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Roseomonas Bacteremia: A Clue To Underlying Severe Combined Immunodeficiency

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ABSTRACT

Background:
Severe combined immunodeficiency (SCID) is a rare but fatal inborn error of immunity characterized by profound defects in cellular and humoral immunity. It predisposes infants to severe, recurrent opportunistic infections. Unusual or low-virulence pathogens may serve as early clinical clues to an underlying immunodeficiency. Roseomonas species are slow-growing, gram-negative bacilli with low pathogenic potential and are rarely implicated in paediatric infections and predominantly affect immunocompromised hosts.

Case Report:
A 40-day-old male infant with an unremarkable perinatal history presented with high-grade fever and elevated inflammatory markers. Evaluation showed leukocytosis, raised C-reactive protein and multiple hepatic microabscesses on ultrasonography. Cerebrospinal fluid analysis was unremarkable. During hospitalisation, he developed multiple pustular skin lesions and blood culture grew a slow-growing Roseomonas species identified by the VITEK system. Antibiotics were adjusted according to susceptibility which led to clinical improvement and resolution of hepatic abscesses. Owing to the unusual organism and hepatic abscesses, an inborn error of immunity was suspected. At nine months of age, the child was hospitalised again with recurrent lower respiratory tract infections, pneumonia, mucocutaneous candidiasis and failure to thrive. Immunological workup revealed markedly reduced CD3 and CD56 cells with hypogammaglobulinemia, absence of the thymic shadow on chest radiography. Genetic studies confirmed X-linked SCID due to a hemizygous stop-gain mutation in IL2RG gene. He was referred for hematopoietic stem cell transplantation.

Conclusion:
Paediatricians should maintain a high index of suspicion for inborn errors of immunity in infants presenting with unusual or opportunistic infections including Roseomonas infection. This will enable early diagnosis and appropriate interventions at an early and appropriate time.

Keywords: Hepatic Abscess, Opportunistic Infection, Roseomonas, Severe Combined Immunodeficiency, X-linked SCID

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INTRODUCTION

Severe combined immunodeficiency syndrome (SCID) is an uncommon inborn error of immunity, that typically results in fatality during infancy and predisposes individuals to severe, life-threatening infections.[1] SCID has a low incidence rate, estimated at approximately 1 in 50,000 to 1 in 1,00,000 live births.[2] The most common indicator of immunodeficiency in children is recurrent infections occurring more frequently than expected and affecting multiple sites.[3] Additionally, a single severe, opportunistic, or unusual infection can serve as an initial indicator of immunodeficiency. Roseomonas is a gram-negative, slow-growing, aerobic, non-fermentative bacteria characterized by their distinctive pink-pigmented colonies. They are generally opportunistic pathogens with limited pathogenic potential. A notable increase in reported human infections over the past two decades, particularly among immunocompromised individuals have been reported.[4] Here, we present the case of a 40-day old child diagnosed with T- B+ NK- SCID.

CASE REPORT

A 40-day-old male infant, weighing 4.75 kg, was referred with a 5-day history of fever and urinary tract infection, with urine culture showing insignificant growth of Klebsiella pneumoniae. The child had been treated for 2 days with piperacillin-tazobactam, and amikacin. Clinical examination did not reveal an obvious source of infection, although the liver was palpable 3 cm below the right costal margin. Blood investigations revealed anemia, leukocytosis, and significantly elevated C-reactive

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protein. The child was started empirically on cefuroxime, amikacin, and antipyretics. Cerebrospinal fluid analysis was normal. The child had ongoing fever spikes without a clear focus, hence high-resolution ultrasound of the abdomen and pelvis revealed multiple hepatic abscesses measuring 3-5 mm, likely pyogenic (Figure 1 A). Blood cultures obtained on day 5 showed no significant growth. However, on day 6, the child developed multiple pustular lesions on the ear, trunk, and extremities (Figure 1B). By day 7, blood culture revealed the growth of a slow-growing Roseomonas species, a member of the acetobacteraceae family, which grew within 7 days of incubation. The VITEK identification system confirmed the organism as Roseomonas species (Figure 1C).

