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Intracranial Chronic Subdural Haematoma as a Complication of Epidural Anesthesia

Epidural Anestezinin Komplikasyonu Olarak Gelişen İntrakranial Kronik Subdural Hematom

Nail ÖZDEMİR[1]
Mustafa Kemal ARI[2]
Mustafa Fazıl GELAL[3]
Hamdi BEZİRCİOĞLU[4]

[1,2,4] İzmir Atatürk Training and Research Hospital, Neurosurgery Department, İzmir, Turkey
[3] İzmir Atatürk Training and Research Hospital, Radiology Department, İzmir, Turkey

ABSTRACT

We report the case of a 39-year-old woman with unilateral intracranial chronic subdural haematoma that occurred after epidural anesthesia for excision of a skin scar, complicated by postdural puncture headache. The patient had no history of trauma, headache, coagulation abnormalities, or neurological disorders. Most physicians encountering a case of headache after epidural or spinal anesthesia first think of a postdural puncture headache. Nonetheless, the symptoms subside within 7 days when treated with analgesics and bed rest in the majority of cases. The presence of a continued postdural puncture headache without neurological deterioration, as in this case, should prompt a search for an intracranial lesion.

KEY WORDS: Chronic subdural haematoma, Dural puncture, Epidural anesthesia, Headache

ÖZ

Derideki skar eksizyonu için epidural anestezi uygulanan ve istemsiz dura ponksiyonu ile başağrısının oluştuğu 39 yaşındaki kadın hastada gelişen unilateral kronik subdural hematom vakasını sunduk. Hastanın özgeçmişinde travma, başağrısı, koagülasyon bozukluğu ve nörolojik hastalık yoktu. Birçok hekimin epidural veya spinal anestezi sonrasında gelişen başağrısında ilk düşündüğü şey, dural ponksiyon sonrası beyin omurilik sıvısının boşalımına bağlı gelişen başağrısıdır. Bununla birlikte; vakaların bir çoğunda, analjezik ve yatak istirahati ile tedavi edildiğinde, 7 gün içinde bu durum geçer. Bu vakada olduğu gibi, nörolojik kötüleşme olmasa bile dural ponksiyona bağlı başağrısının devam etmesi halinde, bir intrakranial lezyon araştırılmalıdır.

ANAHTAR SOZCÜKLER: Kronik subdural hematom, Dural ponksiyon, Epidural anestezi, Başağrısı

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Correspondence address:
Nail ÖZDEMİR
E-mail : nailozdemir@mynet.com

INTRODUCTION

Chronic subdural haematoma (cSDH) is a known complication after head trauma in Emergency Department patients with predisposing factors, such as old age, alcoholism, and coagulation abnormalities (2,5). However, cSDH following unintentional dural puncture of epidural anesthesia is a very rare condition.

CASE REPORT

A 39-year-old woman with severe and persistent headache was admitted to our hospital. She had a history of epidural anesthesia for excision of a skin scar 21 days earlier, including unintentional dural puncture. The patient had no history of trauma, headache, coagulation abnormalities, or neurological disorders. One day postoperatively, the patient experienced a frontal headache, which was assumed to be a postdural puncture headache because it was more intense in the sitting position. No associated neurological symptoms or signs were found. She was discharged from hospital, still complaining of the headache, two days later. She returned to the hospital with headache and nausea 15 days after the discharge. The patient had been treated with analgesic tablets and continuing bed rest, but her headache had become worse. In the following weeks the headache became progressively incapacitating and unresponsive to oral analgesics. Ahe was readmitted to our hospital with a severe headache and vomiting 21 days after the epidural anesthesia. There were no signs of neurological deficits. A computed tomography (CT) scan of the brain revealed isodense cSDH material of the right frontal region with 2 cm midline shift (Figure 1A). Cranial magnetic resonance imaging (MRI) showed a 2 cm-thick T1-weighted hypointense, T2-weighted hyperintense cSDH overlying the right fronto-parietal lobe with 2 cm midline shift (Figure 1B, 1C). Preoperative coagulation tests were within normal limits. The patient subsequently underwent right frontal and parietal burr holes with drainage under general anesthesia without complications. The postoperative course was uneventful. Her headache resolved completely. Postoperative cranial MRI and MRI angiography did not reveal an associated aneursym or arteriovenous malformation. The MRI showed marked resolution of the cSDH with correction of midline shift. The patient was discharged on the third postoperative day.

