

## CASE REPORT

## Anteromedial thalamic infarct: a rare presentation

Syed H Shabbir,<sup>1</sup> Faryal Nadeem,<sup>2</sup> Daniel Labovitz<sup>1</sup><sup>1</sup>Department of Neurology, Montefiore Hospital and Medical Center, Bronx, New York, USA<sup>2</sup>Department of Medicine, Capital Health Regional Medical Center, Trenton, New Jersey, USA**Correspondence to**

Dr Syed H Shabbir, syedhshabbir@gmail.com

Accepted 17 April 2018

**SUMMARY**

We report a rare presentation of an anteromedial thalamic infarct in a 50-year-old woman with acute onset left eye ptosis, vertical gaze paresis and confusion. MRI identified an acute left anteromedial thalamic infarct with a severe left P1 stenosis. Thalamic infarcts are associated with marked neurobehavioural disturbances with dominant thalamic lesions causing language deficits, verbal perseveration, memory disturbances, abulia and disorientation. Ocular movement deficits can also be present and typically accompany paramedian lesions. Rarely, patients can develop an ipsilateral ptosis. We discuss these symptoms and review the literature.

**BACKGROUND**

The clinical presentation is vital in the evaluation, diagnosis and management of neurological disease. An understanding of brain anatomy, disease and associated features can help guide whether additional workup needs to be done. We report a rare constellation of symptoms resulting from an anteromedial thalamic infarct.

**CASE PRESENTATION**

A 50-year-old woman presented with acute onset left eye ptosis, diplopia and confusion. Her medical history was notable only for hypertension for which she was taking low dose amlodipine. On examination, she had a moderate left eye ptosis and impaired vertical movement of the left eye associated with diplopia. There was no diurnal fluctuation or fatigability, anisocoria or anhidrosis. She had minimal word finding difficulty with rare paraphasic errors.

Family reported that she had woken up with these complaints. Initially she was confused, agitated and screaming. She was asking nonsensical questions and speaking of characters in a book as if they were real people who might come visit. The family noticed the ptosis and her speech and brought her into the emergency department for an evaluation. She had no weakness, pain, numbness, paresthesias, ataxia, hyporeflexia or gait instability.

**INVESTIGATIONS**

The patient had stable vital signs, and routine blood work was unremarkable. MRI revealed an acute left anteromedial thalamic infarct with a severe left P1 stenosis (figures 1 and 2). There were no other areas of acute infarction.

**TREATMENT**

She underwent a comprehensive stroke workup, had no other vascular risk factors, patent foramen ovale (PFO) or other embolic source. She was treated with aspirin, statin and optimisation of her antihypertensive regimen.

**OUTCOME AND FOLLOW-UP**

Her left eye ptosis and vertical gaze paresis persisted at discharge. On follow-up, she reported often having trouble shifting her gaze when looking up and down from notes to a computer screen at work; however, this resolved within a couple months after discharge. Repeat imaging showed recanalisation of the left posterior cerebral artery (PCA).

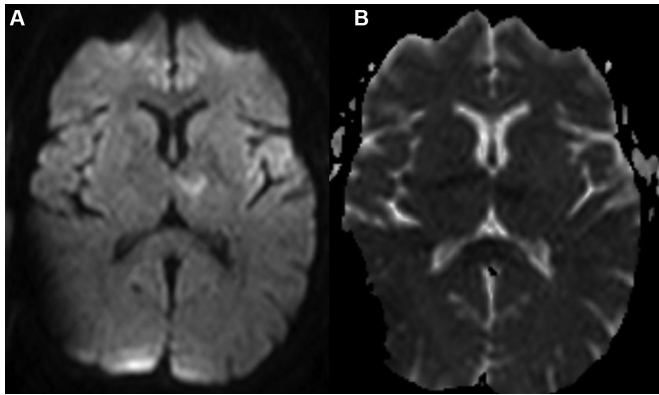
**DISCUSSION**

Studies of thalamic vascular supply and associated syndromes identify four major vascular territories. The tuberothalamic artery (also known as the polar artery) originates from the middle third of the posterior communicating artery and primarily supplies the anteromedial thalamus, though there are notable interpersonal variations and as many as one-third of the normal population derives supply from the paramedian artery.<sup>1</sup> The associated clinical syndrome is characterised by marked neurobehavioural disturbances with dominant thalamic lesions causing language deficits, verbal perseveration, memory disturbances, abulia and disorientation.<sup>2,3</sup>

Rarely, anteromedial thalamic lesions can also cause an ipsilateral ptosis. A search of the English language literature found only a few case reports with unilateral or bilateral ptosis.<sup>4-7</sup> Another recent case report of a 74-year-old woman who presented with a left eye ptosis and found to have a left anteromedial thalamic infarct was attributed by the authors to a Horner's syndrome from extension of the lesion to the hypothalamus. However, the patient was without any associated signs or symptoms of Horner's, and the isolated ptosis may have stemmed from the thalamic infarction.<sup>8</sup> This finding is attributed to a disruption of the supranuclear motor circuit to the levator palpebrae. A 'cerebral ptosis' in the setting of a hemispheric insult is well established and ascribed to a cortical region that controls eyelid elevation. It is theorised that any interruption of this supranuclear pathway can cause an ipsilateral ptosis. Ptosis resulting from an anteromedial thalamic lesion possibly involves the topographic correlate to the part of the precentral cortex responsible for eyelid elevation.<sup>5,9</sup>



**To cite:** Shabbir SH, Nadeem F, Labovitz D. *BMJ Case Rep* Published Online First: [please include Day Month Year]. doi:10.1136/bcr-2017-223404



**Figure 1** (A) Diffusion-weighted MRI showing acute left anteromedial thalamic infarct. (B) Apparent diffusion coefficient sequence with corresponding area of low intensity consistent with an acute infarct.

