aortic balloon through a 14F sheath coming from the right groin (white arrow) and its inflation (black arrow) well above the renal arteries with the subsequent angiogram through an 8F seath coming fom the left groin.. B. The deployment of the main graft coming from the left just below the renal arteries (white arrow), with the occlusion balloon (black arrow) maintained above them. C. After withdrawal of the delivery system, an aortic balloon is advanced inside the endograft from the left and inflated inside the main body of the endograft, below the renal arteries. Subsequently, the cannulation of the contralateral gate is achieved (black arrow) , D. Final angiogram after the successful deployment of the bifurcated graft.

DISCUSSION

The primary goal of development of a rAAA protocol is to use EVAR as the initial treatment for rAAAs in most cases, which is the current recommendation of several vascular societies.[2,3] Hypotension or hemodynamic instability on presentation should not be considered a contraindication for rEVAR. A secondary aim of the protocol is through introducing a stepwise approach, to standardize the early patient's management from the time of presentation to the initiation of the procedure in the operating room, independently of the final treatment choice.

The establishment of such a protocol is not always easy and requires an on-call multidisciplinary team of various medical specialties and surgical staff, as well as the ability of each Hospital to perform adequately both treatment modalities in terms of personnel capabilities and logistics. Pre-hospital and emergency room personnel should be aware on the use of permissive hypotension, warming and intubation avoidance, factors that may avoid exacerbation of hemodynamic instability. Anesthesiologist should be informed to keep patients awake and apply permissive hypotension until an adequate control of bleeding is achieved. Those performing the procedure should be experienced in EVAR as well as in open repair of AAAs. An always-available inventory of various endografts, wires and catheters is also essential for implementation of a rEVAR programme.

Patient selection seems to play a significant role on outcome after rAAA treatment. In the IMPROVE study, crossover between the two treatment arms was common, showing that factors as hemodynamic instability and anatomic suitability are critical for the therapeutic decision.[12] Selection of patients for achieving favorable results for rEVAR can only be accomplished with the establishment of a standardized protocol. Some studies suggested that an EVAR-driven protocol for treating rAAAs is associated with an improved outcome not only for the EVAR treated patients, but as well as for those treated by OR.[13-14] Moore et al. by assessing 126 patients with rAAA demonstrated that a predefined strategy that includes rEVAR was associated with improved mortality.[15] These results are in accordance with those published by Mehta et al. who showed that emergent EVAR of hemodynamically stable and unstable patients was associated with a reduction in mortality (from 51% to 18%) once a standardized protocol was established.[16] Both studies were conducted in high-volume centers with highly experienced vascular teams.

CONCLUSION

With a standardized team approach, hemodynamically stable and unstable patients with rAAA can be treated by endovascular means. Unstable patients with rAAA may be particularly benefited by EVAR and should not be excluded from repair. Successful implementation of a systematic protocol relies on a devoted experienced team of surgeons, anesthesiologists, nurses, and surgical staff, coordination of pre-hospital and emergency room care, and an adequate stent graft inventory.

REFERENCES

1 Hoornweg LL, Storm-Versloot MN, Ubbink DT, Koelemay MJ, Legemate DA, Balm R. Meta analysis on mortality of ruptured abdominal aortic aneurysms. Eur J Vasc Endovasc Surg. 2008;35:558-70

2 Chaikof EL, Dalman RL, Eskandari MK, Jackson BM, Lee WA, Mansour MA, et al. The Society for Vascular Surgery practice guidelines on the care of patients with an abdominal aortic aneurysm. J Vasc Surg. 2018;67:2-77.e2

3 Wanhainen A, Verzini F, Van Herzeele I, Allaire E, Bown M, Cohnert T, et al. European Society for Vascular Surgery (ESVS) 2019 Clinical Practice Guidelines on the Management of Abdominal Aorto iliac Artery Aneurysms. Eur J Vasc Endovasc Surg. 2018 Dec 5

4 van Beek SC, Conijn AP, Koelemay MJ, Balm R. Editor's Choice - Endovascular aneurysm repair versus open repair for patients with a ruptured abdominal aortic aneurysm: a systematic review and meta-analysis of short-term survival. Eur J Vasc Endovasc Surg. 2014;47:593-602

5 Ellard L, Djaiani G. Anaesthesia for vascular emergencies. Anaesthesia. 2013;68 :72-83

6 Kothandan H, Haw Chieh GL, Khan SA, Karthekeyan RB, Sharad SS. Anesthetic considerations for endovascular abdominal aortic aneurysm repair. Ann Card Anaesth. 2016;19:132-41

7 Mouton R, Rogers CA, Harris RA, Hinchliffe RJ. Local anaesthesia for endovascular repair of ruptured abdominal aortic aneurysm. Br J Surg. 2019;106:74-81

8 Lee CW, Bae M, Chung SW. General considerations of ruptured abdominal aortic aneurysm: ruptured abdominal aortic aneurysm. Korean J Thorac Cardiovasc Surg. 2015;48:1-6

9 Broos PP, Stokmans RA, Cuypers PW, van Sambeek MR, Teijink JA; ENGAGE Investigators. Effects of Anesthesia Type on Perioperative Outcome After Endovascular Aneurysm Repair. J Endovasc Ther. 2015;22:770-7

10 Ulug P, Sweeting MJ, von Allmen RS, Thompson SG, Powell JT; SWAN collaborators. Morphological suitability for endovascular repair, non-intervention rates, and operative mortality in women and men assessed for intact abdominal aortic aneurysm repair: systematic reviews with meta-analysis. Lancet. 2017 ;389:2482-2491

11 Chee YE, Liu SE, Irwin MG. Management of bleeding in vascular surgery. Br J Anaesth. 2016;117 :85-94

12 IMPROVE Trial Investigators, Powell JT, Sweeting MJ, Thompson MM, Ashleigh R, Bell R, Gomes M, Greenhalgh RM, et al. Endovascular or open repair strategy for ruptured abdominal aortic aneurysm: 30 day outcomes from IMPROVE randomised trial. BMJ 2014;348:f7661

13 Haveman JW, Karliczek A, Verhoeven EL, Tielliu IF, de Vos R, Zwaveling JH, et al. Results of streamlined regional ambulance transport and subsequent treatment of acute abdominal aortic aneurysms. Emerg Med J 2006;23:807-10

14 Kapma MR, Groen H, Oranen BI, van der Hilst CS, Tielliu IF, Zeebregts CJ, Emergency abdominal aortic aneurysm repair with a preferential endovascular strategy: mortality and cost-effectiveness analysis. J Endovasc Ther 2007;14:777-84

15 Moore R, Nutley M, Cina CS, Motamedi M, Faris P, Abuznadah W. Improved survival after introduction of an emergency endovascular therapy protocol for ruptured abdominal aortic aneurysms. J Vasc Surg 2007;45:443-50

16 Mehta M, Taggert J, Darling RC 3rd, Chang BB, Kreienberg PB, Paty PS, et al. Establishing a protocol for endovascular treatment of ruptured abdominal aortic aneurysms: outcomes of a prospective analysis. J Vasc Surg 2006;44:1-8

INVITED COMMENTARY

Ruptured Abdominal Aortic Aneurysm Treatment; it is time to change things

Konstantinos Papazoglou, Ioakim Giagtzidis

Vascular Surgery Unit, 5th Department of Surgery, Medical School, Aristotle University of Thessaloniki, Hippokratio Hospital, Thessaloniki, Greece

The treatment approach of Ruptured Abdominal Aortic Aneurysm (RAAA) still remains a controversial field in vascular surgery community mainly, because of the mortality it carries. We still cannot recognize the point of no return for each individual patient, although this is inevitable for some patients who arrive at the emergency department. And since mortality is always present and mostly unaffected, there will always be a challenging effort to improve treatment results.

As presented by Matsagkas et al.[1] the new approach with the endovascular treatment of the ruptured AAA offers several important advantages and is recommended from Vascular societies globally. Minimizing trauma, avoiding general anesthesia, and difficult dissections during aortic exposure as well as avoid clumping in the presence of retroperitoneal hematoma are some of the benefits that indicate possible improvement in survival following the new approach. Even more as we noticed with our experience during the last two decades, the evolution in the performance and efficiency of endovascular grafts has widened the anatomical applicability, safety and technical success rate of the procedure.[2] Low profile, suprarenal fixation with barbs and accurate deployment are important features that increase the chances of successful RAAA treatment.

Unfortunately, the multicenter randomized controlled trials conducted failed to prove the superiority of endovascular approach[3-5] which is observed in the results of experienced vascular units.[6] There are some explanations of this fact. First of all, as thoroughly described by the authors, a carefully designed protocol in a dedicated vascular center, concerning this new approach that is dependent on technology and proper equipment, is necessary as already shown in several centers.[7] As easy as it sounds, such an algorithm is quite difficult to be established, especially in a Greek Hospital which is usually understaffed and luck of technical recourses is a usual condition. Another very important aspect of the procedural success is the experience of the operator which is often limited concerning endovascular treatment. The latter, was one of the main reasons for selecting open treatment and crossover between the groups in many trial cases, leading to bias in patient selection. As described from a number of dedicated centers[8,9] and also the authors opinion, the absolute endovascular first approach in all RAAA cases, could be a better solution in order to reduce overall mortality, leaving open conversion and repair in case of EVAR complication and technical failure. This can be achieved with chimney renal reperfusion or with debranching of the abdominal branches after graft deployment in cases of short or no proximal neck. As already described, this group of patients, even with open surgery and suprarenal clumping, carry extremely high mortality.[10]

Author for correspondence:

Prof. Konstantinos Papazoglou

E-mail: kpapas@auth.gr
ISSN 1106-7237/ 2019 Hellenic Society of Vascular and Endovascular Surgery Published by Rotonda Publications

One of the most important developments in the diagnosis and treatment of AAA, open or endovascular in my opinion is the evolution of spiral computed tomography and the widespread use of CT angiography. I would consider a necessary step the 15 min. delay of the procedure in order to have a detailed anatomical picture of the aneurysm, the proximal neck and the iliac arteries. Having in mind the above information the vascular surgeon can perform the operation quicker and safer, whether he is planning open or "endo" approach. Even more with today's technology, CT scan images, can be sent online from a peripheral district hospital during patient transfer from another hospital in order to evaluate the aneurysm, plan the operation and organize delivery of the necessary endografts and other necessary equipment in case there are no available in OR storage. Mean time from rupture to death is almost 12 hours, so some extra minutes are not decisive in the vast majority of cases.[11] On the other hand though the interval from rupture to diagnosis, to treatment, which can take several hours is extremely important for the survival and care must be taken to provide quick diagnosis and transportation within an organized health care system.

As described from the authors, hypotensive resuscitation, in combination with coagulation control could be important preoperative medical support steps in order to reduce blood loss until the operation is carried out. Even more, graft evolution with newer generation low profile grafts and controlled suprarenal fixation are contributing today to higher operative success rates.

Finally as described according to the experience of the vascular surgeon proper use of local anesthesia, percutaneous approach, graft selection (Bifurcated/ Aorto-uniiliac) and balloon occlusion bleeding control are technique developments that can improve the results, but have to be evaluated thoroughly in the future. Another important factor which should be taken into consideration, in applying EVAR in RAAA, is the abdominal compartment syndrome which carries very high mortality. Again, early diagnosis, quick transfer and proper management of the patient, has to be included in the treatment protocol.

Concluding, it seems that endovascular RAAA treatment offers a unique opportunity to improve the results in this critical and urgent medical condition but in order for these results to widely appear, new trials incorporating the newer developments and experience on the field are needed and at the same time, establishment of a treatment algorithm in every vascular center is required.

REFERENCES

1 Matsagkas M, Kouvelos G, Spanos K, Bouzia A, Arnaoutoglou E, Giannoukas A. Ruptured abdominal aortic aneurysm repair: introducing a step by step protocol. HJVES 2019;2: 55-58

2 Papazoglou K, Mallios A, Rafati F, Zambas N, Karkos C. Endovascular treatment of ruptured abdominal aortic aneurysms with the Endurant device. Ann Vasc Surg 2013;27: 162-167

3 IMPROVE trial investigators, Powell JT, Sweeting MJ, Thompson MM, Asleigh R, Bell R, et.al. Endovascular or open repair strategy for ruptured abdominal aortic aneurysm: 30 days aoutcome of IMPROVE randomised trial BMJ 2014; 348:f7661

4 Reimerink JJ, Hoornweg LL, Vahl AC, Wisselink W, van den Brock TA, Legemate DA et al. Endovascular repair versus open repair of ruptured abdominal aortic aneurysms: a multicenter randomized controlled trial . Ann Surg 2013; 258: 248-256

5 Desgranges P, Kobeiter H, Katsabian S, Boufi M, Gouny P, Favre J-P et al. ECAR (Endovasculair Chirurgiedans les Anevrysmesaorto-iliaques Rompus): a French randomized controlled trial of endovascular vs. open surgical repair of ruptured aorto-iliac aneurysms. Eur J Vasc Endovasc Surg 2015; 50: 303-310

6 Van Beek SC, Conjin AP, Koelemay MJ, Balm R. Endovascular aneurysm repair versus open repair for patients with a ruptured abdominal aortic aneurysm: A systematic review and meta-analysis of short-term survival. Eur J Vasc Endovasc Surg 2014; 47: 593 -602

7 Anjum A, von Allmen R, Greenhalgh R, Powell JT. Explaining the decrease in mortality from abdominal aortic aneurysm rupture. Br J Surg 2012; 99:637-45

8 Mayer D, Aeschbacher S, Pfammatter T, Veith FJ, Norgren L, et al. Complete replacement of open repair for ruptured abdominal aortic aneurysms by endovascular aneurysm repair: a two center 14-year experience. Ann Surg 2012; 256: 688-695

9 Karthikesalingam A, Wanhainen A, Holt PJ, Vidal-Diez A, Brownrigg JRW, et al. Comparison of long-term mortality after ruptured abdominal aortic aneurysm in England and Sweden. BJS 2016; 103: 199-206

10 Veith FJ, Lachat M, Mayer D, Malina M, Holst J, et al. Collected world and single center experience with endovascular treatment of ruptured abdominal aortic aneurysms. Ann Surg 2009; 250: 818-824

11 Chaikof EL, Blankensteijn JD, Harris PL, White GH, Zarins CK, et al. Reporting standards for endovascular aneurysm repair. J VascSurg 2002; 35: 1048-1060.

Open repair of ruptured abdominal aortic aneurysm in the endovascular era

Lazar B. Davidovic[1,2], Igor B. Koncar[1,2]

[1]School of Medicine. University of Belgrade
[2]Clinic for Vascular and Endovascular surgery. Clinical Center of Serbia, Belgrade

Abstract:

Published literature is supporting endovascular repair (EVAR) of ruptured abdominal aortic aneurysm (RAAA) and in the recent years, papers about open repair (OR) of RAAA are scares. Does OR of RAAA has its' own place in endovascular era.

Substantial rate of RAAA are not anatomically suitable which confirms necessity to conquer techniques of open repair, even nowadays. Fast diagnosis and bleeding control followed by rational tactics and reconstruction strategies to perform safe procedure in optimal time are very important. Postoperative care and medical management of hemodynamic condition of the patient with thorough assessment of coagulation and timely diagnosis of any complication are important for improved survival. This paper presents some of the detailed technical tricks used in high volume RAAA center that has treated over 1500 patients during last 25 years.

Providing both options, would allow careful selection and probably optimal results. Lack of experience in elective procedures is devastating experience of new generation of vascular surgeons and therefore education of young vascular residents and surgeons in open aortic surgery is of an utmost importance.

INTRODUCTION

Published literature is supporting endovascular repair (EVAR) of ruptured abdominal aortic aneurysm (RAAA) and in the recent years, papers about open repair (OR) of RAAA are scarce. In the following text some literature data and our institutional protocol based on more than 1500 patients with RAAA operated by OR since 1992 will be presented. (Figure 1)

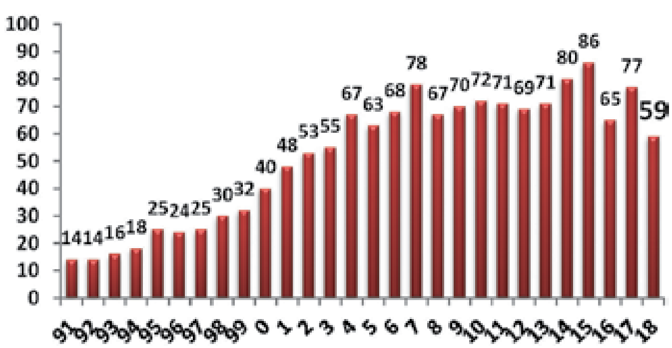


Figure 1. A repair of RAAA 1992-2018 at the Clinic for Vascular and Endovascular Surgery, Clinical Center of Serbia.

INDICATIONS FOR OPEN REPAIR OF RUPTURED AAA

As in elective cases EVAR has the same and very important advantages regarding treatment of ruptured AAA (REVAR): avoiding laparotomy, aortic cross clamping and general anesthesia, together with significantly lower blood loss[1]. However, multicenter randomized controlled trials showed relatively unexpected results[2-6]. The first of them is AJAX trial that has been performed in 10 hospitals in the Netherlands[2]. Out of total number of 520 RAAA 116 were randomized. According to this study, majority of severe complications were more frequent in open surgical group, but not significantly. Then, prolonged postoperative mechanical ventilation, perioperative blood loss and consumption of blood products have been significantly higher in open surgical group. However, there was no difference regarding the length of ICU and total hospital stay while mean time of OR was shorter than mean time of EVAR. Finally this trial did not show a significant difference regarding 30 day mortality between EVAR and OR of RAAA (21% in the EVAR and 25% in the OR group)[2]. An IMPROVE trial has randomized 613 (316 for EVAR and 297 for OR) RAAA from 30 centers[3-5]. That trial, did not also show significant difference regarding 30-day mortality (35% for EVAR and 37% for OR group), duration of procedure (The median length of the EVAR was 180 minutes, while 199 minutes for OR) and 30 day cost, between endovascular and open groups. At the same time in cases under local anesthesia EVAR has been associated with a lower mortality than those under general anesthesia. (Mortality in local anesthesia group was 13%, while 34% was in general anesthesia group)[3]. ECAR trial that has been performed in 14 hospitals in France between 2008 and 2013, randomized 107 out of 524 patients with RAAA[6]. In this study 30 day mortality after open and endovascular repair of RAAA was also equal (18% in the EVAR group versus 24% in the OR group)[6]. In AJAX and ECAR trials less than 25% of all patients with identified RAAA, were randomized[3,4]. According to IM-

Author for correspondence:

Lazar B. Davidovic

Clinic for Vascular and Endovascular Surgery, Clinical Center of Serbia, Koste Todorovića Street 8, 11000 Belgrade, Serbia
Tel: +38 1113065176, Fax: +38 1113065177
ISSN 1106-7237/ 2019 Hellenic Society of Vascular and Endovascular Surgery Published by Rotonda Publications

PROVE, trial 30-day mortality after REVAR at relatively hemodynamic stabile patients with good aortic anatomy was 25%. Still, this group represents only 60% of patients with RAAA[3]. Namely, patients who were not suitable for EVAR and those with severe hemodynamic instability have not been included. Consequently these trials are not representing real life conditions and it is difficult to follow them in clinical routine practice, even though randomized controlled trials are the best option to compare different methods and procedures in the era of evidence based medicine[7].

In the real life, more than 80% of hemodynamically unstable patients with RAAA, if not treated immediately upon admission, will die within two hours[6]. Prerequisites for EVAR are multidetector computed tomography (MDCT) examination, available endovascular team, stent grafts and material. In some countries and/or hospitals it is difficult or impossible to provide these conditions within two hours upon admission, and yet, without it EVAR is not possible while natural outcome of RAAA is fast. REVAR is associated with relatively significant incidence of abdominal compartment syndrome, which is followed by a mortality rate of 60%[9]. REVAR is also associated with high cumulative risk of secondary interventions during the follow up period[10]. There is no significant difference regarding long term survival and quality of life between open and endovascular repair of RAAA[11, 12]. All being said, OR of RAAA is still very important. But, can we improve early surviving? Yes actually, we can. In the past 26 years, we managed to decrease the 30-day mortality since more than 50% between 1991 and 2001, to 28% in the last two years [13-15].

TECHNICAL CONSIDERATIONS

During OR of RAAA we use modified Crawford`s strategy that includes fast diagnosis, permissive hypotension, non-selective supraceliac aortic cross clamping, cell saving and auto transfusion, as well as fast and simple aortic replacement[15].

Diagnosis

In unstable patients with abdominal or low back pain who have pulsatile abdominal tumor and profound shock, we perform emergency surgery after ultrasonography confirmation of RAAA. MDCT is performed prior to emergency surgery only to patients with suspected RAAA or extensive suprarenal and thoracoabdominal anerurysm or in hemodinamicaly stable patients especially when diagnostic dilemmas are present or endovascular solution is option due to comorbid conditions. Thanks to previous strategy we significantly reduced the mean time from arrival to emergency room to entering an operating suite, from more than two hours during the first time of our investigation, to just 43 minutes in the past 3 years[13-15]. Unfortunately we are not able to influence time since symptoms or first medical examination. In our country no helicopter transportation is routinely used for these patients. Our hospital is 24/7/365 aortic emergency referral center and all doctors in the country are informed which is saving time due to avoiding repetitive call to different hospitals. It is of interest to note that durndown rate in our experience is very low, less than 5%, and it is only considered in patients with malignant extensive diseases or old age with poor pre-rupture condition. Hemodinamic status is not reason for turndown in our clinical practice[15,16]. (Figure 2)

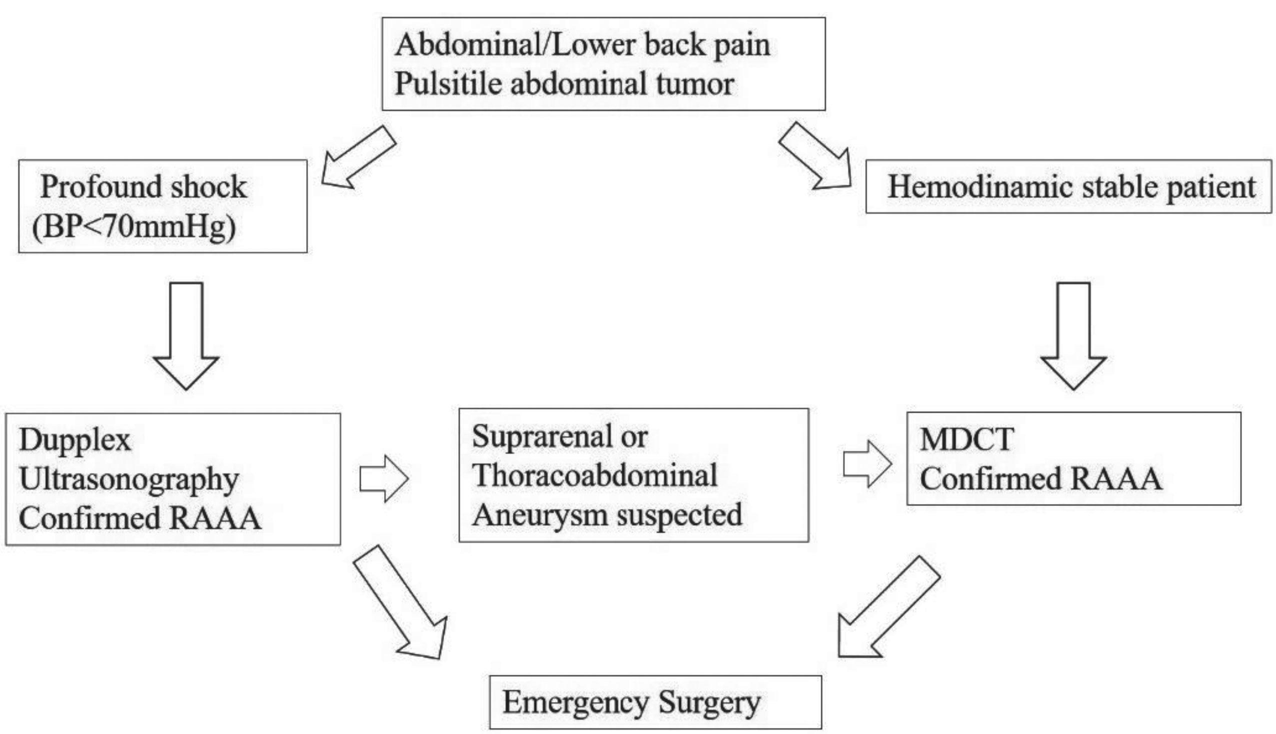


Figure 2. Diagnostic algorithm used in our settings prior to open repair of ruptured abdominal aortic aneurysms.

Permissive Hypotension

One of the biggest mistakes in the initial RAAA treatment, both during transport and upon admission, is an aggressive restitution of circulatory volume. It increases arterial pressure that, in addition, annuls the initial retroperitoneum tamponed and leads to new bleeding with conversion of retroperitoneal rupture into intraperitoneal one. Crawford was the first to insist on "permissive hypotension"[16]. Volume should be compensated to a level required to maintain consciousness and to prevent ST depression[17].

