Hong Kong College of Physicians
Case report for Interim Assessment
Specialty Board of Advanced Internal Medicine (AIM)

For AIM Training, case reports should be submitted in the prescribed format together with the application form for Interim Assessment at least EIGHT Weeks before the date of Interim Assessment

Name of candidate (print and sign):
Hospital and Unit: Specialty:
Name of supervisor (print and sign):
Date(s) and place (hospital) of patient encounter: October 2020
Date of report submission: March 2021

Case report

Note: Failure to follow the prescribed format (including the number of words) results in a FAILURE mark (score between 0 and 4) for the Case Report.

Title: A patient with vertebral artery dissection

Case history:

A 61-year-old gentleman presented to the hospital for dizziness and upper limb numbness in October 2020. He reported vertiginous dizziness for 2 weeks with unsteady gait and tendency of leaning to the left side. Left upper limb numbness began on the day of presentation. There was no limb weakness. There was no history of head trauma, fall or neck manipulation. He denied headache or neck pain. The patient enjoyed good past health. He was a chronic smoker, and the owner of an advertisement company. He denied using excessive alcohol or illicit drugs.

On examination, vital signs were within normal limits. The level of consciousness was normal. Neurological examination revealed impaired right saccade eye movement, impaired left eye abduction, and nystagmus towards left lateral gaze. He had left facial palsy, left past-pointing, impaired right sided pain and left sided touch sensation. Limb power and reflexes were normal. Cardiovascular and respiratory examinations were unremarkable. The National Institutes of Health Stroke Scale (NIHSS) score was 4/42.

Initial laboratory results including complete blood count, clotting profile, renal and liver function were unremarkable. Computed tomography (CT) scan of the brain on admission showed no intracranial hemorrhage. He was managed as acute ischemic stroke with dual antiplatelet therapy including aspirin and clopidogrel. Ultrasound doppler of extracranial neck vessels showed absent left vertebral artery flow. CT angiogram of brain and neck (Figure 1) was performed, showing almost total occlusion of the V4 part of left vertebral artery, shortly after the origin of left posterior inferior cerebellar artery to the terminal left vertebral artery, and the terminal left vertebral artery was faintly opacified. Left posterior inferior cerebellar artery, left anterior inferior cerebellar artery and basilar artery were patent. The diagnosis of left vertebral artery dissection was established.

The patient was continued on dual antiplatelet therapy for 3 weeks in total, followed by aspirin alone. Cardiovascular risk factors were screened, and he was started on rosuvastatin for hyperlipidemia. Intensive physiotherapy was performed. He was discharged at 2 months after presentation, requiring walking frame for mobility and Ryle's tube feeding due to severe dysphagia. Follow-up magnetic resonance (MR) angiography 4 months after presentation showed recanalization of the left vertebral artery. Magnetic resonance imaging (MRI) of the brain showed chronic infarction in left lateral medulla. (Figure 2) He continued to receive rehabilitation service in day hospital and was able to walk unaided with supervision 4 months after presentation. He resumed oral diet after a videofluoroscopic swallowing study (VFSS). He still suffered from dizziness but it was in improving trend.

Discussion and literature review

Vertebral artery dissection is an important cause of stroke in young patients but may also happen at any age group. The mean age of presentation is around 45 years. The annual incidence rate is 0.97 per 100,000 population. It is less common when compared to internal carotid artery dissection, another form of cervical artery dissection, which annual incidence rate is 1.72 per 100,000 population.[1] The incidence rate is likely underestimated, as asymptomatic vertebral artery dissections are usually undiagnosed.

