ISSN 2706-6819 Vol. 7, Issue 1: 56-63, DOI: 10.25236/IJFM.2025.070109

Adult Lumbar Langerhans Cell Histiocytosis: A Case Report

Yiwen Xu[1,a,#], Jie Huang[1,b,#], Hao Huang[1,c,#], Ze Gao[1], Xinhui Chi[1], Zijian Cheng[2], Mingxie Chen[3], Jing Wang[1,*]

[1]The First Affiliated Hospital of Jinan University, Guangzhou, 510623, China
[2]School of Basic Medical Sciences, Jinan University, Guangzhou, 510623, China
[3]Dongguan Tungwah Hospital, Dongguan, 523120, China
[a]x2613824893017@163.com, [b]jiehuang0313@163.com, [c]ericsonh@163.com
[#]Yiwen Xu, Jie Huang and Hao Huang have contributed equally to this work
[*]Corresponding author: wangjing11277@126.com

Abstract: *Langerhans cell histiocytosis (LCH) is an extremely rare inflammatory myeloid tumor disease. This article reports a 25-year-old male patient who sought medical attention for one month due to persistent lower back pain. Imaging examination suggests the possibility of tumor like lesions in the L3 vertebral body, which was ultimately diagnosed as LCH through surgical pathological biopsy. This case can provide important reference for clinical doctors and other frontline medical personnel, enrich the existing knowledge base of adult lumbar LCH, improve understanding of the disease, and reduce misdiagnosis rate.*

Keywords: *Langerhans cell histiocytosis, adult spine, osteolytic lesions, surgical treatment*

1. Introduction

Langerhans cell histiocytosis (LCH) is an exceedingly rare inflammatory myeloid tumor characterized by the clonal proliferation and infiltration of Langerhans cells within the mononuclear phagocyte system [1-3]. The clinical manifestations of LCH are diverse, with common symptoms including bone pain, rashes, respiratory difficulties, and abnormal growth and development. LCH is more prevalent in children and adolescents, with an incidence rate of 3.0-5.0 per million, while in adults, the incidence rate is 1.0-2.0 per million, and it is more common in men than women [4-6]. Based on the organs and systems affected, LCH can be classified into single-system LCH (SS LCH) and multi-system LCH (MS LCH). According to the 2017 WHO classification, LCH can be further categorized into non-specific, isolated, and multifocal bone lesions with uncertain benign or malignant outcomes, while disseminated lesions are classified as malignant [7]. Commonly affected organs include the bones (50%-70%), lungs (30%-60%), endocrine system (20%-50%), and skin (20%-40%). Among bone lesions, the skull is most frequently involved, while spinal involvement is relatively rare [8-9]. Reports of adult lumbar LCH are limited [10-13]. This article presents a case of a 25-year-old male patient diagnosed with LCH, exhibiting osteolytic lesions in the L3 vertebra, and reviews relevant literature to enhance clinical understanding of the disease, promote early diagnosis and treatment, and ultimately save lives.

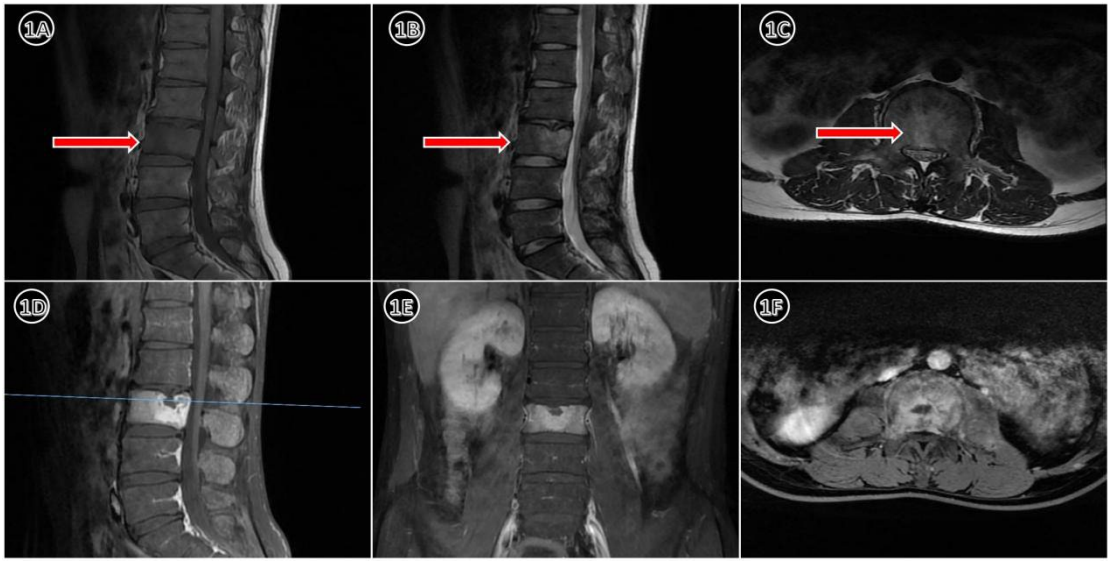
2. Case report

The patient is a 25-year-old male who presented to the Spinal Surgery Department on November 19, 2024, with complaints of "lower back pain for over a month, progressively worsening over the past week." Approximately one month prior, the patient experienced an unexplained fever, with a peak temperature of 38°C. After half a day, the fever subsided, but he developed persistent back pain, which was most severe at night. The pain was constant and intense, prompting the patient to self-medicate with painkillers, which provided some relief. However, the soreness and swelling worsened after physical activity or when turning over. There was no radiating pain, numbness, or weakness in the lower limbs, nor was there any urinary or fecal incontinence.

