Case Report

Spontaneous Resolution of Nontraumatic Acute Spinal Subdural Hematoma

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Spinal subdural hematoma (SSDH) is an extremely uncommon condition. Causative factors include trauma, anticoagulant drug administration, hemostatic disorders, and vascular disorders such as arteriovenous malformations and lumbar punctures. Of SSDH cases, those that do not have any traumatic event can be considered cases of nontraumatic acute spinal subdural hematoma, which is known to have diverse clinical progress. Treatment typically consists of surgical decompression and cases in which the condition is relieved with conservative treatment are rarely reported. We report two nontraumatic acute spinal subdural hematoma patients who were successfully treated without surgery.

Key Words : Nontraumatic acute spinal subdural hematoma · Spinal hemorrhage · Conservative treatment.

INTRODUCTION

Spinal subdural hematoma (SSDH) is a rare condition in which the spinal subdural hematoma compresses the spinal cord and the cauda equina, causing symptoms that may occur in relation to trauma, hemostatic disorders, anticoagulant drug administration, arteriovenous malformations, tumors, and lumbar punctures. Various unknown causes of SSDH have also been reported. Its clinical symptoms are sudden pain, hypesthesia, motor paralysis, and neurologic defects in some cases. Treatment generally consists of surgical decompression. We experienced two cases of nontraumatic acute spinal subdural hematoma (NASSDH), treated conservatively without surgery.

CASE REPORT

Case 1

A 55-year-old female patient came to the emergency room due to serious back pain that occurred during sleep. The patient had a past history of taking antihypertensive drugs and drugs to treat diabetes mellitus. Blood tests conducted in the emergency room did not reveal anything in particular. Paralysis of both lower extremities occurred after the patient came to the hospital and was classified as grade 1. An examination found hypesthesia at the T8 level and below and the deep tendon reflexes were reduced. We performed a chest computed tomography (CT) scan, but aortic dissection was not observed. We suspected hypokalemic periodic paralysis of both lower extremities and conducted laboratory studies; however, the results were negative. We then suspected thoracic myelopathy and thus provided steroid mega-dose therapy and conducted magnetic resonance imaging (MRI). The motor paralysis was gradually relieved so that the motor function were grade 2 on the right side and grade 3 on the left side four hours after the onset of symptoms. It was grade 4 on both sides after five hours. The MRI showed acute subdural hematoma at levels C2-T6 (Fig. 1A, C). Paralysis almost completely disappeared by 55 hours after onset. A cervical MRI conducted at that time revealed reduced cervical-dorsal SSDH (Fig. 1B, D).

Case 2

A 38-year-old male patient experienced headache, back pain, cold sweating, and dizziness that began when he was lifting weights one month earlier. These symptoms were relieved after he was treated at a clinic of pain medicine near his home. Three days later, he came to the emergency room with vertigo, which was relieved with intravenous hydration. Approximately one month later, his chest and back pain was aggravated by stretching exercises and he came to the emergency room. A brain MRI with contrast enhancement was conducted, which revealed thickening of the dura mater. The patient was then admitted to the neurologic department of the hospital with suspected spontaneous intracranial hypotension. The chest and back pain worsened and
acute urinary retention occurred. An MRI was conducted, revealing an acute subdural hematoma at levels C6-T5. The patient was then transferred to the neurosurgery department (Fig. 2A, D). At that point, his motor functions were intact and there was hypoesthesia at T2 and below. He was also experiencing deep tendon reflexes and ankle clonus reflexes accentuation. After steroid therapy, the symptoms were relieved for approximately two weeks and the patient was discharged. He was followed up with an MRI at two and six weeks after discharge. The hematoma observed earlier gradually decreased and almost completely disappeared and the patient’s clinical condition greatly improved (Fig. 2B, C, E, F).

