CASE REPORT

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LANGERHANS CELL HISTIOCYTOSIS OF THE SPINE: T12 VERTEBRAL DESTRUCTION AND SPINAL CORD COMPRESSION IN A 49-YEAR-OLD ADULT: A CASE REPORT

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ABSTRACT

Langerhans cell histiocytosis (LCH) is a rare clonal disorder that predominantly affects children, while adult spinal involvement is uncommon. We report a 49-year-old woman presenting with thoracic back pain and progressive lower extremity weakness. Imaging revealed a destructive lytic lesion of the T12 vertebra with epidural extension causing spinal cord compression, initially suggestive of infection or metastasis. Histopathological examination confirmed LCH. The patient underwent posterior decompression, instrumentation, and T12 hemicorpectomy, resulting in rapid neurological improvement. This case highlights the diagnostic challenge of adult spinal LCH and the importance of timely surgical management in the presence of neurological compromise.

Keywords: Langerhans cell histiocytosis, eosinophilic granuloma, spinal cord compression

INTRODUCTION

Langerhans cell histiocytosis (LCH) is a rare disease characterized by the clonal proliferation of Langerhans cells[1]. It is widely variable in the clinical manifestation; it may manifest as a single bone lesion or as a multisystemic disorder that may be fatal[2]. Generally, LCH affects children under the age of 15, with an incidence of approximately 8.9 per million in this age group. In contrast, its occurrence in adults is much rarer, with an estimated incidence of 0.07 per million annually[3].

The symptoms of LCH are diverse and depend primarily on the organ system involved. LCH could be unifocal or multifocal. When the bone is affected, it is known as eosinophilic granuloma[4]. In the skeletal system, the most commonly involved parts are the skull, followed by the femur, mandible, pelvis, and spine[5]. Lung involvement is the most common presentation in LCH, seen in over half (51%) of such cases, bone (38%) and skin (7%)[6]. Spinal involvement in adult LCH is relatively rare, and epidural extension is particularly uncommon[7]. In adults, LCH presents as a multisystem disease in approximately 69% of cases, while 31% exhibit single-system involvement[6].

Treatment of LCH varies according to disease extent and location and may include conservative management, local or surgical interventions, and systemic therapy[8]. Because spinal LCH can mimic infection or metastatic disease, diagnosis is often challenging. Herein, we report a 49-year-old woman with T12 vertebral LCH initially suspected to be an inflammatory or metastatic lesion, underscoring the diagnostic complexity of this rare condition.

CASE PRESENTATION

Written informed consent was obtained from the patient. A 49-year-old woman presented with a five-month history of lower thoracic back pain and progressive left lower extremity weakness, accompanied by gait disturbance for one month. Her medical history included hypertension and hypothyroidism.

Physical examination revealed lower thoracic tenderness and grade 4 motor weakness in the left lower limb. Laboratory findings showed a white blood cell count of 4.55×10^9/L, C-reactive protein level of 19 mg/dL, and erythrocyte sedimentation rate of 60 mm/h, without fever or evidence of systemic infection.

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Radiological Findings

Magnetic resonance imaging showed a pathological fracture of the T12 vertebra with approximately 40% height loss, posterior element destruction, and an epidural mass occupying the left T11-T12 neural foramen, resulting in spinal cord displacement and canal stenosis. These findings initially suggested infectious spondylitis.

Contrast-enhanced computed tomography (CT) revealed a 33×23 mm lytic lesion in the left T12 vertebral body extending to the posterior elements and paravertebral region, with destruction of the posterior 12th rib . Fluorodeoxyglucose-positron emission tomography (FDG-PET)/CT demonstrated a solitary hypermetabolic lesion without additional involvement; therefore, no systemic treatment was administered (Figure 1).

Biopsy and Histopathological Findings

A preoperative biopsy of the T12 vertebra was obtained using a 16-G tru-cut needle. Histopathological examination revealed histiocyte-like cells with reniform nuclei and multinucleated giant cells within an eosinophil-rich inflammatory stroma. The lesional cells showed immunohistochemical positivity for S-100 and CD1a, consistent with LCH. Intraoperative biopsy confirmed the same findings (Figure 2).

