Case Report

A case of neck pain in emergency department

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ABSTRACT

Neck pain is a common presentation to the emergency department (ED) and a prominent source of disability in the adult population, accounting for about 10–20% of the population. Neck pain can be challenging for ED clinicians to accurately diagnose and manage. The vital role of ED remains in diagnosing the musculoskeletal red flag conditions such as cervical spine fractures or dislocations, radiculopathies, and disk disruptions. However, most non-traumatic causes of neck pain do not have a specific pathoanatomical etiology that can be identified on imaging to identify the cause of pain in the absence of red flags signs. In this article, we would like to discuss one such case, wherein our patient, a 49-year-old gentleman presented to our ED with neck pain which rapidly progressed to quadri paresis within a short duration of ED stay and the diagnostic hurdles faced and the outcome of the patient.

Key words: Dissection, Ischemic stroke, Neck pain

Although neck pain is a common presentation in emergency department (ED), vertebral artery dissection (VAD) presenting with neck pain as a symptom, which progressed to evolve into an ischemic stroke is a game changer as most of our stroke protocols don't include a particular subset of patients presenting with cervical artery dissection (CeAD) and the relative safety and efficacy of fibrinolysis in them. There have been reports of spontaneous dissections and the majority of them are intracranial in nature. Incidental minor trauma is a common trigger but rather goes unnoticed. VAD is thought to be responsible for at least 20% of ischemic strokes in young individuals [1]. Ischemic stroke due to CeAD requires meticulous evaluation and a decision on thrombolysis should be considered based on the patient's hemodynamics and risks.

CASE REPORT

A 49-year-old gentleman, a farmer with no known comorbidities, presented to the ED with a history of sudden onset of neck pain, gradual in progression, radiating to shoulders followed by infrequent episodes of dizziness since the previous day, followed by neck pain, and acute onset of bilateral upper and lower limb weakness (Right>Left) on the day of presentation. He was apparently normal the previous day. On ED primary survey, he complained weakness of all four limbs with a power of 4 by 5. He had no complaints of headache in the past, ptosis, miosis, anhydrosis, no prior trauma history, blurring of vision, seizures, headaches, or vomiting, and no risk factors such as alcohol consumption or smoking.

On examination, he had a pulse rate of 58/min, blood pressure of 170 over 100 mm of Hg, 100 % saturation in room air, all peripheral pulses were palpable with no radio-femoral delay, and a capillary blood glucose level of 117 mg/dl. On physical examination, the patient was conscious and oriented with a Glasgow coma scale (GCS) of 15/15, the National Institute Of Health Stroke Scale (NIHSS) was 4, moderately built, and nourished. No clinical features of anemia or hyperbilirubinemia were noted. He experienced rapidly progressing weakness on the right side, with a power of 0 by 5 in the right upper and lower limbs, 4 by 5 in the left upper limb, and 3 by 5 in the left lower limb, after only a short stay in the ED.

The electrocardiogram revealed normal sinus rhythm and echo revealed normal left ventricular systolic function with grade-1 LV diastolic dysfunction and left ventricular hypertrophy. In view of sudden onset weakness, acute ischemia stroke was included in our differentials and activated the stroke protocol. Our stroke protocol magnetic resonance imaging revealed acute infarct of the right cerebellar hemisphere, right VAD, cervical

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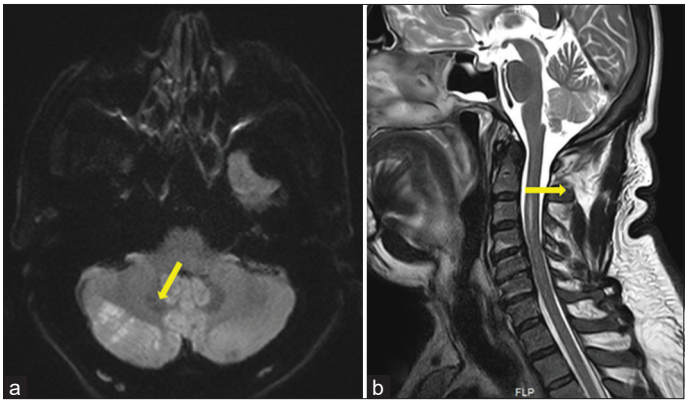

