Balloon angioplasty of the A_2 segment of the anterior cerebral artery for recurrent, symptomatic atherosclerotic disease

Case report

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Intracranial atherosclerotic disease accounts for 5–10% of ischemic strokes in the US. Lesions located in the anterior cerebral artery territory are infrequently reported. Patients in whom medical therapy fails are at a high risk for recurrent ischemic events, in which case intracranial angioplasty or stenting may be a reasonable therapy. There is a paucity of literature describing angioplasty of fixed atherosclerotic lesions affecting the anterior cerebral artery territory, and especially the A_2 segment. This case illustrates that this vessel segment may be treated with balloon angioplasty.

Key Words • balloon angioplasty • intracranial atherosclerotic disease • interventional neurology • stroke • cerebrovascular disorder

Intracranial atherosclerotic disease of the ACA territory and especially of the A_2 segment is an infrequent source for recurrent cerebrovascular infarcts. When medical management fails, this segment of the intracranial vasculature may be treated with balloon angioplasty. Here, we report such a case.

Case Report

History and Examination. This 63-year-old right-handed African-American woman with hypertension, hyperlipidemia, and diabetes presented with intermittent episodes of dysarthria as well as proximal right upper- and lower-extremity numbness and weakness. The patient reported 10 such episodes on the day of admission, which were almost always associated with urination; each episode lasted ~ 2 minutes and completely resolved. The neurological examination revealed mild right arm and right leg weakness. Head CT scanning findings were unremarkable. An MR imaging study of the brain showed a few areas of restricted diffusion consistent with acute infarcts (Fig. 1 left) in the left centrum semiovale and frontal lobe. A CT perfusion study revealed no perfusion defect, 50% stenosis of the extracranial left ICA, mild to moderate diffuse multivessel intracranial atherosclerosis, and a focal severe stenosis of the proximal left A_2 segment. A 2D catheter angiogram showed 40–50% left ICA stenosis and 40–50% focal left A_2 segment stenosis. The remainder of the stroke workup revealed normal sinus rhythm and low-density lipoprotein of 180. A trans-thoracic echocardiogram showed normal findings. After a 2-day hospital stay, the patient was discharged home without residual deficits on neurological examination. She was placed on a regimen of aspirin/dipyridamole and atorvastatin. Five days later, she was readmitted with recurrent right arm and leg weakness and numbness with dysarthria that resolved spontaneously after 30 minutes. Additional MR imaging showed new areas of infarction in the left ACA territory (Fig. 1 right). A 3D cerebral angiogram with reconstruction now revealed 75% A_2 segment stenosis of the left ACA (Fig. 2A). Because the patient had recurrent symptoms attributable to the A_2 segment lesion and at this point medical management had failed, intervention was undertaken.

Operation. After placement of a 6 Fr guide catheter in the left ICA, a Gateway 1.5 × 15–mm balloon (Boston Scientific) failed to cross the A_1 segment. Subsequently,
a Sprinter 1.5 × 6-mm balloon (Medtronic) was successfully navigated across the left A2 stenosis (Fig. 2B) over a 0.014-in microguidewire and inflated to 6 atm for 30 seconds. A control angiogram revealed nonopacification of the left A2 segment. Therefore, verapamil 4.5 mg was injected intraarterially for suspected vasospasm. A Hype rglide 4 × 10-mm balloon (ev3, Inc.) was then navigated across the A2 segment, and serial overlapping inflations were performed, resulting in reestablished antegrade flow with < 25% residual stenosis (Fig. 2C).

Postoperative Course. The next day, the patient was discharged home on a regimen of clopidogrel; she was asymptomatic, and findings from the neurological examination were normal. A 2D cerebral angiogram obtained at 8 months showed a residual < 40% stenosis of the left ACA A2 segment. The patient remains asymptomatic 13 months postangioplasty.

Discussion

Intracranial atherosclerosis is common in the US, particularly in patients of African-American, Asian, or Hispanic background.8 There are several possible mechanisms of stroke due to ICAD including hypoperfusion, branch vessel local thrombosis, and distal artery-to-artery embolization, all of which appear to be interwoven.3

Aggressive medical management remains the first line treatment in most cases of ICAD. The Warfarin-Aspirin Symptomatic Intracranial Disease (WASID) trial demonstrated that warfarin is not superior to aspirin in preventing new strokes and showed an increased rate of hemorrhage with warfarin.5 Unfortunately, similar to our case, optimal medical management has been reported to fail in ~ 20% of patients with ICAD > 50%.

Surgical bypass has been used for symptomatic patients with intracranial ICA or MCA stenosis or occlusion and was proven ineffective.5 The next therapeutic option for patients in whom the medical treatment has failed is intracranial angioplasty with or without stent placement. In a retrospective study of 36 patients with symptomatic ICAD treated with angioplasty, the periprocedural death and stroke rate only was 8.3%, while the annual stroke rate in the angioplasty-related territory was 3.36%.7 In another series of 60 patients with symptomatic ICAD treated with angioplasty alone, stenting alone, or angioplasty followed by stenting, the periprocedural stroke and death rate was 4.8%, while the annual stroke rate was 1.8%.11 Restenosis occurs not uncommonly after intracranial angioplasty, with a frequency ranging between 24 and 50%.11

Intracranial atherosclerotic disease of the ACA has been infrequently reported. In the Northern Manhattan Stroke Study, ~ 20% of the cases of ICAD-related infarcts were attributed to ACA and posterior cerebral artery disease in the Hispanic population and 0% in African-American and Caucasian population.1 The natural course of the disease is not well defined, but some studies have suggested that intracranial ACA, MCA, and posterior cerebral artery lesions are more likely to progress than intracranial ICA lesions. The main challenges of performing interventions in this area are the small caliber of the ACA (ranging from 0.9 to 4.0 mm with an average of 2.6 mm)2 and the sharp angulation of the ACA origin, complicating navigation and increasing the risk of vessel rupture. A common indication for intervention in the ACA territory is vasospasm-related stenosis after subarachnoid hemorrhage. However, this intervention in the ACA territory is less successful (34% success rate) than in other major intracranial vessels (73–100% success rate).9 Angioplasty of the ACA for management of intracranial atherosclerosis has rarely been reported. In
their series, Marks et al.\textsuperscript{7} did not report on any patient with ACA lesions, whereas Wojak et al.\textsuperscript{12} reported 1 case with ACA stenosis that was complicated by wire perforation. In Japan, Touho\textsuperscript{10} reported 2 cases of A\textsubscript{2} stenosis in which percutaneous angioplasty was attempted but failed due to an inability to introduce the catheter into the stenotic segment. In the US, Janjua et al.\textsuperscript{6} reported the first successful case of A\textsubscript{2} disease managed with percutaneous angioplasty.

In our patient, the discrepancy of reporting results of the A\textsubscript{2} stenosis between the 2 angiograms obtained in the 1-week interval are explained by the optimized projection with the addition of 3D angiography on the second angiogram, resulting in a more accurate “least-diameter” projection. It is also possible that the patient’s stenosis may have been due to a thrombus or hemorrhage in the wall of the artery. However, the finding of persistent residual stenosis at the A\textsubscript{2} segment on 8-month follow-up angiogram is highly suggestive of local ICAD rather than an embolic or mural thrombus.

Conclusions
Intracranial atherosclerotic disease of the ACA territory and especially of the A\textsubscript{2} segment is uncommon, but it may be the source for recurrent cerebrovascular infarcts. This case illustrates that in the setting of failed medical therapy, this segment of the intracranial vasculature may be treated with balloon angioplasty.

Disclosure
Dr. Norbash owns stock in Boston Scientific.

References

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