

Histopathologic Description of Wingspan Stent in Acute Ischemic Stroke

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Abstract

Objective and Importance We report the histopathologic examination of Wingspan stent in acute ischemic stroke.

Clinical Presentation A 75-year-old female presented with acute left-hemiplegia due to right carotid terminus occlusion. Mechanical embolectomy was unsuccessful.

Intervention A Wingspan stent was placed from the distal intracranial carotid artery to the proximal middle cerebral artery stem and established partial antegrade flow. The patient died of malignant infarction on post-stroke day 7. At autopsy, embolized calcified atherosclerotic plaque fragments were noted within a non-occlusive thrombus over which the Wingspan stent was deployed. There was no evidence of intimal or media dissection or perforator ostium occlusion.

Conclusion Our case provides a rare pathological description of intracranial stent placement in the setting of acute ischemic stroke.

Keywords Angioplasty · Intracranial stenosis · Wingspan stent · Stroke

Introduction

Emergent balloon angioplasty or stent placement has been recently described as an alternative therapeutic approach in the treatment of acute ischemic stroke [1–4]. Self-expanding open-cell stents have an emergent role in the treatment of acute stroke [5]. We report the histopathologic examination of Wingspan stent in acute ischemic stroke.

Case Report

A 75-year-old woman with a history of hypertension presented to an outside hospital with sudden onset left hemiplegia. Neurological examination demonstrated left hemiplegia, neglect, and right gaze preference. Initial CT brain was negative. Blood pressure was 263/98 mmHg. She was transferred to our institution 6 h after the onset of symptoms, with a National Institutes of Health Stroke Scale score of 18. The patient arrived beyond the intravenous tissue plasminogen activator (tPA) and intra-arterial tPA window. Cerebral angiogram showed a right internal carotid artery (ICA) terminus (“carotid T”) occlusion (Fig. 1a). Mechanical embolectomy was unsuccessful. The patient subsequently underwent balloon (Gateway, 1.5 × 15 mm) angioplasty without success. Finally, a Wingspan stent (4.5 × 15 mm) was placed from the distal ICA to the proximal middle cerebral artery (MCA) stem (M1 segment). Thrombolysis in myocardial infarction (TIMI) 2 antegrade flow was established at 8 h after stroke onset (Fig. 1b). The patient’s post-procedure neurological examination was

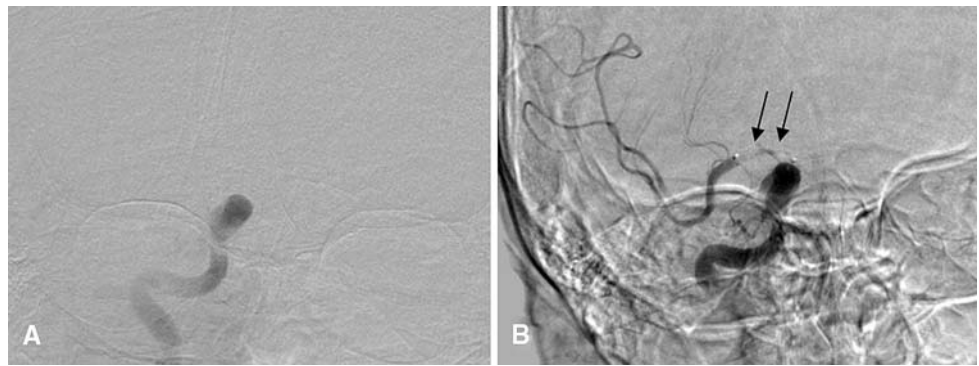
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Fig. 1 Cerebral angiogram. **a** Right carotid “T” occlusion. **b** After Wingspan stent placement, distal MCA flow is achieved (*double arrows*)



unchanged. Hemiraniectomy was declined by the family. Repeat CT brain showed an evolving right MCA territory infarct with mass effect (Fig. 2). The patient died of malignant infarction on post-stroke day 7.

Pathological Findings

On gross examination, the brain showed a right MCA infarct with bilateral cerebral edema, subfalcine herniation, right uncal hemorrhage, Duret hemorrhages in the pons and midbrain, and a right posterior cerebral artery infarct. The

stent containing segment of the right MCA/ICA was sent to Boston Scientific (Natick, MA, USA) for processing. The EXAKT processing method of saw-grind-surface polish and surface stain is required to cut the Wingspan stent (EXAKT TECHNOLOGIES Inc. 7416N. Broadway Extension, Suite E Oklahoma City, OK 73116-9066). This method creates H&E surface stained microscopic sections for analysis that are 40–50 μ thick.

