Acute carotid stent thrombosis (ACST): a review study

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Abstract:

Objective: Acute carotid stent thrombosis (ACST) is a rare complication of carotid artery stenting (CAS), occurring within 30 days after CAS and can lead to devastating, even lethal consequences. The purpose of this study is to review the current literature on ACST and investigate the potential causative factors and the available therapeutic strategies.

Methods: A search of multiple electronic health databases was conducted. A total of 37 cases reporting acute ACST were retrieved.

Results: There were 33 male and 4 female patients [mean age 68.54 years (SD ±7.98 y)]. 21.62% (8 cases) referred to asymptomatic carotid stenosis. The left internal carotid artery (ICA) was the predominant site of carotid stenosis (24/37 cases, 64.86%). In 13(35.1%) of cases ACST occurred intra-procedurally, in 10 (27%) within the first 24 hours, in 13 (35.1%) within the first week after CAS. In 1 (2.7%) case there was late thrombosis 3 weeks after CAS. Inadequate antiplatelet therapy, thrombotic predisposition, or antiplatelet resistance, were described as possible causes in 42.3% of cases. The most common technical intraprocedural anatomic causes leading to ACST were ICA dissection, overlapping stents, plaque protrusion along the stent, and thrombosis of embolic protection devices (EPDs). Treatment varied according to the time of carotid stent thrombosis, the cause, and the patient's neurologic status. Antiplatelet therapy in combination with antithrombotic agents (heparin, warfarin, argatroban), intravenous infusion of platelet glycoprotein IIb/IIIa inhibitors (GPIs), thrombolysis, mechanical thrombectomy, thromboaspiration with thrombolysis, open surgery and re-angioplasty were reported as therapeutic strategies. In the majority of cases clinical improvement with resolution of neurological symptoms or residual neurological deficit was observed. Two deaths (5.4%) were reported.

Conclusion: Although, there is no consensus regarding the management of ACST, thrombolysis, mechanical thrombectomy, thromboaspiration with thrombolysis, open surgery and re-angioplasty have all been used with promising results. The critical points for preventing the devastating outcome are immediate recognition of the thrombosis and rapid revascularization of the damaged tissue.

Keywords: Carotid stent; acute thrombosis; stroke; revascularization

INTRODUCTION

Acute carotid stent thrombosis is a rare complication of carotid artery stenting (CAS), the prevalence of which varies between 0.5% to 0.8%.[1] It is a devastating complication that can lead to a stroke and death and occurs within 30 days after CAS.[1-4] This serious complication requires early diagnosis and prompt restoration of cerebral perfusion via internal carotid artery (ICA) recanalization to limit ischemic brain damage.[5] There are several case reports in the literature, but no large series and there are no specific guidelines regarding the ideal strategy for treating ACST. The purpose of this study is to review the current literature on this devastating complication occurring after CAS to investigate potential etiological factors and to explore available therapeutic strategies and outcomes of treated patients.

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doi: 10.59037/nyv9m480
ISSN 2732-7175 / 2023 Hellenic Society of Vascular and Endovascular Surgery Published by Rotonda Publications

METHODS

A multiple search of electronic health databases, including MEDLINE/PUBMED and EMBASE, was performed for all articles published up to June 2022 on carotid stent thrombosis after CAS. Only cases with ACST were included in our study. ACST was defined as those thromboses occurring in the first hours or days after the procedure. Late thromboses (> 30days after CAS) were excluded from the study.

Search strategy

Studies were identified by searching electronic databases and scanning bibliographic references of articles. The National Library of Medicine's Medline database was searched using the PubMed interface and SCOPUS from through June 2022. No language constraints were used. Keywords were selected using medical subject headings (MeSH) for PubMed and MeSH/Emtree for Scopus. The keywords "acute", "carotid stent", and "thrombosis", were used. Databases were searched with an unrestricted search strategy, applying exploded MeSH

and keywords combined with the Boolean operator AND to retrieve relevant reports. A second-level search included a manual screen of the reference lists of the articles identified through the electronic search.

Age and sex, degree and location of stenosis, incidence, perioperative antiplatelet medication, cause of ACST, type of treatment, and outcome were retrieved and analyzed.

