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Case report

Arcanobacterium haemolyticum as an Unusual Etiology of Hepatic Abscesses: A Case Report

María Paula Herrera-Centeno,[1*] Karol Valeria García-González,[2] Óscar M. Muñoz.[3]

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[1] General Practitioner and Surgeon, Internal Medicine Resident, Department of Internal Medicine, Pontificia Universidad Javeriana, Hospital Universitario San Ignacio. Bogotá, Colombia.
[2] Medical Student, Pontificia Universidad Javeriana, School of Medicine. Bogotá, Colombia.
[3] Internist, Professor in the Department of Internal Medicine. PhD in Epidemiology, Department of Internal Medicine, Pontificia Universidad Javeriana, Hospital Universitario San Ignacio. Bogotá, Colombia.

***Correspondence:** María Paula Herrera-Centeno.
mp-herrera@javeriana.edu.co

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Abstract

Introduction: *Arcanobacterium haemolyticum* is an infrequent Gram-positive, catalase-negative bacillus, typically associated with pharyngitis and mild cutaneous infections. Systemic infections caused by this microorganism are uncommon, and hepatic abscess formation is particularly rare. **Case report:** We present the case of a 30-year-old man with human immunodeficiency virus (HIV) infection on antiretroviral therapy who presented with diarrhea, fever, and abdominal pain. During hospitalization, jaundice, hepatomegaly, and cholestasis were observed. Magnetic resonance cholangiography revealed hepatic abscesses and portal thrombophlebitis. Blood cultures were positive for *A. haemolyticum*, with no additional isolates. Given the inability to perform drainage, antibiotic therapy was administered for six weeks, resulting in an adequate clinical response and progressive resolution of the lesions. **Conclusions:** *A. haemolyticum*, typically considered a low-virulence pathogen, can cause severe invasive infections, including hepatic abscesses and pylephlebitis, and should be considered a potential etiologic agent in immunocompromised populations.

Keywords

Arcanobacterium; hepatic abscess; venous thrombosis; HIV infections; sepsis.

INTRODUCTION

Hepatic abscess is an infectious inflammatory space-occupying lesion of the liver[(1)]. The bacteria most frequently associated with the development of hepatic abscesses are gram-negative bacilli, enterococci, and *Streptococcus spp*[(1,2)]. However, over recent decades, less common pathogens have been described, posing diagnostic and therapeutic challenges, particularly in immunosuppressed patients, individuals with diabetes, or those with alcoholism[(2)]. Among these pathogens, *Arcanobacterium haemolyticum* is a catalase-negative gram-positive bacillus usually associated with pharyngitis and cutaneous infections[(3)], especially in young adults. Clinical presentations of *A. haemolyticum* involving deep or visceral infections are unusual, while endocarditis, osteomyelitis, meningitis, and pneumonia are more commonly reported[(3)]. We present the case of a patient with multiple hepatic abscesses secondary to *A. haemolyticum* infection, highlighting the importance of recognizing this atypical pathogen in hepatobiliary infections among immunosuppressed patients.

CLINICAL CASE

A 30-year-old male patient presented with an eight-day history of frequent, low-volume diarrheal bowel movements with mucus and no blood, associated with nausea and vomiting of food contents, as well as subjective fever, chills, and poor oral intake tolerance. He had a history of human immunodeficiency virus (HIV) infection diagnosed three years earlier, with good adherence to treatment with tenofovir/emtricitabine/efavirenz (TDF/FTC/EFV), and a most recent CD4 count of 230 cells/μL. Review of systems revealed odynophagia at the onset of symptoms. On admission, the patient appeared in fair general condition and was febrile, hypotensive, jaundiced, and had diffuse abdominal pain with voluntary guarding and hepatomegaly. Initial laboratory tests showed severe thrombocytopenia and hyperlactatemia. Gastrointestinal sepsis was suspected; therefore, fluid resuscitation was initiated and antibiotic therapy with ceftriaxone 2 g IV daily was started for a planned five-day course. A gastrointestinal FilmArray panel was negative.