Since the onset of symptoms, the patient has lost approximately 7.5 kg. His medical history is negative for chronic or infectious diseases. The patient had intracranial tumor resection 25 years ago

ISSN 2706-6819 Vol. 7, Issue 1: 56-63, DOI: 10.25236/IJFM.2025.070109

(specific surgical details unavailable) but denies any history of other surgeries or trauma. Family and personal histories are unremarkable. On physical examination, the patient displayed normal physiological curvature of the spine without deformities. There were no signs of abnormal pigmentation, eczema, or ulcers on the back. Muscle tension was noted in the lower back, but no masses were palpated. Tenderness was present over the L3 spinous process (+), but there was no radiating pain in the lower limbs. Muscle strength in both lower limbs was normal, with intact physiological reflexes and no pathological reflexes.

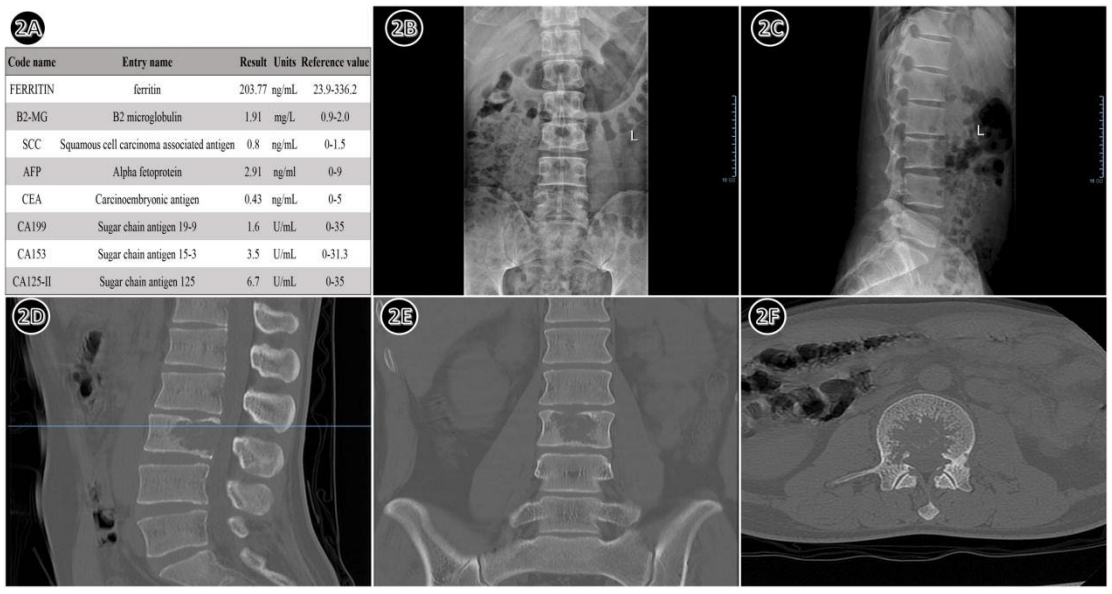


The sagittal plane shows strong signal changes in the L3 vertebral body on T1 weighted images (1A), slightly strong signal changes on T2 weighted images (1B), and slightly strong signal changes on coronal T2 weighted images (1C), with a size of approximately 2.5cmx2.6cmx2.4cm. Enhanced scanning shows obvious uneven enhancement of the lesion, indicating infectious lesions. It is recommended to follow up with short-term treatment to exclude tumor lesions (1D-1F)

Fig.1 Lumbar Magnetic Resonance Imaging (MRI)