RESULTS

The search identified 277 records in total after applying the inclusion criteria. All studies were reviewed and a total of 25 studies, describing the outcome of 37 patients with ACST, met the inclusion criteria.[1-3,6-9,11,13,15-30] The included studies were case reports and small series. In the present study, 33 out of 37 patients were male with a mean age of 68.54 years (SD ±7.98, min 44, max 82 y), and only 21.62% (8 cases) were treated with CAS for asymptomatic carotid stenosis. In the majority of cases (89.19%) mono or dual antiplatelet therapy was used pre-procedurally. The left internal carotid artery (LICA) was the predominant site of carotid stenosis (24/37 cases, 64,86%). In 13 (35.1%) cases ACST occurred intra-procedurally, while in 10 (27%) patients, stent thrombosis occured within the first 24 hours. The remaining 13 (35.1%) cases reported ACST within the first week after CAS. In 1(2.7%) case there was a thrombosis 3 weeks after CAS. Overall, two deaths (5.4%) were reported.

Etiology

The cause of thrombosis was not defined in 11 out of the 37 cases **(Table 1).**

Inadequate antiplatelet therapy, thrombotic predisposition or antiplatelet resistance were described as possible causes in 11(42.3%) of cases (table 1). Three cases of hypercoagulation (3/26, 11.1%) were described due to malignancy [15,22,27] and one more was attributed to atrial fibrillation by Setacci et al.[21]

Technical intraprocedural anatomical causes which led to ACST were identified as follows: ICA dissection in 2 cases[11], plaque protrusion across the stent in one patient[27], overlapping stents in 3 cases (due to malposition in two of them and a long common carotid stenosis in one case)[27], percutaneous transluminal angioplasty (PTA) failure in 3 reports[11,20,25] and embolic protection devices (EPD) complications in 5 patients [1,16,24] (4 cases of EPD thrombosis and 1 case of ICA dissection). A combination of the abovementioned causes was reported in 4 cases **(Table 1)**.

Table 1: Cases with acute carotid stent thrombosis.

	First Author	Year	Age/ sex	Site of Stenosis /Grade	S/A	Onset	Preprocedural antiplatelet/ antithrombotic drugs	Cause of ACST (according to Authors)
1	**Tong**[8]	2000	44/M	LICA/70%	A	Intraprocedurally	ND	ND
2	**Chaturvedi**[2]	2001	63/F	LICA/sever	S	12 hours	ASA	ND
3	**Chaturvedi**[2]	2001	57/M	LICA/ND	S	3 days	NONE	ND
4	**Hamann**[9]	2002	76/M	RICA/70%	S	4 days	ASA and heparin	Antiplatelet therapy ceased
5	**Owens**[18]	2003	65/M	LICA/70%	S	Intraprocedurally	ND	ND
6	**Bush**[7]	2003	68/M	RICA /90%	S	15 minutes	DAPT	ND
7	**Steiner Boker**[6]	2004	64/F	RICA/85%	S	Intraprocedurally	DAPT	Hypercoagulation
8	**Setacci**[21]	2005	82/M	LICA/80%	S	2 days	DAPT O	ND
9	**Setacci**[21]	2005	78/M	LICA/85%	S	4 days	DAPT	DAPT therapy ceased, thrombocythemia
10	**Setacci**[21]	2005	72/M	LICA/80%	S	2 hours	ASA and heparin	AF, multiple cardiac embolism
11	**Masuo**[19]	2006	71/M	LICA/severe	S	3 days	DAPT and heparin	lacking periproced. antithrombotic medication
12	**Buhk**[22]	2006	78/M	LICA/80%	S	3 weeks	DAPT → heparine	Antiplatelet therapy ceased, bladder cancer
13	**Seo**[25]	2008	63/M	LICA/85%	A	Intraprocedurally	DAPT	postdilatation filling stent defect
14	**Iancu**[11]	2010	65/M	LICA/80%	S	Intraprocedurally	DAPT	postdilatation carotid dissection
15	**Iancu**[11]	2010	70/M	RICA/80%	S	Intraprocedurally	DAPT	Balloon burst
16	**Dhall**[17]	2010	62/M	LICA/90%	S	Intraprocedurally	DAPT	ND
17	**Choi**[23]	2012	69/M	RICA/83%	S	9 days	DAPT	ND
18	**Choi**[23]	2012	68/M	LICA/72%	S	4 days	DAPT	DAPT resistance
19	**Kanemaru**[15]	2013	77/M	RICA/ 90%	A	6 days	DAPT and cilostazol	Hypercoagulation/ bladder cancer
20	**Markatis**[13]	2012	67/M	LICA/95%	S	2 days	CLO	DAPT Discontinuation
21	**Kim**[24]	2013	75/M	LICA 90%	S	Intraprocedurally	DAPT	EPD thrombosis
22	**Kim**[24]	2013	73/M	ND	A	Intraprocedurally	DAPT	EPD thrombosis
23	**Kim**[24]	2013	51/M	ND	S	Intraprocedurally	DAPT	EPD thrombosis

