Case Report

Bilateral spontaneous internal carotid artery dissection managed with endovascular stenting – A case report

Manoj Kumar Agarwala*, Azeez Asad, Naveen Gummadi, Sundar Chidambaram, J. Venkateswaralu
Apollo Hospitals, Hyderabad, India

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ABSTRACT

Carotid artery dissection (CAD) is a frequent cause of stroke, accounting for up to 25% of all ischemic strokes in young and middle-aged patients. It may be traumatic or spontaneous, with multi-factorial etiology. A tear in the arterial wall causes intrusion of blood within its layers, producing intra-luminal stenosis, or aneurysmal dilatation. Thrombo-embolism arising from this anatomic disruption has been postulated as the essential stroke mechanism in CAD. Bilateral internal carotid artery dissection (ICAD) has been rarely reported. Antiplatelets and anticoagulation remain standard therapy for CAD. However, in patients with either expanding pseudoaneurysms, severe flow compromise, worsening symptoms despite anticoagulation or contraindication to anticoagulation, endovascular stenting is beneficial.

We describe a patient with ischemic stroke from spontaneous bilateral ICAD with completely occluded left ICA. Having failed medical therapy with antiplatelets and anticoagulants due to extensive loss of carotid vascular supply, he was managed successfully with endovascular stenting with good neurological recovery.

1. Case description

A 45-year-old male presented with sudden onset of headache, neck pain, and left hemiparesis following a forceful cough. He had no seizure or loss of consciousness. He had no conventional risk factors such as diabetes, hypertension, and smoking. He had been treated for a week with antiplatelets (aspirin and clopidogrel) and enoxaparin in a private hospital and subsequently referred to our stroke unit. On admission, the patient was confused, dysphasic, and had left hemiparesis. His NIH Stroke Scale (NIHSS) score was 16 and modified Rankin Scale (mRS) was 4. Biochemical analysis showed normal blood glucose, normal renal and thyroid function, and normal lipid and homocysteine levels. Carotid Doppler showed occlusion of left ICA just distal to its origin and significant luminal narrowing of right ICA. Cerebral MRI revealed bilateral subcortical periventricular acute infarcts. MR angiography showed bilateral ICAD with total occlusion of the left ICA immediately after its origin.

The patient continued to deteriorate neurologically despite antiplatelet and anticoagulant therapy, requiring elective intubation and mechanical ventilation. A 4-vessel angiogram showed bilateral ICAD just distal to the origin, extending to cavernous part of ICA with near-total occlusion of left ICA and 90% occlusion of right ICA (Fig. 1A–C). We decided to treat this patient with carotid angioplasty plus stenting in view of his deteriorating neurological status. The patient was pretreated with clopidogrel and aspirin. Under local anesthesia, an 8-French multipurpose guide catheter (Cordis Corporation, Florida, U.S.A.) was placed in the common carotid artery. Using high-resolution angiography, the true lumen of the dissected vessel was identified. A 0.014-in. floppy-tipped coronary guide wire (Balance Middle Weight, Abbott Vascular, U.S.A.) was passed distal to the lesion. Then, a progreat microcatheter (Terumo, Japan) was inserted distal to the lesion, and the wire was removed. About 0.5 ml of contrast was gently injected through the microcatheter to confirm the position of the tip in the true lumen. We initially took 6 mm × 120 mm (Protege RX, EV3, Covidien Ltd, Ireland) carotid stent to deploy in the cavernous part of ICA. However, because of the tortuosity, the stent could not be negotiated. Then 4 mm × 23 mm (Multilink ZETA, Abbott Vascular, U.S.A.) and 4 mm × 33 mm (Multilink ZETA, Abbott Vascular, U.S.A.) overlapping coronary stents were deployed in the...
cavernous part of ICA. Subsequently, 6 mm × 120 mm (Protege RX, EV3, Covidien Ltd, Ireland) self-expanding carotid stent was deployed to cover the rest of dissection. These three tandem stents placed in left ICA from its origin to cavernous part of ICA spanned the entire length of the dissection.