Causes of vertebral artery dissection can be traumatic or spontaneous. Prior cervical trauma could be identified in 40% of patients with cervical artery dissections. Cervical manipulation therapy and extreme head movements are the more frequently reported types of cervical trauma.[2] Spontaneous arterial dissection may be related to underlying arteriopathy such as fibromuscular dysplasia, or other connective tissue diseases such as Ehlers-Danlos syndrome type IV and Marfan's syndrome. Vascular risk factors, including hypertension, diabetes mellitus, hyperlipidemia and the use of oral contraceptive pills, are also associated with arterial dissection.[3] Spontaneous vertebral artery dissection most often occurs in the V2 (pars transversaria) or V3 segment (atlas loop). V4 segment (intracranial) dissection only occurs in 11% of patients.[4]

Patients with vertebral artery dissection usually present with ischemic stroke, transient ischemic attack (TIA), occipital or posterior neck pain. Posterior circulation ischemic signs and symptoms are common, including gait disturbance, nystagmus, sensory deficits, Horner's syndrome, cranial nerve palsies and dysphagia. Some patients may experience milder symptoms like headache, vertigo, or tinnitus. Subarachnoid hemorrhage may also occur exclusively in intracranial vertebral artery dissection due to rupture of the adventitia. Cervical radiculopathy is another rare complication, due to compression of cervical nerve root by arterial enlargement from subadventitial mural hematoma or a dissecting aneurysm.[4]

The diagnosis of vertebral artery dissection requires a high clinical suspicion with careful history taking and physical examination, then confirmed by targeted neuroimaging. Modalities of neuroimaging include conventional digital subtraction angiography, CT angiography, MR angiography, and duplex ultrasonography.

Conventional digital subtraction angiography (DSA) has been considered as the gold standard for the diagnosis of arterial dissection. Classical findings include the double-lumen sign (the presence of a false lumen or an intimal flap) and the pearl and string sign (segment of stenotic lumen).[5] However, DSA is an invasive procedure and is associated with procedural risks. Also, it does not allow direct visualization of the artery wall and may miss dissections in which false lumen is not opacified by contrast medium.[3]

The advance in diagnostic radiology allows the increasing use of non-invasive imaging modalities including MR angiography (MRA) and CT angiography (CTA). In CTA, dissection can be easily identified by 3D reconstruction images. Intimal tear and intramural hematoma can be identified in the axial imaging. In MRI, one can visualize any intramural hematoma in T1- and T2-based imaging, assess the vascular lumen in angiography, and look for evidence of ischemic infracts in diffused weighted imaging (DWI) and fluid attenuation inversion recovery (FLAIR) imaging.[6] The sensitivity and specificity of MRA and CTA are comparable. The positive predicted value and negative predicted value of CTA are very good at 65-100% and 70-100% respectively.[7] MRI is more sensitive in detecting acute infarct, especially for small and posterior circulation infarcts . CTA is the most efficient modality in diagnosing vertebral artery dissection due to the widespread availability and rapid acquisition of images. Risks of CT are rare but include radiation risk especially in children and pregnant ladies, and contrast nephropathy.

Ultrasonography has the least contraindication and is the most inexpensive imaging modality. It is primarily used to assess the extracranial part of cervical vessels, in which flow alteration, intramural hematoma, luminal thrombus and intimal flap may be visualized. It has limited intracranial assessment due to poor penetration of ultrasonic wave through bone. Its sensitivity is dependent on the severity of the artery stenosis, and the technical expertise in detecting the subtle ultrasonic findings. In routine practice, duplex ultrasonography is less performed than CTA or MRA for the diagnosis of vertebral artery dissection, but maybe helpful when the other neuroimaging choices are not available.[8]

Treatment of vertebral artery dissection can be divided to acute treatment to re-establish blood flow to brain tissue at risk, and secondary prevention of stroke recurrence.

