

CASE REPORT

Three cases of Spontaneous Vertebral Artery Dissection (SVAD), resulting in two cases of Wallenberg syndrome and one case of Foville syndrome in young, healthy men

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SUMMARY

First patient, presented with sudden onset of headache, left hypoacusia and right hemiparesis, posteriorly developing gaze-evoked nystagmus and worsening right-sided weakness. Diagnosis of vertebral artery dissection and Foville Syndrome were made through clinical assessment and CT-carotid angiogram-MR angiography. Second patient, presented with four episodes of pain over left side of the nose and left eye pain over 1 month; admitted for acute facial pain without limb weakness. During admission, tingling over V1/V2 facial territory, vertigo, hypotension, uvula deviation and right lower limb numbness. CT-carotid angiogram confirmed vertebral artery with dissection. MRI revealed left lateral medullary infarct. Third patient, presented with sudden onset of left facial numbness and right upper limb weakness; 1 day after, right arm and leg hypoesthesia with hoarseness. MRA revealed dissection of left distal vertebral artery and MRI showed infarction in lower medulla oblongata.

BACKGROUND

These three cases are important because of several reasons:

- ▶ They demonstrate the on-going clinical pattern of vertebral artery (VA) dissection.

- ▶ They demonstrate the lack of specificity of plain CT scans for diagnosis while reminding us of the importance of a well done clinical history and examination.
- ▶ They demonstrate the variety of manifestations of the same pathology.

CASE PRESENTATION

Patient 1: A 39-year-old man, with no medical history, presents with sudden onset of headache, left hypoacusia, dysarthria, right-gaze deviation and right upper and lower limb weakness. Head CT on admission failed to show ischaemia, but rather a dominant left VA (LVA) that appeared to be hyperdense, indicative of thrombosis. CT-carotid angiogram (CTCA) revealed a very narrow basilar artery and distal (LVA) with surrounding thrombosis that corresponds to the dominant VA seen on plain CT scan (figure 1). Four days after onset, he develops posterior cervical neck pain, gaze-evoked lateral nystagmus and worsening hemiparesis. MR angiography (MRA) was performed, showing focal dissections involving the proximal V4 segment of the LVA and a low-volume well-established acute infarct in the median pons, clinically corresponding to an Inferior Medial Pontine Syndrome (IMPS) or Foville syndrome (figure 2).

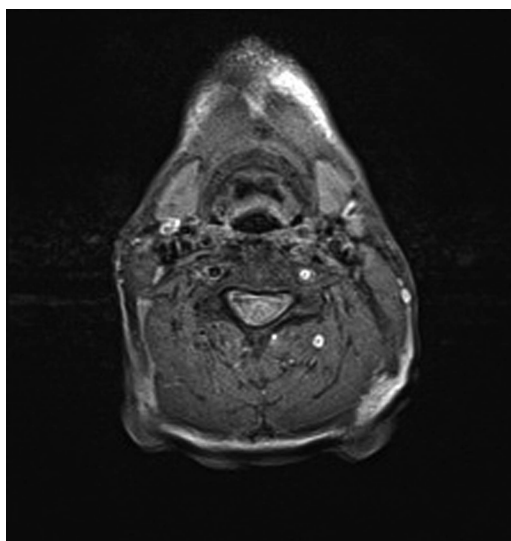


Figure 1 MRI T1 axial: thrombus around the left vertebral artery.

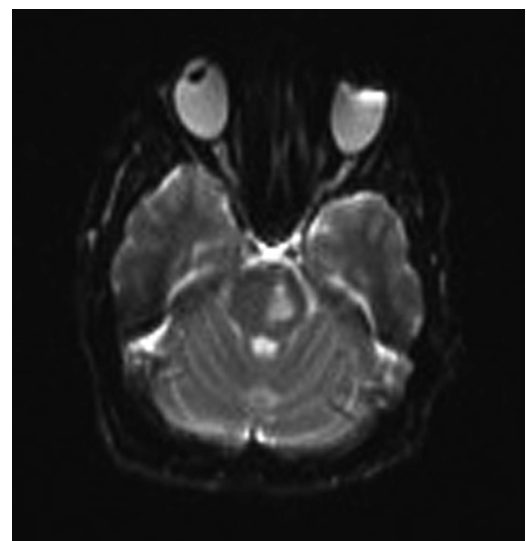


Figure 2 Diffusion-weighted MRI: left medial pons infarct.