Figure 1: (a) (Axial section) diffusion-weighted images of brain showing areas of restricted diffusivity in the right posterior inferior cerebellar hemisphere (yellow arrow); (b) (sagittal section): Diffusion-weighted images of spinal cord showing areas of restricted diffusivity in the ventral cervical cord

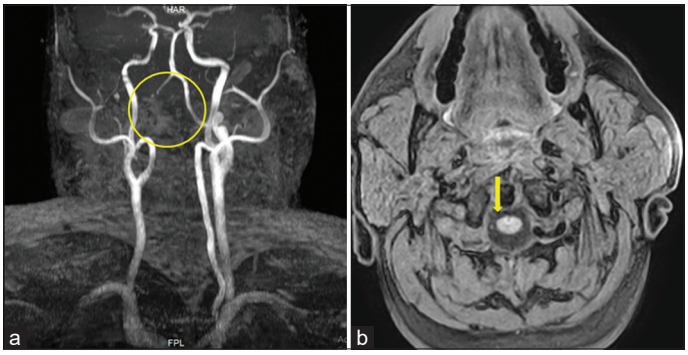

Figure 2: (a) Non contrast MR angiogram showing non visualization of flow-related signals in V1, V2, and V3 segments of the right vertebral artery and minimal distal flow in V4 segment (Yellow circle); (b): Non-contrast MR angiogram (T1) Vessel wall imaging shows non-visualization of V3 segment of the right vertebral artery with eccentric thickening (Yellow arrow)

cord, and anterior spinal artery territory infarct (Figs. 1-4). Acute to subacute infarcts are redemostrated showing T2/FLAIR hyper intensity and altered diffusivity along vermin, paravermian region, and right inferior cerebellar hemisphere with temporal changes in the signal.

We made a calculated risk and opted for thrombolysis with rTPA. After obtaining written consent, thrombolysis was performed with 20 mg of intravenous tenecteplase. He was continuously monitored for any signs of clinical deterioration or worsening from his baseline GCS. He was examined 1 h after lysis, and clinical improvement was appreciated with a power of 4 by 5 bilaterally and no deterioration in GCS from baseline. He was treated with atorvastatin, aspirin with clopidogrel, low molecular weight heparin, labetalol, and neuroprotective agents during his hospital stay.

Follow-up CT cerebral angiography was done which revealed V1–V3 segment vertebral artery occlusion, and right cerebellar infarct with no hemorrhagic transformation. After satisfactory clinical improvement, he was shifted to the ward and managed conservatively with physiotherapy and supportive care and was discharged to home on the 5th day of illness. NIHSS was 0 on discharge.

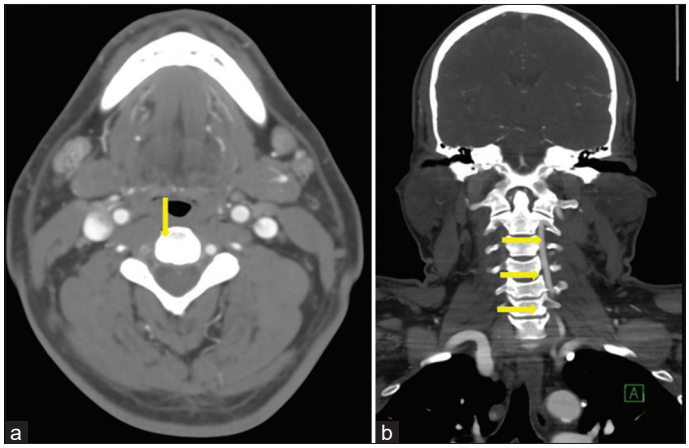

Figure 3: CT angiogram axial (a) (Yellow arrow) and coronal reconstructed MIP images (b) (Yellow arrow) - confirmed the MRA findings with incomplete opacification of the right vertebral artery and suspicious floating intraluminal thrombus

