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A Diagnostic Road to Damascus: A Case Of Conversion To Pontine Infarct

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Abstract: *A pontine stroke could be missed on the initial brain imaging and Hoover's sign often fails to differentiate between organic and functional limb weakness. We present a case of a 58-years-old lady who initially was thought to have a functional weakness which later turned out to be left hemi pontine infarct. She presented to the emergency department with right hemiplegia. On physical examination, she had muscle power of MRC grade 0/5 on her right upper and lower limb. Although brain imaging on admission to the hospital was normal with positive Hoover's sign and hand on face drop test, repeat imaging revealed a subtle left hemi pontine ischemia which 6 days later turned into an established infarct due to fusiform aneurysmal dilatation of the basilar artery. The final diagnosis was left hemi pontine infarct. Perseverance with repeat brain imaging saved a near-miss for the medical team.*

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I. Introduction

Pontine stroke although it accounts for a small percentage of all ischemic events but unfortunately is associated with a variable range of neurological deterioration.[1] Most alarmingly could be missed on initial brain imaging and thusly, repeat imaging scans are generally preferred for assessment of any brainstem strokes.[1,2] Early diagnosis of a brainstem lesion can be a grueling task because of their relatively smaller in size appearance on the brain imaging during the initial stage of ongoing ischemic brain insult and often take days to weeks before changes are evident on imaging studies.[3] Usual risk factors are hypertension, diabetes, high cholesterol. Pathogenesis includes long term hypertension or diabetes leading to lipohyalinosis of the small perforating arteries of pons causing infarction, atherosclerosis of small and large arteries, aneurysmal dilatation of the basilar artery.[4,5] Clinical spectrum of presentation depends on lesion location and size ranging from cross paralysis syndrome to less common unilateral weakness or pure sensory stroke.[4] This case is one in which the patient appeared to have a functional disorder on initial assessment but turned out to have a hemi pontine infarct, repeated imaging saved the medical team from avoiding a near miss and saving the patient from catastrophic consequences.

II. Case report

A 58 years old lady presented to the emergency department with a sudden, severe occipital headache which developed in seconds during a swim in a pool that afternoon. She was able to move out of the pool independently although later found unable to move her right side of the body. She had neither neck rigidity nor photophobia.

Her physical examination revealed she was conscious. Her heart rate was 84beats per minute and regular in rhythm. She had a blood pressure of 152/90mm Hg. On auscultation, there was no cardiac murmur. Neurological examination revealed an NIHSS score of three. Muscle power was MRC grade 0/5 on her right upper and lower limbs and normal on her left side. Muscle tone and jerks were normal in all four limbs. She had intact sensory and cerebellar function. Cranial nerves were intact with no facial asymmetry. She had a positive Hoover's sign and a positive hand on face drop test.

Initial lab investigations included a full blood count, lipid profile, HbA1c as well as liver and renal function tests. All blood tests came back within normal ranges. ECG showed a sinus rhythm. Both CT and MRI of her head appeared normal on the day of her admission (Figure 1,2). Therefore, initially, the neurological deterioration was thought to be due to a functional disorder. However, due to the persistence of her unilateral weakness repeat magnetic resonance imaging performed on the following day revealed subtle left hemi pontine ischemia which 6 days later showed an established infarct in left hemi pons (Figure 3,4). Images were reviewed by a neuroradiologist. CT angiogram intracranial including CT carotids and aortic arch was obtained and demonstrated subtle fusiform aneurysmal dilatation of the basilar artery towards its craniad aspect with narrowing of its terminal extent which is within the vicinity of the pontine arterial origin. The patient was

treated initially with aspirin after pontine infarction was identified. Later on, she was started on atorvastatin and clopidogrel for secondary prevention. In light of the intracranial CT angiogram findings, neurosurgery was consulted and recommended medical management without any need for surgical intervention. She was also reviewed by the speech and language therapist and found to have no abnormalities of her swallow, or in eating and drinking. She was reviewed regularly by the physiotherapist.

The patient was discharged home after a 29-days hospital course with minimal motor weakness improvement. Discharge prescriptions included clopidogrel 75mg daily, atorvastatin 40mg daily, amlodipine 5mg daily, metformin (modified release) 1g twice daily, and lansoprazole 30mg daily. She was instructed to follow up in the stroke clinic with a 7-day heart rate tape which was found to be normal during her follow up. She was found to have significant improvement in her neurological deficit as she was able to mobilize independently with the muscle power of MRC grade 4/5 on her right upper and lower limb during a repeat follow up in the stroke clinic 6 months later from her discharge from the hospital. The final diagnosis was left hemi pontine infarct.

