CASE SERIES

Aortic Dissection Presenting as Acute Ischaemic Stroke and Thrombolysed: A Case Series

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Abstract

Acute aortic dissection (AAD) masquerading as ischaemic stroke can be a challenging diagnosis for clinicians and can pose complications for the patient. Type A dissections extending into the vessels of the brain and neck can cause cerebral hypoperfusion leading to strokes or stroke-like symptoms. Thrombolysis of ischaemic strokes caused due to aortic dissection can be catastrophic for the patient. We present a series of two cases of aortic dissection presenting as strokes which were thrombolysed and underwent surgical intervention for dissection but did not suffer catastrophic complications. We do not recommend to rule out dissection in acute stroke patients before thrombolysis.

Keywords: Acute stroke, Aortic dissection, Case report, Thrombolysis.

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Introduction

More than 200 years ago, Morgagni first described acute aortic dissection (AAD) which still remains one of the most challenging clinical diagnoses.[1] Only about 5–30 cases per million people per year are diagnosed with AAD.[2] Aortic dissections can be divided into type A (involving the ascending aorta) and type B (distal to left subclavian artery) according to the Stanford classification.[3] Ischaemic strokes are caused by type A dissections in 5–10% of the cases.[4] This usually occurs due to the extension of dissection into the common carotid arteries, thromboembolism or cerebral hypoperfusion.[5] We discuss two cases which presented to our emergency department (ED) with stroke, they were thrombolysed and were subsequently diagnosed with AAD followed by surgical interventions.

Case Description

Case 1

A 53-year-old hypertensive man presented to our ED with a history of sudden onset of aphasia followed by loss of consciousness 2 hours prior to arrival. The history was elicited from the wife of the patient. The patient was not compliant with his medications. On arrival, his blood pressure was 170/80 mm Hg, pulse was 110/minute, respiration – 20/minute, saturation 98% on room air, temperature 98.7 F, and a random sugar of 117 mg/dL. The patient had a Glasgow coma score (GCS) of 7 with the motor component being 3, pupils were equal and reactive and plantars were extensor. The rest of the systemic examination was unremarkable. The patient was immediately intubated and ventilated and shifted for magnetic resonance imaging (MRI) of the brain which revealed hyperintensities in bilateral cerebellar regions on diffusion-weighted imaging (DWI) with corresponding hypointensity on apparent diffusion coefficient (ADC) images suggestive of acute cerebellar infarct (Fig. 1). An additional brain stem infarct in the midbrain/pons was a strong clinical possibility and as a patient was within the time window with no obvious contraindication, along with the neurology team and after taking appropriate consent, the patient was prepped and thrombolysed with recombinant tissue plasminogen activator (rTPA) at the standard dose of 0.9 mg/kg body weight.

As a protocol, the patient underwent computed tomography angiography (CTA) of head and neck vessels to rule out basilar occlusion. It revealed aortic dissection involving the ascending aorta, arch, and descending aorta with extension into the brachiocephalic artery and right common carotid (Fig. 2). Urgent cardiothoracic surgery teams were consulted who planned conservative management of the patient in view of poor neurological status and post-thrombolysis status. The patient was taken against medical advice to another hospital where he underwent Bentall's procedure and died after 23 days of inpatient stay due to septic shock and acute renal shutdown.

Case 2

A 56-year-old diabetic, hypertensive, hypothyroid male presented to our ED with chief complaint of right hip pain radiating to his right

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Disclaimer: The views expressed in the submitted case report are the author's own and not an official position of the institution.

Patient consent statement: The author(s) have obtained written informed consent from the patient for publication of the case report details and related images.

