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CASE REPORT

Lumbar puncture complicated by spinal epidural hematoma in a child with leukemia

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Key Clinical Message

We report a case of spinal epidural hematoma (SEH) preceded by diagnostic lumbar puncture (LP) in a 5-year-old boy with acute lymphoblastic leukemia. MRI confirmed the presence of SEH between T7 and L5 levels, but the patient showed fast recovery during the next hours and conservative management was elected.

Keywords

Acute lymphoblastic leukemia, lumbar puncture, spinal epidural hematoma, thrombocytopenia.

Introduction

Spinal epidural hematoma (SEH) is a rare condition, mostly caused by trauma, anticoagulant therapy, arteriovenous malformations, hypertension, blood dyscrasia, pregnancy and childbirth, spinal surgery, and invasive spinal procedures [1–4]. The clinical features of SEH are that of acute spinal cord compression, along with prolonged and/or severe headache [5–8], while incidence of paraplegia after LP has been recorded in several studies to vary between 0.0005 and 0.02 % [3, 9].

The reasonable approach on treating thrombocytopenic children with blood malignancies undergoing LP is prophylactic platelet transfusion prior to any surgical procedures. A trigger of not lower than 10×10^9 /L platelet count has been suggested by previous retrospective studies, in order to avoid serious bleeding events [9–12]. Currently, magnetic resonance imaging (MRI) is the diagnostic method of choice, and is also used in monitoring the course of SEHs [13, 14]. Studies on SEHs preceded by LP in children with leukemia are lacking, so we hereby report a notable case of a young thrombocytopenic patient with acute lymphoblastic leukemia (ALL).

Case Description

A 5-year-old boy was referred to our hospital with a history of poor general health, following a febrile upper respiratory tract infection, with an unusual and prolonged course. Physical examination revealed lymphadenopathy, hepatosplenomegaly, and multiple ecchymoses. Neurological examination was normal, while no headache was reported. Blood laboratory studies showed thrombocytopenia and anemia. Flow cytometry of bone marrow aspirate resulted in a diagnosis of common ALL. At diagnosis, LP was performed, which indicated central nervous system (CNS) involvement, and he started therapy with ALL IC - BFM 2009 treatment protocol. LPs on days 15 and 33 of the first phase and on day 10 of the second phase of induction chemotherapy protocol showed remission of CNS involvement.

During the second phase of chemotherapy, the onset of vomiting, triggered by motion and accompanied by headache, set the clinical suspicion of CNS pathology (i.e.,

relapse or infection), while papilledema was absent in fundoscopy. Complete blood count showed thrombocytopenia (16×10^9/L) and neutropenia. LP was conducted *lege artis*, and cerebrospinal fluid cytochemical analysis revealed neither pathological findings, nor hemorrhagic elements. The patient reported postspinal headache and lower back pain, symptoms that were relieved, and initially responded to analgesics and the infusion of fluids. After strict maintenance of a supine posture for two hours, the boy found difficulties in maintaining the upright posture, and urine loss occurred, along with a diarrhea. Lower back pain reappeared, but was well tolerated. Neurological symptoms continued to worsen for the next 4 hours, leading to paraparesis, decreased sensation of both legs, and urinary retention. MRI scanning revealed the presence of extensive epidural hemorrhagic collection in the thoracolumbar region of the spinal cord between T7 and L5 levels, which occupied the entire spinal canal, and compressed the spinal cord and its sac (Fig. 1A). Intramedullary edema between T8 and T12 levels was noticed as well. The brain MRI showed no pathological findings. Platelet transfusion and fresh frozen plasma (FFP) were administered immediately, with parallel infusion of intravenous dexamethasone.

The patient showed fast recovery during the next hours and medical conservative management was elected. During the next days, the boy showed gradual improvement of his neurologic syndrome: sensation of lower extremities was restored completely and relatively soon, whereas motility of legs improved gradually. Despite the initial improvement, urinary retention followed a slower course of recovery (set for 1.5 month in Foley catheter, 4 intermittent self-catheterizations per day for the next month, which were decreased to 1 or 2 nowadays). The MRI on day 4 showed small reduction in hematoma width and localized partial absorption of the hemorrhagic display with respective hemosiderin deposits (Fig. 1B). Two months later, MRI scanning showed significant decrease in hematoma limits (width of 2.5 mm; between T12 and L5 levels) and absence of any focal lesions or signs of compression in the spinal cord (Fig. 1C). Last MRI took place 6 months after the event, showing complete resorp-