III. Discussion

The brainstem is the posterior portion of the brain. It is composed of midbrain, pons, and medulla.[4] Common risk factors for pontine infarct are the same as for any other type of stroke which includes smoking, diabetes, hypertension, hypercoagulable state, vasculitis, hypercholesterolemia, history of ischemic heart disease, atrial fibrillation.[4] The aetiopathogenesis of pontine infarct ranges from atherosclerotic occlusion of the small and large arteries to cardiogenic emboli.[4] Lipohyalinosis of the small perforating arteries of the pons as well as a basilar artery aneurysm can lead to the development of infarction within the pons.[4,6] Clinical presentation depends on anatomical and arterial territories involved. Various presentations of pontine infarction are - contralateral motor hemiparesis or hemiplegia, ipsilateral abducens or facial nerve palsy, ipsilateral ataxia/hemiataxia, pure sensory stroke (ipsilateral facial sensory disturbances/contralateral hemisensory loss), tetraplegia, locked-in-syndrome, intranuclear opthalmoplegia.[4] Hoover's test on the other hand performed by the clinician often fails to differentiate between organic and functional causes of limb weakness particularly if the patient has components of both.[7]

IV. Conclusion

This case report discusses the significance of repeated brain imaging if initial imaging is negative for ischemic changes in a patient who presented with worsening neurological symptoms and was diagnosed with left hemi pontine infarction. Further studies, utilizing structural and functional magnetic resonance imaging are needed to provide better insights into the underlying pathophysiology of pontine infarction. This case serves to emphasize the need to maintain a high index of suspicion for brainstem infarcts despite initial negative brain scans, as small lesions can present with significant neurological deficits. Prompt neurovascular evaluation and treatment can help to prevent devastating neurological deficits and avoid near misses.

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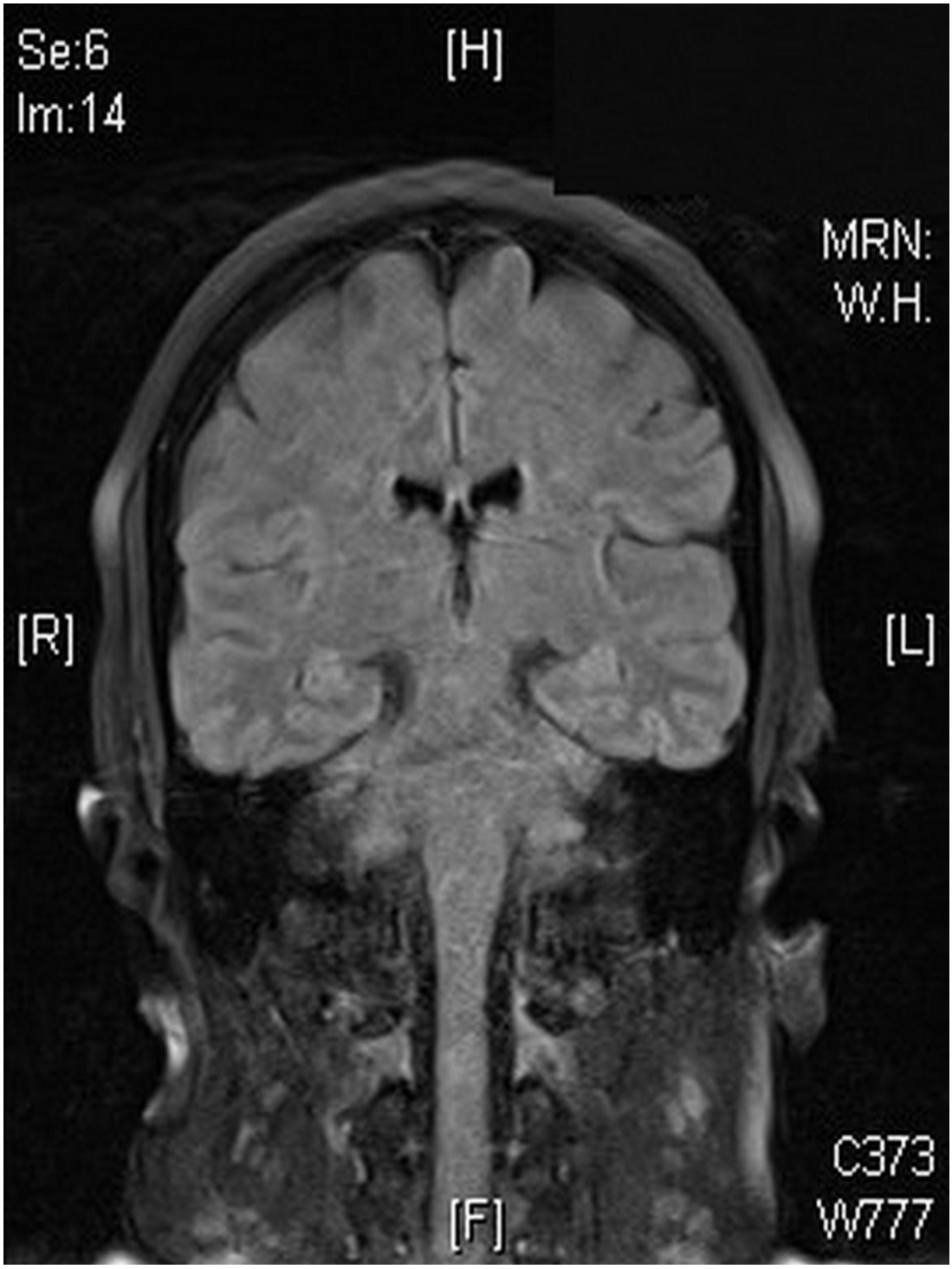