Abnormalities of ocular movements resulting from thalamic infarcts typically involve paramedian lesions. In a case series of 18 patients with thalamic infarcts, the highest frequency of horizontal and vertical gaze disturbances, including skew deviation, was associated with paramedian lesions. In this series, 10 patients had either an anterior or medial lesion of which only three had a vertical gaze palsy.<sup>10</sup> Another review of patients with isolated thalamic infarcts identified nine patients with involvement of the anteromedial territory. Among those who had a dominant thalamic lesion, 75% were described to have a vertical eye paresis.<sup>2</sup> The mechanism underlying this finding

is not completely understood though possibly attributable to frontocortical fibres decussating in the medial thalamus.<sup>11 12</sup>

To our knowledge, this is the first report of a patient presenting with this constellation of symptoms resulting from an anteromedial thalamic infarct.

**Learning points**

- ▶ The anteromedial thalamic syndrome is characterised by marked neurobehavioural disturbances with dominant thalamic lesions causing language deficits, verbal perseveration, memory disturbances, abulia and disorientation.
- ▶ Rarely these lesions can cause ptosis and/or vertical gaze paresis, for which the pathophysiology is not completely understood

**Contributors** SHS: design, draft and revision. FN: draft and critical revision of content. DL: critical revision of content.

**Funding** The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

**Competing interests** None declared.

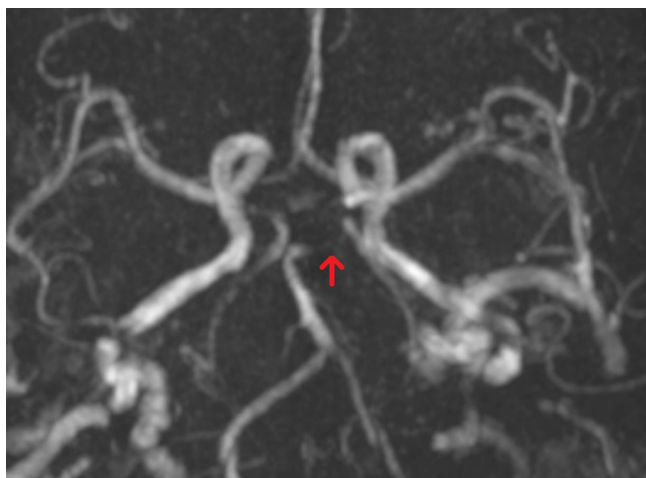
**Patient consent** Obtained.

**Provenance and peer review** Not commissioned; externally peer reviewed.

© BMJ Publishing Group Ltd (unless otherwise stated in the text of the article) 2018. All rights reserved. No commercial use is permitted unless otherwise expressly granted.

**REFERENCES**

- 1 Schmahmann JD. Vascular syndromes of the thalamus. *Stroke* 2003;34:2264–78.
- 2 Carrera E, Michel P, Bogousslavsky J. Anteromedian, central, and posterolateral infarcts of the thalamus: three variant types. *Stroke* 2004;35:2826–31.
- 3 Graff-Radford NR, Damasio H, Yamada T, et al. Nonhaemorrhagic thalamic infarction. Clinical, neuropsychological and electrophysiological findings in four anatomical groups defined by computerized tomography. *Brain* 1985;108 (Pt 2):485–516.
- 4 Ebert AD, Vinz B, Görtler M, et al. Is there a syndrome of tuberothalamic artery infarction? A case report and critical review. *J Clin Exp Neuropsychol* 1999;21:397–411.
- 5 Kausar H, Antonios N. Combined thalamic ptosis and astasia. *J Clin Neurosci* 2013;20:1471–4.
- 6 Kim EJ, Lee DK, Kang DH, et al. Ipsilateral ptosis associated with anterior thalamic infarction. *Cerebrovasc Dis* 2005;20:410–1.
- 7 Lampl Y, Gilad R. Bilateral ptosis and changes in state of alertness in thalamic infarction. *Clin Neurol Neurosurg* 1999;101:49–52.
- 8 Azabou E, Drexel L, Honnorat J, et al. Ipsilateral ptosis as main feature of tuberothalamic artery infarction. *Neurol Sci* 2009;30:69–70.
- 9 Schmidtke K, Büttner-Ennever JA. Nervous control of eyelid function. A review of clinical, experimental and pathological data. *Brain* 1992;115(Pt 1):227–47.
- 10 Steinke W, Sacco RL, Mohr JP, et al. Thalamic stroke. Presentation and prognosis of infarcts and hemorrhages. *Arch Neurol* 1992;49:703–10.
- 11 Clark JM, Albers GW. Vertical gaze palsies from medial thalamic infarctions without midbrain involvement. *Stroke* 1995;26:1467–70.
- 12 Weidauer S, Nichtweiss M, Zanella FE, et al. Assessment of paramedian thalamic infarcts: MR imaging, clinical features and prognosis. *Eur Radiol* 2004;14:1615.



**Figure 2** Loss of flow-related signal involving the P1 segment of the left posterior cerebral artery (PCA) (arrowhead). The right PCA has a fetal origin and demonstrates normal flow-related signal.

Copyright 2018 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.  
 BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact [consortiasales@bmjgroup.com](mailto:consortiasales@bmjgroup.com)

Visit [casereports.bmj.com](http://casereports.bmj.com) for more articles like this and to become a Fellow