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CORRESPONDENCE

Delayed tine Displacement of a CASPER Carotid Artery Stent due to Styloid Process Compression

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Background

Eagle syndrome results from an elongated styloid process or calcified stylohyoid ligament causing impingement [1, 2]. This interaction causes injury to the carotid artery which may result in pseudoaneurysm, dissection or focal stenosis [1]. A similar interaction may occur when carotid stents are placed due to the alteration in biomechanics.

Stent fracture and deformation have been reported for laser-cut stents, with overall rates of fracture and deformation ranging from 3% at 1 year, to 50% at 4 years, and this can be complicated by restenosis in up to 36% [3, 4]. Fractures are associated with densely calcified arteries, angulated arteries and open cell stent designs [3–5]; however, most published case series are based on stents in the proximal internal carotid artery and carotid bulb, a location which experiences high torsional strain and has exposure to external compression [3–5].

The CASPER (Microvention Terumo, Aliseo Viejo, CA, USA) carotid stent is a woven stent, which is a design considered to be more conformable than laser-cut stents; however it may still be susceptible to focal deformations. This article presents a case of early stent deformation, unusual in that it occurred with this newer generation stent, in the first 2 weeks, and in the high cervical internal carotid artery. It is believed that the styloid process may have played a role in its pathogenesis.

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Case Presentation

A 50-year-old male smoker with no significant medical history presented with 4 h of right hemiparesis and aphasia. The National Institutes of Health Stroke Scale (NIHSS) score was 13. Noncontrast and perfusion computed tomography (CT) of the head and CT angiography of the neck were performed, demonstrating no flow into the left cervical internal carotid artery (ICA) and partially occlusive thrombus in the proximal M1 segment. The patient underwent endovascular thrombectomy under conscious sedation; the occlusion was crossed with a Synchro2 Standard 0.014″ wire (Stryker Neurovascular, Fremont, CA, USA), Headway-27 and Sofia Plus 6 French catheters (Microvention Terumo, Aliseo Viejo, CA, USA). Removal of the intracranial thrombi required 4 stentriever passes due to the volume of thrombus. Final revascularization was consistent with a modified thrombectomy in cerebral ischemia (mTICI) score of 3. Cervical ICA digital subtraction angiogram (DSA) revealed a spiral dissection extending from distal cervical ICA to the lateral segment, which had been the likely initial cause of carotid occlusion. The patient was loaded orogastrically with aspirin 600 mg and clopidogrel 600 mg, and the dissection was jailed with a 5 × 35 mm PED-SHIELD (Medtronic, Minneapolis, MN, USA) and angioplastied with a Transform 7 × 7 mm balloon (Stryker Neurovascular). As the extracranial ICA was large, the stent construct was extended inferiorly with a CASPER RX 7 × 18 mm (Microvention Terumo) to mid-C1 level, covering the entirety of the dissected segment (Fig. 1).

Postprocedure, the patient awoke with no neurological deficits except a left partial Horner's syndrome. The postoperative imaging only demonstrated small left globus pallidus and caudate head hypodensities, with patent stent construct which was well apposed throughout. The patient was discharged home after 2 days.

The patient subsequently represented 2 weeks postprocedure with ongoing left-sided headache and neck pain, worse on right lateral rotation with cervical flexion. He had not experienced any interval cervical trauma. The neu-

