5-24-2021

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NRJ

The Neuroradiology Journal
0(0) 1–2

DOI: 10.1177/19714009211017785
journals.sagepub.com/home/neu
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Under pressure: pressure-dependent pontine compression by a dolichoectasia basilar artery, a case report

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Abstract

Basilar artery dolichoectasia can lead to ischemic stroke through thrombosis of small perforating vessels of the brainstem. Here we report the case of a patient with transient paramedian pontine syndrome in the setting of a hypertensive crisis, finding a dolichoectasia basilar artery compressing on the ventral surface of the pons. The outcome was near-complete resolution of deficits after blood pressure control. We propose increased basilar artery pulse pressure as a novel mechanism of transient compression of the brainstem by a dolichoectasia artery.

Keywords

Dolichoectasia, basilar artery, transient compression, pressure-dependent, pons

Introduction

Basilar artery dolichoectasia (BAD) is the most common type of intracranial arterial dolichoectasia. Most patients with BAD (75%) go on to develop at least one event of ischemic stroke, hemorrhage, brainstem compression, hydrocephalus,[1,2] or arterial dissection,[3] all of which are more or less static and have not been described to fluctuate. Here we present the first case reported in the literature of fluctuating neurological deficits related to BAD in the setting of a hypertensive crisis.

Case

Here we present the case of a 79-year-old woman with a medical history of left posterior cerebral artery infarct five years before her presentation with residual right-sided homonymous hemianopia. Upon presentation to the emergency department, approximately seven hours after a sudden onset of right-sided weakness and a left facial weakness, acute ischemic stroke was first suspected. Notably, her blood pressure on presentation was 235/112, requiring nicardipine infusion for adequate control.

By the time she presented to the emergency department her right-sided weakness had resolved, but she had severe weakness on the left side of her face, particularly prominent in the inferior two-thirds, and left conjugate gaze palsy. CT angiography of the head and neck showed a dilated and tortuous basilar artery—the presence of a BAD. She was admitted to the acute stroke unit for blood pressure control and for identification of a presumed lesion in the left paramedian pons. Follow-up imaging showed no acute infarct, and the pons was compressed by the presence of the BAD (Figure 1).

After her blood pressure was reduced by about 25% (with a subsequent slower decrease), her left conjugate gaze palsy completely resolved, and her left facial palsy improved but did not completely resolve.

Discussion

Recently it has been described a so-called vertebral artery compression syndrome,[4] which comes from direct compression of the brainstem structures, particularly the medulla or the pons,[2] but is also described to compress cranial nerves[5] or even to lead to hydrocephalus,[6] although this presentation of transient symptoms related to hemodynamic stress has not been previously described.

There are several pathophysiological elements that take place with BAD, beginning with intermittent blood flow reversal (two-way flow) that leads to reduced blood flow on a dilated arterial system, making it ripe for in situ thrombosis to occur. To this, we add the fact

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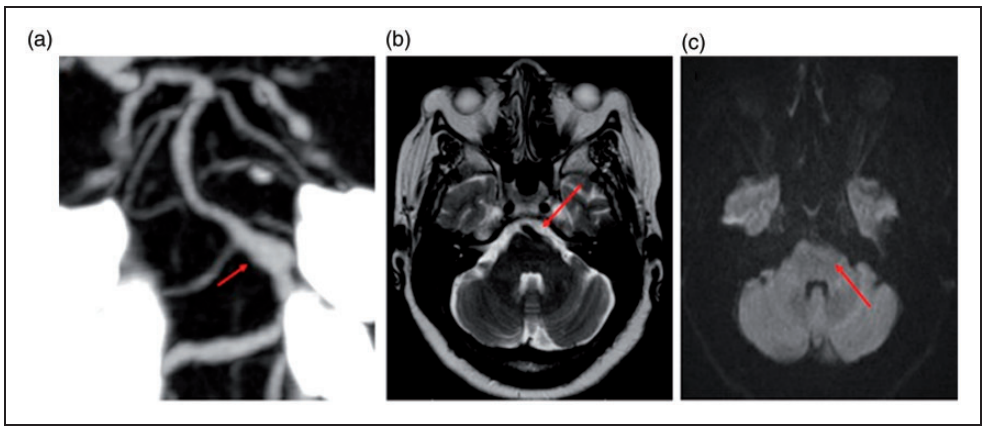

Figure 1. CT angiogram that shows dilated basilar artery at the level of the lower pons measuring 9x6.3 mm with the most prominent dilation overlying the left lower pons (a). MRI brain T2-sequence showing a flow void that corresponds to the basilar artery (white arrow) deforming the ventral surface of the pons (b), and on the diffusion-weighted images (DWI) we can see that there is no evidence of acute infarct (c).

that dolichoectasia mechanically pulls and twists the small perforating arteries stemming from the vertebral and basilar arteries.[7,8]

We hypothesize that in our case the mechanism leading to this phenomenon was increased pulse pressure, which caused compression of structures surrounding the artery facilitated by the muscular layer and adventitia thinning known to occur in BAD.[9,10] This type of mechanical compression to the brainstem and surrounding structures is facilitated by chronic stiffening of arterial walls that takes place normally with age and is enhanced by arterial hypertension.[10] This is different from the mechanisms described above in clinic-pathological studies of ischemic infarcts due to BAD, where the pathophysiological mechanism is usually thrombosis of brainstem perforating arteries.[9]

Conflict of interest

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

Informed consent

The investigators obtained written consent from the patient prior to the submission of this work.

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References

1. Gutierrez J, Bagci A, Gardener H, et al. Dolichoectasia diagnostic methods in a multi-ethnic, stroke-free cohort: results from the northern Manhattan study. *J Neuroimaging* 2014; 24: 226–31.
2. Passero SG and Rossi S. Natural history of vertebrobasilar dolichoectasia. *Neurology* 2008; 70: 66–72.
3. Lv X, Yu J, Zhang W, et al. Acute hemorrhagic cerebral artery dissection: Characteristics and endovascular treatment. *Neuroradiol J* 2020; 33: 112–117.
4. Li Q, Xie P, Yang WS, et al. Vertebral artery compression syndrome. *Front neurol* 2019; 10: 1075.
5. Yang XS, Li ST, Zhong J, et al. Microvascular decompression on patients with trigeminal neuralgia caused by ectatic vertebrobasilar artery complex: technique notes. *Acta neurochir* 2012; 154: 793–7; discussion 7.
6. Siddiqui A, Chew NS and Miszkiel K. Vertebrobasilar dolichoectasia: a rare cause of obstructive hydrocephalus: case report. *Brit J Radiol* 2008; 81: e123-6.
7. Kumral E, Kisabay A, Atac C, et al. The mechanism of ischemic stroke in patients with dolichoectatic basilar artery. *Eur J Neurol* 2005; 12: 437–44.
8. Passero S and Filosomi G. Posterior circulation infarcts in patients with vertebrobasilar dolichoectasia. *Stroke*. 1998; 29: 653–9.
9. Pico F, Labreuche J and Amarenco P. Pathophysiology, presentation, prognosis, and management of intracranial arterial dolichoectasia. *Lancet Neurol* 2015; 14: 833–45.
10. Pico F, Labreuche J, Seilhean D, et al. Association of small-vessel disease with dilatative arteriopathy of the brain: neuropathologic evidence. *Stroke*. 2007; 38: 1197–202.
11. Mokhber N, Shariatzah A, Avan A, et al. Cerebral blood flow changes during aging process and in cognitive disorders: A review. *Neuroradiol J* 2021; 19714009211002778 ahead of print.