

# Postpartum Complicated by Transverse Myelitis

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## ABSTRACT

*Transverse myelitis is a very rare neurological condition associated with immunologic and infectious conditions causing interruption of the neuroanatomical pathways in a transverse plane in the spinal cord. Herein is described the fatal case of a patient that developed transverse myelitis after a Caesarean delivery, probably related to the epidural analgesia.*

**Keywords:** Epidural, pregnancy, postpartum, transverse myelitis

# Postparto Complicado por Mielitis Transversa

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## RESUMEN

*La mielitis transversa es un trastorno neurológico poco común, asociado con problemas inmunológicos e infecciosos que causan interrupciones de las vías neuroanatómicas en el plano transversal de la médula espinal. Aquí se describe el caso fatal de una paciente que desarrolló mielitis transversal luego de un parto con cesárea, probablemente en relación con la anestesia epidural.*

**Palabras claves:** Epidural, embarazo, postparto mielitis transversa

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## INTRODUCTION

Transverse myelitis (TM) is a very rare condition, with an annual incidence of 1.3 to 8 cases per million. This condition is usually associated with immunologic and infectious processes that can trigger the disease (1).

Pregnancy, by itself, is characterized by immunological changes such as increase in the secretion of interleukin-10 that downregulate the production of B-cell immunity, making the patients prone to autoimmune conditions like lupus or neuromyelitis optica. In most of the cases, the aetiology of transverse myelitis remains a mystery, but there are some reports of the association of infections or the use of neuroaxial anaesthesia (1, 2).

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## CASE REPORT

A 27-year old female, one-week post-Caesarean delivery, arrived at the hospital emergency room complaining of severe pain in the right lower extremity with no paraesthesia or changes in muscular strength. An initial workup for deep venous thrombosis by Doppler ultrasound was negative. The patient was reassured and was discharged home on analgesics. Three days later, she returned to the emergency room due to increased severity of pain in the same extremity and a new onset of dyspnoea with chest pain. Her medical history was remarkable for a recent Caesarean delivery done under combined spinal/epidural anaesthesia. On physical examination she was morbidly obese, her vital signs were remarkable for temperature of 101 F (38°C), pulse 84 beats/minute, respiratory rate 45/minute, oxygen saturation 91% and blood pressure 158/83 mmHg. On auscultation, she had scattered bilateral diffuse crackles and scattered bibasilar rales. Her abdomen was soft and nontender with the Caesarean scar well healed. Her extremities had mild bilateral pedal oedema. On neurological examination, she was alert and oriented to time, person and place with marked deep tendon reflexes in the right lower extremity. A head and chest computed tomography (CT) was done, with no signs of intracranial

haemorrhage or midline shift. The chest CT reported diffuse ground-glass appearance suggesting pulmonary oedema and no signs of pulmonary embolism.

The laboratory results at the time of admission showed haemoglobin 9.7 g/dL; haematocrit, 29.5%; white blood cell (WBC),  $12.5 \times 10^9$  and platelets  $254 \times 10^9$ . D-dimer was 7. Sodium was 139 mmol/L, potassium 3.6 mmol/L, chloride 104 mmol/L, bicarbonate 19, blood urea nitrogen (BUN) 13 mmol/L, creatinine 0.7 mmol/L, blood glucose 85 mmol/L, and calcium 8.4 mmol/L. Lactic acid was 1.3. Liver function tests revealed: total protein 6.5 g/dL, albumin 3.1 g/dL, alanine aminotransferase (ALT) 59, aspartate aminotransferase (AST) 42, alkaline phosphatase 150  $\mu$ , total bilirubin 0.6 mmol/L and conjugated bilirubin 0.2 mmol/L.