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Patient 2: A 60-year-old man, with no medical history, presents over a period of 1 month, four episodes of pain over the left side of the nose (V2 region), left eye pain and dizziness, each lasting approximately 5 min. No limb weakness or neck pain was noted. On admission, he had severe left-sided nose pain and dizziness. Head CT scan on admission failed to reveal ischaemic infarct. Three days after admission, the patient developed left side facial numbness with ‘tingling’ over V1/V2 territory, vertigo, hypotension, deviation of the uvula and with right lower limb numbness. CTCA confirmed occlusion of the V3 and V4 segments of the LVA consistent with dissection (figure 3). Subsequent MRI revealed left lateral medullary infarct (figure 4A) and left cerebellar infarct (figure 4B).

Patient 3: A 40-year-old, previously healthy man, presents with sudden onset of left facial numbness, dysarthria and right upper limb weakness and numbness. Head CT on admission failed to show ischaemia. One day after admission he presents with right hemibody hypoesthesia, increasing ‘tingling’ in the left side of the face and hoarseness. MRI revealed area of patchy restricted diffusion in the left lower medulla oblongata (figure 5). MRA revealed a dominant right VA and dissection of the left distal VA without changes in the basilar artery (figure 6).

INVESTIGATIONS

CT, CT-CA, MRI, MRA

DIFFERENTIAL DIAGNOSIS

- ▶ I M P S (Foville syndrome).
- ▶ Lateral medullary syndrome (Wallenberg syndrome).
- ▶ VA dissection and thrombosis of posterior circulation.

TREATMENT

Undoubtedly, anticoagulation treatment for VA dissections at the distal or V4 level is controversial. Such treatment carries a relative increased risk of side effects if the dissection extends into the V4 region (risk of subarachnoid haemorrhage). Nevertheless, anticoagulation may be appropriate if there is rapid blood flow through severely narrowed vessels on transcranial Doppler despite the use of aspirin, completely occluded

vessel, recurrent stroke-like episodes or if there is free-floating blood clot visible on scans.

Treatment for VA dissection cannot be standardised. It requires a patient-based approach. Specific treatments must be planned in these cases.

OUTCOME AND FOLLOW-UP

Good outcome in all three cases. Good response to inpatient rehabilitation and no further thromboembolic events after anti-aggregation and anticoagulation schemes.

DISCUSSION

VA dissection has become increasingly recognised as an important cause of stroke, especially in young and middle age patients.¹ The most important clinical symptoms of a spontaneous VA dissection are unilateral headache and neck pain in 55–

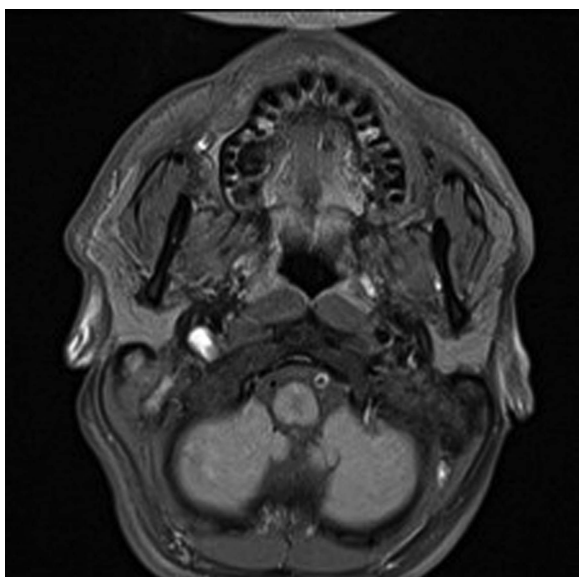


Figure 3 MRI T1 axial: thrombus around the left vertebral artery.

