Postpartum Spinal Cord Injury in a Woman With HELLP Syndrome

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Abstract

Objective: To report a rare cause of spinal cord injury.

Study design: Case report.

Case report: A 36-year-old woman presented with acute onset of paresis of the upper and lower extremity (level C5, ASIA B) the day after delivering a healthy daughter (39 weeks' gestation). Prior to giving birth, she was admitted with gestational hypertension. Directly postpartum, blood pressure increased markedly, and hemolysis, elevated liver enzymes, and low platelet count (HELLP) syndrome was diagnosed. The next day, tetraplegia developed. Magnetic resonance imaging showed multiple epidural and subdural hematomas in the cervical and thoracic spinal canal extending from C2 to T8, with displacement and compression of the cord. Three weeks after treatment with intravenous labetalol, she made a significant recovery (level T1, ASIA D).

Conclusions: This rare cause of spinal cord injury shows the importance of closely monitoring pregnant patients with hypertension complicated with HELLP syndrome both pre- and postpartum.

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Key Words: Spinal cord injuries; Myelopathy; Postpartum; Tetraplegia; HELLP syndrome; Gestational hypertension; Hematoma, spontaneous, epidural, subdural

INTRODUCTION

Spontaneous spinal epidural hematoma (SSEH) associated with pregnancy is a rare cause of spinal cord compression. There have only been 9 previously published cases of SSEH occurring during pregnancy (1–3). We present what we believe is the first reported case of SSEH associated with hemolysis, elevated liver enzymes, and low platelet count (HELLP) syndrome occurring directly postpartum.

CASE REPORT

A 36-year-old, otherwise healthy, pregnant woman (G1P0) was admitted to a local hospital with gestational hypertension, which had developed over the last 3 weeks. She gave birth to a healthy daughter at 39 weeks of gestation without any complications. Within hours, she developed abdominal pain, accompanied by a marked

increase in blood pressure. The next morning, she complained of headache, pain in the neck, and back pain, with an even further increase in blood pressure. Laboratory tests showed signs of HELLP syndrome. Within 1 hour, tetraplegia developed (level of injury C5, ASIA B, bilateral upper extremity motor score of 20 and a lower extremity motor score of 0, WISC II level 0). Magnetic resonance imaging (MRI) of the spine 5 hours after the onset of tetraplegia showed multilevel epidural and subdural hematomas in the cervical and thoracic spinal canal extending from C2 to T8, with displacement and compression of the spinal cord (Figures 1–3).

She was treated with intravenous labetalol for hypertension; no neurosurgical intervention was conducted regarding the hematomas. During admission, she showed a gradual improvement in motor function and sensation.

At the time of transfer to our spinal cord injury (SCI) department, 2 weeks postpartum, she had a T1 level of injury, ASIA D, upper extremity motor scores of 23 on the right and 19 on the left side, and lower extremity motor scores of 18 on both sides with no sensory loss. She had started walking with a walking aid (WISC II level 13) and had persistent difficulties in maintaining balance and coordination. In self-care activities, she only needed some

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Figure 1. Sagittal T2-weighted MRI of the cervical and thoracic spine, taken 5 hours after onset of tetraplegia, showing epidural and subdural hematomas compressing the spinal cord at the C5-C6 level (white arrow).

Figure 2. Sagittal T2-weighted MRI of the cervical and thoracic spine, taken 5 hours after onset of tetraplegia, showing an epidural hematoma compressing the spinal cord at the T1 level (white arrow).

Figure 3. Transversal T2-weighted MRI of the thoracic spine at the T4 level, taken 5 hours after onset of tetraplegia, showing an epidural hematoma compressing the spinal cord with displacement of the spinal cord to the right (white arrow).

help in washing her back and lower legs. Her bladder was managed by an indwelling catheter, and bowel function was normal. She needed assistance with the daily care of her daughter.

At admission, intermittent catheterization for bladder management was started. Progressive improvement in motor function, balance, coordination, and bladder function was noted throughout the rehabilitation period. Five weeks postpartum, at discharge to home, her SCI level and ASIA score were unaltered. However, the upper extremity motor scores had increased to 25 bilaterally, and the lower extremity scores had increased to 24 on the right and 22 on the left side. She was completely mobile and could walk independently carrying her daughter (WISCI II level 20). No help was needed in self-care activities, and bladder and bowel function were normal. After discharge, muscle strength, physical condition, balance, and coordination training were continued in her own environment.

Four weeks after discharge to home, muscle strength and physical condition were further improved. She had only minimal problems with balance and coordination, which did not cause any problems in daily life. She was completely independent and could also look after her daughter independently. Follow-up MRI, 12 weeks after the onset of tetraplegia, did not show any late sequelae.

DISCUSSION

SSEH is a rare cause of spinal cord compression, and SSEH associated with pregnancy is even more uncommon (1–3). The hypothesis for the pathogenesis of SSEH is rupture of epidural veins situated in the low-pressure epidural space secondary to sudden increases in intra-abdominal

or intrathoracic pressure; this is usually associated with bleeding disorders (4). The etiology of SSEH during pregnancy (a state of hypercoagulability) remains unclear (1). The changes in hemodynamics during pregnancy may increase epidural venous pressure and cause rupture and hemorrhage (1,2).

Clinically, SSEH presents as severe localized back pain, often with a radicular component (3). Onset of pain may be related to minor straining but is mostly spontaneous. Neurological symptoms appear rapidly and may progress to SCI. When neurological abnormalities develop, MRI of the spinal cord and a coagulation profile should be performed (3). MRI is recommended for identifying the level of injury, presence of hemorrhage and edema, and degree of cord compression (5). The most appropriate treatment of SSEH is surgical decompression of the spinal cord (2,3). Postoperatively, a rehabilitation specialist should be involved in establishing the rehabilitation objectives and plans to ensure optimal recovery.

This woman had several factors pre- and postpartum that are associated with or linked to the development of SSEH: gestational hypertension, giving birth, and HELLP syndrome, in which one of the key factors is a low platelet count. It could be that the combination of these factors increased her risk for developing SSEH and could also explain the multiple epidural and subdural hematomas on various levels of the spinal canal, in contrast to the single SSEH in previously reported cases (1–3). She developed SSEH characterized by the usual clinical

symptoms, but this occurred postpartum and not, as previously reported, during pregnancy (1–3). No surgical decompression was conducted because of the surgical risk incurred by low platelet count and hematomas on several levels from C2 to T8.

CONCLUSIONS

This rare cause of SCI shows the importance of closely monitoring pregnant women with hypertension complicated with HELLP syndrome for both pre- and postpartum complications. Onset of neurological symptoms warrants prompt and thorough workup to maximize clinical outcomes.

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