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Case Report

Spontaneous Spinal Subarachnoid Hemorrhage with Spontaneous Resolution

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Spontaneous spinal subarachnoid hematoma (SSH) is a rare entity to cause spinal cord or nerve root compression and is usually managed as surgical emergencies. We report a case of spontaneous SSH manifesting as severe lumbago, which demonstrated nearly complete clinical resolution with conservative treatment. A 58-year-old female patient developed a large SSH, which was not related to blood dyscrasia, anticoagulation, lumbar puncture, or trauma. Patient had severe lumbago but no neurologic deficits. Because of absence of neurologicl deficits, she was treated conservatively. Follow-up magnetic resonance (MR) image showed complete resolution. Conservative treatment of SSH may be considered if the patient with spontaneous SSH has no neurologic deficits.

KEY WORDS : Spinal subarachnoid hematoma · Spinal cord · Spontaneous.

INTRODUCTION

A spinal subarachnoid hematoma (SSH) is rare cause of spinal cord or cauda equina compression. It is usually associated with several well-known predisposing factors, including coagulation abnormality, use of anticoagulants, arteriovenous malformation (AVM), spinal artery aneurysm, lumbar puncture and trauma[1,3,5,9]. It may also occur spontaneously, but the incidence is extremely rare [3,7,14,17]. We report a very rare case of spontaneous SSH, which developed then regressed spontaneously.

CASE REPORT

A 58-year-old female patient developed a large SSH that was not related to bleeding diathesis, anticoagulation, lumbar puncture, or trauma. Patient's general condition was good except severe lumbago. She suddenly developed low back pain but she had no leg pain or neurologic deficit including bladder dysfunction. She had no trauma history. Physical examination demonstrated no apparent motor weakness except for the severe low back pain. Patient had mild headache and stiffness of the neck among the first symptoms. There was no fever or leukocytosis. Diffuse high signal intensity was seen on T2-weighted MR images (Fig. 1), and high signal intensity was seen on T1-weighted MR images (Fig. 2) from L2 to S2 in the spinal cord suggesting late subacute stage hematoma. Computed tomography (CT) of the head showed no abnormality (Fig. 3). There were no any pathologic findings in whole spine T2-weighted MR image. Short-term follow-up MR imaging with gadolinium enhancement after 8 days was done for evaluation of any lesion that could be causes of SSH. However, it

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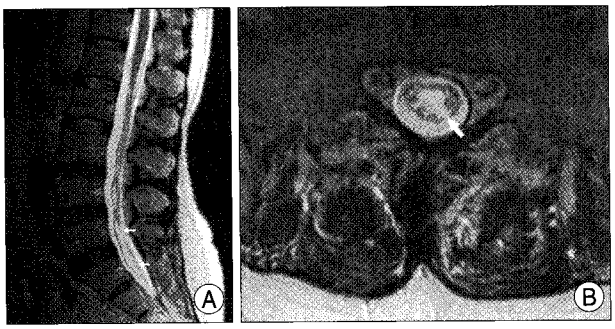

Fig. 1. A : Initial sagittal T2-weighted magnetic resonance (MR) image showing a large amount of subarachnoid hematoma (arrows) with diffuse high signal intensity from L-2 to S-2. B : Initial axial T2-weighted MR images showing a large amount of subarachnoid hematoma (arrow) with high signal intensity at the S-1 level.

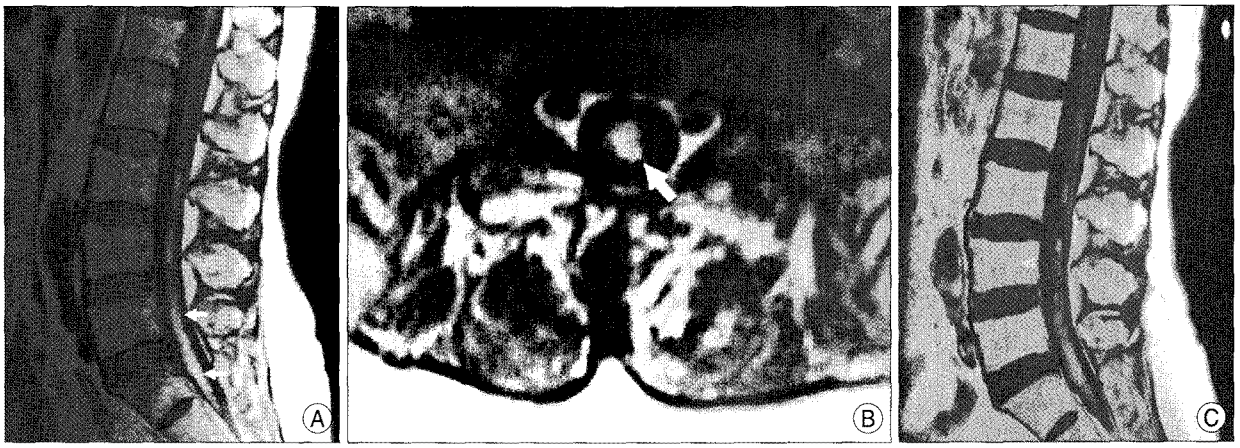

Fig. 2. A : Initial sagittal T1-weighted magnetic resonance (MR) image showing a large amount of subarachnoid hematoma (arrows) with diffuse high signal intensity from L-2 to S-2. B : Initial axial T1-weighted MR images showing subarachnoid hematoma (arrow) with high signal intensity at the S-1 level. C : Gadolinium-enhanced T_1-weighted MR images revealing no abnormal focal enhancement.