The patient was admitted with the diagnosis of pulmonary oedema due to severe pre-eclampsia. She was started on ceftriaxone and azithromycin as prophylaxis recommended by the infectious disease specialist and she required ventilatory support. Due to the elevated blood pressure and the findings suggestive of pulmonary oedema on the CT, a 24-hour urine collection was done considering pre-eclampsia. The determination of proteinuria was 200 mg, ruling out severe pre-eclampsia. An echocardiogram showed ejection fraction of 57.11%. The patient required full mechanical ventilation support with sedation due to respiratory distress 24 hours later. She continued to spike fever in the ranges of 39°C and 40°C, with blood cultures, bronchial lavage, urine and cerebrospinal fluid cultures being negative. Rapid plasma reagin (RPR) and influenza (A, B) rapid tests were performed. Serology for autoimmune diseases (anti DNA, anti Smith, SSA, SSB, Scl-70) were all negative.

Her white blood cell count increased to  $19.4 \times 10^9$  and the infectious diseases specialist recommended the addition of ciprofloxacin, vancomycin and oseltamivir for possible influenza. The patient underwent a second lumbar puncture which showed that the cerebrospinal fluid (CSF) was colourless, and blood cultures and gram stain were negative. There was increase in protein at 115 g/dL, glucose 90, lactic acid 65, CSF index 0.65, CSF IgG: 5.1 mg/dL, CSF albumin: 7.2 mg/dL, CSF IgG/albumin: 0.12, serum IgG/albumin: 0.2, CSF IgG synthesis rate: 4.5 mg/24 hours. Her blood pressure was normal.

The sedation was stopped after her respiratory parameters improved, but the patient was unable to be weaned from the ventilator because she was found to be quadriplegic. A full neurological examination revealed loss of muscular strength and sensation below the neck, with absence of deep tendon reflexes and no bowel or bladder control. Despite the previous findings, she was able to appreciate vibration of the tuning fork in both elbows and knees and as a remarkable finding the patient was complaining of “banding like pressure around her trunk”. An ophthalmologic evaluation found bilateral optic disc hyperaemia with no areas of haemorrhage and obscured peripheral vessels in the nasal

fields of both eyes, consistent with papillitis. There were no other signs of ophthalmic involvement.

Since the patient was sedated and it was impossible to determine the progression of the paralysis the neurology service started intravenous steroids suspecting Guillain-Barre syndrome. Magnetic resonance imaging (MRI) studies showed an extensive lesion of the spinal cord, which enhanced with gadolinium on T1 weighted and had high signal intensity on T2 weighted sequences, suggestive of a transverse myelitis with acute inflammation of the cord. The patient persisted with fever and her muscular strength deteriorated and she had a cardiac arrest and could not be resuscitated.

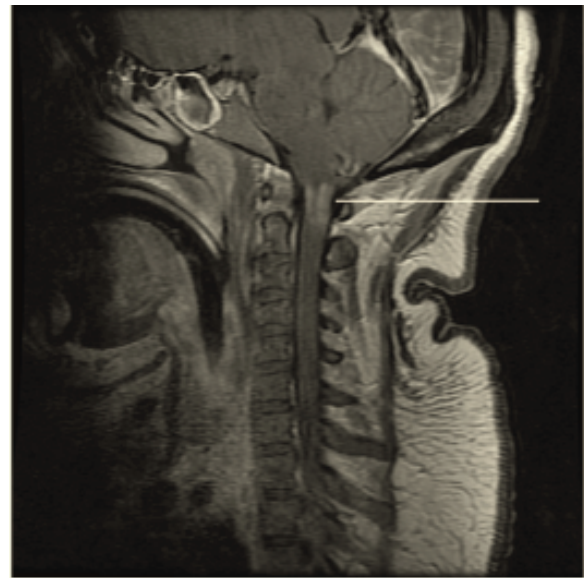


Fig 1: Sagittal plane, T2 weighted sequence images showing widespread inflammation of the spinal cord causing gadolinium-enhancing signal abnormality at the level of the medulla oblongata and C1.