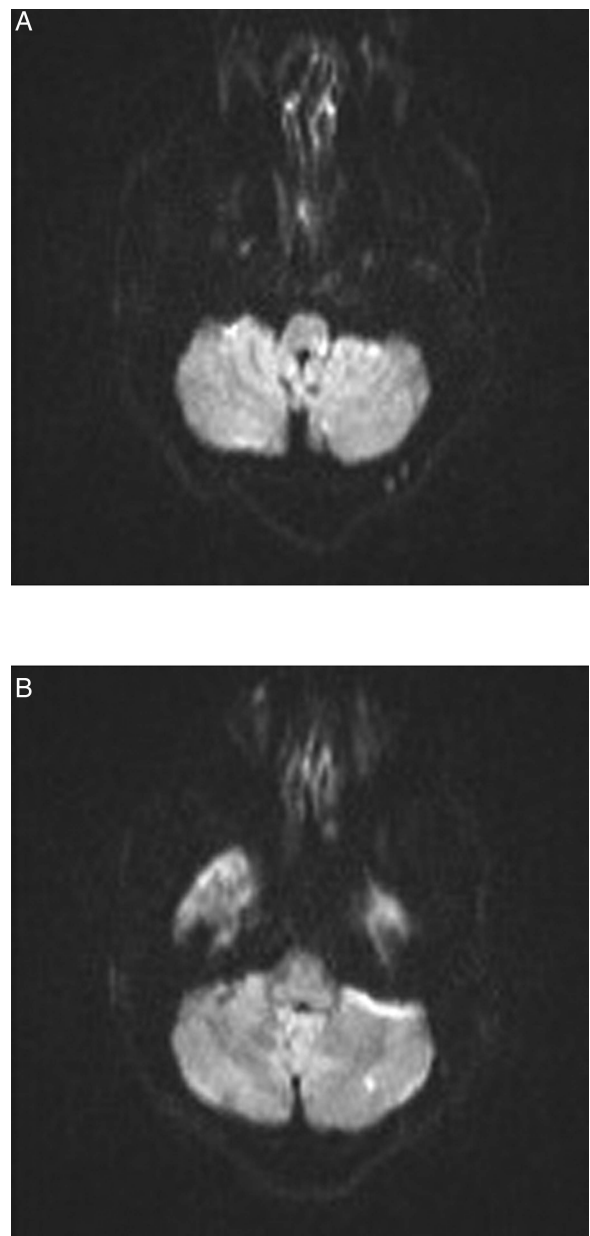


Figure 4 (A) Diffusion-weighted MRI: discrete left medulla oblongata infarct. (B) Diffusion-weighted MRI : infarction in left cerebellar hemisphere.

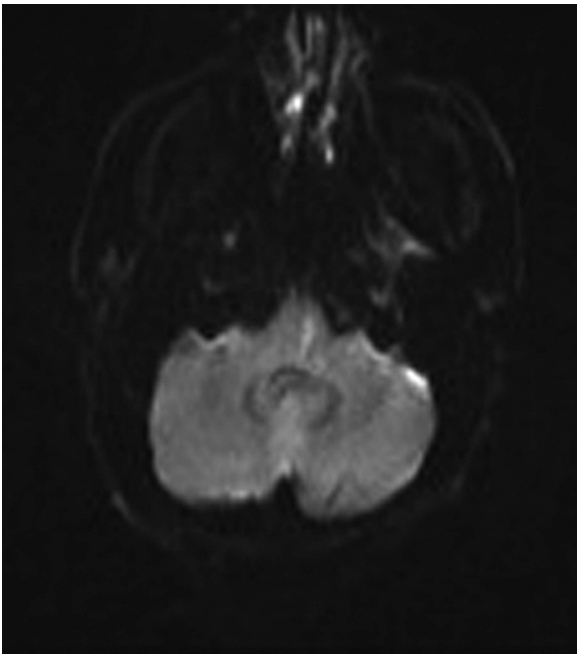


Figure 5 Diffusion-weighted MRI : infarction in left lower medulla oblongata.



Figure 6 MR angiography (postcontrast coronal image): dissected and narrow left vertebral artery. Note: patent and well-perfused basilar artery, possibly indicating an arterial–arterial embolus formation travelling from the distal left vertebral artery towards the left lower medulla oblongata.

60%.² In 23–43% the symptoms are identical to those of Wallenberg syndrome.^{3 4} MRI detects the intraluminal thrombus and intimal flap that are characteristic to VA dissection and in some cases, a hyperintensity of the vessel wall seen on T1-weighted axial images is considered as pathognomonic.^{5 6}

Learning points

- ▶ Vertebral artery dissection (VAD) can be a clinically on-going process, with different clinical features appearing at different stages.
- ▶ Contrary to what is usually believed, neck pain is not always present in VAD.
- ▶ At plain CT scan it is usually not enough to diagnose brainstem infarcts and VAD, clinical suspicion is paramount. More so, in special cases CT-carotid angiogram, at certain stages of the disease, fails to show dissection (MR angiography) might be needed.
- ▶ If the patient presents with changing neurological symptoms, it is possible that dissection of the VA is on-going.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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