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Acute thromboses and occlusions of dual layer carotid stents in endovascular treatment of tandem occlusions

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ABSTRACT

Purpose To evaluate the occurrence and risk factors of acute in-stent thrombosis or stent occlusion in patients with tandem occlusions receiving intracranial mechanical thrombectomy and emergent extracranial internal carotid artery stenting with a dual layer carotid stent.

Methods Multicenter retrospective data collection and analysis of stroke databases of seven comprehensive stroke centers from three European countries.

Results Overall, 160 patients (mean (SD) age 66 (12) years; 104 men (65%); median (IQR) baseline NIHSS 14 (9–18); IV lysis, n=97 (60.6%)) were treated for a cervical carotid artery occlusion or stenosis using a CASPER stent (MicroVention), and received mechanical thrombectomy for an intracranial occlusion between April 2014 and November 2018. During the procedure or within 72 hours, formation of thrombus and complete occlusion of the CASPER stent was observed in 33/160 (20.8%) and in 12/160 patients (7.5%), respectively. In 25/33 (75.8%) and in 9/12 patients (75%), respectively, this occurred during the procedure. No statistically significant difference was observed between patients with and without thrombus formation with regard to pre-existing long term medication with anticoagulants or intraprocedural administration of heparin, acetylsalicylic acid (ASA), or heparin and ASA. Favorable early neurological outcome was similar in patients with (n=15; 45.5%) and without (n=63; 49.6%) thrombus formation at the CASPER stent.

Conclusion Acute thrombosis or occlusion of CASPER stents in thrombectomy patients receiving emergent extracranial internal carotid artery stenting for tandem occlusions were observed more often during the procedure than within 72 hours of follow-up, were less frequent then previously reported, and showed no impact on early neurological outcome.

INTRODUCTION

Endovascular thrombectomy after large vessel ischemic stroke has proven to be an effective treatment.[1] According to a meta-analysis of individual patient data from five randomized trials, 10.7% of patients with intracranial large vessel occlusion need to be treated for a tandem occlusion.[1] Tandem occlusions are defined by an occlusion of the middle cerebral artery or the terminus of the internal carotid artery with an additional preceding high grade stenosis or occlusion of the cervical internal carotid artery. In such cases of mechanical thrombectomy (MT) of the intracranial occlusion, angioplasty or stenting of the preceding ipsilateral carotid stenosis or occlusion is required.

Currently there are different stent designs available for carotid artery stenting (CAS): single layer stents (SLS) and the recently introduced dual layer stents (DLS). SLS are well established and have been in use for several years.[2] The stent design of SLS has an impact on procedural stroke risk and death, favoring a closed cell design over open cell design for symptomatic carotid stenosis undergoing CAS.[3] DLS use a second micromesh layer and have been designed for better plaque coverage and reduction of debris dislodgement.[4] CAS using a DLS appears to be feasible and safe, and moreover in combination with a distal embolic device may result in a lower rate of periprocedural diffusion weighted imaging lesion burden compared with previous historic studies.[5 6] For these reasons it appears to be reasonable to use DLS in acute stroke patients with tandem occlusions.

Recently, a retrospective single center study showed a rate of 45% for thrombotic occlusion of DLS (CASPER stent, Carotid Artery Stent designed to Prevent Embolic Release; MicroVention, Aliso Viejo, California, USA) within 72 hours of emergent CAS during treatment of a tandem occlusion.[7] This very high rate of acute occlusions appears alarming, especially as all DLS were patent in the final angiogram at the end of the thrombectomy procedure.[7] Another retrospective single center study observed acute in-stent thrombus formation in 52.4% of patients.[8] However, as the sample sizes in the two studies are small and these studies appear to show a higher-than-anticipated occlusion rate, we hypothesize that the rate of occlusion is lower and rather comparable to previously reported occlusion rates in single layer stents and propose a retrospective multi-center evaluation to increase the power to assess this.

METHODS

For this analysis, stroke databases from seven comprehensive stroke centers in three European countries were retrospectively searched for patients with acute ischemic stroke due to a tandem occlusion who received endovascular treatment between April 2014 and November 2018. Patients were identified by the respective inhouse registry of neuroendovascular interventions.

Inclusion criteria

Patients meeting the following criteria were selected for this analysis.

Patients who received at least one intracranial thrombectomy maneuver and underwent emergent CAS using a CASPER stent.

