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Case Report

A Curious Case Of Encephalopathy In A Post Partum Female

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ABSTRACT

Autoimmune encephalitis is an uncommon cause of altered sensorium in the post partum period and is usually diagnosed with an autoimmune panel. This case reports seeks to highlight the entity of an autoimmune panel negative encephalitis which can present with certain atypical features as in our case. A 29 year old female presented to us 8 days post partum with fever and altered sensorium. After ruling out the common causes of encephalopathy post partum an MRI brain was done which showed a demyelinating lesion in the right internal capsule and an MRI spine showed a hyperintensity in the cervical spine. CSF showed low glucose but a negative biofire initially with a similar picture in the repeat sample. Repeat MRI showed extension of the lesion in the cervical cord. Autoimmune panel was negative but steroids were started. She showed an excellent response to steroids and her sensorium improved. She was discharged home on oral steroids, and the demyelinating lesion had resolved on the repeat MRI after discharge. The response to steroids and the resolution of the lesion in our case suggest a high possibility of autoimmune panel negative encephalitis . When all other causes are ruled out and even when the autoimmune panel is negative, the possibility of autoimmune encephalitis cannot be ruled and may be present in upto 44 % of such cases.

Keywords: Steroids, autoimmune encephalitis, demyelinating lesion

Introduction

Encephalopathy, a disorder affecting the structure and function of the brain, typically presents a myriad of challenges for healthcare professionals. However, the postpartum period, known for its inherent physiological changes, adds an additional layer of complexity to this enigma. The immediate postpartum phase, marked by hormonal fluctuations, vascular adaptations, and immunological shifts, creates a delicate interplay that demands vigilant attention. When encephalopathy manifests in this context, it raises critical questions about potential causative factors, ranging from obstetric complications to autoimmune responses. Understanding the intricate interconnections between the peripartum period and neurological dysfunction is paramount for unraveling the mystery at hand. This case not only poses a diagnostic challenge but also underscores the need for a comprehensive approach that considers the unique physiological landscape of the postpartum state.

Case description

A 29 year old female presented to us with history of fever, non-projectile vomiting and altered sensorium for 3 days. She had also complained to her relatives about headache and mild blurring of vision the day before. She was undergone an emergency lower uterine caesarian section under spinal anaesthesia 8 days ago in view of non progression of labour, which was uneventful from an anaesthetic and surgical point of view. She was

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discharged the next day with oral antibiotics. She had a past history of Pulmonary Tuberculosis 11 years ago, after which she finished 6 months of anti-tubercular therapy and was declared cured. She did not have any other significant past history. On arrival to the Emergency department, patient was haemodynamically stable and her ABG showed high lactates with a compensated metabolic acidosis. She was shifted to our ICU for further management. A working diagnosis of hospital acquired infecion was made and she was prophylactically started on Meropenem and Teicoplanin. On examination of her Central nervous system, there were no localising or long tract signs. CT scan of brain did not reveal any significant abnormalities. A USG of her abdomen only showed a bulky involuting uterus. MRI showed a subacute ischemia of the right corona radiata. MR Venogram showed focus of diffusion restriction in right posterior limb of internal capsule extending upto periventricular centrum semiovale white matter suggestive of subacute ischaemic changes but without any thrombosis. VEP study was suggestive of bilateral demyelinating type of reticulo-optic pathway dysfunction. EEG tracing suggested a diffuse non-specific encephalopathic process. Infective causes were ruled out. A lumbar puncture was planned and done the next day which showed 150 cells, 83 mg/dl proteins, 40 mg/dl glucose and a negative ADA. Gene expert and CSF biofire were negative. Thrompophilia screen was sent and Lupus anticoagulant was found to be positive. Cryptococcal Antigen, Anticardiolipin antibodies, Leptospira and Borriela IgG IgM and Scrub IgM were all found to be negative. ANA screening and autoimmune encephalitis panel were also negative. Carotid Doppler and Holter monitoring were done and did not reveal any significant findings. ATTs and IV Methylprednisolone at 40 mg OD were started. ATTs were subsequently withdrawn as CSF Gene Expert was negative. A repeat lumbar puncture was done 3 days following the first one and showed protein of 113 mg/dl and glucose of 40 mg/dl. A repeat MRI brain and MRI whole spine were done. MRI brain showed a diffuse symmetrical white matter hyperintensity in FLAIR, T2 AND DWI sequence, extending from the bilateral centrum semiovale extending down to the posterior limb of internal capsule. MRI spine showed a T2/STIR hyperintensity in the cervical cord, mainly at the anterolateral aspect and the cervical cord was found to be thickened and edematous. EEG done on the same day showed a diffuse non-encephalopathic process. Within 24 hours of starting Methlprednisolone, she started improving symptomatically. Her sensorium started improving and she started obeying commands. IV Steroids were continued for 5 days. She was discharged subsequently on tapering doses of oral steroids and was asymptomatic on follow up. Her MRI after 1 week of discharge had shown a resolution of the demyelinating lesion.