Microscopic Description

The stented MCA lumen was patent at all levels. In the distal M1 segment, there was asymmetric plaque near the ostia of a small arterial bifurcation (lenticulostrate perforator) (Fig. 3a, b). In the proximal M1 segment with a portion of the ostial ACA lumen, Wingspan struts were present in both the MCA and ACA (Fig. 3c). Strut members were closely apposed ($< 100 \mu$) to the intimal surface in the MCA while three struts in the ACA exceeded a 100μ gap width. There was non-occlusive subacute thrombus in the MCA stent and focal intimal lipid plaque at the ACA ostia. In the segment of the ICA terminus, stent struts appeared to be in close apposition ($< 100 \mu$) to the intimal surface (Fig. 3d). Non-occlusive mural thrombus, age estimated at 4–8 days, appeared to have existed prior to stent placement and occupied an estimated 40–50% of the lumen area. Tracks through the thrombus correlated with strut placement, and thrombus was compressed between some of the struts and the lumen. Within the thrombus were fragments of calcified embolized plaque. At all levels, the media and adventitia were unremarkable without evidence of dissection. Stent struts were not covered by neointima due to the early time duration after deployment.

Discussion

The histopathological changes after endovascular treatment of acute stroke with angioplasty and stenting are poorly

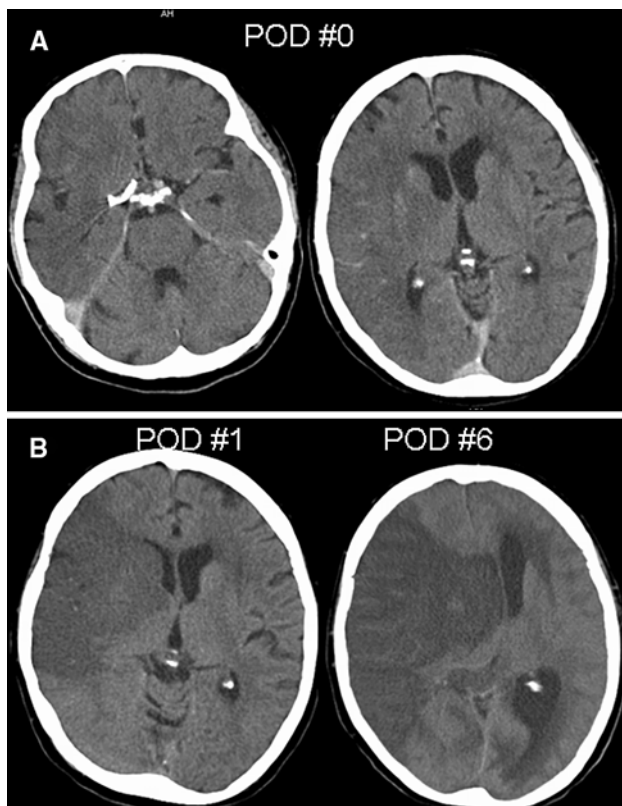
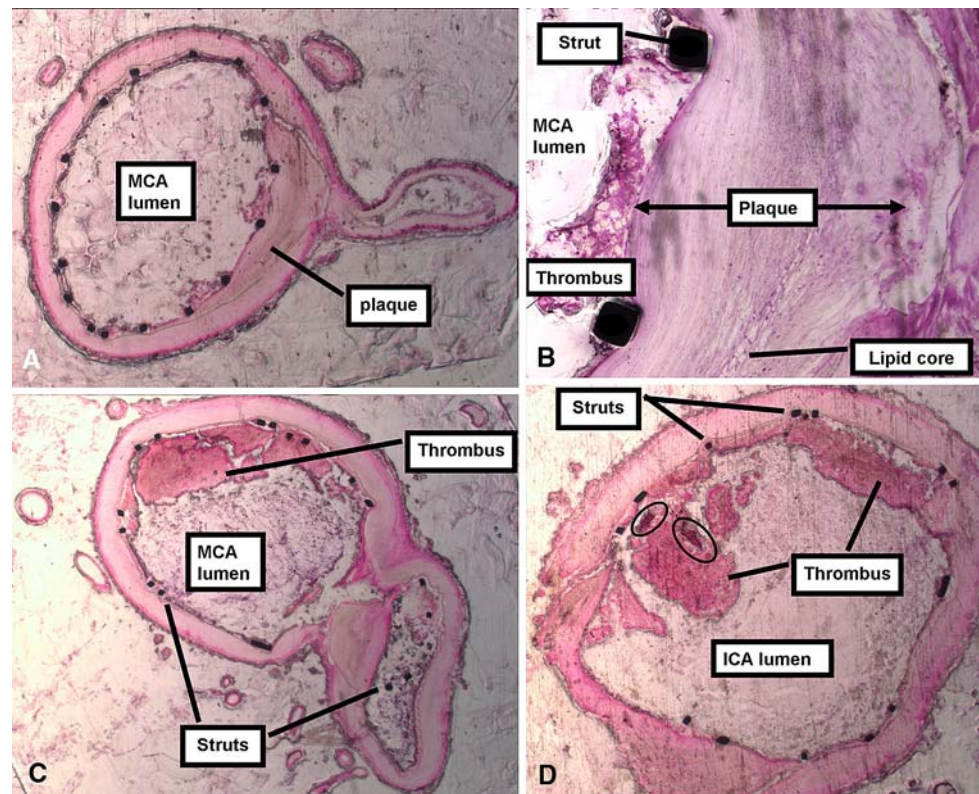


