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Case Report

It's all in the History - Hemiplegic Migraine versus Stroke due to Arterial Dissection: A Case Report

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ABSTRACT

A 42-year-old right-handed woman with a history of hypertension and migraine presented with acute onset headache with left-sided weakness. Imaging showed right MCA territory infarction with Internal carotid artery (ICA) dissection with circumferential luminal thickening into the petrous segment. A repeat angiogram five days later revealed new left vertebral artery dissection in V3 and V4 segments. She was managed with dual antiplatelet therapy and her symptoms improved after standard stroke care. We report this case to highlight the importance of taking a good history in patients with previous migraines presenting with acute headaches and focal neurology.

Keywords: Hypertension and migraine; MCA territory infarction; Internal carotid artery; Left vertebral artery dissection; Focal neurology

Introduction

An internal carotid artery (ICA) dissection leading to petrous segment dilation is a rare entity with only a few case reports. We report an interesting case of a young woman who had a background history of chronic migraine headaches associated with visual aura and her symptoms improved after taking rest in a dark room. She presented acutely with a headache and focal neurological features and was found to have an internal carotid artery dissection with acute ischemic stroke.

Case Presentation

A 42-year-old right-handed woman presented with a gradual onset, persistent headache of one-day duration. She described this headache as a 'cold headache' as if her head had been on ice and this was different from her usual migraine headaches. This headache was associated with binocular blurring and greying of vision. She was taken to her local acute hospital where she was also found to have weakness in her left arm and leg. She had never experienced limb weakness along with migraine headaches in the past. She had a non-contrast CT head which was unremarkable. When she was about to be discharged home with a presumed diagnosis of migraine attack, she developed sudden onset left facial droop and slurring of speech. She was referred to our regional specialist stroke center, for further evaluation and management.

Her past medical history included migraine and hypertension. She was on ramipril and as required sumatriptan. She didn't smoke, had a moderate alcohol intake of 5 units per week and was not employed. Her usual migraines were chronic in nature with a pulsating character and visual aura of seeing flashing lights. There was no history of recent neck trauma.

Upon our assessment, she was alert and orientated with normal vital signs. The neurological deficits on our assessment were left facial droop and left arm and leg weakness which contributed to the score of NIHSS of 8. A CT angiogram (CTA) of the aortic arch and carotids (and intracranial vessels) was performed as all her symptoms were within 6 hours of time of onset. The CTA **(Figure 1)** revealed right Internal Carotid Artery dissection 17 mm past carotid bifurcation. No large vessel occlusion of both extracranial and intracranial vessels was found.

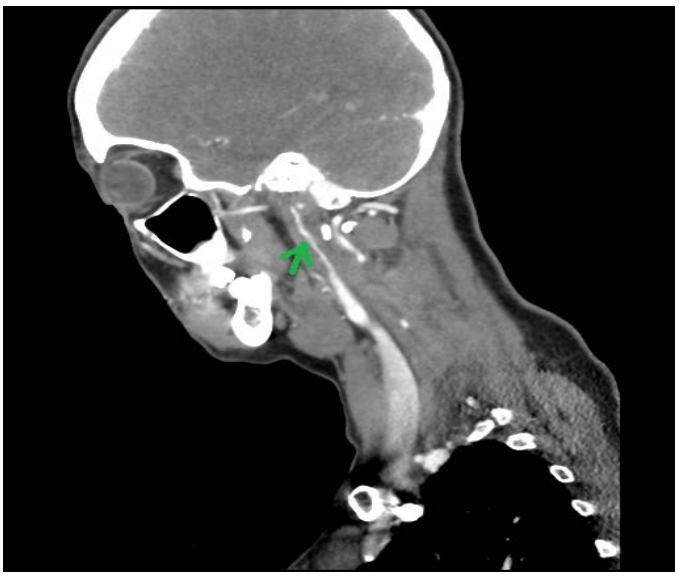

Figure 1: Right ICA dissection on CT angio (arrow). Rat tail appearance due to reduced lumen size of blood vessels because of dissection.

She was admitted to the hyperacute stroke unit (HASU) for further management. An MRI head with Angiogram **(Figure 2)** was performed which revealed acute right MCA territory infarct in the region of right insula, centrum semi-ovale and corona radiata in the right frontal lobe and aneurysmal dilatation of the petrous segment of the right internal carotid artery leading to possible occlusion of the right extracranial ICA into the petrous segment.

She was treated with dual antiplatelet therapy (aspirin 75mg plus clopidogrel 75mg) with a follow-up plan of monitoring via scans and optimizing the antiplatelet regimen as per scan findings. A repeat MR Angio 5 days later to monitor "pseudoaneurysm" showed left distal V3 segment vertebral artery (VA) dissection extending up to the border with V4. A neurovascular MDT took place and it was advised that imaging is not suggestive of pseudoaneurysm and the area of abnormality is likely an ICA dissection flap instead. The patient had no neck trauma or pain during her stay with us. The right ICA dissection appearances did not change.