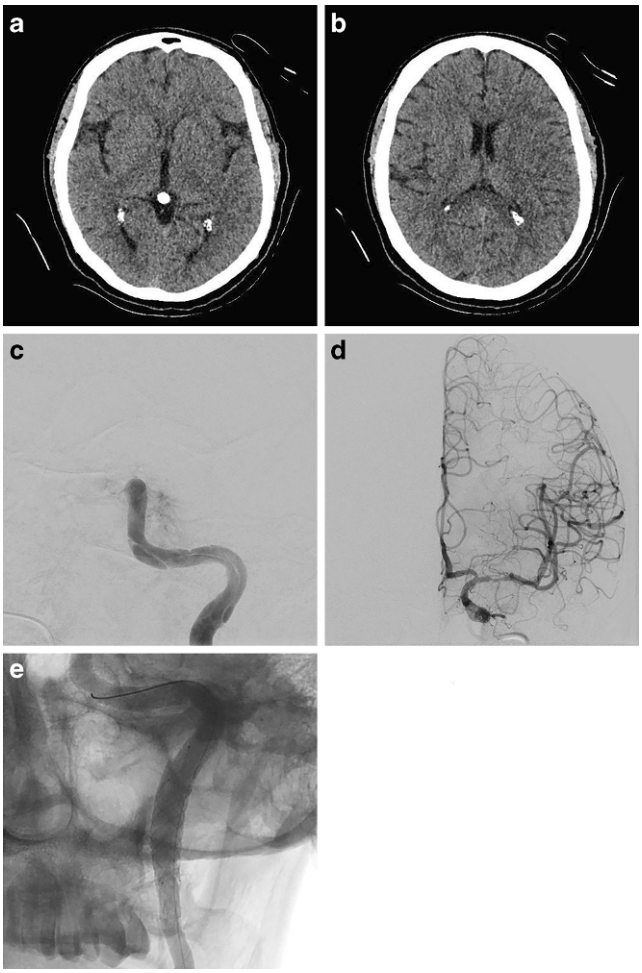

Fig. 1 CT head, axial reconstructions at the **a** level of the basal ganglia and **b** mid-thalamic level on initial presentation, demonstrating no established infarct. **c** Left ICA angiogram AP oblique projection at the level of the petrous canal demonstrating the dissection which was the underlying cause for the thromboembolism. **d** Townes projection left ICA angiogram demonstrating recanalization and mild spasm in the left M1 segment from stentriever thrombectomy. **e** Unsubtracted AP oblique angiogram of the left ICA stent construct demonstrating its apposition to the vessel wall

rological examination found a slightly worsened Horner's syndrome. A repeat CT and CTA of the head and neck were performed as work-up for the persistent neck pain. These studies demonstrated new medial displacement of the most inferolateral tine of the CASPER stent, protruding into the vessel lumen (Fig. 2). Due to this appearance, a catheter cerebral angiogram was performed to assess for any dynamic interaction. This confirmed that the inferolateral stent tine was displaced and found that the carotid to styloid process distance was decreased by 30% on right lateral neck rotation, but further rotation was limited by patient pain and was hence not pursued (Fig. 3). The styloid process was 31 mm long on the left and 32 mm long on the right (normal 20–25 mm).

Outcome

As the buckling of the stent strut was non-flow limiting, a conservative approach was taken. The patient's symptoms and partial Horner's symptomatology improved over subsequent weeks, with no further stroke. A 6-month catheter angiography demonstrated no further distortion of the stent construct, but one stent strut continues to protrude into the lumen of the internal carotid with associated narrowing (Fig. 4). It is known that stent struts not opposed to a vessel wall remain nonendothelialized for longer. To reduce this risk of stent thrombosis the patient will be kept on long-term dual antiplatelet therapy, until there is a contraindication requiring aspirin monotherapy. Ongoing follow-up will be performed to monitor for further stent distortion and in-stent restenosis.

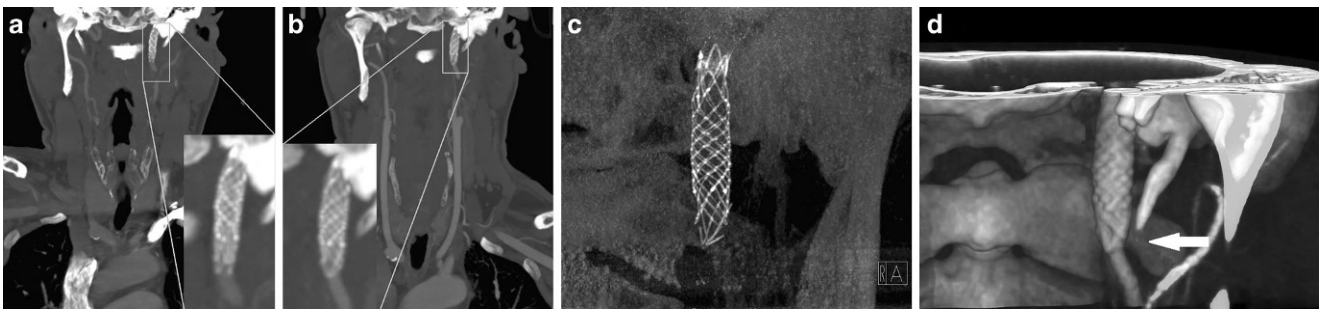

Fig. 2 **a** Day 1. Baseline CT angiography of the neck, coronal maximum intensity projection demonstrating the position of the stent, with the inferior stent configuration demonstrated in the inset. **b** Day 15. Follow-up CT angiography demonstrating the altered stent tine configuration (inset). **c** Day 22. Cone beam CT demonstrating altered stent tine configuration. **d** Day 15. 3D volumetric render of CT angiography showing the close relation of the stent and the tip of the styloid process (head straight)