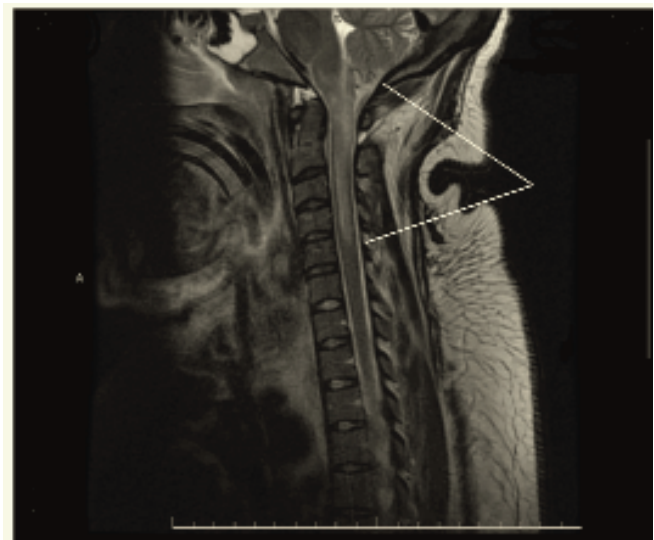


Fig 2: T2 weighted image sequence, showing an area of intramedullary high signal intensity at the level of C5 extending to the pons.

## DISCUSSION

Defining transverse myelitis is probably easier than determining the aetiology. Transverse myelitis is characterized by an inflammatory demyelinating disease that produces a disruption in ascending or descending neuroanatomical pathways in a transverse plane of the spinal cord; producing focally motor, sensory and autonomic dysfunction with clinical findings directly related to the level of the anatomic lesion (3).

Thirty per cent of all the cases of TM are defined as idiopathic, most of the common causes arise from an autoimmune mediated phenomenon as in paediatric cases of TM where up to 60% of the cases are associated with vaccines, or in adults as a result of an infectious process or the presence of concomitant autoimmune demyelinating process like Devic's disease or multiple sclerosis (4).

The use of neuroaxial anaesthesia has been associated with TM, as is reported in a few cases described in the literature. This is based on the fact that the patients with damaged demyelinated fibre are more susceptible to the neurotoxicity of the anaesthetic drugs (5).

Once the clinical picture of myelopathy is established, imaging studies are recommended to determine if there is any recognizable factor like compression, malignancy, vascular or metabolic factors. The use of MRI is the preferred mode of assessment and it needs to be performed along the length of the spinal cord. The typical lesions associated with TM are characterized to span at least two vertebral segments, visualized on MRI. This modality also helps to delineate if there is any source of infection or injury related to invasive procedures like the use of epidural analgesia as in our case (4).

The use of serum markers like the autoantibody marker NMO-IgG which targets the astrocytic water channel, aquaporin – 4, is specific for neuromyelitis optica and has no role in plain TM (1).

The use of corticosteroids is the standard first line of treatment, however, due to the rarity of the disease, there are

no well-designed studies evaluating its use, but the reason why they are used is because of the extrapolation of the studies using corticosteroids in patients with multiple sclerosis. The usual doses are 1000 mg of methylprednisolone daily, for three to five days (2, 3). Plasma exchange is a more aggressive therapy that is reserved for patients not responding to the use of steroids and can be considered as experimental and is related to severe complications including hypotension, electrolyte imbalance, coagulopathy, thrombocytopenia and catheter-related thrombosis. It should be reserved only for cases refractory to steroids (3).

The prognosis for TM is usually obscure, but 50–70% of the patients may have a partial recovery, but the rest usually will have severe sequels or can even die of complications related to ventilation or poor mobilization as happened with our patient (1, 3).

The diagnosis of TM and the aetiology is challenging, in our case all the cultures and the serology for infective agents were negative, along with the serology for autoimmune diseases. As a matter of exclusion, the TM was attributed to the use of regional analgesia.

Further research is required to clarify the obscure aetiology of this disease and the relation with regional analgesia.

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