Surgical Procedure

The patient was positioned prone under general anesthesia, and intraoperative neuromonitoring was used throughout the procedure. A midline posterior approach from T10 to L2 was performed after fluoroscopic level confirmation. Intraoperatively, extensive destruction of the left T12 vertebral body, pedicle, and lamina with exposure of the spinal canal was observed , and marginal excision of the soft-tissue mass was carried out.

Pedicle screws were placed at T10, T11, T12 (right side only), L1, and L2 using a freehand technique under fluoroscopic guidance. Left T12 hemicorpectomy and laminectomy were

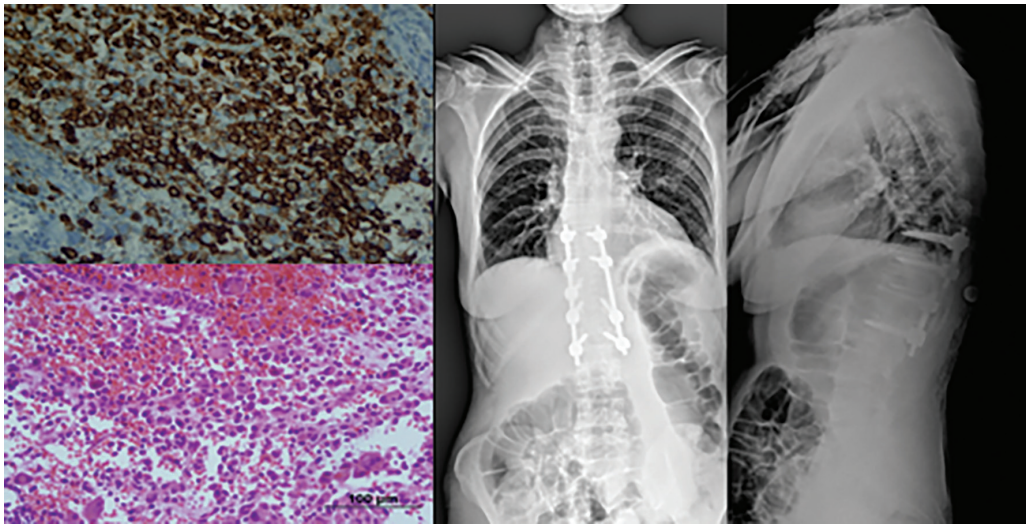


Figure 1. Axial magnetic resonance imaging demonstrating a lytic lesion involving the left side of the T12 vertebral body and posterior elements, with associated epidural and paravertebral soft-tissue extension causing spinal canal compromise. Axial contrast-enhanced computed tomography shows a lytic lesion in the left half of the T12 vertebral body extending to the posterior elements and paravertebral region, with destruction of the posterior portion of the 12th rib. Axial FDG-PET/CT demonstrates a hypermetabolic lytic lesion involving the left T12 vertebra and adjacent posterior rib arch. FDG-PET/CT: Fluorodeoxyglucose-positron emission tomography/computed tomography