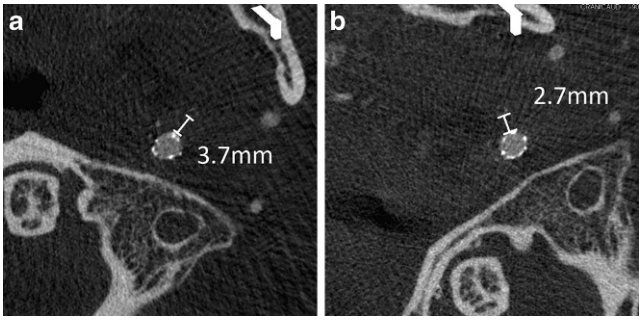

Fig. 3 Day 22. Cone beam CT, axial reconstructions at the level of C1, showing the decrease in distance between the tip of the styloid process and the stent, in **a** neutral head position and **b** head right laterally rotated position (limited by patient pain)

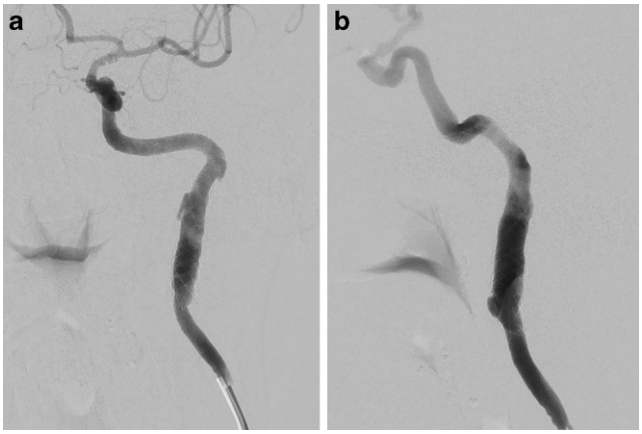

Fig. 4 Day 174. Digital subtraction angiogram of the left ICA stent construct in **a** AP oblique and **b** lateral oblique views showing mild stenosis but no further stent deformation

Discussion

This case is unusual in that the deformation of the stent occurred within 2 weeks of placement. Given the patient had not experienced any interval cervical trauma, in the presence of the longer styloid processes bilaterally the only potential etiology that could be ascertained was that of compression from the ipsilateral styloid process. This mechanism of stent deformation mimics Eagle syndrome, which would explain increasing pain on lateral head rotation.

Whilst direct contact of the styloid process with the ICA or carotid stent was not visualized during this study, the patient had to hold his head in a right laterally rotated and flexed position to pain tolerance whilst performing a cone-beam CT. The patient previously could have turned further, and deformed the stent, however it was not felt necessary to put the patient in further discomfort and risk further stent deformation to prove the interaction.

At the time of initial diagnosis of stent distortion, several treatment strategies were considered for the patient. Balloon angioplasty may re-oppose the stent tine to the vessel wall, but it was felt that this was not justified as the stent may distort again via the same mechanism. Initially placing the proximal stent margin inferior to the styloid may have placed a stronger section of stent to resist the styloid compression, since in our experience the CASPER stent has less radial force at the proximal and distal ends compared to traditional laser-cut carotid stents; however, as the patient was nearly asymptomatic it was considered unreasonable to expose the patient to the risks of placing a further stent.

To our knowledge, this case is the first published case of CASPER stent deformation, and of symptomatic stent deformation possibly due to styloid impingement.

Learning Points

- Stents placed in the high cervical carotid are susceptible to deformation.
  The extremities of the CASPER stent are vulnerable to external compression resulting in deformation.
- The size and location of the styloid process and stylohyoid ligaments may be a consideration when planning endovascular stenting of the carotid arteries to minimize the risk of stent deformation.
- Knowledge of the potential for stent disruption by the styloid process in certain patients will enable careful observation and follow-up imaging decisions.

Compliance with ethical guidelines

Conflict of interest D. Tan, M. Crockett and A.H.Y. Chiu declare that they have no competing interests.

Ethical standards All procedures reported in this article were in accordance with the ethical standards of the National Health and Medical Research Council Australia and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. The patient provided informed consent for publication of images and text regarding the medical condition.

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