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**Acute carotid stent thrombosis: A case report and review of literature**

Zhang JB *et al.* ACST in CAS

Jian-Bin Zhang, Xue-Qiang Fan, Jie Chen, Peng Liu, Zhi-Dong Ye

**Abstract**

**BACKGROUND**

Acute carotid stent thrombosis (ACST) is a rare but devastating complication in carotid artery stenting (CAS) procedure. The aim of this article is to report a case and review cases of ACST reported in literatures, then to investigate risk factors and management strategies for ACST.

**CASE SUMMARY**

We reviewed the treatment process of a patient with ACST after CAS. Then multiple databases were systematically searched to identify studies reporting ACST from 2005 to 2020. The demographic data, risk factors, treatment strategies, and prognosis were extracted and analyzed.

**CONCLUSION**

The reason for ACST is multi-factorial. Proper patient selection, normative anti-platelet treatment and perfect technical detail may decrease the incidence of ACST. Several treatment strategies, such as thrombolysis, mechanical thrombectomy and open surgery could be selected for the treatment of ACST. Limited data showed that carotid endarterectomy (CEA) is effective and have favorable results.

**Key Words:** Acute carotid stent thrombosis; Carotid artery stenosis; Carotid artery stenting; Case report

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**Core Tip:** The reason for acute carotid stent thrombosis (ACST) is multi-factorial. Proper patient selection, normative anti-platelet treatment and perfect technical detail may decrease the incidence of ACST. Several treatment strategies, such as thrombolysis, mechanical thrombectomy and open surgery could be selected for the treatment of ACST. Limited data showed carotid endarterectomy is effective and have favorable results.

## <sup>1</sup> INTRODUCTION

Carotid artery stenting (CAS) has emerged as an alternative to carotid endarterectomy (CEA) in treating carotid artery stenosis<sup>[1]</sup>. With the development of instruments and techniques, CAS is more and more widely used. The most common reported complications for CAS were thromboembolic events. Acute carotid stent thrombosis (ACST) refers to thrombus formation in carotid artery during or after CAS procedure, which is rare but may be devastating<sup>[2]</sup>. The etiology, treatment strategy and prognosis are not well reported in the past.

In the current study, we reported the treatment process of a case with ACST caused by stent cutting plaque. The patient gave consent to use his medical record for educational and publication purpose. Then we reviewed the current published literatures reporting cases of ACST, aiming to investigate the etiologies, management strategies, and prognosis for ACST.

## CASE PRESENTATION

### *Chief complaints*

Dizziness and transient left limb weakness for 2 mo.

### *History of present illness*

Dizziness and transient left limb weakness for 2 mo.

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### *History of past illness*

The patient denied any history of hypertension, diabetes mellitus, or coronary atherosclerotic disease, but had a history of smoking for more than 40 years (20 cigarettes/d).

### *Personal and family history*

### *Physical examination*

Abdominal palpation showed the presence of pulsatile mass, auscultation showed murmur at right carotid artery.

### *Laboratory examinations*

### *Imaging examinations*

Carotid ultrasonography and computed tomography angiography (CTA) showed 95% stenosis of right internal carotid artery (ICA). Aortic CTA showed infrarenal AAA with maximum diameter of 53 mm and mural thrombus formation.

### **FINAL DIAGNOSIS**

The patient was diagnosed with combined symptomatic ICA stenosis and infrarenal AAA.

## TREATMENT

For the ICA stenosis, the pre-operative CTA showed the plaque is relatively soft with less calcification. According to plaque morphology (the PLAC Scale) on CT angiography<sup>[3,4]</sup>, soft plaque and lower grade of calcification were associated with better stenting outcome. Though with transient ischemia attack (TIA) 2 mo ago, we prefer to carry out CAS for the patient. For the combined infrarenal AAA, it is classical with enough landing zone. The estimated time for endovascular aneurysm repair (EVAR) procedure is not long. So we plan to perform right CAS and standard EVAR simultaneously. Before the procedure, 100 mg aspirin and 75 mg clopidogrel daily was given for 5 d. The procedure was performed under general anesthesia. During procedure, activated clotting time was 302 s after administration of 6000 IU unfractionated heparin. The angiography showed 95% stenosis at the proximal part of right ICA. After pre-dilatation with 4.0-20 mm and 5.0-20 mm balloon (AVIATOR, Abbott) sequentially, under the protection of embolism protection device (EPD, Emboshield NAV6, Abbott). A taper stent (7-9-40 mm, XACT, Abbott) was implanted. Post-procedure angiography showed contrast agent retention in the stent and the distal of ICA was obliterated. ICA spasm and plaque debris occluded EPD was suspected. Then we gave the patient 30 mg papaverine and retrieved the EPD. Subsequent angiography showed the ICA was patent with 40% residual stenosis because of spasm. After another 3000 IU unfractionated heparin was administered, standard EVAR was performed with stent graft system (2316170, ENDURANT, Medtronic) implanted smoothly. After EVAR, we performed another carotid angiography to evaluate whether the ICA spasm alleviated. However, we found acute thrombus formation in the stent (Figure 2). Worrying about thrombus migration and distal embolization, we did not try to aspirate thrombus through the catheter. Instead, we decided to perform CEA immediately to remove the carotid stent and thrombus thoroughly. Then we carried out the standard CEA procedure with shunt (Aesculap, Braun) implantation and patch

(Edwards) angioplasty. During CEA procedure, we can see the stent was occluded by the plaque debris.

