Acute spontaneous spinal epidural hematoma (ASSEDH) is one of the rarest of all kinds of bleedings occurring within the craniospinal cavity [20,34]. Despite its characteristic presentation with sudden pain and rapidly progressive neurologic deficit, the clinical diagnosis of ASSEDH has been difficult, often being made at the time of operation.

In the following report, the author describes a new case of ASSEDH. The aim is to draw attention to this rare clinical entity and discuss the influence of magnetic resonance imaging (MRI) on its diagnosis and treatment.

Case Report

This 79-year-old man presented to our hospital with a history of severe middorsal pain, which started suddenly 1 week earlier when he attempted to lift a heavy object. The pain was followed by heaviness in both legs and difficulty walking. His past medical history was unremarkable, and he was not on any medication.

The patient was initially admitted to a local hospital, where MRI was obtained 5 days after the onset of symptoms, and it showed a fusiform extradural mass lesion at the level of D6 to D8 with significant spinal cord compression. The lesion was isointense with the spinal cord on T1-weighted images (T1WI) and hypo intense on T2-weighted images (T2WI); it enhanced mainly in its periphery following intravenous injection of gadolinium-labeled diethylene triamine pentaacetate (Gd-DTPA) (Figures 1 and 2).

On general examination, the patient was found to be fit with no chest, cardiovascular, or abdominal abnormalities. There was mild tenderness in the middorsal segment of the spine. The neurologic examination revealed normal higher mental functions, cranial nerves, and upper extremities. In the lower extremities, however, the muscle tone was slightly elevated, while the deep-tendon reflexes were weak, and plantar responses equivocal bilaterally. The power of the proximal leg muscles was
Midsagittal MRI of the dorsal spine demonstrating a biconvex intraspinal mass lesion at D6 to D8, compressing the spinal cord. The lesion is isointense on T1WI (A), enhances peripherally after intravenous injection of Gd-DTPA (B), and appears hypointense on T2WI (C).

slightly reduced, and there was considerable impairment of proprioceptive sensation with unsteadiness of gait.

The patient underwent urgent decompressive laminectomy from D6 to D8 with total excision of a dark extradural mass that proved histopathologically to be an organized hematoma without any evidence of a tumor or vascular abnormality.

The postoperative recovery was uneventful. The patient was discharged on the tenth postoperative day with good improvement in the feeling of heaviness in the legs. At last follow-up examination 4 months after the operation, he was asymptomatic and his neurologic status had normalized.

**DISCUSSION**

ASSESDH usually develops on the basis of a predisposing condition, including anticoagulant therapy [20,24,34,39,40], bleeding tendencies of all kinds [21,37,41,43,44], vascular malformations [14,36,48], hypertension [20], immunovasculitis [19], and Paget's disease of the spine [23]. However, in as many as 40% of the cases no obvious cause or predisposing factor is detected [20], as demonstrated by our case. Most of the patients present in their 60s and 70s, but all age groups from 6 months [41] to 86 years may be affected, with a slight predominance of the male sex [20].

The clinical picture of ASSES DH is characterized by sudden onset of severe spinal pain, with or without radicular radiation, followed by rapidly progressive symptoms and signs of cord or cauda equina compression. Rarely, however, patients may present with slowly progressive [7,27] or relapsing [15,25] symptoms, which may add to the diagnostic difficulties of the condition. The neurologic deficits may be mild [39] or complete [20,34] and, based on
the location of the hematoma, they can be bilateral, or show features of a Brown-Sequard syndrome [6,17,37,48] or anterior cord syndrome [20]. In the lumbar region, the symptoms of ASSEDH may be indistinguishable from those of an acute intervertebral disc prolapse [30,35]. The exact origin of ASSEDH is still controversial. While the most popular hypothesis postulates that spinal epidural venous plexus is the source of this type of hematoma, Beatty et al [3] argued that the pressure in the epidural cervical venes is lower than intrathecally, and thus a venous bleeding cannot compress the cervical spinal cord. The reason for the predominant occurrence of ASSEDH in the cervical and dorsal regions of the spine is also not clear [20]. In contrast, the frequent location of ASSEDH posterior to the dura could be better explained theoretically by higher stretch and shear forces during spinal movements on the epidural vessels lying posterior than on those lying ventral to the dura.

