Chronic spinal subdural hematomas

Report of two cases

VIRENDER K. KHOSLA, M.S., M.CH.(NEURO), VIJAY K. KAK, M.S., F.R.C.S., F.R.C.S.(ED),
AND SURESH N. MATHURIYA, M.S., M.CH.(NEURO)

Department of Neurosurgery, Postgraduate Institute of Medical Education and Research,
Chandigarh, India

Two patients with chronic spinal subdural hematomas are described. Both had a fluctuating clinical course, not previously documented in the literature. Surgical evacuation resulted in almost complete recovery. The pathogenesis of spinal subdural hematoma is discussed and the pertinent literature is reviewed.

KEY WORDS • spinal hematoma • subdural hematoma

S PINAL subdural hematomas are rare and only 10 cases of the chronic type have been described in the literature. We are reporting two patients with chronic spinal subdural hematoma, both of whom had a "fluctuating" clinical course, which has not been previously associated with this entity. Besides minor trauma, neither patient had any other abnormality. Although the prognosis is reported as poor in such cases, even with surgery, our patients had a good recovery.

Case Reports

Case 1

This 40-year-old man lost consciousness for 45 minutes on January 9, 1977, while out walking. When he recovered, he noticed weakness of both lower limbs and inability to pass urine.

Examination. Examination the next day revealed an alert patient with paraparesis (muscle power 3/5) and a sensory level at T-10. Blood count, urinalysis, and bleeding and clotting times were normal. Radiouclide studies of the dorsal spine revealed no abnormality. Within 24 hours, the patient showed remarkable improvement (muscle power 4+/5) and was therefore discharged. He remained well for 4 weeks. He then developed fever for a few days, followed by recurrence of his lower-limb weakness.

On examination on February 11, 1977, he had spastic paraparesis with weakness more marked on the left side. He had 3/5 muscle power and was unable to walk. Sensation was absent below T-10, and the bladder was distended. Deep-tendon reflexes were brisk in both lower limbs. Spine films were normal. Radionuclide studies of the dorsal spine were again normal. A hemogram and urinalysis revealed no abnormality. Myelography disclosed a complete extradural block at T-11.

Operation. A laminectomy from T-11 to L-1 was carried out on February 21, 1977. No extradural pathology was found. When the dura was opened, the arachnoid was found to be thick and opaque with adhesions. Incision of these adhesions released about 7 to 10 ml of dark blood with a few clots. The wound was closed after decompression. Histopathological study of the arachnoid showed organizing non-specific inflammation. The patient improved and was able to walk unaided within 2 weeks.

Case 2

This 45-year-old man was admitted with a 1-month history of dull nonradiating neck pain. Neck massage aggravated the pain, which later gradually subsided. A week prior to admission he began to develop a progressive left hemiparesis, difficulty in micturition, and inability to walk without support.
Chronic spinal subdural hematomas

Examination. At admission on January 28, 1983, the patient was fully alert and had no systemic problems. The left hemiparesis persisted, with the upper limb being weaker than the lower, and he had a sensory loss with an acute, subacute, and chronic variety. The acute or subacute types usually present with sudden rapid onset of back pain, with progressive pain at a lumbar puncture site, or with radicular symptoms, fever, and meningismus. A sudden onset and rapid course indicate a vascular etiology. The chronic variety produces progressive painless cord compression without any clue to its vascular origin, and spontaneous remission has been reported. Rarely, these lesions may be found only at autopsy.

Spinal subdural hematoma is more common in females and is most frequently seen in the thoracic and thoracolumbar regions. The commonest age group affected is that between 50 and 70 years old. Acute, subacute, and chronic varieties have all been described. The acute or subacute types usually present with sudden rapid onset of back pain, with progressive pain at a lumbar puncture site, or with radicular symptoms, fever, and meningismus. A sudden onset and rapid course indicate a vascular etiology. The chronic variety produces progressive painless cord compression without any clue to its vascular origin, and spontaneous remission has been reported. Rarely, these lesions may be found only at autopsy.

