Rupture of the infrarenal aorta during the 1st postoperative day after emergent endovascular treatment of acute Type B aortic dissection

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Abstract:

Acute Type B Aortic Dissection (ATBAD) is a vascular emergency requiring prompt diagnosis and treatment to avoid serious complications. Complicated cases are treated invasively, preferably by endovascular means.

We present a 60-year old woman who was admitted due to ATBAD, with the primary entry tear located just distal to the level of the Left Subclavian Artery (LSA) origin. She was initially treated conservatively, however on the 2nd day due to refractory pain she was subjected to emergency TEVAR with LSA coverage.

The procedure was uneventful. During the 1st postoperative day the patient complained for back pain and underwent a new CT indicating successful endograft deployment without remarkable changes from the distal thoracic and abdominal aorta compared to the preoperative imaging. During the same night the patient became hemodynamically unstable and died. Post-mortem CT indicated infra-renal aortic rupture.

ATBAD may result in mortality even if prompt treatment has been undertaken.

INTRODUCTION

Acute aortic syndromes are disorders of thoracic and abdominal aorta, that require urgent evaluation and treatment, with acute aortic dissection (AD) being the most frequent and most lethal in-between them¹. Uncomplicated Acute Type B Aortic Dissections (ATBAD) initially undergo conservative management with close monitoring in an intensive care unit (ICU) setting, aiming to lower blood pressure and heart rhythm, relieve pain and allow for an uneventful recovery during the acute phase^{2, 3}. On the contrary, complicated cases (rupture, malperfusion, aortic enlargement) undergo immediate endovascular repair. Similarly, patients with uncontrolled hypertension or refractory pain, are considered an intermediate risk category and are candidates for emergent treatment. The primary goal of endovascular repair is primary entry tear coverage, to lower false lumen pressure, allow true lumen expansion and promote positive aortic remodeling². Nevertheless, reentry tears may continue to perfuse the false lumen leading to its expansion.

CASE REPORT

We present a 60-year old female patient who was admitted from the emergency department due to ATBAD. CT angiogra-

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doi: 10.59037/y66n1973

ISSN 2732-7175 / 2025 Hellenic Society of Vascular and Endovascular Surgery Published by Rotonda Publications All rights reserved. https://www.heljves.com

phy (CTA) indicated ATBAD with the primary entry tear located just distal to the Left Subclavian Artery (LSA) origin, extending distally to the aortic bifurcation and the left common iliac artery (Figure 1). Additionally intramural hematoma, with multiple ulcerations were noted, extending from the level of the LSA to the thoracoabdominal aorta, where a second entry point was also identified, at the level of the celiac artery (CA) origin, on the posterior aortic wall (at 1800 with regard to the CA origin). The CA, superior mesenteric artery and renal arteries were originating from the true lumen (TL), whereas the inferior mesenteric artery from the false lumen, which however was patent. The patient was transferred to the ICU, where the arterial pressure was normalized and the pain was controlled, with intravenous administered antihypertensive and analgesic treatment. During the 2nd hospital day, the patient complained about pain recurrence and underwent repeat-CTA without remarkable changes, including expansion of the dissection, rupture of the aorta or malperfusion to the abdominal viscera or the limbs. Because of uncontrolled and refractory pain she was subjected to emergency TEVAR with LSA coverage during the same day (Gore TAG 34x100mm, W.L.Gore and Associates, Inc.) (Figure 2).

The procedure was uneventful. During the 1st postoperative day the patient was transferred to the ward in good general condition, with per os controlled pain and blood pressure. In the afternoon of the same day she complained for back pain and underwent a new (3rd) CT angiography, which indicated successful endograft deployment and unremarkable changes in the distal thoracic and the abdominal aorta, compared to the preoperative imaging. During the same night the patient became hemodynamically unstable and died. Post-mortem CT examination indicated rupture of the infra-renal aorta and a large retroperitoneal hematoma (Figure 3).

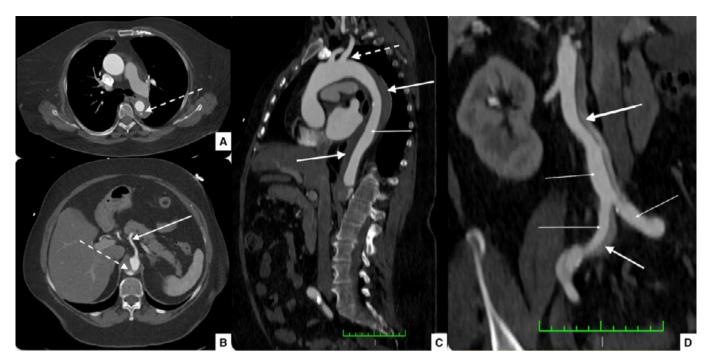


Figure 1: Pre-operative CTA. A: Axial image indicating proximal entry tear (dashed arrow) and intramural hematoma. B: Axial image indicating a second entry tear (dashed arrow) at the level of the celiac artery origin (solid arrow). C: Sagittal image indicating the false lumen and intramural hematoma (thick white arrows), the true lumen (thin white arrow) and the level of the primary first entry tear (dashed arrow), just distal to the origin of LSCA. D: Coronal views of the aorta (thick arrow indicating the false lumen/intramural hematoma, thin arrows indicating the true lumen).