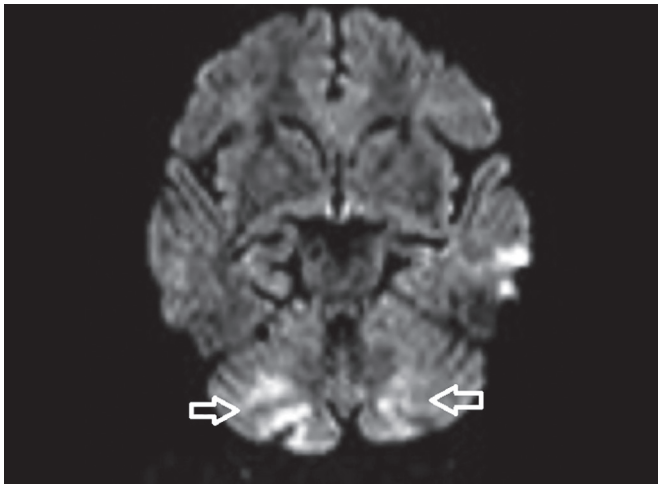

Fig. 1: Magnetic resonance imaging brain showing hyperintensities in bilateral cerebellar regions on diffusion-weighted imaging suggestive of an acute cerebellar infarct

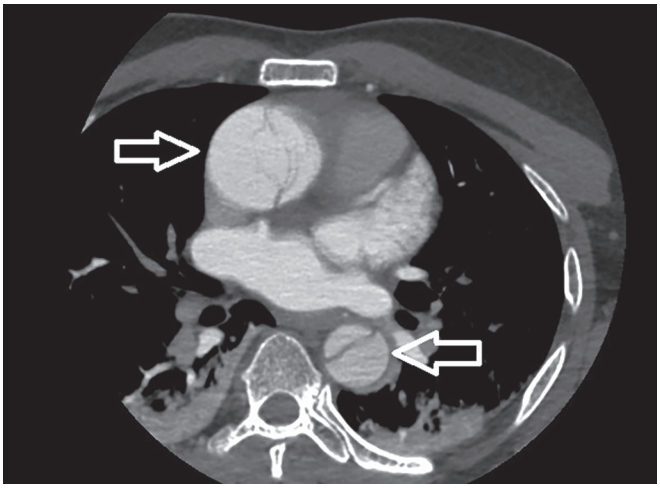

Fig. 2: Computed tomography angiography neck vessels showing aortic dissection involving the ascending aorta and descending aorta

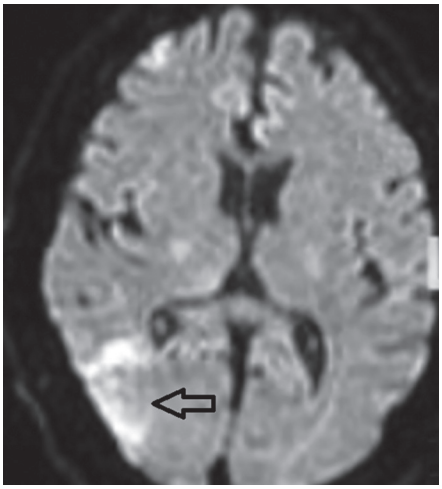

Fig. 3: Magnetic resonance imaging brain with hyperintensities in right high parietal region suggestive of acute ischaemic infarcts in a watershed area

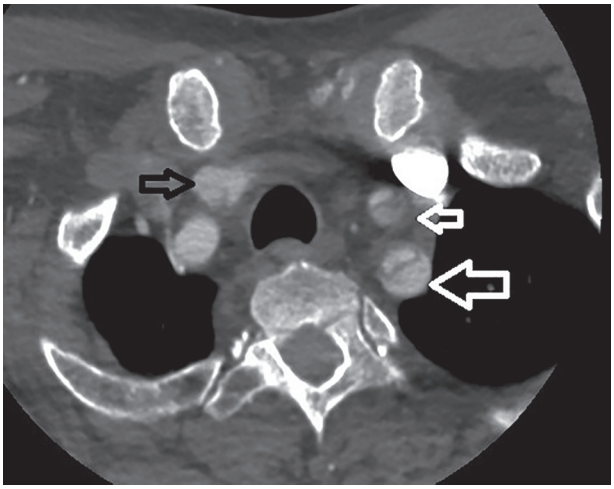

Fig. 4: Computed tomography angiography showing aortic dissection from the aortic root, extending into ascending aorta, arch, descending aorta, and into the proximal portion of the right brachiocephalic artery

