

# Neurogenic shock due to transverse myelitis with Landry's acute flaccid paralysis in a child

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## ABSTRACT

Neurogenic shock is a life-threatening condition mostly associated with cervical and high thoracic spine injury, whereas transverse myelitis leading to neurogenic shock is extremely rare. An 11-year-old boy was admitted to our center with Landry's acute flaccid paralysis and urinary retention, which led the patient to severe respiratory distress. The test indicated a high level of protein and white blood cells in cerebrospinal fluid. He was diagnosed with transverse myelitis and then treated with methylprednisolone. The patient developed hypotension (63/45 mmHg), bradycardia (60 beats per minute), warm flushed skin, and decreased consciousness as a result of neurogenic shock. Fluid resuscitation, noradrenalin, and atropin were indicated, and the shock was well controlled. This is the first case of neurogenic shock due to transverse myelitis with Landry's acute flaccid paralysis in a child we have experienced so far. It highlights that neurogenic shock should be considered in patients with acute transverse myelitis with Landry's acute flaccid paralysis.

**Keywords:** shock, transverse myelitis, Landry's paralysis

## INTRODUCTION

Neurogenic shock is a life-threatening condition characterized by loss of sympathetic tone due to acute spinal cord or central nervous system injury [1]. The sudden loss of autonomic tone cause decreased systemic vascular resistance and vasodilation. The clinical presentations of this shock are commonly hypotension with wide pulse pressure, normal heart rate, or bradycardia [2].

There are various causes of neurogenic shock, including cervical spine injury, spinal anesthesia, Guillain-Barre syndrome, and transverse myelitis [3-9]. While neurogenic shock is mostly associated with cervical and high thoracic spine injury, neurogenic shock occurring from transverse myelitis is extremely rare [1,2,4,8]. Thus, along with the management of neurogenic shock, early diagnosis and treatment of the etiology should always be considered.

Acute transverse myelitis is a rare acquired neuro-immune spinal cord disorder that can manifest

with the rapid development of motor and sensory deficits at any spinal cord level [10-12]. Its incidence is estimated to be around 2 per million children annually [11].

Currently, there is not any report of neurogenic shock caused by transverse myelitis. Therefore, we present a case of neurogenic shock due to transverse myelitis with Landry's acute flaccid paralysis in a child.

## Case report

A previously healthy 11-year-old boy was admitted to the Pediatric Intensive Care Unit in Hue Central Hospital with a history of high fever (40°C), a headache, and acute progressive lower extremity weakness for four days. At the time of admission to our center (day one), the patient had a mild fever, chills, and weakness in both legs. He was conscious with a Glasgow Coma Scale (GCS) of 15. He did not complain of the headache anymore. Neither vomiting nor stiff neck signs were shown. On neurologic

examination, a complete flaccid paralysis was revealed in both lower extremities, whereas muscle power in both hands and forearms was normal. Sensory testing showed hypesthesia when touching his trunk and all four limbs. The deep tendon reflexes were absent in her patellar and ankles bilaterally. Babinski's reflexes were negative in both lower extremities, and urinary control was normal.

Laboratory findings revealed: white blood cell count 10.27 K/ $\mu$ l, hemoglobin 12.9 g/dl, platelet count 212 K/ $\mu$ l, procalcitonin 0.14 ng/ml. Blood glucose, urea, creatinine, AST, ALT, and electrolytes were within the normal range. Cerebrospinal fluid (CSF) tests showed a white blood cell count of 300/mm<sup>3</sup> (leukocytes were predominant at 70%), an elevated concentration of protein (0.89 g/l), and a normal concentration of glucose (4.07 mmol/l). Routine culture and Gram staining of CSF were negative. The result of the blood culture was negative. Herpes simplex virus, Japanese encephalitis tests were negative. A cardioechography was performed, and the results were normal. The previous computed tomography (CT) scan of the brain did not show any abnormalities.

The patient then developed urinary retention on day two, which required the placement of a foley catheter. He was diagnosed with transverse myelitis due to these findings and treated with methylprednisolone 30 mg/kg/dose once daily.

The patient had planned to perform a magnetic resonance imaging (MRI) of the spinal cord. However, the condition quickly worsened as a result of Landry's acute flaccid paralysis. Flaccid paralysis began to affect the upper extremities (muscle power 2/5) and the respiratory muscles, which led to shortness of breath and then severe respiratory distress. He also was drowsiness (GCS: 10). Intubation was indicated when the oxygen saturation (SpO<sub>2</sub>) was <85%, and the patient underwent invasive synchronized intermittent mandatory ventilation. Simultaneously, the patient developed a shock with hypotension (63/45 mmHg), bradycardia (60 beats per minute), and flushed warm skin.

A diagnosis of neurogenic shock was established after ruling out all other possible causes like sepsis, cardiogenic shock, and hypovolemic shock. The patient was immediately treated with fluid resuscitation, noradrenalin, atropine, and the shock was well controlled within 5 hours. The fever and chills disappeared on day three, and his hemodynamics was stable. His consciousness gradually improved. However, he still required ventilation in spontaneous mode.