Discussion

The common causes of altered sensorium post partum include post-partum haemorrhage, pre-eclampsia, cerebral venous thrombosis and infections. Initially in our patient a provisional diagnosis of sepsis was made and empirical antibiotics were started. As per shields et al. altered mental status is one of the early signs of sepsis and provides a high index of suspicion.[1] However, it was ruled out due to a low procalcitonin value and lack of other parameters of sepsis. Our work up aimed to rule out the more common causes first. Cerebral venous thrombosis was ruled out with a negative MR Venography and pre-eclampsia was ruled out due to absence of its characteristic features. According to a study by Duraipandi Manjubashini et al, Posterior reversible encephalopathy , cerebral venous thrombosis, and postpartum angiopathy were the three most common causes.[2] MRI of the brain, done after a normal CT showed a subacute ischaemic change of the right corona radiata and subsequent MRI showed a demyelinating lesion extending upto the right posterior limb of the internal capsule. As per Ke Qiu et al, in a study of demyelinating lesions occurring during pregnancy and postpartum a few of the common causes included acute disseminated encephalomyelitis, neuromyelitis optica spectrum disorders and other secondary CNS inflammatory demyelinating diseases.[3] Although we found a negative autoimmune encephalitis panel, upto 44% of such patients can have a negative panel.[4] In a study performed in Lady Hardringe Medical College, out of the 76 patients who had presented with neurological disorders in pregnancy were due to CNS infections out of which Tubercular meningitis was the most common cause.[5] Thus a Lumbar Puncture was urgently done to rule out an infectious aetiology. The CSF study

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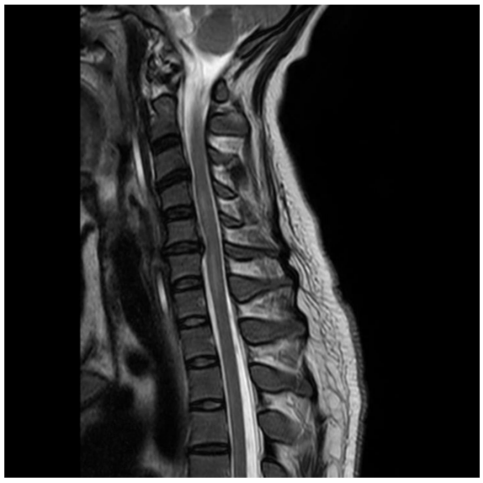
showed glucose of 40 mg/dl. One of the causes of hypoglycorrachia is bacterial meningitis as per Vandana Shrikhar et al which was ruled out by a negative CSF biofire.[6] In another retrospective study of 89 patients with hypoglycorrachia by Chow et al, the most common etiologies were bacterial and fungal meningitis, but non infectious etiologies comprised 33% of the cases.[7] The normal range of CSF glucose to serum glucose can range from 0.43 to 0.74.[8] At the time of checking the CSF glucose the serum glucose was 90 and hence the ratio was 0.44, which was within the normal range. Autoimmune encephalitis is typically associated with a normal CSF glucose.[9] Lyme's disease was another possibility due to the history and the nature of demyelination on the MRI. MD Gerard X Brogren described a case of cerebral vasculitis with a right thalamic infarct, which was similar to the imaging findings in our patient.[10] However in our case Borriela IgG and IgM was negative. As the patient had a past history of pulmonary tuberculosis, empirical Antitubercular therapy was started. Tubercular meningitis both intrapartum and postpartum has been described as by Sadie Namani et all who reported a case of tubercular meningoencephalitis in a pregnant female.[11] However Antitubercular therapy was withheld due as a negative CSF Gene Expert virtually ruled out a tubercular etiology. In two cases described by Ricardo Paquete Oliviera, patients had developed drug induced aseptic meningitis post spinal anaesthesia with hypoglycorrhachia in both the cases.[12] However the demyelinating changes on the MRI in our patient cannot be explained by the same. Repeat MRI done showed an extension of the demyelinating lesion. MRI findings of autoimmune encephalitis are variable and can provide a diagnosis challenge.[13] As we ruled out an infectious etiology we started steroids empirically considering a provisional diagnosis of autoimmune encephaltis. Within 24 hours of starting Dexamethasone 8 mg thrice daily she showed clinical improvement and completely recovered in 3 days. Steroids were then gradually tapered and she was asymptomatic on follow up. As per Christopher E Uy et al autoimmune encephalitis can have a variety of presentations, and have increase in incidence over the last decade.[14] The good response to steroids falls in line with our provisional diagnosis of an autoimmune panel negative autoimmune encephalitis. Several cases of anti NMDA receptor mediated encephalitis have been described in the postpartum period.[15] This is possibly a case of autoimmune panel negative encephalitis in the post partum period though we could not find any association between the same and the low CSF glucose.

Conclusion

The response to steroids and the resolution of the lesion in our case suggest a high possibility of autoimmune panel negative encephalitis. When all other causes are ruled out and even when the autoimmune panel is negative, the possibility of autoimmune encephalitis cannot be ruled and may be present in upto 44 % of such cases.



MRI Cervical Spine showed a T2 hyperintensity extending down the cord

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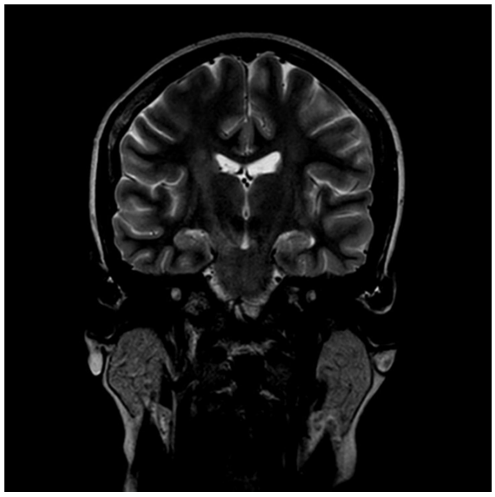

MRI flair showing increased signal intensity at caudal aspect reaching upto the midbrain and pons

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