Traumatic lumbar hematomyelia causing spinal shock: A case report


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Abstract

Background: Spinal cord hemorrhage or hematomyelia can arise from trauma or non-trauma causes, adverse effect of anticoagulation therapy and ruptured vascular malformation. Bleeding in epidural space is common and complications such as acute spinal cord syndrome can arise due to the compression and destruction of the spinal cord. Symptoms of hematomyelia might be varied depending on location of the lesion, often accompanied with acute radicular pain. In this case study, we reported a case of spinal shock after traumatic lumbar hematomyelia.

Case: A 13-year-old girl presented to Dr. Moewardi General Hospital with inferior paraparesis, bladder and bowel incontinence, a day after falling on her glutes. Physical examination revealed reduced motoric and sensory function below 12th thoracic dermatome, with no patellar and reduced Achilles reflexes. Conventional X-ray was unremarkable and further investigation with MRI showed a hematoma on thoracal 10 to 12 vertebrae. Motor and sensory functions were improved after 12 days administration of corticosteroids and further improvement was seen almost immediately after decompression surgery.

Objective: To describe a case of spinal shock caused by traumatic lumbar hematomyelia

Method: This is a case report study describing spinal shock which improved after administration of corticosteroid and decompression surgery.

Conclusion: Spinal shock could be caused by traumatic lumbar hematomyelia, which is characterized by loss of motor, sensory, and bladder function, and decompression surgery improved the prognosis.

Keywords: case report; spinal shock; traumatic hematomyelia

Introduction

Spinal cord hemorrhage or hematomyelia can arise from trauma and/or non-trauma causes, adverse effect of anticoagulation therapy and ruptured vascular malformation (Akpınar et al., 2016; Berkowitz, 2022).

Symptoms of hematomyelia may be varied depending on location of the lesion, often presented with acute pain with radicular radiation (Claro et al., 2018). Clinical manifestations of hematomyelia are sensory problems, paralysis, and autonomic dysfunction (Sabouri et al., 2022). Spinal shock is a transient extinction of reflexes and muscle tone below the level of injury. Spinal shock was reported on 14-50 cases per million annually worldwide (Ziu & Mesfin, 2022) and can be caused by compression and destruction of the spinal cord following hematomyelia (Akpınar et al., 2016). Shaban et al. (2018) suggested that decompression through surgical procedure is considered as the definitive treatment for hematomyelia. Through this case report, we present a case of a 13-year-old girl with hematomyelia showing clinical improvement after surgery and oral medications.

Case Presentation

A 13-year-old girl presented to the Neurology Clinic, Dr. Moewardi General
Hospital, Surakarta with inferior paraparesis after falling on her gluteus. Despite the pain, she was initially still able to walk. However, a tingling sensation was felt on both her legs right after the fall and, on the following day, she started to have inferior paraparesis, and she could not stand up, and was only able to move her legs. She also had bladder and bowel incontinence the day after the incident. She was brought to hospital, hospitalized for three days, and was referred to Dr. Moewardi General Hospital.

She had no previous history of malignancy, blood and coagulation disorder, infection, or prior blood-thinner medication.

**Intervention or Clinical Examination**

**Ethical consideration**

To obtain information regarding this case, written informed consent was obtained from the patient. The copies of the written consent were available.

**Clinical Findings**

On the physical examination, she was awake, alert, and oriented with Glasgow Coma Scale of E4V5M6 with normal vital signs. Meningeal signs and cranial nerve were unremarkable. She had inferior paraparesis with motor strength of 333 on the right leg and 111 on the left leg. We used Manual Muscle Test ranging from 0-5 with this patient, 3 being able to do active movements against gravity and a score of 1 for flicker or traces of contraction. Muscle tone in all extremities was normal. We also observed reduced pain and light touch sensation below the level of thoracal 12 (Th12) spinal cord. Proprioceptor sensation was reduced in her left leg, but preserved in her right leg. No patellar reflexes were found during examination and the Achilles reflexes were reduced on both lower extremities. No pathologic reflexes were found.