The value of MRI in the diagnosis of ASSEDH has been well demonstrated in several recent publications [2,4,6,9,10,12-14,16,21,26-30,32,35,39,41,45-48]. MRI gives accurate information not only concerning the location and extension of the hematoma, but also the degree of the cord compression, as well as any preexisting lesion that might have been the source of the bleeding, such as an arteriovenous malformation [14]. Typically, an ASSEDH appears on MRI as a biconvex mass that is separated from the underlying neural structures by a curvilinear low-signal structure, representing the dura mater [6]. The T1 and T2 signals of ASSEDH vary based on the clot, age, size, and oxygenation [22]. Within the first 24 hours after onset, ASSEDH is usually isointense with the spinal cord on T1WI and hyperintense on T2WI, but after 24 hours it gives high signal on T1WI and low signal (similar to that of the cerebrospinal fluid) on T2WI [6]. Old hematoma (more than 21 days) shows, in contrast, high signal intensity on the T1WI and mixed (low and iso) signal intensity on T2WI [27]. The intravenous injection of Gd-DTPA mostly results in peripheral enhancement [12] similar to that shown in our case, but a central enhancement may be seen occasionally [10].

It is interesting to note that since the first report on MRI in the diagnosis of ASSEDH was published in 1987 [45], some 51 cases of ASSEDH have been described in the literature [1,2,4-6,9-18,21,23,26-33,35,38-42,44-50]. A comparison of this figure with the estimated 260 cases of ASSEDH that had been documented during the period from the time of the first report in 1869 until 1986 [34] shows that the mean incidence of reported cases of ASSEDH has increased since 1987 from 2.2 to 6.4 new cases per year. More striking, however, is a remarkable concomitant rise in the rate of ASSEDH cases recovering spontaneously from 1.5% (4 out of 260 cases) [8,24,25,43] before 1987 to 29% (15 out of 51) [1,6,11,17,18,21,30,31,41,47,50]. These findings suggest that MRI has greatly facilitated the diagnosis of ASSEDH allowing for more cases with a benign natural course to be detected. Such cases seem to have often escaped clinical diagnosis in the past because of the relative paucity of their symptoms and signs, or the very early onset of recovery. Consequently, the traditional unreserved pleading for urgent neurosurgical decompression in ASSEDH may not be justifiable in all cases. Instead, the choice of treatment should be decided for each case individually, based on the severity of the neurologic impairment, the course of the illness from the time of onset to the time of diagnosis, and the MRI findings. A conservative approach under close neurologic observation is recommended for patients with no, or only mild and not disabling, neurologic deficit and for patients who show definite progressive improvement prior to the MRI diagnosis of ASSEDH. Occasional difficulties in differentiating ASSEDH from other intraspinal space-occupying lesions may be overcome by serial MRI studies demonstrating progressive resolution of the clot. A repeat MRI is also useful in excluding any underlying structural lesion that could have been missed in an earlier examination. In contrast, patients presenting with disabling neurologic deficits will continue to need urgent neurosurgical decompression.

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REFERENCES


COMMENTARY

This article nicely reviews the subject of spontaneous spinal epidural hematoma. The diagnosis is made more readily since the advent of MRI. In fact, I have seen three recent cases in our own practice. The MRI appearance of the hematoma can be quite variable, depending upon the age of the clot, and we did a great deal of pondering about the nature of the epidural mass in several cases. As the author points out, operation is not always necessary. The epidural hematoma in one of our patients resolved spontaneously with good neurologic recovery.

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This is a well-written article reviewing this neurologic condition. Professionally, I find ASSEDH easy to diagnose and easy to treat, with MRIs being widely available for spinal diagnoses. In the past, we would see spinal epidurals following anticoagulation therapy, particularly in younger people when they are anticoagulated for their prosthetic heart valves and less commonly for cerebrovascular insufficiency. We have had MRI available in our area since 1987, and since then, we seem to be seeing less of this condition, possibly because internists are using less anticoagulation or at least different types of anticoagulation regimens for underlying medical conditions.

Our two most recent cases of acute spinal epidural hematomas in the past month were:

(1) Following a lumbar epidural steroid injection in a 75-year-old woman by an experienced anesthesiologist. There was no underlying bleeding diathesis or medical disease that we could ascertain in this elderly woman; she became paraplegic within 3–4 hours. Possibly this clot was arterial in origin. At surgery, we discovered that the clot extended from T10 to L3.

(2) My associate, along with an orthopedist, operated on a woman who had a Chance fracture at L1, following a motor vehicle trauma. There was a small spinal epidural associated with that fracture, although the woman remained neurologically intact.

In our community, we rarely see ASSEDH. The spinal epidural hematomas we see are usually associated with injuries.

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