CASE REPORT

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Spinal Epidural Hematoma After Spinal Anesthesia

Mustafa HACIÖMEROĞLU[a], Simge YILDIZ[a]

[a]Erciş Şehit Rıdvan Çevik State Hospital, Clinic of Anesthesiology and Reanimation, Van, Türkiye

ABSTRACT Spinal epidural hematoma (SEH) is a rare but severe complication of central neuraxial blocks (CNB), with an estimated incidence of 1:150.000 to 1:200.000. SEH can cause significant neural tissue compression, potentially leading to permanent neurological damage. A 35-year-old man underwent tibia and fibula fracture surgery under spinal anesthesia without intraoperative complications. On 2nd postoperative day, the patient developed abdominal pain, followed by bilateral lower extremity muscle weakness on day 3, progressing to paraparesis. Magnetic resonance imagining (MRI) revealed an epidural hematoma extending from L1 to S1, compressing the spinal cord. Conservative treatment was initiated, and the patient fully recovered. SEH should be considered when postoperative neurological symptoms arise, especially after neuraxial blocks. Despite the multifactorial etiology and absence of clear risk factors, epidural vascular structure and anesthesia technique may play a role. MRI remains the diagnostic gold standard, and early neurosurgical consultation is crucial. Conservative management may be considered in selected cases.

Keywords: Spinal Epidural Hematoma; spinal anesthesia; orthopedic procedures

Spinal epidural hematoma (SEH), first described by Jackson in 1869, is one of the rare but severe complications of central neuraxial blocks (CNB). An estimated 1:150.000 to 1:200.000 is the incidence of SEH associated with CNB. Epidural hematoma that occurs after CNB can caused by a trauma to the epidural veins, spontaneous hemorrhage or congenital vascular malformation.[1] The literature identifies several risk factors including hypertension, age, perioperative anticoagulant medication, pregnancy, a history of spinal cord injury, coagulopathy, spinal canal deformity, multiple punctures, difficult catheter placement, and needle size.[2] SEH typically presents with a sudden onset and may result in severe compression of neural structures. This compression can occur either due to direct mechanical pressure or through ischemic mechanisms. The condition commonly manifests as intense back pain accompanied by motor and sensory impairments, which can progressively lead to paraplegia. The most appropriate treatment of SEH is nearly always surgical, with rapid decompression of the spinal cord being the most effective treatment. If decompression is not performed within 8 hours, permanent neurological damage may occur.[3] However, there have been reports of spontaneous recovery in some cases of SEH, which complicates the decision between surgical and conservative management.[4] We report the case of a patient without comorbidity, normal coagulation function, and no history of anticoagulant therapy who developed paraparesis due to an SEH 3 days after spinal anesthesia. Remarkably, the condition resolved rapidly and completely without surgical intervention.

CASE REPORT

A 35-year-old Caucasian male (ASA II) presented with tibia and fibula fracture and was promptly transferred to the operating room for intramedullary nail-

Correspondence: Mustafa HACIÖMEROĞLU
Erciş Şehit Rıdvan Çevik State Hospital, Clinic of Anesthesiology and Reanimation, Van, Türkiye
E-mail: mustafaahaciomeroglu@gmail.com

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ing. He had no history of disease or surgery. The patient had no prior history of back issues and could walk unlimited distances without experiencing dyspnea, angina, or claudication. His smoking history was 10 pack-years, with no family history of coagulation disorders.

On examination, his vital signs were within normal limits and stable. Airway assessment revealed a Mallampati class II. Cardiovascular examination showed normal heart sounds, and the chest was clear on auscultation. Neurologic evaluation was unremarkable, and there were no detectable spinal abnormalities. Preoperative laboratory findings included a hemoglobin level of 14.7 g/dL, a platelet count of 259.000/mm³, an international normalized ratio (INR) of 1.1, and an activated partial thromboplastin time (aPTT) of 27.4 seconds. All other investigations were normal and not clinically significant.

