Spinal Epidural Hematoma Induced By Leukemia

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Spinal epidural hematoma, a potentially devastating problem, requires rapid diagnosis and urgent surgical intervention. Two types of spinal epidural hematoma have been described in the literature: traumatic and nontraumatic.1 Traumatic spinal epidural hematoma has been attributed to spine surgery,2 epidural catheter placement,3 lumbar puncture,4 and chiropractic manipulation.5 Nontraumatic, or spontaneous, epidural hematoma has been associated with anticoagulation,6 thrombolytic agents,7 antiplatelet medications,8 hemophilia,9 among other causes.10-13 There is a single reported case of spontaneous epidural hematoma is attributed to underlying malignancy.14

This article reports a case of nontraumatic spinal epidural hematoma secondary to the blast crisis phase of chronic myelogenous leukemia.

CASE REPORT

A 26-year-old man presented approximately 6 hours after the onset of back pain with lower extremity weakness and urinary difficulty. Three years prior to presentation, he was diagnosed with chronic myelogenous leukemia. After undergoing initial treatment with interferon and hydrea, he was lost to follow-up.

Examination revealed complete paraplegia at the tenth thoracic level and palpable splenomegaly. White blood cell count was 475,000 with peripheral blood smear showing 40% myeloblasts with granulocytosis. The platelet count was elevated to 662,000. Urgent spinal magnetic resonance imaging (MRI) demonstrated a large posterior epidural mass extending from T9-T11 with marked spinal cord compression (Figure).

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Spinal Epidural Hematoma Induced

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Intravenous methylprednisolone was started and leukopheresis was performed. Emergent surgical spinal canal decompression was achieved by resection of the T8 through T12 laminae. A large posterior epidural hematoma extending from T8-T11 was evacuated. Prior to wound closure, two closed suction drains were placed deep into the fascia.

Postoperatively, no immediate return of neurologic function occurred. Daily leukopheresis was continued, and the chemotherapeutic agents, gleevac and hydrea, were administered. On postoperative day 3, suction drains were removed. Within 1 hour of drain removal, profuse bleeding from the incision and drain insertion sites occurred. Urgent operative exploration showed hematoma reaccumulation due to diffuse hemorrhage from the epidural venous plexus along the decompressed spinal canal. Aggressive use of bipolar electrocautery and gelfoam allowed for reasonable hemostasis. The incision was again closed over suction drains.

Following the second surgical procedure, repetitive leukopheresis was performed and multiple platelet transfusions were administered. Although no further bleeding occurred, neurologic status did not improve. The patient remained in the hospital for 2 weeks following the second surgical procedure and continued to receive chemotherapy and leukopheresis. At discharge, white blood cell count had decreased to 34,000. Three weeks after discharge, the patient returned to the hospital with severe abdominal pain. Computed tomography (CT) demonstrated a psoas hematoma. Serial CT revealed a stable hematoma, and the patient was discharged. At 3-month follow-up, the patient’s neurologic...
status remained stable with complete paraplegia at the tenth thoracic level.

**DISCUSSION**

Spinal epidural hematoma is a rare clinical entity. The annual incidence is estimated at 0.1 per 100,000 patients per year. Approximately 300 cases of spinal epidural hematoma have been reported. In adults, spinal epidural hematoma is localized predominantly to the lumbar region and dorsal to the dural sac. Up to 50% of all cases arise spontaneously in the absence of trauma.

Although multiple nontraumatic etiologies for spinal epidural hematoma have been identified, only one case of a spinal epidural hematoma attributed to malignant disease has been reported. Hayem et al described an epidural hematoma in a patient with myeloma involving the vertebral body at the same spinal level. This patient, however, was treated with low-molecular weight heparin prior to the onset of epidural bleeding.

Chronic myelogenous leukemia is a hematopoiesis disorder due to a genetic defect in the pluripotential stem cell. In the United States, >6000 new cases are diagnosed each year, accounting for 6%-15% of adult leukemias. Thirty percent of patients are diagnosed while asymptomatic as a result of splenomegaly findings on physical examination or a blood count with leukocytosis. Patients may initially present with fatigue, fever, weight loss, excessive sweating, or bone pain.

Although >90% of patients with chronic myelogenous leukemia present in benign or chronic phases, many patients eventually progress to blast crisis, which is diagnosed when ≥30% myeloblast cells are present in the bone marrow, peripheral blood, or both. An overwhelming number of granulocytes produced during a blast crisis can lead to leukostatic sludging in the circulatory system and end organ damage from vascular insufficiency. Thrombocytosis with associated platelet dysfunction is also common in many patients with chronic myelogenous leukemia.

Successful management of a spinal epidural hematoma requires rapid diagnosis. In patients with suspected spinal epidural hematoma, the diagnostic imaging modality of choice is MRI. T1-weighted images demonstrate an epidural collection of blood with an isointense or slightly hyperintense signal to that of the spinal cord. On T2-weighted images, the hematoma displays a signal of mixed intensity. Mass effect on the spinal cord is generally visible. Gadolinium contrast is helpful in further characterizing such epidural collections.

Acute spinal epidural hematoma treatment depends on clinical presentation. Patients with rapid onset and acute loss of neurologic function are best treated with emergent surgical decompression. In select patients with minimal neurologic compromise or an improving neurologic examination, addressing the underlying cause of the hematoma may provide effective non-operative treatment.

The prognosis of patients with spinal epidural hematoma depends on: 1) the degree of neurologic compromise at presentation, and 2) the time from symptom onset to surgical decompression. Lawton et al demonstrated that surgery performed >12 hours after symptom onset is unlikely to improve the neurologic status of patients. However, spontaneous recovery has also been reported.

In our patient, the primary cause of neurologic injury was most likely...
mechanical compression of the spinal cord by the large epidural hematoma. It is also possible that leukostatic sludging from the marked granulocytosis further compromised the blood supply to the cord parenchyma. Therefore, our treatment approach incorporated emergent surgical decompression and treatment of the underlying disorder.

REFERENCES