



The First Reported Case of Infant Soft Tissues Infection Caused by *Legionella Maceachernii*: A Case Report and Literature Review

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Abstract: *Legionella spp.* predominantly *Legionella pneumophila*, are recognized respiratory pathogens, while soft tissue infections caused by non-pneumophila species remain exceptionally rare. We present the first documented case of *L. maceachernii* soft tissue infection in an infant worldwide. The patient presented with fever accompanied by occipital, anterior thoracic, and wrist masses. Diagnosis was confirmed through metagenomic next-generation sequencing (mNGS) of tissue samples with histopathological correlation. Initial empiric therapy with vancomycin and cefotaxime yielded no clinical improvement. Subsequent mNGS analysis of cerebrospinal fluid and lesional tissue identified *L. maceachernii* infection, prompting targeted antimicrobial therapy with levofloxacin and rifampicin that resulted in clinical resolution. A review of historical cases reveals that *Legionella* soft tissue infections typically occur in immunocompromised hosts or those receiving immunosuppressive therapies, and this association prompted an investigation into possible congenital immunodeficiency in our patient. Whole exome sequencing coupled with Sanger sequencing validation identified a pathogenic mutation in the *IL2RG* gene, confirming X-linked severe combined immunodeficiency (X-SCID) in the infant and carrier status in the mother. This case highlights three paradigm-shifting concepts in pediatric infectious disease management, including 1) *L. maceachernii* should be included in the differential diagnosis of pediatric soft tissue infections refractory to standard therapy, 2) underlying immunodeficiency must be systematically evaluated in pediatric patients with atypical *Legionella* infections, and 3) the diagnostic utility of mNGS in identifying fastidious pathogens and underscore the importance of genomic investigations in elucidating immunological comorbidities.

Keywords: *Legionella maceachernii*, tissue, infant, X- SCID

Introduction

Legionella spp., aerobic Gram-negative bacilli, emerged as significant human pathogens following the 1976 Philadelphia outbreak where 182 American Legion convention attendees developed mysterious pneumonia, resulting in 29 fatalities (16% mortality).¹ This seminal event prompted the Centers for Disease Control and Prevention to deploy its largest investigative team to date, culminating in Joseph McDade's isolation of *Legionella pneumophila* - marking the genus' first scientific documentation.² Subsequent taxonomic advances have identified over 65 species, with *L. pneumophila* responsible for 90% of human infections, predominantly causing pneumonia.³ Non-pneumophila species (eg, *L. longbeachae*, *L. bozemanii*, *L. micdadei*) account for 2–7% of cases, typically manifesting as opportunistic infections in immunocompromised hosts.⁴

The pathogenic success of *Legionella* relies fundamentally on its Dot/Icm (Defect in organelle trafficking/Intracellular multiplication) type IV secretion system,^{5–7} through which *Legionella* delivers effector proteins to the host cell, holds the vesicle transport between the endoplasmic reticulum and the Golgi apparatus and interferes with the signaling pathways of the host cell to provide an optimal environment for its own growth.⁸ Such sophisticated host-pathogen interplay enables intracellular proliferation while evading lysosomal degradation—a survival strategy particularly effective in alveolar macrophages.^{9–12}

We present the first documented pediatric case of *Legionella maceachernii* soft tissue infection in a 4-month-old male with X-linked severe combined immunodeficiency (X-SCID). The infant presented with fever and multifocal masses (occipital, anterior thoracic, wrist) were diagnosed through metagenomic next-generation sequencing (mNGS) of tissue biopsies with histopathological correlation. Comprehensive genomic sequencing revealed a hemizygous IL2RG mutation confirming X-SCID. This dual discovery of a novel *Legionella* manifestation and underlying immunodeficiency provides critical insights into pediatric host-pathogen dynamics. Our findings emphasize three key imperatives: 1) expanding differential diagnoses for pediatric soft tissue infections, 2) underlying immunodeficiency must be systematically evaluated in pediatric patients with atypical *Legionella* infections, 3) implementing advanced molecular diagnostics for fastidious pathogens, such as mNGS.