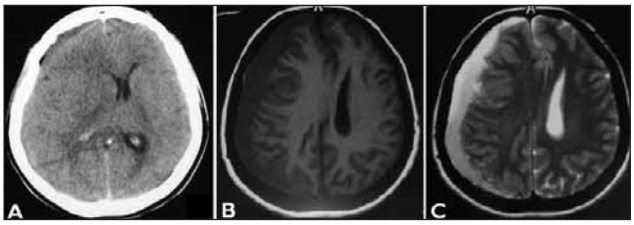

Figure 1: *Brain CT (**A**) showed an isodense cSDH material of the right frontal region with 2 cm midline shift. Cranial MRI showed a 2 cm-thick T1-weighted (**B**) hypointense, T2-weighted (**C**) hyperintense cSDH overlying the right fronto-parietal lobe with 2 cm midline shift.*

DISCUSSION

Intracranial subdural haematomas that appear later than 3 weeks after injury are called chronic and produce symptoms within the first week that clinically resemble acute subdural haematoma, while the symptoms of those that appear 1 to 3 weeks after injury are very much like cSDH (9).

cSDH following dural puncture is a very rare complication of epidural anesthesia. In 2006, Zeidan et al. (11) published their review of the literature containing 21 patients who developed intracranial subdural haematoma after unintentional dural puncture following epidural anesthesia. Only 2 of the 21 published cases of intracranial subdural haematoma were in non-obstetric patients (1,3). Twelve of these 21 cases were cSDH patients. There was only one patient with cSDH following epidural anesthesia in non-obstetric patients (3). We here present the second case of cSDH after unintentional dural puncture following epidural anesthesia in non-obstetric patients. However, the true incidence of cSDH after epidural anesthesia may be greater than published as most patients with headache are probably treated without further investigation.

A dural fistula can remain open for many weeks after dural puncture and the associated volume of cerebrospinal fluid (CSF) may be over 200 ml per day. The size of the needle and degree of dural tear is important. The extent of the leak increases with needle diameter. CSF leakage decreases both intraspinal and intracranial CSF pressure. Rapid CSF loss through the fistula created by the larger spinal needle may be responsible for this situation. The cause of cSDH is low CSF pressure following dural puncture leading to traction and tearing of thin-walled dural blood vessels (5,10,11). The cSDH may have been due to CSF hypotension in our patient.

Bilateral cSDH is more common in patients with prolonged coagulation times (6). Lee et al (4) reported that the shape and posture of the cranium can predict the location of the cSDH. Patients who have a symmetrical cranium develop bilateral cSDH more frequently than those who have an asymmetrical cranium. Therefore, the underlying coagulation times and cranial morphology may potentially be contributing factors in addition to the size of the dural tear. Aneursyms or arteriovenous malformations are thought to be contributing factors in the pathogenesis of cSDH (7,8). Several other etiologies of cSDH such as head trauma, coagulation disorders, anticoagulant drugs, brain atrophy, or alcoholism were ruled out in our patient. Postoperative cranial MRI and MRI angiography also revealed no associated aneursym or arteriovenous malformation.

Most physicians encountering a case of headache after epidural or spinal anesthesia first think of postdural puncture headache (PDPH). Postspinal headache develops due to leakage of cerebrospinal fluid (CSF) in 70% of the cases. The pain is characteristically relieved in the supine position, and associated with neck stiffness. Photophobia, diplopia and mild deafness are also commonly reported. The symptoms subside within 7 days when treated with analgesics and bed rest in the majority of cases (5,10).

The diagnostic procedure of choice for the evaluation of a cSDH is CT of the brain. Isodense cSDH is even more likely to result in misdiagnosis. Cranial MRI is more sensitive and specific than contrast CT in identifying isodense cSDH and obviates the risks of an intravenous contrast agent (9). Although there was an isodense cSDH in the cranial CT of our patient, we made the diagnosis as it had caused midline shift. Nevertheless, our patient's MRI scan revealed a thicker and larger cSDH than CT. Midline shift may also be absent as a result of bilateral cSDH formation.

Presumably, our patient's repeated presentations did not lead to a diagnosis because there were no symptoms of deteriorating mental status or focal neurological deficit. Our patient's persistent headache and vomiting were clues that led us to investigate an intracranial pathology. The presence of a suspected PDPH without neurological deterioration, as in this case, should prompt a search for an intracranial lesion.

CONCLUSION

Physicians that come across severe and persistent headache lasting more than a week in a patient with unintentional dural puncture following epidural anesthesia should take into account the possibility of intracranial lesion such as intracranial subdural haematoma.

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