2A

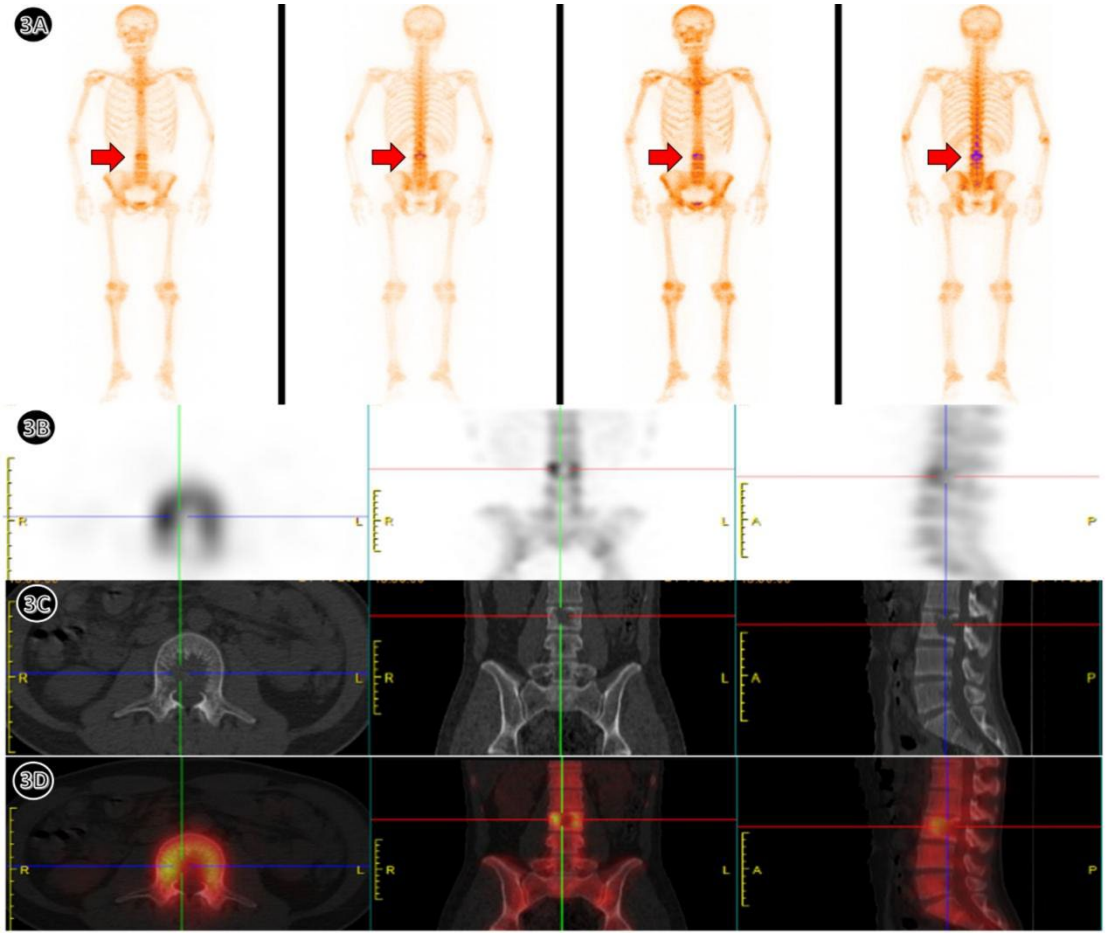
Code name	Entry name	Result	Units	Reference value
FERRITIN	ferritin	203.77	ng/mL	23.9-336.2
B2-MG	B2 microglobulin	1.91	mg/L	0.9-2.0
SCC	Squamous cell carcinoma associated antigen	0.8	ng/mL	0-1.5
AFP	Alpha fetoprotein	2.91	ng/ml	0-9
CEA	Carcinoembryonic antigen	0.43	ng/mL	0-5
CA199	Sugar chain antigen 19-9	1.6	U/mL	0-35
CA153	Sugar chain antigen 15-3	3.5	U/mL	0-31.3
CA125-II	Sugar chain antigen 125	6.7	U/mL	0-35



Tumor markers were all within normal range (2A). Lumbar DR showed a slight decrease in density in the posterior part of the L3 vertebral body (2B-2C). Lumbar CT showed osteolytic bone destruction in the L3 vertebral body, involving bilateral pedicle bones, with residual bone ridges visible inside. The upper edge of the vertebral body was locally depressed with hardened edges. 2/3 of the intervertebral discs herniated downwards, and the canal was slightly narrowed, suggesting a tumor like lesion (2D-2F)

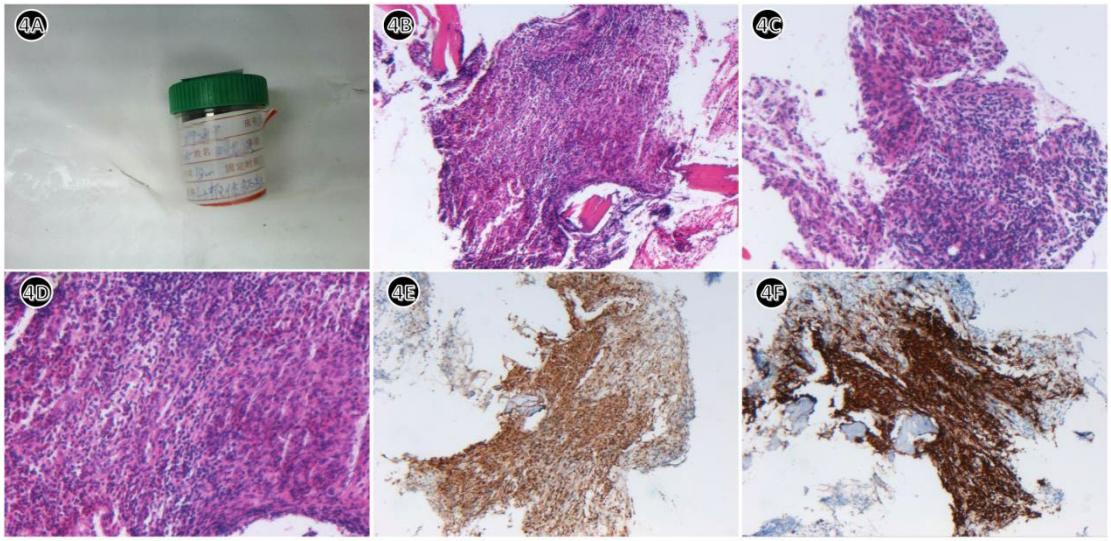
Fig.2 Tumor markers and lumbar digital radiography (DR), lumbar computed tomography (CT)

ISSN 2706-6819 Vol. 7, Issue 1: 56-63, DOI: 10.25236/IJFM.2025.070109



L3 vertebral body osteolytic bone destruction with abnormal radioactive concentration at the edge, first consider tumor lesions (3A-3D)