Acute treatments include thrombolysis and endovascular treatment. Thrombolysis with intravenous altepase is now an established treatment for acute ischemic stroke. For those with extracranial cervical artery dissection and acute ischemic stroke, thrombolysis is considered safe. However, in those with intracranial arterial dissection, the usefulness and hemorrhage risk of thrombolysis remain uncertain.[9] Endovascular treatments include proximal occlusion, aneurysm trapping, coil embolization and stenting. Intradural vertebral artery dissection, especially those complicated with dissecting aneurysm, carries a higher risk of subarachnoid hemorrhage and rebleeding in the first 24 hours. In addition to avoiding antithrombotic treatment, endovascular treatment may be considered for such high-risk patients. For those with recurrent stroke despite medical therapy, the benefit of extracranial endovascular treatment is not well established.[8]

Secondary prevention aims to lower the rate of recurrent stroke. The pathogenesis of stroke in vertebral artery dissection is postulated to be embolic from thrombus forming at the dissection site. Both antiplatelet and anticoagulation have been used as antithrombotic treatment for stroke prevention in vertebral artery dissection. The CADISS study evaluated the effectiveness of antiplatelet agents and anticoagulants in stroke prevention in cervical artery dissection, including extracranial carotid and vertebral artery dissections. For antiplatelet arm, aspirin, clopidogrel, dipyridamole or dual combination were used. For anticoagulation arm, heparin followed by warfarin were used. There was no difference between treatment groups in recurrent stroke risk or the rate of recanalization.[10] The duration of antithrombotic therapy is not well established. Antithrombotic treatments are generally discontinued after 3 to 6 months of treatment. Long term antiplatelet therapy may be considered in the setting of residual radiological abnormalities or presence of risk factors of recurrent arterial dissections.

Neurological outcome of vertebral artery dissection is usually good with most of the dissections healed upon repeated imaging.[1] Those with low NIHSS score on admission and young age enjoy a better clinical outcome.[4]

In conclusion, this is a case of vertebral artery dissection presenting with posterior circulation ischemia. The diagnosis requires high clinical suspicion and specific imaging modality including CT angiography or MR angiography. Patients can be treated with antithrombotic agents including antiplatelet agents or anticoagulants.

Tables and figures (where applicable) (no more than two figures)

Figure 1. CT angiography at diagnosis, showing almost total occlusion of the V4 part of left vertebral artery

