Answer

Michael Wansbrough, MD, MSc(Epi), DTM&H, DLSHTM;* John Rizos, MD†

The answer is B: MRI to image the cervical cord. The patient was observed in the emergency department (ED) overnight, while awaiting magnetic resonance imaging (MRI) the next day. By morning his neurologic exam had improved considerably. He had some mild residual weakness in his biceps (4/5 bilaterally) but otherwise normal strength, sensation and reflexes. He developed coffee-ground emesis, which was treated with intravenous fluids and pantoloc prior to transfer to the MRI suite. The MRI showed increased cord signal at C3–C4 (Fig. 1). This was felt to be consistent with cord ischemia, transverse myelitis or a demyelinating condition.

At this point urgent neurology and gastroenterology consultations were requested. The patient went on to have a magnetic resonance angiogram (MRA) that showed lack of flow in the right vertebral artery (Fig. 2) and mural thrombus in the vertebral artery wall (Fig. 3), consistent with a vertebral artery dissection. Urgent gastroscopy revealed distal esophagitis with a linear ulceration that was not felt to contraindicate anticoagulation. Since imaging revealed retrograde flow into the stenotic vertebral artery, the neurologist elected to treat with antiplatelets agents (ASA and clopidogrel) as opposed to the more commonly recommended heparin and warfarin regimen.

The diagnosis of vertebral artery dissection (VAD) can be challenging, and delay is common. Classic presentation involves the onset of a sudden, severe posterior head or neck pain, suggestive of a subarachnoid hemorrhage. This is usually followed by symptoms and signs of posterior circulation ischemia that can be delayed hours to weeks after the initial pain of the dissection, making the diagnosis difficult.[1] Headache or neck pain may be relatively mild, gradual in onset and similar to previous headaches the patient has suffered, including migraines. The diagnostic difficulty is compounded by reports that up to 80% of "spontaneous" vertebral artery dissection are preceded by minor neck trauma involving hyperextension or rotation of the neck.[2] This makes it easy to ascribe the patient's symptoms to a muscular strain. These events include sneezing, coughing, vomiting, yoga, chiropractic manipulation, overhead painting, wrestling, minor falls and minor motor vehicle accidents.[1,2]

The ischemic manifestations are related to decreased flow in a stenotic vessel or secondary to embolic events emanating from the mural thrombus. Imaging and transcranial Doppler studies provide evidence that the latter is most responsible, rationalizing anticoagulation as the standard treatment of cervical artery dissection.[1] Lateral medullary syndrome is the most common stroke syndrome associated with vertebral artery dissection, but the cerebellum or occipital cerebrum may be affected. Isolated spinal cord ischemia, as seen in our patient, is now more frequently recognized than in the past. VAD and carotid artery dissection (CAD) are more common in younger age groups, with a peak incidence in the fifth decade of life.[1]

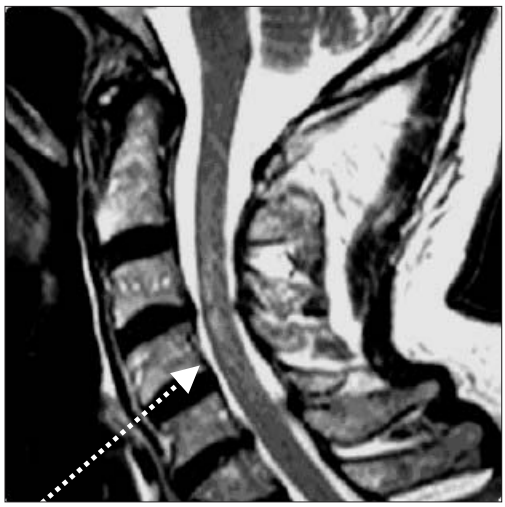

Fig. 1. MRI, showing increased cord signal (the "whiter zone") at C3-C4 (arrow) and above.