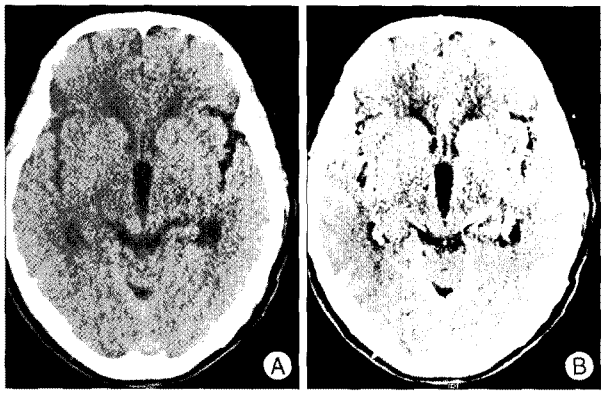

Fig. 3. A : Computed tomography (CT) of head. Precontrast image revealing no subarachnoid blood clot in arachnoid cistern. B : Contrast-enhanced CT scans demonstrating no abnormal enhancement of intracranial vessel.

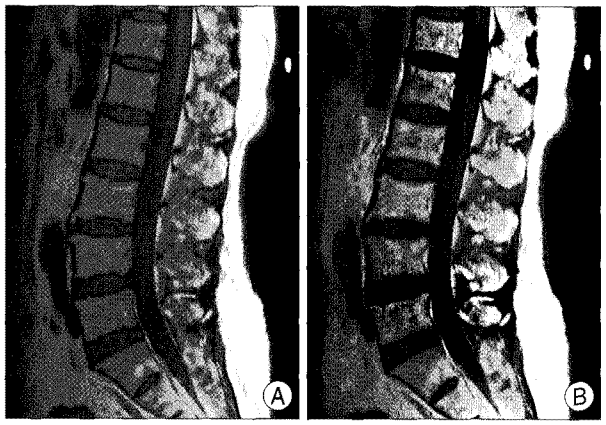

Fig. 4. (A) Sagittal T1-weighted and (B) gadolium-enhanced T1-weighted magnetic resonance images after 6 months, showing no abnormal enhancement.

failed to reveal the causes. The hematologic studies were normal including complete blood count, prothrombin time, partial prothrombin time, international normalized ratio, bleeding and clotting times, platelet count and coagulation factor analysis. She was not on anticoagulant therapy. The patient recovered completely after conservative treatment and discharged without any neurologic deficit. T_1-weighted MR imaging with gadolinium 6 months after onset showed complete resolution of hematoma and no findings of AVM and no tumor-like enhancement (Fig. 4). She has not complained any clinical symptoms for 49 months after initial diagnosis.

DISCUSSION

Subarachnoid hematoma affecting the spinal cord is very rare. According to the recent literature, Domenicucci et al.[3] reported 69 cases of SSH, but they found only 12 spontaneous ones. They were most often located in thoracolumbar areas; none of the cases of SSH at the lower lumbar and sacrum level have been reported. All except one reported to date had severe underlying diseases such as severe coagulopathy, malignant tumors including leukemia, degenerative vasculopathies[7]. Other reports described SSH due to spinal artery aneurysms[1,9,11], dural arteriovenous fistula (AVF)[8], melanoma[12], etc[15]. To the best of our knowledge, this is the first report about spontaneous SSH at the lower lumbar and sacrum level of which exact cause was not found. The causes of SSH are controversial. The most frequent causes of SSH are coagulopathies[3], lumbar puncture for diagnostic or anesthesiological purpose[13], trauma[5], vascular malformations[1,4,8,11], and spinal tumors[2,12,16]. In rare cases, they may be spontaneous[3,7,14,17]. Some authors believe that the causes of bleeding are rupture of the arteries and radicular veins, especially in iatrogenic SSHs[10]. Another theory is that the SSH may originate from primarily within the subarachnoid space. The spinal arachnoid mater is a connec-

tive tissue membrane closely attached to the dura mater and reflected off the surface of the spinal cord to ensheath blood vessels as they transverse the subarachnoid space[10]. In the case of a sudden rapid increase of abdominal or thoracic pressure, these are responsible for similar pressure variations in the spinal canal with rupture of the vessels, particularly the valveless radiculomedullary veins that cross the subdural and subarachnoid space. Some authors found cases of simultaneous SSH and spinal subdural hematoma (SSdH), which support theory that spontaneous SSdH might originate in the subarachnoid space, dissect the arachnoid membrane, and spread into the subdural space[14]. The lesion should be differentiated from other subdural hematoma, tumorous condition and vascular malformation including AVM and aneurysm. In this case, spinal angiography might have confirmed whether the patient had any vascular malformation or not. However, MR imaging with gadolinium enhancement was checked twice during follow-up periods, which failed to reveal any abnormal findings. Moreover, review of literature demonstrated that most cases having the vascular malformation such as AVM and aneurysm have abnormal signal in MRI before spinal angiography[1,4,8,9,11].

The differentiation between a subdural and subarachnoid hematoma remain difficult and at times only surgical exploration will prove the exact location of the hematoma. However, pure SSH like this case can be easily differentiated from the subdural hematoma. Subarachnoid hematomas are located within the thecal sac without an inverted "Mercedes star sign" that is typical finding in subdural hematoma[6]. A subdural hematoma may have a semicircular appearance, and tend to be more crescentic on axial images.

In general, acute SSH is a potentially dangerous condition and may have disastrous consequences. Though there are several case reports of SSH with spontaneous resolution, urgent decompressive surgery has been the primary treatment for SSH when the neurological state progressively deteriorates and conservative treatment is an option for only a few selected patients with minimal neurological impairment. MR imaging contributed essential information for diagnosis and follow-up after conservative treatment.

CONCLUSION

A spontaneous SSH, although very rare, should be considered in the diagnosis of spinal compression syndrome. Conservative treatment can be recommended for patients with stable neurological status.

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