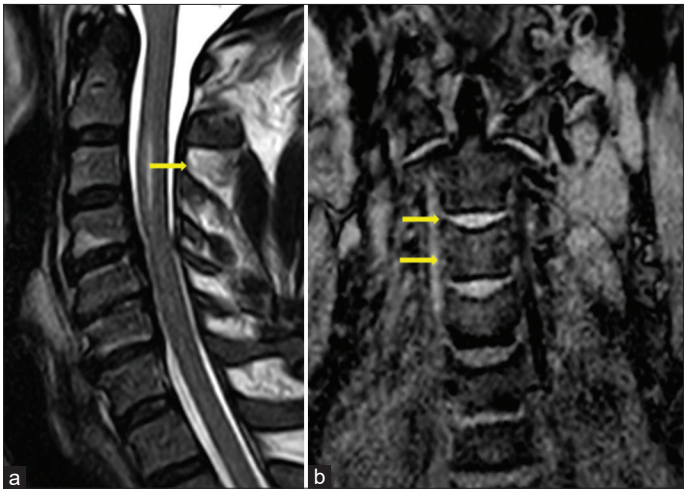

Figure 4: (a) T2 Sagittal image of the cervical cord shows more prominent linear high signal along ventral aspect of the cord from C2 to C4 levels during immediate post lysis follow-up study after 24 h (Yellow arrow); (b) Coronal T1 vessel wall imaging shows eccentric linear high signal along medial aspect of the right VA at C2–C4 concerning for intra-mural hematoma (yellow arrow)

DISCUSSION

Although stroke care has been well designed and formulated over years to perfect, prevent, and reduce long-term disability, at times, ED physicians come across common illnesses presenting in the most uncommon ways. To start with, as a rule, neck pain in ED is not always benign. VAD as a cause of neck pain still remains a diagnosis of exclusion in ED. CeAD presenting as ischemic stroke accounts for approximately 2% and 10–25% in patients <50 years of age [2]. Subsequent ischemic stroke develops due to thromboembolic complications secondary to dissection [3].

CT angiography is the initial test of choice as it can demonstrate posterior fossa ischemia or subarachnoid hemorrhage. The door-to-needle time, in this case, was 3 h, 10 min. The thrombolysis was preferred over mechanical thrombectomy, because other vertebral artery and basilar artery were found to be patent.

Fibrinolytic therapy in patients with ischemic stroke still remains a clinical debate. Numerous studies have indicated that thrombolysis may improve the prognosis by restoring perfusion of cerebral blood flow. Although the safety and efficacy have not been estimated in any of the randomized controlled trials in these subset group of patients. The most dreadful complication of thrombolysis is the development symptomatic intracranial hemorrhage, which occurs in 2–9% of patients [4]. In patients with CAD, an additional concern is the risk of enlargement of the intramural hematoma causing further deterioration of vertebral perfusion and hemodynamics [5].

Although stroke encounters are more likely in the ED, determining the cause might be challenging particularly in young stroke. Acute stroke, especially in young patients under 45 years of age, should raise red flags, which was the case in our instance. The management guidelines for VAD are currently limited. Early detection, a high suspicion of stroke, proactive management, and a dedicated stroke team are all important elements in a patient's prognosis. As far as young stroke is concerned, all possible life-threatening conditions should be considered and treated for the same. It was observed that thrombolysis seems to be equally safe and efficacious to non-thrombolysis in CeVD-induced acute ischemic stroke patients [6]. The only FDA-approved medicine for acute stroke is alteplase. Tenecteplase has potential advantages over alteplase in terms of fibrin selectivity and half-life, despite the fact that it is not FDA-approved for acute stroke [7]. Tenecteplase was also found to have associated with significantly better reperfusion and clinical outcomes than alteplase in stroke patients who were selected on basis of CT perfusion imaging [5]. Added to that, tenecteplase is superior to alteplase for patients with more severe stroke from a large vessel disease [8].

CONCLUSION

No specific trials implying thrombolysis in patients with CeAD have been conducted. As a result, in these patients, a combined clinical discussion with a neurologist and an interventional radiologist on the next course of action is critical.

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