

Single Case

Extensive Nail Changes Are a Possible Clue Indicating Multisystem Involvement in Childhood Langerhans Cell Histiocytosis: A Case Report and Literature Review

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Keywords

Extensive nail lesions · Multisystem Langerhans cell histiocytosis · Risk organ

Abstract

Introduction: Langerhans cell histiocytosis (LCH) is a rare myeloid neoplasm that can involve nearly any organ, leading to multisystem damage. Nail involvement in LCH is particularly uncommon. Here we report a case of a young boy with multisystem LCH initially presenting with nail changes. **Case Presentation:** We described a boy who presented with a 2-year history of asymptomatic changes characterized by onycholysis, subungual hyperkeratosis, and purpuric striae affecting most fingernails and toenails, initially attributed to onychomycosis. Two years later, he developed multisystem involvement affecting the pituitary gland, lungs, skin, liver, and spleen. The patient succumbed shortly after histopathological confirmation via skin biopsy due to massive gastrointestinal hemorrhage secondary to cirrhosis-induced portal hypertension. **Discussion:** Nail lesions may serve as the initial manifestation of LCH, often preceding other characteristic disease symptoms. This early presentation provides critical diagnostic opportunities for timely intervention. Consideration of LCH, biopsy, and comprehensive evaluation of organ involvement is essential to reduce the rate of misdiagnosis and the potential for unrecognized high-risk disease. **Conclusion:** Nail involvement in LCH, while rare, may serve as an early clinical indicator of multisystem disease.

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Introduction

Langerhans cell histiocytosis (LCH) is an inflammatory myeloid neoplasia characterized by the over-expansion or differentiation of pathogenic CD1a⁺/CD207⁺ (also known as langerin) dendritic cells, resulting in single or multiple organ dysfunction [1, 2]. The sites of involvement include bone, skin, pituitary gland, liver, spleen, bone marrow, lung, lymph nodes, and central nervous system except for the pituitary gland [3]. Epidemiologic data indicate an annual incidence of 2–9 cases per million children under 15 years, with peak onset occurring between ages of 1–3 years [4, 5]. LCH is classified into two distinct subtypes: single-system disease (SS-LCH) and multisystem disease (MS-LCH), defined by the number of affected organ systems. In multisystem LCH, two or more organs are involved at diagnosis, with or without risk of organ involvement (spleen, liver, and bone marrow). The 5-year overall survival (OS) rates in the single-system LCH (SS), multisystem LCH without risk organ involvement (MS-RO⁻), and multisystem LCH with risk organ involvement (MS-RO⁺) groups were 99.8%, 98.4%, and 77.0%, respectively, and the 5-year reactivation rates were 17.9%, 33.5%, and 34.3%, respectively [6]. LCH also has a significant impact on patients' health-related quality of life, especially in terms of emotion and cognition [7]. Therefore, early diagnosis and appropriate treatment are important for improving the prognosis and long-term quality of life of LCH patients.

Nail involvement remains a rare manifestation of LCH. Current evidence is limited to sporadic case reports in peer-reviewed literature. Notably, large cohort studies and systematic reviews focusing on pediatric LCH populations have not systematically addressed the clinical characterization of nail lesions [6, 8, 9]. Arturo Bonometti et al. [10] summarized the 39 cases of LCH with associated nail involvement by querying the PubMed database. This work showed that most patients were young boys, and the majority of patients had more than one nail involvement and displayed multisystem disease, especially in the pediatric sub-cohort [10]. Besides, nail lesions were present as the initial lesions of LCH in 92.3% of these cases and were associated with skin and lung involvement and considered a sign of poor prognosis [10]. Here, we describe a 6-year-old boy with nail changes as the initial sign of multisystem LCH and review the literature associated with this rare clinical manifestation.