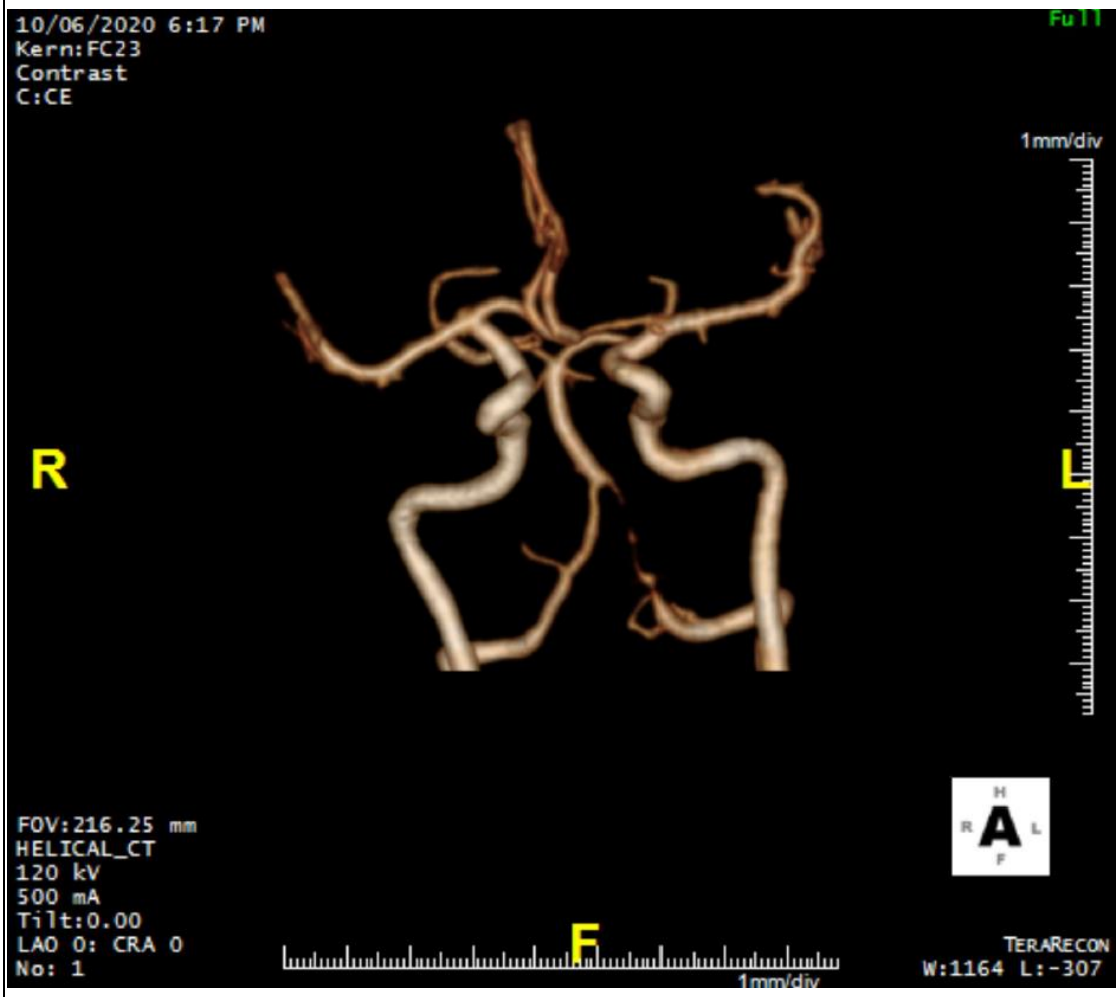
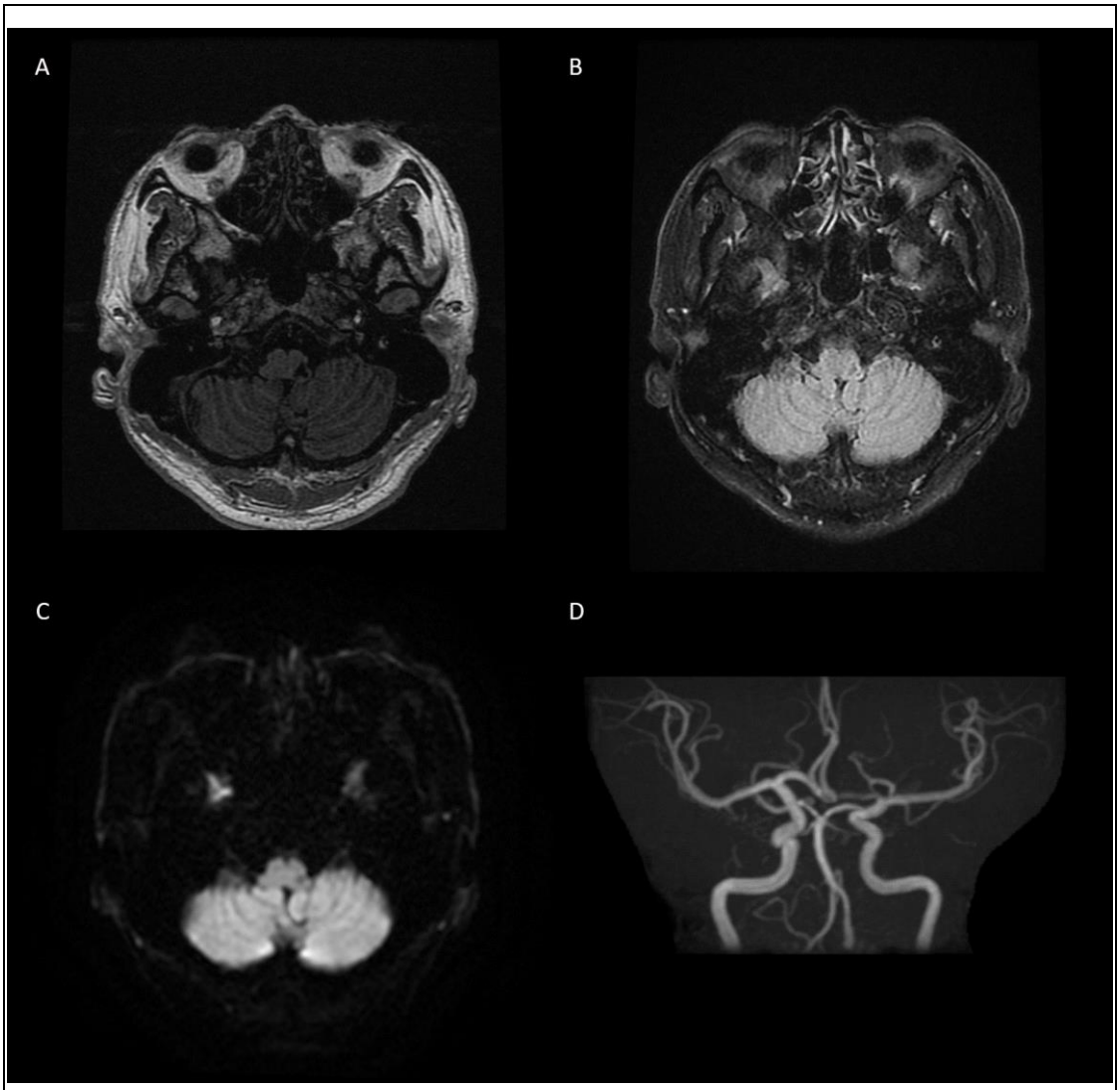