Patients who underwent inhouse neurological examination as per the National Institutes of Health Stroke Scale (NIHSS) score before the procedure, and assessment of the degree of disability or dependence according to the modified Rankin Scale (mRS) before discharge.

Patients who had follow-up imaging of the brain and cervical vessels using CT, CT angiography, MRI, MR angiography, or Doppler ultrasonography to assess stent patency as well as occurrence and degree of intracranial hemorrhage and infarction within 72 hours of treatment or—if earlier—until discharge.

Endovascular procedures

Endovascular procedures were performed at the discretion of the treating neurointerventionalist. There were no limitations concerning the use of stent retrievers or direct thromboaspiration for MT of the intracranial occlusion, or periprocedural administration of antiplatelet therapy or anticoagulants. Furthermore, there were no prespecified conditions with regards to the chronology of MT, CAS, and, if applicable, conduct of percutaneous transluminal angioplasty. Intravenous thrombolytic drugs were administered and dosed at the discretion of the treating neurologist following national and international guidelines.

Study device

The CASPER stent is a dual layer, closed cell structured carotid artery stent with flexible weave and, on each end, flared ends. The inner layer has a micromesh design with an average pore size of 390–700 µm, originally designed to hold back plaque. The CASPER stent is fully resheathable and repositionable up to 50% of its deployment length.

Study endpoints

The primary endpoints of this study were the rate of occurrence of acute thrombus formation at the CASPER stent as well as occlusion of the CASPER stent following emergent extracranial internal carotid artery stenting in patients receiving endovascular stroke treatment for a tandem occlusion in the anterior circulation. Occlusion was defined as complete thrombotic occlusion of the CASPER stent, and acute thrombus formation was defined as any formation of thrombus at the CASPER stent either during the intervention or within 72 h hours (or, if earlier, until hospital discharge) detected on DSA images or on follow-up examinations.

Secondary endpoints were the rate of early neurological outcome (ie, improvement in stroke symptoms at hospital discharge: modified Rankin Scale (mRS) score of 0–2 or NIHSS score ≥10 (Solitaire With the Intention For Thrombectomy (SWIFT) trial criteria))[9] and symptomatic intracranial hemorrhage according to the criteria of the European Cooperative Acute Stroke Study (ECASS II/III).[10 11] Furthermore, the assumed etiology of cervical artery stenosis or occlusion (arteriosclerotic vs dissection), the interventional approach (antegrade=CAS first followed by MT vs retrograde=MT first followed by CAS), and concurrent antiaggregation with heparin, acetylsalicylic acid (ASA), and glycoprotein IIb/IIIa inhibitors (Tirofiban) was investigated.

Ethical approval, data acquisition, and statistical analysis

This study was conducted according to the principles of the Declaration of Helsinki. The stroke patient databases are independently approved by the respective local ethics committee of and individually maintained by the participating centers. Due to the emphasis on patient safety and quality control as well as the retrospective character of the data collection and analysis, written informed consent was waived.

Data collection was performed using a standardized electronic data entry form. A centralized plausibility assessment and, if necessary, specific inquiries were conducted to ensure data validity before statistical analysis. Data are shown as mean (SD) or median (IQR), as appropriate. Differences in means and medians were tested using the Student's t test. The χ^2 test was performed to determine differences in frequencies. All statistical analyses were performed using IBM SPSS Statistics 21.0.0.0 (Armonk, New York, USA).

RESULTS

A total of 160 patients were included in this analysis. In the patient population, mean age was 66 years (SD 12), baseline median NIHSS score was 14 (IQR 9–18), and 97/160 (60.6%) patients received intravenous thrombolysis with recombinant tissue plasminogen activator (tPA). Overall, there was a predominant number of male patients (n=104/160, 65%). Baseline, demographic, and imaging characteristics of the overall patient population and those patients with (Pt_+) and without (Pt_-) thrombus formation are shown in table 1.

Primary endpoint

The rate of occurrence of acute thrombus formation at the CASPER stent was 33/160 (20.6%). In 12/33 patients with acute thrombus formation (36.4%), an occlusion of the carotid artery stent was observed; this corresponded to 7.5% of all patients included in this analysis. In 25/33 (75.8%) patients with thrombus formation and in 9/12 (75.8%) patients with stent occlusion this occurred during the endovascular procedure (please see table 1). All other stent-thromboses or -occlusions were detected via Doppler ultrasonography, which was done in all patients before discharge.

