CASE REPORT

POST PARTUM MULTI ORGAN INFARCTION SYNDROME- A RARE CASE

Malagouda R Patil[1], Arjun V A[2], Suman G R[3], Sriharsha kulkarni[4]

[1] Assistant professor, Department of Nephrology, [2] Junior Resident, Department of Medicine, [3,4] Assistant professors, Department of Medicine, S. S. Institute of Medical Sciences & Research Centre, Davangere

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Abstract

Postpartum hemorrhage (PPH) is the leading cause of maternal mortality in developing world. In the most severe cases, it may lead to anterior pituitary ischemia with delay or failure of lactation. Occult myocardial ischemia, acute kidney injury, dilution coagulopathy, and death may also occur. Spinal cord infarction is a rare but often devastating disorder caused by a wide array of pathologic states. We report a case of severe postpartum hemorrhage causing hypotension leading to multi organ infarction involving brain, spinal cord, liver and kidney. The patient was managed with hemodialysis and other conservative management which brought her to recovery.

Key words: Acute kidney injury, Anterior spinal artery syndrome, Post partum hemorrhage, Multi organ infarction, Acute shock liver

Introduction

Postpartum hemorrhage (PPH) is the leading cause of maternal mortality in developing world. In most severe cases it may lead to anterior pituitary ischemia, occult myocardial ischemia, acute kidney injury, dilutional coagulopathy, and death may also occur. Spinal cord infarction is a rare but often devastating disorder caused by a wide array of pathologic states. Patients typically presents with acute paraparesis or quadriparesis with impaired bladder and bowel control. We report a rare case of severe postpartum hemorrhage causing hypovolemia and systemic hypotension leading to multi organ infarction.

Case study

A 27 year old female P2L1D1, on 2nd post natal day presented to emergency with history of altered behavior for a brief period of 12 hours and oliguria after delivery in government hospital. Obstetric history revealed severe pre eclampsia with abruption of placenta with significant PPH and intra uterine fetal demise. She was intubated in outside hospital in view of low general condition. On examination, she had severe pallor, mild pedal edema, Pulse rate-104/min, Blood Pressure-150/90mmhg, respiratory rate-14 cycles/min and was afebrile. Saturation was 100% on room air with artificial manual breathing unit (AMBU). On CNS examination, she was conscious and cranial nerve examination could not be done. Fundus examination showed papilledema. Power was three (3/5) in upper limbs and two (2/5) in lower limbs with absent all superficial reflexes. Sensory system examination revealed dissociated anesthesia in the form of impaired pain and temperature sense with preservation of light touch, vibration and joint position sense. Respiratory, cardiac and gastro intestinal examination was within normal limits. Investigations at admission showed haemoglobin-4.3g/dl, total leucocyte count- 14,200cell/cmm, platelet count-35,000/cmm. Peripheral smear did not show any evidence of intra vascular hemolysis. Serum sodium-134, Potassium- 3.5, blood urea-109mg, serum creatinine-3.0mg, serum lactate dehydrogenase(LDH) – 26,170IU/L. ABG shows PH- 7.0, PcO2-34, Po2 99,

Address of Correspondence :
Dr. Arjun V A
Junior Resident
Department of Medicine
S. S. Institute of Medical Sciences & Research Centre, Davangere
E-mail - arjunsporter@gmail.com

and Hco3- 8. Serum bilirubin-2.6mg/dl (direct-1.0mg/dl, indirect-1.6mg/dl), total protein-3.8g/dl, ALB-1.3g/dl, AST- 14,965U/L, ALT-4570U/L and alkaline phosphatase-177U/L. Prothrombin time was 21 sec and INR was 2.4. Urine examination showed 2+ proteinuria and 3-5 red blood cells/cmm. Anti nuclear antibody (ANA), antibody against double stranded DNA (ds DNA) were all negative. Ultrasonography whole abdomen showed multiple hypo echoic lesions in the liver (fig.1) suggestive of infarction, bulky postpartum uterus and normal sized kidneys with grade II corticomedullary differentiation with mild ascites. The patient was started with intravenous broad spectrum antibiotics and diuretics. The patient was initiated on hemodialysis from the second day of admission in view of volume overload and acidosis. The patient was transfused a total of six units packed red blood cells, four units of fresh frozen plasma and nine units of random donor platelets in first two days. The power in both upper and lower limbs decreased to zero (0/5) on second day of admision. From third day, patient showed improvement in both upper and lower limbs power, improvement in platelet counts and decline of liver enzymes. On the fifth day the patient had normal power in both upper and lower limbs and was extubated. MRI brain showed hyperintense image of pituitary on T1WI and iso to hypo intense on T2WI suggestive of pituitary apoplexy (fig. 2). The patient started pouring urine after 2 weeks and now after 6 weeks, having creatinine around 2-3 mg/dl. The patient was started on thyroid and steroid replacement therapy.