Case Report

A 42-day-old male infant was admitted to the Children's Hospital Affiliated to Shandong University on December 18, 2024, presenting with a 15-day history of occipital mass and 4-day recurrent fever (peak temperature 39.8°C). Initial laboratory evaluation at an external facility revealed leukocytosis (WBC $18.25 \times 10^9/L$) and elevated C-reactive protein (CRP 76.43 mg/L). On admission, physical examination identified three distinct lesions: a 4 × 6 cm firm, ill-defined occipital mass; a 1 × 1 cm dark red wrist nodule; and a 5-mm chest wall nodule (Figure 1A–C). Color ultrasound Doppler of surface masses showed that hyperechoic nodules were detected in the subcutaneous part of the occipital region, the subcutaneous part of the left wrist, and the subcutaneous part of the right chest wall (Figure 1F–H). Cardiac color ultrasound Doppler showed that a cystic solid mass echo was detected in the pericardium of the posterior wall of the left

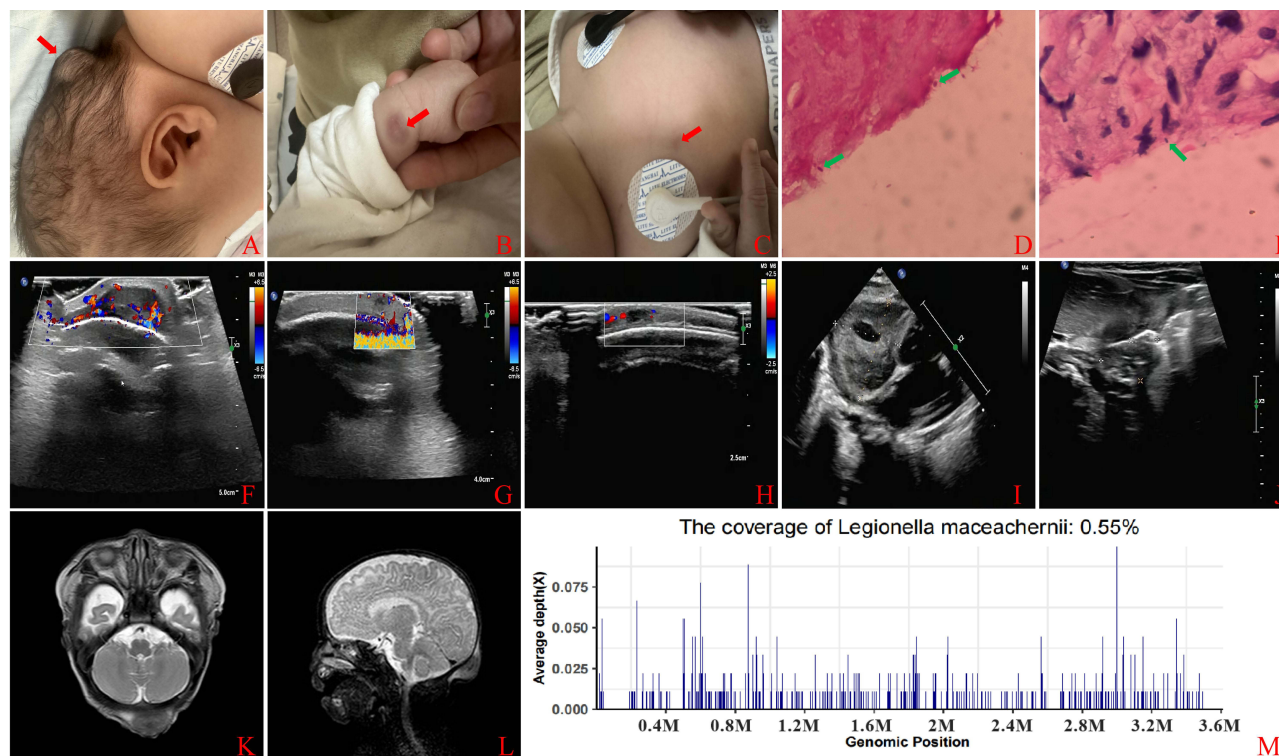


Figure 1 Clinical findings and metagenomic next-generation sequencing (mNGS) results of the pediatric patient. (A–C): nodules (indicated by red arrows) in the occipital region, wrist region, and anterior chest wall of the pediatric patient. (D): histopathological findings on Hematoxylin and Eosin (H&E) staining of the puncture biopsy tissue (green arrows point to bacterial organisms). (E): gram staining results of the puncture biopsy specimen (green arrows point to bacterial organisms). (F): a hypoechoic nodule measuring about 25 × 26 × 9 mm was detected in the occipital subcutaneous area immediately adjacent to the bone cortex. (G): a hyperechoic nodule measuring about 8 × 5 × 5 mm was detected subcutaneously in the left wrist. (H): a hyperechoic nodule measuring about 5 × 4 × 3 mm was detected subcutaneously in the right chest wall. (I): echoing cystic solid mass of approximately 34 × 27 × 15 mm in the pericardium of the posterior wall of the left ventricle, with indistinct borders. (J): an irregular solid hypoechoic mass measuring approximately 24 × 15 × 11 mm was detected posterior to the posterior mediastinal mass, with unclear borders, uneven internal echoes, and localized compression of lung tissue. (K and L): a mass-like iso-slightly shorter T1, slightly longer/iso T2 signal shadow was seen under the soft tissue of the scalp in the posterior occipital region, with a size of about 25.8 × 16.2 × 18.5 mm. (M): sequence comparison and coverage analysis of mNGS sequenced sequences with those of *L. maceachernii*.