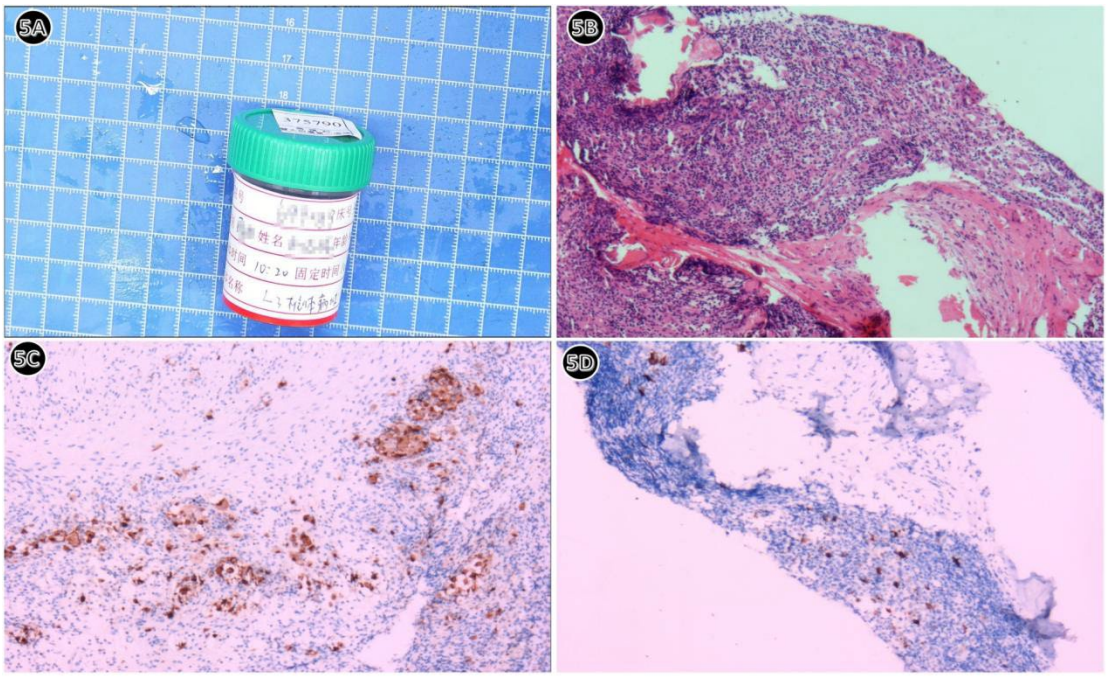
Fig. 3 SPECT/CT tomography imaging



A piece of gray yellow gray brown fragmented tissue with a diameter of 1cm (4A); 4B-4D: Monocyte like cells and abundant eosinophil proliferation are observed under the microscope, accompanied by more lymphocytes, plasma cells, and fibroblasts (4B: HE, x40; 4C, 4D: HE, x100; 4D: HE, x40); 4E, 4F: Immunohistochemical staining showed S-100 (+), CD1a (+), Langerin (+), CD56 (-), CD68 (-), CD34 (-), ERG (-), TTF-1 (-), Syn (-), P53 (approximately 80%+), Ki-67 (approximately 10%+), consistent with Langerhans cell histiocytosis (x40)

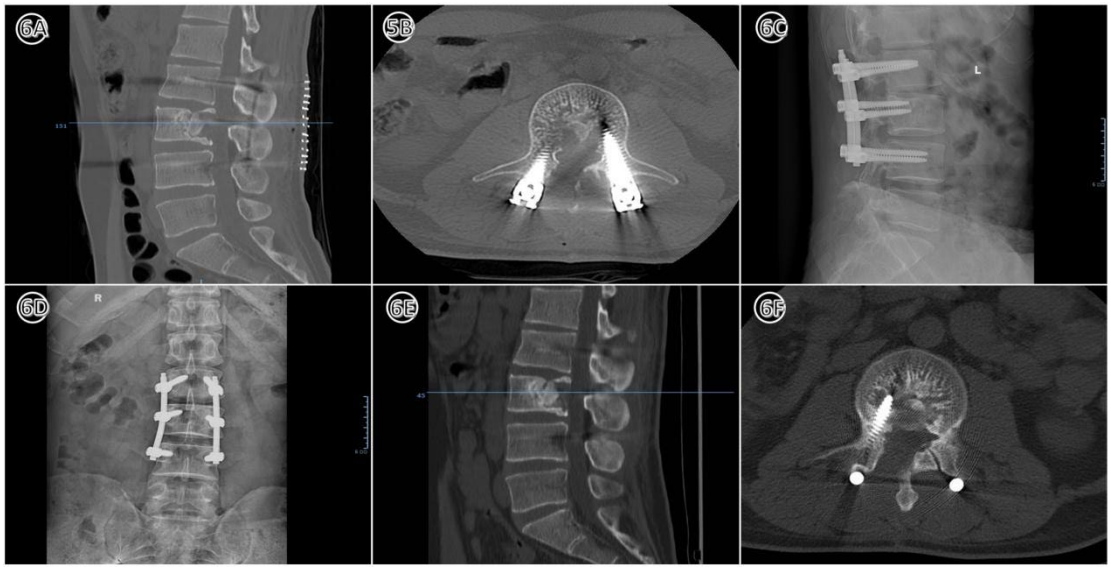
Fig. 4 Pathology of biopsy tissue from the lesion of the L3 vertebral body.

ISSN 2706-6819 Vol. 7, Issue 1: 56-63, DOI: 10.25236/IJFM.2025.070109



Gray white tissue block, diameter 1cm (5A); 5B: Monocyte like cells and abundant eosinophil proliferation were observed under the microscope, accompanied by more lymphocytes, plasma cells, and fibroblasts (5B: HE, x40); 5C, 5D: Immunohistochemical staining showed BRAF (+), S-100 (+), CD1a (+), Langerin (+), consistent with Langerhans cell histiocytosis (x40)

Fig. 5 Intraoperative biopsy pathology of lesion tissue in lumbar vertebrae 3



Postoperative CT scan show: Postoperative changes in L3 vertebral body osteolytic lesions can be seen in the sagittal (6A) and axial (6B) directions; Postoperative DR at 1 month (6C, 6D); One month after surgery, CT scan showed internal fixation shadow in the L2-L4 vertebral body, without any loosening or displacement. The degree of soft tissue swelling around the surgical area improved compared to before (6E, 6F)

Fig. 6 Postoperative imaging review

Lumbar magnetic resonance imaging (MRI) revealed irregular clustered signals in the L3 vertebral body (Figure 1A-1C). Enhanced scanning suggested possible infectious lesions in the L3 vertebral body and pedicle, prompting a recommendation for follow-up after short-term treatment to rule out tumor involvement (Figure 1D-1F).

