

Late Coronary Ostial Stent Fracture and Embolism Causing an Acute Thrombotic Occlusion of the Carotid Artery With Cerebral Infarction

Apostolos Th. Mamopoulos, MD; Thomas Nowak, PhD; Heinrich Klues, PhD; Bernd Luther, PhD

A 56-year-old woman presented 30 minutes after onset of a right arm paresis. The cardiovascular risk factors included high cholesterol levels and heavy smoking, leading to a posterior myocardial infarction 6 years earlier, with stenting of a subtotal right coronary artery (RCA) ostial stenosis (3×13 mm bare metal stent). A symptomatic 70% RCA ostial restenosis, 9 months after initial percutaneous transluminal coronary angioplasty, led to the deployment of a 3×18 mm Cypher stent (Cordis, Warren, New Jersey) within the former RCA stent, postdilated (3.5×10 mm and 4.0×20 mm balloon, 25 bar) with a satisfactory result, the proximal end of the drug eluting stent (DES) protruding into the aorta (Figure 1A). The carotid arteries were known to be free of stenotic lesions. After the admission in the neurology department, a computed tomography examination revealed a significant reduction in the perfusion of the right brain hemisphere without cerebral infarction, the subsequent computed

tomography-angiography showed a thrombotic occlusion of the extracranial right internal carotid artery, with a patent intracranial segment (Figure 1B). The cause of the occlusion was not recognized. An ultrasound examination confirmed the presence of recent thrombotic material occluding the carotid artery. Because of the recent onset of the symptoms and lack of cerebral infarction, we decided that immediate surgery was the best course of action. The patient underwent a standard carotid artery thrombectomy under general anesthesia. After the arteriotomy of the internal carotid artery, the thrombotic nature of the occlusion was evident. During the successful thrombectomy with a 2F, 4 mm Fogarty catheter we came upon a stent embedded in thrombus (Figure 2A–2C). With strong backflow from the internal carotid artery, we closed the arteriotomy with a vein patch under shunt protection. The right internal carotid artery was patent without evidence of intracranial embolism. The patient

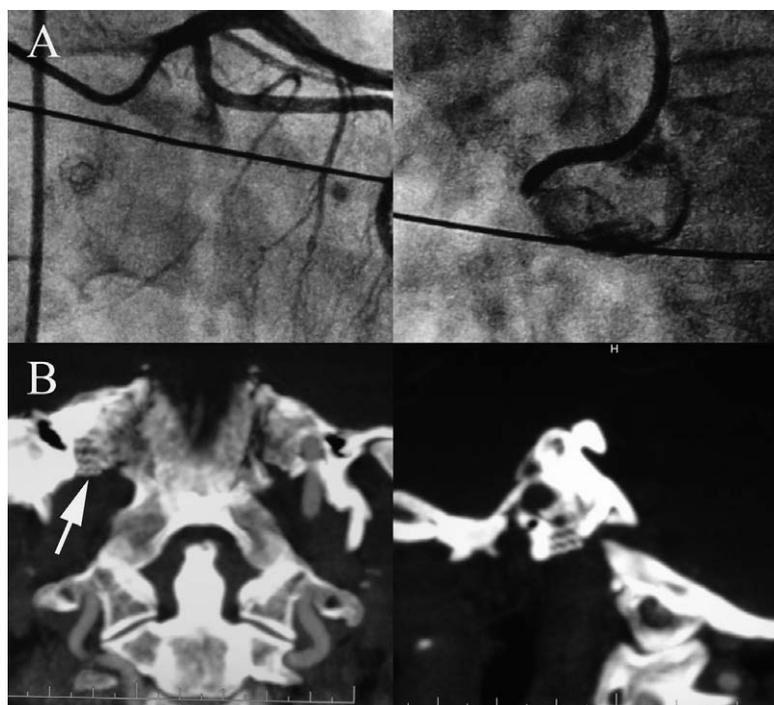


Figure 1. A, A drug eluting stent placed at the ostium of the right coronary artery, inside a bare metal stent, with its proximal end protruding into the aortic lumen, 6 years before the fragmentation and embolism of its proximal end. B, A fragment of the drug eluting stent lodged in the carotid siphon on the right side (white arrow), leading to an acute thrombotic occlusion of the arterial lumen. The carotid artery on the left side shows a normal perfusion. The cause of the occlusion was not recognized preoperatively.