*Assistant Professor, Department of Family and Community Medicine, University of Toronto, Toronto, Ont.; Staff physician, Department of Emergency Medicine, Credit Valley Hospital, Mississauga, Ont., and Mount Sinai Hospital, Toronto, Ont.; Mis. Director, Canadian Network for International Surgery, Vancouver, BC
†Staff physician, Department of Emergency Medicine, Credit Valley Hospital, Mississauga, Ont.

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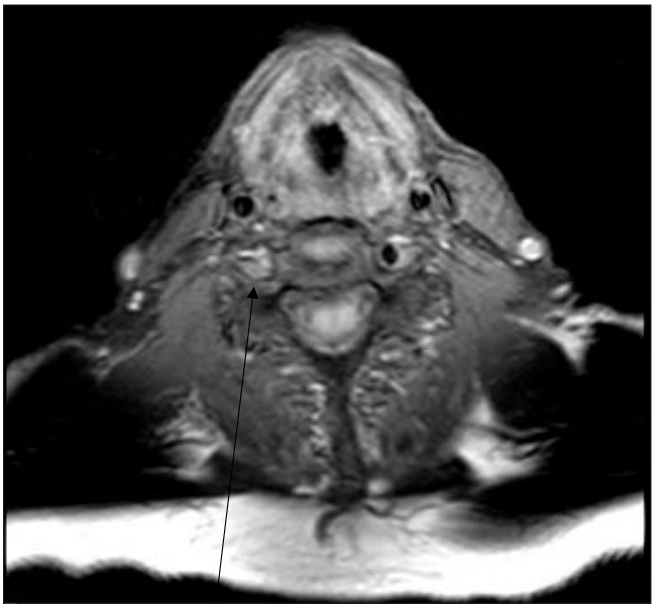

Fig. 2. MRA shows lack of flow in the right vertebral artery.

The traditional method of diagnosing VAD or CAD has been conventional angiography. This has now been generally replaced with MR angiography. MRA is less invasive, and its axial images can demonstrate mural thrombus that establishes the diagnosis.[1] Doppler ultrasound may show non-specific abnormal flow patterns but usually require conventional or MR angiography to confirm the diagnosis.

Heparin followed by warfarin for 3 to 6 months is the standard treatment for vertebral or carotid artery dissection, unless there are contraindications like intracranial extension of the dissection.[1] The presence or absence of residual vessel abnormalities at 3 to 6 months will guide therapy beyond this juncture and may include the continuation of anticoagulation, the replacement of warfarin with an antiplatelet agent, or the cessation of medical treatment altogether.[1] Although randomized trials evaluating the benefit of anticoagulation or anti-platelet agents have not been reported, it is believed that the natural history of this condition is improved with such therapy. Only rarely, when there is progressive ischemia despite full anticoagulation, are surgical or endoscopic treatments undertaken.

The prognosis for VAD is good in the approximately 90% who do not succumb to the initial stroke.[2] In the remaining patients there is usually a complete neurological recovery. The age of the patient, co-existing disease, and the presence of collateral circulation affect prognosis. The headache associated with VAD resolves in about 90% of patients but may continue for years.[1] Our patient's complete recovery was in part due to the retrograde flow of blood into the affected vessel.

Fig. 3. The MRA also showed mural thrombus in the vertebral artery wall.

Vertebral artery dissection is often misdiagnosed as musculoskeletal pain or migraine. Even when a more serious diagnosis like subarachnoid hemorrhage is considered, the traditional methods to diagnose this condition (CT and/or lumbar puncture) will often be negative, and the diagnosis depends on a high index of suspicion. VAD should be considered in younger patients presenting with stroke-like syndromes — particularly when accompanied or preceded by headache or neck pain. The identification of a preceding rotational or hyperextension injury, however trivial, increases the likelihood of this rare but dangerous condition.

Competing interests: None declared.

References

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Key words: headache; cervical spinal cord ischemia; neck pain; vertebral artery dissection; vascular injury

Correspondence to: mwansbrough@sympatico.ca; fax 416 203-2502