	First Author	Year	Age/ sex	Site of Stenosis /Grade	S/A	Onset	Preprocedural antiplatelet/ antithrombotic drugs	Cause of ACST (according to Authors)
24	**Munich**[16]	2014	70/M	LICA/90%	S	Intraprocedurally	DAPT	EPD thrombosis
25	**Köklü**[26]	2015	73/M	LICA/95%	S	24 hours	ASA	DAPT resistance
26	**Moulakakis**[27]	2017	67/M	RICA/90%	S	1 hour	DAPT	Dissection from EPD, 360˚ RICA kinking
27	**Moulakakis**[27]	2017	74/M	LICA/ 80%	S	2 hours	Nadroparin calcium	overlapping stents, malignancy
28	**Moulakakis**[27]	2017	73/F	LCCA/80%	S	3 days	DAPT	overlapping stents, malignancy
29	**Moulakakis**[27]	2017	66/M	LICA/80%	S	4 days	DAPT	overlapping stents
30	**Cvjetko**[29]	2017	68/F	RICA/70%	ND	90 minutes	DAPT	ND
31	**Cvjetko**[29]	2017	80/M	LICA/70%	A	4 days	DAPT	ND
32	**Moulakakis**[1]	2018	66/M	RICA/90%	S	Intraprocedurally	ASA	Plaque protrusion across the stent
33	**Moulakakis**[1]	2018	72/M	RICA/severe	A	1 hour	ND	ND
34	**Hu**[20]	2018	79/M	LICA occlusion	A	Intraprocedurally	DAPT	Stent not fully adhered to vessel
35	**Kahyaoglu**[3]	2018	66/M	LICA/70%	S	3 hours	DAPT	DAPT Discontinuation
36	**Toljan**[30]	2019	55/M	LICA occlusion	A	2 hours	DAPT	CYP2C19 & MDR1
37	**Wei wei**[28]	2021	69/M	LICA/90%	S	5 days	DAPT	CYP2C19 heterozygote

F- female, M- male, ND- not determined, ASA- aspirin, CLO- clopidogrel, , DAPT: Dual antiplatelet therapy(ASA and clopidogrel or ticlopidine), A-asymptomatic, S- symptomatic, LICA- left internal carotid artery, RICA- right internal carotid artery, CEA- carotid endarterectomy, EPD- embolic protection device

Therapeutic Management

Antiplatelet therapy in combination with antithrombotic agents

Eight patients were treated with antiplatelet therapy (single or dual) in combination with antithrombotic agents (low molecular weight heparin, warfarin, argatroban) or intravenous (IV) infusion of GPIs.[8,15,25,26,27] Data on the use of GPIs are relatively limited, and fatal intracranial hemorrhage has been reported as a complication[5]. Abciximab was used alone or in combination with other thrombolytic agents and showed efficacy in dissolving an in-stent thrombus (IV bolus dosage of 0.25mg/kg and a 0.125mg/kg intra-arterially, followed by IV infusion of 0.125mg/kg/min for further 12 hours).[6,7,8]

In the majority of cases, resolution of neurological symptoms or residual paresis was observed. In one case treated with aspirin alone, death occurred **(Table 2)**.[2]