Figure 1: MRI Brain showing no acute ischemic changes

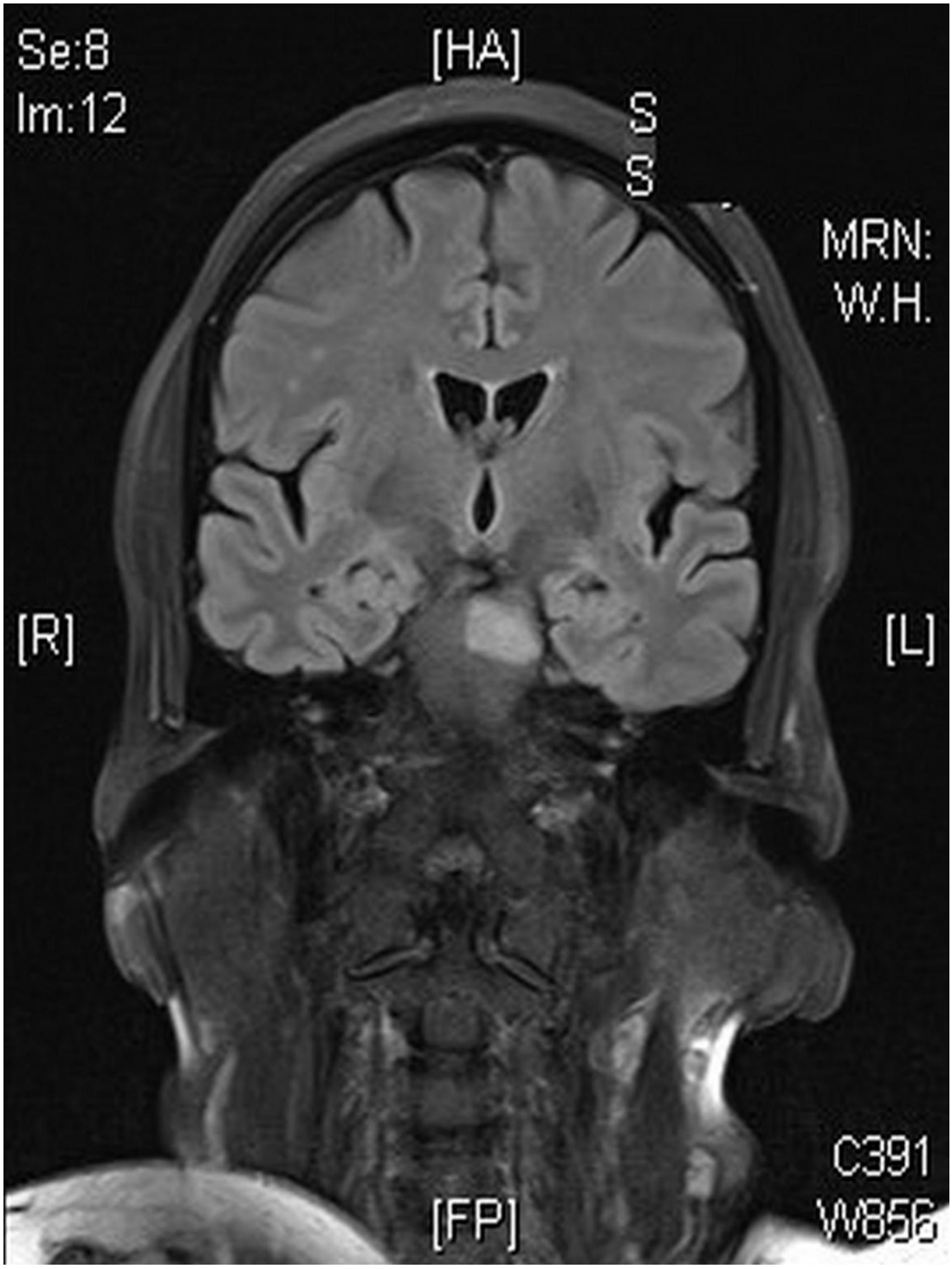

Figure 2: Repeated MRI brain demonstrating left hemi pontine infarction

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