leg since 1 hour prior to arrival. On examination, he had a blood pressure of 120/70 mm Hg, pulse rate – 80/min, random sugar of 200 mg/dL and a pain score 8/10. On examination, the patient had right hip tenderness and had a restricted range of motion of the right leg with a straight leg raise test restricted to 10 degrees. His right dorsalis pedis was also feeble. Rest of the peripheral pulses and systemic examination was unremarkable. He was given appropriate intravenous analgesia. A provisional diagnosis of suspected acute disc prolapse v/s acute arterial occlusion was made and the patient was shifted to radiology for X-rays and arterial Doppler. Just before the Doppler exam, it was noticed that the patient was not able to lift his left arm. On examination, he had developed fresh onset left facial palsy, power in left upper limb 3/5, poor grip, and left leg power 4/5 on the MRC scale. Suspecting a fresh stroke, the patient was immediately shifted for an urgent MRI which revealed hyperintensities in the right high parietal region (MCA/PCA watershed zone), right paraventricular region in DWI with corresponding hypodensity in ADC sequences suggestive of acute ischaemic infarcts (Fig. 3). Neurology consent was sought and after appropriate consent, the patient was thrombolysed using rTPA (90 mg). At the end of the infusion, the patient had a decline in neurological status. He was shifted for urgent non-contrast CT brain and CTA of brain and neck. The scans revealed aortic dissection starting from the aortic root and extending into ascending aorta, arch, and descending aorta and seen extending into the proximal portion of the right brachiocephalic artery, left common carotid as well as subclavian arteries (Fig. 4). Urgent cardiothoracic surgery consultation was done. The patient was taken up for high-risk Bentall's procedure after reversal and was given epsilon amino-caproic acid, cryoprecipitates, and random donor platelet infusion. Post-operatively, the patient was tracheostomised and had a good neurological recovery. The patient was discharged in stable condition after 10 days.

Discussion

Both cases highlight the complex nature and presentations of AADs. This can get even more complicated if the patient presents with neurological symptoms. AAD masquerading as acute strokes in today's era of thrombolysis can pose major clinical dilemmas for the physician as well as cause complications for the patient. Acute ischaemic strokes must be thrombolysed with rTPA within 3–4.5 hours from the time of onset of symptoms.[6,7] Reducing the door-to-needle (DTN) time to less than 30 minutes improves favourable outcomes for the patients.[8,9]

Post-thrombolysis, such patients may develop an extension of the flap into the pericardium and lead to tamponade and death.[10] The treatment of choice for type A dissections is an emergent surgical repair because of the risk of aortic rupture and catastrophic haemorrhage.[11] Although the half-life of rTPA is very short (72 minutes)[12] in the setting of aortic dissection, it can lead to massive haemorrhage pre-, intra- or post-operatively. Adverse effects of rTPA can be reversed using epsilon amino-caproic acid 4–5 gm over 1 hour or tranexamic acid 1 gm over 10 minutes and 10 units of cryoprecipitate (containing Factor VIII) infused over 10–30 minutes.[6]

Aortic dissections can be diagnosed by bedside transthoracic echocardiography (TTE) which is noninvasive yet quick.[13] Acute ischaemic stroke warrants early thrombolytic intervention but to rule out stroke mimics and contraindications before administering rTPA is even more crucial. None of the patients we have presented here worsened or had excessive morbidity due to bleeding complications. In the absence of suggestive history or any clinical signs suggesting a dissection, it becomes very difficult to diagnose it bedside. Our case presentations suggest that acute stroke patients due to aortic dissections if thrombolysed can be managed appropriately without complications. We do not recommend ruling out dissection at the risk of increasing DTN times in acute stroke patients. But it would be prudent for emergency physicians to clinically suspect a dissection as a stroke mimic and proceed accordingly.

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