Diagnostic Examination: The high sensitivity C-reactive protein (HsCRP) level was 16.84 mg/L, lactate dehydrogenase (LDH) 108 U/L, and alkaline phosphatase (ALP) 204 U/L. Tumor markers,

ISSN 2706-6819 Vol. 7, Issue 1: 56-63, DOI: 10.25236/IJFM.2025.070109

including alpha-fetoprotein (AFP), carcinoembryonic antigen (CEA), and carbohydrate antigen 19-9 (CA199), did not show significant abnormalities (Figure 2A). Digital radiography (DR) of the lumbar spine revealed a localized decrease in density in the L3 vertebral body, warranting further examination. No signs of vertebral slippage were noted in dynamic imaging (Figures 2B and 2C). Lumbar computed tomography (CT) revealed abnormalities in the L3 vertebral body and pedicle. Combined with MRI findings, a tumor lesion was suspected, and a biopsy was recommended (Figures 2D-2F). Whole-body bone scintigraphy (ECT) suggested osteolytic bone destruction in the L3 vertebral body with abnormal radioactive uptake at the edges, raising the possibility of a tumor-like lesion (Figures 3A-3D).

Treatment Process: Upon admission, the patient underwent physical and auxiliary examinations, and pathological bone tissue was obtained for biopsy. The postoperative pathological examination showed focal proliferation of mononuclear-like cells and numerous eosinophils, along with increased lymphocytes, plasma cells, and fibroblasts. Immunohistochemical staining was positive for S-100 (+), CD1a (+), and langerin (+), and negative for CD56, CD68, CD34, ERG, TTF-1, Syn, and P53 (approximately 80% positive). Ki-67 was approximately 10% positive. These findings were consistent with Langerhans cell histiocytosis (Figure 4A-4F).

Based on the patient's medical history, physical examination, and auxiliary tests, the final diagnosis was:

1) Langerhans cell histiocytosis of the L3 vertebral body

2) Pathological fracture of the L3 vertebral body

After consulting with the treatment team and excluding relevant surgical contraindications, and obtaining informed consent from the patient and family, the patient underwent "L3 lesion scraping surgery, L2-L4 pedicle screw fixation, and iliac bone grafting" on December 2, 2024.

Treatment Results and Outcomes: Postoperatively, the patient's lower back pain improved, and the muscle strength in both lower limbs returned to level 5, with no abnormal muscle tone or sensory deficits. The postoperative pathology confirmed focal proliferation of monocytes and numerous eosinophils, along with a higher number of lymphocytes, plasma cells, and fibroblasts. Immunohistochemistry results were: S-100 (+), CD1a (+), langerin (+), BRAF (+), confirming Langerhans cell histiocytosis (Figure 5A-D). Lumbar CT imaging showed clear bone grafting in the L3 vertebral body and stable internal fixation (Figures 6A and 6B).

Full communication with the patient and their family was conducted, recommending referral to the oncology department for further specialized treatment, and relevant precautions were provided before discharge. One month after surgery, the patient returned for a follow-up. Compared to before surgery, the patient's lower back pain had significantly improved, and no other discomforts were noted. DR and CT of the lumbar spine showed satisfactory surgical outcomes, with visible changes in the L3 vertebral body post-bone grafting, and stable fixation from L2 to L4 without loosening or displacement (Figures 6C-F). A follow-up visit was recommended in two months to continue monitoring the patient's progress.

3. Discussion

Langerhans cell histiocytosis (LCH) is an extremely rare clonal proliferative disorder first reported by Lichtenstein in 1953. Historically, this disease has been referred to by various names, including "eosinophilic granuloma of bone," "Hand-Schüller-Christian disease," "Letterer-Siwe disease," and "systemic histiocytosis X" [14-15]. LCH primarily affects children and is relatively uncommon in adults, with a male-to-female incidence ratio of approximately 1.2:1 [16]. Although the exact cause of LCH remains unclear, current research suggests it is associated with immune dysfunction or specific gene mutations. Previous studies have highlighted the activation of the MAPK pathway as the molecular basis for LCH's tumor characteristics, with the BRAFV600E mutation being the most prevalent (64%), followed by the BRAFindel mutation (29%) [17-18]. Histologically, LCH is characterized by the infiltration of dendritic cells.

LCH commonly affects bones (50%-70%) and skin (20%-40%), but may also involve multiple organs and systems, such as the lungs, spleen, liver, gastrointestinal tract, hematopoietic system, and central nervous system [19]. The classification of LCH is based on the location and extent of lesions, typically divided into single-system involvement (SS-LCH) and multi-system involvement (MS-LCH). In cases of multi-system involvement, further classification occurs based on whether high-risk organs

ISSN 2706-6819 Vol. 7, Issue 1: 56-63, DOI: 10.25236/IJFM.2025.070109

(such as the liver, spleen, or bone marrow) are affected, distinguishing between high-risk (Risk Organ+ MS-LCH, RO+ MS-LCH) and low-risk (Risk Organ- MS-LCH, RO- MS-LCH) forms. However, lumbar LCH in adults remains relatively rare. Reports suggest that LCH most commonly affects the thoracic spine (54%), followed by the lumbar spine (35%) and cervical spine (11%) [20-21]. Due to the extensive organ involvement and the variety of clinical manifestations associated with LCH, combined with the lack of specific symptoms, it is prone to misdiagnosis or delayed diagnosis.