Figure 2. MRI brain 4 months after presentation, showing lesion over left lateral medulla, which is hypointense on T1-weighted image (A), isointense on T2/FLAIR image (B), and no restricted diffusion in DWI image (C), compatible with chronic infarction. MR angiogram (D) shows recanalization of left vertebral artery



Reference (not more than 10)

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4) Arnold Marcel et al., "Vertebral Artery Dissection," Stroke 37, no. 10 (October 1, 2006): 2499–2503
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Intracranial Vertebrobasilar Artery Dissection," Stroke 30, no. 5 (May 1, 1999): 1083–90

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7) James M. Provenzale and Basar Sarikaya, "Comparison of Test Performance Characteristics of MRI, MR Angiography, and CT Angiography in the Diagnosis of Carotid and Vertebral Artery Dissection: A Review of the Medical Literature," American Journal of Roentgenology 193, no. 4 (October 1, 2009): 1167–74
8) José Biller et al., "Cervical Arterial Dissections and Association with Cervical Manipulative Therapy: A Statement for Healthcare Professionals from the American Heart Association/American Stroke Association," Stroke 45, no. 10 (October 2014): 3155–74
9) Powers William J. et al., "Guidelines for the Early Management of Patients With Acute Ischemic Stroke: 2019 Update to the 2018 Guidelines for the Early Management of Acute Ischemic Stroke: A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association," Stroke 50, no. 12 (December 1, 2019): e344–418
10) Hugh S. Markus et al., "Antiplatelet Therapy vs Anticoagulation Therapy in Cervical Artery Dissection: The Cervical Artery Dissection in Stroke Study (CADISS) Randomized Clinical Trial Final Results," JAMA Neurology 76, no. 6 (June 1, 2019): 657–64

No of words in Case History and Discussion (excluding references):____1463_____

(should be between 1000-2000)

Declaration

I hereby declare that the case report submitted represents my own work and adheres to the prescribed format. I have been in clinical contact with the case selected. The case report has not been submitted to any assessment board or publication and it is NOT related to my second specialty(ies), if any. My consent is hereby given to the College to keep a copy of my case report, in written and/or electronic, at the College Secretariat and allow the public to have free access to the work for reference.

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(signature of Trainee)

Endorsed by Supervisor *

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(signature of Supervisor)

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