Secondary endpoints

A statistically significant difference with regard to pre-existing long term medication with anticoagulants or intraprocedural medication with ASA, heparin, or a combination of heparin and ASA was not observed (see Table 2). Patients who had formation of thrombus at the CASPER stent were more likely to receive glycoprotein IIb/IIIa inhibitors during CASPER placement (Pt_-: n=31/127 (24.4%); Pt_+: n=19/33 (57.6%); P<0.001, see table 3). However, administration of glycoprotein IIb/IIIa inhibitors was predominantly a rescue treatment, as the rate of planned administration of glycoprotein IIb/IIIa inhibitors was comparable

Table 1 Baseline, demographic, and imaging characteristics of the overall patient population, and those patients with and without thrombus formation

	All patients (n=160)	Patients without thrombus formation (n=127; 79.4%)	Patients with thrombus formation (n=33; 20.6%)	P value
Age (years) (mean (SD))	**66 (12)**	**67 (11)**	**62 (11)**	**0.024***
Men (%)	104 (65)	82 (64.6)	22 (66.7)	0.822†
Initial NIHSS score (median (IQR))	14 (9–18)	14 (9–17)	15 (11–19)	0.128*
Intravenous tPA (%)	97 (60.6)	72 (56.7)	25 (75.8)	0.046†
Unknown time of symptom onset (%)	59 (36.9)	49 (38.6)	10 (30.3)	0.363†
ASPECTS (median (IQR))	8 (7–9)	8 (7–9)	7 (7–9)	0.290*
Procedural aspects				
Treatment with general anesthesia (%)	141 (88.1)	111 (87.4)	30 (90.9)	0.579†
Cervical internal carotid artery occlusion (%)	92 (57.5)	72 (56.7)	20 (60.6)	0.685†
Placement of CASPER before mechanical thrombectomy (%)	117 (73.1)	95 (74.8)	22 (66.7)	0.090†
Presumed cause of cervical internal carotid artery stenosis or occlusion				
Arteriosclerosis (%)	137 (85.6)	113 (89)	24 (72.7)	0.018†
Dissection (%)	23 (14.4)	14 (11)	9 (27.3)	
Location of intracranial occlusion				
Occlusion site right, (%)	68 (42.5)	55 (43.3)	13 (39.4)	0.685†
Carotid T (%)	31 (19.4)	33 (26)	10 (48.5)	0.031†
M1 (%)	81 (50.6)	67 (52.8)	14 (42.4)	
M2 (%)	30 (18.7)	27 (21.3)	3 (9.1)	
Time from stroke onset to groin puncture (min) (median (IQR))‡	203 (140–276)	207 (155–311)	152 (125–252)	0.114*
Final mTICI score				
0-2a, (%)	11 (6.9)	7 (5.5)	4 (12.1)	0.192§
2b, (%)	77 (48.1)	60 (47.2)	17 (51.5)	
3, (%)	72 (45)	60 (47.2)	12 (36.4)	
Early outcome parameters				
Favorable early neurological outcome (%)	78 (48.8)	63 (49.6)	15 (45.5)	0.671†
Inhouse mortality (%)	11 (6.9)	10 (7.9)	1 (3)	0.327†
Incidence and anatomic distribution of intracranial hemorrhages on follow-up NCCT¶				
1a, (%)	20 (12.5)	14 (11)	6 (18.1)	0.117§
1b, (%)	19 (11.9)	16 (12.6)	3 (9.1)	
1c, (%)	8 (5)	8 (6.3)	0	
2, (%)	10 (6.25)	10 (7.9)	0	
3a, (%)	1 (0.6)	1 (0.8)	0	
3b, (%)	1 (0.6)	1 (0.8)	0	
3c, (%)	8 (5)	6 (4.7)	2 (6.1)	
3d, (%)	2 (1.25)	2 (1.6)	0	
Symptomatic intracranial hemorrhage (%)	15 (9.4)	15 (11.8)	0	0.036†

*t test, two sided.
†χ^2 test, two sided.
‡For patients with known symptom onset.
§Mann–Whitney U test, two sided.
¶Multiple assignments possible, if more than one bleeding location within the same patient was detected; according to the Heidelberg Bleeding Classification.
ASPECTS, Alberta Stroke Program Early CT Score; mTICI, modified Treatment in Cerebral Infarction; NCCT, non-contrast CT; NIHSS, National Institutes of Health Stroke Scale; tPA, tissue plasminogen activator.

between patients without thrombus formation (n=31/127, 24.4%) and patients with thrombus formation during the further course of the endovascular procedure (n=5/33 (15.2%); P=0.872).