Approach

A trans-peritoneal approach through a long midline incision is the mostly used during OR of RAAA. This approach is more comfortable for anesthetists, especially if patients are hemodynamically unstable, or even if they require reanimation[17-20]. This approach enables easier exploration of the abdominal cavity, as well as dissection of the iliac and femoral arteries, especially on the right side. The patient is positioned supine on the operating table. The operative field is prepped and draped from the nipples to the knees before introduction of general anesthesia with consequent relaxation (that can reduce intrabdominal pressure due to relaxation of muscles and promote further bleeding). Immediately after intubation follows a midline incision, made from xyphoid (it can be excised if necessary) to the pubis[20,21]. In patients with RAAA and extensive proximal pathology towards suprarenal or even thoracoabdominal individual strategy is made based on patient condition, MDCT findings and surgeons' and his team experience.

Bleeding Control

We perform OR of RAAA routinely under supraceliac aortic cross clamping. That is a fast, efficient and safe proximal bleeding control, which, in addition, enables to prevent iatrogenic injuries in the presence of huge retroperitoneal hematoma. An experienced vascular surgeon needs less than 10 minutes - from the initiation of laparotomy to supraceliac aortic cross clamping[13-15]. This procedure begins with resection of the left triangular ligament and retracting the left lobe of the liver to the right. Then, the gastro hepatic omentum is opened to allow entry into the laser sac. The nasogastric tube is used to identify the esophagus and proximal part of the stomach, which are retracted to the left. The final step, before aortic cross clamping, is splitting or resecting of the crura of the diaphragm[18-21]. One should be advised that, during this procedure, the first assistant retracts esophagus and stomach downward to the left. Excessive retraction during supra celiac aortic cross clamping might cause spleen injury. In most cases, spleen repair is unsuccessful and requires early re-intervention, due to prolonged hemorrhage. Because of that, we always perform splenectomy in such cases.

The removing and relocation of the proximal clamp from initial supraceliac to infrarenal position, is not recommendable, however in cases of convenient anatomy this can be done in selected cases. Namely infrarenal aortic cross clamping requires additional dissection through retroperitoneal hematoma that increases risk of iatrogenic injury of duodenum, aorta, inferior vena cava, etc. In our last article we have found that supraceliac aortic cross clamping longer than 35 minutes increased an early mortality[13-15]. In cases of prolonged supraceliac or suprarenal aortic cross clamping, we recommend renal protection using cold renoplegic solution (500 ml NaCl, 5000 IU Heparin, 125mg Urbazon, 30 ml 20% Manitol). Initially, 250ml of this solution is administered into each kidney, with the procedure being repeated if the kidney circulation is not established after 30 minutes.

In the presence of large retroperitoneal hematoma the dissection of iliac arteries should be also avoided to prevent iatrogenic injuries of the ureters and iliac veins[13-15]. Instead that the distal bleeding control can be performed, by placement of balloon occlusive catheters into both iliac arteries after opening of the aneurysm sac. (Figure 3)

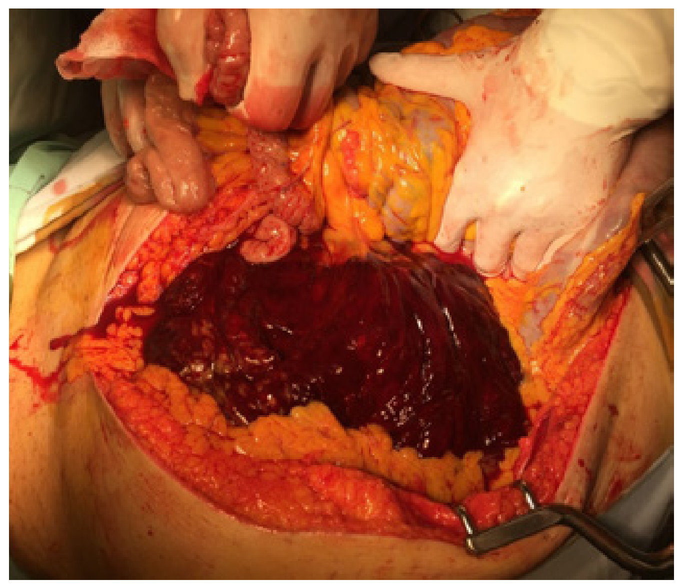


Figure 3. A huge retroperitoneal hematoma caused by AAA rupture which makes it difficult or even impossible the dissection of the infra- and juxta-renal aorta as well as iliac arteries.

The Opening of the Aneurysm Sac

After proximal and distal clamping an omentum and transverse colon are retracted cephalad, while the small bowel is packed in the right hemi-abdomen. If more working space is necessary, the small bowel can be temporary eviscerated out of the abdominal cavity. In this case adequate protection with either warm moist towels or sterile plastic bags is necessary[13,14]. The aneurysm sack is opened longitudinally. The course of that incision is important. Namely in cases of ruptured AAA where anatomical landmarks are not perfectly clear, it is important to open the aneurysm to the left (side of the patient) to avoid injury to the duodenum. The opening of the aneurysm sac is followed by removal of the thrombus and by suture of patent's inferior mesenteric (IMA) and lumbar arteries[18-21]. The usage of self-retaining retractor placed in the aneurysm sac allows ligature of lumbar arteries, as well as suture of both proximal and distal anastomosis.

Inferior mesenteric artery

According to our experience during OR of RAAA, IMA should be ligated. That ligature has to be done at IMA origin from the aneurysm sac to preserve left colic artery [18,19] (Figure 4).

Figure 4. The ligature of the inferior mesenteric artery at origin from the aneurysm sac to preserve left colic artery.

Aortic Repair

Vascular reconstruction during OR of RAAA should be performed in the simplest method possible. The usage of bifurcated graft can increase mortality[2-4], but that was not confirmed by our last study[13-15]. Anyway it is important to keep at least one of the hypogastric arteries patent to prevent colonic ischemia.

At patients with cardiac diseases, de-clamping may lead to myocardial infarction or cardiac insufficiency. Bearing this in mind, it is apparent that cooperation with anesthetist is extremely important. Namely, prior to decamping, the volume should be substituted optimally to avoid hypotension and to prevent hypo-perfusion of brain and kidneys[18-21].

An evacuation of retroperitoneal hematoma

A development of ACS should be avoided by careful manual evacuation of retroperitoneal hematoma. That is followed by separate drainage of an abdominal cavity and retroperitoneal space.

Cell Saving and Auto-transfusion

The intraoperative cell saving and auto-transfusion are obligatory during OR of RAAA. According to our and some other studies the intraoperative cell saving with auto-transfusion reduces significantly the 30-day mortality after OR of RAAA[22-24].

Postoperative complications

Postoperative complications after repair of ruptured abdominal aortic aneurysm might not fit in few paragraphs. These complications are mostly the cause of mortality in these patients since intraoperative death incidence is low[15]. One of the most severe complications is abdominal compartment syndrome and colon ischemia. In our algorithm intraabdominal pressure is followed routinely after RAAA repair while colonoscopy is performed in patients suspected for colon ischemia. In case of obvious signs of acute abdomen explorative laparotomy is preferred. Routine colonoscopy might be option for timely diagnosis however these patients should be followed thoroughly since colon ischemia might occur any time in the early postoperative time[25].

CONCLUSIONS

Besides well-known advantages associated with endovascular repair, multicenter randomized controlled trials did not find significant difference regarding 30-day mortality between open and endovascular repair of ruptured abdominal aortic aneurysm. Endovascular repair offers improved survival when it is anatomically feasible and when haemodinamic condition of the patient allows. Providing both options, in high volume center, would allow careful selection and probably optimal results. Lack of experience in elective procedures is devastating experience of new generation and therefore education of young vascular residents and surgeons in open aortic surgery is of an utmost importance.

REFERENCES

1 Veith F, Powel JT, Hinchliffe RJ. Is a randomized trial necessary to determine whether endovascular repair is the preferred management strategy in patients with ruptured abdominal aortic aneurysms? J Vasc Surg 2010; 52: 1087-93

2 Reimerink JJ, Hoornweg LL, Vahl AC, et al. Endovascular Repair Versus Open Repair of Ruptured Abdominal Aortic Aneurysms: A Multicenter Randomized Controlled Trial. Ann Surg 2013; 258(2): 248-258

3 IMPROVE trial investigators. Observations from the IMPROVE trial concerning the clinical care of patients with ruptured abdominal aortic aneurysm. Br J Surg 2014; 101: 216–224

4 Sweeting MJ1, Ulug P2, Powell JT3, Desgranges P4, Balm R5; Ruptured Aneurysm Trialists Ruptured Aneurysm Trials: The Importance of Longer-term Outcomes and Meta-analysis for 1-year Mortality. Eur J Vasc Endovasc Surg. 2015 Sep;50(3):297-302

5 Sweeting MJ1, Balm R2, Desgranges P3, Ulug P4, Powell JT4; Ruptured Aneurysm Trialists Individual-patient meta-analysis of three randomized trials comparing endovascular versus open repair for ruptured abdominal aortic aneurysm. Br J Surg. 2015 Sep;102(10):1229-39

6 Desgranges P, Kobeiter Katsahian HS, et al. ECAR (Endovasculaire ou Chirurgie dans les Anévrysmes aorto-ili-

aques Rompus): A French Randomized Controlled Trial of Endovascular Versus Open Surgical Repair of Ruptured Aorto-iliac Aneurysms. Eur J Vasc Endovasc Surg 2015; 50:303-310

7 Naylor AR. Trans-Atlantic Debate: Does endovascular repair offer a survival advantage over open repair for ruptured abdominal aortic aneurysms? Eur J Vasc Endovasc Surg 2015, 49: 116-128

8 Lloyd GM, Bown MJ, Norwood GA et al. Feasibility of preoperative computer tomography in patients with ruptured abdominal aortic aneurysms: A time-to-death study in patients without operation. J Vasc Surg 2004; 39:788-91

9 Mehta M, Paty PSK, Byrne J, et al. The impact of hemodynamic status on outcomes of endovascular abdominal aortic aneurysm repair for rupture. J Vasc Surg 2013; 57:1255-60

10 Hechelhammer L, Lachat ML, Wildermuth S, et al. Midterm outcome of endovascular repair of ruptured abdominal aortic aneurysms. J Vasc Surg 2005; 41:752-7

11 Eefting D, Ultee KHJ, Von Mejienefeldt GCI, et al. Ruptured AAA: state of the art management. J Cardiovasc Surg 2013; 54 (Suppl.1, No.1):47-53

12 Kapma MR, Dijksman LM, Reimerink JJ, et al. Cost-effectiveness and cost-utility of endovascular versus open repair of ruptured abdominal aortic aneurysm in the Amsterdam acute aneurysm trial. Br J Surg 2014; 101: 208–215

13 Marković M, Davidović L, Maksimović Ž, et al. Ruptured Abdominal Aortic Aneurysm. Predictors of Survival in 229 Consecutive Surgical Patients. HERZ 2004; 29(1):123-9.

14 Davidović L, Marković M, Kostić D, et al. Ruptured Abdominal Aortic Aneurysms: Factors. Influencing Early Survival. Ann Vasc Surg 2005; 19 (1):29-3

15 Markovic M, Tomic I, Ilic N, et al. The Rationale for Continuing Open Repair of Ruptured Abdominal Aortic Aneurysm. Ann Vasc Surg 2016; 36:64-73

16 Crawford SE. Ruptured abdominal aortic aneurysms: An Edidtorial. J Vasc Surg 1991; 13:348

17 Roberts K, Revell M, Youssef H, et al. Hypotensive resuscitation in patients with ruptured abdominal aortic aneurysms. Eur J Vasc Endovasc Surg 2006; 31:339-44

18 Chaikof EL, Brewster DC, Dalman RL, et al. The care of patients with an abdominal aorticaneurysm: The Society for Vascular Surgery practice guidelines. J Vasc Surg 2009; 50 (8S)

19 Wanhainen A, Verzini F, Van Herzeele I, et al. European Society for Vascular Surgery (ESVS) 2019 Clinical Practice Guidelines on the Management of Abdominal Aorto-iliac Artery Aneurysms. Eur J Vasc Endovasc Surg 2019, -, 1-97

20 Woo EY, Damrauer SM. Abdominal aortic aneurysms: Open surgical treatment. In: Cronenwett Jl, Johnston KW, editors. Rutheford`s Vascular Surgery 8th Edition. Philadelphia: Saunders Elsevier; 2014. p. 2024-2045

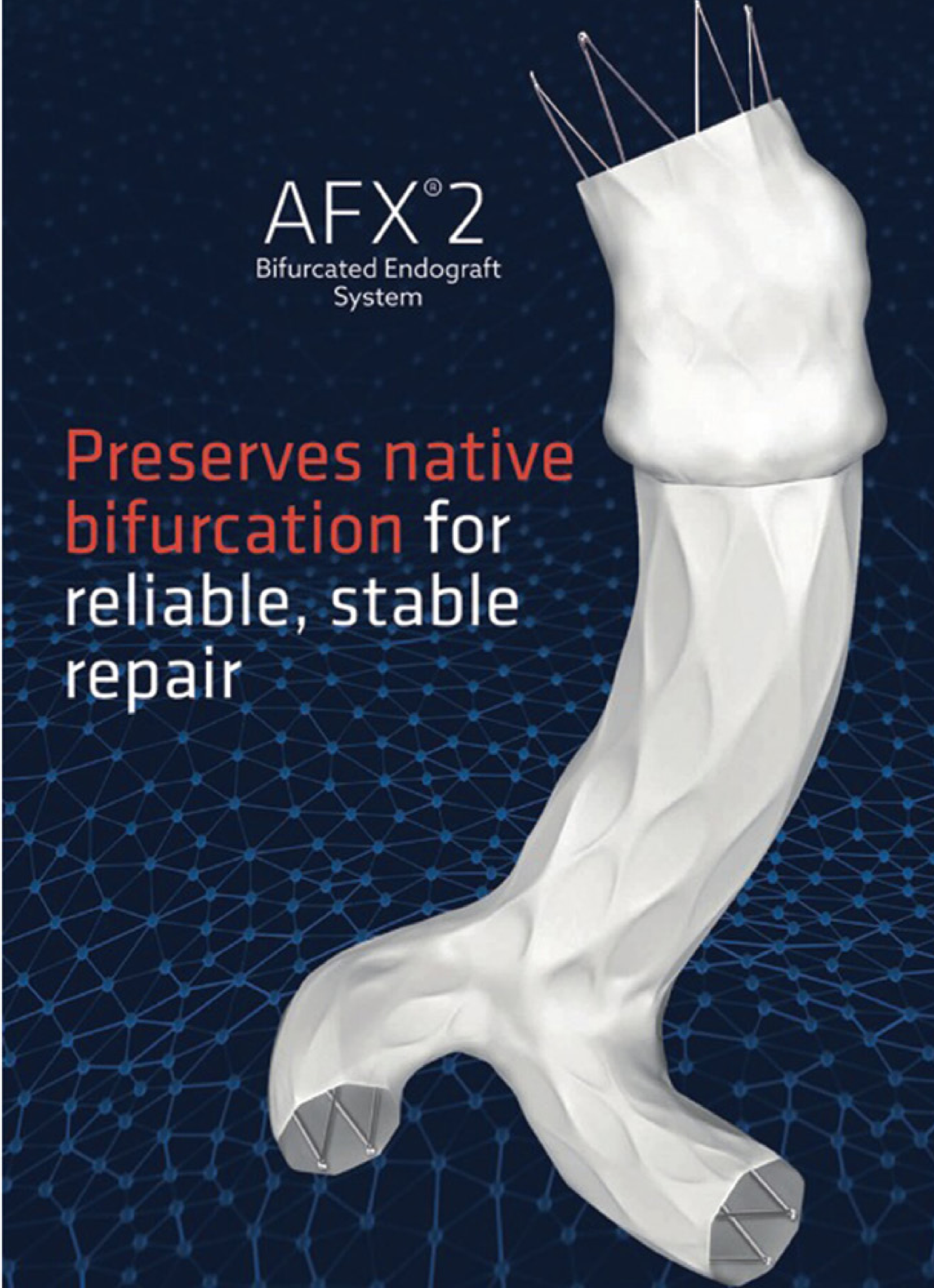
21 Davidovic L Open Repair for infrarenal AAA. In: Francesco Speziale (ed). Management of Abdominal Aortic Aneurysms. Edizioni Minerva Medica Turin-Italy 2016, ISBN: 878-88-7711-883-7. pp 16-27

22 Davidovic L, Dragas M, Cvetkovic S, et al. Twenty years of experience in the treatment of spontaneous aorto-venous fistulas in a developing country. World J Surg 2011; 35(8):1829-34

23 Markovic M, Davidovic L, Savic N, et al. Intraoperative Cell Salvage versus Allogeneic Transfusion during Abdominal Aortic Surgery: Clinical and Financial Outcomes. Vascular 2009; 17(2): 83-92

24 Shantikumar S, Patel S, Handa A. The role of cell salvage autotransfusion in abdominal aortic aneurysm surgery. Eur J Vasc Endovasc Surg 2011, 42:577-584

25 von Meijenfeldt GCI, Vainas T, Mistriotis GA, Gans SL, Zeebregts CJ, van der Laan MJ. Accuracy of Routine Endoscopy Diagnosing Colonic Ischaemia After Abdominal Aortic Aneurysm Repair: A Meta-analysis. Eur J Vasc Endovasc Surg. 2018 Jul;56(1):22-3


AFX®2
Bifurcated Endograft
System
Preserves native bifurcation for reliable, stable repair

Initial Experience with Chimney EVAR for the Treatment of Para- and Juxta-Renal Aneurysms

Miltiadis Matsagkas[1],Petroula Nana[1], Georgios Kouvelos[1], Konstantinos Spanos[1], Aikaterini Bouzia[2], Konstantinos Mpatzalexis[1], Eleni Arnaoutoglou[2], Athanasios Giannoukas[1]

[1]Department of Vascular Surgery, Faculty of Medicine, School of Health Sciences, University of Thessaly, Larissa, Greece
[2]Department of Anesthesiology, Faculty of Medicine, School of Health Sciences, University of Thessaly, Larissa, Greece

Abstract:

Introduction: Endovascular aortic aneurysm repair using parallel grafts (chimney technique, ChEVAR) is an off-the-shelf alternative technique in patients with challenging aortic anatomy when fenestrated or branched devices are contraindicated or unavailable. This study reports the outcomes of ChEVAR for para-renal abdominal aortic aneurysm (AAA) treatment in a single center.

Methods: This is a retrospective analysis of prospectively collected data. All patients suffering from a pararenal AAA and treated with ChEVAR between May 2016 and February 2019, wereincluded.Proximal landing zones precluded any standard endovascular intervention and all patients were considered as high risk for open aortic surgery. As main outcomes technical success, endoleak type Ia, overall mortality, freedom from target vessel occlusion,as well as reinterventions were recorded and analyzed.

Results: Thirty patients (28 males; mean age 72 years) underwent Ch-EVAR.In 23 cases, patients were primarily treated while 7 patients had a previous aortic procedure. Target vessels (TVs) included 51 renal and 11 superior mesenteric arteries. 11 patients received three chimneys, 10 patients two chimneys and 9 patients one chimney. The median pre-operative proximal neck length increased from3mm (range, 0-8mm), to 24.5 mm (range, 18-34 mm)using the chimney technique. Technical success was 100%. Thirty-day mortality was 10% (3/30), whileno early re-intervention was needed. During follow-up (range, 1-30 months), the survival rate was calculated at 73% (SE 9.9%), TVs primary patency rate at 95% (SE 5%) and freedom from chimney graft-related re-interventions was 94.7% (SE 5.1%) at 24 months. In three cases, a gutter endoleak was detected on the initial CTA and spontaneously resolved in all patients. In 2 cases, a type Ia endoleak was detected (93.3% at 24 months, SE 4.6%). These patients are under close surveillance (2nd and 3rd post-operative month respectively).

Conclusion: The chimney techniqueallowsthe endovascular treatment of para-renal aneurysms according to each patient's specific anatomy. It seems a feasible and safe option at least during the early follow-up period. Despite the minimal invasive nature of the procedure, these patients remain fragile and need a meticulous perioperative care.

INTRODUCTION

Nowadays, the numerous advanced endovascular treatment techniques offer a minimal approach in patients with complex aortic aneurysm anatomy.[1] Chimney technique (chimney EVAR, ChEVAR) using parallel grafts is an endovascular approach that hasincreased its popularity during last decade.[2] ChEVAR is an off-the shelf technique in the treatment of pararenal abdominal aortic aneurysms (AAA), initially used in emergent cases or as a bailout technique. Furthermore, when proximal or distal anatomy rules out a treatment with fenestrated or branched devices, ChEVAR is an alternative management in complex aneurysms.[3,4]

Author for correspondence:

Miltiadis Matsagkas, MD, PhD, FEBVS

Department of Vascular Surgery, Medical School,
University of Thessaly, Larissa University Hospital,
Mezourlo, Larissa, Greece
Tel: +30 6945585876, E-mail: milmats@gmail. com
ISSN 1106-7237/ 2019 Hellenic Society of Vascular and Endovascular Surgery Published by Rotonda Publications

Despite the wider application andincreasing experience in the use of ChEVAR in complex aortic aneurysm treatment, there are persistingdilemmas concerning overall technical success, endoleak Ia and chimney stent patency, as well as long-term ChEVAR's durability and safety.[2] In clinical aspects, occurrence of adverse events during the early follow-up is associated with renal impairment or fatal superior mesenteric artery occlusion.[4]

The aim of this study was to report the early-outcomes of ChEVAR for the treatment of para-renal AAAs at a single tertiary vascular center.

METHODS

Study Cohort

From May 2016 to February 2019, 30 consecutive high-risk patients (28 men; mean age 72 years) with AAAs extending to or involving renal arteries were treated using the chimney technique. All patients had proximal landing zones precluding any standard endovascular intervention and were at high risk for open aortic surgery.

All cases were electively treated. In cases where FEVAR or branched devices were indicated, patients' decision to deny a long waiting of 4 to 8 months for the fenestrated or branched endograft was considered. A preoperative CTA of the thoracic and abdominal aorta down to femoral arteries was performed in all patients. During preoperative planning, CTAs were assessed using center lumen line measurements for both the aorta and the TVs, usingdedicatedsoftware (3Mensio Medical Imaging BV, Bilthoven, Netherlands). Patients' demographics, preoperative and postoperative anatomic parameters, perioperative characteristics and outcome data were collected prospectively. Informed consent was obtained from all patients. The study was approved by the Institutional Review Board.

Periprocedural characteristics

Intraoperative management

All operations were realized under general anesthesia. After the insertion of the sheaths, 50-100IU/kg of unfractionated heparin were administrated to the patient. After the first operative hour, activated clotting time (ACT) was calculated and repeated each 30 minutes. In the case that ACT target (200-300%) was not achieved, a further bolus administration of heparin was demanded (50IU/kg). Cerebral oximetry was applied in all cases as a standard of care.

Access

In all cases, bifemoral access was used for the insertion of the main endograft. Concerning the parallel grafts, in cases of one chimney a left brachial access was preferred with percutaneous puncture under ultrasound guidance, of the peripheral brachial artery. Left axillary artery was dissected and two parallel sheaths were inserted when two chimneys were applied. Right axillary artery was additionally used in cases of three chimneys. In all cases, a complete CTA of the aortic vessels, thoracic, abdominal aorta and iliac arteries was preoperatively demaned and the anatomy of the upper and lower access was carefully evaluated and taken into consideration.

Type of stents

A balloon expandable covered stent was preferred in all cases where patient's anatomy permitted a successful stenting with a maximal stent length up to 57 mm. In any other case, whenlonger stents were demanded, a self-expanding covered stent was used. Relining with self-expanding stents was applied according to surgeon's preference or in cases where an inadequate angulation of the inserted stent was detected in the intra-operative angiography.

Antiplatelet therapy

Double antiplatelet therapy, clopidogrel 75mg and aspirin 100mg,was initiated in all patientsat least for the first post-operative month, except those under anticoagulant regiment with DOACs or VKA antagonist, where single antiplatelet treatment was preferred. The duration of the double antithrombotic therapy was reevaluated in 1st and 6th month of follow-up according to patient's characteristics and surgeon's preferences.

Follow-up

A standardized follow-up protocol including CTA of the abdominal aorta and iliac arteries and laboratory testing was performed between 2nd and 30th post-op day, at 12 months and yearly, thereafter. Duplex ultrasonography with plain x-rays was used as standard follow-up method at 6-month follow-up, as well as in cases of uncomplicated CTA at discharge, at 1st month. In any other case, a CTA was demanded.

Outcomes

As main outcomes technical success, presence of endoleak type Ia, overall mortality, freedom from target vessel occlusion, as well as reinterventions were recorded and analyzed. Endoleak type Ia was considered as any high flow endoleak provoked by an inadequate proximal sealing between the main graft and the aortic wall. On the other hand, as gutter endoleaks were considered all low flow endoleaks created between the main and the parallel grafts.

Statistical Analysis

Continuous data were reported as a mean ± standard deviation. Categorical data were expressed as absolute numbers and percentage of prevalence (%) in the study cohort. In the statistical analysisfor continuous variables the independent t-test for normally distributed data and the Mann-Whitney U test for nonparametric data were used. The Pearson x2 test or the Fisher exact test was used for categorical variables, as appropriate. Survival times were initially compared among groups with the log-rank test and Kaplan- Meier curves were generated. P value was considered significant when it was <0. 05. Statistical analysis was performed by SPSS 22. 0 for Windows software (IBM Corp, Armonk, NY).