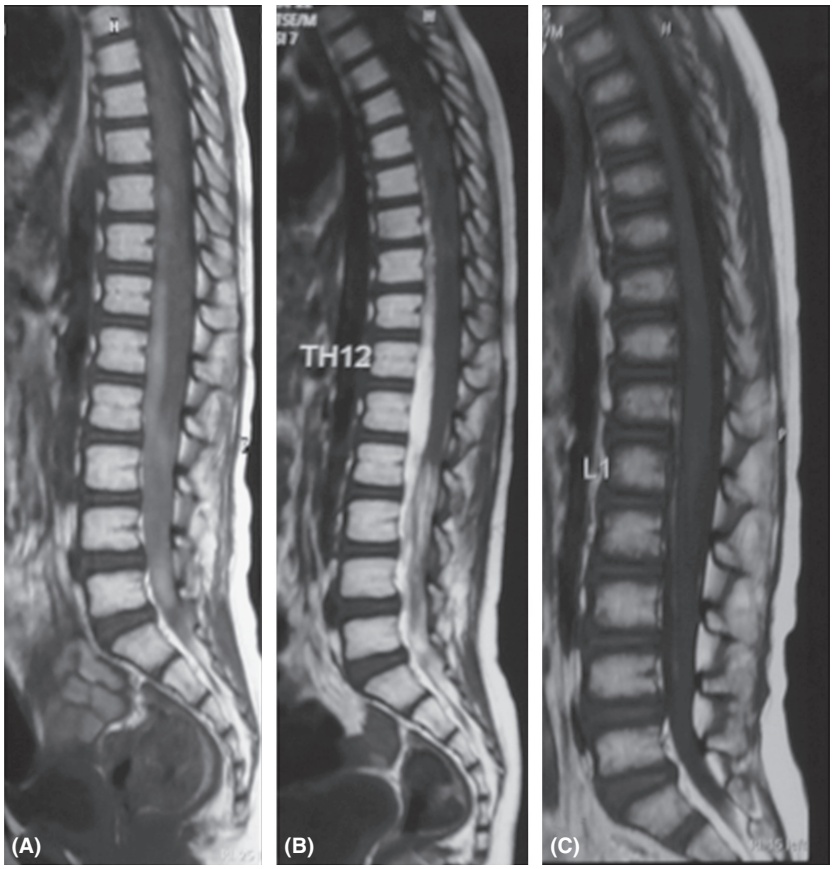

Figure 1. Comparative MR T1-weighted TSE SENSE images of the spine on days: 1 (A), 4 (B) and 60 (C) after the incident.

tion. The patient continued his therapy for leukemia and is now in maintenance therapy, reporting only a minor neurologic deficit, with regard to control of urination.

Discussion

The pathophysiology of SEH still remains unclear, but is best described as the result of internal rupture of the Batson vertebral venous plexus, and it can rapidly develop severe neurologic deficit. Regardless of the setting, symptomatic SEHs need urgent surgical decompression [13]. Constant clinical evaluation in conjunction with early MR findings (such as hypersensitivity on T2-weighted images of the involved spinal cord and/or contrast enhancement), can guide the attending physicians to a conservative or a more radical way of treatment. In our case, fast recovery from SEH clinical features combined with respective MR findings led us to the use of a conservative way of treatment. Regarding the need for LP in this patient, it was considered essential, because the boy was diagnosed with high risk ALL, involving the CNS, and there was history of severe infections (e.g. *E. coli* sepsis). Apparently, PLT and FFP transfusion together with intravenous dexamethasone contributed to a better outcome in our patient.

LP has rare (0 to 1.87%) but serious complications, not taking into account local back pain and headache following such a procedure. These include both nonhemorrhagic (vertebral disc infection, spinal nerve root herniation, meningitis, and cerebellar tonsil herniation) and hemorrhagic complications (spinal subdural, subarachnoid, and epidural hematomas) [10, 11]. The unmodifiable risk factors for traumatic and bloody LP include black race, age younger than 1 year, a traumatic or bloody previous LP, performed within the past 2 weeks, and a previous LP performed, when the platelet count was 50×10^9/L or less. Modifiable risk factors include procedural factors reflected in treatment area, a platelet count of 100×10^9/L or less, an interval of 15 days or less between LPs, and a less experienced practitioner [15]. Other potential risk factors include anticoagulant therapy, CNS pathology, and disseminated intravascular coagulation [10]. The British Committee for Standards in Haematology, Blood Transfusion Task Force, produced guidelines, suggesting the limit of a platelet count of $\geq 50 \times 10^9$/L, to safely proceed with LP [16]. Several retrospective studies in thrombocytopenic pediatric patients with ALL suggest that it may be safe to perform LPs at platelet counts $>10 \times 10^9$/L [10, 17, 18], especially when other risk factors are determined or suspected. Other studies in this field [9, 11, 19, 20] set higher thresholds (i.e. $20–40 \times 10^9$/L) for prophylactic platelet transfusion, although there is a published case of a 12-year-old male with ALL and thrombocytopenia with a count of 42×10^9/L, who was given platelet transfusion immediately before the LP, but developed an extended spinal subdural hematoma [12].

In conclusion, we would like to stress the importance of achieving a safe level of platelet count, through prophylactic platelet transfusion. The gold standard in treating SEH is surgical decompression. Alternatively, conservative management should be chosen very carefully, and monitored intensively, utilizing MR imaging [21].

Conflict of Interest

The authors declare that there are no conflicts of interest.

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