Thrombolysis ± Re-Angioplasty

In 12 patients thrombolysis was performed which was unsuccessful in 4 of them and further treatment was needed. Thrombolysis in patients with acute ischemic stroke attributed to ICA occlusion can be infused either intra-arterially or IV. Several thrombolytic agents such as streptokinase, urokinase, tenecteplase, or rtPA have been used for the treatment of acute ACST intra-arterially with complete, partial, or even unsuccessful clot resolution.[2,7,10,11,12,13] A clinical improvement and a patent stent in the majority of cases were described **(Table 2)**. Studies have recorded the beneficial effect of IV thrombolysis[9,10], however, an increase in death and intracranial bleeding were also documented.[6] In one case treated with urokinase, a death occurred **(Table 2)**.[2]

3 cases were treated with re-angioplasty or stenting. Owens et al reported thrombolysis and repeat angioplasty, leading to ICA thrombosis and rupture.[18] Masuo et al described an intraprocedural ACST treated with redo in stent PTA using an EPD, followed by intravenous heparin infusion for 7 days with a residual mild hemiparesis.[19] Finally, Hu et al reported intraprocedural in stent thrombosis treated with microcatheter infusion of rt-PA and re-angioplasty.[20]

Mechanical thrombectomy or thromboaspiration with or without thrombolysis

8 patients underwent mechanical thrombectomy with or without thrombolysis. The Penumbra system showed promising results with complete recanalization and neurological improvement in 3 patients **(Table 2)**.[24] Thromboaspiration combined with thrombolysis or anticoagulants (abciximab) has shown clinical improvement and stent recanalization.[16,17,28,30] It offers an alternative, however EPD occlusion has been reported.[15,16] Dhall et al have reported complete patency restoration with the use of a thromboaspiration catheter before EPD withdrawal.[17] Mechanical thrombectomy and stenting were associated with higher recanalization rates and improved functional status when compared to intra-arterial thrombolysis.[12]

Open Surgical Exploration

12 patients underwent surgical exploration **(Table 2)**, due to described major neurological deterioration.[1,13,18,21,23,29] Strategies such as carotid thromboendarterectomy and stent explantation, open surgical thrombectomy without stent removal, primary or patch closure, use of shunt or not, and even bypass from superficial temporal artery to middle cerebral artery (STA-MCA) were reported.

Table 2: Cases with acute carotid stent thrombosis. F- female, M- male, ND- not determined, ASA- aspirin, CLO- clopidogrel, ,