Postprocedural long-term medication is listed in Table 3. An antiplatelet monotherapy with ASA was given to 13 (8.1 %) patients following the procedure. In 6/13 patients a stent-thrombosis was observed; 5 of which were detected during the interventional procedure.

DISCUSSION

According to the HERMES meta-analysis, approximately every 10th patient with intracranial large vessel occlusion needs to be treated for an additional cervical artery stenosis or occlusion.[1]

Table 2 Pre- and intraprocedural medication of the overall patient population, and in patients with and without thrombus formation

	All patients (n=160)	Patients without stent thrombosis (n=127; 79.4%)	Patients with stent thrombosis (n=33; 20.6%)	P value
Long term anticoagulation medication administered prior to hospital admission (n (%))	41 (25.6)	34 (26.8)	7 (21.2)	0.500
Heparin administered during CASPER placement (n (%))	62 (38.8)	49 (38.6)	13 (39.4)	0.958
ASA administered during CASPER placement (n (%))	92 (57.5)	69 (54.3)	23 (69.7)	0.122
Heparin+ASA administered during CASPER placement (n (%))	36 (22.5)	28 (22)	8 (24.2)	0.805
GP2b/3a inhibitor administered during CASPER placement (including rescue treatment) (n (%))	50 (31.3)	31 (24.4)	19 (57.6)	<0.001
GP2b/3a inhibitor administered during CASPER placement (planned/without rescue) (n (%))	36 (24.7)	31 (24.4)	5 (15.2)	0.872
GP2b/3a inhibitor administered as rescue treatment after CASPER placement (n (%))	14 (8.8)	0	14 (42.4)	<0.001
CASPER placement without planned administration of heparin, ASA, or GP2b/3a inhibitor (n (%))	31 (19.4)	28 (22)	3 (9.1)	0.093

All P values were calculated using the χ^2 test, two sided.
ASA, acetylsalicylic acid; GP2b/3a, glycoprotein IIb/IIIa inhibitors.

Awareness of thrombus formation and secondary thrombotic occlusion in these stroke patients receiving emergent CAS is of major concern. In this multicenter retrospective analysis, we report a rate of acute thrombotic occlusion of 7.5% for a dual layer carotid artery stent system in patients with acute ischemic stroke receiving emergent CAS using the CASPER stent and additional MT for treatment of a tandem occlusion. In addition, 13.1% of thrombus formations were observed at the carotid stent side but did not result in stent occlusion.

The rate of acute thrombus formation of single layer carotid artery stents during endovascular stroke treatment of tandem occlusions has been reported previously, ranging from 1.3% to 17%.[7 12–15] Furthermore, the rate of long term (ie, 30 days or later) in-stent restenosis and occlusion after SLS CAS seems to be between 7.4% and 9% under comparable clinical conditions.[12 13] Early internal carotid artery occlusion (ie, within 7 days) after emergent CAS following treatment of a tandem occlusion was reported in 10.3% of patients.[16] Unfortunately, the later internal carotid artery occlusions could not be attributed to a specific stent design as both (SLS and DLS) were reported as one group.[16]

Our results are in contrast with a previous single center retrospective study showing a significantly higher rate of CASPER stent occlusions within 72 hours after treatment of tandem occlusion of about 45%.[7] An especially noteworthy aspect is that in this previously published study, all stents were reported to be patent on the final angiograms. Yilmaz *et al* hypothesized that the high rate of DLS occlusion could potentially be attributable to the absence of a standard antiplatelet regimen or the fact that intravenous recombinant tPA was administered significantly more often in patients treated with SLS. Our findings contradict these explanations as in the current analysis, due to the multicenter approach, there were widespread differences with regard to the intraprocedural medication regimens. Additionally, patients who showed thrombus formation at the CASPER stent received intravenous recombinant tPA more often. The latter finding in our cohort might be confounded by the fact that these patients were significantly younger and potentially had less contraindications for administration of intravenous recombinant tPA. Unfortunately, this explanation cannot be proven as past medical history (in contrast with previous medication) was not recorded systematically for this analysis.

In patients receiving only ASA post CASPER treatment stent-thrombosis was observed more often. However, since 5 of these stent-thrombosis were observed during the interventional procedure, the ASA monotherapy might not be the cause, but rather a consequence to reduce the risk of hemorrhage in patients with a poor prognosis.