RESULTS

Patients' demographics and anatomic characteristics

A total of 30 patients (28 men, age 72 years, range 68-81 years old) underwent chimney procedures. In 23 cases, chimney technique was used as primary treatment (Image 1, 2) while in 7 patients a previous aortic procedure has been recorded (Image 3). One patient underwent previous open repair for AAA and was re-operated with ChEVAR for a para-anastomotic aneurysm. In the remaining 6 cases, a previous failed EVAR with endoleak type Ia was the indication of treatment. The preoperative aneurysm diameter was calculated at 67 mm (range 51-91mm) with a preoperative proximal neck length at 3mm (range, 0-8mm)while the neck length using the chimney technique changed to 24. 5 mm (range, 18-34 mm).

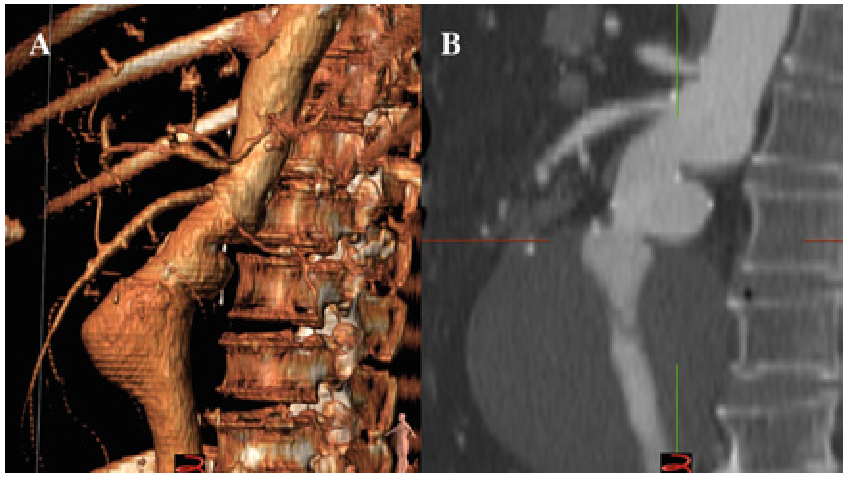


Image 1. Preoperative CTA: 8cm pararenal AAA with complex proximal neck anatomy (Panel A, 3D reconstruction, Panel B, Longitudinal axis)

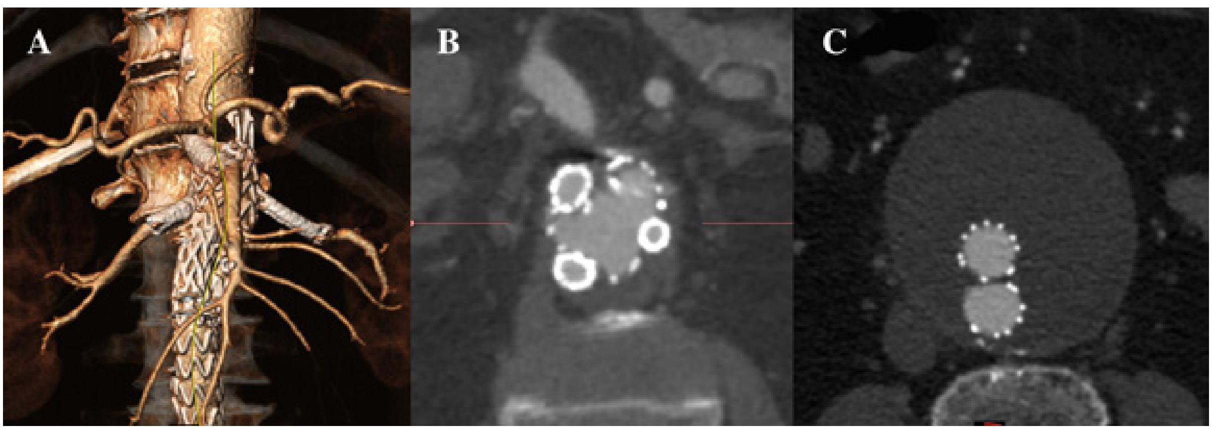


Image 2. Post-operative CTA: The patient presented in Image 1 was treated with a triple chimney. All grafts are patent (Panel A & B). The post-operative CTA revealed no endoleak (Panel C).

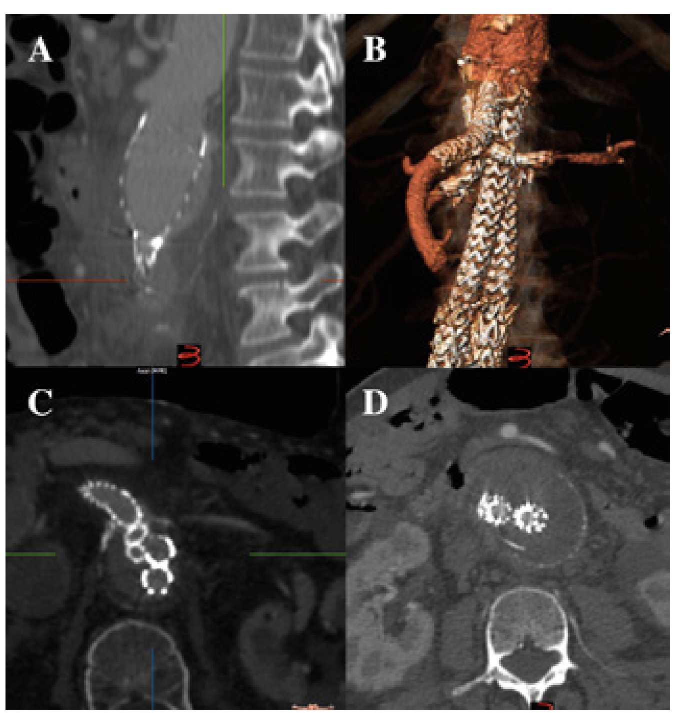


Image 3. Preoperative CTA of a patient with failed EVAR (Panel A, EndoleakIa). The patient was treated with a triple chimney and a Nellix device (Panel B & C). ChEVAR resolved the endoleak as presented in the post-operative CTA (Panel D).

Perioperative characteristics

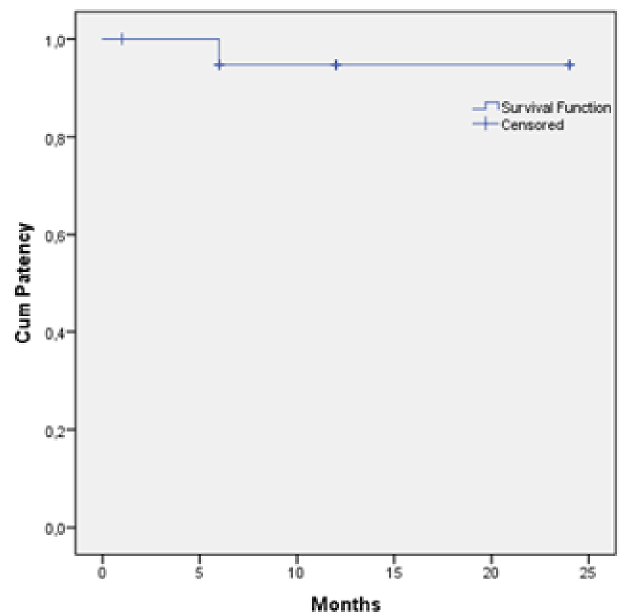
Target vessels were51renal (RA) and 11superior mesenteric arteries (SMA). 11 patients treated usingthree chimneys, 10 patients two chimneys and 9 patients one chimney. 18 patients received an Endurant endograft (Medtronic Inc, Minneapolis, MN), 9 patients a Nellix sealing device (Endologix-Inc, Irvine, CA),2 patients an Incraft device (Cordis, Cardinal Health, Dublin, UK) and 1 patient a Bolton Relay endograft (Vascutek, Terumo Aortic, Glasgow, UK). The decision for the endograft used was according to patient's specific anatomic characteristics and surgeon's preference. The oversizing of the main aortic graft was varied between 23% and 30% (mean: 26,7%). Totally, we used for primary stenting 11 covered balloon expandable stents for the SMA and 49 for the RAs (14 BeGraft,Bentley Innomed,GE, 7 Atrium V12, Maquet SAS, FR, 28 LifeStream, C. R. Bard, USA), and 2 self-expanding covered stents for the RA (Viahban, W. L. Gore, USA) with a range between 5 and 10mm. Additionally, we used for relining self-expanding covered stents, 6 for the SMA and 15 for the RA (E-luminex, C. R. Bard, USA). Completion angiography showed no type Ia endoleak in any of the patients at the end of the procedure. Technical success was 100%.

Median perioperative time was calculated at 214 minutes (range 180-360 minutes) while the median radiation exposure time was 43. 5 minutes (range 30-102 minutes). The median contrast volume used was 200ml (range 120-300ml). Blood loss was within acceptable limits as median transfusion volume was 535ml (2RBC/patient). After the procedure, patients were usually transferred directly to the ward. However, some patients had to be admitted to the ICU, usually due to pre-existing comorbidities. For these patients, median stay at the Intensive Care Unit (ICU) was 1day (range 0-10 days).

The overall 30-day mortality was 10% (3/30). An aneurysm related death was recorded due to severe systemic inflammatory response syndrome (SIRS). The patient expressed an extensive SIRS, a few hours after the end of the operation (an Endurant endograft was used in this case), admitted immediately in the ICU, but unfortunately, he never recovered and eventually died10 days later due to multi-organ failure. All 3 chimneys remained patent without any endoleak at 6th postoperative day when he underwent a CTA. Two patients (an 80-year-old and a 57-year-old male patient) died due to myocardial infarction on the 2nd and the 5th post-operative day, respectively. Both suffered from severe coronary artery disease, even though with preserved EF preoperatively. During hospitalization, 3 myocardial infarctions (2 fatal) and 2 strokes were recorded. One patient suffered a left ischemic stroke due to embolization probably from the aortic arch. 3 target vessels were stented in this patient, via both axillary arteries. A second patient suffered a stroke associated with visual blurriness and mild instability due to embolization from the vertebral artery. Left renal artery was the target vessel in this patient. Left axillary artery was used the catheterization.

Kaplan-Meier estimation forTVprimary patency rate was 95% at24months (SE 5%) (Figure 1). One renal stent occlusion was detected at 6th month follow-up. The patient remained asymptomatic with a creatinine at 1. 7mg/dl during follow-up. After extended discussion with him, he refused any further intervention. He is under nephrological surveillance. Two target vessel stenoses were diagnosed during the 1st month follow-up, concerning 1 renal and 1 superior mesenteric artery. The SMA stenosis was treated successfully by endovascular means during the 2nd post-operative month using a balloon expandable stent (Image 4).

Figure 1



Months	0	1	6	12	24
Patients at risk	30	29	18	12	3
Events	0	0	1	1	1
Percentage			95%		95%
SE			5%		5%

Figure 1: The cumulative patency rate was 95% at 24 months according to Kaplan Meier

The patient with the renal stenosis was treated conservatively ashe remained asymptomatic. A mild renal impairment was identified with amaximum post-stenosis creatinine at 1. 7mg/dl with a preserved GFR >60ml/kg/1. 73m^2 and urine output (creatinine baseline at 0. 9). Threegutter endoleaks werediagnosed with CTA before patient's discharge. All of themresolved spontaneouslyduring the 1st month. Furthermore, at the initial CTA, type Ia endoleaks were identified in 2 patients treated with triple chimney and they are still under surveillance (2nd and 3rd month respectively). The cumulative endoleak type Ia and freedom from chimney graft-related reinterventions rate was 93. 3% (SE 4. 6%) and 94. 7 % (SE 5. 1%) at 24 months, respectively.

Three patients died during the follow-up period and the overall survival rate during follow-up was calculated at 73%at 24 months (SE 9. 9%, Figure 2). A patient died the 6th post-operative month due to pneumonia. This patient suffered a stroke with left hemiparesis the 2nd post-operative day (1 of the 2 patients that suffered a post-operative stroke). An em-

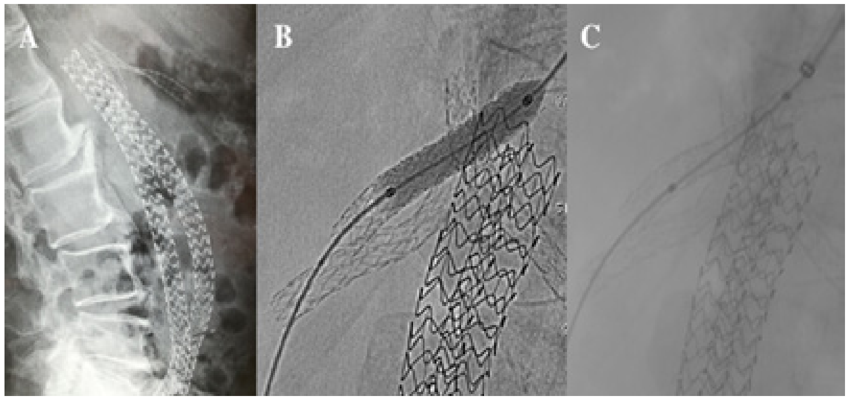
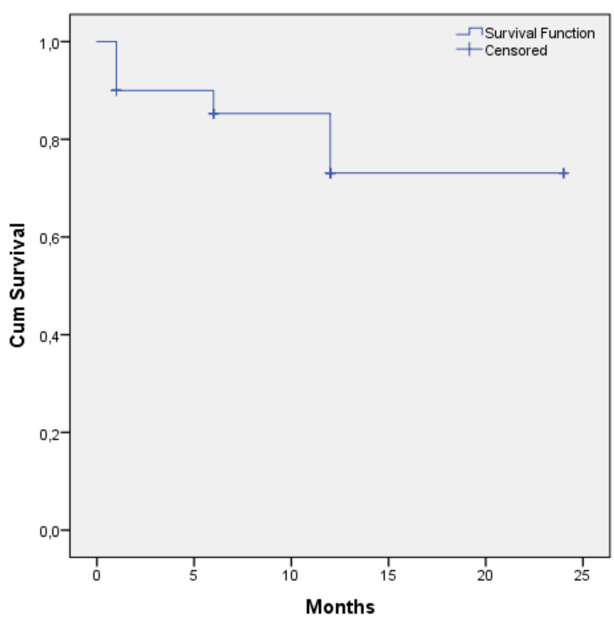


Image 4. Plain X-rays revealed a post-operative SMA stenosis in a patient treated with ChEVAR (Panel A). The patient was treated with a balloon expandable stent (Panel B & C).

bolic etiology is the most possible diagnosis, as the patient had a mild aortic arch atheromatosis and both axial artery accesses were used during a triple-chimney procedure. The second patient suffered a fatal myocardial infarction the 5th post-operative month. He had a medical history significant for coronary artery disease. Another patient died two years after the procedure because of alcoholic cirrhosis. No late aneurysm related death was recorded.

Figure 2



Months	0	1	6	12	24
Patients at risk	30	27	18	12	3
Events	0	3	4	6	6
Percentage		90%	85. 3%	73%	73%
SE		5. 5%	6. 9%	9. 9%	9. 9%

The cumulative survival rate was 73% at 24 months according to Kaplan-Meier

DISCUSSION

Endovascular repair of pararenal abdominal aortic aneurysms has gain popularity as an attractive alternative to open repair.[5] ChEVARincreases the eligibility of endovascular aneurysm repair in complex aneurysm anatomy with acceptable mortality rates and less morbidity than open repair.[6] ChEVAR seems a safe option, with comparable morbidity and mortality to FEVAR, especially in emergent cases or when custom-made grafts are unavailable or contraindicated.[5] In high volume centers, early mortality is as low as 1. 17% for the chimney technique and 0. 58% for fenestrated EVAR.[5]

In this study technical success was 100%. Along this line, in PERICLES registry and PROTAGORAS study, the intraoperatively technical success rate was 97. 1% and 100%, respectively.[2,7] With more than 500 patients treated with the chimney technique and 898 parallel grafts implanted in target vessels in PERICLES registry, ChEVAR seems a feasible option in pararenal aneurysm repair.[2] Despite of the encouraging perioperative results, long-term durability and stent patency is an oft-debated issue in these cases. During median follow-up, patency achieves acceptable rates, more than 95%.[8] Similarly, in our study the cumulative TV primary patency rate was 95% at24 months (SE 5%).

TVs graft occlusion seems a rare condition, mostly occurring the early post-operative period, within the first 4 months in the current literature.[4] In most renal cases, it is a silent complication, associated with mild or reversible renal impairment. Otherwise, superior mesenteric artery occlusion could be a lethal event, demanding urgent laparotomy.[4] In this study, one renal occlusionseen at 6 months associated with mild renal injurywas treated conservatively. During the follow-up, freedom from reinterventions was 94. 7%at 24 months (SE 5. 1%), which consort with the current literature.[9, 10]

Type Ia endoleak is characterized as the Achilles's heel of the chimney technique. Excessive or insufficient main stent-graft oversizing as well as an inadequate seal zone length (<20mm) are the main factors associated with high flow Ia endoleak in ChEVAR.[11] This classification of endoleak type Ia according to its mechanism may instigate a more appropriate management, as in these cases a further intervention is obliged.[11] Lindblad et al report an incidence of 13%endoleak type Ia in patients treated with parallel grafts in abdominal aortic aneurysms during early follow-up.[12] In this study, two patients were diagnosed with type Ia endoleak and they are under close surveillance (2nd and 3rd post-operative month respectively). In both cases, an inadequate proximal seal zone (<20mm) is recorded. The cumulative freedom from endoleak type Ia was 93. 3% at 24 months (SE 4. 6%)

On the other hand, the challenging issue regarding the inevitable formation of "gutters" between the parallel and main grafts provides more uncertainty regarding the technical and clinical role of these type Ia endoleak. 13Gutter endoleaks are physically low-flow endoleaks, not associated with sac enlargement, with a tendency to disappear during early follow-up. 13Only a few patients, presenting this probably benign type of endoleak, seem to require reintervention. 13In PERICLES registry, the persistence rate of gutter endoleaks at 17. 1 months follow-up was eliminated at 2. 9%. 11 Also, in this study, all gutter-related type Ia endoleaks (3/3) were resolved spontaneously within first month. 13. 3% of patients presented a gutter endoleak. With the wider use of the chimney technique, more technical aspects, as stent graft position and angulation, are investigated, promising an amelioration of the current results. 14More studies are needed to identify the role of gutter endoleaks, as in the current literature they are generally described and included in type Ia endoleaks, despite their different natural history and clinical impact.

Chimney technique is a readily dispensable solution for the treatment of not only intact pararenal aneurysms but also type I endoleaks in failed EVAR.[15] Especially in this series, two Nellix devices were used in previous failed EVAR. No complication was recorded in technical or clinical aspects. Nellix device seems to offer a good adaptation in previous endografts and was associated with no endoleak (type Ia, or gutter) in our series. In ASCEND registry, 3 endoleaks within 154 patients and 90 days of follow-up were detected (1type Ia and 2 type Ib). The freedom from type Ia endoleaks was 95. 7% and the freedom from all endoleakswas 94. 2% at 1 year.[16] Youssef et al reported also encouraging results with 100% technical success rate and no endoleak[17], while other small series also support these results.[18,19] Even in cases of failed ChEVAR with Nellix, a more proximal extension with a second Nellix is already described with encouraging results in the 2-year follow-up.[20]

As perioperative mortality in ChEVAR is similar to open repair and fenestrated EVAR, within the acceptable rate of 3. 1%, the parallel graft technique remains an attractive alternative in the treatment of pararenal aneurysms.[10] Especially, in elder patients, late outcomes, including overall mortality, are similar to standard EVAR.[21] High early mortality rate (10%) in this series may be explained by the small number of high-comorbidity patients during the primary period of the learning curve. Analogous results are presented also by Coscas et al during their early experience report (4/16).[22] During the follow-up period, 3 non–aneurysm related deaths were recorded. The fragility of these patients is an aspect that should not be disregarded. All patients were characterized as unfit for open repair while a complex aneurysm anatomy remains an independent factor of progressed disease. A high major adverse event rate up to 8. 5% is described in the literature.[1] In this study, 2 patients suffered a stroke (2/30, 6. 6%). In the current literature, bilateral upper extremity access, aneurysm rupture and an operation time >290 minutes are associated with a significantly increased risk for adverse events, with an incidence of clinical cerebrovascular events at 1. 9%.[1] Stroke after ChEVAR is associated with a high mortality rate.[1]

The major limitation of this study was its retrospective nature. The quite small number treated with ChEVAR and the short-term follow-up may also lead to bias. The high early post-operative mortality rate (3/30) represents an acceptable number when the age and comorbidities of these patients are kept in mind. Furthermore, the heterogeneity of endografts, devices and stent grafts used during the procedures influence the conformability of our results and hamper disciplinarian conclusions.

CONCLUSION

Chimney technique permitsthe management of para-renal AAAs according to each patient's specific anatomy. It seems a feasible and safe option at least during the early follow-up period. Despite the minimal invasive nature of the procedure, these patients need a meticulous perioperative care. Patients suffering from extended aortic aneurysm disease remain fragile during early follow-up, needing a close surveillance.

Acknowledgements: None

No conflict of interest.

REFERENCES

1 Bosiers M, Tran K, Lee J, Donas K, Veith F, Torsello G et al, for the PERICLES Registry Collaborators, Incidence and prognostic factors related to major adverse cerebrovascular events in patients with complex aortic diseases treated by the chimney technique, JVS, 2018, 1372-9

2 Donas K, Lee J, Lachat M, Torsello G, Veith F, on behalf of the PERICLES investigators Collected World Experience About the Performance of the Snorkel/Chimney Endovascular Technique in the Treatment of Complex Aortic Pathologies, The PERICLES Registry, Ann. Surg. , 2015, 262 (3): 546-53

3 Wanhainen A, Verzini F, Van Herzeele I, Allaire E, Bown M, Cohnert T et al, European Society for Vascular Surgery (ESVS) 2019 Clinical Practice Guidelines on the Management of Abdominal Aorto-iliac Artery Aneurysms, Eur J Vasc Endovasc Surg, 2018, 1-97

4 Usai M, Torsello G, Donas K, Current Evidence Regarding Chimney Graft Occlusions in the EndovascularTreatment of Pararenal Aortic Pathologies: A Systematic Review with Pooled Data Analysis, J Endovasc Ther, 2015, 22 :396-400

5 Donas K, Torsello G, Bisdas T, Osada N, Schonefeld E, Pitoulias G, Early Outcomes forFenestrated and Chimney Endografts in the Treatment of Pararenal Aortic Pathologies Are Not Significantly Different: A Systematic Review with Pooled Data Analysis, J Endovasc Ther, 2012, 19: 723–8

6 Bruen K, Feezor R, Daniels M, Beck A, Lee A, Endovascular chimney technique versus open repair of juxtarenal and suprarenal aneurysms, JVS, 2011, 53 (4): 895-904

7 Donas K, Torsello G, Piccoli G, Pitoulias G, Torsello G F, Bisdas T et al, The PROTAGORAS study to evaluate the performance of the Endurant stent graft for patients with pararenal pathologic processes treated by thechimney/snorkel endovascular technique, JVS, 2016, 63 (1): 1-7

8 Moulakakis K, Mylonas S, Avgerinos E, Papapetrou A, Kakisis J, Brountzos E et al, The chimney graft technique for preserving visceral vessels during endovascular treatment of aortic pathologies, JVS, 2012, 55 (5):1497-1503

9 Lachat M, Veith F, Pfammatter T, Glenck M, Bettex D, Mayer D et al, Chimney and Periscope Grafts Observed Over 2 Years After Their Use to Revascularize 169 Renovisceral Branches in 77 Patients With Complex Aortic Aneurysms. J Endovasc Ther, 2013, 20: 597-605

10 Chinsakchai K, Prapassaro T, Salisatkorn W, Hongku K, Moll F, Ruansetakit C et al, Outcomes of Open Repair, Fenestrated Stent Grafting, and Chimney Grafting in Juxtarenal Abdominal Aortic Aneurysm: Is it Time for a Randomized Trial?, Ann Vasc Surg, 2018, DOI: https://doi.org/10. 1016/j. avsg. 2018. 08. 097

11 Donas K, Criado F, Torsello G, Veith F, Minion D on behalf of the PERICLES Registry Collaborators, Classification of Chimney EVAR–Related Endoleaks: Insights from the 430 PERICLES Registry, J Endovasc Ther, 2016, DOI: 10. 1177/1526602816678994

12 Lindblad B, Bin Jabr A, Holst J, Malina M, Chimney Grafts in Aortic Stent Grafting: Hazardous or Useful Technique? Systematic Review of Current Data, Eur J Vasc Endovasc Surg, 2015, 50: 722-31

13 Ullery B, Tran K, Itoga N, Dalman R, Lee J, Natural history of gutter-related type Ia endoleaks after snorkel/chimney endovascular aneurysm repair, JVS, 2017, 65(4): 981-90

14 Tran K, Ullery B, Itoga N, Lee J, Polar orientation of renal grafts within the proximal seal zone affects risk of early type IA endoleaks after chimney endovascular aneurysm repair, JVS, 2018, 67 (4): 1034-41

15 Ronchey S, Fazzini S, Scali S, Torsello G, Kubilis P, Veith F et al, Collected 445 Transatlantic Experience from the PERICLES Registry: Use of Chimney Grafts to Treat Post-EVAR Type Ia Endoleaks Shows Good Midterm Results, J Endovasc Ther, 2018, 25(4): 492-8

16 Thompson M, Youssef M, Jacob R, Zerwes S, Reijnen M, Szopinski P et al, Early Experience with Endovascular Aneurysm Sealing in Combination with Parallel Grafts for the Treatment of Complex Abdominal Aneurysms: The ASCEND Registry, J Endovasc Ther, 2017, 24(6): 764-72

17 Youssef M, Zerwes S, Jakob R, Salem O, Dünschede F, Vahl C et al, Endovascular Aneurysm Sealing (EVAS) and Chimney EVAS in the Treatment of Failed Endovascular Aneurysm Repairs, J Endovasc Ther, 2017, 24(1): 115-20

18 Martinelli O, Fresilli M, Irace L, Venosi S, Jabbour J, Picone V et al, An alternative approach for treating a type Ia endoleak after conventional EVAR usingthe Nellix Endovascular Aneurysm Sealing, Ann Vasc Surg, 2018, 49: 316. e5-316. e10

19 Paraskevas K, Karouki M, Rehman A, Torella F, McWilliams R, England A et al, Endovascular Aneurysm Sealing (EVAS) Alone or in Combination with Chimney Grafts (chEVAS) for Treating Complications of Previous Endovascular Aneurysm Repair (EVAR) Procedures, Cardiovasc Intervent Radiol, 2018, 41(7): 1015-20

20 van de Velde L, Zoethout A, Lardenoije J W, Reijnen M, Secondary Endovascular Aneurysm (EVAS) Sealing in Combination with Chimney Grafts to Treat Failed Chimney EVAS, J Endovasc Ther, 2019, 15:1526602819830420

21 Tran K, Lee A, McFarland G, Sgroi M, Lee J, Complex endovascular aneurysm repair is associated with higher perioperative mortality but not late mortality compared with infrarenal endovascular aneurysm repairamong octogenarians, JVS, 2019, 69(2): 327-33

22 Coscas R, Kobeiter H, Desgranges P, Becquemin J P, Technical aspects, current indications, and results of chimney grafts for juxtarenal aortic aneurysms, JVS, 2011, 53(6): 1520-27

A multiscale model for hemodynamic properties' prediction after fenestrated endovascular aneurysm repair. A pilot study

Stavros Malatos[1], Anastasios Raptis[2], Michalis A. Xenos[1,2], George Kouvelos[3], Athanasios Katsargyris[4], Athanasios Giannoukas[2,3], Eric L. Verhoeven[4], Miltiadis Matsagkas[2,3]

[1]Department of Mathematics, Section of Applied Mathematics and Engineering Research, University of Ioannina, Ioannina, Greece
[2]Laboratory for Vascular Simulations, Institute of Vascular Diseases, Larissa, Greece
[3]Department of Vascular Surgery, Faculty of Medicine, School of Health Sciences, University of Thessaly, Larissa, Greece
[4]Department of Vascular and Endovascular Surgery, Paracelsus Medical University, Nuremberg, Germany

Abstract:

Aim: Fenestrated endovascular abdominal aortic aneurysm repair (FEVAR) has been increasingly applied for the treatment of anatomically suitable short-necked, juxtarenal, and suprarenal abdominal aortic aneurysms (AAA). Blood flow in the visceral vessels is maintained after FEVAR, but deployment of both the device and bridging covered stents might give rise to hemodynamic conditions that could impair the long-term success of the treatment. The aim of the present study is the development of a multiscale model that can reproduce hemodynamic flow in endograft models after FEVAR.