ventricle. The border was unclear (Figure 1I); an irregular solid hypoechoic mass was detected posterior to the posterior mediastinal mass, with unclear border, heterogeneous internal echogenicity, and localized compression of lung tissue (Figure 1J). Cranial MRI showed a mass-like iso-slightly shorter T1, slightly longer/iso T2 signal shadow under the soft tissue of the scalp in the posterior occipital region (Figure 1K and L).

Laboratory findings included leukocytosis (WBC $19.57 \times 10^9/L$), elevated C-reactive protein (CRP 148.40 mg/L), hypogammaglobulinemia (IgA < 0.058 g/L, IgM 0.141 g/L), and profound lymphopenia: total T cells $60/\mu L$, CD4⁺T cells $4/\mu L$, CD8⁺T cells $1/\mu L$, NK cells $17/\mu L$, and elevated B cells ($1025/\mu L$). The infant was heavily infected and was empirically given vancomycin (0.075 g IV pumped q8h 12.18–12.21) in combination with cefotaxime (0.25 g IV pumped bid 12.18–12.21) for anti-infective and immunoglobulin supportive therapy. Cerebrospinal fluid examination: colorless, slightly turbid, Cl⁻ 115 mmol/L, glucose 0.73 mmol/L, protein quantification 1102 mg/L, white blood cell counts $1.165 \times 10^9/L$, percentage of single nucleated cells 48.6%, and percentage of lobulated nucleated cells 51.4%, suggesting intracranial infection, and immunoglobulin was added on top of the original antibiotic to strengthen immune support. Dexamethasone was added to the original antibiotic for anti-inflammation, while the cerebrospinal fluid was sent for mNGS detection. Cerebrospinal fluid mNGS sequencing results returned *L. maceachernii* (Figure 1M).

L. maceachernii is an intracellular bacterium, after discussion, it was decided to give the infant levofloxacin and rifampicin anti-infective treatment (0.049 g intravenous pump q12h, 12.21–12.30). As the nature of the mass was unknown, it was considered to be an infectious or neoplastic mass, and ultrasound-guided biopsy was performed to clarify the nature of the mass, and Gram-negative rod-shaped bacilli were found in both Gram and HE stains of the puncture tissue (Figure 1D and E), and at the same time, the puncture tissue was sent to mNGS sequencing, which showed that it was *L. maceachernii*. Tissue and CFS used BCYE medium, but the cultures were all negative.