Lumbar spinal anesthesia was performed at the L2-3 level using a 25G Quincke spinal needle, administering 12.5 mg of bupivacaine on the first attempt, and achieving a T10 level of anesthesia. The operation was uneventful. In the follow-up examination of the patient in the ward setting, it was observed that the motor, sensory, and autonomic blockades resolved completely after 4 hours, and the patient's neurological examination was normal. The patient was mobilized on the same day but developed nonspecific abdominal pain 24 hours postoperatively, which was evaluated by the general surgery and internal medicine teams, and was thought to be secondary to slow bowel motility. The patient, who commenced a program of mobilization exercises, subsequently exhibited motor weakness in both lower extremities at 72 hours postoperatively. In the neurological examination of the lower extremities, the motor power was 3/5 on the left and 1/5 on the right. On examination, the patient was afebrile with stable vital signs. Bilateral deep tendon reflexes were absent. Sensory testing showed a loss of pinprick and light touch sensation below the L2 dermatome on the right and below L1 on the left. Laboratory results revealed a platelet count of 200.000/mm³, an INR of 1.2, and an aPTT of 27.6 seconds. The white blood cell count was measured at 11.300/mm³.

Urgent magnetic resonance imaging (MRI) of the thoracic and lumbar spine identified an extensive epidural hematoma spanning from L1 to S1 (Figure 1, Figure 2). T2-weighted images showed iso-hypointense and T1-weighted images showed iso-hyperintense areas with minimal enhancement after intravenous contrast material injection. The axial sections revealed bilateral spinal compression, more dominant and prominent on the right side.

After consulting with neurosurgery and radiology, a spinal computed tomography angiography was performed, showing normal spinal circulation. A risk-benefit analysis was conducted to assess the feasibil-

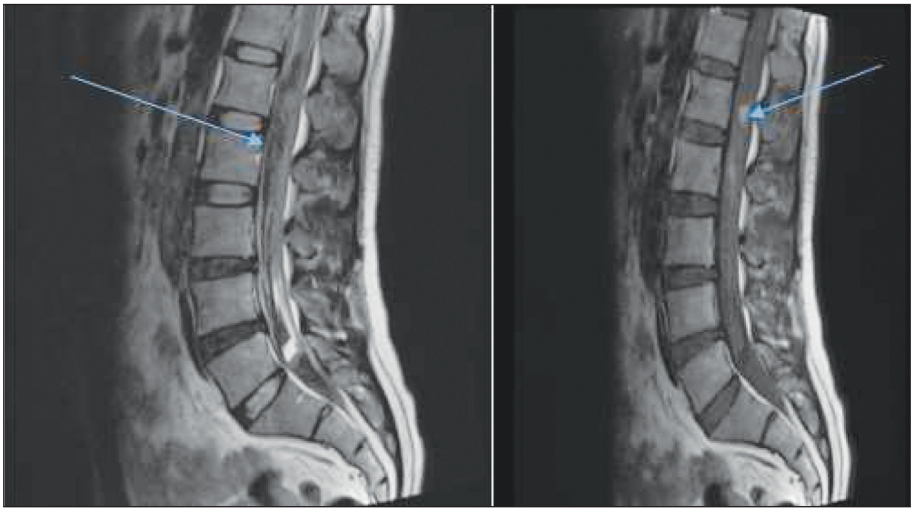

FIGURE 1: Spinal epidural hematoma T2-T1 sequences on MR

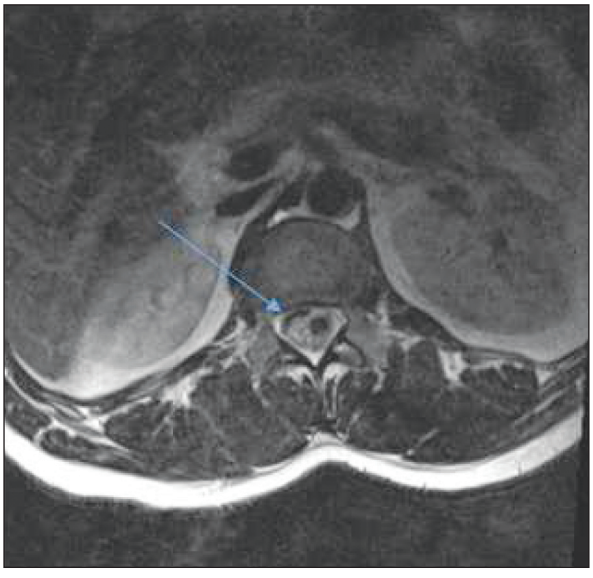

FIGURE 2: Spinal cord compression on MRI

ity of surgery given the hematoma size. The patient was subsequently treated with a conservative approach. In the weeks that followed, the patient showed consistent improvement with ongoing physiotherapy and mobilization exercises. An MRI scan performed on the 6th postoperative day revealed that the hematoma was isolated at the L1-L2 level and that the patient's muscle strength had increased. On postoperative day 15, his motor strength was 4/5 in all lower extremity muscle groups. Deep tendon reflexes were present bilaterally. The patient was discharged with a modified physical therapy program, following a normal neurological examination on postoperative day 20. Informed written consent was acquired for the purpose of publication. In the patient's 3th month follow-up MRI, it was observed that the compression had fully resolved, and the patient exhibited no longer any symptoms or signs that would indicate the presence of the condition. This was confirmed by both clinical and radiological evaluations.