To accurately diagnose spinal LCH, a comprehensive evaluation of multiple factors—such as the patient's medical history, clinical manifestations, imaging results, and pathological findings—is essential. The clinical presentation of spinal LCH is diverse and non-specific, with common symptoms including swelling and bone pain. Thus, imaging plays a crucial role in supporting the clinical diagnosis. Imaging characteristics of spinal LCH display distinct patterns: X-rays and CT scans often reveal osteolytic lesions, typically presenting as "map-like" or "worm-like" bone destruction. As the disease progresses, vertebral collapse, wedge-shaped deformities, or flattening may occur, and some lesions may show marginal sclerosis [22]. However, MRI lacks sufficient specificity for diagnosing spinal LCH. Affected vertebrae typically appear as low or isointense on T1-weighted images (T1WI) and hyperintense or slightly hyperintense on T2-weighted images (T2WI). Many cases also show significant enhancement on contrast-enhanced MRI scans [23], which is consistent with the imaging findings in this case (Figure 1D-1F).

Despite these characteristic features, other diseases may present with imaging findings similar to those of LCH. For instance, multiple myeloma (MM) may exhibit "chisel-like" bone destruction without bone sclerosis or periosteal reaction, with laboratory tests showing positive urinary light chains (BJP). Similarly, Ewing's sarcoma may present with "worm-like" bone destruction, which must be differentiated from LCH [24-25]. Thus, pathological examination is critical for diagnosis. Currently, lesion biopsy remains the gold standard for diagnosing spinal LCH. Immunohistochemical staining typically reveals positivity for S-100 protein, CD1a, and Langerin (CD207), with some cases demonstrating Birbeck granules [26-27], consistent with the immunohistochemical findings in this case (Figures 4, 5).

The treatment options and prognosis of Langerhans cell histiocytosis (LCH) can vary significantly, and there is currently no standardized treatment regimen [28]. While some patients may experience spontaneous remission without treatment, most cases are curable. However, certain cases, such as Risk Organ Multi-System LCH (RO+ MS-LCH), may progress rapidly and have a poor prognosis. Once spinal LCH is diagnosed, treatment plans should be tailored to the distribution, severity, and overall health status of the disease. The primary goal of treatment is typically to control the disease, alleviate symptoms, and prevent further bone damage. Standard treatment modalities include local surgical resection, radiotherapy, and drug therapy. For solitary localized lesions, surgical resection may be the treatment of choice. However, in cases with extensive or multiple lesions, a combination of radiotherapy and drug therapies, such as chemotherapy or hormone therapy, may be required [29]. In recent years, targeted therapy and immunotherapy have been investigated and applied to LCH, offering new treatment options for some patients [30-32]. During treatment, close monitoring for changes in the condition, recurrence, and periodic follow-up examinations are crucial.

In summary, this case involves a young male patient presenting with lower back pain, with spinal imaging revealing worm-like bone destruction and uneven enhancement in the affected vertebrae. Based on biopsy results, a final diagnosis of adult lumbar LCH (SS-LCH) was confirmed. Although spinal LCH is rare, and standardized treatment protocols are lacking, available options are limited. However, after thorough evaluations, it was determined that the lesion was confined to a single lumbar vertebra. The patient underwent surgical treatment, achieving favorable therapeutic outcomes. Post-surgery, the patient received further specialized treatment.

Reviewing the relevant literature and reflecting on the diagnosis and treatment of this patient, several key insights emerge regarding spinal LCH, a condition often misdiagnosed or missed clinically: lumbar LCH is exceptionally rare in adults and easily overlooked. Therefore, orthopedic physicians must remain vigilant when evaluating patients, especially when similar symptoms arise. Through detailed imaging studies, accurate pathological diagnosis, and comprehensive clinical assessment, misdiagnosis can be avoided, leading to timely treatment and improved patient prognosis and quality of life.

ISSN 2706-6819 Vol. 7, Issue 1: 56-63, DOI: 10.25236/IJFM.2025.070109

4. Conclusion

This case represents an exceptionally rare instance of adult spinal Langerhans cell histiocytosis (LCH). The diagnosis primarily depends on pathological biopsy, with particular attention to the expression of S-100 protein, CD1a, and CD207, in addition to imaging techniques such as CT and MRI, and tumor marker detection. Currently, there is no standardized treatment protocol for such cases. After thorough departmental discussions and an extensive review of the literature, we conclude that for cases of spinal LCH involving a single lesion confined to one system, surgical treatment should be considered the preferred approach. This includes local lesion resection and spinal stability reconstruction, followed by standardized systemic treatment post-surgery.

References

[1] Sato A, Kobayashi M, Yusa N, Ogawa M, Shimizu E, Kawamata T, Yokoyama K, Ota Y, Ichinohe T, Ohno H, Mori Y, Sakaida E, Kondo T, Imoto S, Nannya Y, Mitani K, Tojo A. Clinical and prognostic features of Langerhans cell histiocytosis in adults. Cancer Sci. 2023 Sep;114(9):3687-3697. doi: 10.1111/cas.15879. Epub 2023 Jun 26. PMID: 37364599; PMCID: PMC10475785.

[2] Yang PP, Hu SY, Chai XY, Shi XM, Liu LX, Li LE. Adult localized Langerhans cell histiocytosis: A case report. World J Clin Cases. 2023 Dec 6;11(34):8164-8169. doi: 10.12998/wjcc.v11.i34.8164. PMID: 38130781; PMCID: PMC10731173.