During the treatment, the infant's soft tissue mass was reduced, but the infant still had recurrent fever. Taking into account the infant's clinical manifestations and various examinations, considering that extrapulmonary infections of *Legionella spp.* often occur in immunocompromised populations, we hypothesized that the infant might have a defect in the immune system, and we performed genetic tests on the infant and his parents to clarify the diagnosis. Whole exome and Sanger sequencing of the infant and his parents revealed a hemizygous mutation in the *IL2RG* gene: the change in nucleotide 718 from thymine T to cytosine C (c.718T > C) resulted in a change in amino acid 240 from tryptophan to arginine (p. Trp240Arg), no mutation in this locus in the father and a heterozygous mutation in this locus in the mother (Figure 2). Based on the above results, the infant was diagnosed with X-SCID. Due to the infant's severe infection and poor immune function, hematopoietic stem cell transplantation is the first choice for the treatment of X-SCID, and he has now gone to a higher-level hospital.

Discussion

Legionella usually causes pulmonary infections, but soft tissue infections are rare, and this case report is the first soft tissue *L. maceachernii* infection found in infant. We searched PubMed with the keywords “*Legionella* and soft tissue” and compiled previous case reports of a total of 10 patients (excluding our patient),^{13–22} no immunocompromised or related underlying disease (n=1),¹³ received organ or stem cell transplantation (n=4),^{14–17} taking cortisol hormones or immunosuppressive drugs (n=7),^{15,16,18–22} required surgical debridement or incision and drainage after soft tissue infection (n=4).^{16,17,20,22} There were 6 patients received treatment and recovered,^{13,16–20} 1 patients developed new lesions,²² and 3 patients died,^{14,15,21} the mortality rate reported in 10 cases was as high as 30%. As a result, soft tissue infections caused by *Legionella* warrant clinical attention (Table 1).

Legionella is an opportunistic pathogen in aquatic environments, commonly found in freshwater environments (eg lakes, hot springs, artificial water supply systems), and can parasitize protozoa, such as amoebae, or invade mammalian alveolar epithelial cells and monocyte macrophages and reproduce therein, ultimately leading to death of the host cells.²³ *Legionella* has a low risk of infection in healthy people, but immunocompromised individuals (organ transplant, HIV patients) may become infected by inhaling aerosols containing the bacteria (atomized droplets from contaminated water sources such as showerheads, air-conditioning cooling towers, humidifiers, etc.), which can lead to pneumonia or other respiratory infections, with symptoms that include fever, coughing, and respiratory distress, and can lead to multiple organ failure in severe cases. Currently, *Legionella* infections are identified by isolation culture, pathologic examination, serologic testing, mass spectrometry, PCR, DFA and mNGS.³ *Legionella spp.* is susceptible to macrolides

