Dear Sir,

We would like to present a case of ‘Spontaneous late onset extradural hematoma in spine: post-traumatic’. Spinal epidural hematomas (EDHs) have been reported to occur in patients with anticoagulant therapy, hypertension and following trauma.\textsuperscript{1-3} Subdural hematoma and subarachnoid bleeding following trauma also have been reported rarely in literature. We recommend that in the presence of significant hematoma in spinal cord causing compression, surgical evacuation is safer and would relieve the symptoms.\textsuperscript{3} In this article, we report a fortunate case of recovery after an unusual spine cord decompression. We also review the current literature concerning diagnosis and treatment of EDH in spine.

A 70 years old male patient was sent to our emergency room (ER) with a chief complaint of sudden onset of quadriplegia since 1 hour prior to presentation. He had a history of road traffic accident (RTA) on 24 September 2014, following which he got sustained injury to neck and right lower limb. He was taken by relatives to the nearest hospital and investigated. Investigation suggestive of no abnormal changes in X-rays and he was discharged on the same day. After the period of 10-day on 3 October 2014, he came to us with quadriplegia. Sagittal magnetic resonance imaging (MRI) showing disk herniation at C4-5, C5-6 and C6-7 level and EDH from C7 to T4.

The patient complained of neck pain. On neurological examination, the patient was conscious and alert with quadriplegia of power 3/5. There is no muscle atrophy of the extremities. Bowel and bladder function was intact. Other physical examinations were normal. The hematological data, including platelet count and function of the coagulation cascades, were normal. Since a cervical spinal pathology was suspected, cervical spinal X-ray and MRI were performed. Cervical spinal MRI showed an isointense, mass situated on the dorsolaterally, extending from C7 to T4 on T1-weighted images. The T2-weighted image displayed a heterogeneous signal within the lesion (Fig. 1). A dorsolateral hyperdense biconvex lentiform mass extending from C7 to T4, with compression of the cord with cord edema seen with multiple level annular bulging of disk at C4-C5, C5-C6, C6-C7 which causing compression of nerve root. Intraoperatively, D1-D2-D3 spinous process excised, laminectomy done on exploration the ligamentum flavum was contused, discolored. After excision of ligamentum flavum, a large EDH was found which was compressing the cord. Liquid blood with solid clots were found and evacuated. The dura resumed its normal position and showed good respiratory pulsations (Fig. 2).

No discrete bleeding point was identified, neither tumor nor abnormal vessels were found in the epidural space. The limb weakness was markedly improved immediately after the operation.

Spontaneous spinal epidural hematoma is rarely the cause of spinal cord compression. It was first described by Blauby in 1808.\textsuperscript{9,10} Acute spinal EDH can be classified as spontaneous or secondary to trauma.\textsuperscript{11} The infrequent occurrence of spontaneous spinal EDH makes its clinical diagnosis unlikely before surgical exploration has been undertaken.
pain and the subsequent development of neurological signs will suggest a differential diagnosis that includes disk prolapse, extradural abscess, dissecting aortic aneurysm and extradural tumor. Acute vascular lesions of the spinal cord, such as hematomyelia and spinal artery thrombosis are not usually associated with pain while the neurological sequelae immediately follow the occlusion. The main differential diagnosis is a prolapsed intervertebral disk and this is important, because the direction of surgical approach would then be anterior in the cervical region. The clinical features of spinal EDH and disk prolapse are similar whether they occur at the cervical, thoracic or lumbar levels. Pain is the usual presenting symptom and may well have a radicular distribution. The onset of neurological signs may be delayed by hours, days or in a small percentage of cases by weeks. The pain often subsides with the onset of neurological symptoms which usually take the form of a flaccid motor weakness, urinary retention and disturbance of sensation. Variations in the neurological deficit have been reported including the central cord symptom, Brown-Séquard syndrome and rarely no sensory deficit. The cause of the free interval (lucid interval) is not easy to explain, especially considering that it may be hours or weeks in duration. In our case, the free interval was 12 days. The only effective treatment is prompt and adequate decompression of the spinal cord by laminectomy and evacuation of the hematoma. Results show that this will cure or greatly improve the majority of patients, especially in the younger age group. Delayed decompression usually means a poor neurological result and conservative management is not recommended. In our cases, the spinal cord was compressed by the hematoma but cord pulsation returned when it was evacuated. Patient has made a very satisfactory recovery. Spinal EDH occurring spontaneously or after any mode of trauma has been attributed most often to a venous source. Predisposing factors include: coagulopathy, anticoagulation, vascular anomaly, disk herniation, Paget disease of bone, valsalva maneuver and, possibly, hypertension. Extravascular hematoma is a rare cause of spinal compression. This is important in order that the correct surgical approach be made. The condition often responds favorably to early decompression as in our case. Outcome after surgery is somewhat variable. Several authors recommend early decompressive surgery and suggest that if the operation is performed before interference with blood supply to the cord occurs, the prognosis is favorable. The neurological recovery after surgery varies with the severity of the preoperative impairment and the interval between presentation and operation. Post-traumatic concomitant spinal hematomas are rare as evidenced in the literature, outcome depends on time to operation and prognosis is impacted by age and preoperative deficit. Because of the high risk of poor outcome without treatment, EDH should always be a diagnostic consideration in patients whose presentation is even slightly suggestive. Rapid, appropriate treatment of these patients can often lead to complete recovery of function, whereas any delay in appropriate treatment can be catastrophic. Surgical evacuation of the hematomas is safer and would relieve symptoms.

REFERENCES


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