[3] Guyot-Goubin A, Donadieu J, Barkaoui M, Bellec S, Thomas C, Clavel J. Descriptive epidemiology of childhood Langerhans cell histiocytosis in France, 2000-2004. Pediatr Blood Cancer. 2008 Jul;51(1):71-5. doi: 10.1002/pbc.21498. PMID: 18260117.

[4] Rigaud C, Barkaoui MA, Thomas C, Bertrand Y, Lambilliotte A, Miron J, Aladjidi N, Plat G, Jeziorski E, Galambrun C, Mansuy L, Lutz P, Deville A, Armari-Alla C, Reguerre Y, Fraitag S, Coulomb A, Gandemer V, Leboulanger N, Moshous D, Hoang-Xuan K, Tazi A, Heritier S, Emile JF, Donadieu J. Langerhans cell histiocytosis: therapeutic strategy and outcome in a 30-year nationwide cohort of 1478 patients under 18 years of age. Br J Haematol. 2016 Sep;174(6):887-98. doi: 10.1111/bjh.14140. Epub 2016 Jun 7. PMID: 27273725.

[5] Baumgartner I, von Hochstetter A, Baumert B, Luetolf U, Follath F. Langerhans'-cell histiocytosis in adults. Med Pediatr Oncol. 1997 Jan;28(1):9-14. doi: 10.1002/(sici)1096-911x(199701)28:1<9::aid-mpo3>3.0.co;2-p. PMID: 8950330.

[6] Yağci B, Varan A, Cağlar M, Söylemezoğlu F, Sungur A, Orhan D, Yalçin B, Akyüz C, Kutluk T, Büyükpamukçu M. Langerhans cell histiocytosis: retrospective analysis of 217 cases in a single center. Pediatr Hematol Oncol. 2008 Jun;25(5):399-408. doi: 10.1080/08880010802107356. PMID: 18569842.

[7] Swerdlow SH, Campo E, Harris NL, et al. WHO Classification of Tu mours of Haematopoietic and Lymphoid Tissues[M]. 4th ed.Lyon, France: IARC Press, 2017.

[8] Aricò M, Girschikofsky M, Généreau T, Klersy C, McClain K, Grois N, Emile JF, Lukina E, De Juli E, Danesino C. Langerhans cell histiocytosis in adults. Report from the International Registry of the Histiocyte Society. Eur J Cancer. 2003 Nov;39(16):2341-8. doi: 10.1016/s0959-8049(03)00672-5. PMID: 14556926.

[9] Hassan BW, Moon BJ, Kim YJ, Kim SD, Choi KY, Lee JK. Langerhans cell histiocytosis in the adult lumbar spine: case report. Springerplus. 2016 Aug 23;5(1):1398. doi: 10.1186/s40064-016-3006-7. PMID: 27610317; PMCID: PMC4994818.

[10] Dhillon CS, Tantry R, Ega SR, Pophale C, Medagam NR, Chhasatia N. Langerhans Cell Histiocytosis in the Adult Lumbar Spine - A Case Report and Literature Review. J Orthop Case Rep. 2020 Dec;10(9):28-32. doi: 10.13107/jocr.2020.v10.i09.1892. PMID: 34169013; PMCID: PMC8046439.

[11] Hassan BW, Moon BJ, Kim YJ, Kim SD, Choi KY, Lee JK. Langerhans cell histiocytosis in the adult lumbar spine: case report. Springerplus. 2016 Aug 23;5(1):1398. doi: 10.1186/s40064-016-3006-7. PMID: 27610317; PMCID: PMC4994818.

[12] Schär RT, Hewer E, Ulrich CT. Langerhans Cell Histiocytosis of the Adult Cervical Spine: A Case Report and Literature Review. J Neurol Surg A Cent Eur Neurosurg. 2019 Jan;80(1):49-52. doi: 10.1055/s-0038-1655769. Epub 2018 Jun 11. PMID: 29890538.

[13] Lou Y, Zhao C, Cao H, Yan B, Chen D, Jia Q, Li L, Xiao J. Multiple Langerhans cell histiocytosis with spinal involvement. Bone Joint J. 2023 Jun 1;105-B(6):679-687. doi: 10.1302/0301-620X.105B6.BJJ-2022-1129.R1. PMID: 37257861.

[14] LICHTENSTEIN L. Histiocytosis X; integration of eosinophilic granuloma of bone, Letterer-Siwe disease, and Schüller-Christian disease as related manifestations of a single nosologic entity. AMA