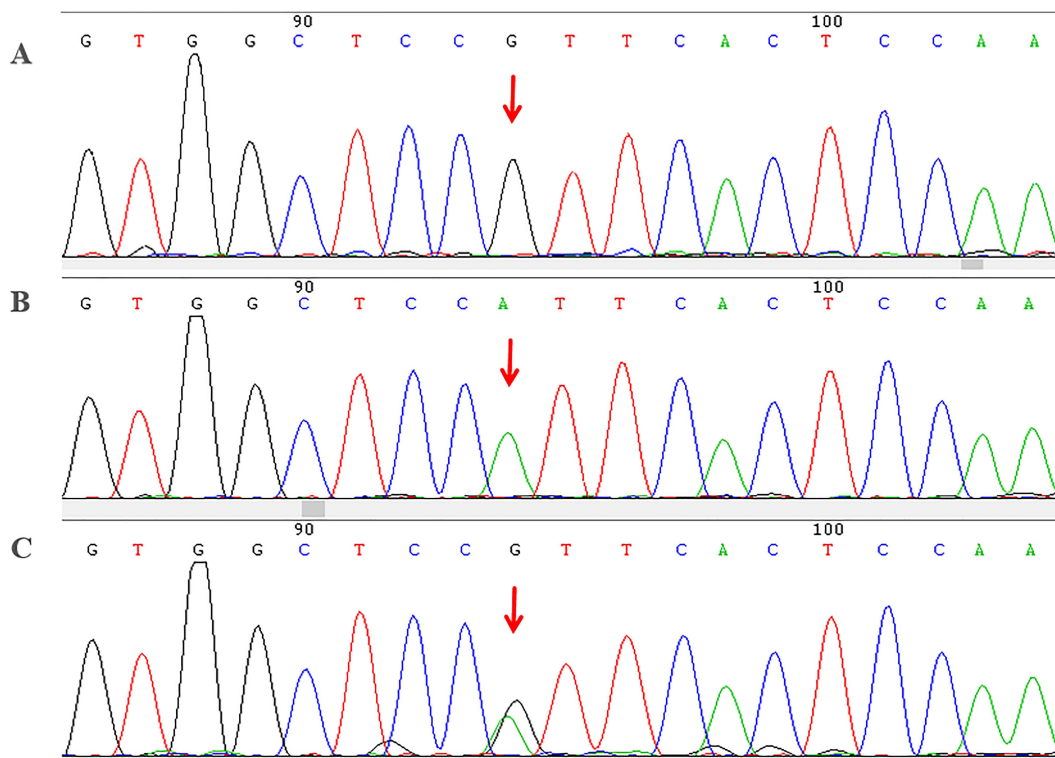


Figure 2 Chromatograms showing a mutation in the X-SCID gene. **(A)**: deletion of “T” at position 718 in the patient (indicated by red arrows). **(B)**: wild-type c. 718T sequence in the patient’s healthy father (indicated by red arrows). **(C)**: heterozygous c. 718delT deletion in patient’s mother (indicated by red arrows).

(erythromycin, azithromycin and clarithromycin), fluoroquinolones and rifampicin.^{24,25} *L. maceachernii* was detected in the cerebrospinal fluid and puncture tissue mNGS of the infant in this case, and we also found gram-negative rod-shaped bacilli in the histopathological sections of the child, but unfortunately, the child’s blood, puncture tissue, and CSF were negative after 14 days of culture due to the extremely low number of *bacilli* and the difficulty of culturing clinical specimens of non-pneumophila *Legionella* infections in vitro. It has been reported in the literature that among *Legionella*-infected patients, those with immunosuppression are more susceptible to non-pneumophila *Legionella* infections than immunocompetent patients,³ combined with the child’s laboratory tests and pathogenetic testing, we further hypothesized that the child may have an immune system defect, and further genetic sequencing was performed on the infant and his parents, and the results confirmed our conjecture.

X-SCID is a rare inherited immune system disorder that is the most common type of severe combined immunodeficiency disease (SCID), accounting for about 50% of SCID cases. The disease is inherited in an X-linked recessive pattern and is caused by mutations in the *IL2RG* gene located on the X chromosome (Xq13.1) and mainly affects males (because males have only one X chromosome), and female carriers are usually asymptomatic but may pass the mutated gene to their offspring. The *IL2RG* gene encodes a common γ chain, a protein that is a core component of various interleukin receptors (eg, IL-2, IL-4, IL-7, IL-9, IL-15, IL-21).²⁶ Within a few months after birth, the infant has recurrent and severe bacterial, viral, fungal infections (such as pneumonia, chronic diarrhea, thrush, BCG vaccination, etc.), and other symptoms are growth retardation, intractable rash, etc. and most untreated infant die of severe infection within 1 year of age. The treatment of choice is hematopoietic stem cell transplantation (HSCT), the earlier the transplantation the higher the survival rate of the patient, and the preferred donor is matched sibling or umbilical cord blood.²⁷ In this case, the mother had a hemizygous mutation in the *IL2RG* gene: from thymine T to cytosine C (c. 718T > C), resulting in the change of amino acid 240 from tryptophan to arginine (p. Trp240Arg), for missense mutations, we used the SWISS-MODEL database (<https://swissmodel.expasy.org>) to query the 3D model data of wild-type and mutant proteins of *IL2RG* gene, visualized with PyMOL (<https://pymol.org>), and the predictive model obtained is shown in Figure 3.