We wish to suggest an etiological classification (Table 1) which should help in creating clinical awareness and thus assist in improving therapeutic results. Hematological disorders coupled with trauma (including lumbar puncture) are present in 84% of all spinal subdural hematomas, and the remaining 16% fall into a miscellaneous category. Thrombocytopenia, leukemia, hemophilia, and anticoagulant therapy are significant predisposing factors in 38% of all reported cases. In the miscellaneous category of spinal subdural hematomas (not associated with trauma or

---

**Fig. 1.** Preoperative myelograms in Case 2. Left: Anteroposterior projection showing a complete block at C-2. The subarachnoid space on the left is enlarged by a cup-shaped filling defect. Right: Lateral projection showing the filling defect situated posteriorly.
vascular anomalies), anticoagulant therapy and lumbar puncture were associated in 38% of cases with subarachnoid hematoma, in 44% of cases with subdural hematoma, in 88% of cases with both subdural and subarachnoid hematomas, and in 57% of cases with epidural hematoma. The importance of lumbar puncture in such situations is well emphasized.

Trauma, accidental or iatrogenic, accounts for about 46% of all spinal subdural hematomas. Severe spinal injury rarely produces subdural hematoma. Systemic disease and arteriovenous malformations producing "spontaneous" subdural hematoma have been reported. Certain unusual spinal hematomas have also been documented. A wide range of myelographic findings have been described, varying from intradural filling defects to a complete block; but no diagnostic criteria are defined. At times, even the distinction between extradural, subdural, and subarachnoid hematoma may not be possible. Appearances suggestive of a subdural injection of contrast medium or normally placed ipsilateral axillary pouches with subarachnoid contrast material displaced to the opposite side are good leads to a correct diagnosis. Cerebrospinal fluid was abnormal in all cases tested.

Controversies exist regarding the source of bleeding and mechanism of formation of spinal subdural hematoma. Various possibilities have been put forward. Although Gillilan and Batson did not mention any "subdural vessels," these have been described by Djindjian, et al., and Manelfe and, in spite of their small caliber, these vessels have been suggested as sources of bleeding. "Indirect forces" and "unusual forces" have been implicated in the formation of spinal subdural hematomas. Although fine subdural vessels are not accepted as a source of bleeding, a tear in these vessels with the added effect of flow reversal and "indirect forces" may lead to hematoma formation. Hemorrhage originating solely in the subarachnoid space has also been reported. Dilution by cerebrospinal fluid usually prevents clotting of blood within the subarachnoid space, but subarachnoid blood can enter the subdural space through an arachnoid tear. A large subarachnoid clot causing spinal block can predispose to further clotting, with additional enlargement of the clot and rupture into the subdural space. Some factors that encourage formation of subarachnoid clot have been described. Formation of acute or subacute spinal subdural hematoma is possibly due to a combination of various factors as described above, rather than to a single cause.

The pathogenesis of chronic spinal subdural hematomas, however, remains ill-understood. Related mostly to minor trauma, they are usually found in the elderly, but children are not exempt. A fluctuating clinical course, as observed in both our patients, has not been documented in the literature, but two similar case histories were found. Although Russell and Benoit (personal communication, 1984) have suggested that enlargement of a chronic spinal subdural hematoma may induce this clinical course, the literature offers no proof to this effect. Computerized tomographic studies of this entity are not available in the literature, but should be of immense help. Even if the hematoma increases in volume, it is not known whether the mechanism is akin to that of its cranial counterpart. We suggest that most of the features, like a progressive deficit with fluctuating symptoms and poor recovery, are the result of arachnoiditis or arachnoidal fibrosis causing vascular insult. Enlargement of the hematoma and the subsequent mass effect are of lesser importance. The degree of recovery is directly related to the vascular insult to the cord. Both of our patients showed good recovery due to timely surgery.

Spinal subdural hematoma should be considered as a possible diagnosis in cases of progressive spinal neurological deficit with hematological disorders, minor trauma, or lumbar puncture. Only clinical awareness will lead to early diagnosis, timely surgery, and good results. A delayed diagnosis often results in a poor outcome.

References


V. K. Khosla, V. K. Kak and S. N. Mathuriya
Chronic spinal subdural hematomas

23. Klauer R: Cited in Reference 31

Manuscript received December 27, 1984.
Address reprint requests to: Virender K. Khosla, M.S., M.Ch.(Neuro), Department of Neurosurgery, Postgraduate Institute of Medical Education and Research, Chandigarh 160012, India.