### **OUTCOME AND FOLLOW-UP**

The patient was conscious and limbs can move freely after awoke from general anesthesia after CEA. The patient also has no neurological deficit at 1-year follow-up

### **DISCUSSION**

CAS has become an alternative treatment to CEA for carotid artery stenosis. The most common complication for CAS is peri-procedure stroke<sup>[31]</sup>. The incidence of ACST is rare, which is reported to be 0.5%-2%<sup>[21]</sup>. Though rare, delayed recanalization of ICA or inappropriate management may lead to devastating consequence.

The etiology of ACST is multi-factorial. Inadequate, cease, or resistance of anti-platelet agent is an important reason. The incidence of acetylsalicylic acid resistance is 5.5%-45% and incidence of clopidogrel resistance has reported to be 4%-44%<sup>[12]</sup>. Pre-operative evaluation of sensitivity to anti-platelet agent is helpful. Light transmittance aggregometry (LTA) is the golden standard to measure platelet aggregation.

Hyper-coagulable state including cancer history, protein C deficiency, taking oral contraceptives, heparin induced thrombocytopenia, and thalassemia is also common. For these patients, additional anti-coagulative agent may be necessary. Local factors such as plaque protrusion in stent and debris occluding the EPD occurred occasionally. Embolic events occurred in atrial fibrillation patients, accidentally after CAS, is rare<sup>[5]</sup>. Iatrogenic injury, such as balloon burst during the procedure, should be avoided<sup>[22]</sup>.

In our present patient, no evidence of hyper-coagulable state was discovered. Pre-operative dual anti-platelet agent was given for 5 d. During CAS and EVAR procedure, we gave the patient sufficient unfractionated heparin and the activated clotting time was suitable for interventional therapy. Stent cutting plaque could be found during CEA. So we think the initiating factor of platelet aggregation may be the exposure of thrombogenic material after plaque rupture<sup>[13]</sup> in our case. The presence of TIA

indicated that the plaque is not stable. But we did not attach enough importance to it and underestimated the risk of CAS. From our present case, we want to emphasize again that pre-operative plaque evaluation is very important for decrease the rate of plaque rupture and subsequent ACST. Vulnerable or ulcerated plaque is more suitable for CEA instead of CAS. It is a pity that we did not evaluate whether the patient was resistant to aspirin or clopidogrel.

The present procedure was performed under general anesthesia for possible breath control during EVAR. This may decrease the patient's blood pressure and prolong operation time, which is another hidden reason for ACST. General anesthesia also made it impossible to perceive the patient's neurological deficit in time. For CAS procedure, local anesthesia with close intra-operative neurologic status monitoring is more suitable.

The best treatment strategy for ACST is controversial. Multiple methods have been reported, all without large series experience. Catheter directed thrombolysis with alteplase, urokinase, tenecteplase is mostly used. Most of the patients achieved successful complete or partial recanalization. Administration of abciximab intravenously or intra-arterially also has been reported to be effective<sup>[18,28]</sup>. 'Facilitated thrombolysis' consisting of reduced dosage of de-thrombolysis (abciximab) and fibrinolysis (alteplase) <sup>3</sup> may be beneficial with respect to both arterial recanalization and suppression of recurrent thrombosis<sup>[29]</sup>.

Other endovascular management strategies including catheter thrombo-aspiration, balloon angioplasty and subsequent stent implantation<sup>[5,10,13,16]</sup>. These methods are helpful for carotid revascularization, but balloon angioplasty and subsequent stent implantation may cause extra intima injury or damage stent structure.

The reported open surgical treatment including STA-MCA bypass and CEA. STA-MCA bypass is considered as second-line treatment, which may be beneficial to patient with ACST for whom intra-arterial thrombolysis is not available<sup>[14]</sup>. CEA with thorough removal of stent and thrombus have shown favorable results<sup>[24]</sup>. We believe that urgent stent and thrombus removal should be performed for patients with plaque rupture and



ACST to prevent thrombus extension and subsequent cerebral infarction. Unlike traditional CEA, extended exposure of carotid artery is required both proximally and distally in these situations. The ICA should be clamped first to prevent thrombosis dislocation when dissected the carotid artery. Fogarty catheter may be necessary to pull out the thrombus in distal ICA. Another reason that we choose CEA is that we did not have enough experience in endovascular treatment for ACST.

Mechanical thrombectomy using Penumbra 4Max aspiration catheter and rheolytic thrombectomy system is also reported to be effective. Limited experience showed that mechanical thrombectomy could effectively recanalize ICA with ACST, without the irritation of balloon angioplasty or stent deployment. The hemorrhagic risk is lower than thrombolysis. It also has the advantage of easily switch to other treatment methods if failed<sup>[21,28,30]</sup>.

## **CONCLUSION**

The reason for ACST is multi-factorial. Proper patient selection, normative anti-platelet treatment and perfect technical detail may decrease the incidence of ACST. Several treatment strategies, such as thrombolysis, mechanical thrombectomy and open surgery could be selected for the treatment of ACST. Limited data showed that carotid endarterectomy (CEA) is effective and have favorable results.

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