During neurological examinations, we revealed some degree of hypoesthesia on the Th12 level and below, which was secondary to hemorrhage on level Th 10-12. According to the Frankel and Asia Scale, her left leg was classified into class B or sensory only, where some sensory sensation was still present below the lesion. The right leg had more than 50% of muscle strength more than grade 3 which was classified into class D (Bennett et al., 2022; van Middendorp et al., 2011).

**Laboratory and Imaging Findings**

Hematology laboratory results were unremarkable with coagulation parameter and leukocyte parameter within normal range, excluding both coagulation and infection etiology. Conventional thoracolumbar X-ray was performed and showed no fractures or mass compressing the spinal cord (Figure 1).

Magnetic resonance imaging (MRI) was then performed (Figure 2) and showed irregular intramedullary lesion at thoracal 10 to 12 spinal cord which appeared hyperintense in T1W1, but hypointense in T2W1 and T2STIR, indicating intramedullary spinal cord hemorrhage.

**Results**

After admission to the hospital, she remained under observation for several days to see the progression of the disease and was scheduled for decompression surgery. She was observed to keep thirty degrees head up position, stretch out every two hours to prevent decubitus ulcers and lower
the risk of falling. Indwelling foley catheter was used due to bladder incontinence. During the observation, her ability to move her leg improved slightly which might be due to the reabsorption of the hematoma and passing of the acute spinal shock phase. Her right leg had a motoric function of 111 and improved up to 333 in 10 days. Dexamethasone 10 mg b.i.d. was administered for two days and later switched to methylprednisolone 125 mg q.i.d. for three days. Methylprednisolone was then tapered off every three days and switched to oral after a total administration for 12 days. She was then scheduled for decompression surgery. Explanation about the surgery was given before the surgery to the patient and her family. Following the surgery, she was given oxygen via nasal cannula, while her breathing pattern and saturation were monitored. Yellow hand band was also given to inform others of her risk in falling which was calculated from the Morse Fall Scale as 47 and she had a high risk of falling. Her motoric ability improved to 333 on her left leg. Patient was sent home after 15 days as inpatient.

Discussion

Hematomyelia or spinal cord hemorrhage is considered as a rare etiology for post-traumatic neurology deficit cases with limited literature (Gahlot & Elhence, 2020). The epidemiology of neurogenic shock is difficult to assess as it is still unknown how hemorrhagic shock and other injuries impact the hemodynamic effects of spinal cord injury. The global incidence of traumatic spinal injury is estimated around 768,473 annually worldwide, which is higher in low and middle income countries compared to high income (Kumar et al., 2018). Trauma Audit and Research Network identified that, from 490 isolated spinal cord injuries, 19.3% had spinal shock (Dave, 2023). For every one million people, t 30-40 had spinal shock, around 8000-10000 cases per year in Indonesia (Soertidewi et al., 2006).

Hematomyelia mostly occurs due to vascular malformations, vasculitis, hypo coagulation disorders, or trauma (Ramazanov et al., 2021). Intramedullary hemorrhage is rare in children compared to epidural hematomas (75%), subarachnoid hemorrhage (15%), and subdural spinal hematomas (4.1%) (Gahlot & Elhence, 2020; Nunes et al., 2016). Cervical spine is the typical location for traumatic hemorrhages in children, whereas it usually occurs in thoracic and lumbar spine for adult cases. Nonetheless, the locations may vary from patient to patient. In this case, the hemorrhage was in the lumbar area despite the patient being a 13-year-old girl. The hemorrhage occurred without compression or fracture from the trauma. Acute bleeding happened after trauma in the microcirculation of the spinal cord, since large vessels are usually spared due to their higher amount of collagen and elastin within its wall (Shaban et al., 2018).

