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Case report

Spontaneous spinal epidural haematoma: a rare cause of quadriplegia in the post-partum period

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Spontaneous spinal epidural haematoma (SSEH) is a rare cause of neurological deficit in the pregnant and post-partum patients. However, SSEH with associated myelitis presenting as quadriplegia and respiratory paralysis in the post-partum period has never been reported. We report the development of acute onset quadriplegia progressing to respiratory arrest in a 24-yr-old woman 2 weeks after normal vaginal delivery. There was no history suggestive of any coagulopathy (inherited or acquired), eclampsia, pre-existing neurological deficit, or iatrogenic manipulations such as spinal/epidural injections. Magnetic resonance imaging revealed a posterior epidural haematoma extending from C4–C7 and areas of signal changes in spinal cord from cervicomedullary junction to D5 level (suggestive of demyelination). We highlight this rare cause of quadriplegia; focusing on the altered dynamics of the epidural vasculature in the peripartum period leading to SSEH.

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Neurological complications in the peripartum period are commonly due to eclampsia, intracranial haemorrhage, cerebral venous thrombosis, epilepsy, and myasthenia gravis.[1] Quadriplegia as a result of spontaneous spinal epidural haematoma (SSEH) in the post-partum period is a rare occurrence, more so in the absence of predisposing factors. A thorough search of relevant literature in English language revealed six cases of SSEH. None of these was associated with demyelination of the spinal cord or brain stem. We report the development of acute onset quadriplegia associated with respiratory paralysis in a young woman 2 weeks after a normal vaginal delivery. This report aims to highlight SSEH with cord demyelination as a possible cause of quadriplegia in the post-partum period.

Case history

A 24-yr-old, previously healthy woman gave birth to her second child by normal vaginal delivery. Her antenatal course had been uneventful. On 15th post-partum day, she suddenly experienced pain in the neck which radiated to the shoulders. This was also associated with weakness of all four limbs. Within the next 2 days, she became bedridden and also experienced difficulty in swallowing and breathing. On post-partum day 17, she became unresponsive and was rushed to the emergency department of our institute. On admission, she had a Glasgow Coma Scale (GCS) of 3/15. Pupils were semi-dilated and sluggishly reacting to light. She had a power of Grade 0/5 in all four limbs. Reflexes could not be elicited. She had a non-invasive arterial pressure of 90/50 mm Hg and heart rate of 58 beats min^{-1}. Arterial blood gas (ABG) drawn on admission revealed: pH, 7.056; P_{O_2}, 83 mm Hg; P_{CO_2}, 76 mm Hg; HCO_3, 21.5 mmol $litre^{-1}$; Na^+, 136 mEq $litre^{-1}$; K^+, 4.0 mEq $litre^{-1}$.

Immediately, her airway was secured with an endotracheal tube and ventilation assisted. She was shifted to our neurology intensive care unit (ICU) for further management.

In the ICU, as her arterial pressure continued to remain low, dopamine was started and maintained in the range of 5–8 $\mu g\ kg^{-1}\ min^{-1}$ to target mean arterial pressure of

80 mm Hg. Her GCS improved with the institution of haemodynamic and respiratory support and dopamine was gradually tapered off during the next 2 days.

On admission to ICU, lumbar puncture at L3/4 level revealed normal opening pressure and drained xanthochromic fluid. Sugar was 51 mg dl^{-1}, protein 200 mg dl^{-1}, red blood cells 115 ml^{-1}, and white blood cells 0. Cerebrospinal fluid cryptococcal antigens, venereal disease research laboratory test, and acid fast bacilli were negative. Viral markers for hepatitis B virus antigen (HBsAg), hepatitis C virus, and human immunodeficiency virus were also negative. Antibody to double stranded DNA (anti-dsDNA) was negative. Her coagulation tests including bleeding time, clotting time, prothrombin time, activated partial thromboplastin time, and platelet counts were normal. Magnetic resonance imaging (MRI) revealed cord signal changes (suggestive of demyelination) extending from cervicomedullary junction to D5 level and a posterior epidural haematoma extending from C4–C7 (Fig. 1). T2-weighted images revealed hyperintense signal changes in medulla. Constructive images in steady-state images showed no flow void. No enhancement was seen within the cord. There was evidence of small specks of haemorrhage within the cord.

Her neurological deficit remained non-progressive over the next 2 weeks. She remained quadriplegic with a sensory level at C_2 dermatome. Neurosurgeons opinion was taken regarding evacuation of haematoma. It was suggested on seeing the MRI that the haematoma had been organized and was not expanding. It would have probably resolved. Since there was a long window period between the occurrence of the complication and the patient coming to our hospital, the damage to the cord had already occurred. Surgery was considered to not benefit the patient.

