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Case Report-Medicine

An Unusual case of flaccid Quadriplegia

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Abstract

This case describes a 47 years old Obese post menopausal lady with sudden onset flaccid quadriparesis with sparing of posterior column sensation .She has a prior history of Diabetes mellitus ,systemic hypertension ,Dyslipidemia , and hypothyroidism on regular .She was admitted for 1 day history of fever and malaise, laboratory parameters suggestive of a Possible a viral ethnology .She developed sudden onset of weakness, initially started on her Lower limb and progressing to her upper limb within few hours along with bladder involvement which appeared on the next day of hospitalisation prompt thinking of a demyelination like pattern after ruling out mechanical causes with a radiograph .In the background of a fever prompted thinking of a fast progressing demyelination like a GBS/ADEM and even started IVIG as there is always some time delay for MRI to arrive. The MRI report suggested of Thrombosis of ASA territory involving the anterior paramedian aspect of Medulla extending up to the upper cervical cord. She remained quadriplegic thereafter and 6 months later despite receiving antiplatelets, Physiotherapy and other supportive treatments. The case described here elaborates the clinical presentation and outcome of ASA thrombosis. ASA thrombosis though rare is be thought as a differential of acute onset flaccid quadriparesis in an acute setting .The clinical course and devastating nature of illness should be aware off. Its necessary to diagnose this syndrome in the earliest when patient presents with acute onset of motor weakness sparing the posterior column sensation

Keywords: ASA thrombosis, Areflexia, Quadriplegia

Introduction

Spinal cord infarction though rare is caused by number of pathological causes presenting as acute onset of quadriparesis and paraparesis depending of site of involvement .The most common form of this clinical presentation of a spinal cord infarction is anterior spinal artery (ASA) syndrome [1,2,3]. Anterior cord syndrome (also known as Beck's syndrome or anterior spinal artery syndrome) .The diagnosis is made clinically and supported by radiological imaging to exclude other causes. In ASA syndrome the primary blood supply to the anterior portion of the spinal cord, is interrupted, causing ischemia or infarction of the spinal cord in the anterior two-thirds of the spinal cord and medulla oblongata. It is characterized by loss of motor function below the level of injury, loss of sensations carried by the anterior columns of the spinal cord namely pain and temperature but with preservation of sensations carried by the posterior columns sensations. It may also be associated with autonomic dysfunction, bowel and bladder involvement and chest pain and ECG changes and rarely Unilateral presentations [4,5,6]. The vascular supply of the spinal cord relies on three longitudinal arterial trunks: the anterior spinal artery, which originates at cervical levels from the vertebral arteries, and the posterolateral spinal arteries. At the thoracic and lumbar levels, the anterior spinal artery is additionally supplied by segmental aortic vessels [7,8]. The most important feeding artery of the thoracolumbar spinal cord is the great anterior radiculo-medullary artery, also known as the Artery of Adamkiewicz [9,10]. This artery supplies the lower two-thirds of the spinal cord via the anterior spinal artery [11,12]. If this artery is injured or unintentionally interrupted (dominant vascular supply to the anterior spinal cord), it might lead to ischemia of the ventral horn, ventral commissura, and the sympathetic centers of the intermediolateral region; it manifests as anterior spinal artery syndrome with impaired motor and sensory function of the bilateral lower extremities and loss of urinary and fecal continence [13,14].