Manifestation of hematomyelia depends on the level of spinal cord affected, which usually appears abruptly (Ramazanov et al., 2021). In this case, her chief complaint was loss of ability to move her lower extremities, primarily in the left leg, with some sensory sensation loss defined as incomplete paraplegia. The differences of neurological deficit in both legs might be caused by the hematoma which was more extensive in the left side of the leg. Another prior case of traumatic spinal cord injury with monoplegia of the right arm also reported an infarct on the right hemi cord on level cervical 4 (C4)-C5. The right arm showed no power in muscle testing and the left arm showed a mild weakness (Birua, 2019).

Our patient had spinal shock characteristics which were defined as loss of motor and sensory functions below the level of lesion secondary to complete or relatively complete spinal cord lesion (Salazar-Muñoz et al., 2019). Spinal cord pathway rearrangement causes loss of function that happens acutely post-injury. Accumulation of protein post trauma in the medulla also contributes to the process of spinal shock. After the process abates, the function will return to normal. Since there was no
hemodynamic instability (hypotension, bradycardia, and hypothermia) found, neurogenic shock could be excluded (Knj, 2018).

Disease progression of spinal shock is divided into four phases. The first phase (0-24 hours after injury), identified as areflexia/hyporeflexia phase, shows none or decreased deep tendon reflex, delayed plantar reflex followed by cutaneous reflexes and sympathetic dysfunctions (Ditunno et al., 2004; Ko, 2018). These changes are due to motor neuron hyperpolarization. The second phase (1-3 days after injury) is characterized by prominent cutaneous reflexes without deep tendon reflexes, and is proposed due to denervation super-sensitivity. Super sensitivity mechanisms include reduced excitatory neurotransmitter reuptake, increased synthesis, and insertion of receptors into postsynaptic membrane, decreased ion channel function and degradation of receptor, and altered synthesis and composition of receptors (Knj, 2018). In the third phase (4 days-1 month after injury), deep tendon reflexes have returned and Babinski sign may appear, reflecting the growth of axon-supported synapse. Ankle jerk appears before Babinski sign and knee jerk. Cutaneous reflexes such as bulbocavernosus reflex, cremasteric reflex, and anal wink typically appear by the end of this period (Ditunno et al., 2004). Lastly, soma-supported synapse growth occurs in the fourth phase (12 months). Hyperactivity occurs in cutaneous and deep tendon reflexes in response to minimal stimuli. This mechanism is driven by synapse and short axon growth in empty synaptic endings in supraspinal neurons (Knj, 2018).

Three weeks after injury, patient was in the third phase of spinal shock. Loss of patellar reflex (Lumbar (L2)-L4 level) and returned Achilles reflex (L2-Sacral 2 (S2) level) aligned with loss of sensation in L2-Sacral level and below that level and represented recovery of reflexes in caudo-rostral pattern (Figliuzzi et al., 2022; Salazar-Muñoz et al., 2019). The recovery of superficial reflexes varies ranging from an hour up to months for deep tendon reflexes. Recent studies showed recovery pattern of reflexes with the following order: delayed plantar reflex, bulbocavernosus reflex, cremasteric reflex, ankle jerk, Babinski sign, and lastly knee jerk (Ziu & Mesfin, 2022).

Acute bladder and bowel incontinence were associated with the level of spinal cord hemorrhage. Lesion in Th10-L2 level will cause incontinence in bowel and bladder due to sympathetic loss which is in the thoracolumbar area (Francis, 2007). Spinal shock caused the suppression of autonomic and somatic activity of bladder, being a-contraction, and areflexic (Salazar-Muñoz et al., 2019).

