

## A Rare Complication of Pre -Eclampsia and Severe Postpartum Hemorrhage – Spinal Cord Infarction Leading To Anterior Spinal Artery Syndrome

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### I. Introduction

Spinal cord infarction is a rare but often devastating disorder caused by a wide array of pathologic states. Anterior spinal artery (ASA) syndrome is a common manifestation of acute ischemic cord infarction. It is caused by hypoperfusion of the anterior spinal artery, leading to ischemia in the anterior two thirds of the spinal cord.<sup>1</sup> Patients typically present with acute paraparesis or quadriparesis with impaired bladder and bowel control. We report a case of severe postpartum hemorrhage causing hypovolemia and systemic hypotension leading to spinal cord infarction. To the best of our knowledge, this is first case report of ASA syndrome due to postpartum hemorrhage.

### II. Case Report

A 28 year old P3L2 woman, delivered four days back at primary healthcare center, referred for weakness and loss of sensations in all four limbs and decreased urine output since delivery.

The present and past medical history of the patient was unremarkable. In the latest pregnancy, she was detected to have hypertension but did not comply with the treatment. She developed severe hypertension and blood loss during delivery and had received four units of whole blood, as per documentation in referral letter.

On admission, her PR-90/min, BP-190/120mmhg, pallor+. CNS examination showed normal higher functions with complete flaccid paralysis of both arms and legs with absent deep tendon and abdominal reflexes. Planter reflex was extensor bilaterally. Sensory system examination revealed “dissociate anaesthesia” in form of impaired pain and temperature sense with preservation of light touch, vibration and joint position sense. She had impaired bowel and bladder function and was catheterized for urinary retention.

Investigations at admission showed haemoglobin-8.2g/dl, total leucocyte count- 13000cell/cmm, platelet count-1,19000/cmm, blood urea-126mg, serum creatinine-4.2mg, serum uric acid- 6.6mg and serum LDH – 325IU/L. Serum bilirubin, AST, ALT and alkaline phosphatase were within normal limits. Urine examination showed 1+ proteinuria. Anti nuclear antibody (ANA), antibody against double stranded DNA (ds DNA), anti phospholipid antibody (APLA) IgM and IgG, anti cardiolipin antibody (ACLA) IgM and IgG were all negative.

Ultrasonography showed bulky postpartum uterus with normal sized kidneys. Renal Doppler showed mild increased resistance in both renal arteries with normal velocity. MRI cervical spine showed patchy altered signal intensity involving anterior and central cord, extending from medulla to cervical cord upto

C- 7 level ( fig 1-3 ). It appeared isointense on T1W images and hyperintense on T2W images. It showed restriction on DW images, suggesting spinal cord infarction in the ASA territory. Screening of brain and rest of the spinal cord revealed no abnormality.

Patient was treated with oral antihypertensives including tab Amlodipin 10 mg and tab Labetolol 200mg. The renal failure seemed to result from hypovolemia, responded well to intravenous rehydration followed by inj frusemide and did not require dialysis. CECT Angiography of spinal artery was performed after normalization of renal function on Day 7 showed no abnormality, indicating the profound systemic hypotension as the culprit of anterior spinal artery ischemia.

Follow-up after a 2-month period of intensive physiotherapy, neurological examination showed spastic quadriparesis with grade 3 muscle power in all groups.

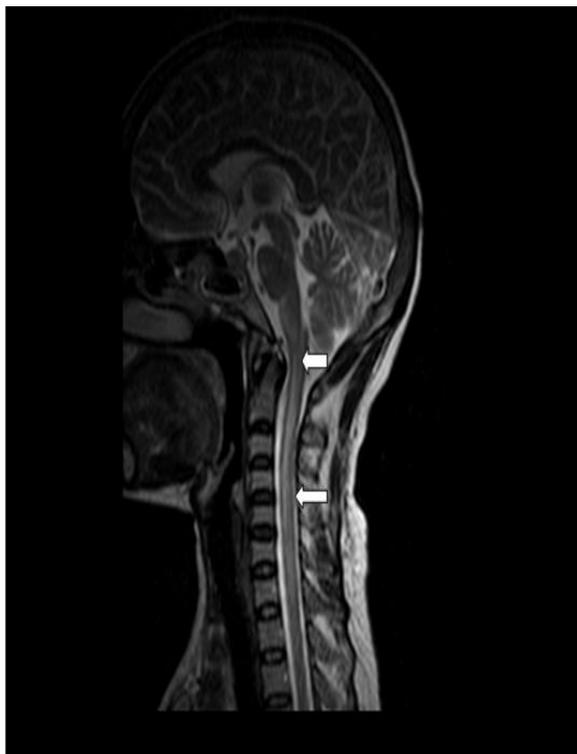
### III. Discussion

As with cerebral infarction, the onset of spinal cord infarction is typically abrupt.<sup>2</sup> The neurologic presentation of spinal cord infarction is largely defined by the vascular territory involved. The most common clinical presentation of a spinal cord infarction is anterior spinal artery syndrome. The causes of ASA syndrome include aortic surgery, atherosclerosis, diabetic arteriopathy, vasculitides, sickle cell disease, cervical spinal trauma and prolonged arterial hypotension due to any cause. The usual presentation of ASA syndrome is an acute and painful myelopathy. The acute stages are characterized by flaccidity and loss of motor function and pain/temperature sensation with relative sparing of vibration and position sense because of the preresvation of the posterior columns. Orthostatic dysfunction may be present and can manifest as hypotension, sexual dysfunction, and bowel-bladder dysfunction.<sup>3,4</sup>

Management of ASA syndrome is supportive with intensive rehabilitation. There is no evidence of use of steroids in ischemic spinal cord infarction.

### IV. Conclusion

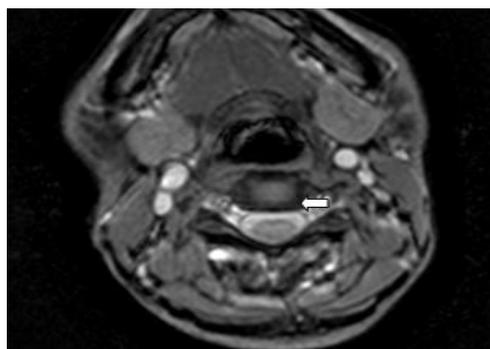
Although rare, ASA syndrome should be considered in any woman who has a history of postpartum hemorrhage with prolong hypotension and present with neurological deficit. Every possible attempt should be made to maintain perfusion pressure in spinal arteries, so as to prevent this dreaded complication.



SAGITTAL T2 WEIGHTED MRI OF CERVICAL SPINE SHOWING HYPERINTENSE SIGNAL IN SPINAL CORD EXTENDING FROM MEDULLA TO C7 LEVEL



SAGITTAL T2 WEIGHTED MRI OF SPINE SHOWING HYPERINTENSE SIGNAL IN SPINAL CORD EXTENDING FROM MEDULLA TO C7 LEVEL.



AXIAL T2 WEIGHTED MRI OF CERVICAL SPINE SHOWING HYPERINTENSE SIGNAL IN CENTRAL REGION OF SPINAL CORD

### **References**

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