	First Author	Stroke severity and symptoms	Treatment	Outcome
1	**Tong**[8]	None	Abciximab IV bolus	Resolution of neurological symptoms /stent patent
2	**Chaturvedi**[2]	None	750000IU urokinase	**Death**
3	**Chaturvedi**[2]	Right hemiplegia	ASA	**Death**
4	**Hamann**[9]	Hemiparesis, left hemianopsia	70mg rt-PA & heparin	Resolution of neurological symptoms/ stent patent
5	**Owens**[18]	Right upper extremity weakness	Thrombolysis, repeat angioplasty/ICA thrombosis & rupture- stent removal	Motor deficits
6	**Bush**[7]	Unconscious, hypotension, seizures	Mechanical thrombectomy & iv abciximab	Resolution of neurological symptoms /stent patent
7	**Steiner-Boker**[6]	Mild left facial paresis & dysarthria	Intracarotid 5mg rt-PA - intraarterial & iv for 12 hours abciximab	Neurological improvement/ stent patent
8	**Setacci**[21]	Crescendo TIAs	Endarterectomy and Stent removal	Neurological improvement
9	**Setacci**[21]	Decreased level of consciouness	Endarterectomy and Stent removal	Gradual improvement from the stroke
10	**Setacci**[21]	Left hemiplegia	Surgical thrombectomy without stent removal	Good recovery/ stent patent
11	**Masuo**[19]	Right hemiparesis, motor aphasia	In-stent PTA & EPD& iv heparin for 7 days	Mild right hemiparesis
12	**Buhk**[22]	Aphasia, right hemiparesis	Antiplatelet therapy & heparin	Minor hemiparesis & slight aphasia/ thrombus resolution, stent patent
13	**Seo**[25]	None	IV tirofiban	No neurological deficit/ stent patent
14	**Iancu**[11]	Hemodynamic instability, contralateral hemiplegia, unconsciousness	Bolus streptokinase	No neurological deficit/ stent patent
15	**Iancu**[11]	Unconsciousness	Intrathrombus tenecteplase & balloon angioplasty	No neurological deficit/ normal intrastent velocities
16	**Dhall**[17]	Seizures, hemodynamic instability	Urokinase, abciximab, thromboaspiration	Clinical improvement/ stent recanalization
17	**Choi**[23]	Hemiparesis, dysarthria, left facial palsy	STA-MCA anastomosis	Left hemiparesis & dysarthria
18	**Choi**[23]	Hemiparesis, dysarthria, right facial palsy	Iv rt-PA, intra-arterial thrombolysis, STA-MCA anastomosis	Right hemiparesis
19	**Kanemaru**[15]	None	ASA & CLO& cilostazol & warfarin & argatroban	Thrombus resolution
20	**Markatis**[13]	Right paresis, numbness	Heparin, endarterectomy, stent removal, thrombectomy, primary closure	Minor numbness of three digits on the right hand
21	**Kim**[24]	None	Mechanical thrombectomy (Penumbra system)	Complete recanalization
22	**Kim**[24]	Global aphasia	Mechanical thrombectomy (Penumbra system)	Complete recanalization/ neurological improvement
23	**Kim**[24]	Hemiparesis, impaired mentality	Mechanical thrombectomy (Penumbra system)	Complete recanalization/ neurological improvement
24	**Munich**[16]	ND	Unsuccessful intraarterial verapamil & abciximab, thrombus aspiration	Good recovery/residual 25-30% stenosis
25	**Köklü**[26]	Right hemiplegia, dysarthria	Unfractionated heparin & ticlodipine	Residual paresis
26	**Moulakakis**[27]	Left hemiparesis	Thrombus aspiration, surgical exploration, eversion CEA with stent removal	Clinical improvement, residual arm paresis
27	**Moulakakis**[27]	Aphasia, right hemiparesis	Intrathrombus urokinase & stenting	Clinical improvement, residual arm paresis
28	**Moulakakis**[27]	Right arm paresis	Tinzaparin	Residual arm paresis/30 months post-op no paresis
29	**Moulakakis**[27]	Aphasia, right hemiparesis	ASA, CLO nadroparin	Mild speech impairment
30	**Cvjetko**[29]	Contralateral hemiplegia, aphasia	Endarterectomy, stent removal, primary closure	Rankin 1

	First Author	Stroke severity and symptoms	Treatment	Outcome
31	**Cvjetko**[29]	TIAs	Endarterectomy, stent removal, Fogarty thrombectomy	Rankin 3
32	**Moulakakis**[1]	Left hemiparesis	Endarterectomy & stent removal	Good recovery
33	**Moulakakis**[1]	Hemodynamic instability	Unsuccessful intraarterial thrombolysis & actilyse, endarterectomy & stent removal	No neurological deficit
34	**Hu**[20]	None	rt-PA through microcatheter, redilatation of the stent	No neurological deficits/ stent patent
35	**Kahyaoglu**[3]	Motor aphasia, right hemiplegia	CLO, unfractionated heparin iv, intaarterial rt-PA	Left arm weakness/ stent recanalization
36	**Toljan**[30]	Global aphasia, right hemiparesis	Thrombus aspiration, alteplase, iv eptifibatide	Good recovery/ stent patent
37	**Wei wei**[28]	Right limb weakness, right facial paralysis, hemianalgesia	Thrombus aspiration & tirofiban	Rankin 1

DAPT: Dual antiplatelet therapy(ASA and clopidogrel), A-asymptomatic, S- symptomatic, LICA- left internal carotid artery, RICA- right internal carotid artery, CEA- carotid endarterectomy, EPD- embolic protection device