Case Presentation

A 6-year-old boy had a 2-year history of nail disease which presented as onycholysis, subungual hyperkeratosis, and purpuric striae on most of his fingernails and toenails (shown in Fig. 1). These manifestations were initially attributed to onychomycosis. His nail lesions were unresponsive to various oral and topical antifungal treatments. However, he did not revisit the dermatologist because the lesions were asymptomatic, and he was generally well at that time. Nineteen months before diagnosis, he developed unexplained abdominal pain, accompanied by fever. Hematological examinations showed slight increases in alanine transaminase and aspartate aminotransferase, along with increased white blood cells, neutrophil count, and elevated alkaline phosphatase and gamma glutamyl transferase levels. Abdominal ultrasonography revealed mild hepatomegaly with bile duct dilation, along with echogenic shadowing debris in the gallbladder consistent with cholelithiasis. Based on clinical presentation and imaging findings, he was diagnosed with cholangitis and cholelithiasis, and subsequently received combination therapy with antibiotics and ursodeoxycholic acid. However, the symptoms of abdominal pain were frequently recurrent. He was admitted to hospital and received laparoscopic biliary tract exploration and liver biopsy. The hematoxylin-eosin and Masson staining showed “onion skin-like” fibers surrounding the small bile ducts or



Fig. 1. Clinical manifestations of LCH. Purpuric lesions and subungual hyperkeratosis on fingernails (a), toenails (b), and purpuric lesions on the instep skin of the left foot (b). c Cirrhotic portal hypertension presenting with severe varicose abdominal vein and abdominal swelling, a few purpuric lesions are also visible on the abdominal skin.

septal bile ducts, with the infiltration of a few lymphocytes and eosinophils (shown in Fig. 2), which resembled the features of primary sclerosing cholangitis [11]. Histopathological analysis revealed positivity for CD68 and α -smooth muscle actin (α -SMA), with negative staining for IgG and IgG4. Following comprehensive exclusion of infectious, hereditary, and immune-mediated sclerosing cholangitis through etiological, genetic, and immunological testing, the hepatologist confirmed the diagnosis of primary sclerosing cholangitis with biliary cirrhosis. Notably, LCH-associated markers including CD207, CD1a, and S100 were not assessed. His abdominal pain significantly improved after the surgery for common bile duct flushing and cholecystostomy, and he was discharged from the hospital. Six months before diagnosis, his parents found several asymptomatic and hemorrhagic lesions on his abdomen and instep, but he did not return to the hospital for re-examination due to the COVID-19 pandemic.

One month prior to diagnosis, the patient presented to the hepatology department of Hunan Children's Hospital with recurrent abdominal distension and a new onset of polyuria and polydipsia. Hematological examination revealed a white blood cell count of $4.14 \times 10^9/L$ (normal range $4.3 \sim 11.3 \times 10^9/L$), neutrophil count of $2.75 \times 10^9/L$ (normal range $1.6 \sim 7.8 \times 10^9/L$), hemoglobin levels of 104 g/L (normal range 118–156 g/L), platelet count of $78 \times 10^9/L$ (normal range $167 \sim 453 \times 10^9/L$), total bilirubin 31.5 $\mu\text{mol/L}$, direct bilirubin 19.7 $\mu\text{mol/L}$, albumin of 29.7 g/L (normal range 35–55 g/L), alanine transaminase of 55.5 IU/L (normal range 7–30 IU/L), aspartate aminotransferase of 97.7 IU/L (normal range 14–44 IU/L), alkaline phosphatase of 475 IU/L, gamma glutamyl transferase of 399.8 IU/L (normal range 5–19 IU/L). Abdominal ultrasonography showed cirrhosis with portal hypertension, esophagogastric fundic varices, and splenomegaly. Brain magnetic resonance imaging showed an enlarged pituitary gland, thickened pituitary stalk, and bilateral and symmetrical slight T1-weighted hyperintensity in basal ganglia. Auditory brainstem responses exhibited elevated thresholds for the stimulation. The skin biopsy at the abdomen revealed cluster-like arrangements of CD207-, CD1a-, and S100-positive lesion cells (shown in Fig. 3), with BRAF V600E positive, which supported the diagnosis of LCH. Besides, the immunohistochemical examination, using the liver specimen obtained by the laparoscopic surgery 1 year earlier, demonstrated that the LCH specific marker CD207 was positive, and CD1a was negative. The comprehensive evaluation exhibited that multiple organs were affected, including the pituitary, temporal bone, lung, skin, liver, and spleen. He was finally diagnosed with MS-RO⁺. One or two 6-week courses of induction chemotherapy with weekly vincristine 1.5 mg/m² i.v. bolus and orally prednisone 40 mg/m²/day (V-P) plus cytarabine 100 mg/m²/day for 5 days a week followed by continuation therapy were administered given the involvement of risk organs, according to the LCH-III protocol [12]. He had severe liver damage and finished preoperative examinations for LT, which were planned to be performed