Given the findings of jaundice and hepatomegaly, a liver profile was ordered, showing aspartate aminotransferase (AST): 26, alanine aminotransferase (ALT): 32, alkaline phosphatase (ALP): 350, total bilirubin (TB): 1.66, direct bilirubin (DB): 1.16, and indirect bilirubin (IB): 0.50, consistent with a cholestatic pattern. Abdominal computed tomography demonstrated haustral dilatation, pneumobilia, and intrahepatic biliary duct dilatation (which had not been documented on the initial hepatobiliary ultrasound), as well as a 9 mm cecal appendix and portal pylephlebitis, probably secondary to septic emboli. Treatment with piperacillin-tazobactam 4.5 g IV every 6 hours was initiated, and the patient underwent exploratory laparotomy because of suspected appendicitis; however, no abnormalities were identified.

Due to persistent thrombocytopenia and splenomegaly, a portal Doppler ultrasound was requested to evaluate for portal hypertension. The study revealed acute thrombosis of the already known left branch of the portal vein, and full anticoagulation was initiated after surgery.

As no clear infectious focus had been identified as the source of the septic pylephlebitis, and given the presence of pneumobilia and intrahepatic biliary duct dilatation, magnetic resonance cholangiography was performed (**Figure 1**). The study showed an abnormal hepatic parenchymal signal with an image suggestive of a developing abscess in the periphery of hepatic segments VI and VII. In addition, blood culture results ultimately documented bacteremia caused by *A. haemolyticum*. Therefore, the condition was interpreted as pharyngeal (more common) and gastrointestinal (colitis) colonization with secondary bacteremia in the context of an immunosuppressed patient, ultimately leading to abscess formation and septic thrombophlebitis.

A six-week course of antibiotic therapy with ampicillin/sulbactam 3 g IV every six hours was completed. Follow-up abdominal computed tomography performed at three weeks documented enlargement of one of the hepatic collections, reaching a volume of 54 mL (**Figure 2**). However, imaging characteristics suggested that the abscess was organized, making drainage unfeasible. Given the favorable clinical course, resolution of abdominal pain, disappearance of diarrheal bowel movements, improvement in liver function tests (**Table 1**), and sustained reduction in the inflammatory response, antibiotic therapy was continued. A subsequent follow-up computed tomography scan performed two weeks later (**Figure 3**) demonstrated a

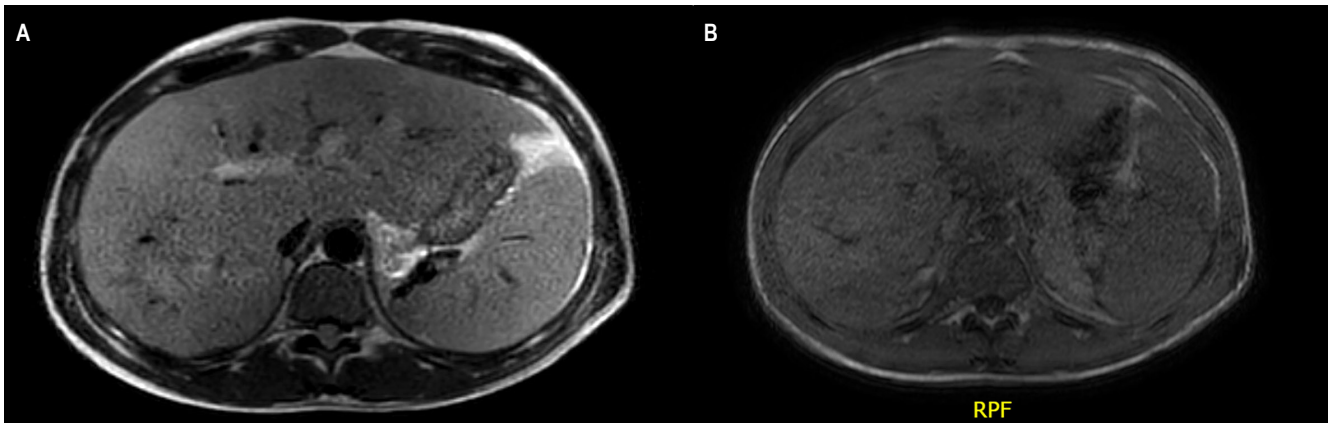

Figure 1. Magnetic resonance cholangiography. **A.** Peripheral focal lesion in hepatic segment VII, suggestive of an organizing abscess. **B.** Poorly defined peripheral hepatic lesions in segments VI–VII. Author's file.

reduction in the size of the hepatic abscesses, and follow-up blood cultures were negative. A total of six weeks of antibiotic treatment was completed. Outpatient follow-up at three months demonstrated complete resolution of symptoms.