Because of the limitation of MRI in patients with mechanical ventilation, he underwent a spinal CT scan instead of an MRI. The CT report did not show spinal inflammation but ruled out common causes of spinal cord injury such as trauma and tumors.

After five days of methylprednisolone therapy, steroid intake was changed to oral administration. The urinary control returned to normal. Muscle power improved in the upper extremities, but his breathing did not meet the expectation, and the muscle power of the lower extremities did not improve significantly. Follow-up blood test components were within the normal range, and CSF results returned to normal.

## DISCUSSION

Neurologic shock, a rare disorder in adults and children, has been described as a sudden disruption of the sympathetic nervous system [1-4]. In the United States of America, traumatic spinal cord injuries affect between 8,000 and 10,000 persons each year. The Trauma Audit and Research Network discovered 490 isolated spinal cord injuries in a survey of isolated spinal cord injuries. Of these, only 19.3 % of individuals suffered from neurogenic shock [4].

The causes of neurogenic shock include spinal cord injury, Guillain-Barre syndrome, and transverse myelitis [1]. Neurogenic shock has been reported in children with trisomy 21, skeletal dysplasia, and tonsillopharyngitis in the pediatric population. In a trauma patient, neurogenic shock is an exclusionary diagnosis. Hemorrhagic shock is the most common cause of hypotension, according to The American College of Surgeons' Advanced Trauma Life Support. After being adequately controlled, neurogenic shock should be evaluated [2]. This is the first case of neurogenic shock due to transverse myelitis with Landry's acute flaccid paralysis in a child we have experienced so far. Although MRI findings and cerebrospinal fluid immunoglobulin investigation were lacking, a diagnosis of transverse myelitis was confirmed based on the clinical presentation and CSF results. After ruling out meningitis, encephalitis, or Guillain-Barre syndrome.

In this case, we were able to diagnose neurologic shock after excluding other types of shock. Firstly, we excluded hypovolemic shock because the patient did not manifest severe hemorrhage or plasma loss via urine or burn. Secondly, the patient had no history of allergies, cutaneous symptoms (e.g., sudden onset of angioedema, flushing, pruritus), or respiratory symptoms (e.g., stridor, wheezing, retractions). We also ruled out cardiogenic shock because of the lack of clinical manifestation or reduced cardiac function on echocardiography. In addition, the patient presented with hypotension, bradycardia, and progressive respiratory failure at the same time. As a result, we diagnosed neurologic shock and initiated treatment for this type of shock. Moreover, pulse methylprednisolone was also initiated intravenous-

ly for five days before being changed to oral steroid treatment. There was a rapid improvement of the clinical presentation within five days. This patient's blood pressure was maintained at 85-90 mmHg, and his muscle power gradually improved in the following days.

Neurogenic shock treatment aims to normalize the heart rate and blood pressure. Three popular therapies for neurogenic shock are discussed below [1-3]. Firstly, intravenous fluid therapy is the most common treatment for low blood pressure. It helps keep blood pressure in check by replenishing fluid levels in veins. Secondly, vasopressors can help tighten blood arteries and raise blood pressure if intravenous fluid therapy is ineffective. Norepinephrine, phenylephrine, dopamine, and epinephrine are some of the most widely utilized vasopressors. Thirdly, atropine may be indicated if the heart rate is too low. By blocking parasympathetic activity, it aids in the normalization of heart rate.

The American Association of Neurological Surgeons and the Congress of Neurological Surgeons' Guidelines for the Management of Acute Cervical Spine and Spinal Cord Injuries also recommend that mean arterial blood pressure should be kept between 85 and 90 mmHg for the first seven days after a spinal cord injury [13]. In our case, along with the treatment of transverse myelitis, we managed the neurogenic shock with fluid resuscitation, noradrenalin, and atropine. Therefore, the prelimi-

nary outcomes were favorable. After five days of methylprednisolone therapy, steroid intake was changed to oral administration. The urinary control returned to normal. Muscle power improved in the upper extremities, but his breathing still did not meet the expectation, and the power of the lower extremities did not significantly change. Follow-up blood test components were within the normal range, and CSF results returned to normal. These changes in clinical manifestation and CSF results strengthened the diagnosis of transverse myelitis.

The patient required long-term treatment with medications and physical therapy.

Our study has some limitations. The patient did not undergo an MRI of the spine, and cerebrospinal fluid immunoglobulin G (IgG) index test was not performed. Nevertheless, these investigations are significant to prove the inflammatory process of the spinal cord in transverse myelitis.

## CONCLUSION

Neurologic shock occurring from transverse myelitis should be considered in the case of Landry's acute flaccid paralysis. Administration of vasopressors and inotropic agents is the primary treatment for neurogenic shock. Along with managing neurologic shock, early diagnosis and treatment of transverse myelitis essentially contribute to improving outcomes.

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