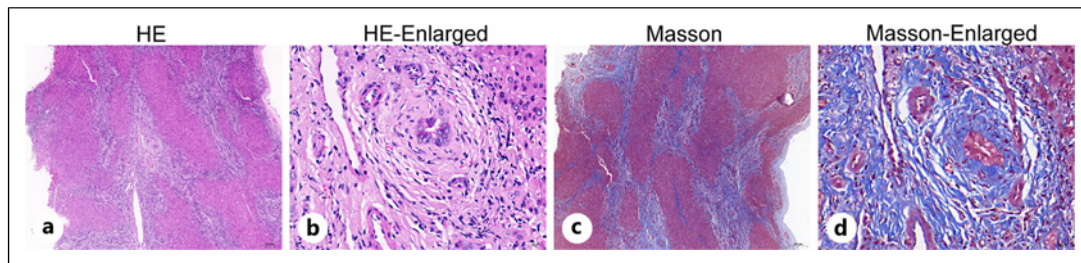


Fig. 2. Histological examinations of liver biopsy. HE and Masson staining show destruction of lobular structures (**a**), localized pseudo-bullet formation (**c**), and “onion skin-like” fibers surrounding the small bile ducts or septal bile ducts (**b**, **d**).

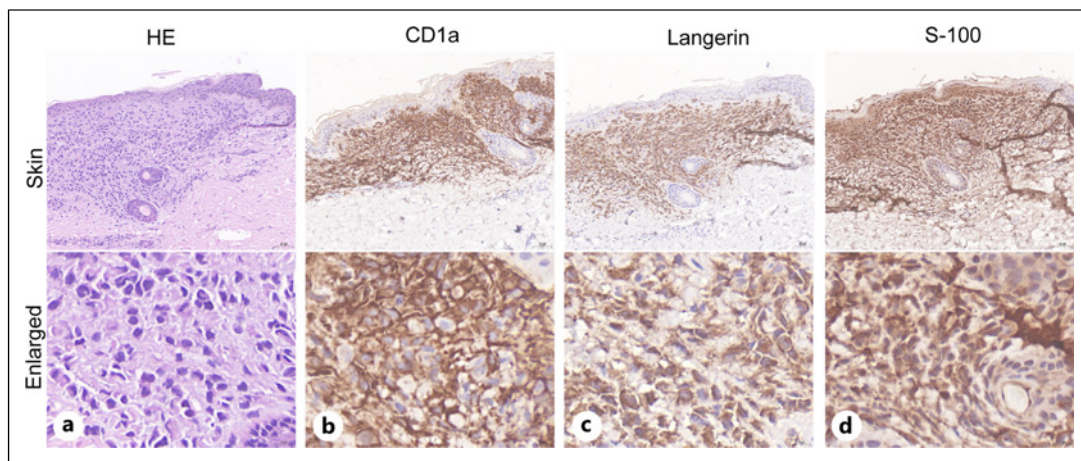


Fig. 3. Histological and histochemical examination of the skin biopsy. **a** HE staining reveals neoplastic histiocytes with pale cytoplasm and kidney-shaped nuclei. Immunohistochemistry demonstrates strong positive expression of CD1a (**b**), langerin/CD207 (**c**), and S100 (**d**).

when the disease was in remission. Unfortunately, he died of massive gastrointestinal bleeding caused by cirrhotic portal hypertension 33 days after the initiation of induction therapy with V-P plus cytarabine.

