Spinal Chronic Subdural Hematoma Mimicking Intradural Tumor in a Patient with History of Hemophilia A: Case Report

Hemofili A Hastasında İntradural Tümör Taklit Eden Spinal Kronik Subdural Hematom: Vaka Sunumumu

ABSTRACT

A 22-year-old man with medical history of Hemophilia A was admitted with a 3-month history of low back pain radiating to the right leg. Neurological examination revealed no abnormalities. Spinal magnetic resonance imaging (MRI) with gadolinium enhancement revealed an intradural extramedullary mass at the level of L1 reported as an intradural tumor. The patient was operated after Factor VIII replacement. Intraoperatively, the lesion was found to be a pure subdural hematoma. The histopathological examination revealed pure chronic hematoma. Postoperatively the patient’s complaints showed improvement and he was discharged with no complications. Although MRI is the gold standard of diagnosis for spinal intradural tumors, some mass lesions remain difficult to diagnose. Spinal chronic subdural hematoma should be considered in the differential diagnosis in these patients, especially in those with coagulopathies, even in the absence of a history of trauma.

KEYWORDS: Hemophilia A, Spinal cord, Neoplasm, Intradural Tumor, Chronic, Subdural hematoma

ÖZ


ANAHTAR SÖZCÜKLER: Hemofili A, Spinal kord, Neoplazm, İntradural Tümör, Kronik, Subdural hematoma
INTRODUCTION

Most cases of spinal subdural hematomas (SDH) are of the acute or subacute type and to date approximately 90 cases have been reported in the literature (1). On the other hand, spinal chronic subdural hematomas (SCSDH) are extremely rare with only 27 cases reported (3). Although some of these cases (2 cases) have been documented in patients under anticoagulant therapy, the etio-pathogenesis remains unclear. The radiological features of SCSDH in MRI are distinct but some mass lesions are still difficult to diagnose (2).

We report a case of SCSDH in a patient with medical history of hemophilia A that was radiologically mimicking an intradural tumor.

CASE REPORT

A 22-year-old man was admitted with a 3-month history of low back pain radiating to the right leg with no history of trauma. The patient had a medical history of hemophilia A. Neurological examination revealed no abnormalities. X-ray examinations of the patient were normal. Spinal magnetic resonance imaging (MRI) with gadolinium enhancement revealed an intradural extramedullary mass at the level of L-1 with spinal cord compression. The lesion was round with clear margins, hyperintense in T1- and hypointense in T2-weighted images, with peripheral contrast enhancement and was reported to be an intradural tumor with hemorrhagic compartment (Figure 1A,B,C,D,F). Surgery was planned. Factor VIII replacement therapy was started due to abnormalities of partial thromboplastin time (PTT) and international normalization ratio (INR) in hematological investigations and continued until normal values were achieved. During surgery, the patient was placed in the prone position. After locating the L-1 level by fluoroscopy, the lesion was approached by a midline incision and T-12 and L-1 laminectomy. Under magnification of the operating microscope, the dura was opened and the lesion exposed with its semi-transparent round membrane covering a core of a semisolid mass of blood clot and yellow colored hemosiderin (Figure 2). The covering membrane was then dissected easily from the surrounding arachnoid bands. The lesion was carefully inspected under the operating microscope and no neoplastic pathology was seen. It was evaluated as pure hematoma and sent for histopathological examination after total resection. Histopathological examination confirmed the diagnosis of pure chronic hematoma with an organized neomembrane. Postoperatively, the patient’s complaints showed improvement with no neurological deficits. Factor VIII replacement therapy was continued until the patient was discharged on postoperative day 8 without complications. Radiological follow-up with spinal MRI showed no residual pathology and decompression of the spinal cord with normal anatomy (Figure 3).
DISCUSSION

Spinal acute and subacute SDHs are usually attributed to trauma, blood dyscrasia, anticoagulation, lumbar puncture, and vascular malformations (3). However, the etio-pathogenesis of SCSDH remains unclear. Tow mechanisms are thought to contribute to the pathogenesis of SCSDH. The primary lesion is an initial subarachnoid hemorrhage that eventually dissects into the subdural space, and the subarachnoid hemorrhage is washed out by cerebrospinal flow (6). The other theory states that spinal SDH may be related to redistribution of blood from the intracranial subdural space, and its expansion may be secondary to chronic changes (6). However, only three reports exist on SCSDH associated with intracranial chronic SDH (3). While an acute SDH is isointense with the spinal cord on T1-weighted images and shows a mixed signal on the T2-weighted images, a high signal is typically observed on both T1- and T2-weighted images in the late chronic stage. MRI signal changes in SCSDH are related to the presence of methemoglobin as a result of evaluation in the clot (2). No contrast enhancement is observed in SCSDH on T1-weighted images. The differential diagnosis of SCSDH includes traumatic disc protrusion, bleeding in a neoplasm, or extra-axial abscess. An intravenous contrast medium is necessary to exclude these other diagnoses (2). Although spontaneous remission is reported in some cases (5), evacuation of the clot is suggested to prevent ischemic changes due to compression (1). The prognosis is reported to be fair or good after early surgical decompression. The clinical features of SCSDH, such as progressive deficit along with fluctuating symptoms and poor recovery, are the result of arachnoiditis or arachnoidal fibrosis that causes vascular injury (1). Most cases of spinal SDH cases are of the acute or subacute type (1). On the other hand SCSDHs are uncommon and only 27 cases have been reported in the literature (2,6). Among these, there is only one report of SCSDH radiologically mimicking an intradural tumor with contrast enhancement (4) and no reports of any patient with medical history of blood dyscrasia.

CONCLUSION

Although spinal MRI is the gold standard of diagnosis in spinal cord neoplasms, some mass lesions remain difficult to verify. SCSDH should be considered in the differential diagnosis in these patients, especially in cases with coagulopathies, even in the absence of a history of trauma.

REFERENCES