



SURGICAL MANAGEMENT OF TRAUMATIC VERTEBRAL BODY COMPRESSION FRACTURE WITH CORD COMPRESSION IN A THALASSEMIC PATIENT. CASE REPORT

Neurosurgery

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ABSTRACT

Extramedullary hematopoiesis associated with thalassemia causing spinal cord compression is a rare event in the course of the disease. Management of these patients remains controversial. We present a case of traumatic vertebral body compression fracture with cord compression in association with thalassemia, which was treated successfully with spinal decompression and fusion, fixation using implants and bone graft.

KEYWORDS

Extramedullary Hematopoiesis, Thalassemia, Mri Spinal Cord

CASE REPORT

A 25 years female, known case of thalassemia presented in emergency with history of fall in bathroom followed by severe back pain and unable to move both lower limbs. Her physical examination revealed anemia, characteristic thalassemic facial features. Neurological examination showed paraplegia, tone both lower limbs increased, both plantar extensors, sensory loss below L1 dermatome. She had urinary retention with perianal sensory loss. X-ray lumbosacral spine (Fig. 1) revealed compression fracture D12 vertebral body. MRI Lumbosacral spine (Fig. 2,3) showed compression fracture of D12 with cord compression. All vertebral bodies showed low to intermediate signal intensity signifying replacement of fatty marrow with hematopoietic tissue. Hematologist consulted and blood, along with iron chelating agent started accordingly. Initially, Inj. Methyl prednisolone infusion given, but didn't reveal any improvement. Surgical option (spinal instrumentation & decompression) discussed with patient & her family members, and they opted for surgery. On operation, bones were relatively soft, D10, D11, L1, L2 titanium trans-pedicular screws placed under C-arm fluoroscopic guidance with relatively good purchase, D12 laminectomy performed, with removal of reddish brown (epidural) hematopoietic tissue from left posterolateral aspect of dura (Fig. 4, 5), decompression of cord done. Bilateral rods applied & postero-lateral G-bone graft (calcium hydroxyapatite granules) fusion done. Post operatively she became pain free and at time of discharge, neurological improvement in power of right lower limb 2/5, left lower limb 3/5. Foley's catheter removal trial was given but failed. At 6 months follow up she can stand and walk with Taylor's brace for about 50 meters, and passes urine normally.

IMAGES



FIGURE.1

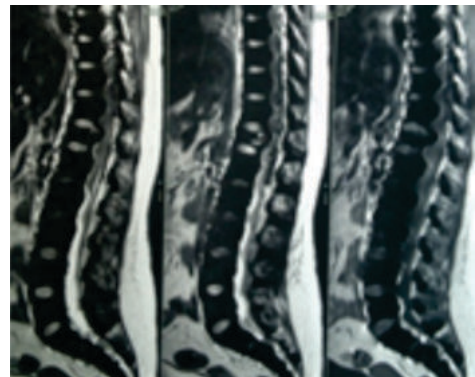


FIGURE.2

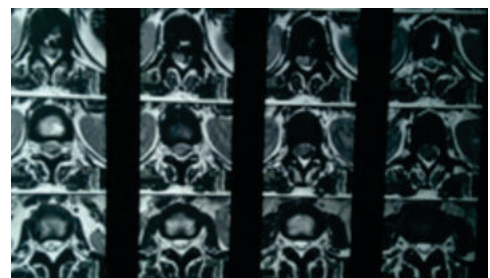


FIGURE.3

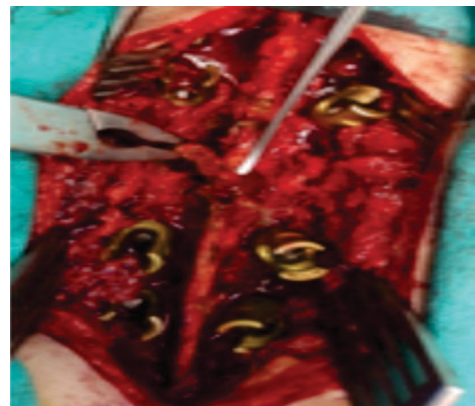


FIGURE.4



FIGURE.5

DISCUSSION

Thalassemia characterized by reduced or absent synthesis of one or more globin chains of hemoglobin. Beta thalassemia associated with marked osseous changes, attributed to bone marrow expansion and cortical thinning caused by massive erythropoiesis.

Extramedullary hematopoiesis are commonly seen in liver, spleen, kidney, and lymph nodes, so as to combat long standing anemia. The development of bone marrow from primitive cell rests or direct extension from the adjacent vertebral bone marrow is the most likely etiology of spinal extramedullary hematopoiesis¹⁻⁴. Extramedullary hematopoietic tissue causing cord compression was first described by Gatto et al⁵ in 1954. MRI is the best method for demonstrating spinal cord compression. On T1-weighted images, extramedullary masses of signal intensity slightly higher than that of the adjacent bone marrow of the vertebral bodies⁶. Adults with beta thalassemia major frequently have low BMD, increased bone turnover, and an increased risk for fractures & bone pain.¹³

There is considerable controversy regarding the proper management of spinal cord compression in thalassemic patients. Treatment options include surgical decompression, blood transfusions, radiotherapy (RT), or a combination of these modalities⁶⁻⁸. Low-dose RT alone or partial excision and repeated blood transfusion have been reported with good response^{4,6,7,9}.

Removal of the epidural mass causes decompression of the spinal cord and improves chances of neurological recovery. Limitations of surgery is in total excision due to the diffuse nature of the process^{6,10,11}, implant failure, CSF leak, neurological deterioration, wound dehiscence (poor wound healing in anemia). Radiation inhibits hematopoietic activity and causes shrinkage of the mass and reduction of the cord compression¹².

In our opinion, surgery is the treatment of choice in the event of acute and severe neurological deficits precipitated by spinal trauma.

CONCLUSION

Extramedullary hematopoiesis occurs in thalassemic patients, and sometimes it causes spinal canal compromise. Various treatment options for compromised spinal canal in thalassemic patients has been described in literature which includes surgical decompression, RT, blood transfusion, and any of these in combination.

Surgical spine canal decompression and fusion, fixation is necessary in patients who have spinal instability and progressive neurological deficit or acute paraplegia, so as to start rehabilitation.

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