Discussion

We reported a special case of LCH initially presented with nail lesions. LCH remains diagnostically challenging due to its insidious disease progression and heterogeneous clinical manifestations that often mimic benign conditions, particularly in the early stages with nonspecific findings [1, 13, 14]. Although nail involvement is rare in LCH, the majority of documented cases present with nail lesions as the initial clinical manifestation [10]. Current evidence primarily derives from isolated case reports and small case series documenting this presentation. Ashena et al. [15] described a case of a 27-month-old girl with lesions of all fingernails and toenails at 17 months of age, who suffered from lung involvement 10 months later and unfortunately died with the feature of respiratory distress despite several rounds of systemic treatment. In addition, Ashena et al. [15] also summarized the classification of 14

Table 1. Literature review of LCH cases with nail changes

Number	Age	Sex	Classification	Nails involved, <i>n</i>	Nail biopsy	Organ system involvement	Outcome	Follow-up, months	Reference
1	2 yr 3 mo	F	MS-RO ⁻	20	No	Skin and nails, lung, bone, middle ear	Dead	22	Ashena et al. [15]
2	2 yr 4 mo	M	MS-RO ⁺	10	No	Skin and nails, bone, middle ear (otitis media), (terminal stage: bone marrow, liver, lymph nodes)	Dead	30	Kahn et al. [16]
3	2 yr	M	MS-RO ⁺	20	No	Skin and nails, liver, spleen, lung, bone	Dead	10	Kahn et al. [16]
5	2 yr 5 mo	M	MS-RO ⁺	20	No	Skin and nails, liver, lung, spleen, middle ear (chronic otitis media), bone marrow (thrombocytopenia)	Dead	12	Kahn et al. [16]
6	1 yr 6 mo	N/A	MS-RO ⁺	>1	Yes	Skin and nails, liver, lung, spleen, internal auditory canal, and lymph nodes	Remission	N/A	Kumar et al. [17]
7	13 yr	M	MS-RO ⁻	20	Yes	Skin and nails, lung	Remission	10	Yazc et al. [18]
8	9 mo	M	MS-RO ⁻	>1	No	Skin and nails, bone, lung	Remission	8	de Berker et al. [19]
9	30 yr	M	MS-RO ⁻	>1	No	Skin and nails, lung	N/A	N/A	Jain et al. [20]
10	41 yr	M	SS	1	Yes	Skin and nails	Stable	14	Bonometti et al. [10]
11	38 yr	M	SS	>1	No	Skin and nails	Remission	12	Wollina et al. [21]
12	12 yr	M	MS-RO ⁻	>1	Yes	Skin and nails, pituitary	Remission	18	Aghazadeh et al. [22]
13	10 yr	M	SS	>1	Yes	Skin and nails	Remission	N/A	Bender et al. [23]
14	1 yr 6 mo	M	MS-RO ⁺	>1	Yes	Skin and nails, liver, bone, lung	Dead	N/A	Somasundaram et al. [26]

cases of LCH with nail changes published in the literature and the results showed that 93% (13/14) of cases were multisystem LCH, including 79% (11/14) of cases with high risk. Kahn et al. [16] reported 3 patients with nail involvement in a series of 15 cases, and 2 patients died rapidly due to systemic involvement and the third one died after a period of transient remission. Notably, nail manifestations were identified as a poor prognostic indicator in this cohort.

Kumar et al. [17] reported a case of extensive nail changes in an 18-month-old child with multisystem LCH, including skin, liver, lungs, bone, spleen, internal auditory canal, and lymph nodes. Chemotherapy induced resolution of nail and cutaneous lesions with concurrent regression of other organs, though long-term outcomes including survival data and prognostic evaluation remained undocumented [17]. Yazc et al. [18] reported a case of a 13-year-old male diagnosed with multisystem LCH manifesting nail dystrophy, cutaneous lesions, and pulmonary involvement. The patient had a good prognosis after several rounds of systemic chemotherapy combined with intrapleural bleomycin, with no evidence of skin, nail involvement, and lung lesions after 10 months of follow-up. Berker et al. [19] described an infant manifesting multisystem LCH with nail and skin lesions, as well as skeletal bone involvement. Systemic treatment combined with topical steroid creams resulted in resolution of the bone and skin lesions, though residual permanent onychodystrophy (complete nail plate loss) persisted in several nails. Collectively, these clinical observations demonstrate that nail manifestations exhibit no significant correlation with disease severity or prognostic outcomes in LCH.

