SPINAL EPIDURAL HEMATOMAS
EXPERIENCES WITH THREE PATIENTS

JOHN J. LOWREY, M.D.
Department of Neurological Surgery, Straub Clinic, Honolulu, Hawaii

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In the past three years, I have explored 3 patients with spinal epidural hematomas unassociated with spinal fractures or a bleeding diathesis.

A search of the literature indicates that this condition is rare. Sadka\textsuperscript{11} and Schultz \textit{et al.}\textsuperscript{12} in 1953 in separate articles listed the same 12 cases. Sadka\textsuperscript{11} added 2 and Schultz \textit{et al.}\textsuperscript{12} 4 more cases. Since then reports of 3 more cases have been found in the English literature. Aymes \textit{et al.}\textsuperscript{1} reported 1 (2 reported patients did not fit the above criteria) in 1955, Nichols and Manganiello\textsuperscript{9} 1 in 1956, and Maxwell and Puletti\textsuperscript{8} 1 in a child in 1957, making a total of 21. The last authors listed Jackson's case in 1869. This is not included, as the reference was not available. The cases are summarized in Table 1 taken directly from Sadka's\textsuperscript{11} article and adding the subsequent cases including the 3 reported here. The table is similar to Kaplan and Denker's\textsuperscript{7} earlier table in 1949.

Age and sex do not appear to be factors. Patients from 1\textsuperscript{3} to 79 years of age are reported. There were 15 males and 8 females. The sex of the 18-month-old baby is not stated.

Any segment of the spine may be involved, and the hematoma in 1 patient (Reid and Kennedy\textsuperscript{10}) extended from the 3rd cervical to the 2nd sacral vertebra. In most patients the hematoma extended over 2 or 3 segments.

HISTORY AND PHYSICAL FINDINGS

As seen from Table 1, most patients associated some mild trauma or strain with the onset of symptoms. The initial symptom was almost invariably pain in the neck or back at the level of hemorrhage, followed within a matter of hours, days or even weeks by varying degrees of motor and sensory paralysis. Weakness was sometimes delayed in onset, then rapidly progressive, resulting in complete paraplegia. In other patients the weakness was slowly progressive or occasionally intermittent.

DIAGNOSIS

Roentgenograms of the spine were not helpful. Lumbar puncture showed anything from a complete block to normal dynamics. The spinal fluid showed levels of total protein from normal to several hundred milligrams per cent. Myelography is the most useful procedure, but even this may be normal, as reported by Sadka\textsuperscript{11} in both of his cases. Lumbar puncture may be followed by a temporary improvement in motor and sensory signs leading to an unfortunate delay in treatment.

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Laminectomy is obviously the only method of treatment, and the earlier performed the better because many patients with slowly developing signs may suddenly get worse. The age and risk of the patient should not delay us from offering the only treatment that may lead to recovery in these patients. Surgery unfortunately was delayed 2 days in the first patient here reported because first, it was felt her lesion was caused by a thrombosis of the cord and second, because her age of 71 years and her fixed chest (rheumatoid arthritis) were felt to contraindicate surgery. The age and fused spine resulting from rheumatoid arthritis in this patient are surprisingly similar to those in Ver Brugghen’s patient.16

PROGNOSIS

As one would predict from Tarlov’s work, the patients with slowly developing incomplete lesions do better. Some patients showed poor recovery even when the cord looked normal. The extensive clots in some patients probably interfered with the nutrient vessels to the spinal cord. Many patients had to be transferred to suitable hospitals for laminectomy and the changing neurological signs are difficult to follow. The suddenness of onset in some patients suggests arterial bleeding. The bleeding in Case 3 reported here was probably arterial.

ETIOLOGY

In some patients, bleeding apparently arises from a previously existing hemangioma. This was apparently the explanation in the case of Nichols and Manganiello.9 The intermittent attacks of pain in the chest for 7 months prior to the acute episode suggest a pre-existing lesion in this patient.

Most patients with this condition, however, have had no previously existing symptoms at the level of the lesion and have nothing to suggest a hemangioma at the time of surgery. In the case reported by Maxwell and Puletti,8 the discrete clot arose from a single epidural vein. The source of bleeding usually is not discovered. The complete lack of prior symptoms at the site of the lesion argues against a pre-existing hemangioma.

CASE REPORTS

Case 1. K.B., a 71-year-old housewife, was seen a few hours after she fell off a chair, striking the right side of her head slightly. She was not unconscious and immediately got up and walked but complained of pain in her neck. She noted weakness of the legs within an hour and paralysis within 4 hours.

Neurological Examination. There was tenderness at C7. There was good movement of the right arm and left upper arm. The left forearm and hand were paralyzed. There was paralysis of the left leg and weakness of the right leg. Sensation was intact. The patient had a completely fused spine from old healed rheumatoid arthritis. There was no expansion of the chest. Lumbar puncture showed normal dynamics. The fluid contained no cells and a total protein of 41 mg. per cent.