Table 1. Evolution of Laboratory Parameters During Follow-up

Laboratory Tests	Admission	Day 3	Week 3	Week 6
Leukocytes (/µL)	9,800	17,330	4,600	5,000
Hemoglobin (g/dL)	13.5	11.9	10.0	12.3
Platelets (/µL)	42,000	29,000	345,000	400,000
AST (U/L)	25	127	26	17
ALT (U/L)	26	59	32	15
ALP (U/L)	185	329	350	130
Total bilirubin (mg/dL)	5.59	8.18	4.88	1.25
Creatinine (mg/dL)	1.54	1.12	0.88	0.98
Lactic acid (mmol/L)	3.2	2.2	1.3	0.8

ALT: alanine aminotransferase; AST: aspartate aminotransferase; ALP: alkaline phosphatase. Author's own research.

DISCUSSION

We present the case of a systemic *A. haemolyticum* infection in a patient with HIV infection, manifesting as sepsis accompanied by septic pylephlebitis of the portal vein and the formation of hepatic abscesses. The coexistence of this microorganism in the bloodstream, together with the observed thrombotic and hepatic complications, represents an unusual presentation distinct from the more commonly reported clinical picture of self-limited pharyngeal infections in young adults or mild cutaneous infections[2–4]. This clinical presentation may be associated with the patient's immunocompromised status, which predisposed him to invasive infection[4]. The literature reports few cases of hepatic abscesses caused by *A. haemolyticum*[2,3], suggesting an uncommon tropism for hepatic parenchyma.

In this case, the most likely pathophysiological sequence involved bacteremia secondary to pharyngeal or gastrointestinal colonization, followed by hepatic seeding and subsequent formation of multiple abscesses. The local inflammatory process and endothelial infection likely favored the development of pylephlebitis and portal vein thrombosis, findings that support the septic progression and organ dysfunction observed.

Early microbiological diagnosis enabled targeted antimicrobial management. Identification of *A. haemolyticum* in blood cultures, together with its susceptibility profile to penicillins(6–8), prompted optimization of antibiotic therapy to ampicillin/sulbactam, resulting in an adequate clinical and radiological response. This finding highlights the importance of maintaining active microbiological surveillance when unusual gram-positive cocci or bacilli are identified in immunosuppressed patients, avoiding underestimation of these organisms as contaminants.

Regarding management, hepatic abscesses associated with pylephlebitis usually require prolonged antibiotic therapy (four to six weeks)[5,9], with drainage when feasible. In the present case, radiological follow-up demonstrated

