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Coexistence of Spinal Ependymoma with Chronic Spinal Subdural Hematoma

Spinal Ependimoma ile Kronik Spinal Subdural Hematom Birlikteliği

Doğa GÜRKANLAR
Mehmet DANEYEMEZ
İlker SOLMAZ

Department of Neurosurgery,
Gülhane Military Medical Academy,
Ankara, Turkey

ABSTRACT

Although primary intradural-extramedullary ependymomas arising from the cauda equina or filum terminale are not rare, spinal ependymoma as a possible cause of chronic spinal subdural hematoma (CSSDH) following lumbar puncture has not been reported in the literature previously. A patient with progressive neurological deficit following lumbar puncture due to a CSSDH is presented. Possible mechanisms of spinal subdural hematoma are discussed. Magnetic resonance imaging (MRI) study of the lumbar region revealed the coexistence of chronic spinal subdural hematoma with the spinal tumor (ependymoma). The CSSDH was evacuated and the tumor was resected totally. Surgical evacuation of hematoma and total resection of the tumor resulted in almost complete recovery. A patient with progressive neurological deficit following a lumbar puncture should make us consider the coexistence of CSSDH with a spinal ependymoma.

KEY WORDS: Ependymoma, Lumbar puncture, Subdural hematoma, Spinal tumor.

ÖZ

Filum terminale vaya cauda equinadan köken alan primer intradural-ekstramedüller ependimomalar nadir görülmeseler de lomber ponksiyon sonrası kronik spinal subdural hematoma neden olan spinal ependimomaya daha önce literatürde rastlanmamıştır. Burada lomber ponksiyonu takiben gelişen kronik spinal subdural hematom (KSSDH) nedeni ile progresif nörolojik defisitli bir hasta sunulmuştur. Spinal subdural hematom oluşum mekanizmaları tartışılmıştır. Lomber bölgenin manyetik resonans görüntüleme (MRG) çalışması, KSSDH ile spinal tümör (ependimoma) birlikteliğini ortaya koymuştur. KSSDH boşaltılmış ve tümör tamamen çıkarılmıştır. KSSDH'un boşaltılması ve tümörün tamamının çıkarılması ile hasta neredeyse tamamen iyileşme göstermiştir.Lomber ponksiyonu takiben ilerleyen nörolojik defisit gelişen bir hastada KSSDH ile spinal ependimoma birlikteliğini akla gelmelidir.

ANAHTAR SÖZCÜKLER: Ependimoma, Lomber ponksiyon, Subdural hematom, Spinal tümör

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Correspondence Address:
Doğa GÜRKANLAR
Gülhane Askeri Tıp Akademisi
Beyin ve Sinir Cerrahisi Kliniği
Ankara, TURKEY
Phone : +90 312 304 53 18
Fax : +90 312 304 53 00
E-mail : dgurkanlar@hotmail.com

INTRODUCTION

CSSDH is not a common entity and only about 15 cases have been reported in the literature. Anticoagulant therapy, blood dyscrasias, trauma, vascular malformations, lumbar puncture and surgery have been proposed as causative factors. There have also been spontaneous cases (3,6,14,22).

We report the coexistence of CSSDH with a spinal intradural-extramedullary tumor (ependymoma) that occurred following lumbar puncture and discuss the possible mechanisms of hematoma formation.

CASE REPORT

A 15-year-old boy was admitted to our outpatient clinic with a history of left leg weakness following two lumbar punctures performed two months ago at the L4-5 level to find the etiology of high temperature. A few days after these lumbar punctures, he began suffering from progressive left leg weakness.

Although the patient was suffering from left leg weakness, motor strength was reduced in both legs (Grade 3/5 in the left and Grade 4/5 in the right). He also had hypoesthesia on the left L3-4-5-S1 dermatomes. Deep tendon reflexes were reduced in both lower extremities. There was no Babinski's sign.

His lumbar MRI revealed an intradural–extramedullary spinal tumor at the L3-L4 and L5 levels together with CSSDH that filled the tecal sac under the L5 level (Figure 1).

