Spinal Subdural Hematoma: A Rare Complication of Spinal Anesthesia: A Case Report

ABSTRACT
Spinal hematoma is an infrequently described complication of spinal anesthesia. It has been described in the literature in patients with a deranged coagulation profile in the form of systemic diseases (e.g. chronic renal failure, liver failure) or anticoagulant therapy. Whatever the reason, it will have disastrous consequences in the form of permanent paraplegia or persistent neurological deficit if not treated promptly. One therefore needs to maintain a high index of suspicion at all times in any patient who has undergone spinal anesthesia and who exhibits any sign or symptom of a neuraxial hematoma.

We report a case of post-spinal puncture subdural hematoma in an otherwise healthy male without any predisposing factor.

KEY WORDS: Complication, Paraplegia, Spinal anesthesia, Spinal subdural hematoma

Spinal hematoma spinal anestesinin oldukça az görülen bir komplikasyonudur. Literatürde pıhtılaşma mekanizmasını etkileyen sistemik hastalığı (kronik böbrek yetmezliği, karaciğer yetmezliği) veya antikoagülan tedavi alan hastaları alan hastalarda spinal anestesi sonrası gelişen spinal hematom tanıltanmıştır. Sebebi ne olursa olsun spinal hematom hızla tanı konularak tedavi edilmekte ise kalıcı paraplejije yol açmaktadır; bu nedenle spinal anestesi alan hastaların muayenesi her zaman spinal hematom olasılığı акılda tutularak yapılmalıdır.

Bu yazida, herhangi bir sebeb olmaksızın spinal anestesi sonrası gelişen spinal hematom vakası sunulmaktadır.

ANAHTAR SÖZCÜKLER: Komplikasyon, Parapleji, Spinal anestesi, Spinal subdural hematoma
INTRODUCTION

Bleeding into the spinal canal is the most serious complication associated with spinal anesthesia as the spinal canal is a concealed and non-expandable space(11). Despite surgical decompression and aggressive rehabilitation, the majority of reported neurological deficits have been permanent6 if not treated in time(12).

We report a case of post-spinal puncture subdural hematoma in an otherwise healthy male without any predisposing factor.

CASE REPORT

A 35-year-old male was admitted to our institute with complaints of complete paraplegia and a history of attempted spinal anesthesia 3 days back at another hospital for fixation of fractured shaft of femur on the right side. He had a history of two attempts at spinal anesthesia during which he felt a shock-like sensation in both the lower limbs followed by pins and needles like sensation. The procedure was abandoned immediately and he was transferred out of the operating room. A history of bloody tap could not be elicited. He soon started complaining of progressive ‘numbness’ followed by complete paraplegia over the next 8 hours. The general physical and systemic examination was unremarkable except for the neurological findings. He was not receiving anticoagulant therapy in any form. Neurological findings included spastic paraplegia with complete sensory loss below the L1 dermatome. Tone was markedly increased, knee jerks were exaggerated and both the plantars were upgoing.

Hematological investigations were normal and MRI spine revealed an epidural collection from T12-L3 level without any contrast enhancement (Figure 1 and 2). The patient was immediately taken up for surgery. There was no collection in the epidural space but the dura was tense, bulging and bluish in color. The dura was opened and hematoma evacuated. Arachnoid membrane was intact and the spinal cord and cauda equina were apparently normal. There was no post operative CSF leak.

Aggressive rehabilitation was started from the 2nd postoperative day but even after a month he showed no improvement in neurological function. One month post operative he was discharged on rehabilitative aids.

DISCUSSION

The safety of neuraxial blocks has been well established and acknowledged over a series of studies and trials, even with anticoagulation and antiplatelet therapy(1,11). Intraspinal hematoma, though rare, is an established complication of lumbar puncture and a source of concern for the anesthesiologist, because it can cause permanent neurological deficits(2,6). As far as localization of the hemorrhage is concerned, there are still doubts in the literature regarding the site of bleeding and the blood collection. It may occur in the extradural, subdural or subarachnoid compartments of which the epidural space is the commonest site(2,3). Spinal subarachnoid hematoma is extremely uncommon. In an analysis of causative factors, 54% of the cases with acute spinal subdural hematoma were associated with a bleeding disorder, which was either secondary to a defect in the haemostatic mechanism (leukemia, hemophilia, thrombocytopenia, cryoglobulinemia, hemorrhagic diathesis, polycythemia) in 19% or to acute or chronic anticoagulant therapy in 35% of the cases. In addition to the coagulative alteration, LP acted as an iatrogenic cause in 33% of the cases. In another 14%, the cause of the acute spinal subdural hematoma was purely iatrogenic (the result of LP), although in
7% of these cases there were coexisting conditions such as pregnancy, dialysis treatment, diabetes mellitus, arterial hypertension, and alcoholism, either singly or combined(2). Most of these purely iatrogenic reports have involved at least some difficulty during dural puncture or the return of bloody cerebrospinal fluid(3). The localization of the acute spinal subdural hematoma has been reported infrequently(2). It has been described as being extraarachnoidal, appearing to be “in between two layers of dura,” which suggested a “potential intradural space” as the site of the bleeding and subsequent hematoma(9). The extraarachnoidal aspect of acute spinal subdural hematoma is also confirmed by preoperative neuroradiological imaging, in which it often appears as extradural bleeding(2) as was present in our case also. Typically, it begins with severe lumbar pain, which progresses to flaccid paralysis. Absence of lumbar pain does not exclude the possibility of an intraspinal hematoma as was apparent in our case(10). In our case the paraplegia was of spastic nature suggesting an UMN type of neurological deficit, which can be attributed to the extraarachnoidal origin and cephalad migration of the hematoma. Spinal hematoma should always be considered early in the differential diagnosis of patients who present with motor and sensory deficits with a history of neuraxial intervention and should be diagnosed and treated aggressively(10). Though MRI confirms the presence of a large mass compressing the spinal roots, the exact localization can be confirmed on surgical exploration(2), 8. Early surgical intervention and evacuation is indicated, however in some patients with minimal symptoms conservative treatment may play a role(7). Good results may be expected in patients with mild preoperative neurological deficits. Results are poor in patients with subarachnoid hematoma, severe preoperative deficits and in those where surgery has been delayed as in our patient(2,7). Early diagnosis and urgent surgical treatment improve the possibility of neurological recovery(1,5). It is known that surgical decompression more than eight hours after the beginning of the symptoms is associated with a worse prognosis(1). The anatomical localization of acute spinal subdural hematoma influences the prognosis if the hematoma is confined to an intradural space or if it produces a SAH. In the latter case, the prognosis is worse as in addition to the mass effect of the hematoma separated from the subarachnoid space, there is also rupture of the internal membrane and consequent SAH, which has negative effects of bleeding on the spinal cord or cauda equina(2). When the collection of blood is large, there may be local pain, flaccid paraparesis, impairment of sensation in the lower limbs and sphincter disturbances. In this setting, prompt evacuation of the hematoma is mandatory to prevent a permanent neurological deficit4.

This case report highlights the importance of maintaining a high index of suspicion in cases of spinal anesthesia presenting with symptoms suggestive of spinal hematoma even if the patient is devoid of any predisposing factor. Early diagnosis and timely intervention will result in improved outcome.

REFERENCES