Fig. 2 Noncontrast CT brain on post-operative day (POD) # 1 and # 6 shows evolution of malignant right MCA infarct

Fig. 3 Histopathological findings. **a** Distal M1 segment. The MCA is patent with multifocal patches of small mural microthrombi (1–3–5 O'clock) and asymmetric plaque near the ostia of a small arterial bifurcation. **b** Magnified view of the plaque. **c** Proximal M1 segment with a portion of the ostial ACA lumen. Wingspan struts are present in both the MCA and ACA. There is a non-occlusive subacute mural thrombus in the MCA. **d** ICA terminus segment. Within the thrombus are fragments of calcified embolized plaque (*ovals*). Linear tracks through the thrombus correlate to strut placement



understood. Although pathologic data inferred from the coronary literature can be applied to intracranial arteries, there are many anatomic limitations of direct extrapolation to cerebral vasculature. Compared with coronary vessels, cerebral arteries lack a well-formed external elastic lamina and are relatively fixed from movement due to small branching arteries [6]. Disruption of perforating arteries during parent vessel intervention may result in hemorrhagic complications. Coronary stents are balloon-mounted stents deployed using the technique optimal “sizing.” The suggested mechanism of acute luminal gain in cardiac angioplasty is mainly arterial stretching, associated focal wall trauma, and lesion dependent plaque modification (cracking and redistribution) [7]. In the cardiac literature, the histology of bare metal coronary stents demonstrate plaque compression by stent struts, with early thrombus formation and inflammation followed by delayed neointimal growth [8].

The use of primary angioplasty and balloon-mounted stents in intracranial vessels has been reported with many limitations. Angioplasty alone may be associated with vessel dissection that may not be evident during the intervention, and this is more likely during “optimal sizing”, which may be more likely to cause intimal and medial tears. Schumacher et al. [9] reported histology after MCA angioplasty, and found plaque disrupted and separated from the underlying media (consistent with dissection) and focal intramural hemorrhage within the eccentric atheroma of the

vessel. Angioplasty-related dissection of the vessel wall could lead to vessel re-occlusion, and stenting may theoretically prevent this by reapposition of the layers dissected by angioplasty. However, a major perceived risk of balloon-mounted stents is the “snow plowing” effect (forceful plaque displacement causing perforator vessel occlusion at the ostium) [10]. Jiang et al. [11] reported perforator strokes in 3% of patients who were treated with balloon-expandable intracranial stenting for symptomatic atherosclerotic disease. Phatouros et al. [12] reported pathology 10 days after angioplasty and stenting of a basilar artery stenosis using a cardiac stent and demonstrated hemorrhage within the plaque over the rostral end of the stent. Delayed in-stent restenosis is also a concern, and Lopes et al. [13] reported focal narrowing at the MCA stent lumen due to neointimal growth 6 months after placement.

The angioplasty technique used in this case was “sub-optimal angioplasty”, which inflates the balloon to less than 80% of the parent vessel size. The “submaximal angioplasty” technique has theoretical advantages over balloon-expandable stent that may allow it to be better suited for intracranial vessels [14]. Self-expanding stent deployment requires less force on the vessel wall compared to balloon-mounted stents, and this would theoretically decrease the risk of parent vessel dissection, rupture, and “snow plowing” effect [14]. In our case, histopathology shows a patent perforating branch originating within the stented artery and there was no evidence of angiographic or

pathologic dissection in this acute setting. Though it is possible that sustained radial force of self-expanding stents or neointimal proliferation may theoretically occlude perforators in a delayed fashion, preliminary reports suggest that this does not appear to be the case. Leung et al. [15] reported no new clinical stroke or late perforator infarcts on follow-up MRI in 24 patients with self-expanding stent placed for high-grade MCA stenosis.

We acknowledge the limitations of our study, including the retrospective nature of the case report and the pathological limitations of the EXAKT method, which does not allow high-quality resolution histology on plaque morphology due to the thickness of the sectioning method. Histological descriptions of in-stent pathology are limited, and the goal of this report is to provide the description of an open-cell-design stent on an acute intracranial occlusion. In our patient, the mechanism of stroke was likely related to plaque rupture, as evidenced by embolized calcified atherosclerotic plaque fragments present within a non-occlusive thrombus in the stented segment. The cause of stroke remains unknown; as there was no evidence of a cervical carotid lesion angiographically, a proximal embolic source from aortic arch atheroma is a reasonable presumption, especially given the calcified material noted pathologically at the site of occlusion. Interestingly, the stent struts compressed the thrombus against the vessel wall as evidenced by the track marks left by the struts. The radial force of the self-expanding stent appears to be adequate for clot disruption without significant injury to the plaque or vessel wall. To our knowledge, this case represents the first histopathological description of a self-expanding stent in the setting of acute stroke.

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