Case Report

This case describes a 47 Years old lady who presented to the General medicine OPD with complaints of fever, head ache, generalised myalgia, and malaise for one day duration. Fever was mild degree, present for one day duration. It was associated with mucoid nasal discharge and generalised myalgia. She gives a history of Type 2 Diabetes mellitus , Systemic Hypertensive , Dyslipidemia and Hypothyroid well controlled on oral medications . On Examination she appeared as morbidly obese lady (BMI-36) with mild pallor and febrile with a temperature of 99.4 F, Her Blood pressure was170/100 mm Hg, rest of vitals were within normal limit. Systemic examination appeared normal and her Laboratory investigations showed only mild elevation of CRP and blood sugars .She was admitted and treated with symptomatic medications and fluids as for a viral Fever .The very next day she developed sudden onset of flaccid weakness, initially started on her both Lower limb and progressing to her upper limb progressed over to a locked in state within few hours .Examination of her cranial nerves and higher mental function were normal . She had retentions of urine for which she had to be catherised and bowel incontinence .She had intact posterior coloumn sensations but her pain

and temperature perceptions were reduced in both limbs .This combination of acute flaccid weakness of all limbs without any cranial nerve involvement with dissociated anaesthesia prompted a vascular accident rather more prominent than a demyelination phenomenon (atypical GBS). Laboratory investigations repeated were normal .CSF analysis and EEG were within normal limits .The Magnetic resonance imaging of brain and spinal cord was performed which suggested a Thrombosis of Anterior spinal artery (ASA) territory involving the anterior paramedian aspect of Medulla extending up to the upper cervical cord.