Under consideration of the previously published results concerning thrombus formation and occlusion of SLS and a very low rate of thromboembolic events and stent occlusions in patients undergoing scheduled CAS using a CASPER stent,[6 17] we believe that the higher rate of CASPER stent occlusion reported by Yilmaz *et al* and Bartolini *et al* might potentially be attributed to other factors which have not yet been investigated (eg, blood velocity profile, vascular anatomy (ie, tortuosity of the common and internal carotid artery), blood coagulation parameters, and residual stenosis after stenting).

There are limited data concerning thrombus formation and clinical outcomes in patients with carotid artery dissection and intracranial large vessel occlusion receiving emergent CAS. As patients with carotid artery dissections are usually of younger age and prone to develop thromboembolic ischemic events,[18] this might be a possible explanation for the high percentage of these patients in our subgroup of patients with thrombus formation.

In this retrospective analysis, patients with carotid T occlusions were more likely to develop acute in-stent thrombosis. A possible explanation for this finding is that carotid T occlusions

Table 3 Postprocedural long-term medication of the overall patient population, and in patients with (Pt_+) and without thrombus formation (Pt_-)

	All patients(n=160)	Pt_+(n=127; 79.4%)	Pt_-(n=33;20.6%)	P value
ASA (n (%))	13 (8.1)	7 (5.5)	6 (18.2)	0.029
ASA + Clopidigrel(n (%))	93 (58.1)	82 (64.6)	11 (33.3)	0.001
ASA+ Ticagrelor(n (%)	40 (25)	29 (22.8)	11 (33.3)	0.115
No long-term anticoagluation or antiaggregation medication (n (%))	14 (8.8)	9 (7.1)	5 (15.2)	0.134

All p-values were calculated using $\chi 2$ test, 2-sided

lead to a markedly reduced or even no blood outflow into the anterior and middle cerebral artery and thereby creates stagnation of blood in the internal carotid artery. This stagnation could trigger activation of the coagulation cascade and therefore make these patients more prone to developing acute in-stent thrombosis.[19] This finding needs to be investigated further, as this endorses a retrograde approach (ie, intracranial MT before cervical carotid artery CAS) to ensure internal carotid artery outflow in patients with carotid T occlusions or M1 occlusions with hypoplastic ipsilateral posterior communicating or anterior cerebral arteries.

A favorable early clinical outcome was seen in nearly half of the patients, and symptomatic intracranial hemorrhage in <10% of cases. Both findings are consistent with data from a current meta-analysis.[20] An interesting finding in our analysis is that despite the fact that intravenous recombinant tPA and glycoprotein IIb/IIIa inhibitors were administered more often in the patient group with thrombus formation, none of the patients were reported to have suffered a symptomatic intracranial hemorrhage. A potential explanation might be reperfusion hemorrhage in patients without thrombus formation at the stent[21 22] or discontinuation of anticoagulation in patients with stent occlusion.

Limitations

Our study has several limitations because of its retrospective, single arm design. Although this is the largest sample size of acute thrombus formation in patients undergoing CAS and MT for tandem occlusion, the multicenter design might have introduced some bias with regard to site specific medication and interventional treatment regimens or national guidelines.

As this research was conducted on departmental funding only, there was no imaging core laboratory or on site monitoring to validate data provided by the participating centers. Hence the occurrence of discrete thrombus formation might be underreported; however, the rate of clearly visible thrombus formation and thrombotic occlusion should be unaffected. Additionally, the possibility of plaque protruding through the stent struts into the patent lumen mimicking thrombus formation could not be ruled out; however, as DLS were specifically designed to withhold plaque with their micromesh layer, this possibility appears to be less relevant.

Another limitation is the missing long term follow-up data concerning stent patency and clinical outcome beyond discharge. Furthermore, there are no data concerning treatment of tandem occlusions with a different type of dual layer carotid artery stent to compare with, and our data do not support a class effect.

CONCLUSION

Acute thrombosis or occlusion of CASPER stents in thrombectomy patients receiving emergent extracranial internal carotid artery stenting for tandem occlusions may occur and can be observed more often during the procedure rather than within 72 hours of follow-up, but showed no impact on early neurological outcome. The previously published higher rates of acute CASPER stent occlusions were not reproducible. The lack of follow-up data after patient discharge limit the power of this analysis regarding long-term patency of the stent. Nonetheless, to prevent acute thrombus formation and occlusion, perhaps a stricter antiplatelet or anticoagulation regimen or even longer angiographic surveillance need to be considered in the future when this type of DLS is used for treatment of patients with tandem occlusions.

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