Discussion

Infarctions in multiple organs is a very rare clinical finding, occurring commonly due to disseminated intravascular coagulation and other hereditary or acquired conditions of thrombophilia.[1] In most of the cases, multi organ infarction is due to the thrombosis in the arterial system. Sudden cardiac arrest and hypotension can also lead to infarctions in the multiple organs due to acute cessation of blood supply. [2] There is literature evidence of anterior spinal artery syndrome after sudden cardiac arrest and hypotension after major vascular surgeries. [2] In our patient we had infarctions in the liver, anterior spinal artery territory infarction, pituitary apoplexy and acute cortical necrosis. The anterior spinal artery syndrome is most common clinical presentation of a spinal cord infarction. 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. [2] 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.[2] Management is supportive with intensive rehabilitation which gives fairly good outcome in cases of hypotension induced ASA syndrome. Pituitary apoplexy is rare endocrine emergency which can occur due to infarction or haemorrhage of pituitary gland. [3] Patients usually present with headache, vomiting, altered sensorium, visual defect and/or endocrine dysfunction. [3] Bilateral cortical necrosis is a severe and often irreversible form of acute tubular necrosis that is associated with septic abortion and placental abruption. [4] Cortical necrosis can involve the entire renal cortex, often leading to irreversible renal failure, but more commonly involvement is patchy and incomplete. In such cases, a protracted period of oligoanuria is followed by variable return of renal function. The treatment of acute cortical necrosis in pregnancy is supportive with prompt restoration of fluid volume and dialysis when needed. [4] In our case the sudden hypotension which was precipitated by post partum hemorrhage lead to infarctions in many vital organs like brain, spinal cord, liver and kidney, also the functions of liver, spine and kidney recovered over time except pituitary, which needed hormone replacement. The above discussion shows that, the management of multi organ infarction in post partum period is supportive and in most of the cases the functions recover over time. So, the possibility of ASA syndrome with other organ infarction should be kept in any patient who develops sudden onset quadriparesis after an event of sudden hypotension.

Risk factors for postpartum hemorrhage include a prolonged third stage of labor, multiple delivery, episiotomy, fetal macrosomia, and history of postpartum hemorrhage. [5] Diagnosis of ASA syndrome needs very high clinical suspicion as it mimics many other conditions like guillain barre syndrome and critical illness polyneuropathy. Both obstetricians and intensivists should aggressively contribute in prevention and timely management of these cases which can prevent mortality, lifelong morbidity and disability of patients.

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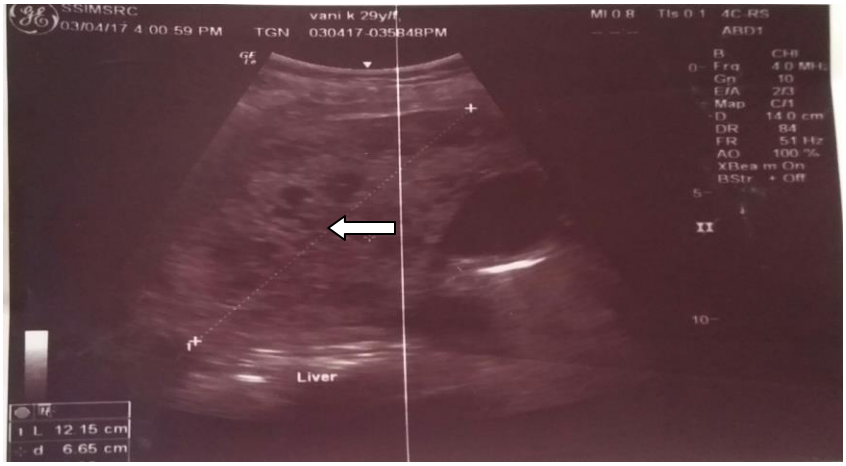

Fig. 1: Ultra-sonography of abdomen showing multiple hypo-echoic areas in liver parenchyma suggestive of infarction

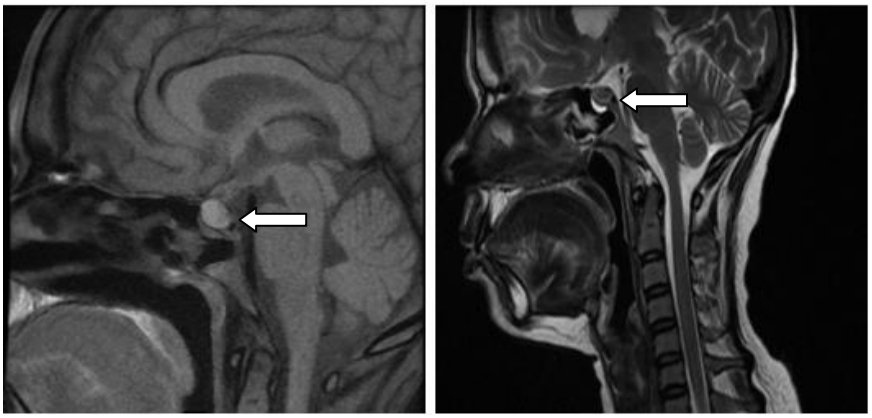

Fig. 2. MRI brain showing hyperintense image of pituitary on T1WI and iso to hypo intense on T2WI of pituitary apoplex

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