Good flow was achieved with reconstitution of the luminal diameter of the left ICA (Fig. 2A, B). Collaterals from the left ICA filled the right ICA. Clopidogrel along with aspirin was continued. Stent patency was evaluated with serial Doppler scans. The patient was weaned off ventilator support over the next 3 days and intensive physiotherapy was continued. He walked with minimal support at discharge. At one-month follow-up, he had total recovery of left hemiparesis, normal language function and NIHSS score 2 and mRS of 1. Carotid Doppler after three months showed patent left carotid stents and complete healing of right carotid dissection.

2. Discussion

Spontaneous carotid dissection is rare; the annual incidence for all age groups in a community-based setting was 2.6/100,000. A bilateral ICAD at presentation is rarer, approximately 2–10% of all ICADs. A number of risk factors have been investigated, including fibromuscular dysplasia (FMD), infections, cystic medial necrosis, and genetic factors. Most patients with ICAD present with cerebral or retinal ischemic symptoms. Local symptoms such as headache, neck pain, and cranial nerve signs including hypoglossal nerve lesions or Horner’s syndrome arouse the suspicion of ICAD. Conventional angiography has traditionally been the gold standard in the diagnosis of arterial dissections. This typically shows a long segment of irregular and eccentric narrowing of the vessel lumen. Intimal flaps or double lumen may be seen.

MRI techniques, being non-invasive, with excellent resolution, are replacing conventional angiography. Findings of carotid dissection include increase in the external diameter of the artery, with luminal narrowing, aneurysms, and intramural hematoma. The intimal flap is more readily evident.

Computed tomography angiography (CTA) can show the typical findings of ICAD – a narrowly eccentric lumen surrounded by a crescent-shaped mural thickening and annular enhancement, an intimal flap or a dissecting aneurysm. Ultrasoundography is useful in the initial assessment and serial imaging of patients with CAD. An intimal flap floating in the lumen is found in less than one third of cases. The most common spectral Doppler finding, however, is a high-resistance flow. Asymptomatic CADs do not require any intervention. Antiplatelets and anticoagulation remain the cornerstone of management in symptomatic patients with acute ICAD. Surgical or endovascular treatment is usually reserved for patients who have persistent ischemic symptoms despite anticoagulation or have contraindication to anticoagulation.

Endovascular treatment is associated with less risk than with surgical treatment. The main indications for stent therapy include failure of medical therapy (presence of persistent or recurrent ischemic symptoms, or progressive neurological deterioration), expanding or symptomatic pseudoaneurysm, contralateral carotid...
occlusion, impending stroke attributable to significant occlusion with poor collateral circulation, or a contraindication to anticoagulation. In our patient, there was near-total occlusion of left ICA with significant stenosis in the right ICA, probably accounting for poor collateral flow and persistent ischemic symptoms.

Advantages of endovascular therapy with stenting include obliteration of the false lumen by opposing the dissected segment of the vessel wall. It allows immediate recanalization of the culprit arteries, with instantaneous reperfusion of the ischemic brain. Once successful stenting is done there is no role for anticoagulation. In our case, due to the extensive dissection of left ICA and non-availability of such long stents, 3 tandem stents were used to cover the entire length of the dissection.

Technical difficulties are the major limitation of endovascular stenting in ICAD. The reported technical failure rate is about 5.2%. Risks associated include embolization of thrombotic fragments during stent deployment, reperfusion injury, worsening, or extension of the dissection in the undermined vessel wall and consequent rupture. Post-procedural complications of in-stent stenosis or occlusion are rare as the stenosis is non-atherosclerotic and stent deployment for dissection is less traumatic and performed at lower pressure. No procedure-related major morbidity or mortality has been reported with stenting in ICAD patients.

3. Conclusion

Carotid artery dissections are a noteworthy cause of ischemic stroke, particularly in young patients. Recent advances in noninvasive imaging have made the diagnosis easier. However, the management of carotid artery dissections is still a gray area. Endovascular therapy with stenting may be offered to high-risk selected patients who have failed medical treatment. The excellent neurological recovery in our patient with bilateral carotid dissection testifies to the fact that endovascular approach with stenting (including multiple stents) should be considered in select group of patients, not responding to other forms of treatment.

Conflicts of interest

The authors have none to declare.

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