**DISCUSSION**

Nontraumatic spinal hemorrhage is a rare condition that can be epidural, subdural, or medullary and in terms of frequency, it occurs in that order. Most cases are acute and neurologic defects quickly manifest. The major symptoms are sudden pain, motor paralysis, and sensory paralysis. These symptoms are progressive in many cases. An MRI is an appropriate method of identifying lesions in the spinal cord. Generally, using slight contrast enhancement, iso-hyperintensity is found on T1 weighted images (WIs) and heterogeneous hyperintensity is found on T2WIs. It is known that if no vascular disorders, such as arteriovenous malformations, are found upon MRI, selective spinal angiography will not be performed. Treatment typically consists of surgical decompression and management of basal diseases, such as blood disorders.

The cause and the origin of NASSDH are undefined. According to several authors, a forgotten effort can increase both the intrathoracic and intraabdominal pressure of the vessels, particularly the radiculomedullary veins crossing the subdural and the subarachnoid space. If the cerebrospinal fluid (CSF) does not instantly neutralize this force, the sudden increase in pressures can cause rupture of the vessels located on the inner surface of the dura, resulting in a subdural hemorrhage. Domenicucci et al. have described the presence of combined subdural and subarachnoid hemorrhage in the same patient. This has led them to suggest that subdural hemorrhage may have originated in the more vascular subarachnoid space and passed through the thin and delicate arachnoid membrane. However, the dilution and redistribution of the CSF prevent clot formation unless the hematoma is large enough to block the CSF flow. On the contrary, the origin of the hematoma can be the subdural space and then can permeate through the arachnoid membrane, giving rise to an associated subarachnoid hemorrhage. There is a ques-
tion whether this fine network of vessels can be the source of hemorrhage.

On the review of the literature, 106 cases of NASSDH were analyzed. There was good prognosis for 42% of the 59 patients who underwent surgical treatment. The remaining patients either had a poor prognosis or died. Kyrilakides et al. reported that patients with rapidly progressive paralysis were diagnosed with NASSDH and emergency laminectomies and hematoma evacuations were performed. At four months after surgery, the patients showed complete recovery of motor functions while showing remaining spasticity and overactive bladder. The good prognosis of receiving conservative treatment for NASSDH was 43%. The remaining 57% of conservative treated group died or had poor prognosis. Some deaths were related to chronic spinal subdural hematoma; others were unrelated to NASSDH.

A review of cases in which treatment was conservative revealed that patients’ symptoms were gradually relieved within several hours. Representative cases were described by Oh et al. and Hentschel et al., who reported quick recoveries within 10 minutes, four hours, and three hours after the onset of symptoms for NASSDH patients with motor grade 3 or 4 who did not have serious neurologic defects and who received non-surgical treatment.

As mentioned above, NASSDH patients show diverse treatment results and prognosis. The type of treatment depends greatly on patients’ initial neurologic condition and whether basal diseases are present. Although it is clear that in NASSDH, surgical treatment of progressive spinal cord diseases should be considered, such treatment may aggravate damage to the spinal cord and hemorrhaging may be accelerated after surgery due to hemostatic disorders.

Based on the literature review, there was no objection to surgical treatment when patients: 1) had basal diseases such as vascular malformations, 2) had progressive neurologic defects and did not show any signs of relief, or 3) showed serious spinal cord compression upon MRI. Based on analysis of cases in which conservative treatment was successful, it can be assumed that there were no basal diseases such as vascular malformations, edema was observed to be more serious than spinal cord compression upon MRI, neurologic defects were not aggravated, and patients responded to steroids and other medications.

Of the two cases presented in this study, the first patient showed gradual recovery from motor paralysis after being treated with steroids. Thus, surgical treatment was deemed unnecessary. The second patient experienced pain and hypoesthesia as the major symptoms and had minor motor paralysis. Thus, non-surgical treatment was selected. According to Maeda et al., if symptoms are relieved after treatment for acute phase edema and if initial neurologic conditions are Frankel Grade D or higher, conservative treatment is recommended.

CONCLUSION

We reported two cases of NASSDH in which symptoms were relieved by non-surgical treatment. We therefore suggest that conservative treatment to be selectively considered for all cases of NASSDH.

References