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Case Report

Retrograde embolism from the descending thoracic aorta causing stroke: An underappreciated clinical condition

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ABSTRACT

The mechanism of retrograde aortic blood flow is a complex and underreported clinical phenomenon. Complex plaques of the aortic arch are considered high-risk sources of cerebral emboli.[1] Aortic plaques situated in the descending thoracic aorta are however often overlooked and in fact can be more frequent potential sources of cerebral embolism through the mechanism of retrograde aortic blood flow. We present the case of an elderly Caucasian female who experienced recurrent posterior circulation embolic strokes where the only possible underlying etiology was found to be an atheroma in the descending thoracic aorta, possibly showering retrograde emboli.

1. Case presentation

A 69 years old female presented with complaints of sudden onset diplopia, difficulty of eye movements and right hemiparesis. She was discharged from the hospital a day prior to her current admission with the diagnosis of a vertebrobasilar transient ischemic attack (TIA). She also had multiple other hospital admissions for stroke in the past: bilateral basal ganglia infarction a week prior, left internal capsular infarction six months prior and a few bilateral lacunar infarctions diagnosed about a year prior to the current admission. Other history included hypertension, hyperlipidemia, moderate aortic stenosis and atypical chest pain episodes. She was a former smoker with smoking history of 20 pack years. Her home medications included aspirin, metoprolol, hydrochlorthiazide, simvastatin and amlodipine. She was also on clopidogrel since the time of her last hospital admission for TIA. Initial vital signs were stable. Her systemic examination was remarkable for a grade 2/6 systolic ejection murmur at the aortic area consistent with her history of aortic stenosis, left internuclear ophthalmoplegia, right sided weakness (Power of 3/5 on right lower extremity and 4/5 on right upper extremity), and impaired right sided sensations with exaggerated right biceps reflex (3+). Gait was unsteady.

Routine laboratory studies including hemogram and basic metabolic panel were unremarkable. ESR, CRP, Homocysteine levels and Fasting lipid profile were normal. Antinuclear antibody and Rapid plasma reagin were negative. An outpatient hypercoagulability work-up done five months prior was unremarkable. Admission electrocardiogram showed a normal sinus rhythm with no acute abnormalities. A Computerized Tomographic (CT) scan of the brain showed a small acute to subacute left thalamocapsular infarction and chronic bilateral lacunar infarcts with no evidence of

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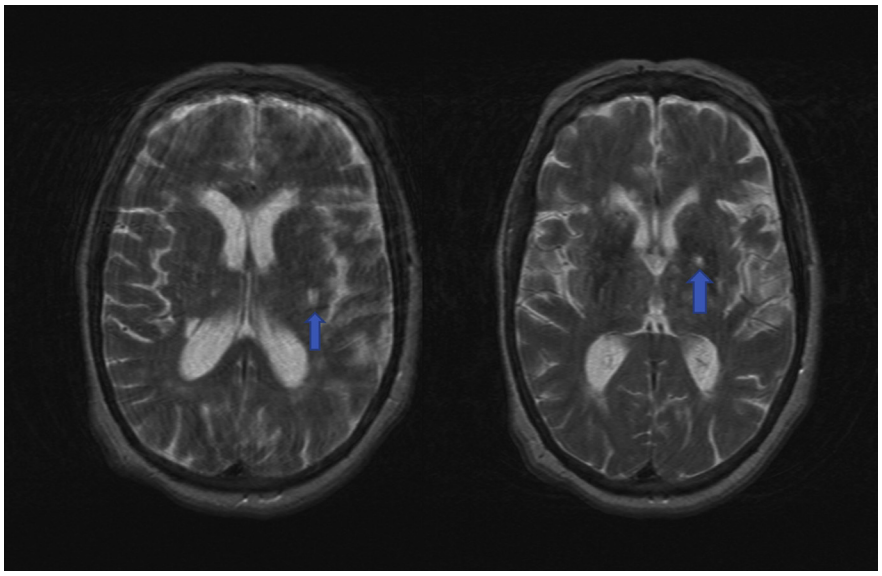

Fig.1 – Magnetic Resonance Imaging (MRI) of the brain showing multiple chronic bilateral lacunar infarcts.

hemorrhage. Magnetic Resonance Imaging (MRI) of the brain showed multiple chronic bilateral lacunar infarcts [Fig. 1] and a fresh infarct in the pontine region as represented by a small hyperintensity on the Diffusion Weighted Imaging (DWI) with corresponding hypointensity on the Apparent Diffusion Coefficient (ADC) mapping [Fig. 2]. Bilateral Cerebrovascular duplex scan showed non-stenotic common carotid, internal carotid, external carotid and vertebral arteries. Magnetic Resonance Angiography (MRA) of the head and neck also showed no evidence of significant carotid or vertebral artery stenosis [Fig. 3]. Transthoracic 2D-echocardiography with bubble study showed normal biventricular function (left ventricular ejection fraction of 60–65%) and largely normal valvular function except evidence of moderate aortic stenosis. Due to a clinical suspicion for the presence of a mobile thrombus/atheromatous plaque in the aortic arch given the patient's history of recurrent posterior circulation embolic strokes, a CT angiography of the chest (with a 3-Dimensional angiographic reformation) was performed. CT angiography showed a normal ascending aorta and aortic arch but revealed a large complex ulcerated atheromatous plaque in the proximal descending thoracic aorta (DAo), multiple other small calcified and noncalcified plaques/thrombi throughout the DAo and also a 4 cm partially calcified aneurysm with a superimposed thrombus in the DAo [Fig. 4]. Transesophageal echocardiogram (TEE)

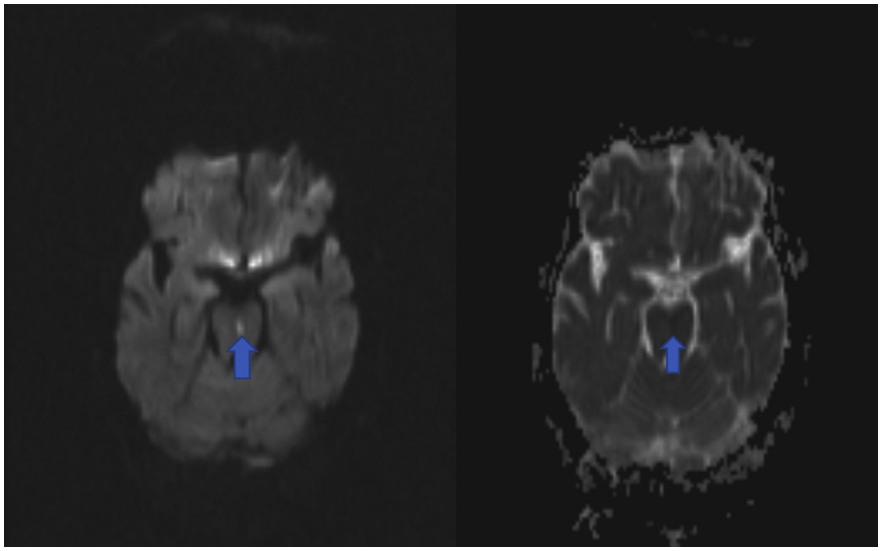

Fig. 2 – Magnetic Resonance Imaging (MRI) of the brain showing a fresh infarct in the pontine region as represented by a small hyperintensity on the Diffusion Weighted Imaging (DWI) with corresponding hypointensity on the Apparent Diffusion Coefficient (ADC) mapping.