DISCUSSION

Acute carotid stent thrombosis remains a rare complication of CAS, occurring during or after less than 1% of procedures.[1] According to the available literature, 37 cases reporting acute carotid stent thrombosis in the early period (<30 days) have been recognized. Many factors have been imlicated as responsible for ACST. Our study showed that the predominant factors for carotid stent thrombosis are inadequate antiplatelet therapy, thrombotic predisposition, and the antiplatelet resistance.[3] Furthermore, technical intraprocedural anatomical causes predisposing to carotid stent thrombosis are local vessel dissection or intimal injury leading to platelet adhesion and thus thrombus formation[11,25], plaque protrusion promoting thrombosis, kinking and even coiling of the distal part of ICA[27], vasospasm, stent underexpansion which impairs stent's thombogenicity itself[28], overlapping carotid stents[27] and EPD occlusion.[4,16,24] It is supported that EPD occlusion and carotid stent thrombosis are sequential events as they have been attributed to similar mechanisms.[4,16,24]

Our study showed heterogeneity in the type of treatment used, the severity of the patient's neurological condition, and the extent of brain damage. In the majority of cases, clinical improvement was observed with a resolution of neurological symptoms or residual neurological deficit. Two deaths (5.4%) were reported. However, this rate might be underestimated by the fact that ACST cases with a fatal outcome may have been avoided for publication. Thrombolysis, mechanical thrombectomy, thromboaspiration with thrombolysis, open surgical therapy, and re-angioplasty have all been used, depending on the time of thrombosis, cause, and neurological status of the patient, with promising results. The main goal of ACST treatment is to promptly remove the source of thrombus to limit ischemia time, reperfusion injury, and potential post-procedure related intracranial hemorrhage.

Emergent removal of the thrombosed stent is indicated in ACST patients with immediate neurological deterioration, especially when the in-stent thrombosis is not associated with intracranial carotid branch thrombosis. The emergency operation has the risk of clot dislocation due to surgical maneuvers and therefore demands extended site exposure and careful preparation to protect distal cerebral embolization.[1]

Several studies have documented the beneficial effect of intra-arterial thrombolysis.[2,6,7,9-13] It can be used rapidly especially when ACST occurs intraprocedurally. Although local intra-arterial thrombolysis might have a high recanalization rate, however, intraluminal manipulation of wires and catheters combined with thrombus lysis can cause clot fragmentation and therefore distal cerebral occlusion. The residual thrombus within the stent predisposes to restenosis or even re-thrombosis of the stent. Intra-arterial thrombolysis with redo PTA or stenting may be another alternative for treating ACST.[18-20] It should be emphasized that redo PTA or stenting itself carries a risk of damaging the stent's stability. In addition, one study showed that overlapping carotid stents predispose to thrombosis.[1]

A combination of methods such as mechanical thrombectomy with thrombus aspiration may be used according to ASA/AHA guidelines in patients with large segment occlusion who have not responded to intravenous thrombolysis and should be performed only by certified experts.[31] According to literature data, mechanical thrombectomy and stenting may be associated with higher recanalization rates and improved functional status when compared to intra-arterial thrombolysis[12].

In the most comprehensive review up to date in which 26 cases of early carotid stent thrombosis were presented an algorithm for the management has been proposed.[1] This algorithm distinguishes whether the complication occurred intra- or post-procedurally. In the latter case, an imaging study initially such as carotid duplex examination, followed by digital substraction angiography or computed tomographic angiography in suspicion of distal extension of carotid thrombosis is recommended to evaluate the level of thrombosis. Once confirmed, then percutaneous mechanical thrombectomy, thromboaspiration and thrombolysis or thrombolysis with or without IIb/IIIa receptor inhibitors are suggested. Then, if partial or residual intrastent thrombosis with no distal cerebral extension is documented surgical exploration can be used. If carotid stent thrombosis occurs intraprocedurally immedi-

ate treatment with percutaneous mechanical thrombectomy, thromboaspiration and thrombolysis or thrombolysis with or without IIb/IIIa receptor inhibitors should be initiated. If the thrombus is refractory, surgical exploration should be considered. Tirofiban has been suggested mainly due to its short platelet-bound half-life and long plasma half-life predisposing to lower hemorrhagic risk[1].

In conclusion, acute carotid stent thrombosis is a rare complication of CAS with potentially devastating even lethal outcomes. Although, there is no consensus on its management, thrombolysis, mechanical thrombectomy, thromboaspiration with thrombolysis, open surgical treatment, and re-angioplasty have all been used, depending on the stent thrombosis time and cause with promising results. The critical points in preventing a catastrophic outcome are immediate recognition of thrombosis and rapid revascularization of the damaged tissue.

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