Bladder dysfunction manifestation in post spinal cord injury might be varied depending on its location, supra-sacral, sacral, or infra-sacral, or mixed. Supra-sacral bladder dysfunction is an upper motor neuron (UMN) neurogenic dysfunction when there is blocked communication between sacral reflex arc and pontine micturition center (PMC). The lower motor neurons are damaged in sacral or infra-sacral spinal cord injury (SCI) which interrupts voiding reflex arc and leads to areflexic detrusor muscle and flaccid external urethral sphincter. Mixed type is classified to type A, which is more common than type B. Type A neurogenic bladder is caused by damaged detrusor nucleus causing detrusor areflexia and intact pudendal nucleus producing hypertonic external urinary sphincter. Contrarily, detrusor muscles are spared and pudendal nucleus are damaged in type B causing flaccid external urinary sphincter and spastic bladder (Dorsher & McIntosh, 2012).

In this case, incontinence happened due to supra-sacral lesion causing UMN symptoms called detrusor sphincter dyssynergia (DSD). Detrusor and urethral sphincter became hyperreflexia and increased the pressure within the detrusor (Perez et al., 2022). Complete spinal cord lesions at the T6-S2 level usually develop involuntary bladder contractions without sensation and DSD. Catheter was used to avoid large post-void residual volume which could lead to further complication such as urinary tract infection and autonomic dysreflexia (Chen et al., 2022). Head up position is needed to reduce intracranial pressure due to facilitation drainage of reverse blood flow from intracranial (Pertami et al., 2017). Yellow hand band is used to warn others of fall risk. This is given for someone ≥65 years old, hospitalized for fall-related injuries, and suffered from moderate to severe injuries that reduce mobility and independence (Wisconsin Hospital Association, 2007). Postoperative status and neurological diseases are one of the leading causes of falls. Process of assessment and risk detecting are effective in achieving reduction in falls (Montejano-Lozoya et al., 2020). There are three primary strategies to prevent falls: identify patient at risk, place bed or chair alarms on patients, and run to alarms. However these strategies are known to be ineffective and more studies are needed (King et al., 2018).

Spinal cord secondary injury entails inflammation, edema, ischemia, electrolyte imbalances, lipid peroxidation, and glutamate excitotoxicity leading to death of spinal cord tissue. It can be prevented by steroids or decompression surgery within 24 hours (Caneco et al., 2021). Decompression surgery is still encouraged if possible. However, in cases where surgery is less beneficial to perform, rehabilitation may improve patient’s outcome and function. To prepare her for surgery, she needed to be explained about why she needs surgery, what the operating room looks like, and what she will feel and do after surgery. Visualization and using neutral words are recommended. Informing the patient’s parent or guardian is also recommended for pediatric patients. Visualization using blocks, doll houses, and stuffed animals by telling the story of what will happen to the doll (as herself) can help the explanation and indicate that the equipment is safe (Kaakinen et al., 2015).
Physiotherapy and strength training for three months, twice a day for 30 minutes each, may improve patient’s ability to regain their strength (Oh et al., 2018). Three months are recommended according to the rehabilitation phases, acute subacute and long-term phase. In this case, patient’s physiotherapy was not recorded; however, it was recommended upon discharge. When the vital signs are stable, physical rehabilitation is recommended to increase the patient’s strength (Pegat et al., 2015). Concomitant with the strength training, there will be limitations of daily activities of the patient. Supportive care and surroundings are recommended. Dexamethasone and methylprednisolone are widely used to prevent neural secondary injury despite the controversies on their effect. Steroid are known to increase risk of pneumonia and hyperglycemia compared to the minimum benefit in spinal cord injury (Canseco et al., 2021). Methylprednisolone’s neuroprotective effects are due to lipid peroxidase and inflammatory cytokines inhibition. This follow up after discharge of this patient was not recorded and observed, which limits this study.

Conclusions
Clinical symptoms of hematomyelia can affect sensoric, motoric, and autonomic systems, including bladder and bowel incontinence. Hematomyelia can cause spinal shock due to spinal cord pathway rearrangement and build-up of protein which leads to the shock. Decompression surgery is recommended to increase the prognosis of the patients. Minimizing the risk of falling and explanation of a procedure using visualization is recommended.

Declaration of Interest
All authors declare that they have no conflict of interests.

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Data Availability
Available on reasonable request from the corresponding author’s email.

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