DISCUSSION

SEH can occur spontaneously or as a result of coagulation disorders, regional anesthesia techniques, or trauma. When associated with neuraxial anesthesia, SEH is more frequently reported after epidural anesthesia compared to spinal anesthesia and is often linked to coagulation abnormalities or the use of anticoagulants.[1] The occurrence of SEH following spinal anesthesia is extremely rare, and the understanding of this complication remains limited. Conducting prospective, randomized studies on this topic is challenging due to the large sample sizes required. Identified risk factors include female gender, age, traumatic and multiple attempts at puncture, anticoagulant use, vascular malformations, and hypertension.[5] Additionally, while vessel injury during spinal anesthesia has been reported, its documentation remains challenging.

While back pain is the most frequently reported symptom of SEH, it is important to note that its absence does not rule out the condition. In a 1994 review of 61 cases, back pain was the initial symptom in only 38% of patients. Early and proactive MRI screening could facilitate prompt diagnosis in such

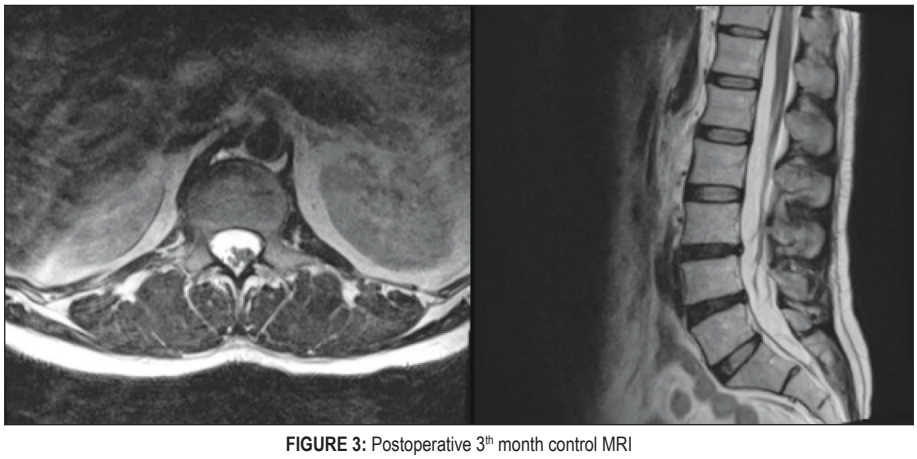

FIGURE 3: Postoperative 3th month control MRI

cases, and this approach should be considered the standard of care.[6] Unfortunately, in this instance, timely MRI imaging could not be performed due to the patient's inability to lie down. Notably, our patient did not present with back pain but instead experienced vague abdominal discomfort. Another critical point to consider is that the hematoma had extended across the entire vertebral column while the patient was in the supine position. It is reasonable to suggest that the compression effects may have manifested after the second day, once the patient began standing.

The management of SEH can be approached through 2 distinct pathways: surgical and conservative. In the presented case, a conservative management approach was chosen, involving repeated neurological assessments, based on the observed improvement in symptoms following the MRI. This improvement may be explained by the hematoma fluid leaking through the intervertebral foramina, reducing pressure on the spinal cord. The duration and dosage of methylprednisolone treatment were determined by the neurosurgeon's clinical judgment and experience, rather than being guided by specific protocols. Several reports have documented the resolution of SEH without the need for surgical intervention, with growing evidence supporting this approach in certain cases. Most patients managed conservatively showed improvement during the initial evaluation.

The possibility of SEH should always be considered when neurological symptoms arise in the postoperative period, especially following neuraxial blockade. A thorough neurological examination is essential whenever SEH is suspected. MRI is the diagnostic tool of choice, and prompt neurosurgical consultation is crucial for determining the appropriate course of action. In some cases, conservative management may be an option.

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Conflict of Interest

No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.

Authorship Contributions

All authors contributed equally while this study preparing.

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