ISSN 2706-6819 Vol. 7, Issue 1: 56-63, DOI: 10.25236/IJFM.2025.070109

Arch Pathol. 1953 Jul;56(1):84-102. PMID: 13057466.
[15] Hassan BW, Moon BJ, Kim YJ, Kim SD, Choi KY, Lee JK. Langerhans cell histiocytosis in the adult lumbar spine: case report. Springerplus. 2016 Aug 23;5(1):1398. doi: 10.1186/s40064-016-3006-7. PMID: 27610317; PMCID: PMC4994818.
[16] Gulati N, Allen CE. Langerhans cell histiocytosis: Version 2021. Hematol Oncol. 2021 Jun;39 Suppl 1(Suppl 1):15-23. doi: 10.1002/hon.2857. PMID: 34105821; PMCID: PMC9150752.
[17] Badalian-Very G, Vergilio JA, Degar BA, MacConaill LE, Brandner B, Calicchio ML, Kuo FC, Ligon AH, Stevenson KE, Kehoe SM, Garraway LA, Hahn WC, Meyerson M, Fleming MD, Rollins BJ. Recurrent BRAF mutations in Langerhans cell histiocytosis. Blood. 2010 Sep 16;116(11):1919-23. doi: 10.1182/blood-2010-04-279083. Epub 2010 Jun 2. PMID: 20519626; PMCID: PMC3173987.
[18] Chen J, Zhao AL, Duan MH, Cai H, Gao XM, Liu T, Sun J, Liang ZY, Zhou DB, Cao XX, Li J. Diverse kinase alterations and myeloid-associated mutations in adult histiocytosis. Leukemia. 2022 Feb;36(2):573-576. doi: 10.1038/s41375-021-01439-3. Epub 2021 Oct 5. PMID: 34611301.
[19] Guyot-Goubin A, Donadieu J, Barkaoui M, Bellec S, Thomas C, Clavel J. Descriptive epidemiology of childhood Langerhans cell histiocytosis in France, 2000-2004. Pediatr Blood Cancer. 2008 Jul;51(1):71-5. doi: 10.1002/pbc.21498. PMID: 18260117.
[20] Abu-Bonsrah N, Goodwin CR, Crane GM, Abiola G, Sciubba DM. Langerhans cell histiocytosis of the cervical spine. Spine J. 2016 Jan 1;16(1):e11-2. doi: 10.1016/j.spinee.2015.08.010. Epub 2015 Aug 11. PMID: 26276243.
[21] Hassan BW, Moon BJ, Kim YJ, Kim SD, Choi KY, Lee JK. Langerhans cell histiocytosis in the adult lumbar spine: case report. Springerplus. 2016 Aug 23;5(1):1398. doi: 10.1186/s40064-016-3006-7. PMID: 27610317; PMCID: PMC4994818.
[22] Huang Juan, Fu Yufei Classification and imaging manifestations of Langerhans cell histiocytosis [J]. Journal of Clinical Radiology, 2022, 41 (10): 1977-1981.
[23] Zeng Fanping, Huang Zifeng, Li Chunyan Imaging characteristics analysis of spinal Langerhans cell histiocytosis [J]. Chinese Journal of Science and Technology Database (full-text edition) Medical and Health, 2023 (6): 79-82.
[24] Zhao L, Yuan W, Yang F. Imaging-based diagnosis for extraskeletal Ewing sarcoma: A case report. Asian J Surg. 2024 Dec 3:S1015-9584(24)02531-4. doi: 10.1016/j.asjsur.2024.10.197. Epub ahead of print. PMID: 39632191.
[25] Virk J, Hillengass J. Imaging in multiple myeloma. Presse Med. 2024 Dec 9;54(1):104263. doi: 0.1016/j.lpm.2024.104263. Epub ahead of print. PMID: 39662760.
[26] Hwang YR, Lee KM, Kim HG, Na K. Diagnostic Challenge in Rapidly Growing Langerhans Cell Histiocytosis with Aneurysmal Bone Cyst in the Maxilla: A Case Report. Diagnostics (Basel). 2022 Feb 3;12(2):400. doi: 10.3390/diagnostics12020400. PMID: 35204491; PMCID: PMC8870746.
[27] St Claire K, Bunney R, Ashack KA, Bain M, Braniecki M, Tsoukas MM. Langerhans cell histiocytosis: A great imitator. Clin Dermatol. 2020 Mar-Apr;38(2):223-234. doi: 10.1016/j.clindermatol.2019.10.007. Epub 2019 Oct 24. PMID: 32513402.
[28] Dai Jiawen, Cao Xinxin Progress in the treatment of adult Langerhans cell histiocytosis [J]. Chinese Journal of Internal Medicine, 2023,62(1): 97-102. DOI: 10.3760/cma.j.cn112138-20220106-00014.
[29] Miao C, Zhang L, Sun M, Ma C. Pituitary gland and bone involvement of Langerhans cell histiocytosis in a boy and brief review of the literature. Hell J Nucl Med. 2018 May-Aug;21(2):148-150. doi: 10.1967/s002449910806. Epub 2018 Jul 12. PMID: 30006648.
[30] Wang JN, Liu T, Zhao AL, Pan BJ, Sun J, Li J, Zhou DB, Cao XX, Duan MH. Phase 2 study of oral thalidomide-cyclophosphamide-dexamethasone for recurrent/refractory adult Langerhans cell histiocytosis. Leukemia. 2022 Jun;36(6):1619-1624. doi: 10.1038/s41375-022-01555-8. Epub 2022 Mar 31. Erratum in: Leukemia. 2023 Mar;37(3):723. doi: 10.1038/s41375-023-01812-4. PMID: 35361865.
[31] Jan M, Sperling AS, Ebert BL. Cancer therapies based on targeted protein degradation - lessons learned with lenalidomide. Nat Rev Clin Oncol. 2021 Jul;18(7):401-417. doi: 10.1038/s41571-021-00479-z. Epub 2021 Mar 2. PMID: 33654306; PMCID: PMC8903027.
[32] Haroche J, Cohen-Aubart F, Emile JF, Arnaud L, Maksud P, Charlotte F, Cluzel P, Drier A, Hervier B, Benameur N, Besnard S, Donadieu J, Amoura Z. Dramatic efficacy of vemurafenib in both multisystemic and refractory Erdheim-Chester disease and Langerhans cell histiocytosis harboring the BRAF V600E mutation. Blood. 2013 Feb 28;121(9):1495-500. doi: 10.1182/blood-2012-07-446286. Epub 2012 Dec 20. PMID: 23258922.