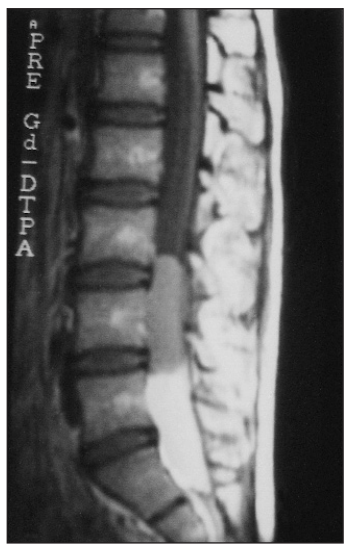

Figure 1: T1WI image revealing both the tumor and the subdural hematoma. Note the level between the tumor (above) and the subdural hematoma

He was operated on under general anesthesia. L3 and L4 total laminectomy, evacuation of subdural hematoma and total excision of the tumor with the arachnoid capsule were performed.

During the operation we observed that the tumor was vascular and that the dura around the tumor was rich in vessels that were probably the feeders of the tumor. We also observed defects on both the dura and the arachnoid capsule of the tumor. There was bleeding from the vessels around the tumor following CSSDH drainage.

Hematoxylin and Eosin studies revealed a spinal ependymoma.

After the operation he was free of pain, his motor weakness improved dramatically and he was able to walk without any help.

DISCUSSION

CSSDHs are rarely reported in the literature and there is no clear pathogenic mechanism of SSDH. They have been associated with many factors including lumbar puncture, anticoagulant therapy, blood dyscrasias, trauma, vascular malformations and surgery (3,6,14,22). Russel and Benoit found only 10 of 59 SSDHs to be of the chronic variety (22).

Lumbar puncture and anticoagulant therapy are associated with subarachnoid hematoma in 38%, with subdural hematoma in 44%, with subdural and subarachnoid hematoma in 88% and with epidural hematoma in 57% of the cases (21). Accidental or iatrogenic trauma was the causative factor in 46% of all spinal subdural hematomas (8).

There are controversies about the mechanism of spinal subdural hematoma formation. Gillian and Batson did not mention any subdural vessels (1,11) while subdural vessels were described by Djindjian et al. (4) and Manelfe (18). These vessels have been suggested as sources of bleeding despite their small caliber (7,12). After Djindjian et al. and Manelfe, Brandt (3) and Khosla (14) reported that spinal subdural space lacks vessels of sufficient size to be the source of bleeding. Russell et al. reported that a tear in fine subdural vessels with the added effect of flow reversal and indirect forces that may lead to hematoma formation (22). Based on the anatomical observations of Haines et al., the vulnerable inner spinal dura could be torn under certain circumstances, such as after lumbar punctures in a patient with a pre-existing coagulopathy (11).

There are a lot of descriptions of the myelographic findings in spinal subdural

hematomas, but there are no defined diagnostic criteria (2,12,15). In the past, the diagnosis of spinal hematoma has been made on a high degree of clinical suspicion and myelographic findings. More recently, MRI has proved very useful (20); it is non-invasive, has superior contrast resolution to CT, and shows the spinal cord and the entire spine to determine the extent of the spinal hematoma (10).

In our case, lumbar puncture alone could have been the reason of CSSDH formation by tearing the vessels in the subdural space around the tumor but bleeding from the tumor should be taken into account as an additional factor as we observed a defect in the arachnoid capsule of the tumor following lumbar puncture. The defect in the arachnoid capsule of the tumor following lumbar puncture could have let the blood flow into the subdural space and dilution by cerebrospinal fluid probably prevented clot formation within the subarachnoid space (6,7,16,19). Although primary intradural-extramedullary ependymomas arising from the cauda equina or filum terminale are not rare (23), spinal tumors were not included in the etiological classification of SSDH created by Khosla et al. (14).

It is known that the prognosis for spinal SDH is good if an evacuation or drainage is performed before irreversible damage occurs. Surgical outcomes are reported to be good in 42% of patients with spinal SDH, and our patient's neurological deficits also improved after the hematoma evacuation (5). However, in some cases of spinal SDH with mild symptoms, recovery might occur spontaneously with non-operative treatment (13, 17).

In conclusion, a patient with progressive neurological deficit following a lumbar puncture with a chronic SSDH should make us consider a spinal ependymoma besides other etiologies.

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