Bilateral Medial Medullary Infarction in a Patient with Basilar Artery Fenestration – Cause or Coincidence?

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Introduction

Fenestration refers to localized duplication of a vessel. Basilar artery fenestration is a congenital variant with an incidence of 5%. Medial medullary infarction (MMI) is a rare stroke syndrome accounting for 0.5-1.5% of all strokes and bilateral involvement is uncommon. The most common presentation of bilateral MMI is the sudden onset of a variable constellation of findings which can include quadriparesis/quadriplegia, dysarthria, nystagmus, sensory disturbances, hypoglossal palsy and bulbar dysfunction.

Case presentation

A 56-year-old female with no significant cerebrovascular risk factors except for a remote history of breast cancer in remission, presented within 5 hours of sudden onset, whole body paresthesia sparing the face, oscillopsia, nausea and vomiting. Initially neurological examination was remarkable only for bilateral vertical nystagmus. MRI brain disclosed a V-shaped diffusion restriction in the medial medulla with ADC correlation. CT angiography of the head revealed proximal basilar artery fenestration at the level of medulla. The next day, patient developed asymmetric quadriparesis, dysarthria, hypophonia and dysphagia. Repeat MRI demonstrated evolution of medial medullary infarction with FLAIR hyperintensity. An extensive stroke work-up was unremarkable, except for a patent foramen ovale without evidence of venous thrombosis.

Discussion

Pontine and lateral medullary infarctions have been reported in patients with basilar artery fenestrations. However, to our knowledge, this is the first reported case of bilateral medullary infarction. Hemodynamic alterations and turbulence are thought to make fenestrated artery a more common site for thrombosis compared to a normal artery. It is also noted that partial endothelium-lined intraluminal septa appearing as spurs are seen in fenestrated arteries. These are hypothesized to cause turbulent blood flow leading to thrombosis and possible embolization from the fenestrated artery. Paradoxical embolic etiology, though a possibility in our case was thought to be unlikely in the absence of venous thrombosis or cortical infarction on DWI.

Conclusion

Our case, as well as several others, show association between basilar artery fenestration and strokes in the brainstem. Knowledge of this rare entity is important to consider when pursuing workup for medullary lacunar strokes.

References