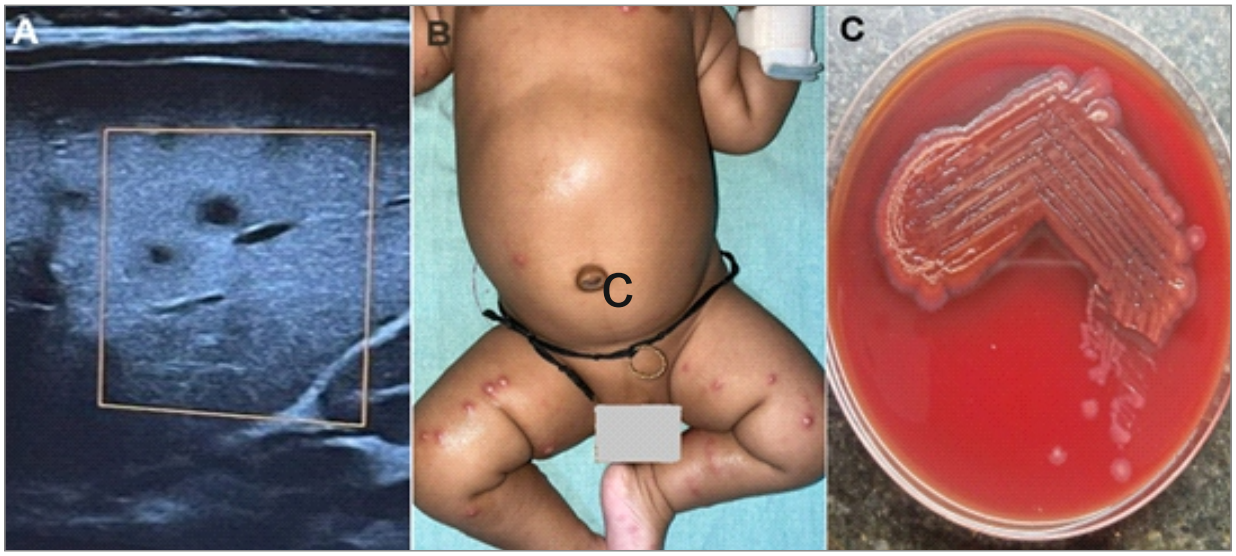

Figure 1.(From Left to right) A) High-resolution ultrasound of 40-day old male with persisting fever spikes showing multiple hepatic micro-abscesses B) Multiple pustular lesions over the trunk and extremities at 45 days of life C) Blood agar showing pink colored colonies of Roseomonas species.

The child showed clinical improvement, and a repeat ultrasound revealed small hypoechoic lesions in the liver, indicating resolving abscesses. On follow-up after 2 weeks, the child was afebrile, had gained weight, and a repeat abdominal ultrasound was normal. Due to hepatic abscesses and an unusual bacterial infection, the possibility of an inborn error of immunity was considered. The child had a history of recurrent respiratory tract infections, urinary tract infections, and cutaneous infections, all requiring oral antibiotics. At 9 months of age, the child presented with fever and cough. Blood tests showed moderate microcytic hypochromic anemia, neutrophilic leukocytosis, thrombocytosis, and elevated inflammatory markers. The child was treated with antibiotics, antipyretics, and bronchodilator nebulizations. A nasopharyngeal PCR swab was positive for Pneumocystis jiroveci and Klebsiella pneumoniae. Additionally, the child had hypopigmented patches on the neck and trunk, suggestive of a candidial infection. An immunodeficiency panel revealed low levels of CD3, CD56 cells, and immunoglobulins, suggesting SCID. A chest X-ray confirmed the absence of the thymus. The child was started on antibiotics, antivirals, and monthly intravenous immunoglobulin (IVIG) infusions. He was referred for hematopoietic stem cell transplantation (HSCT) at a specialized center. Whole exome sequencing revealed an IL2RG-related disease, with a stop-gain variant observed in exon 7 of IL2RG in the hemizygous state, inherited from the mother.