Methods: We reconstructed pre- and post-operative computed tomography scans of two patients treated with a custom-made fenestrated endograft. Blood flow properties were obtained by computational fluid dynamics (CFD) simulations for the post-FEVAR cases. Specifically, pressure drop, velocity, wall shear stress and mean helicity were measured during the cardiac cycle.

Results: No hemodynamic extremities with respect to the specialized wall shear stress (WSS) and helicity, based indexes were observed. A coherent helical field characterized blood flow topology during the cardiac cycle, with two counter-rotating helical structures. Regions of lowest time averaged WSS (TAWSS) in combination with high oscillatory shear index (OSI) are the areas after the splanchnic vessels at the aortic wall. Flow in the superior mesenteric and renal arteries remains almost unaffected after FEVAR. Important indices such as TAWSS, OSI and helicity are retained to almost normal levels. Low WSS values followed by high OSI values suggest that specific regions are sensitive to flow separation and probably to thrombus formation.

Conclusion: This pilot study shows the possibility of predicting non-invasively the hemodynamic performance of FEVAR utilizing CFD. FEVAR does not seem to alter target vessel and aorta perfusion significantly allowing good perfusion of visceral arteries.

INTRODUCTION

Fenestrated endovascular aneurysm repair (FEVAR) was first reported in 1999 for the treatment of a juxtarenal aortic aneurysm[1]. The technique was initially developed to treat high-risk patients unfit for open surgery and anatomically unsuitable for standard EVAR. Gradually, FEVAR has been increasingly applied for the treatment of all anatomically suitable short-necked, juxtarenal, and suprarenal aortic aneurysms. High volume centers have reported excellent perioperative and midterm outcomes[2,3]. However, FEVAR has also been associated with complications, such as loss of a visceral branch patency, stent fracture, endoleak, and graft migration[2-4].

Complications after FEVAR, such as target vessel thrombosis, could be associated to potential alteration in hemodynamics. Computational Fluid Dynamics (CFD) facilitates the identification of the hemodynamic conditions that might promote the advent of complications, focusing in regions of expected disturbances with high-resolution hemodynamics. Despite the increased popularity of CFD simulations in endovascular community, there are few studies delineating the hemodynamics in fenestrated endografts[5-8]. Additionally, Ou et al. highlighted hemodynamic variations in renal arteries with different stent orientations, concluding that appropriate cephalic deployment of a custom-made fenestrated endograft is a feasible strategy to preserve hemodynamic stability and durability[9]. Kandail et al. quantified the hemodynamic impact of flared renal stents on the performance of fenestrated endografts[2,4].

Author for correspondence:

Michalis A. Xenos

Assoc. Prof., Section of Applied Mathematics and Engineering Research, Department of Mathematics, University of Ioannina, Ioannina, Greece, 45110
Tel: +30 26510 008262,
E-mails: xenosmichalis@gmail.com, mxenos@uoi.gr
ISSN 1106-7237/ 2019 Hellenic Society of Vascular and Endovascular Surgery Published by Rotonda Publications

The modeling of such hemodynamic flows requires specialized multiscale models due to the complexity of physiology. There have been proposed holistic models of the arterial system[10,11]. Detailed three-dimensional time-dependent models have been developed, describing a particular part of the cardiovascular system. In these models the waveforms obtained from 1D / 0D models, used as boundary conditions for the 3D simulations[12,13]. The aim of the present study is the development of a multiscale model that can reproduce hemodynamic flow in endograft models after FEVAR.

MATERIAL AND METHODS

In this study, a multiscale model, composed of 1D and 3D parts that can reproduce hemodynamic flow in endograft models after FEVAR is presented. Through computational simulations, the blood flow in an indicative FEVAR case is characterized, drawing attention to the arteries that are supplied with blood through the fenestrations in the splanchnic vessels. The model provides high-resolution hemodynamic analytics, such as, time-averaged wall shear stress (TAWSS), oscillatory shear index (OSI), and local normalized helicity (LNH).

We studied the computed tomography (CT) scans, preoperatively and 1 month after FEVAR of 2 patients with a para-renal AAA treated electively by FEVAR. The patients underwent implantation of a custom-made fenestrated device at the Department of Vascular Surgery, Paracelsus Medical University, Nuremberg, Germany. Both patients gave written consent for utilization of the imaging data. The first patient underwent a double FEVAR (two fenestrations for the renal arteries and one scallop for the superior mesenteric artery, SMA). The second patient underwent FEVAR with three fenestrations (two for the renals and one for the SMA) and one scallop for the celiac axis, as depicted in Figure 1**.** Both patients received balloon expandable bridging stent-grafts in all target vessels.

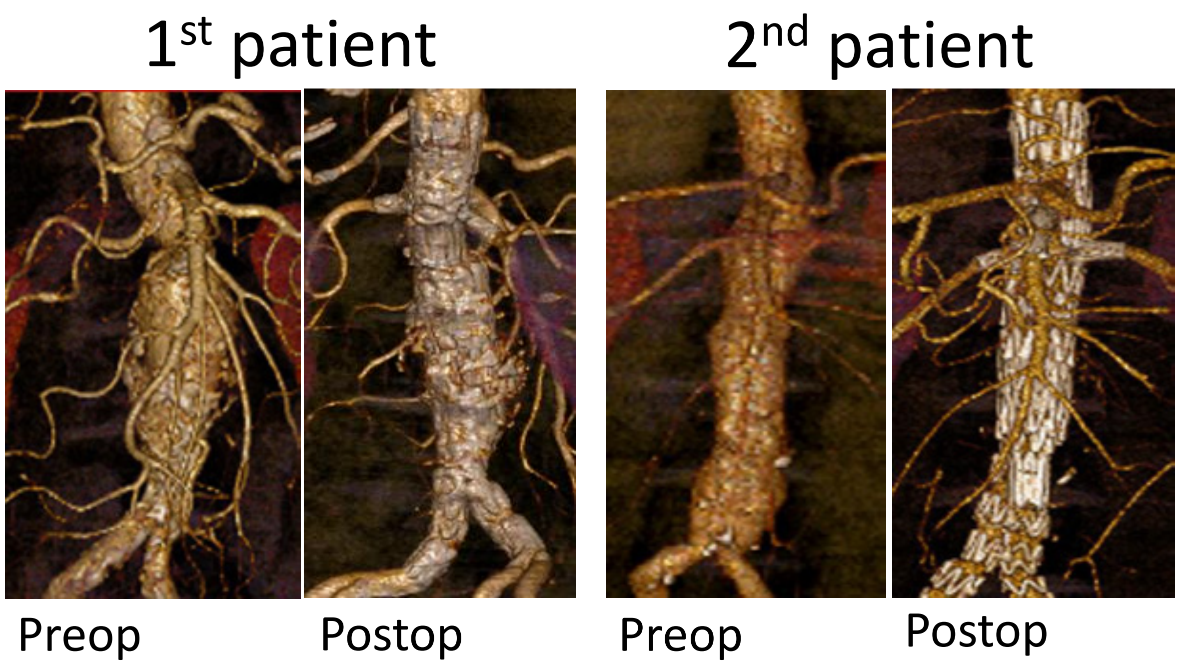


Figure 1. CT scans of the two patients pre- and post-operative cases.

3-dimensional patient-specific FEVAR model

Modelling approaches

The 3D model of the fenestrated stent-graft system, including the renal and superior mesenteric arteries and the celiac axis, was constructed using the CT scan of the treated patient. The reconstruction of the DICOM images into a three-dimensional model that preserves the geometry of the lumen postoperatively, was performed in the image processing and reconstruction software, Mimics (Materialise, Leuven, Belgium). The reconstructed geometry of the fenestrated endograft was subsequently meshed with tetrahedral elements in the software package ICEM CFD (Ansys Inc., Canonsburg, PA). After mesh independence test, a numerical grid of 2 million cells was considered sufficient to accurately predict the hemodynamic flow in detail.

Governing equations

The numerical simulations were performed in the software package Ansys Fluent (Ansys Inc., Canonsburg, US). Blood was considered as Newtonian fluid with density, ρ=1050 Kg/m^3 and kinematic viscosity, $\nu = 3.2\times10^{-6}$ m^2/s. The unsteady and incompressible blood flow is governed by the coupled non-linear system of transport equation such as the continuity and Navier-Stokes equations:

$$\begin{cases} \nabla \cdot \boldsymbol{u} = 0, \\ \dfrac{\partial \boldsymbol{u}}{\partial t} + (\boldsymbol{u} \cdot \nabla)\boldsymbol{u} = -\dfrac{1}{\rho}\nabla p + \nu \nabla^2 \boldsymbol{u}, \end{cases} \qquad (1)$$

where **u** is the velocity vector, ν is the kinematic viscosity, ρ is the density and p is the blood pressure.

Boundary conditions for the 3D model

1-dimensional holistic model

To perform CFD simulations, boundary conditions are required at the inlet and outlets of the model. The 3D model of the fenestrated endograft has one inlet, at the suprarenal abdominal aorta, and several outlets, at the superior mesenteric artery, celiac axis, renal arteries and common iliac arteries. Given the lack of patient-specific information regarding flow or pressure at the inlet and the outlets, we developed a 1D model that can provide the required waveforms at the points of interest for subsequent use in the 3D hemodynamic simulations. Specifically, the 1D model is composed of a system of elastic cylindrical tubes that enables the mathematical description of blood flow in an extended subset of the arterial system, as displayed in Figure 2a. The tubes are inter-connected with each other, meaning that the outflow from the preceding tube defines the inflow to the succeeding one, along with appropriate conditions at bifurcations[10,14]. As initial conditions the pressure and the flow waveforms in the ascending aorta were introduced in the model. The 1D model can predict for each vessel the flow (q), pressure (p) and cross-sectional area (A). The model relies on literature data for the definition of the Young's modulus, E, on each tube, while the wall thickness, h, and base diameter, A_0 of each segment are patient-based. To include blood flow through the microvascular system (smaller arteries, arterioles and capillaries), a lumped-parameter model (3-element Windkessel) is attached at each end of the network of tubes[13]. Figure 2 shows a graphical representation of the multiscale model that relies on 0D, 1D parts to export boundary condition for the high-resolution 3D hemodynamic simulations after FEVAR.

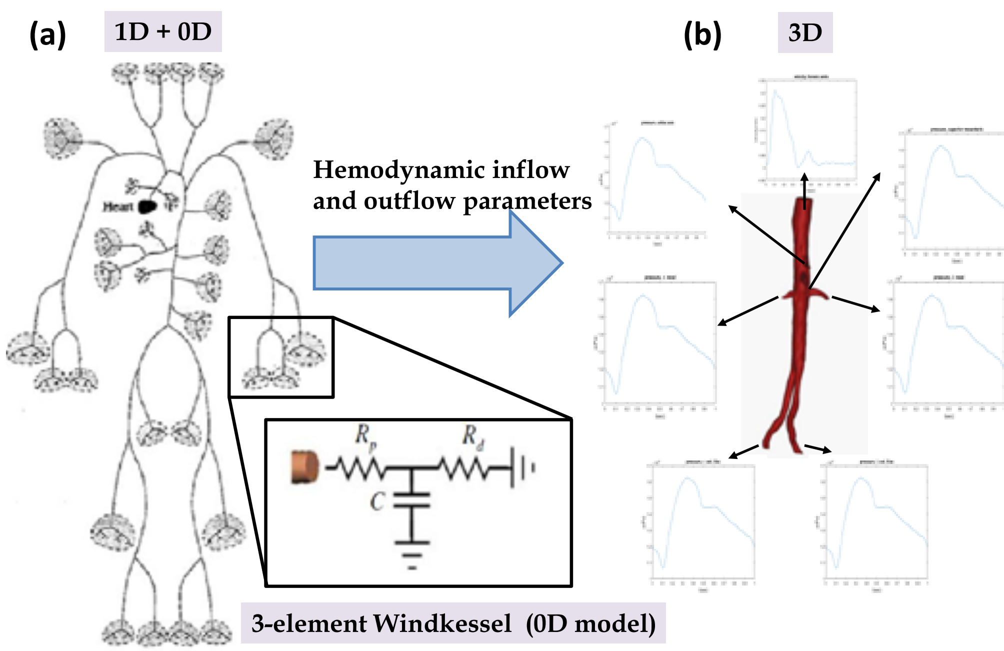


Figure 2. The multiscale arterial blood flow model.

Numerical solution

The governing partial differential equations[1] of blood flow are discretized with the Finite Volume method. The resulting system of algebraic equations is solved iteratively using the SIMPLE pressure-velocity coupling scheme and convergence was achieved when residual error of each equation was equal to 10^{-5}. The cardiac cycle was considered to last 1s, with a fixed time step of Δt = 0.0035s. The simulations were performed for four cardiac cycles in two Intel Xeon processors (E5645, 2.40GHz, 12MB Cache, 5.86GT/s Intel QPI) of a Dell™ Precision™ T7500 workstation. We exclusively utilized the results of the fourth cardiac cycle avoiding any dynamic disturbances of the numerical solution in the initial three cycles.

Hemodynamic parameters

WSS-related properties. Besides wall shear stress (WSS), additional WSS-derived properties were calculated. Specifically, the hemodynamic analysis includes the time averaged wall shear stress (TAWSS) over a cardiac cycle,

$$\mathrm{TAWSS}(\boldsymbol{s}) = \frac{1}{T}\int_0^T |WSS(\boldsymbol{s},t)|\,dt, \qquad (2)$$

and the oscillatory shear index (OSI),

$$\mathrm{OSI}(\boldsymbol{s}) = 0.5\left[1 - \frac{\frac{1}{T}\left|\int_0^T WSS(\boldsymbol{s},t)\,dt\right|}{\frac{1}{T}\int_0^T |WSS(\boldsymbol{s},t)|\,dt}\right], \qquad (3)$$

where *T* is the duration of cardiac cycle, ***s*** the generic location at the vessel wall and *t* is time[15,16].

Helicity-related parameters. Helicity, *H(**s**, t)*, is an index applied to quantify the interplay between rotational and translational motion of blood and is defined as the scalar product of the velocity field ***v****(s, t)* and the vorticity field, ***ω****(s, t)* at a location, ***s***, in the flow field[17,18]. The local normalized helicity (LNH) is defined as,

$$\mathrm{LNH} = \frac{\boldsymbol{v}(s,t)\cdot\boldsymbol{\omega}(s,t)}{|\boldsymbol{v}(s,t)\cdot\boldsymbol{\omega}(s,t)|}\,. \qquad (4)$$

The quantity LNH, is a function of space and time and is an indicator of the intensity of helical structures and their direction of rotation[17]. Specifically, when the absolute value of LNH is one, then the flow is purely helical, and when it is zero, blood flow is characterized symmetric. The sign of LNH dictates the right or left-handed direction of the rotation of helical structures.

RESULTS

The proposed multiscale model of the arterial system is able to provide information on the flow, pressure and changes in the cross section of the entire arterial system, Table 1. It can be used to describe significant hemodynamic phenomena based on appropriate non-invasive patient-based measurements.

Arteries	Mean Flow, q *(ml/s)*	Mean Arterial Pressure, p *(mm Hg)*	Cross-sectional variation, A (%)
Ascending aorta	99.8	95.91	3.53
R. subclavian & brachial	7.49	86.88	4.90
R. com. carotid	7.49	78.21	6.21
Aortic arch (I)	84.91	95.01	3.68
Thoracic aorta	70.13	93.97	4.09
Celiac axis	0.54	93.85	4.11
Superior mesenteric	10.11	90.06	4.73
Abdominal aorta (II)	59.47	93.42	3.75
R. renal	10.72	87.71	5.06
R. com. & external iliac	19.66	90.50	2.44
R. Internal iliac	4.53	79.57	1.16
R. Femoral	15.13	86.54	2.10

Table 1. Quantification of mean flow, mean blood pressure and percentage change in cross-sectional area, as derived from the model in basic arteries during the cardiac cycle.

WSS-related parameters. The spatial WSS profile at peak systole is displayed in Figure 3. It is shown that low WSS magnitude applies on the main body of the stent-graft, WSS < 10 Pa, and higher WSS, in the range of 15-40 Pa, is observed along the surface of the limbs, Figure 3 (a). On the wall of renal arteries, celiac axis and superior mesenteric arteries, the magnitude of WSS is in the range of 30-35 Pa, Figure 3 (b)-(e).

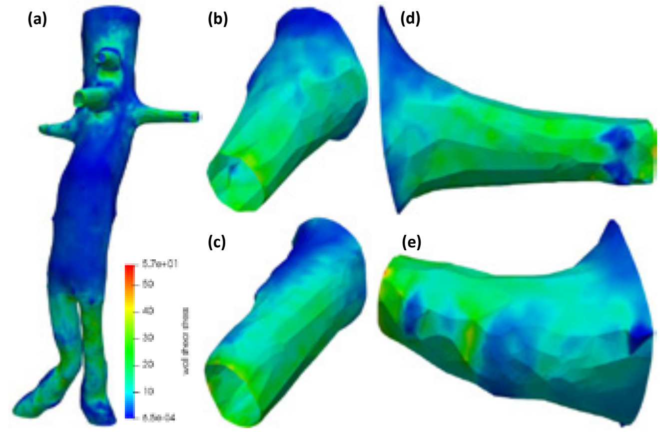


Figure 3. Wall shear stress at the maximum of the systolic phase. (a) WSS in extended graft (abdominal and iliacs), (b) WSS in the celiac axis, (c) WSS in the superior mesenteric artery, (d) WSS in the left renal artery, and (e) WSS in the right renal artery.

The distribution of TAWSS on the surface of the lumen showed that areas with higher values appear in the smaller arteries, iliacs, mesenteric, celiac axis and renal arteries, in the range of 4 - 8Pa, while smaller values of 2 - 4Pa, Figure 4 (a), are observed on the main body of the device. In the superior mesenteric, celiac axis and renal arteries, Figure 4 (b)-(e) the value of TAWSS ranges from 2 - 9 Pa**.**

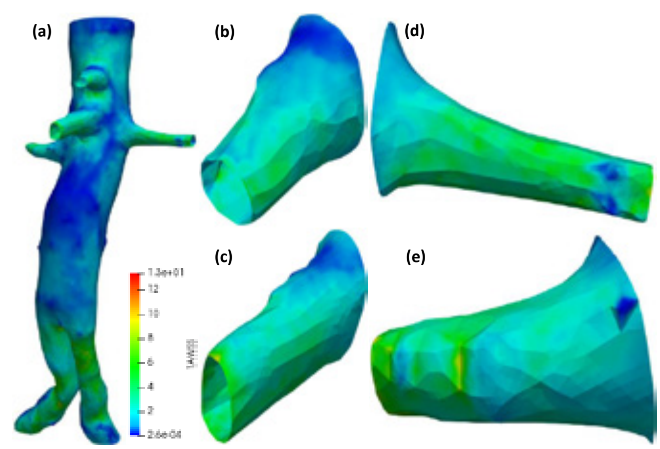


Figure 4. The profiles of time-averaged wall shear stress, (TAWSS) during cardiac cycle. (a) TAWSS throughout the graft (abdominal and iliacs), (b) TAWSS in the celiac axis, (c) TAWSS in the superior mesenteric artery, (d) TAWSS in the left renal artery and (e) TAWSS in the right renal artery

The OSI has been proposed as a parameter that can give an estimate of in-graft thrombus formation risk in correlation with WSS and TAWSS[19] and characterizes whether the WSS vector is aligned with the TAWSS vector throughout the cardiac cycle. Elevated OSI levels are often accompanied by low TAWSS, and together they serve as indicators for vascular injury or dysfunction[20], as depicted in Figure 5. An OSI value close to zero (OSI < 0.2) indicates that flow is unidirectional at that location throughout the pulsatile cycle whereas a high OSI value (OSI > 0.2) indicates that the flow oscillates forward and backward for the same periods of time during the entire cardiac cycle[21]. At high OSI values blood flow induces the retrograde flow, causing a greater change in flow direction near the wall. In addition, this regions has a higher probability of developing lesions, as OSI values>0.2 trend to the development of endothelial dysfunction. Thus, OSI is a relatively sensitive hemodynamic indicator of regional vascular remodeling. In Figure 5, the areas in the renals mesenteric, celiac axis and iliac arteries exhibits low OSI. On the contrary regions such as the entrance of these arteries and a large portion of the aortic lumen reveal an intense OSI.

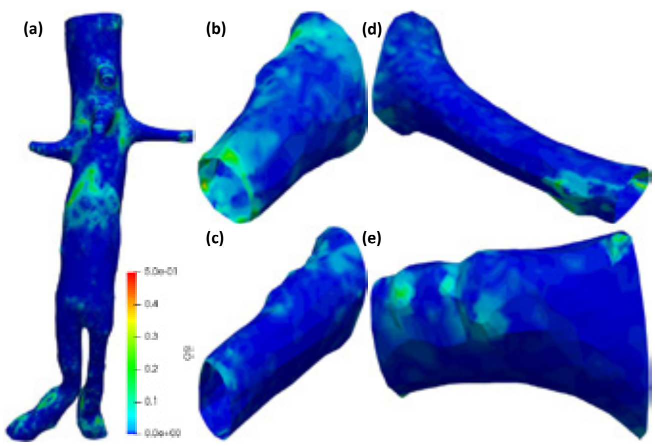


Figure 5. The profiles of oscillatory shear index (OSI) during cardiac cycle. (a) OSI throughout the graft (abdominal and iliacs), (b) OSI in the celiac axis, (c) OSI in the superior mesenteric artery, (d) OSI in the left renal artery and (e) OSI in the right renal artery.