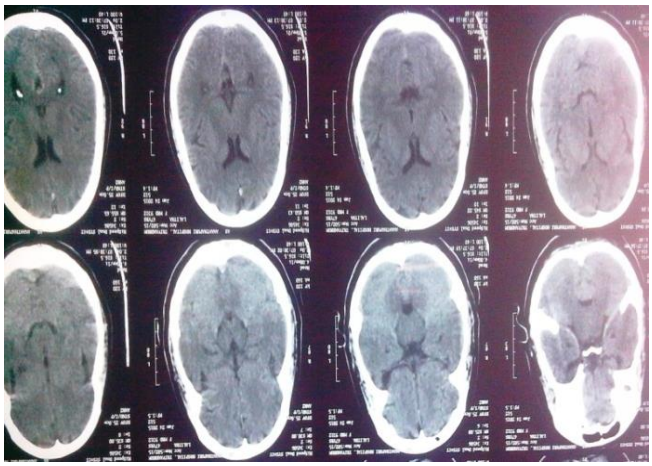

Fig 1. Plain CT brain appears normal

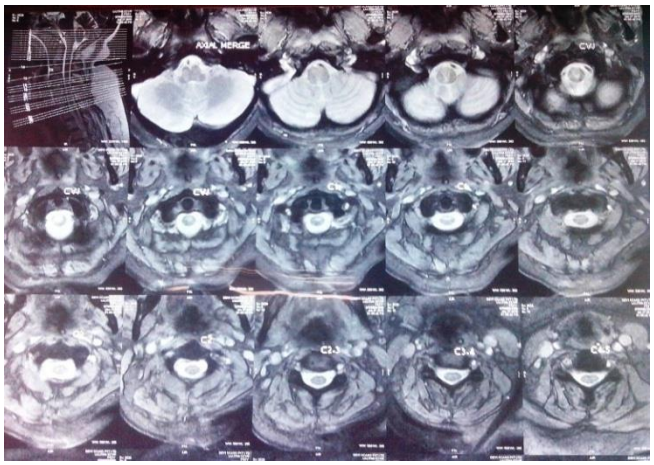

Fig 2. MRI Cervical Spinal cord

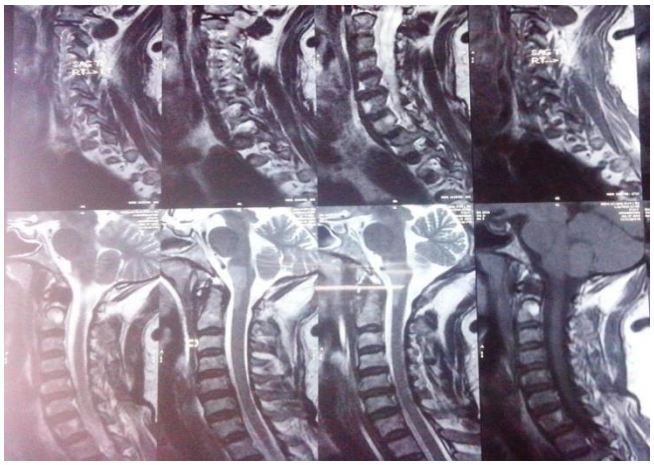

Fig 3. MRI T2 Spinal and brain stem images

Fig 2,3,4 ; MRI Brain and Cervical Spine showed T2 and Flair Hyper intensity with diffusion restriction involving the anterior paramedian aspect of Medulla extending up to the upper cervical cord suggestive of an acute infract involving Anterior spinal artery (ASA) territory(total length of involvement is 3.5 cmts) .She was managed with antiplatelets and statins along with regular physiotherapy and treatment of her co morbid conditions. On follow up after 6 months she remained paraplegic with minimal neurologic recovery.

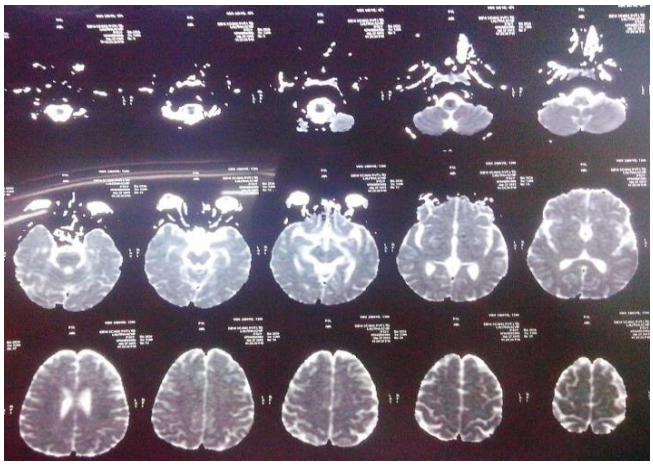

Fig 4. MRI DWI images brain.

Discussion

ASA syndrome refers to the typical neurological syndrome of acute painful flaccid paralysis with bladder involvement secondary to occlusion of the anterior spinal artery . The causes of this devastating syndrome include hypercoagulable states, anti phospholipid antibody syndrome, Sickle cell anemia, Down syndrome, atlanto-axial dislocation, embolism, trauma ,Abdominal aneurism repairs ,syphilis and vasculitis which were ruled out by appropriate investigations. Adult patients usually have risk factors like hypertension, diabetes, smoking, hypercholesterolemia or degenerative spine disease. Aortic dissection and aortic surgeries can result in fulminant ASA thrombosis [3-5].There is no definite guidelines for thrombolytic therapy in anterior spinal artery infract most recommended treatment is antiplatelets and heparin analogues .But the preferred therapy in acute phase would be the use of thrombolytic therapy which was not considered in our patient as she had presented in a atypical fashion mimicking demyelination like GBS [12]. Antiplatelets and low molecular weight heparin can be used along with supportive measures and physiotherapy.The overall prognosis remains poor with majority of patients ending up with some degree of disability [3-5]. In a study on the clinical outcome of ASA ischemia, 17.1% patients were ambulatory at the time of discharge which speaks against a universally bad prognosis. Patients who were younger were observed to have a better outcome. Severe initial impairment and female sex were found to be independent predictors for poor outcome in another study both of which are factors which are going to play a significant part in the neurological recovery of our patient. Our patient remained quadriplegic with minimal motor recovery after 6 months despite treatment.

Conclusion

The case described here elaborates the clinical presentation and outcome of ASA thrombosis. ASA thrombosis though rare is be thought as a differential of

acute onset flaccid quadriparesis in an acute setting .The clinical course and devastating nature of illness should be aware off. Its necessary to diagnose this syndrome in the earliest when patient presents with acute onset of motor weakness sparing the posterior column sensation.

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