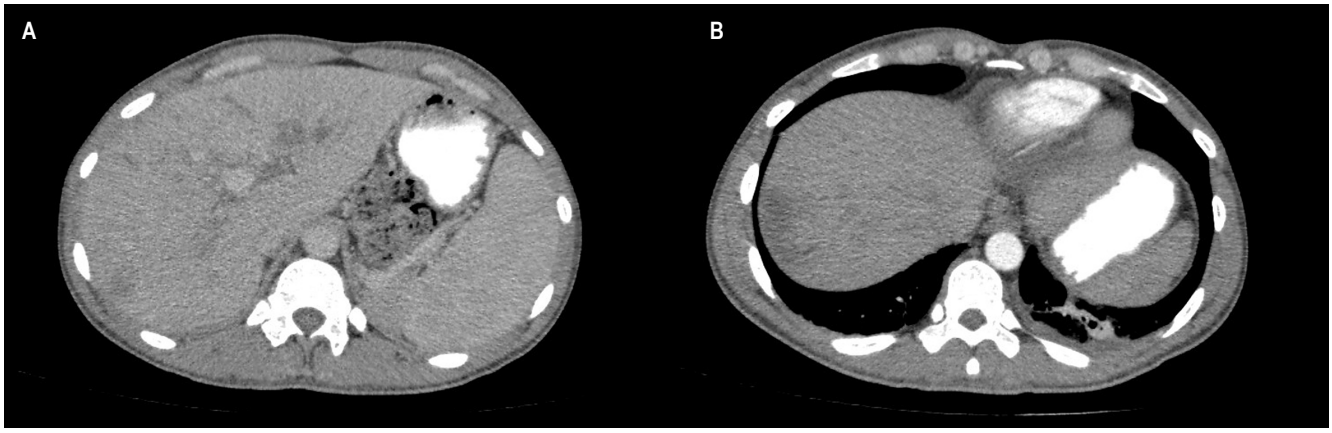

Figure 2. Contrast-enhanced computed tomography of the abdomen and pelvis. **A.** Peripheral caudal collection in segment VII measuring 19 × 19 × 16 mm (L × AP × T), consistent with a hepatic abscess, with an approximate volume of 8 mL. **B.** Additional collection in segment VII, located more superiorly and with poorly defined borders, measuring 57 × 59 × 31 mm, with an approximate volume of 54 mL. Thrombosis of the left portal vein and right portal branches. Heterogeneous enhancement of the hepatic parenchyma, predominantly during the arterial phase. Splenomegaly. Author's file.

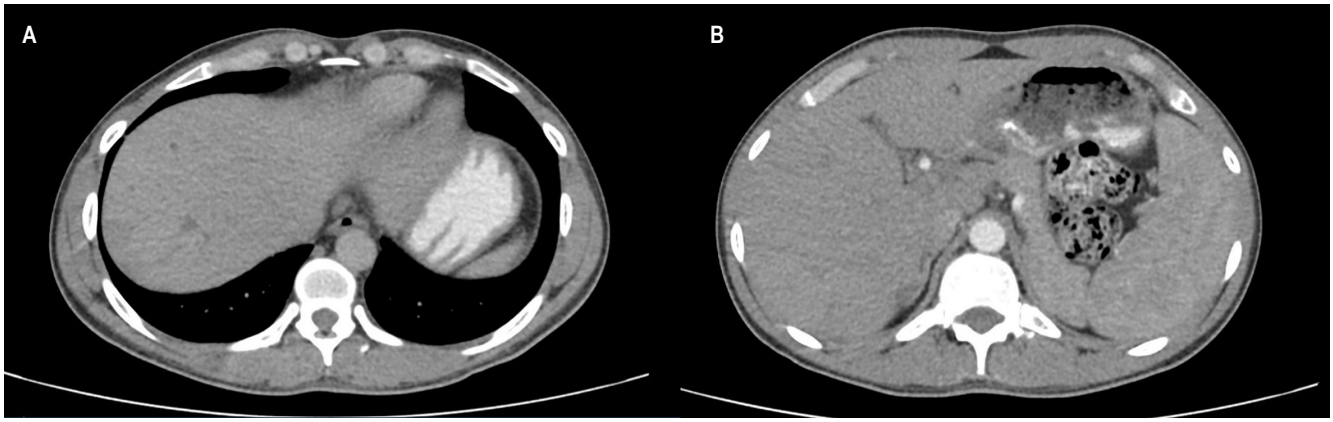

Figure 3. Follow-up computed tomography of the abdomen and pelvis. **A.** Irregular subcapsular hypodense lesion in segment VII measuring 23 × 18 mm. **B.** Additional rounded hypodense lesion in segment VII measuring approximately 15 × 18 mm. Findings were primarily attributable to hepatic abscesses, with a reduction in size compared with the previous study. Author's file.

apparent initial enlargement followed by regression, probably reflecting the liquefaction and organization process of the abscess. Since the lesions were not amenable to percutaneous drainage, exclusive medical management was chosen, with favorable clinical evolution and progressive reduction of the lesions during follow-up.

This case provides additional evidence of the capacity of *A. haemolyticum* to cause severe invasive infections outside the respiratory tract, particularly in immunocompromised hosts. It also emphasizes the need to consider this pathogen in the differential diagnosis of bacteremia caused by unusual gram-positive organisms and of hepatic infections without an evident biliary source.

CONCLUSION

This case demonstrates that *A. haemolyticum*, typically considered a low-virulence pathogen, can cause severe invasive infections, including hepatic abscesses and pylephlebitis, underscoring the importance of early microbiological identification and targeted antimicrobial management in immunocompromised populations.

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