Helicity-related parameters. In the main body of the fenestrated device there are two, almost uniformly distributed helical structures in different directions, Figure 6 (a). In the smaller arteries and at the entrance of the graft, these helical structures are separated into individual smaller structures, Figure 6 (b)-(e). The order of average helicity is |LNH|≤0.2.

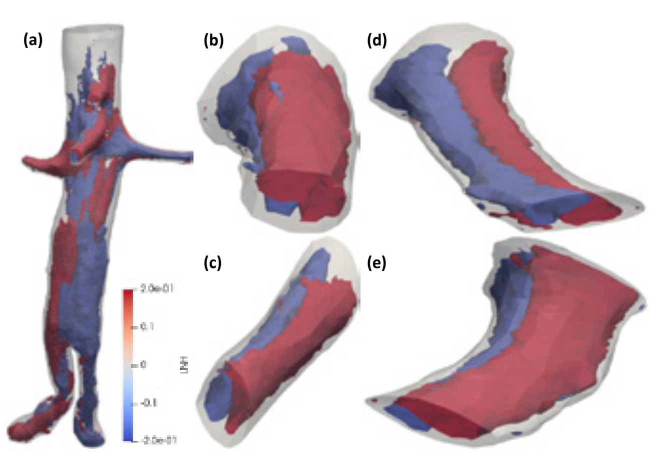


Figure 6. The local normalized helicity (LNH) during the cardiac cycle. (a) LNH throughout the graft (abdominal and iliacs), (b) LNH in the celiac axis, (c) LNH in the superior mesenteric artery, (d) LNH in the left renal artery, and (e)) LNH in the right renal artery.

The above hemodynamic indices are similar for the two patients, so we have decided to highlight the results obtained from one of the two patients. We observe that the second patient exhibits slightly lower WSS values in the abdominal part of the stent-graft, the remaining part exhibits similar behavior. The second patient also presents lower OSI values but similar LNH behavior with the first patient.

DISCUSSION

FEVAR has matured and became a valid alternative to open surgery, especially in high-risk surgical patients. In the short term, FEVAR success largely depends on the accurate preoperative planning, and the appropriate device design[22], while in the long-term, it seems important to maintain graft integrity to avoid complications such as target vessel thrombosis or endograft's migration. Data obtained by CFD simulations may theoretically aid to improve those devices and eventually lead to a better long-term outcome.

CFD simulations may aid to evaluate the durability of fenestrated stent-grafts and seem cost-effective and reliable when used in conjunction with physiologically correct boundary conditions[4]. In recent years, a number of multiscale approaches have been proposed to accurately describe blood flow dynamics of compliant arterial vessels[11,13,23]. Multiscale models are widely used as boundary conditions for advanced 3D patient-based models through simple Windkessel or lumped models for the rest of the circulation. Simplified 1D models, have been coupled with 3D model to determine the hemodynamic characteristics and represents a significant step forward for realistic representation of the cardiovascular system[24-28]. In this study, a multiscale model was developed composed of a 1D simplified model to describe blood circulation. The data from the simplified model are used as boundary conditions for advanced 3D patient-based FEVAR models. For the microvascular system, a lumped-parameter model is attached at each end of the circulation network. This pilot study shows the possibility of predicting non-invasively the hemodynamic performance of FEVARs utilizing CFD.

The notion of wall shear stress (WSS) is important in the clinical practice due to the fact that a pathological profile of shear stresses can impede atherogenesis, thrombosis, adhesion of leukocytes, smooth muscle proliferation and endothelial apoptosis[29]. Additionally, the parallel action of high and low shear stresses at the aortic wall in connection with irregularities of the lumen, could initiate the activation of the platelets that could eventually lead to thrombosis[30-33]. For the stented cases, the presence of low shear stresses might lead to in-stent restenosis and possibly to limb occlusion that in turn will force the patient to undergo additional intervention[29]. In previous studies has been shown that the postoperative structures reveal a reduction in the maximum WSS in the abdominal part of the graft compared to the normal cases[34,35]. Additionally, TAWSS has a significant difference between repaired and healthy cases during the cardiac cycle[36]. The combination of TAWSS and OSI could reveal regions more susceptible to thrombus formation due to flow separation. The results of the present study demonstrate that the regions characterized by the lowest TAWSS in combination with high values of OSI are the areas after the splanchnic vessels at the abdominal aortic wall.

Helical flow has been reported to play a positive role facilitating blood flow transport, suppressing disturbed blood flow, preventing accumulation of atherogenic lipoproteins on the arterial surfaces, enhancing oxygen transport from the blood to the arterial wall, thus reducing the adhesion of blood cells on the surface[37]. The helical arrangement of the flow visualized at renals, superior mesenteric and celiac axis, has its energetic meaning in the compensative attempt of curling the forward blood flow, to fill the space due to flow separation at the wall. The results of the present study indicate that a coherent helical field characterizes blood flow topology during the entire cardiac cycle, with two counter-rotating helical structures[17]. Additionally, a high level of LNH is instrumental in suppressing low velocity/stagnation regions leading to a healthy flow profile in the FEVAR. In a nutshell, the study reveals no significant changes according to the hemodynamic indices. The clinical outcome of this pilot study is that FEVAR does not alter target vessel and aorta perfusion significantly allowing good perfusion of visceral arteries. Additional study including a large cohort of patients is required to infer unquestionable clinical implications.

Limitations

The elastic properties of the graft material were not taken into account in the 3D patient-based structure and the surface of the lumen was modeled as rigid. Blood could be modeled as non-Newtonian fluid, separating red blood cells from plasma, which were considered in the current study as a single continuum medium. The geometric and structural parameters employed in the 1D arterial model was based on bibliographic data. A future arterial model could be constructed with a geometry and elasticity database derived from patient-based measurements, and the model predictions would be compared with non-invasive measurements.

CONCLUSION

The findings of this study show that the flow in the superior mesenteric and renal arteries remains almost unaffected after FEVAR retaining all important indices to normal levels. However, low values of WSS followed by high values of OSI could suggest that these regions are more sensitive to flow separation and may become prone to thrombus formation.

Conflict of Interest

E.L.G.V. is consultant for Cook and Atrium.

REFERENCES

1 Browne T, Hartley D, Purchas S, Rosenberg M, Van Schie G, Lawrence-Brown M. A fenestrated covered suprarenal aortic stent. European journal of vascular and endovascular surgery. 1999;18(5):445-9

2 Kandail HS, Hamady M, Xu XY. hemodynamic Functions of Fenestrated stent graft under resting, hypertension, and exercise conditions. Frontiers in surgery. 2016;3:35

3 Verhoeven EL, Katsargyris A, Oikonomou K, Kouvelos G, Renner H, Ritter W. Fenestrated Endovascular Aortic Aneurysm Repair as a First Line Treatment Option to Treat Short Necked, Juxtarenal, and Suprarenal Aneurysms. European journal of vascular and endovascular surgery : the official journal of the European Society for Vascular Surgery. 2016

4 Kandail H, Hamady M, Xu XY. Effect of a Flared Renal Stent on the Performance of Fenestrated Stent-Grafts at Rest and Exercise Conditions. Journal of Endovascular Therapy. 2016;23(5):809-20

5 Suess T, Anderson J, Danielson L, Pohlson K, Remund T, Blears E, et al. Examination of near-wall hemodynamic parameters in the renal bridging stent of various stent graft configurations for repairing visceral branched aortic aneurysms. Journal of vascular surgery. 2015

6 Kandail H, Hamady M, Xu XY. Patient-specific analysis of displacement forces acting on fenestrated stent grafts for endovascular aneurysm repair. Journal of Biomechanics. 2014;47(14):3546-54

7 Avrahami I, Brand M, Meirson T, Ovadia-Blechman Z, Halak M. Hemodynamic and mechanical aspects of fenestrated endografts for treatment of Abdominal Aortic Aneurysm. European Journal of Mechanics B-Fluids. 2012;35:85-91.

8 Jones SM, Poole RJ, How TV, Williams RL, McWilliams RG, Brennan JA, et al. Computational fluid dynamic analysis of the effect of morphologic features on distraction forces in fenestrated stent grafts. Journal of vascular surgery. 2014;60(6):1648-56. e1

9 Ou J, Tang A, Chiu T, Chow K, Chan Y, Cheng S. Haemodynamic variations of flow to renal arteries in custom-made and pivot branch fenestrated endografting. European Journal of Vascular and Endovascular Surgery. 2017;53(1):133-9

10 Olufsen MS, Peskin CS, Kim WY, Pedersen EM, Nadim A, Larsen J. Numerical simulation and experimental validation of blood flow in arteries with structured-tree outflow conditions. Annals of biomedical engineering. 2000;28(11):1281-99

11 Reymond P, Merenda F, Perren F, Rufenacht D, Stergiopulos N. Validation of a one-dimensional model of the systemic arterial tree. American Journal of Physiology-Heart and Circulatory Physiology. 2009;297(1):H208-H22

12 Formaggia L, Gerbeau J-F, Nobile F, Quarteroni A. On the coupling of 3D and 1D Navier–Stokes equations for flow problems in compliant vessels. Computer methods in applied mechanics and engineering. 2001;191(6-7):561-82

13 Kim HJ, Vignon-Clementel IE, Figueroa CA, LaDisa JF, Jansen KE, Feinstein JA, et al. On Coupling a Lumped Parameter Heart Model and a Three-Dimensional Finite Element Aorta Model. Annals of Biomedical Engineering. 2009;37(11):2153-69

14 Stergiopulos N, Young D, Rogge T. Computer simulation of

arterial flow with applications to arterial and aortic stenoses. J Biomech. 1992;25(12):1477-88

15 Gallo D, Vardoulis O, Monney P, Piccini D, Antiochos P, Schwitter J, et al. Cardiovascular morphometry with high-resolution 3D magnetic resonance: First application to left ventricle diastolic dysfunction. Medical engineering & physics. 2017;47:64-71

16 Morbiducci U, Kok AM, Kwak BR, Stone PH, Steinman DA, Wentzel JJ. Atherosclerosis at arterial bifurcations: evidence for the role of haemodynamics and geometry. Thromb Haemost. 2016;115(3):484-92

17 Morbiducci U, Ponzini R, Rizzo G, Cadioli M, Esposito A, De Cobelli F, et al. In vivo quantification of helical blood flow in human aorta by time-resolved three-dimensional cine phase contrast magnetic resonance imaging. Annals of Biomedical Engineering. 2009;37(3):516-31

18 Morbiducci U, Ponzini R, Gallo D, Bignardi C, Rizzo G. Inflow boundary conditions for image-based computational hemodynamics: Impact of idealized versus measured velocity profiles in the human aorta. Journal of Biomechanics. 2013;46(1):102-9

19 Di Achille P, Tellides G, Humphrey J. Hemodynamics-driven deposition of intraluminal thrombus in abdominal aortic aneurysms. International journal for numerical methods in biomedical engineering. 2017;33(5):e2828

20 Papadopoulos KP, Gavaises M, Pantos I, Katritsis DG, Mitroglou N. Derivation of flow related risk indices for stenosed left anterior descending coronary arteries with the use of computer simulations. Med Eng Phys. 2016;38(9):929-39

21 Hardman D, Semple SI, Richards JM, Hoskins PR. Comparison of patient-specific inlet boundary conditions in the numerical modelling of blood flow in abdominal aortic aneurysm disease. International journal for numerical methods in biomedical engineering. 2013;29(2):165-78

22 Ou J, Chan Y-C, Chan CY-T, Cheng SW. Geometric alteration of renal arteries after fenestrated grafting and the impact on renal function. Annals of vascular surgery. 2017;41:89-95

23 Xiao N, Humphrey JD, Figueroa CA. Multi-scale computational model of three-dimensional hemodynamics within a deformable full-body arterial network. Journal of computational physics. 2013;244:22-40

24 Vignon-Clementel IE, Figueroa CA, Jansen KE, Taylor CA. Outflow boundary conditions for three-dimensional finite element modeling of blood flow and pressure in arteries. Comput Method Appl M. 2006;195(29-32):3776-96

25 Vignon IE, Taylor CA. Outflow boundary conditions for one-dimensional finite element modeling of blood flow and pressure waves in arteries. Wave Motion. 2004;39(4):361-74

26 Steele BN, Taylor CA, editors. Simulation of blood flow in the abdominal aorta at rest and during exercise using a 1-d finite element method with impedance boundary conditions derived from a fractal tree. Proceedings of the 2003 ASME Summer Bioengineering Conference; 2003

27 Blanco PJ, Feijóo RA. A 3D-1D-0D Computational model for the entire cardiovascular system. Computational Mechanics, eds E Dvorking, M Goldschmit, M Storti. 2010;29:5887-911

28 Crosetto P, Reymond P, Deparis S, Kontaxakis D, Stergiopulos N, Quarteroni A. Fluid–structure interaction simulation of aortic blood flow. Computers & Fluids. 2011;43(1):46-57

29 Papaioannou TG, Stefanadis C. Vascular wall shear stress: basic principles and methods. Hellenic J Cardiol. 2005;46(1):9-15

30 Boyd AJ, Kuhn DC, Lozowy RJ, Kulbisky GP. Low wall shear stress predominates at sites of abdominal aortic aneurysm rupture. Journal of vascular surgery. 2015;63(6):1613-9

31 Bluestein D, Dumont K, De Beule M, Ricotta J, Impellizzeri P, Verhegghe B, et al. Intraluminal thrombus and risk of rupture in patient specific abdominal aortic aneurysm - FSI modelling. Computer methods in biomechanics and biomedical engineering. 2008;12(1):73-81

32 Biasetti J, Hussain F, Gasser TC. Blood flow and coherent vortices in the normal and aneurysmatic aortas: a fluid dynamical approach to intraluminal thrombus formation. Journal of the Royal Society Interface. 2011;8(63):1449-61

33 Biasetti J, Gasser TC, Auer M, Hedin U, Labruto F. Hemodynamics of the normal aorta compared to fusiform and saccular abdominal aortic aneurysms with emphasis on a potential thrombus formation mechanism. Annals of Biomedical Engineering. 2010;38(2):380-90

34 Raptis A, Xenos M, Georgakarakos E, Kouvelos G, Giannoukas A, Labropoulos N, et al. Comparison of physiological and post-endovascular aneurysm repair infrarenal blood flow. Computer methods in biomechanics and biomedical engineering. 2017;20(3):242-9

35 Raptis A, Xenos M, Georgakarakos E, Kouvelos G, Giannoukas A, Matsagkas M. Hemodynamic profile of two aortic endografts accounting for their postimplantation position. Journal of Medical Devices. 2017;11(2):021003

36 Tasso P, Raptis A, Matsagkas M, Rizzini ML, Gallo D, Xenos M, et al. Abdominal Aortic Aneurysm Endovascular Repair: Profiling Postimplantation Morphometry and Hemodynamics With Image-Based Computational Fluid Dynamics. Journal of biomechanical engineering. 2018;140(11):111003

37 Liu X, Sun A, Fan Y, Deng X. Physiological significance of helical flow in the arterial system and its potential clinical applications. Annals of Biomedical Engineering. 2015;43(1):3-15

CONTINUUM OF CARE
AV

CONQUEST® 40
PTA Dilatation Catheter

LUTONIX® 035
Drug Coated Balloon PTA Catheter

Surgical techniques during extra-anatomical vascular reconstruction to treat prosthetic graft infection in the groin

Nikolaos Kontopodis[1], Emmanouil Tavlas[1], George Papadopoulos[1], Nikolaos Daskalakis[1], Christos Chronis[1], Giannis Dimopoulos[1], Stella Lioudaki[1], Alexandros Kafetzakis[1], Alexia Papaioannou[2], Christos V. Ioannou[1]

[1]Vascular Surgery Unit, Department of Thoracic, Cardiac and Vascular Surgery
[2]Anesthesiology Department, University Hospital of Heraklion, University of Crete Medical School, Heraklion, Greece

Abstract:
Prosthetic graft infection is a serious complication after reconstructive vascular surgery and the most common anatomic location of this condition is the groin. Traditionally, removal of the prosthesis to eradicate the infectious source and subsequent extra-anatomic vascular reconstruction to preserve distal perfusion is considered the safest treatment option. Tunneling of the new graft through uninfected tissues is technically challenging and can usually be performed through three different routes, namely the ilio-psoas muscular lacuna, the obturator foramen and the wing of the iliac bone. We report a single center five-year experience using these techniques emphasizing on technical remarks.

INTRODUCTION

Prosthetic graft infection represents one of the most feared complications in vascular surgery. The goals of managing vascular graft infection involve eradication of the local and systemic septic source and maintenance of adequate arterial perfusion in the distal limb. In cases where either the interruption of the arterial graft does not result in critical limb ischemia due to adequate collateralization or the graft is already blocked, simple excision provides the most straightforward treatment option. On the other hand, in case of patent grafts which are essential for distal limb perfusion, several preservation techniques have been proposed which include serial wound debridement, antibiotic-loaded beads, negative pressure and rotational muscle flaps.[1] The strategy of graft preservation is attractive because it avoids the technical challenges and operative risks associated with in-situ or extra-anatomic vascular reconstruction, but this treatment option should be limited in cases of segmental graft infection sparing the anastomoses, single gram-positive rather than polymicrobial or gram-negative infections and extracavitary locations. Subsequently, lots of patients are not candidates for preservation techniques and in this case graft excision along with in-situ or extra-anatomical reconstruction is indicated. Again, in situ reconstruction has been mainly advocated for less invasive infections especially in intra-cavitary location. The rest of the patients presenting with prosthetic graft infections may benefit from graft excision and extra-anatomical reconstruction through uninfected tissue planes. In these cases, several surgical techniques have been described, but these procedures are usually challenging and accompanied by significant perioperative risks. The groin is the most common site of vascular graft infection and 5% infection rates have been reported.[2] We aim to report a single institution case series of infected prosthetic grafts in the groin, in need for graft excision and extra-anatomic bypass giving emphasis on the surgical techniques used to treat these patients.

Author for correspondence:
Nikolaos Kontopodis, MD, MSc, PhD
Vascular Surgery Unit, Department of Cardiothoracic and Vascular Surgery, University Hospital of Heraklion
PO Box 1352, 711 10, Heraklion, Greece
Tel: +30 281 340 2379, Fax: +30 2810 375365,
E-mail: kontopodisn@yahoo.gr
ISSN 1106-7237/ 2019 Hellenic Society of Vascular and Endovascular Surgery Published by Rotonda Publications

CASE SERIES

During a 5-year period 15 cases of graft infection in the groin were identified at our institution. This accounts for 4.2% of all lower limb arterial reconstructions involving the femoral artery during the same time interval (15/357 limbs). The diagnosis of infection was based on surgical, microbiological, and clinical follow-up findings in all patients. Positive microbiological findings or pus around the prosthesis, exposed graft in a disrupted wound, pseudoaneurysms with overlying cellulitis/inflammation were considered indicative of graft infection. On the other hand, in the absence of compatible operative findings and negative microbiology or in patients who were not operated and presented a negative clinical and imaging follow-up for >4months, the graft was declared uninfected. Among our 15 cases, preservation of the graft was possible in 3 and graft excision in another 3 patients. Excision of the infected prosthesis and extra-anatomical reconstruction was necessary in 9 patients (60% of all infected grafts). Overall these involved 2 axillo-bifemoral, 1 aorto-bifemoral, 1 ilio-femoral, 1 femoro-femoral and 4 femoro-popliteal bypass. All grafts were patent and a consensus was made, based on clinical and imaging data, that simple removal of the graft without additional revascularization would most likely result in critical limb ischemia and significant risk for limb loss. The procedures undertaken in order to revascularize the limb through uninfected tissues were 2 ilio-femoral and 1 ilio-popliteal bypass through the wing of the iliac bone, 1 ilio-femoral and 3 ilio-popliteal bypass through the muscular lacuna underneath the iliopsoas fascia and 1 thoracic (descending) aorta - bifemoral bypass and 1 axillo-bifemoral bypass through the obturator foramen. The conduit was ePTFE in all cases. Peri-procedural mortality was 11% (one patient died due to respiratory tract infection) while during 18 months mean follow-up, there were 3 major amputations. Secondary patency was achieved in 5/8 cases during the same time interval (Table 1). All 3 patients that needed amputation presented graft occlusion with critical limb ischemia and no further options for revascularization.

	Clinical presentation	Prio procedures	Micro-organisms	Management	Graft Reinfection	Graft Patency	Outcome
#1	Szilagyi III, Samson V	Femoro-Femoral	Staphylococcus aureus, Proteus	Ilio-femoral bypass through iliac wing	No	Patent	Deceased, Due to Respiratory tract infection
#2	Szilagyi III, Samson IV	Axillo-BiFemoral	Staphylococcus aureus, Eschericia coli	Thoracic (descending) aorta - bifemoral bypass through right obturator foramen	No	Patent	Alive, good wound closure
#3	Szilagyi III, Samson V	Ilio-Femoral	Staphylococcus aureus	Ilio-femoral bypass underneath muscular lacuna	No	Patent	Alive, good wound closure
#4	Szilagyi III, Samson IV	Aorto-BiFemoral	Enterococcus, Acinetobacter	Graft limb (Previous aorto-bifemoral) to superficial femoral artery bypass through iliac wing	No	Occluded	Alive, Above knee amputation
#5	Szilagyi III, Samson IV	Femoro-Popliteal	Staphylococcus aureus, Pseudomonas aeruginosa	Below Knee ilio-popliteal bypass through iliac wing	No	Patent	Alive, good wound closure
#6	Szilagyi III, Samson IV	Femoro-Popliteal	Enterococcus, Pseudomonas aeruginosa	Below Knee ilio-popliteal bypass through iliac wing	No	Patent	Alive, good wound closure
#7	Szilagyi III, Samson IV	Femoro-Popliteal	Staphylococcus aureus, Acinetobacter	Below Knee ilio-popliteal bypass through muscular lacuna	No	Occluded	Alive, Above knee amputation
#8	Szilagyi III, Samson IV	Femoro-Popliteal	Staphylococcus aureus, Eschericia coli	Below Knee ilio-popliteal bypass through muscular lacuna	No	Occluded	Alive, Above knee amputation
#9	Szilagyi III, Samson IV	Axillo-BiFemoral	Enterococcus, Acinetobacter, Klebsiella	Axilo-bifemoral bypass through obturator foramen	No	Patent	Alive, good wound closure

- **Szilagyi** wound infection classification: I: Infectious involvement of the cutis; II: Infection of the Cutis / Subcutis not involving the graft; III: Graft infection.
- **Samson** classification. I and II similar to previous definitions, III: Graft infection without anastomosis involvement; IV: Graft infection involving the anastomosis, without complications; V: Graft infection associated with complications.

Table 1. Summary of characteristics of patients treated for graft infection in the groin.

From a purely technical perspective, extra-anatomic revascularization and graft excision were always performed during the same operation, with this sequence. Tunneling the new bypass through uninfected tissue planes probably represents the most challenging part of these procedures. Several techniques have been reported to avoid the infected groin and we have used 3 approaches in our series which were based on previous experience with the use of these techniques in our Department.[3,4] The most widely reported approach is the obturator bypass. During this procedure either the common or the external iliac artery can serve as the donor vessel which can be exposed through a standard curvilinear lower quadrant incision and a retroperitoneal approach. The incision usually starts around 1-2 finger-breaths (FB) above the mid-distance between the pubis and the umbilicus and run from the lateral edge of the rectus muscle, obliquely up to 1-2 FB supero-medial to the anterior superior iliac spine (Figure 1).

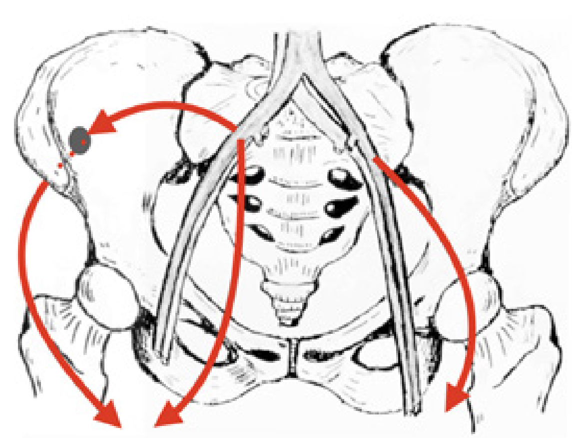


Figure 1. Schematic of the possible extranatomic bypass routes.

Through this incision the abdominal muscles are divided and the peritoneal sac is bluntly mobilized in order to approach the iliac vessels. Through the same incision the obturator foramen is approached. Taking into account that the obturator nerve and artery perforate the obturator membrane at the posterolateral aspect of the foramen, it is advisable to make the surgical incision at its anteromedial side. Since the membrane is very dence, sharp rather than blunt dissection should be applied. Regarding the target vessel, the mid-thigh superficial femoral artery or the popliteal artery are most often used which are exposed through standard surgical incisions. We prefer this approach when the distal target vessel is the superficial femoral or popliteal artery. The profunda femoral artery may also be used but its exposure from the posteromedial side can be technically challenging and risks entering the infected groin. We prefer to make the tunnel in the potential space between the adductor longus and brevis anteriorly and the adductor magnus muscle posteriorely. Since the longus and brevis muscles arise from the anterior while the magnus from the posterior rim of the external surface of the obturator foramen, one can understand that this approach leads directly to the obturator foramen. Direction of tunneling can either be from inferior to superior or vice versa.