Our patient also had a CT angiogram of a renal artery which did not reveal fibromuscular dystrophy (FMD). The Vasculitis screen came back negative. The exact cause of these dissections remained unclear.

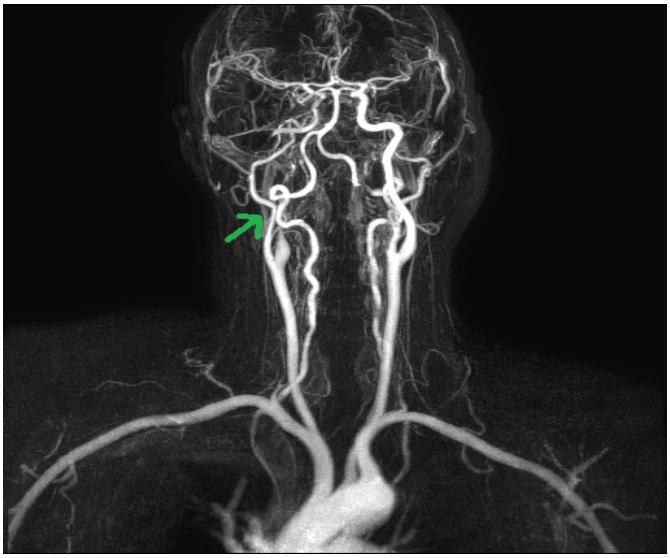

Figure 2: Right ICA dissection on Magnetic Resonance angiogram (green arrow).

She was discharged home as she showed excellent recovery. We reviewed her in the stroke outpatient clinic in 3 months and she was doing very well. A repeat MR angiogram of carotids showed that the right ICA dissection and petrous segment ICA dilation had resolved but left vertebral artery dissection was still persisting. A further follow-up was planned with repeat imaging and a plan to reduce the antiplatelet therapy to aspirin only if improvement was found.

Discussion

About 10-25% of strokes in young and middle-aged patients can be due to carotid artery dissection. The symptoms may vary and include headache, Horner's syndrome, tinnitus and features suggestive of a transient ischemic attack[1]. Incidence[2] of spontaneous CAD is 2 to 3 in 100,000 per year and bilateral CAD is even less as evidenced by only a few case reports in literature.

The risk factors[3] for CAD include minor trauma, migraine, genetic predisposition, connective tissue disorders like Ehlers-Danlos syndrome, Marfan Syndrome and a recent acute infection[4]. Outcomes in Carotid artery dissection (CAD) are usually good with mortality less than 5%[5]. Our patient denied any neck trauma, familial features or recreational drug misuse.

There is no single agreed-upon medical treatment strategy for CAD management. A randomized trial, CADISS[6] investigated the difference of outcomes of CAD in 250 patients comparing the use of antiplatelet therapy and anticoagulation therapy. The trial found no difference in efficacy between the two drug groups in terms of preventing stroke and death in both groups, the trial was however not without limitations. As per a review article by Zafer, et al[7], dual antiplatelet therapy is indicated for extracranial CAD for 3 months followed by single antiplatelet therapy for life long. Our patient was treated with dual antiplatelet therapy with the plan of reducing to single antiplatelet therapy and later follow-ups in the stroke clinic.

There are only a few similar case reports in the literature which have indicated patients having CAD masked under migraine. As per a case report by Sharif et al which includes a literature review, features of a migraine which should prompt consideration of the diagnosis of CAD are a painful Horner's

syndrome, an incomplete Horner's syndrome (miosis, ptosis but no anhidrosis), tinnitus, visual scintillations and cranial nerve palsies[8]. Our patient had clinical features of a headache not like her usual migraine headaches, also had left facial droop, slurring of speech and left arm weakness. Consideration was given to hemiplegic migraine as a potential diagnosis prior to the scans.

MR angiography is the preferred choice[1] for diagnosis of CAD but CT angiogram can be used if there are any contraindications for MR scans. Carotid duplex scan (CUSS) should only be used as a screening tool as it has poor detection of CAD near the skull base and transverse foramina[9].

An MDT approach is recommended for deciding the treatment of carotid artery dissection. Patients with CAD can present with focal neurologic deficits due to ischemia (thromboembolism or arterial occlusion) or subarachnoid hemorrhage (pseudoaneurysm formation and rupture)[10].

Overall, our patient showed good improvement after receiving medical management and rehabilitation. Headache and weakness resolved significantly and she was followed up in an outpatient clinic[11].

Our case study had limitations. There is not much follow up data available for such patients. There is lack of studies in literature clarifying use of single vs dual antiplatelet therapy in such patients. More studies are required to add to the body of knowledge on CAD patient management.

Conclusion

Our management of this case signifies the importance of taking the history of headache presentation in migraine patients. If the headache features are different and there are acute focal or lateralizing neurological symptoms, especially if presenting as an index event, we should always exclude other causes before diagnosing hemiplegic migraines. Prompt specialist referrals 7 and more detailed imaging are essential. An MDT approach should guide ongoing investigations and management for the patient.

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