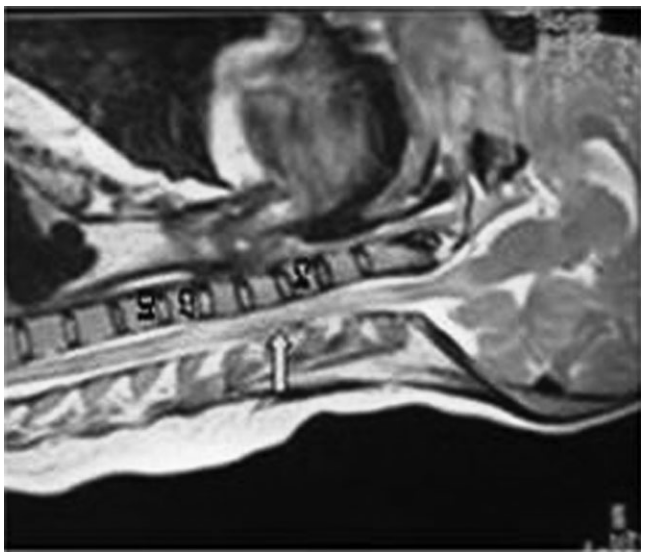

Fig 1 T2-weighted saggital MRI showing cord signal changes from cervicomedullary junction to dorsal vertebrae with posterior epidural haematoma extending from C4 to C7 (white arrow). C4, fourth cervical vertebra; C7, seventh cervical vertebra; D1, first dorsal vertebra.

She continued to require assisted ventilation (synchronized intermittent mandatory ventilation) in view of her poor respiratory efforts. Anticipating prolonged ventilation, a tracheostomy was done and symptomatic and supportive care provided. The neurological status remained unchanged at the end of 45 days.

Discussion

Quadriplegia presenting in the immediate post-partum period is a rare occurrence, particularly in the absence of known predisposing conditions such as pre-existing coagulopathies, anticoagulant therapies, vascular malformations, arteritis, eclampsia or iatrogenic causes such as spinal and epidural injections for labour analgesia or Caesarean section. SSEH is also a rare occurrence during pregnancy with only six cases reported in literature since 1900.[2–6] However, none of these patients presented with associated myelitis. There was no history or signs of trauma on the neck or cervical spine area.

All possible causes of an epidural haematoma were ruled out by investigations in our patient. The progression of the event beginning with localized radicular pain in the neck and arms progressing to motor weakness in the limbs and sensory deficit over a period of 2 days was consistent with the diagnosis of an epidural haematoma/haemorrhage within the cord. The location of the epidural haematoma, that is, in the cervical cord and extending to the brain stem could explain the respiratory compromise and haemodynamic instability associated with the presentation. The aetiology of SSEH remains obscure; there is controversy whether the bleeding is arterial or venous in origin. Carroll and colleagues[2] and Yonekawa and colleagues[3] had hypothesized that SSEH in pregnancy was caused due to the rupture of a pre-existing pathological venous wall in the presence of elevated intra-abdominal pressure. Beatty and colleagues[7] had hypothesized that haemorrhage in cervical and upper dorsal cord was arterial in origin. They concluded that bleeding from a low-pressure venous system could not expand so rapidly and cause compression. They held altered hormonal milieu of the pregnancy responsible for changes in the vessel wall and ligaments leading to the haematoma. Spontaneous rupture of other arteries, namely splenic, coronaries, and vertebral, have also been attributed to high levels of oestrogen and progesterone in peripartum period; however, the hormonal expression is highly variable.[8] We hypothesize that in the absence of any other known predisposing conditions, the haematoma could have been precipitated by the rupture of a weakened epidural artery, possibly by some innocuous trauma to the cervical region which initially went unnoticed by the patient.

Multiple sclerosis (MS) and acute transverse myelitis (ATM) are the two common causes of demyelination in the peripartum period leading to acute onset muscle

weakness.[1] It has been observed that demyelination of the cord associated with MS shows flare-ups in the first 3 months post-partum.[9] Various theories have been put forward for the demyelination, and most attribute them to immuno-modulatory effects of pregnancy on cytokines, chemokines, and T-cell activity.[10 11] It is likely that acute onset demyelination in our patient could have been precipitated by the immuno-modulatory effects of altered hormonal milieu of the post-partum period.

In summary, we report the development of acute onset quadriplegia in a 24-yr-old previously healthy woman associated with respiratory failure. This was possibly due to SSEH and the associated demyelination of brain stem and spinal cord.

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