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Multiple liver abscesses in an adolescent with sickle cell disease, a rare manifestation: A case report

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Abstract

Background: Hepatic abscesses are rare in pediatrics and remain a poorly documented complication in patients with sickle cell disease.

Case presentation: A 15-year-old boy with sickle cell disease presented with acute right upper quadrant pain and was diagnosed with multiple liver abscesses. *A Streptococcus anginosus* was identified from the cyst puncture. Fewer than ten cases of this complication have been reported in children with sickle cell disease.

Conclusion: Hepatic abscesses in children with sickle cell disease are rare but should be considered in the differential diagnosis of fever and/or abdominal pain in these patients. Prompt management includes surgical or percutaneous drainage and prolonged antibiotic therapy.

Keywords: Sickle cell disease; Abdominal pain; Liver abscess; *Streptococcus anginosus*.

Abbreviations: SAG: *Streptococcus Anginosus* Group; SCD: Sickle Cell Disease.

Introduction

Hepatic abscesses are rare in pediatric populations and constitute a poorly documented clinical entity in patients with sickle cell disease (SCD). SCD, the most common genetic disorders worldwide, predisposes children to numerous infectious complications, particularly due to functional asplenia and vascular sequelae related to sickled erythrocytes [1]. While abdominal pain is a frequent symptom during vaso-occlusive crises, it may also signal severe infectious complications such as hepatic abscess. Clinical presentation is often nonspecific, which can lead to diagnostic delay [1,2]. Streptococcus anginosus, a member of the Streptococcus anginosus group (SAG), is an opportunistic pathogen known for its propensity to cause deep-seated visceral abscesses, frequently in association with anaerobic bacteria as part of polymicrobial infections [3,4]. To our knowledge, this is the first reported case of a Streptococcus anginosus hepatic abscess in a pediatric patient with SCD.

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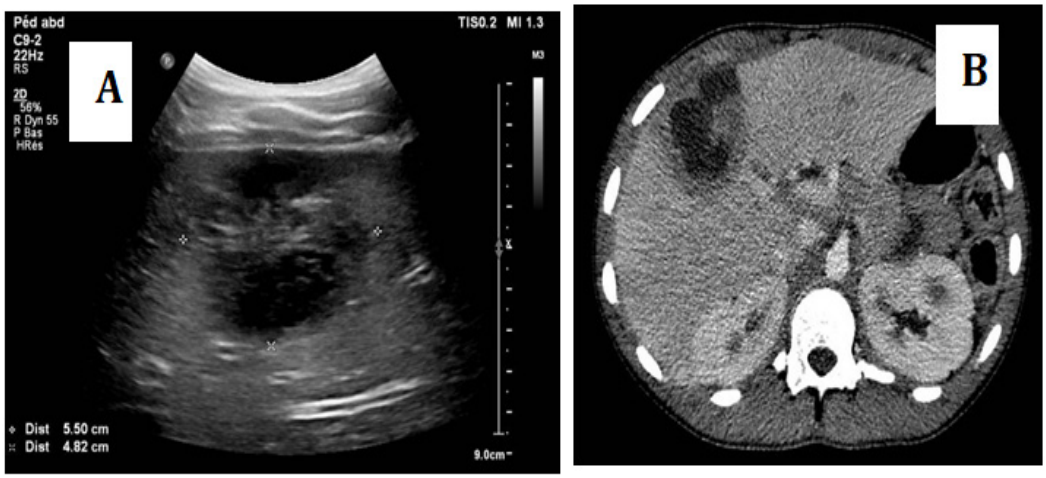

Figure 1: Multifocal hepatic abscesses.
(A) Abdominal CT scan, axial slice: Heterogeneous hepatic lesion located between segments V and VIII, with a centrally hypodense, fluid-like content and a thick peripheral wall.
(B) Abdominal ultrasound: Heterogeneous hypoechoic, poorly vascularized, and multiloculated lesion located between segments V and VIII.

Case presentation

A 15-year-old boy with SCD, chronically treated with hydroxycarbamide, presented to the emergency department with a three-day history of abdominal pain and low-grade fever. He reported significant fatigue over the past month. History revealed poor adherence to hydroxycarbamide therapy in recent weeks. On clinical examination, he exhibited conjunctival jaundice and tender hepatomegaly. Laboratory tests showed marked inflammatory syndrome with a CRP at 110 mg/L, neutrophilic leukocytosis (white blood cell count: 21,000/mm³; neutrophils: 15,000/mm³), stable hemoglobin at 8.5 g/dL and usual hemoglobin electrophoresis (HbA2 1.9%, HbF 28.1%, HbS 70%). Abdominal imaging, including ultrasound and CT scan, revealed three multifocal hepatic abscesses [Fig. 1A, B]. The largest, located in segment V of the right liver lobe, measured 6.0 × 5.9 × 5.8 cm (AP × LL × CC) and was associated with peripheral thrombosis of the anterior and posterolateral branches of the right portal vein. Percutaneous drainage of the main abscess was performed under radiologic guidance, yielding 300 mL of purulent fluid. Empirical broad-spectrum intravenous antibiotic therapy was initiated with a third-generation cephalosporin and metronidazole. Culture of the purulent fluid yielded a multisensitive Streptococcus anginosus, allowing de-escalation to penicillin G and metronidazole.

