

Acute Paraparesis Secondary to Epidural Hematoma Caused by Spinal Anesthesia Given for Lower Segment Caesarean Section

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Abstract

Spinal anesthesia is one of the most common anesthetic procedure performed in operative cases. Neurological deficit is an uncommon but catastrophic complication of spinal anesthesia. Epidural hematoma is the most common cause of such neurological deficit. Unidentified bleeding and clotting disorders increase the risk. On detection such complications need close monitoring. Though many cases can be managed conservatively, progressive deficit warrants quick decompression of spinal canal.

Keywords: Epidural hematoma, Paraparesis, Spinal anesthesia, Laminectomy.

Introduction

The use of spinal anesthesia for surgical procedures dates back to 1885, but it was not until the 1940s when Adriani and associates established safe, standardized techniques that this method of analgesia became popular in obstetrics [1, 2]. Spinal anesthesia is one of the most common anesthetic procedures performed for surgery in the lower part of the body [3]. Neurological deficit is an uncommon but catastrophic complication of spinal anesthesia. Epidural hematoma is the most common cause of such neurological deficit [1]. While such hematomas constitute rare complication, when they develop, they can result in acute spinal cord compression characterized by severe back pain, motor deficits, bowel or bladder dysfunction, and paraplegia. Such a complication is rare, and we report one such rare case of acute paraparesis

developed secondary to spinal anesthesia, which was managed successfully by a prompt surgical intervention.

Case Report

A 26-year-old female P3L4 patient was referred to our center with postpartum hemorrhage and acute onset weakness in both lower limbs. The patient was operated in a peripheral center 5 h ago, where twin delivery had been performed by a lower segment cesarean section under spinal anesthesia. At the time of admission, the patient was in hypovolemic shock with hemoglobin at 4.5 gm%, bleeding time and clotting time were normal, and the platelet count was 1.4 lakh/mm [3]. Bleeding per vagina was controlled by gynecologist, 6 units of blood transfusion were done, and the patient was referred to the orthopedic department for acute onset weakness. On examination, the patient had weakness in both lower limbs but more on the left side (Table 1). Sensation for touch, temperature, pain, and pressure was normal. Deep tendon reflexes on the left side were diminished, and plantar reflex was down going. Suspecting the possibility of epidural hemorrhage, MRI of the lumbar spine was taken, which

showed an epidural hematoma measuring 9 × 8 mm at L2–L3 level encroaching on to the dural sheath causing compression and mass effect (Fig. 1 and 2). Initially, a conservative management was planned and intravenous dexamethasone and methylcobalamin were started and rest advised, but the weakness worsened over next 24 h more in the left limb (Table 1). The patient was taken up for surgery, L2–L3 level laminotomy was done and the hematoma was removed, and nerve roots were decompressed adequately. There was no cord or root contusion. Neurological status improved on the next day and the patient began ambulating with wide-based stable gait on the post-operative day 3.

Discussion

The use of spinal anesthesia for cesarean section is a very common practice all over the world. Damage to the epidural veins during epidural or spinal anesthesia occurs very commonly, but the bleeding is very minimal and temporary which stops by itself through the normal coagulation system of the patient. Spinal anesthesia rarely results in complications such as spinal hematomas [4]. According to obstetric research, 1 out of 183,000

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Figure 1: sagittal MRI showing hematoma

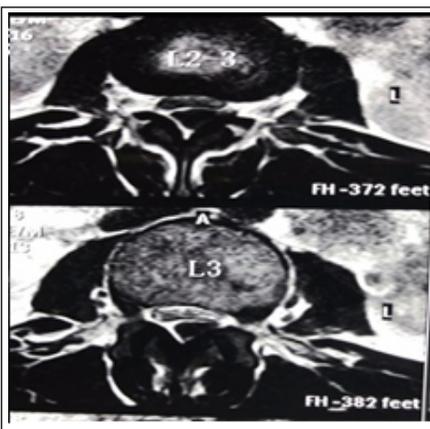


Figure 2: Axial cut showing hematoma compressing on the dural sac

patients developed EDH, which is extremely rare [5]. In addition, pregnancy is defined as a mild hypercoagulation state, which reduces the risk of EDH. In practice, it was found that pregnant women have a lower risk of developing spontaneous EDH than normal adults [2]. However, it remains unclear if certain variables increase the likelihood of these events or if surgical intervention improves outcome [3]. Pre-existing coagulopathy appears to increase the risk of epidural hematoma, it also appears that coagulopathy worsens the prognosis in such cases, thus

highlighting the importance of pre-anesthetic evaluation of any existing coagulation disorders [2,6].

In our case, we can think of few possible causes of an epidural hematoma. The first cause could be the massive blood loss during delivery which could have consumed coagulation factors. The second cause could be the venous dilatation and varicosities of epidural veins which are common during pregnancy [7]. The third cause could be mechanical damage during needle insertion and removal.

Most of the epidural hemorrhages are

self-limiting and asymptomatic. When the bleeding is uncontrolled and excessive, they can present with mild symptoms such as anterior lumbar pain and radiculopathy to severe symptoms such as paraplegia, bowel and bladder involvement, and urinary incontinence, for which immediate management is required since it can lead to permanent sequelae. Early and prompt management of such complication are very important. Although the literature is not clear about indications for surgical intervention, it appears that progressive neurological deficit mandates an early surgical decompression as in our case. It is best to have the operation before paralysis or within a few hours after paralysis has occurred to prevent irreversible ischemia [2]. There are some reports of spontaneous recovery from EDH [8,9]; however, considering the pathological characteristics of a hematoma being clotted, surgical removal seems to be the most reasonable course to follow. Many reports have stated that it is best to have surgery within 24–36 h after symptoms manifest. Whenever possible, we suggest aggressive, proactive management of coagulopathy before any intervention, as this may decrease the incidence of such potentially devastating injuries. In the absence of enough data in the literature, the indication and plan of treatment are highly individualized.

	Hip flexion		Knee extension		Ankle dorsiflexion		Ankle plantarflexion	
	right	left	right	left	right	left	right	left
Day1	04-May	03-May	04-May	03-May	05-May	03-May	05-May	04-May
Day2	04-May	01-May	04-May	01-May	04-May	01-May	05-May	03-May
Post op	05-May	04-May	05-May	04-May	05-May	04-May	05-May	04-May
6 Weeks of Follow up	05-May	05-May	05-May	05-May	05-May	05-May	05-May	05-May

Table 1: MRC grading of lower limb weakness

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