DISCUSSION

Severe combined immunodeficiency (SCID) is characterized by severe T-cell deficiency with variable B and NK-cell involvement, representing the most severe congenital immunodeficiency. Without achieving immunological clinically classified as T-B+ or T-B- SCID depending on B cell reconstitution through interventions like bone marrow transplantation or enzyme replacement, death typically occurs before the age of one. SCID encompasses various types, involvement, all presenting with low to absent T cell levels. The X-linked form is the most common, presenting as T-B+ NK- SCID, characterized by diminished T cells, normal B cell levels, and reduced natural killer cells, as observed in our patient.[5]

Infants with SCID commonly experience recurrent episodes of diarrhea, pneumonia, sepsis, and skin infections within the first months of life, often followed by failure to thrive. Our patient presented at 6 weeks of age with a hepatic microabscesses and uncommon bacterial infection. Viral infections such as varicella, measles, parainfluenza, CMV, Epstein-Barr virus, and fungal infections like Candida and Pneumocystis jirovecii are common.[6]They will typically have hypoplastic thymus tissue, and there is often adenoid, tonsil, and peripheral lymph node hypoplasia.[7] Initially, immunoglobulin levels may appear normal due to maternal antibodies.[8] Analyzing lymphocyte subpopulations aids in identifying the specific type of SCID. Allogeneic hematopoietic cell transplantation (HCT) is curative in the majority of patients receiving a human leukocyte antigen (HLA)-matched sibling donor.[9] Our patient had a rare slow-growing organism infection. A systematic review of all published cases of Roseomonas infections showed an overall mortality of 3%, with mortality attributed to Roseomonas at 1%. Roseomonas species rarely infect humans due to their low pathogenicity, and their clinical characteristics are still not fully understood.[10] Central lines are often common sites of infection for Roseomonas bacteremia in children. Opportunistic infections caused by *R. mucosa* have been associated with skin microbiota.[11] A review of the literature on reported Roseomonas infections in children is summarized in table 1.

S. no	Year	Author	Age (in years/months)/ Sex	Diagnosis	Source
1	2010	Bard JD et al[12]	18y/ M	Recurrent UTI	Blood
2	2017	Mamtora DK et al[13]	10m / F	Neuroblastoma	Blood
3	2018	Kimura K et al[14]	9y/ M	Cerebellar medulloblastoma with febrile neutropenia	Blood
4	2020	Schlappi C et al[15]	5y/M	HbS/β thalassemia status postsurgical splenectomy	Blood
5	2021	Waris RS[16]	17y/M	Meningitis	CSF
6	2022	Reghu A et al[17]	15y/ F	Multi system inflammatory syndrome	Blood
7	2023	Matsuhisa Y et al[18]	10y /M	sebum-deficient eczema	Pus wound culture
8	2024	Agrawal et al[19]	1y/M	Acute gastroenteritis with mild dehydration	Stool
9	2024	Niti Bhai et al[20]	3y/ M	Meningitis	Blood

Table 1: Review of literature on reported Roseomonas infections in children

CONCLUSION:

Early evaluation with a high index of suspicion is key in diagnosing inborn errors of immunity in infants, especially with uncommon bacterial infections such as Roseomonas. Even if a child appears to be thriving, this does not rule out the possibility of inborn errors of immunity. Additionally, chest X-rays should be scrutinized for a narrow mediastinum and the absence of the thymus, both of which can be indicative of inborn errors of immunity. Given the rarity of such presentations, this case contributes valuable insight to the literature on SCID and opportunistic infections, particularly *Roseomonas* bacteremia in immunocompromised children, and highlights the importance of vigilance in recognizing these rare but potentially life-threatening conditions.

Conflicts of Interest: None

Source of funding: None

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Author Contribution:
JSD: Data acquisition, manuscript drafting. **PBK**: Data acquisition, manuscript review and editing. **HPL**: Reviewed the final version of the manuscript and approved it for publication.

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