

“Basilar Web” Causing Basilar Branch Infarction

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We present a young patient with no vascular risk factors with a basilar branch infarction secondary to a shelf-like filling defect of the basilar artery. This defect was present and unchanged on repeat imaging and determined to be most consistent with a basilar web. Similar to carotid webs, a basilar web is believed to be an area of focal intimal fibroplasia that increases the risk of brainstem infarction. Focal fibroplasia of the posterior circulation should be considered when evaluating young adults with posterior circulation strokes of otherwise undetermined cause.

Key Words: Basilar web—Stroke—Basilar artery—Pontine perforator—Carotid web—Stroke in the young

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Case Presentation

A 19-year-old woman presented to the emergency room with sudden onset horizontal diplopia, slurred speech, and gait instability. She was in otherwise good health with no cardiovascular risk factors. Her family and social history were unremarkable. Examination showed moderate guttural dysarthria and difficulty initiating mouth movements. Pupils were normal and visual fields were full. She had limited adduction with preserved abduction in both eyes and accompanied exotropia, greater on the right, consistent with bilateral internuclear ophthalmoplegia. There was bilateral appendicular and gait ataxia.

Basic laboratory testing was unremarkable. Serological tests for human immunodeficiency virus and syphilis were negative. Erythrocyte sedimentation rate, antinuclear antibodies, and antiphospholipid antibodies were either absent or within normal limits. Magnetic Resonance (MR) of the brain showed acute rostral paramedian

pontine infarction (Fig 1). MR angiography of the brain showed a linear filling defect at the top of the basilar, at the level of the infarction. There were no other accompanying vessel wall or luminal abnormalities (Fig 2). Trans-thoracic and trans-esophageal echocardiography, and telemetry were normal. Cerebrospinal fluid analysis, including testing for syphilis and varicella-zoster virus antibodies, were negative. She was given a diagnosis of basilar branch occlusion due to a “basilar web” and managed with aspirin. The filling defect seen on MR angiography was present and unchanged on repeat imaging at 3 days, 3 months, and 11 months after initial presentation. On follow-up at 6 months she was back to near baseline neurological function with only mild residual dysarthria.

The association between paramedian pontine infarction and focal basilar artery dysplasia was first reported by Green and Caplan in 2000.¹ That case was of an otherwise healthy young man who had recurrent pontine infarctions secondary to a shelf-like protruding lesion of the mid-basilar artery described as a “basilar shelf.” Importantly, the lesion was unchanged on repeat imaging 10 years apart. Similarly, our patient is young, has no vascular risk factors, and her shelf-like intraluminal lesion remained unchanged on repeat MR angiography 3 days, 3 months, and 11 months after initial presentation. Further, there was no evidence of intramural hematoma, fusiform dilatation, saccular aneurysm, pearl and string sign, or stenosis of the vessel to suggest a basilar dissection.²

Carotid web is increasingly recognized as an important cause of anterior circulation cerebral infarction in young

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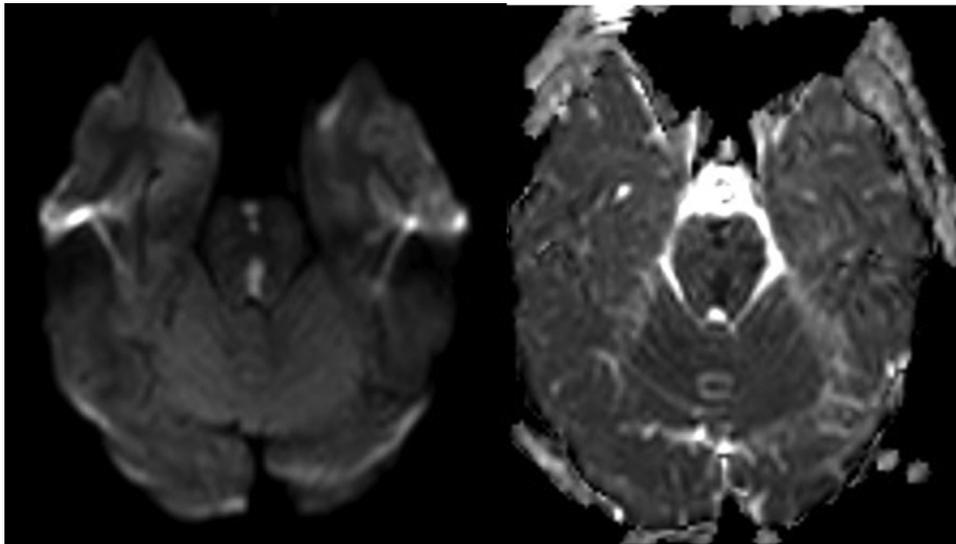


Figure 1. MR of the brain. Diffusion Weighted Image (DWI) and Apparent Diffusion Coefficient (ADC) with axial sections through the rostral pons showing a linear paramedian area of restricted diffusion, consistent with acute infarction.

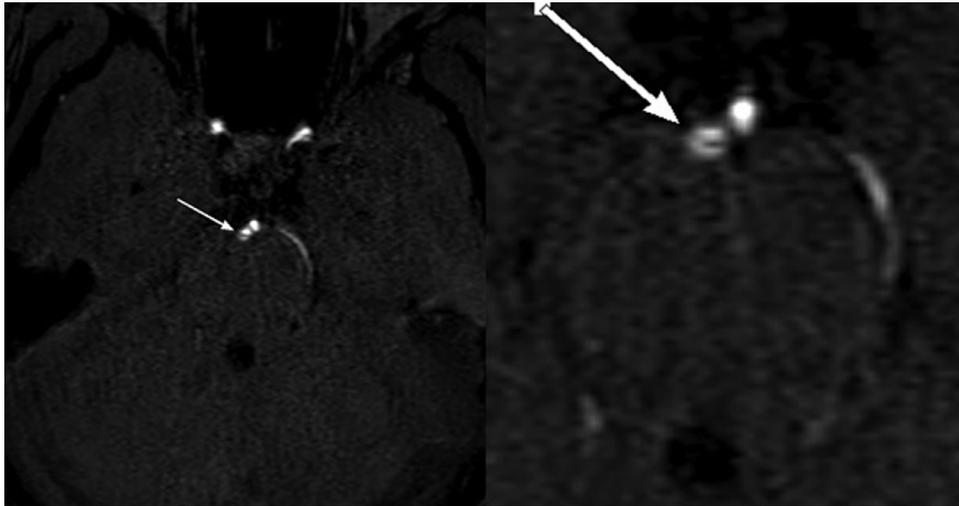


Figure 2. MR angiography at the level of the rostral basilar artery. Two separate axial sections of the source Time of Flight (TOF) MR angiography showing a linear filling defect at the top of the basilar artery that was identified as a continuous intraluminal defect and confirmed by repeat MR angiography at 3 days, 3 months, and 11 months poststroke. The origin of the left posterior cerebral artery is seen next to the basilar artery.

adults. Radiographically carotid webs are commonly described as shelf-like protrusions affecting the posterior aspect of the carotid bulb, while pathologically they are defined by focal intimal fibroplasia.³ We believe that our case and the one described previously are examples of intimal fibroplasia, or focal fibromuscular dysplasia, of the basilar artery. “Basilar web” should be considered when evaluating young adults with posterior circulation infarction of otherwise undetermined cause.

Declaration of Competing Interest

None for all authors.

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