Table 1 Reported Cases of Soft Tissue Infection with *Legionella*

No.	Age	Gender	Presence of Underlying Diseases	Soft Tissue Infection Manifestations	Strains	Methods of Diagnosis	Treatment After Diagnosis
1 ¹³	73	Female	High blood pressure	Abscesses of the right neck, left wrist, left arm	<i>L. cinchonatiensis</i>	PCR, BCYE medium culture	Clarithromycin, rifampicin
2 ¹⁴	27	Female	Pre-B-cell acute lymphoblastic leukemia, after allogeneic cord blood stem cell transplantation	Back and leg pain, diffuse erythema, and subcutaneous mass on the back of the left thigh	<i>L. pneumophila</i>	Tissue culture, DNA sequencing	Azithromycin
3 ¹⁵	56	Male	Hepatitis B combined with hepatocellular carcinoma, after liver transplantation	Swelling and pain in the right forearm, vocal cord lesions	<i>L. micdadei</i>	Pathologic discovery of organisms, PCR	Azithromycin
4 ¹⁶	39	Female	Oral azathioprine and prednisone after kidney transplantation	Cellulitis of the left hand and arm	<i>L. micdadei</i>	DFA fluorescent staining, BCYE medium culture	Surgical debridement, amputation, erythromycin
5 ¹⁷	9	Female	Post-kidney transplant	Left posterior neck mass	<i>L. micdadei</i>	PCR amplification assay	Clarithromycin, surgical incision and drainage
6 ¹⁸	82	Male	Rheumatoid arthritis, oral prednisone, and methylprednisolone injected into the right metacarpophalangeal joint and shoulder	Open wound with swelling and suppuration on the right hand, swelling on the left elbow	<i>L. bozemanii</i>	PCR amplification assay	Moxifloxacin, clarithromycin
7 ¹⁹	66	Male	Follicular lymphoma, INF- α	Erythematous rash on the right side of the chest and abdomen	<i>L. pneumophila</i>	BCYE medium culture, DFA fluorescence	Erythromycin
8 ²⁰	62	Female	Rapidly progressive glomerulonephritis due to necrotizing vasculitis, oral prednisone, cyclophosphamide	Cellulitis of the right lower leg, abscess of the right lateral ankle	<i>L. micdadei</i>	DFA fluorescent staining, BCYE medium culture	Erythromycin IV for 6 weeks, surgical incision and drainage
9 ²¹	65	Female	Interstitial lung disease and idiopathic thrombocytopenic purpura, methotrexate, high-dose hormone therapy	Cellulitis of the right posterior thigh and buttocks	<i>L. pneumophila</i>	BCYE medium culture, DFA fluorescence staining	Azithromycin (used prior to death,)
10 ²²	68	Female	Polymyalgia rheumatica, prednisone and methotrexate	Swelling and erythema of the ring finger of the right hand with pressure and pain, red nodules on the right forearm	<i>L. maceachernii</i>	BCYE medium culture, DFA fluorescence staining	Surgical debridement, levofloxacin for 9 weeks
11 (our case)	42 day	Male	SCID	Subcutaneous nodules on the occiput, wrist, and anterior chest	<i>L. maceachernii</i>	mNGS, pathologic tissue biopsy	Levofloxacin