Nail changes are largely recorded in children and rarely occurred in adults. Jain et al. [20] reported a case of a 30-year-old man with paronychia erythema, swelling, and subungual pustules of the fingernails and toenails, as well as rashes and scales on skin and scalp, and subsequently developed lung involvement, but this case primarily documented disease onset without longitudinal outcome data. Bonometti et al. [10] described a 41-year-old male diagnosed with single-system LCH manifesting exclusive nail and cutaneous involvement. The patient was received only symptomatic treatment and the clinical presentations were stable during the 14 months of follow-up. Wollina et al. [21] documented a 38-year-old man with skin and nail involvement, which diagnosed as cutaneous adult LCH. The lesions were cleared after cladribine and thalidomide treatment, and the patient remained disease-free for 12 months off therapy. However, evidence to support using nail changes as an independent prognostic indicator was unsatisfactory due to the small number of cases and lack of consequential clinical analyses.

Nail lesions in LCH manifest as onycholysis, subungual hyperkeratosis, purpuric striae, nail dystrophy, paronychia, pachyonychia, and longitudinal grooving, which may be accompanied by nail plate deformity. In most cases, nail involvement correlates with cutaneous manifestations, and histopathological features comparable to those seen in skin lesions [22, 23]. In our case, nail lesions, as his initial presentation, were confused with onychomycosis. Notably, the presence of petechiae/hemorrhagic lesions distinguishes LCH from onychomycosis and other dermatological conditions mentioned above [24] as these abnormalities are characteristically absent in fungal nail infections, suggesting that abnormal petechiae/hemorrhagic lesions in nails and skin are a warning sign to suspect LCH. Valdivieso et al. [25] have posited that LCH-related nail pathology is frequently underrecognized as these manifestations are often misattributed to prevalent onychopathies including onychomycosis and behavioral factors like nail-biting. In our case, the nail disease had been unknown for about 2 years due to the lack of comprehensive evaluation and follow-up. Somasundaram et al. [26] report a 1.5-year-old male child with advanced multisystem involvement of liver, bone, lung, skin, and nails. His skin lesions resolved spontaneously, and a nail bed biopsy served as critical evidence

to confirm the diagnosis of LCH, indicating the importance of nail finding in LCH (Table 1).

Some limitations need to be considered. First, the initial presentation of nail lesions was frequently confused with onychomycosis, indicating that the clinical manifestations are diverse and lack specificity. However, it is critical to reevaluate the condition rather than persist with the initial diagnosis when nail lesions show no response to oral or topical antifungal therapies. Second, the patients developed sclerosing cholangitis, which hepatologists attributed to primary sclerosing cholangitis without considering their nail lesions. This oversight highlights the importance of a systematic multidisciplinary evaluation to reach an accurate diagnosis. Third, a nail biopsy was not performed to confirm the nail involvement as a skin biopsy was more appropriate in this case.

Conclusion

Nail lesions may serve as the initial clinical manifestation of LCH, preceding the onset of characteristic systemic symptoms by several months. Recognizing that changes in the nail beds may indicate LCH is critical, and a prompt nail biopsy should be performed to ensure timely diagnosis. Extensive nail changes may be a clue indicating multisystem involvement. Consideration of LCH, biopsy, and comprehensive evaluation of organ involvement is essential to mitigate diagnostic inaccuracies and prevent progression to high-risk disease.

Statement of Ethics

Ethical approval is not required for this single case report in accordance with local/national guidelines. Written informed consent was obtained from the patient's guardian and they consented to the publishing of this case report and all accompanying images. The patient died and CARE Checklist items of 8d,10a, and 12 are not applicable in this case.

Conflict of Interest Statement

The authors declare no conflicts of interest.

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Author Contributions

X.G. and B.Y. drafted the initial manuscript and shared the first authorship. J.H. prepared Figures 1–3. and H.Y. collected the data of laboratory examinations and revised the manuscript. Z.W. conceived of the manuscript. All authors approved the final manuscript as submitted and agreed to be accountable for all aspects of the work.

Data Availability Statement

All data generated or analyzed during this study are included in this article. Further inquiries can be directed to the corresponding author.

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