Regarding the other two surgical approaches, the retroperitoneal exposure of the iliac arteries is performed as already described. If the route through the wing of the iliac bone is used, the internal surface of the iliac wing is approached through the same incision used to expose the iliac arteries, 1-2 FB below the iliac crest. Then a separate short skin incision is performed 1-2 FB below the anterior superior iliac spine at its lateral side, to approach the external surface of the iliac wing. With the aid of a drill, the bone is penetrated from the outside keeping an oblique, upward direction. The void in the bone should be ≈8-10mm wide in order to accommodate a 6-8mm vascular graft without a risk of stenosis (Figure 2).

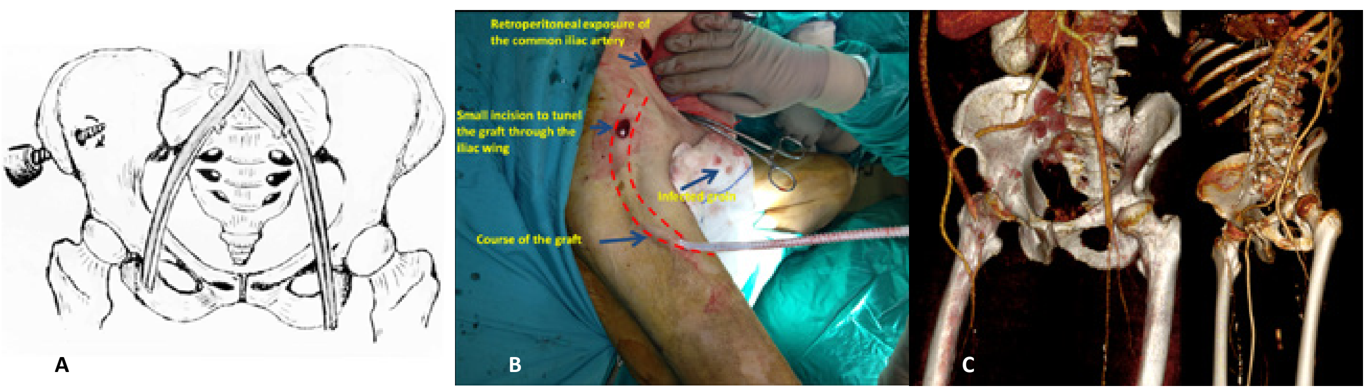


Figure 2. **A.** Schematic of the tunneling of the iliac bone (with a 8-10mm drill) to facilitate passing of the graft.
B. Intra-operative capture emphasizing technical remarks of the procedure.
C. 3D reconstruction of a graft that passed through the wing of the iliac bone and another passed through the obturator foramen.

In the case of tunneling the graft through the muscular lacuna underneath the iliopsoas fascia a transverse incision of the fascia is performed and the route of the tunnel is directed downward and laterally. In this technique the graft is passed lateral to the femoral nerve. Finally, the tunnel reaches the anterolateral side of the upper third of the thigh and then deviates medially to reach the target vessel.

DISCUSSION

In general, prosthetic graft infection in the groin represents a significant source of morbidity after lower limb revascularization procedures and there have been reports suggesting that its occurrence may reach rates as high as 10.6%.[5] A higher risk for major amputation, need for surgical revision and readmission have been recorded for patients with surgical site infection compared to those without.[5] The relative risk for major amputation has been recently reported to be 10-times higher among subjects with graft infection, which is indicative of the significant burden that this complication carries for patients and healthcare professionals.[2] In our series one peri-operative death was recorded, while during 18-months of follow-up 3 major amputations were performed which is close to rates previously reported in the literature.[6] Our results are limited by the low sample volume but most relevant reports share the same limitation. The most recent study on the subject reported on 18 obturator canal bypass procedures performed during 18-years, while others have reported surgical treatment of 14 patients during a similar time interval.[3,6] Nevertheless, besides reporting results in our patient population, the main focus of the present report is to discuss surgical options during extra-anatomical arterial reconstruction in the groin and highlight the technical remarks of these procedures. We believe that these techniques may be useful and relevant in the clinical setting, since removal of the infected prosthesis and extra-anatomical reconstruction (as opposed to simple excision of the prosthesis or graft preservation techniques) is required in the majority of these cases (60% in the current series and 79% in previous reports).[2]

In this regard, we describe three alternative routes to pass a new conduit in patients with a prosthetic graft infection in the groin, needing graft excision and extra-anatomic reconstruction. Each of these techniques has specific advantages and limitations. The obturator bypass cannot be used if infection is present at the medial side of the thigh, involving the adductor muscles. The lateral tunneling during passing a graft through the muscular lacuna can be used in case of limited groin infection not involving the upper thigh. Its origin can be either the common or the external iliac artery. In the presence of extensive groin infection tunneling through the iliac wing provides a better route to avoid entering the groin but may be more technically challenging. Moreover, during this approach using the common iliac artery is recommended as opposed to the external iliac in order to ensure a smooth course of the graft and avoid kinking due to a vertical take-off from the external iliac. An alternative to these techniques is the axillo-femoral or axillo-popliteal bypass, but this is clearly disadvantaged by the significantly longer conduits for which a considerably shorter patency is anticipated.

All patients included in the current series were treated using synthetic grafts, routed through extra-anatomical planes. An alternative option would be to perform in-situ reconstruction using venous conduits, such as the femoral vein, but this technique has mostly been used during treatment of intra-cavitary infections and would only apply in 2/9 of our patients.[7] Moreover, the extent of infection was considered significant and several microbial species (including gram negative) were cultivated in all current cases which could have conferred a higher risk of re-infection in case in-situ rather than extra-anatomical arterial reconstruction was performed. Others have suggested the use of autologous venous grafts even during extra-anatomic revascularization in an attempt to reduce the risk of re-infection.[8] Nevertheless, taking into account that this risk is similarly low if synthetic grafts are used, as long as they are routed extra-anatomically, the additional burden accompanying preparation of autogenous grafts may not be justified. The poor general condition of most of our patients along with the low experience of our center with these techniques, were additional reasons to prefer the use of synthetic grafts.

CONCLUSION

Prosthetic graft infection in the groin is a feared complication in vascular surgery, which usually requires complex surgical procedures to be treated. Despite the fact that the techniques described here may be technically demanding, we believe that they can broaden the armamentarium of the vascular surgeon and aid in the surgical management of this challenging condition.

No conflict of interest.

REFERENCES

1 Dosluoglu HH, Loghmanee C, Lall P, et al. Managementof early (<30 day) vascular groin infections using vacuum-assisted closure alone without muscle flap coverage in a consecutive patient series. J Vasc Surg. 2010;51:1160-6

2 Siracuse JJ, Nandivada P, Giles KA, et al. Prosthetic graft infections involving the femoral artery. J Vasc Surg. 2013;57:700-5

3 Engin C, Posacioglu H, Ayik F, et al. Management of vascular infection in the groin. Tex Heart Inst J. 2005;32:529-34.

4 Katsamouris AN, Giannoukas AD, Alamanos E, et al. Experience with New Techniques for Extraanatomic Arterial Reconstruction of the Lower Limb. Ann Vasc Surg, 2000;14(5): 444-9

5 Davis FM, Sutzko DC, Grey SF, et al. Predictors of surgical site infection after open lower extremity revascularization. J Vasc Surg. 2017;65:1769-1778

6 Bath J, Rahimi M, Long B, et al. Clinical outcomes of obturator canal bypass. J Vasc Surg. 2017;66:160-166

7 Simmons CD, Ali AT, Foteh K, et al. Unilateral inline replacement of infected aortofemoral graft limb with femoral vein. J Vasc Surg. 2017;65:1121-1129

8 Crawford JD, Landry GJ, Moneta GL, et al. Outcomes of unilateral graft limb excision for infected aortobifemoral graft limb. J Vasc Surg. 2016;63:407-13

Influence of obesity and aging on the development of superficial vein thrombosis in patients with primary varicose veins

Christos Karathanos, Petroula Nana, Konstantinos Batzalexis, Georgios Psaras, Konstantinos Spanos, Georgios Kouvelos, Miltiadis Matsagkas, Athanasios D. Giannoukas

Department of Vascular Surgery, University Hospital of Larissa, Faculty of Medicine, School of Health Sciences, University of Thessaly, Larissa, Greece

Abstract:

Introduction: Previous studies have demonstrated the relationship of various factors on etiology of superficial vein thrombosis (SVT), although the association of obesity and aging as independents risk factors for the development of SVT in patients with varicose veins (VVs) remains unknown. The aim of this study was to investigate the role of obesity and aging as risk factors for SVT in patients with primary VVs.

Material and Methods: Retrospective analysis was conducted of prospectively collected data from 230 outpatients with primary VVs. Demographic data, comorbidities and body mass index (BMI) were analyzed. According to medical history, clinical examination and duplex ultrasound (DUS), patients were divided in 2 groups: patients with SVT and VVs, and the control group with VVs but without thrombosis.

Results: 128 patients with a recent documented episode of SVT (85 women, mean age 56.16, SD 13.76) (SVT group) and 102 without any thrombotic event (control group) (75 women, mean age 48.67, SD 12.55) were included. Mean BMI was 27.18kg/m^2 (SD 4.7) in the SVT group and 25.36kg/m^2 (SD 3.6) in control group. Compared to those with a normal BMI (<25kg/m^2), the overweight (25kg/m^2>BMI<30kg/m^2) and obese patients (BMI>30kg/m^2) had an increased risk of SVT by 1.8 - fold (OR 1.85, p=0.038, 95% CI: 1.03-3.32) and 3.3-fold (OR 3.33, p=0.002, 95% CI: 1.53-7.22), respectively. Dislipidemia was associated with a higher risk of SVT (37% vs.18%) (OR 2.3, 95% CI 1.26-4.42). Also, patients > 60yrs showed an increased risk of SVT development by 3.5 fold compared to younger patients. In multiple logistic regression analysis the SVT risk increased by 3.7% (OR 1.037, p=0.001, 95% CI 1.04-1.06) for each year of aging and by 3.5 fold for obese patients (OR 3.5, p=0.003, 95% CI 1.53-8.05).

Conclusions: Obesity and aging appear to increase the risk of SVT development among patients with primary VVs without any other known risk factor.

INTRODUCTION

The prevalence of varicose veins (VVs) in the general population ranges between 20- 60%.[1] Superficial vein thrombosis (SVT) of the lower limbs is a common disease reported to affect 3-11% of the general population. In patients with VVs, the prevalence of SVT ranges from 4- 59%[2,3], although in many cases remains unrecognized and thus their incidence is under reported. Usually SVT is located in the great saphenous vein (GSV) at a rate of 60-80%, while in the small saphenous vein (SSV) occurs more rarely (10-20%).[4] SVT is usually more commonly detected in varicose tributaries than in main GSV trunk. Clinically, it is presented as a sensitive hard cord, circumscribed by a red zone in an area with preexisting varices.[2] Venous thromboembolism (VTE) is a complex disease involving interactions between acquired or inherited predispositions to thrombosis and various risk factors.Recent studies have demonstrated that obesity increases the risk of VTE. [6,7] However it is unclear whether obesity increases risk of SVT in patients with VVs. Another risk factor of VTE is increase in age.[8] Thus, the older the patients, the fewer risk factors are needed for the development of VTE.[9] Currently, the association of obesity and age with SVT in patients with VVs has not been adequately studied. The objective of this study was to evaluate the role of obesity and aging as risk factors for SVT in patients with primary VVs.

Author for correspondence:

Christos Karathanos, MD, MSc, PhD

Department of Vascular Surgery, University Hospital of Larissa, Mezourlo, 41 110 Larissa, Greece
Tel/Fax: +30 2413501739,
E-mail: christoskarathanos@yahoo.gr
ISSN 1106-7237/ 2019 Hellenic Society of Vascular and Endovascular Surgery Published by Rotonda Publications

MATERIALS AND METHODS

Study Design

A retrospective analysis of prospectively collected data of outpatients with an acute or recent episode of SVT and primary VVs referred to our tertiary center between April 2016 and March 2018 was performed. Also patients referred to our department during the same period with VVs without a history of previous SVT were included as control group. Patients with previous episodes of deep vein thrombosis (DVT) or pulmonary embolism (PE), autoimmune disease, malignancy, hepatic or renal insufficiency, recent surgery or trauma, prolonged immobilization, pregnancy, sepsis, use of medications that promotes thrombosis such an oral contraceptives or hormone replacement therapy and patients receiving antithrombotic agents for any other cause such as atrial fibrillation or antiplatelet therapy were excluded.Demographic data, medical history and clinical characteristics were recorded in databases. The data obtained from each patient included gender, age, body mass index (BMI), the presence of other diseases such as hypertension, diabetes mellitus, dislipidemia, history of smoking. Dislipidemia was defined from the presence of serum total cholesterol>200 mg%, LDL>130 mg%, and/or triglycerides>150 mg%. All patients were subjected to renal and hepatic function control, and the levels of blood lipids were measured after 16 hours of fasting.

According to medical history, clinical examination and duplex ultrasound (DUS), patients were divided in 2 groups: patients with SVT and VVs, and the control group with VVs but without thrombosis (SVT or DVT).

Only outpatients with VVs that provided their consent to participate to the study and fulfilled our inclusion criteria were included

Duplex Ultrasound of the Veins

All patients underwent duplex ultrasound examination (DUS) of the superficial and deep veins of the lower limbs. DUS was used to diagnose SVT, to estimate the extent of the thrombus to exclude the presence of deep vein thrombosis (DVT). SVT was diagnosed when superficial veins were not compressible under the probe. DUS were performed by the same vascular surgeon with special interests in duplex scanning.

Body Mass Index

According to the World Health Organization (WHO), definition of obesity is through the BMI. Body mass index was calculated by dividing weight in kilograms by the square of the height in meters (Weight (Kg) / Height (m^2)). Patients were categorized in 3 subgroups: Patients with normal weight (18.5< BMI<24.9 Kg/m^2), overweight (25<BMI<29.9 Kg/m^2) and obese (BMI>30 Kg/m^2).

Statistical analysis

Statistical analysis was carried out with the use of SPSS version 21.0 for Windows, SPSS Inc, Chicago, IL. Chi square tests were applied to initially assess categorical variables. The odds ratios were estimated with confidence intervals (CI 95%). Statistically significant risk factors at p<0.20 were used in a logistic regression model. Interactions of all statistically significant factors at p<0.10 were then examined. All factors entered in the final model had an observed significance level at <0.05.

RESULTS

The study population consisted of 128 patients in the SVT group (43 men, age 56 ± 13) and of 102 patients in the control group (27 men, age 48 ± 12).The GSV was involved in 61 (47%) of the patients,SSV in 1 (0.7%) and varicose tributaries in 66 (52%).

Table 1 shows the main characteristics of the study population. Patients in the SVT group were older than controls (p=0.001). Patients older than 60 years showed an increased risk of SVT by 3.5-fold compared to younger (odds ratio 3.56, p<0.001, 95% CI: 1.90 – 6.68). Mean BMI was 27.18kg/m^2 (SD 4.7) in the SVT group and 25.36kg/m^2 (SD 3.6) in control group. More patients in the SVT group were overweight and obese (Fig1). Compared to those with a normal BMI (<25kg/m^2), the overweight (25kg/m^2>BMI<30kg/m^2) and obese patients (BMI>30kg/m^2) had an increased risk of SVT by 1.8 - fold (OR 1.85, p=0.038, 95% CI: 1.03-3.32) and 3.3-fold (OR 3.33, p=0.002, 95% CI: 1.53-7.22), respectively. Dislipidemia was also associated with a higher risk of SVT (37% vs.18%) (OR 2.3, 95% CI 1.26-4.42).

In multiple logistic regression analysis the SVT incidence increased by 3.7% (OR 1.037, p=0.001, 95% CI 1.04-1.06) for each year of aging and by 3.5 fold for obese patients (OR 3.5, p=0.003, 95% CI 1.53-8.05).

	Diseased Group n (%)	Control group n (%)	p value	Odds Ratio	95% CI
No	128	102			
Gender (Female %)	85 (73.5%)	75 (66.4%)	0.243		
Age(Mean, SD)	48 (12)	56 (13)	0.001		
Age> 60 y	63 (49%)	26 (25%)	<0.001	3.5	1.90-6.68
BMI (Mean, SD)	25 (3.6)	27 (4.7)	0.001		
BMI <24.9 Kg/m^2	44 (34%)	55 (54%)	0.458		
25<BMI<29.9 Kg/m^2	52 (41%)	35 (34%)	0.038	1.85	1.03-3.32
BMI>30 Kg/m^2	32 (25%)	12 (12%)	0.002	3.3	1.53-7.22
Dislipidemia	43 (37%)	18 (18%)	0.054	2.3	1.26-4.42
Smoking	45 (35%)	33(32%)	0.743		
Hypertension	32(26%)	25(24%)	0.598		
Diabetes melitus	23 (20%)	16(16%)	0.672		

Table 1. Main characteristics of the study population

DISCUSION

The main objective of our study was to investigate the role of obesity and aging in patients with primary VVs complicated with SVT without any other known thrombotic risk factor. Our data showed that obesity and aging are associated with an increased risk of SVT development in patients with primary VVs.

The incidence of venous thrombosis rises from 0.001% in childhood to nearly 1% in aged subjects.[8] As described in POST (Prospective Observational Superficial Thrombophlebitis) study the mean age of developing SVT is 60 years and it is generally accepted that people younger than 40 years with a venous thrombotic episode are likely to have a thrombophilia defect.[3]

In most of the studies the mean age of the patients presenting with SVT was lower than in our study. [8,10,11] This may be due the fact that these studies also included patients without VVs, with a previous episode of DVT and/or PE and other risk factors that promotes thrombosis. Moreover in some cases SVT may occur with mild or asymptomatic clinical symptoms and patients may not present for medical attention.

The risk of thrombosis increases with age. [3,12] This may be explained by a combination of factors such as reduced activity, decreased muscle tone, increased morbidity and damaged vein system. Aging causes an increase in fibrinogen, and this leads to formation of thrombin, increase of plasma viscosity and platelet aggregation.[13] Concentration of FVII, IX and others coagulations proteins is increasing without a proportional increase of anticoagulants factors. [13] Interleukin1 (IL-1) and C- reactiven protein (CPR) are increased, suggesting an inflammatory condition, which is important factor in the development of thrombus, particularly in elderly. [23] Furthermore enhanced platelet activity and increase plasminogen activate inhibitor (PAI-1) factor impairs fibrinolytic activity. [13] All the above conclude that the older the patient the fewer risk factors are needed for the development of thrombosis. [3,12]

In recent years, the prevalence of obesity has increased in the western world to a concerning level. At least 250 million people or 7% of the existing population are obese. The number of overweight patients is 2-fold or 3- fold higher. Two –thirds of all men and half of all women in Great Britain are either overweight or obese.[14] In the last decade obesity was more than doubled, with an estimated prevalence of almost 30% of the general population.[15] According to Eurostat data, the prevalence of obesity in Greece is among the highest in the Western Europe. In men above 15 y.o is 26%, and in women 18,2%, which represents the highest and second highest percentage respectively.

Severe obesity is an independent risk factor for the development of VTE, especially when the weight exceeds 175% of the ideal one.[26] Predisposes to venous stasis and is associated with many haemostatic disorders. Obesity enhances thrombosis by increasing prothrombotic factors and impairing fibrinolytic activity.[17,18] Furthermore, obesity is considered as a chronic inflammatory condition that promotes thrombosis, either by increasing IL-1, TNFa and CRP, neither by causing oxidative stress.[18] Several studies have described the association of SVT with obesity.[3,19,20] In our study the overall risk of developing SVT was higher in obese patients, compare to those with normal. The association remained fairly strong after adjustment for age.

Although we recognized obesity as a risk factor of thrombosis in patients with VVs, the presence of a significant number of obese patients may have influenced our results. Over half of our patients were overweight and 25% in the diseased group were morbidly obese. Mobilization of elderly and overweight patients is difficult and this has an obvious influence on their ability to clear thrombus. Obesity also predisposes to postthrombotic syndrome and vein ulcers.[21]

Dislipidemia was also found as a risk factor for SVT. In a recent study, dislipidemia was associated with an increased risk of recurrence of SVT.[22] However, whether obesity on its own or the associated dislipidemia is the triggering factor remains to be clarified. The high levels of nonesterified fatty acids released in obesity increase the hepatic synthesis of triglycerides and very low-density lipoprotein (VLDL), leading to lipid disorder.[23] Lipid disordes are also accompanied by platelet activation and hypercoagulabity with increased factor VII.[24] A possible limitation of our study is that we did not consider the severity of venous disease according to CEAP (Clinical, Etiological, Anatomical, Pathophysiological) classification. Patients with different varicose status were included. Another limitation is that we have not examined the existence / absence of various symptoms in our patients. Also we did not check our patients for insulin resistance, a factor that is associated with a thrombotic predisposition.

CONCLUSIONS

Our study demonstrates that obesity and aging were associated with an increased risk of SVT development among patients with primary VVs without any other known thrombotic risk factors. Larger epidemiological studies are needed to confirm the findings of our study.

REFERRENCES

1 Callam MJ, Epidemiology of varicose veins. Br J Surg 1994;81:167-73

2 Leon L, Giannoukas A, Dodd D, Chan P, Labropoulos N. Clinical significance of Superficial vein thrombosis. Eur J Vasc and Endovasc Surg 2005 ,29:10-17

3 Decousus H, Quere I, Presles E, Becker F, Barreller MT, Chanut M et al: POST (Prospective Observations Superficial Thrombophlebitis) Study Group. Superficial vein thrombosis and venous thromboembolism: a large prospective epidemiological study. Ann Intern Med 2010; 152(4):218-24

4 Lutter KS, Rerr TM, Roedersheimer R, Lohr JM, Sampson MG, Cranley JJ. Superficial thrombophlebitis diagnosed by duplex scanning. Surgery 1991;100;42-46

5 Stein PD, Beemath A, Olson RE. Obesity as a risk factor in venous thromboembolism. Am J Med 2005;118:978-80

6 Oren E, Smith NL, Doggen CJM, Heckbert SR, Lemai-

tre RN. Body mass index and the risk of venous thrombosis among postmenopausal women. J Throm Haem 2006;4:2273-75

7 Rosendaal FR. Thrombosis in the young: epidemiology and risk factors. A focus on venous thrombosis. Thromb Haemost 1997;78:1-6

8 Zollinger RW, Williams RD, Briggs DO. Problems in the diagnosis and treatment of vein thrombosis. Arch Surg 1962;85:34-40

9 Pabinger I, Schneider B. Thrombotic risk in hereditary antithrombin III, protein C, or protein S deficiency. A cooperative, retrospective study. Gesellschaft fur Thrombose- und Hamostasefroschung (GTH) Study Group on Natural Inhibitors.Arterioscler Thromb Vasc Biol 1996;16:742–8

10 Bucciarelli P, Rosendaal FR, Tripodi A, Mannucci PM, De Stefano V, Palareti G, Finazzi G, Baudo F, Quintavalla R. Risk of venous thromboembolism and clinical manifestations in carriers of antithrombin, protein C, protein S deficiency , or activated protein C resistance. Arterioscler, Thromb Vasc Biol .1999;19:1026-33

11 Galanaund JP, Genty C, Presles E, Becker F, Barrellier MT, Chanut M, G et al. Predictive factors for concurrent deep vein thrombosis and symptomatic venous thromboembolism reccurence in case of superficial venous thrombosis. The OPTIMEV study. Thromb Haemost 2011; 105:31-9

12 Wilkerson WR, Sane DC. Aging and thrombosis. Semin Thromb Haemost 2002; 28:555-68

13 House of Commons Health Committee. Obesity – third report of session 2003-2004.Volume 1, 27 (5) 2004

14 Flegal K, Carroll M, Ogden C, Johnson C. Prevalence and trends in obesity among US adults .JAMA 2002; 288: 1723-7

15 Porter JM, Moneta JL. Reporting standards in venous disease: an update. J Vasc Surg 1995; 21:635-645

16 Primrose JN, Davies JA, Prentice CRM, Hughes R, Johnston D. Reduction in factor VII, fibrinogen and plasminogen activator inhibitor-1 activity after surgical treatment of morbid obesity. Thromb Haemost 1992; 68: 396-9

17 Darvall KAL, Sam RC, Silverman SH, Bradbury AW, Adam DJ. Obesity and thrombosis. Eur J Vasc Endovasc Surg 2007;33:223- 33

18 De Moerloose P, Wutschert R, Heinzmann M, Perneger T, Reber G, Bounameaux H. Superficial vein thrombosis of the lower limbs: influence of factor V Leiden, factor II G20210A and overweight. Thromb Haemost 1998;80:239–41

19 Cesarone MR, Belcaro G, Agus G, Georgiev M, Errichi BM, Marinucci R, et al. Management of Superficial Vein Thrombosis and Thrombophlebitis: Status and Expert Opinion. Document. Angiology 2007; 58: 7S-15S

20 Bradbury AW. Epidemiology and etiology of C 4-6 disease. Phlebology 2010; 25 Suppl 1:2-8

21 Karathanos C, Spanos K, Saleptisi V, Tsezou A, Kyriakou D, Giannoukas AD. Recurrence of superficial vein thrombosis in patients with varicose veins. Phlebology 2016;31(7):489-95

22 Eckel R, Barouch W and Ershow A. Report of the national heart, lung, and blood institute national institute of diabetes and digestive and kidney diseases working group on the pathophysiology of obesity-associated cardiovascular disease. Circulation 2002; 105: 2923 -2929

23 Juhan-Vague I and Vague P. Interrelations between carbohydrates, lipids and the haemostatic system in relation to the risk of thrombotic and cardiovascular disease. Am J Obstet Gynecol 1990; 163: 313–323

Applied statistics in vascular surgery Part 2: Correlation analysis and its common misconceptions

Constantine N. Antonopoulos, John D. Kakisis

Department of Vascular Surgery, Medical School, National and Kapodistrian University of Athens, Attikon University Hospital, Athens, Greece

Abstract:

Correlation is a statistical technique assessing the association between two quantitative variables. Before performing a correlation analysis, a scatter plot of the variables used should be drawn to check for linearity. For a normal distribution, the *"Pearson correlation coefficient"* is used, with the assumption that the two variables have to be measured on either an interval or ratio scale, outliers should be taken into consideration and the scatter plot should present fair homoscedasticity. When these assumptions are violated, *"Spearman's rank-order correlation coefficient"* is used, which is the nonparametric alternative version of the Pearson correlation test. Both tests' results range from -1.0 to +1.0, where r > 0 indicates a positive association, r < 0 indicates a negative relationship and r = 0 indicates no relationship. Of note, the closer r is to +1 or -1, the more closely the two variables are related. In case of non-linear (curvilinear) correlation, in which the ratio of change is not constant or when the variable's distribution is not normal, the researcher can perform a logarithmic or other type of transformation for one or both variables. Although very simple in use, correlation analysis carries many misconceptions and misuses. When dealing with them, the researcher should be aware that a conclusion about individuals should never be reached based on group-level data and that correlation does not imply causality.