Figure 2: Intraprocedural image showing deployment of the thoracic endograft. Thick white arrow indicates the brachiocephalic artery, thin white arrow indicates the left common carotid artery, dashed arrow depicts the origin of the left subclavian artery having been covered by the endograft, and star depicting the thoracic endograft

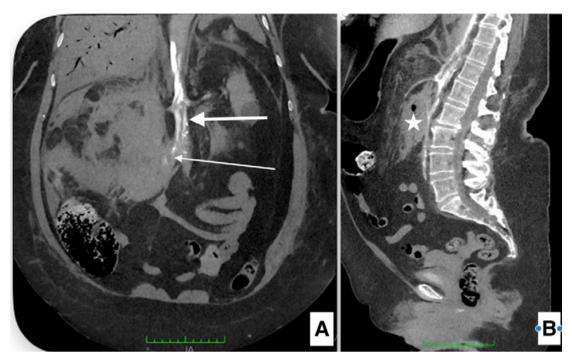


Figure 3: Post-mortem CTA. A: thick white arrow indicating the infrarenal abdominal aorta and thin white arrow depicting the point of aortic rupture. B: white star depicting the large retroperitoneal hematoma.

DISCUSSION

ATBAD results in a 13% in-hospital mortality according to a previous publication from the IRAD registry and most of these deaths occur during the first week after the acute event^{4, 5, 6}. This was the case with the patient reported here as well, who died in the 1st postoperative day, 3-days after the initial symptoms. In our case, the patient had, at first, an uncomplicated ATBAD and thus was treated with intravenous administered β -blockers, vasodilators and morphine. However, due to recurrent and refractory back pain the dissection was characterized as complicated and emergent surgical treatment was decided.

Refractory pain is considered a high risk index after ATBAD, which usually sets the indication for invasive treatment. The term refractory has not been clearly defined in the literature, but it is usually considered when it is not possible to control pain during a >12 hours time period, despite maximal medical therapy⁷. These patients are in an excess risk for mortality, especially when they are managed medically. Specifically, patients with refractory pain and/or uncontrolled arterial hypertension, have been shown to present a 35% in-hospital mortality rate, compared to 1.5% of patients in the low risk group, when treated conservatively. Although overall (regardless of the mode of treatment) mortality rate is again significantly higher among patients with these high risk variables compared to patients without (17.5% vs 4%), the difference is not so wide, thus suggesting that invasive management should be considered for these patients⁵. Moreover, periaortic hematoma has been previously identified as a significant predictor of a poor prognosis, conferring a relative risk around 3 for death after ATBAD, which was present in our patient⁶.

Current therapeutic protocols regarding invasive management of ATBAD indicate that the procedure aims to cover the primary entry tear in order to induce positive aortic remodeling². In our case this was achieved by deploying the endograft just distal to the origin of the left common carotid artery, proximal to the origin of the LSA. This is considered appropriate in cases of acute aortic pathologies, if no specific contraindications exist8. Adequate coverage of the primary entry tear, and successful deployment of the endograft, just distal to the origin of the left common carotid artery was indicated in the post-operative CTA. A 2nd entry tear just opposite the origin of the CA, which was feeding the false lumen, had been noted in all 3 CT scans of the patient, but watchful waiting of this lesion was considered appropriate. If one would decide to cover this as well, not only >35cm of aortic coverage would be required, with a subsequent risk for neurologic complications, but also the origin of the CA would have been covered, while the distal sealing zone till the origin of the SMA would be around 15mm. These factors, along with the fact that the postoperative CTA indicated successful endograft deployment and unremarkable changes in the abdominal aorta contributed to the decision not to undertake a 2nd invasive procedure in this patient.

Among the complications after TEVAR for ATBAD are stroke, spinal cord ischemia, retrograde type A dissection, chronic post-TEVAR aortic dilatation, angulation, migration or collapse of the stent graft, false aneurysm formation, graft erosion, stent-frame fracture and rupture of the aorta². Perioperative aortic rupture after TEVAR can be classified as procedure-related, device-related or due to progression of the disease, as analyzed in Table 1⁹. From the current literature, a low rate of death after endovascular treatment has been reported, that mainly occurs due to aortic rupture (2/3 of cases),

most of which are located in the aortic arch, after retrograde Type A dissections⁹. We are not aware of previous reports on cases presenting infrarenal aortic rupture.

CONCLUSION

Acute type B aortic dissection is a vascular emergency requiring urgent treatment. Complicated cases are preferably treated by endovascular means, but even in the presence of a successful and timely invasive procedure, there is a potential for serious complications which may result in patient's death. To our knowledge only scarce data have been reported regarding abdominal aortic rupture after endovascular treatment of ATBAD.

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