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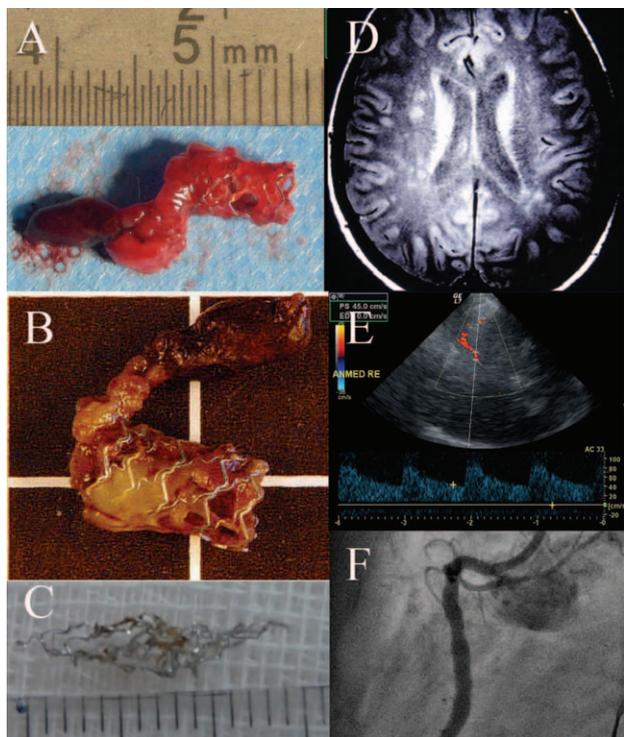


Figure 2. A, B, Intraoperative finding of a 8 mm-long stent fragment embedded in recent thrombus recovered during a thrombectomy of the internal carotid artery. C, Deformed stent fragment after thrombus removal. D, Postoperative diffusion magnetic resonance images showing multiple acute infarctions in the right hemisphere with the patient remaining clinically completely asymptomatic. E, Ultrasound examination of the carotid and middle cerebral artery 3 years later with normal findings. F, The ostium of the right coronary artery remained patent in this coronary angiography 3 years later.

made a complete recovery postoperatively with full reversal of the neurological symptoms. A postoperative magnetic resonance imaging examination showed multiple ischemic lesions in the right hemisphere of the brain (Figure 2D), and a closer look at the preoperative computed tomography-angiography revealed the stent fragment lodged in the carotid siphon. The histological examination showed a 3×8 mm metallic stent embedded in recent thrombus, representing a fragment of the 18 mm-long protruding Cypher stent. The RCA remained patent and the asymptomatic patient was released on the sixth postoperative day. Follow-up, 3 years later, showed a normal perfusion of the right carotid artery and the RCA ostium (Figure 2E and 2F).

Discussion

Coronary stent dislocation with peripheral embolism has been sporadically reported, the overall incidence is very low (0.16%–1.5%). The vast majority of cases involve balloon expandable stent dislodgement from its delivery system during deployment. A late retrograde migration of a coronary stent in the peripheral circulation, on the contrary, is a rarity. Stent fracture with dislodgement of the severed fragment can be a possible cause, with DES at a coronary artery ostium, when protruding into the aortic lumen, being particularly susceptible to peripheral embolism. Studies based showed an increased rate of DES fractures compared

to bare metal stents with a reported clinical incidence of 1.5% to 1.9% of patients (3.2% of stents). Predisposing factors for stent fracture seem to be ostial stents with a protruding proximal end, RCA placement, high-pressure postdilatation with oversized balloons, sirolimus eluting rigid stents with closed cell design, overlapping or longer stents, and increased vessel tortuosity. DES placed to treat ostial stenosis often have the proximal end left protruding to the aortic lumen exposing it to considerable mechanical strain. This mechanical stress can be exacerbated when the protruding stent is placed in another stent, thus increasing the rigidity of the implantation site. An experimental model of the stress inducing forces exerted upon coronary stents at the coronary ostium showed that the Cypher stent suffered significant plastic deformation with considerable decrease in luminal area and a sudden strut fracture in the proximal portion adjacent to the ostium, whereas the mid and distal portions remained unaffected.¹ The majority of dislocated stents usually end up in the lower extremities as low as the pedal arteries, others are found in the descending aorta or the renal arteries. Peripheral embolising stents remain asymptomatic, only rarely they can cause peripheral ischemia.²

There are reported cases of fractured protruding ostial stents with peripheral embolism of the severed proximal end. In 1 such case, a separated fragment of a protruding DES, placed inside another DES at the RCA ostium, was located at the aortic root during angiography. During aortic valve replacement, the stent fragment was found to be anchored to the aortic root by a fibrous strand, which prevented peripheral embolism.³ In 2 other cases, a DES placed at the ostium of the RCA suffered a complete fracture, the severed segment migrated somewhere in the peripheral circulation of the asymptomatic patient. In a fourth case, the aortic end of a protruding DES, placed at the ostium of an aortocoronary bypass, fractured causing in-stent restenosis. The proximal fragment was not completely disarticulated and was removed with an endovascular snare. In another incident similar to our case, a patient underwent DES implantation at the RCA ostium with later placement of a second overlapping DES. The patient was finally referred for a bypass operation. The preoperative examination revealed a 70% to 80% asymptomatic carotid artery stenosis, intraoperatively a 4 mm stent fragment was found lodged in a preexisting plaque at the origin of the internal carotid artery, the patient remained asymptomatic.⁴

To our knowledge, this is the first reported occurrence of a symptomatic late peripheral embolism of a coronary stent fragment in the cerebral circulation and underlines the potential for serious consequences that accompanies coronary stent migration.

Disclosures

None.

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KEY WORDS: carotid artery occlusion ■ cerebral infarction ■ protruding stent ■ stent embolism ■ stent fracture

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