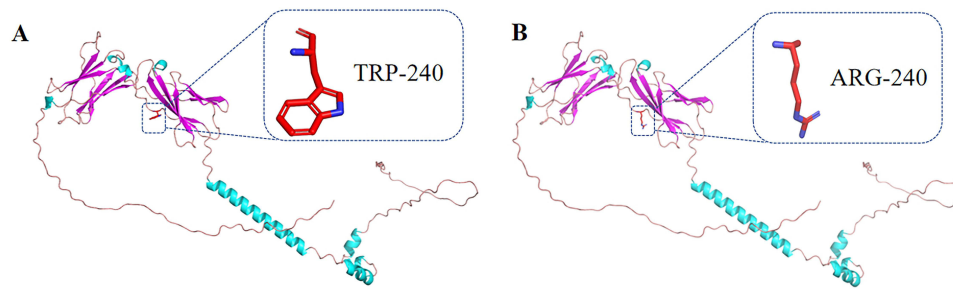


Figure 3 Wild-type and mutant-type crystal structure of the X-SCID gene. **(A)**: wild-type amino acid number 240 consists of tryptophan in the patient's healthy father. **(B)**: mutant-type amino acid number 240 changes from tryptophan to arginine in the patient and patient's mother.

Limitations

This case report has several limitations. Firstly, the definitive diagnosis of *L. maceachernii* infection relied primarily on metagenomic next-generation sequencing (mNGS) of cerebrospinal fluid and lesional tissue. Despite supportive histopathological findings (Gram-negative bacilli), all attempts at conventional culture (blood, tissue, CSF) using BCYE medium were unsuccessful, precluding isolation and antimicrobial susceptibility testing of the pathogen. This highlights the inherent challenges in culturing non-pneumophila *Legionella* species and underscores the diagnostic reliance on molecular methods in such scenarios. Secondly, after 9 days of treatment with levofloxacin in combination with rifampicin, the infant's soft-tissue infection improved, but the mass did not disappear until discharge from the hospital; therefore, we were unable to determine the optimal length of treatment for *L. maceachernii* infection of the soft tissues of immunocompromised children. Thirdly, the nature of the pericardial and mediastinal masses detected on imaging remained undetermined, although they were hypothesized to be related to the underlying immunodeficiency. Soft tissue infections with *Legionella*, particularly *L. maceachernii*, are uncommon, and in the differential diagnosis, attention needs to be paid to the patient's underlying disease, medication use, and, in infant, the presence of immune deficiencies due to congenital disorders needs to be considered.

Conclusion

This case report provides the first case of soft tissue *L. maceachernii* infection in an infant worldwide and reviews the relevant literature. In the soft tissue of infant's infections with *Legionella*, the presence of comorbid immunodeficiency disorders needs to be considered. Our results illuminate the critical intersections between host immunity and atypical pathogen behavior. Our findings extend the clinical spectrum of legionellosis while reinforcing three paradigm-shifting concepts in pediatric infectious disease management, including 1) *L. maceachernii* should be included in the differential diagnosis of pediatric soft tissue infections refractory to standard therapy, 2) underlying immunodeficiency must be systematically evaluated in pediatric patients with atypical *Legionella* infections, and 3) the diagnostic utility of mNGS in identifying fastidious pathogens and underscore the importance of genomic investigations in elucidating immunological comorbidities.

Date Sharing Statement

All data generated or analyzed during this study are included in this published article. It is clarified that the data presented in this study were contributed by Shifu Wang, one of the corresponding authors.

Ethical Approval and Consent Statements

This study underwent rigorous review and was approved by the Ethical Review Committee of Children's Hospital Affiliated to Shandong University (approval no. SDFE-IRB/P-2022017). All procedures were conducted in strict compliance with the Ethical Review of Biomedical Research Involving Human Subjects (2016), the Declaration of Helsinki, and the International Ethical Guidelines for Biomedical Research Involving Human Subjects.

This study has been reviewed and approved by the Research Ethics Committee of the Children's Hospital Affiliated to Shandong University (Approval No. SDFE-IRB/P-2022017), and the legal guardian of the patient has provided written informed consent for publication of the case details and clinical images.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

All authors disclosed no competing interests for this work.

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