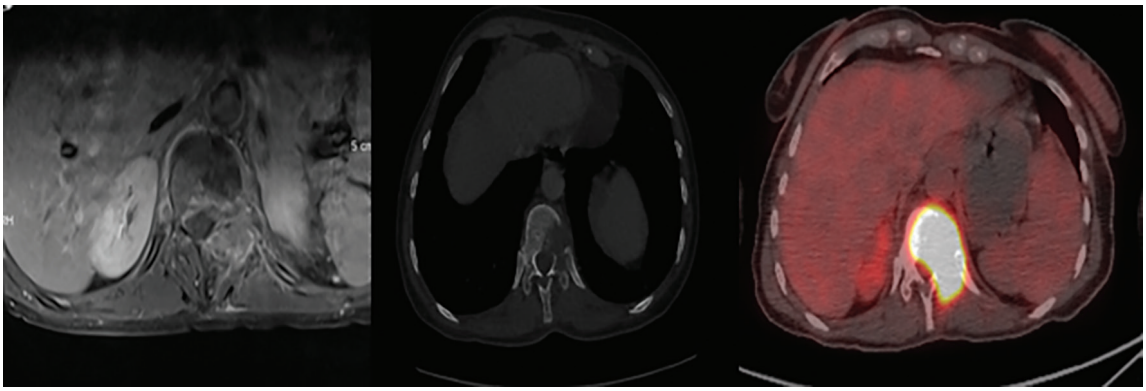


Figure 2. Left: histopathological section of the biopsy demonstrating histiocyte-like cells with eccentric reniform nuclei and multinucleated giant cells, consistent with Langerhans cell histiocytosis. Right: postoperative anteroposterior and lateral thoracolumbar spine radiographs showing posterior spinal instrumentation and fusion spanning from T10 to L2

performed to achieve spinal cord decompression, followed by posterior stabilization with bilateral rods and autologous bone grafting. Final neuromonitoring signals remained unchanged. Postoperative radiographs demonstrated satisfactory alignment and fixation (Figure 2).

The patient was followed for 8 months postoperatively, with no complications observed.

DISCUSSION

LCH is an uncommon disorder in adults and rarely presents with vertebral destruction or spinal cord compression. In their 2025 systematic review, Abdulla et al.[9] identified LCH in 74 patients. This diagnostic difficulty was evident in our patient, whose imaging findings-including a lytic vertebral lesion, paravertebral soft-tissue extension, and rib destruction-initially raised concern for an infectious or metastatic process.

Several radiologic clues may help differentiate LCH from infection or malignancy. Preservation of the intervertebral disc space, a well-defined lytic lesion, and the absence of additional FDG-avid lesions on PET/CT favor LCH over spondylodiscitis or metastatic disease[9]. However, in advanced cases with posterior element destruction or extensive soft-tissue components, as in our patient, the imaging characteristics may become less specific, making histopathological confirmation essential.

Management of adult spinal LCH remains variable due to its rarity. Otsuki et al.[10] achieved successful treatment in four patients using posterior instrumentation alone, without the need for curettage or bone grafting, and in one additional patient with the addition of chemotherapy. Sapkas et al.[11] were concerned about the extraosseous extension and performed excision and bone grafting. While conservative treatment or limited local interventions may be adequate for stable, unifocal lesions without neurological compromise, surgical intervention is recommended in the presence of instability or cord compression[11]. Our patient required posterior decompression and T12 hemicorpectomy due to extensive bone destruction and epidural compression. Surgical stabilization enabled immediate neural decompression and adequate tissue procurement for diagnosis.

Most adults with solitary osseous LCH have favorable outcomes following complete resection, curettage, or just posterior stabilisation[12]. Nonetheless, long-term follow-up is essential, as recurrence or progression to multisystem disease, although infrequent, has been reported[13]. This case underscores the importance of considering LCH in the differential diagnosis of destructive thoracic vertebral lesions in adults. It highlights the role of timely biopsy and surgical management to prevent irreversible neurological deterioration.

CONCLUSION

Adult spinal LCH is rare and may mimic infectious or metastatic disease, causing diagnostic delay. This case emphasizes the importance of considering LCH in destructive vertebral lesions and confirms the role of histopathology. Early surgical intervention is recommended in cases with spinal instability or neurological compromise.

Ethics

Informed Consent: Written informed consent was obtained from the patient.

Footnotes

Authorship Contributions

Surgical and Medical Practices: B.A., N.A., F.E., H.S.C., Concept: B.A., A.A., Design: B.A., A.A., Y.S.B., Data Collection or Processing: B.A., N.A., F.E., H.S.C., Analysis or Interpretation: B.A., N.A., A.A., Y.S.B., Literature Search: B.A., A.A., N.D., Writing: B.A., A.A., N.D.

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