INTRODUCTION

Increase your chocolate consumption and you might win a Nobel Prize?

A very interesting study appeared in 2012 in the distinguished journal "*New England Journal of Medicine*"[1]. The authors collected a list of countries ranked in terms of Nobel laureates per capita from "*Wikipedia*" and data on per capita yearly chocolate consumption from the official website of the "*Association of Swiss Chocolate Manufacturers*". A statistical correlation test was applied thereafter, and, surprisingly, there was a close, significant linear correlation (r = 0.791, P<0.0001) between chocolate consumption per capita and the number of Nobel laureates per 10 million persons in a total of 23 countries. Moreover, the slope of the regression line allowed them to estimate that it would take about 0.4 kg of chocolate per capita per year to increase the number of Nobel laureates in a given country by 1. The authors concluded that "chocolate consumption enhances cognitive function, which is a "*sine qua non"* for winning the Nobel Prize". But should we all change our diet and start consuming more chocolate to increase the chances of winning a Nobel Prize?

Author for correspondence:

Constantine N. Antonopoulos

Department of Vascular Surgery, Athens University Medical School, Attikon University Hospital, Athens, Greece
E-mail: kostas.antonopoulos@gmail.com
ISSN 1106-7237/ 2019 Hellenic Society of Vascular and Endovascular Surgery Published by Rotonda Publications

What does correlation mean?

Correlation is a statistical term, indicating how strongly pairs of variables are related and implicates association between two quantitative variables. A common example is height and weight; taller people are usually heavier than shorter people, which highlights a correlation between height and weight in the humankind. A classical example of correlation in vascular surgery is the size of the aneurysm and its % percentage of risk for rupture; bigger aneurysms tend to rupture more often. When applying a correlation test in statistics, the researcher needs to answer two basic questions; 1) whether this relationship is positive or negative and 2) which is the strength of the relationship? A positive correlation indicates that both variables increase or decrease in parallel (Figure 1), whereas in a negative correlation the change between the two variables occurs in opposing directions so that increase in one is followed by decrease in the other (Figure 2)[2]. As a result, we conclude that height and weight have a positive correlation. On the contrary, it has been reported that the incidence of diabetes mellitus is rising and at the same time the incidence of aneurysms is declining[3], underlying a negative correlation.

Measures of correlation; the correlation coefficient

In order to measure the direction and strength of the association between two variables, a statistical estimator should be used, which is generally called "*correlation coefficient*" (r). It ranges from -1.0 to +1.0; r > 0 indicates a positive association, r < 0 indicates a negative relationship and r = 0 indicates no relationship, while the closer r is to +1 or -1, the more closely the two variables are related. As a general rule, r=-1.0 to -0.5 or r=0.5 to 1.0 indicates a strong correlation, r=-0.5 to -0.3 or

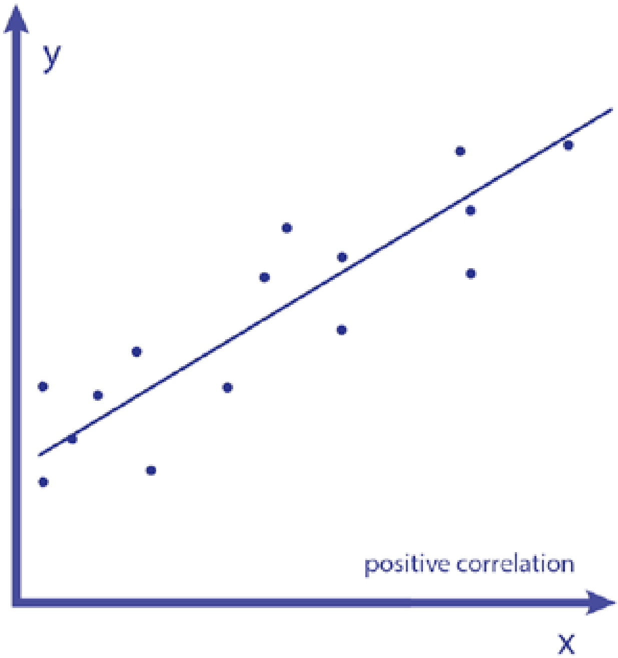


Figure 1.

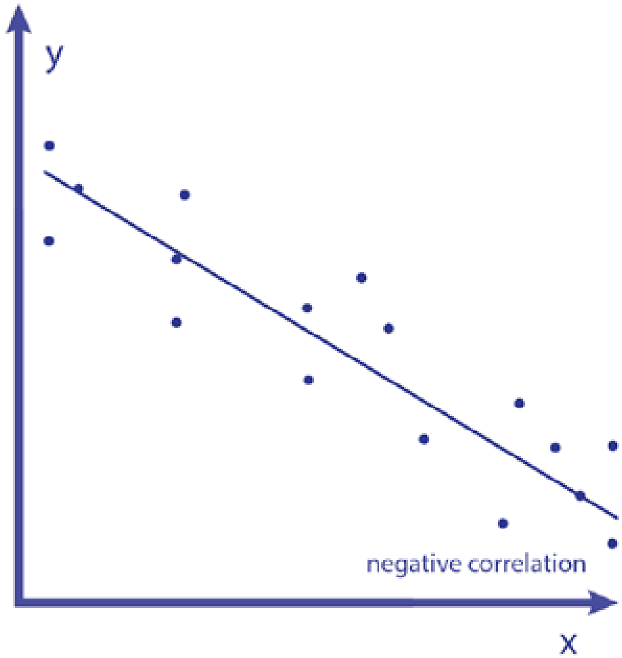


Figure 2.

r=0.3 to 0.5 indicates a moderate correlation, r=-0.3 to -0.1 or r=0.1 to 0.3 indicates a weak correlation, while r=-0.1 to 0.1 indicates a none or a very weak correlation. In any case, p value<0.05 indicates statistical significance, which is the probability that the researcher has found the observed result, or a more extreme one, when the correlation coefficient was in fact zero (null hypothesis). However, someone should be careful, as statistically significant result does not necessarily mean that there is a strong correlation; it simply tests the null hypothesis[2].

Which correlation coefficient should I use?

In cases of normal distribution[4], the "*Pearson product-moment correlation coefficient"*, also called "*Pearson correlation coefficient"* estimates the degree to which a relationship is linear. Its main action is to draw a line of best fit through the data of two variables and indicate how far away all these data points are to this line of best fit. However, it does not represent the slope of the line of best fit. Basic assumptions of *Pearson correlation coefficient* include that the two variables have to be measured on either an interval or ratio scale, while they can be measured in entirely different units. Furthermore, the two variables should form a linear relationship, which can be checked by plotting them on a scatterplot and visually inspect its shape. Usually, the one variable is plotted on the x-axis (horizontally) and the other variable is plotted on the y-axis (vertically).

Moreover, outliers should be taken in to consideration, as they might pose a very large effect on the line of best fit and largely affect the estimate of *Pearson correlation coefficient.* Additionally, the plot should present fair homoscedasticity, which means that the variances along the line of best fit should remain almost the same along the line. Another important characteristic is that *Pearson correlation coefficient* does not take into consideration whether a variable has been classified as a dependent or independent variable. Although *Pearson correlation coefficient* is widely used, it is common in some publications to report the *coefficient of determination, r^2*, which is the square of the *Pearson correlation coefficient r* **(i.e.,** r^2). This index represents the proportion of the variance that is shared by both variables and provides a measure of the amount of variation that can be explained by the model.

The nonparametric version of the Pearson product-moment correlation is called "*Spearman's rank-order correlation coefficient"*, (ρ, also signified by r_s) and measures the strength and direction of association between two ranked variables. This is an alternative to the Pearson correlation index, which is used when assumptions of the Pearson correlation are violated. Of note, in cases of non-linear (curvilinear) correlation, in which the ratio of change is not constant or when the variable's distribution is non-normal, the researcher can perform a logarithmic or other type of transformation for one or both variables.

Other types of correlation coefficient

In cases of ordinal association between two measured quantities, the "*Kendall rank correlation coefficient*", commonly referred to as "*Kendall's tau coeffi*cient", is used and investigates the similarity of the orderings of the data when ranked by each of the quantities[5]. When one variable is continuous and the other variable is dichotomous then the "*point-biserial correlation*" should be used. (eg. correlation between a continuous variable, which is the monthly income measured in Euros and a binary variable, which is gender, with males and females as categories)[6]. Additionally, the "*biserial correlation*", which is different from the "*point-biserial" correlation*", is recommended when the dichotomous (binary) variable has an underlying continuous distribution; for example, if 0=low intelligence quotient (IQ), and 1=high IQ, the researcher should use the biserial and not the point-biserial - correlation.

Major pitfalls in correlation analysis or "*should I consume more chocolate*"?

Although many papers might present data on the correlation between two variables, some important issues require special attention when running a correlation test. First of all, a conclusion about individuals should never be reached based on group-level data. As a result, a correlation coefficient at country level, must not be used to reach a conclusion about the individual level. Therefore, given that no data are known on how much chocolate the Nobel laureates consumed, any conclusions are rather speculative. Another major misinterpretation of correlation is the idea that it implies causality. Correlation only assesses the intensity of association between two variables and never explains the nature of this agreement[7]. The two variables may show a correlation not because they are influenced by each other but because they are both influenced by the same confounder. As a result, chocolate consumption and winning the Nobel Prize do not have a causal relation. In order to point out meaningless correlation, researcher use the term "*nonsense" or "spurious" correlation*" in which "no sensible natural causal interpretation can be provided"[8]. A classification of correlations has been provided by Haig in order to highlight errors in interpreting statistically significant correlations. Consequently, someone has to be very careful when dealing with significant associations.

CONCLUSIONS

The correlation coefficient is a popular measure of the association between two variables and can easily summarize a scatterplot in a single number. Two main estimators are commonly used in research, namely the "*Pearson product-moment correlation"* and its nonparametric alternative version, called "*Spearman's rank-order correlation coefficient"*. Although very simple in implementation and interpretation, researchers should clearly understand the assumptions behind conducting a correlation analysis and explain them in their methods in order to avoid common errors and ecological fallacies.

No conflict of interest.

REFERENCES

1 Messerli FH. Chocolate consumption, cognitive function, and Nobel laureates. N Engl J Med. 2012;367(16):1562-4

2 Swinscow TDV. 11. Correlation and regression. Statistics at Square One. 9th ed: BMJ Publishing Group, Revised by M J Campbell, University of Southampton; 1997

3 Antonopoulos CN, Liapis CD. Aneurysms and Diabetes Mellitus: A Strange Symbiosis? Cardiology. 2018;141(2):123-4

4 Antonopoulos C, Kakisis J. Applied statistics in vascular surgery Part 1: Choosing between parametric and non-parametric tests. HJVES. 2019;1(1)

5 David ST, Kendall MG, Stuart A. Some questions of distribution in the theory of rank correlation. Biometrika. 1951;38(1-2):131-40

6 Linacre J. The Expected Value of a Point-Biserial (or Similar) Correlation. Rasch Measurement Transactions. 2008;22(1):1154

7 Veličković V. What Everyone Should Know about Statistical Correlation. American Scientist. 2015;103:26-9.

8 Haig B. D. What Is a Spurious Correlation? Understanding Statistics. 2 (2)2003. p. 125-32

Tracheo-carotid and oesophageal fistula following prolonged endotracheal intubation

Maria P. Ntalouka[1,2], Miltiadis Matsagkas[3,4], George Kouvelos[3,4], Konstantinos Stamoulis[1], Vasileios Koulouras[5], Elena Arnaoutoglou[1,2]

[1]Department of Anesthesiology, Faculty of Medicine, School of Health Sciences, University of Thessaly, Larissa University Hospital, Larissa, Greece
[2]Department of Anesthesiology, Faculty of Medicine, School of Health Sciences, University of Ioannina, Ioannina University Hospital, Ioannina, Greece
[3]Vascular Surgery Department, Faculty of Medicine, School of Health Sciences, University of Thessaly, Larissa University Hospital, Larissa, Greece
[4]Vascular Surgery Unit, Faculty of Medicine, School of Health Sciences, University of Ioannina, Ioannina University Hospital, Ioannina, Greece
[5]Intensive Care Unit Department of ICU, University Hospital of Ioannina, Ioannina, Greece

Abstract:
Tracheo-carotid and tracheo-esophageal fistula are rare and devastating complications of inappropriate endotracheal tube cuff handling. We report a case of a patient presented with intermitted bleeding episodes through the mouth and the endotracheal tube. The patient suffered the initial episode of hemorrhage 15 days after intubation, which was controlled with volume expansion of the endotracheal tube cuff. Afterwards she underwent several other hemorrhagic episodes, which were also managed each time with further cuff expansion. At last, during many repeated surgical attempts to control the repeated and profound haemorrhage, the diagnosis of tracheo-carotid and tracheo-esophageal fistula were subsequently made. Despite the aggressive efforts, the patient died due to septic shock and multiple organ failure. Optimal endotracheal tube cuff handling followed by immediate and stepwise therapeutic approach in case of a complication, along with the very high suspicion index can prove lifesaving in patients requiring prolonged endotracheal intubation.

INTRODUCTION

The improvement of endotracheal tubes (ETTs) and the advances in mechanical ventilation have shortened the duration of ventilatory support. Cuffed ETTs remain the cornerstone for securing the airway and facilitating gas exchange. Inappropriate ETT handling remains one of the leading causes of complications related to mechanical ventilation, with cuff pressure, accidental extubation, mucosal ulcerations, granulomas, tracheal stenosis, and tracheo-oesophageal fistulae being the most frequent.

The first tracheo-carotid artery fistula following endotracheal intubation was reported in 1984. In 2007 tracheo-oesophageal fistula (TEF) and massive haemorrhage from tracheo-arterial (left subclavian artery) fistula (TAF) described together for the first time.[1, 2] Both TAF (abnormal linking between the trachea and an artery) and TEF (between the trachea and the oesophagus) are rare (0.1-1% and 0.5-1%), with extremely low survival rates (14,3%) and may develop even within a few hours of intubation. The incidence of hemorrhage complicating the fistula between the trachea and the common carotid artery is 4.3% [1, 2, 3, 4, 5].

Author for correspondence:

Maria P. Ntalouka

Department of Anaesthesiology, Faculty of Medicine, School of Health Sciences, University of Thessaly, Larissa, 41334, Greece
Tel: +30 6973688099, Fax: +30 2413502953,
E-mail: maria.ntalouka@icloud.com
ISSN 1106-7237/ 2019 Hellenic Society of Vascular and Endovascular Surgery Published by Rotonda Publications

CASE

A 37-year-old female with Guillain-Barre syndrome (GBS), leading to progressive respiratory failure, was transferred intubated to our hospital from another country, after several failed extubation attempts, due to several episodes of profound bleeding through the mouth and the endotracheal tube (ETT), which were managed with further volume expansion of the ETT cuff each time.

During admission the patient was mechanically ventilated and a small (~150ml) and repeated bleeding through the ETT and the mouth was observed. Chest x-ray revealed a right pneumothorax (a right chest tube was placed), and round distention of trachea (extra thoracic segment) corresponding to the ETT cuff (Fig. 1). Lab findings were not specific, except for a mild impairment of hepatic function (INR:1.22, aPTT:31.9, AST/ALT:199/240, ALP:274, γGT:644, LDH:1461, CPK:230 CK-MB:26). As far as the antibiotics are concerned the patient was treated with ceftazidime which was changed to sultamicillin and finally to cefotaxime (all three of them at maximum dosage).

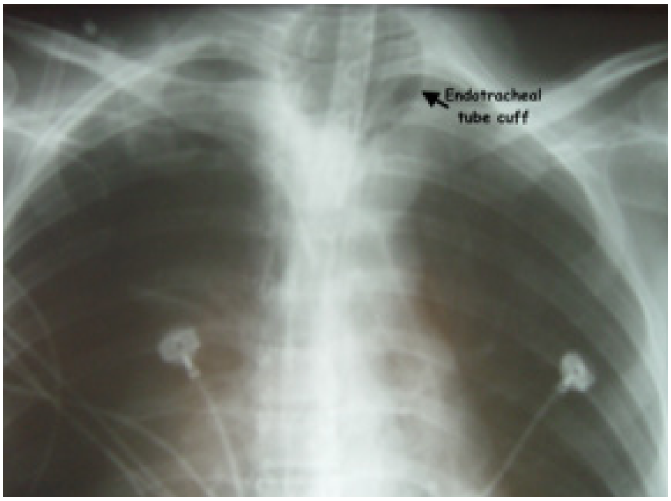


Figure 1. Chest Xray revealing the large endotracheal tube cuff

An urgent angiography of aortic arch revealed leak of contrast from the proximal segment of the right common carotid artery along with great external deviation of the vessel (Fig. 2), and the patient underwent emergency operation. A partial sternotomy and a right oblique cervical incision parallel to sternocleidomastoid muscle confirmed the longitudinal erosion of the right common carotid 1 cm after its origin and revealed a fusiform disruption of the trachea around the ETT cuff, ipsilateral complete erosion of the anterior tracheal wall and protrusion of the cuff through the deficit (Fig. 3). The carotid artery erosion was treated with a patch angioplasty with bovine pericardium and a tracheostomy was performed at the level of the lesion.

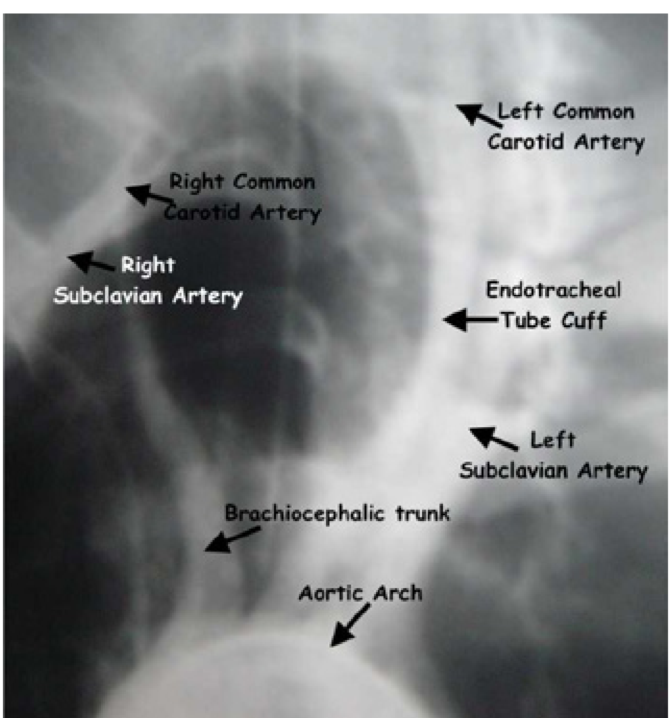


Figure 2. Angiography revealing the right carotid leak of contrast from the proximal segment and the great external deviation of the vessel

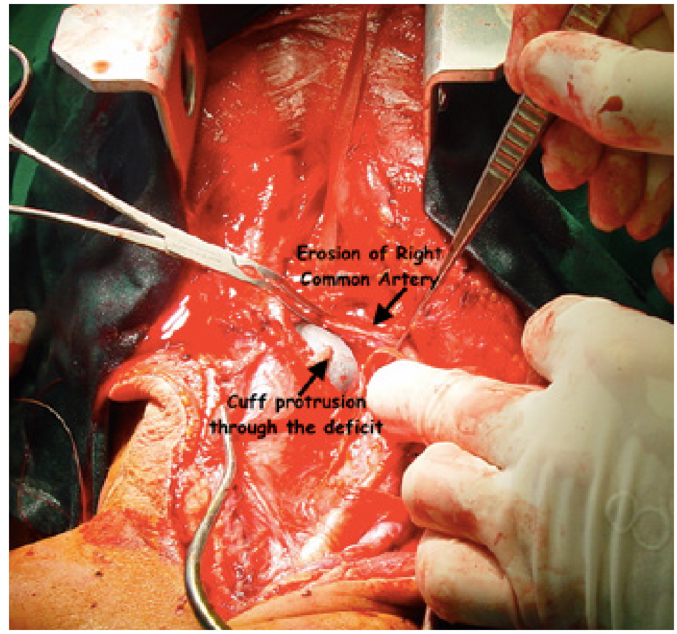


Figure 3. Oblong erosion of the right common carotid 1 cm after its origin, fusiform distention of the trachea around the ETT cuff, ipsilateral complete erosion of the anterior tracheal wall and protrusion of the cuff through the deficit after the partial sternotomy and the supraclavicular incision parallel to sternocleidomastoid muscle.

On the 6th postoperative day two new episodes of bleeding were observed. However, CT angiography revealed no leak of contrast. A second operation through a median sternotomy this time revealed an extensive longitudinal oesophageal erosion along the nasogastric tube with suppuration and mediastinitis, which was treated with left extomosis of the central segment, mediastinal lavage, and insertion of drainage tubes. A new erosion of the right common carotid artery at its origin was revealed and was treated with ligation of the right common carotid and closure of its ostium at the level of innominate artery (Fig. 4).

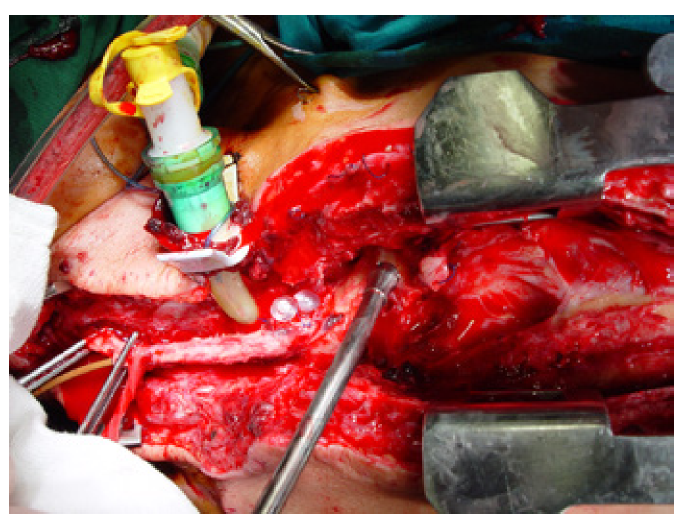


Figure 4. Extensive longitudinal oesophageal erosion along the nasogastric tube treated with left extomosis of the central segment, mediastinal lavage, and insertion of drainage tubes. Right common carotid artery erosion at its origin.

Postoperatively in an attempt to control the septic shock the patient was treated with broad spectrum antibotics and aggressive support. Despite the efforts she unfortunately died twenty-one days after the admission to our Hospital due to septic shock and multiple organ failure.

DISCUSSION

We present the existence of non-traumatic, iatrogenic tracheo-carotid and a tracheo-esophageal fistula due to prolonged endotracheal intubation and inappropriate handling of the ETT cuff volume and pressure (continuous further volume expansion of the ETT cuff). Nowadays, cuffed ETTs remain the cornerstone for securing the airway and facilitating gas exchange, while the improvement of endotracheal tubes and the advances in mechanical ventilation have shortened the duration of ventilatory support [4]. However, prolonged endotracheal intubation and inappropriate ETT handling compose the leading causes of complications related to mechanical ventilation [6, 7].According to the current literature, early tracheoastomy is suggested, if possible at 7 days after initial intubation [8]. Inappropriate handling of the ETT and ETT cuff volume and pressure may lead to mucosal injury, derangement of microcirculation, mucosal ulceration and eventually tracheal wall necrosis, and TAF formation, especially when combined with hypotension, a high-positioned innominate artery and excessive tube movements [1]. When it comes to TEF formation steroids, advanced age, high airway pressure, nasogastric tubes and respiratory and oesophageal infections have been considered as the main causes [7, 9].