The patient improved clinically and the inflammatory syndrome declined steadily up to CRP at 28 mg/L at day 12 but after 15 days of targeted antibiotic therapy, he experienced recurrence of fever and right upper quadrant pain and deterioration of general condition. CRP levels rose again to 61 mg/L. Repeat ultrasound confirmed persistence of the right subcapsular hepatic abscess (unchanged at 6.0 × 3.5 × 6.0 cm) and stability of a second abscess adjacent to the inferior vena cava, without progression of portal vein thrombosis. A second percutaneous drainage procedure was performed and a multi-holed drain was left in place for eight days. The clinical course was favorable, with resolution of fever and gradual reduction of the abscesses on follow-up imaging. In total, the patient received three weeks of intravenous metronidazole and four weeks of intravenous penicillin G. An oral switch to moxifloxacin was then initiated, for a total antibiotic duration of six weeks.

Discussion

Hepatic abscesses are rare entities in children, with an estimated incidence ranging from 3 to 25 cases per 100,000 paediatric hospitalizations [1]. Their clinical presentation is often subtle, particularly in patients with SCD, where abdominal pain and fever are common symptoms that can obscure serious complications.

The pathophysiology of hepatic abscesses in SCD involves several mechanisms: hepatic microinfarctions caused by sickled erythrocytes, functional asplenia predisposing to bacteremia, and iron overload secondary to chronic transfusions [1,2,5].

Literature data demonstrate heterogeneity in the causative pathogens. Reported pathogens include Fusobacterium spp., Staphylococcus aureus, and Klebsiella pneumoniae [3,6,7]. However, hepatic abscesses are generally considered polymicrobial, particularly when involving Streptococcus anginosus, which is frequently associated with anaerobic bacteria that are difficult to isolate due to their extreme oxygen sensitivity [3]. To our knowledge, this case represents the first reported pediatric case of a hepatic abscess caused by Streptococcus anginosus in a child with SCD.

Streptococcus anginosus is part of the SAG, known for its pyogenic tropism and its capacity to cause deep-seated abscesses, particularly in the abdominal region [3]. It is a commensal organism of the orodigestive flora that can become pathogenic in cases of intestinal translocation, especially in immunocompromised individuals or those with chronic illnesses [3,4]. The hepatobiliary system has also been described as a potential entry point for Streptococcus anginosus infections [3]. However, in our patient, the biliary tract appeared normal, and no evident pathway was identified, with sterile blood cultures and no biliary abnormalities.

To date, fewer than ten cases of hepatic abscesses have been reported in children with SCD, most of which involved adoles-

cents. This predominance may be explained by poorer treatment adherence in adolescents and splenic infarction already being well established at that age.

Below, we present an updated version of the table published by Marolf et al. (2016), summarizing previously reported cases in the literature [1].

Publication date	Age / Sex	Isolated bacteria	Presumed pathogenicity
1971	4 years / M	*Peptococcus*	Hematogenous route
1980	5 years / F	*Fusobacterium necrophorum*	Hematogenous route
1993	12 years / F	*Klebsiella spp.*	Hematogenous route or contiguous spread after cholecystitis
1993	10 years / F	*Bacteroides*	Hematogenous route
2005	12 years / M	Aseptic (drained after 5 days of antibiotic therapy)	Iatrogenic infection after liver biopsy or hematogenous route
2006	16 years / F	*MRSA*	Iatrogenic after ERCP and cholecystectomy or contiguous spread after cholecystitis
2016 [1]	16 years / M	*Fusobacterium spp.*	Hematogenous route
2021 [2]	15 years / F	*Klebsiella pneumoniae*	Hematogenous route
2025	15 years / M	*Streptococcus anginosus*	No evident pathway

The management of hepatic abscesses typically relies on a combined approach involving both drainage and antibiotic therapy. When indicated, drainage can be performed percutaneously under ultrasound or CT guidance and is considered the standard method for abscesses of significant size (>3–5 cm), those not responsive to medical treatment, or when clinical improvement is insufficient [5,6]. In complex or multiloculated cases, surgical intervention may be warranted [5]. Given that hepatic abscesses are frequently polymicrobial, empirical antibiotic therapy should provide broad-spectrum coverage targeting the most common causative organisms, including Enterobacteriaceae, anaerobes, and staphylococci. First-line therapy often includes a beta-lactam/beta-lactamase inhibitor combination or a third-generation cephalosporin in association with metronidazole to ensure adequate anaerobic coverage. Antibiotic treatment should then be adjusted based on microbiological culture and sensitivity results. The duration of therapy varies according to clinical response and radiological resolution of the abscess but typically ranges from 4 to 6 weeks, with a switch to oral therapy when the patient's clinical condition permits. Clinical and radiological follow-up is essential to confirm complete abscess resolution and to allow for repeat drainage if required [6].

Conclusion

This case underlines the importance of early abdominal imaging in any SCD patient presenting with unexplained fever or abdominal pain. Although rare, hepatic abscesses should be considered in the differential diagnosis, particularly given the potential involvement of opportunistic pathogens such as Streptococcus anginosus.

Declarations

Acknowledgements: Not applicable.

Ethics approval and consent to participate: This case report is in accordance with the journal's ethics and integrity policies.

Competing interests: The authors declare that they have no competing interests.

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