As far as the prevention of TAF and TEF is concerned, endotracheal tubes with high compliance, low pressure cuff with careful intra-cuff pressure monitoring, intermittent deflation of the cuff, prevention of infection and hypotension, and improvement in the patient's nutritional and immunologic status are strongly advised to be part of the routine management of any patient requiring intubation [7]. In our patient inappropriate ETT handling (an early tracheostomy was not perform) and the continuous increases in the cuff volume, as a measure to control the bleeding, were the main causes for both the fistulae. Prolonged mechanical ventilation, nasogastric tube placement, steroid administration and septic and hemorrhagic shock further compromised the tracheal wall necrosis [1, 3, 7].

Early diagnosis of TAF and TEF is difficult, requiring a high suspicion index. Any patient with an ETT or tracheostomy tube, with >10 ml of haemorrhage from the tip of the tube or unexplained upper airway leak should be immediately evaluated for TAF or TEF, respectively. Once massive intra-tracheal bleeding is observed, the patient should be transferred to the operating room immediately, without manipulating the ETT or expanding the volume of the ETT cuff, for explorative thoracotomy (median sternotomy with cervical expansion) [1, 3, 10].

Both lesions' management remain challenging, with extremely high mortality rates and early treatment is fundamental for survival. The over-inflation of the cuff should be one of our main concerns, followed by digital compression of the artery for TAF. If TEF co-exists, the ETT cuff should be placed distal to the TEF within the trachea, while endotracheal and oesophageal stenting may be necessary [3, 7, 10]. Post-operatively, the cuff's pressure should be 20-30 cmH_2O with the tip of the tube not being compressed against the tracheal wall, while only flexible nasogastric tubes should be used. Frequent cuff pressure monitoring, minimal endotracheal tube movement during suctioning, optimal nutrition, and spontaneous breathing when possible are essential for a favourable result [7].

CONCLUSION

We report the co-existence of non traumatic, iatrogenic TAF and TEF due to inappropriate ETT cuff handling. Preventive measures, high suspicion index, early diagnosis and surgical treatment proves of outmost importance when dealing with TAF and TEF.

REFERENCES

1 Hammed AA, Mohamed H, Al-Mansoori M. Acquired tracheoesophageal fistula due to high intracuff pressure. Ann Thorac Med. 2008; 3: 23-25

2 Elstein M, Legg NJ, Murphy M, Park DM, Sutcliffe MM. Guillain-Barré syndrome in pregnanct. Respiratory paralysis complicated by a fatal tracheo-innominate artery fistula. Anaesthesia 1971; 26: 216-224

3 Kaur D, Anand S, Sharma P, Kumar A. Early presentation of postintubation tracheoesopageal fistula: Perioperative anesthetic management. J Anaesthesiol Clin Pharmacol. 2012; 28:114-116

4 LoCicero J 3rd. Tracheo-carotid artery erosion following endotracheal intubation. J Trauma 1984; 24: 907-909

5 Hung JJ, Hsu HS, Huang CS, Yang SY. Tracheoesophageal fistula and tracheo-subclavian artery fistula after tracheostomy. Eur J Cardiothorac Surg 2007; 32: 676-8

6 Stauffer JL, Olson DE, Petty TL. Complications and consequences of endotracheal intubation and tracheostomy. A prospective study of 150 critically adult patients. Am J Med. 1981; 70: 65-78

7 Grant CA, Dempsey G, Harrison J, Jones T. Tracheo-innominate artery fistula after percutanenous tracheostomy: three case reports and a clinical review. Br J Anesth 2006; 96:127-131

8 Adly A, Youseef TA, El-Begermy MM, Younis HM. Timing of tracheostomy in patients with prolonged endotracheal intubation: a systemic review. Our Arch Otorhinolaryngol 2018; 275: 679-690

9 Benjamin B. Prolonged intubation injuries of the larynx: Endoscopic diagnosis, classification, and treatment. Ann Otol Laryngol 2018; 127: 492-507

10 Komatsu T, Sowa T, Pujinaga T, Handa N, Watanabe H. Tracheo-innominate artery fistula: two case reports and a clinical review. Ann Thorac Cardiovasc Surg 2013; 19: 60-62

Coexistence of infrarenal and supra-celiac aortic aneurysm. Endovascular Treatment using the Chimney technique

Petroula Nana[1,2], Konstantinos Spanos[2], Miltiadis Matsagkas[1,2]

[1]Department of Surgery- Vascular Unit, Faculty of Medicine, School of Health Sciences, University of Ioannina, Ioannina, Greece
[2]Department of Vascular Surgery, Faculty of Medicine, School of Health Sciences, University of Thessaly, Larissa, Greece

Abstract:

During last decade, the complete endovascular approach in complex aortic aneurysm improved the morbidity and mortality rates in these cases. A 69-year-old male patient presented with a coexistence of an infrarenal abdominal aortic aneurysm (AAA) and an additional saccular aortic aneurysm, just above the orifice of the celiac trunk. The two separate aneurysms were treated simultaneously by endovascular means; the infrarenal aneurysm using a conventional infrarenal endograft, while the supra-celiac one combining a short custom thoracic endograft and a covered stent for the celiac axis, applying the chimney technique. The computed tomography angiography of discharge showed a patent celiac stent graft with complete aneurysms exclusion without any endoleak. During a 5-year follow-up, the graft remained patent and the diameter of both sacs was diminished. Nowadays, combining several endovascular techniques, complex aortic pathologies can be treated with a minimal invasive approach.

INTRODUCTION

During the last decade, a widespread interest in the completely endovascular treatment of the complex aortic anatomy aneurysms has been presented.[1] The improved morbidity and mortality rate make more attractive a completely endovascular approach in many cases, especially in patients presenting several comorbidities. Short-term results could be favorably compared to open repair, with a mortality rate at 3.7%, primary patency at 94.8%, and primary-assisted patency at 95.1%.[2] Even in long-term follow-up, a total endovascular repair of complex aortic anatomy aneurysms seems safe and durable with an aneurysm-related survival rate at 91% and late occurring visceral stent occlusion at 4.4% at 5 years.[3]

We herein report a case of a patient diagnosed with two coexisting aortic aneurysms; an infrarenal and a supra-celiac one, treated by endovascular means, using the chimney technique.

CASE PRESENTATION

A 69-year-old man presented with the diagnosis of an infrarenal abdominal aortic aneurysm (AAA) of 56 mm and a suprarenal saccular aneurysm of 40mm, just above the orifice of the celiac axis (Image 1). His medical history was significant for hypertension, dyslipidemia, coronary artery disease with previous CABG and heart failure with an ejection fraction at 40%, under treatment.

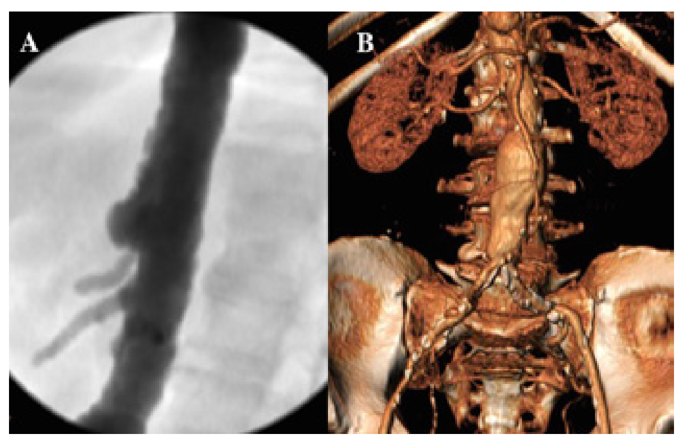


Image 1. Pre-operative CTA revealed a para-visceral saccular aneurysm just above the orifice of the celiac axis (Panel A) and an infrarenal AAA (Panel B).

After an extensive discussion with the patient about the different management options, an endovascular approach with a conventional graft for the infrarenal aneurysm and a custom-made tapered graft, combined with a self-expanding graft for the celiac axis to exclude the supra-celiac aneurysm using the chimney technique was decided. Under general anesthesia, a bifemoral and a left brachial access were used. The celiac trunk was catheterized and stented using the self-expanding covered stent (Viabahn, Gore, 8x100 mm). For the treatment of the suprarenal aneurysm, a custom-made conical graft (Bolton Relay 34-30x80mm) was inserted in the proximal aortic aneurysm. (Image 2) The distances between the splanchnic vessels permitted such a solution, as an adequate

Author for correspondence:

Miltiadis Matsagkas, MD, PhD, FEBVS

Department of Vascular Surgery, Medical School,
University of Thessaly, Larissa University Hospital,
Mezourlo, Larissa, Greece
Tel: +30 6945585876, E-mail: milmats@gmail. com
ISSN 1106-7237/ 2019 Hellenic Society of Vascular and Endovascular Surgery Published by Rotonda Publications

sealing zone of 15mm between the coeliac trunk and the superior mesenteric artery (SMA) was available. The proximal landing zone had a median diameter of 29mm while the distal supra-SMA diameter was 25.7mm. The tapered morphology of the aorta at this level set the indication of a custom-made endograft. For the infrarenal AAA, an Excluder endograft (Gore & Associates, Inc, CA, USA) was used (main body 26-14.5x160 mm, contralateral limb 16x120mm). Completion angiography confirmed successful sac exclusion with no endoleak (Image 3). Post-operatively, the patient remained stable, while the renal or hepatic function were recorded within normal limits. No spinal cord ischemia symptoms were arisen post-operatively. The computed tomography angiography of 5th day showed a complete exclusion of the aneurysms, a patent celiac stent graft and no endoleak. The patient was discharged the 6th post-operative day under double antiplatelet treatment with aspirin 100mg and clopidogrel 75mg daily for the first 6 months and single antiplatelet therapy with aspirin 100mg thereafter. During a 5-year follow-up, the aneurysmal sacs were eliminated, the graft of the celiac artery remained patent, and no endoleak was detected. (Image 4) This case presentation was approved by the Institution's Review Board.

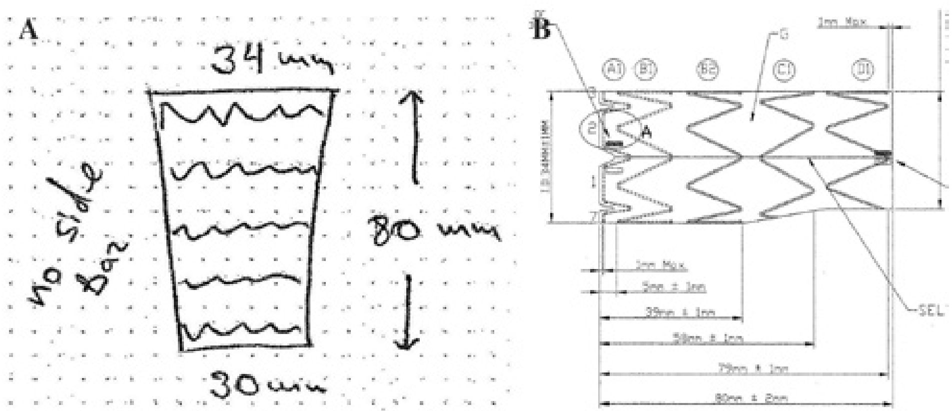


Image 2. A fast custom-made device offered an off-the-shelf solution to the patient (Panel A & B).

Image 3. The celiac trunk was catheterized and stented (Panel A & B). The custom-made endografts was deployed (panel C). Completion angiography revealed the exclusion of the sacs and the patency of the grafts (Panel D).

Image 4. 5-year follow-up CTA confirmed the patency of the grafts (Panel A & B) as well as the exclusion and shrinkage of the sacs (Panel C).

DISCUSSION

Chimney technique has gained popularity as an off-the-shelf technique for the treatment of complex abdominal aortic aneurysms, offering a safe and durable approach when fenestrated or branched endografts are contraindicated or unavailable.[4] It permits a tailor-made solution which is eligible to the specific anatomical characteristics of the patient, using common material that are available at the surgeon's daily routine practice.[5] The off-the-shelf endografts offer the advantage of the faster assessment-to-treatment time in a single or multiple stage procedures when aneurysmal deformation includes the para-visceral aorta.[5]

In this case, standard endovascular aortic aneurysm repair (EVAR) was applied for the infrarenal aneurysm. For the isolated para-visceral aneurysm, a custom-made solution was selected. Despite its minimal diameter (40mm), the saccular morphology of the aneurysm as well as its location, just above the orifice of the celiac axis, were the main indications for an immediate endovascular approach. According to the latest ESVS Guidelines for the management of the aorto-iliac aneurysms, saccular aneurysms should be confronted as a separate aortic pathology. An infection should be excluded in any case. The optimal treatment should be decided according to the patient's specific anatomy and risk factors. The higher risk of rupture should be kept in mind and an early interventional repair may be considered.[6] Furthermore, in this case, the extended comorbidities of the patient eliminated any option of open repair.

Chimney technique was feasible as celiac axis was patent and there was a sufficient distal landing zone at the moment of treatment. Although, no conventional thoracic endograft was applicable in this case, as all thoracic endografts have a minimum length of 100mm. As the parallel graft of the celiac trunk available was 100mm in length, a mismatch between the two endografts was inevitable. Furthermore, the conical formation of the aorta excluded any standard solution. In this case, a fast custom-made tapered endograft of 80mm offered a technically feasible alternative. A more complicated solution, as a branch or fenestrated device could be achievable in this patient. The need for a four-vessel catheterization and stenting for a limited aneurysmal disease, as well as the cost-effectiveness of these devices, enforced the decision through a parallel-graft technique.

In the current literature, there is scarce information concerning the isolated para-visceral aneurysms. A case report of a mycotic para-visceral aneurysm treated with fenestrated endograft has already been reported.[7] Chimney technique offers an alternative solution when fenestrated or branched devices are contraindicated or unavailable in complex aortic anatomies as well as in cases of para-visceral aneurysms.[5] The eligibility of the technique permits an endovascular approach at the majority of the patients with complex aneurysmal disease, excluding only the 17% of them at one series.[5] Furthermore, the short and long-term encouraging data confirm the safety and durability of the method in these cases.[2, 3]

CONCLUSION

Para-visceral aortic aneurysms could be completely treated with endovascular technique. In the few cases reported so far endovascular repair showed promising results, in terms of safety and durability. An extended pre-operative planning and knowledge of all disposable means is demanded to offer an efficient treatment to each patient's anatomic characteristics.

Acknowledgements: None

No conflict of interest.

REFERENCES

1 1. Moulakakis K., Mylonas S., Avgerinos Eet al, The chimney graft technique for preserving visceral vessels during endovascular treatment of aortic pathologies, JVS, May 2012, 55 (5):1497-1503

2 2. Reilly L., Rapp J., Grenon M et al,, Efficacy and durability of endovascular thoracoabdominal aortic aneurysm repair using the caudally directed cuff technique, JVS, 2012, 56 (1):53-64

3 3. Walker J., Kaushik S., Hoffman M et al, Long-term durability of multibranched endovascular repair of thoracoabdominal and pararenal aortic aneurysms, JVS, 2019, 69 (2): 341-7

4 4. Donas K., Torsello G., Bisdas Tet al, Early Outcomes for Fenestrated and Chimney Endografts in the Treatment of Pararenal Aortic Pathologies Are Not Significantly Different: A Systematic Review with Pooled Data Analysis, J Endovasc Ther, 2012, 19: 723-8

5 5. Gasper W., Reilly L., Rapp J. et al, Assessing the anatomic applicability of the multibranched endovascular repair of thoracoabdominal aortic aneurysm technique, JVS, 2013, 57 (6):1553-1558

6 6. Wanhainen A, Verzini F, Van Herzeele I et al, European Society for Vascular Surgery (ESVS) 2019 Clinical Practice Guidelines on the Management of Abdominal Aorto-iliac Artery Aneurysms, Eur J Vasc Endovasc Surg 2018: 1-97

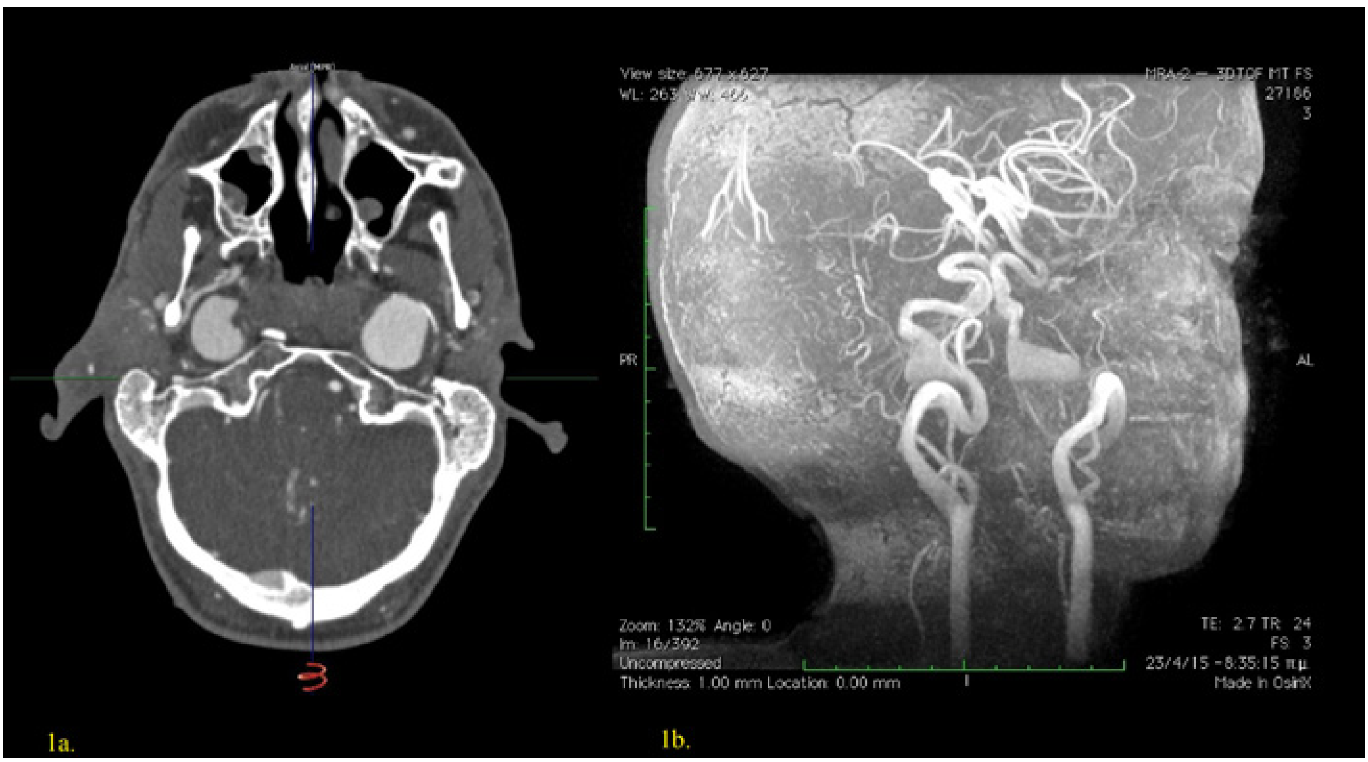
7 7. Kinney EV, Kaednick HW, Mitchell RA et al, Repair of mycotic para-visceral aneurysm with fenestrated stent-graft, J Endovasc Ther, 2000, 7 (3):192-7

VASCULAR IMAGE

Bilateral giant internal carotid artery aneurysms of the cavernous segment in a young male

Stylianos Koutsias[1], Konstantinos Spanos[2], Athanasios D. Giannoukas[2]

[1]Vascular Surgery Department, University Hospital of Larissa, Faculty of Medicine, School of Health Sciences, University of Thessaly, Larissa, Greece
[2]Department of Surgery, Vascular Surgery Unit, School of Medicine, University of Ioannina, Ioannina, Greece



A 21-year old male, with known history of neurofibromatosis type 1, presented to the emergency department with high blood pressure (180/60 mmHg) and headache. Physical examination revealed a bruit and a palpable mass in the neck area bilaterally. Duplex scanning showed bilateral severe carotid artery tortuosity and a suspicion of bilateral internal carotid artery aneurysms in a more distal part of the arteries. A computed tomography angiography confirmed (Image 1a) the presence of bilateral giant internal carotid aneurysms (right side 26mm, left side 34mm in diameter) of the cavernous segment. A magnetic resonance angiography (Image 1b) was undertaken at a later stage to help in the planning of their treatment, however patient denied treatment. This pathology is rare; open repair is accompanied with high morbidity and mortality, while endovascular approach has been suggested recently as another option.

Author for correspondence:

Konstantinos Spanos, MD, MSc, PhD

Department of Vascular Surgery, University Hospital of Larissa, Faculty of Medicine, School of Health Sciences, University of Thessaly, Larissa, Greece
E-mail: spanos.kon@gmail.com, Tel.: +306948570321
ISSN 1106-7237/ 2019 Hellenic Society of Vascular and Endovascular Surgery Published by Rotonda Publications

Forthcoming Events

LIVE 2019
May 9-11, 2019
Larissa Imperial Hotel
Larissa, Greece

68th Congress of the European Society of Cardiovascular and Endovascular Surgery (ESCVS)
May 22-25, 2019
The University Medical Center Groningen
Groningen, The Netherlands

Critical Issues in Aortic Endografting 2019
May 23-24, 2019
Hilton Liverpool City Centre
Liverpool, UK

SVS/VAM 2019
June 12-15, 2019
Gaylord National Resort & Convention Center
National Harbor, MD

20th European Venous Forum Meeting
June 27-29, 2019
Zurich University
Zurich, Switzerland

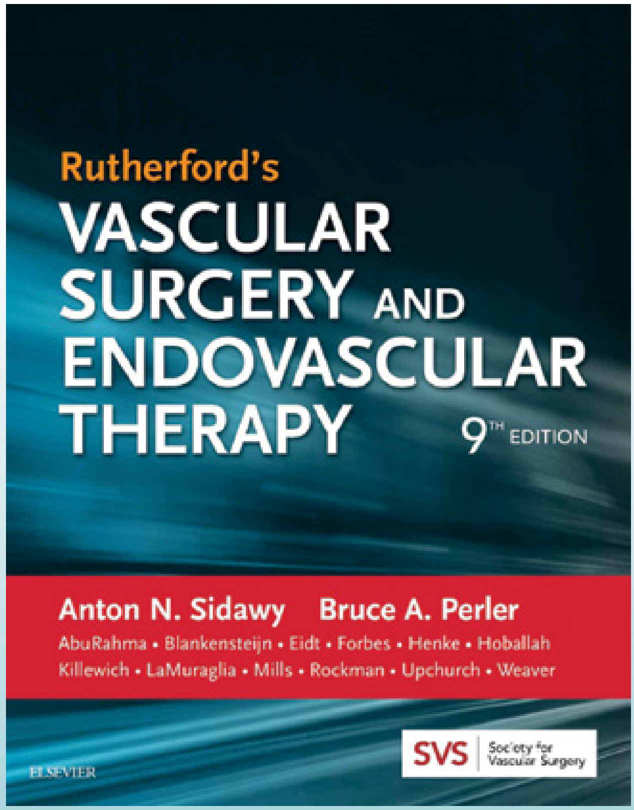
ISTH 2019
July 6-10, 2019
Melbourne, Australia

CIRSE 2019
September 7-11, 2019
Barcelona, Spain

ESVS 2019 - The 33rd European Society for Vascular Surgery Annual Meeting
September 23-27, 2019
Hamburg, Germany

Athens Cardiovascular Symposium
November 8-9, 2019
Athens, Greece

VEITHsymposium
November 19-23, 2019
New York Hilton-Midtown
New York, NY



2- Volume Set

A Health Science Title
Publication Date: 25/05/2018
ISBN-13: 9780323427913
Subject Area: Medicine - Vascular Surgery
PRICE: 330€

Through nine outstanding editions, Rutherford's Vascular Surgery and Endovascular Therapy has been **the gold standard text in this fast-changing, complex field**. Published in **association with the Society for Vascular Surgery**, this state-of-the-art reference by Drs. Anton N. Sidawy and Bruce A. Perler is a must-have for vascular surgeons, interventionalists, vascular medicine specialists, and trainees, as well as general surgeons, interventional radiologists, and cardiologists that depend upon "Rutherford's" in their practice. It offers **authoritative guidance from the most respected and innovative global thought leaders and clinical and basic science experts** in the diagnosis and treatment of circulatory disease.

Rotonda Publications
8 Kamvounion Str, 54621 Thessaloniki, Greece
Tel: +30 2310 212 212, +30 2310 285 785, Fax: +30 2310 280 287

www.ipokratis.gr
e-mail: ipokratis@ipokratis.gr

Hellenic Journal of Vascular and Endovascular Surgery

- Submit your paper at journal's official site: https://heljves.com/
- No fees for paper submission and publication
- Open access to all of journal's content

PUBLICATIONS ROTONDA

Hellenic Society of Vascular and Endovascular Surgery