

Acute Chest Syndrome and Critical Illness-Associated Cerebral Microbleeds in Sickle Cell Disease: A Case Report

Síndrome Torácico Agudo e Microhemorragias Cerebrais Associadas a Doença Crítica em Doente com Doença Falciforme: Relato de Caso

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ABSTRACT

A 33-year-old woman with sickle cell disease presented with severe lumbar pain. Initially stable, she experienced rapid deterioration within the first 24 hours, developing respiratory failure, fever, and impaired consciousness. Chest imaging revealed extensive bilateral infiltrates, and laboratory tests showed severe anemia, thrombocytopenia, and elevated inflammatory markers. She required mechanical ventilation, exchange transfusions, and antibiotics. Parvovirus B19 infection was confirmed. After nine days, she improved, but neurological recovery was delayed. Magnetic resonance imaging revealed cerebral microhemorrhages consistent with critical illness-associated cerebral microbleeds. She was discharged after a 34-day hospitalization. This case highlights two severe and potentially life-threatening complications of sickle cell disease, namely acute chest syndrome and critical illness-associated cerebral microbleeds, underscoring the importance of early recognition and aggressive management.

Keywords: Acute Chest Syndrome; Anemia, Sickle Cell/complications; Cerebral Hemorrhage; Parvoviridae Infections; Parvovirus B19, Human

RESUMO

Uma mulher de 33 anos com anemia falciforme apresentou-se com dor lombar intensa. Inicialmente estável, evoluiu com rápida deterioração nas primeiras 24 horas, desenvolvendo insuficiência respiratória, febre e alteração do estado de consciência. Imagiologicamente, apresentava infiltrados pulmonares bilaterais extensos e os exames laboratoriais evidenciaram anemia grave, trombocitopenia e elevação dos marcadores inflamatórios. Necessitou de ventilação mecânica, exsanguineotransfusões e antibioterapia. Confirmou-se infeção por parvovírus B19. Após nove dias, apresentou melhoria clínica, mas mantinha disfunção neurológica. A ressonância magnética cerebral revelou microhemorragias cerebrais compatíveis com microhemorragias associadas a doença crítica. Teve alta após 34 dias de internamento. Este caso destaca duas complicações graves e potencialmente fatais da anemia falciforme, a síndrome torácica aguda e microhemorragias cerebrais associadas a doença crítica, sublinhando a importância do reconhecimento precoce e da abordagem terapêutica agressiva.

Palavras-chave: Anemia Falciforme/complicações; Hemorragia Cerebral; Infecções por Parvoviridae; Parvovírus B19 Humano; Síndrome Torácica Aguda

INTRODUCTION

Sickle cell disease (SCD) is a genetic hematologic disorder characterized by the presence of hemoglobin S, leading to sickle-shaped red blood cells and multiple complications.¹ Among these, acute chest syndrome (ACS) is one of the most frequent and life-threatening, accounting for approximately 25% of SCD-related deaths.² Acute chest syndrome is defined by new radiographic pulmonary infiltrates with fever and/or respiratory symptoms.¹ In adults, about 78% of ACS episodes result from a vaso-occlusive crisis which can lead to bone marrow infarction and the release of fat emboli into the pulmonary circulation.^{3,4} Mortality rises with age, from 4.3% to 9% in adults.^{5,6}

Additionally, critical illness-associated cerebral microbleeds (CICM) is a rare and newly recognized complication in critically ill patients, often with acute respiratory failure. It is characterized by microbleeds primarily in the subcortical white matter, corpus callosum, and less commonly in the cortex, deep and periventricular white matter, basal ganglia,

and thalami. Pathogenesis remains unclear, but hypoxemia may contribute, as similar microbleeds have been observed in high-altitude exposure. Despite few reported cases, CICM carries high mortality and significant long-term neurological sequelae.⁷

CASE REPORT

A 33-year-old woman with SCD (HbSC) presented to the emergency department with severe lumbar pain refractory to morphine. She was hemodynamically stable, afebrile, with an oxygen saturation of 100% on room air, and a Glasgow Coma Scale (GCS) of 15. Laboratory results (Table 1) showed anemia, reticulopenia, low haptoglobin, elevated LDH, and HbS levels above 30%. Chest radiography showed no infiltrates. Lumbar pain was non-focal, with no skin or joint complaints. She denied recent contact with children or immunocompromised individuals possibly infected with parvovirus B19. Given a suspected vaso-occlusive

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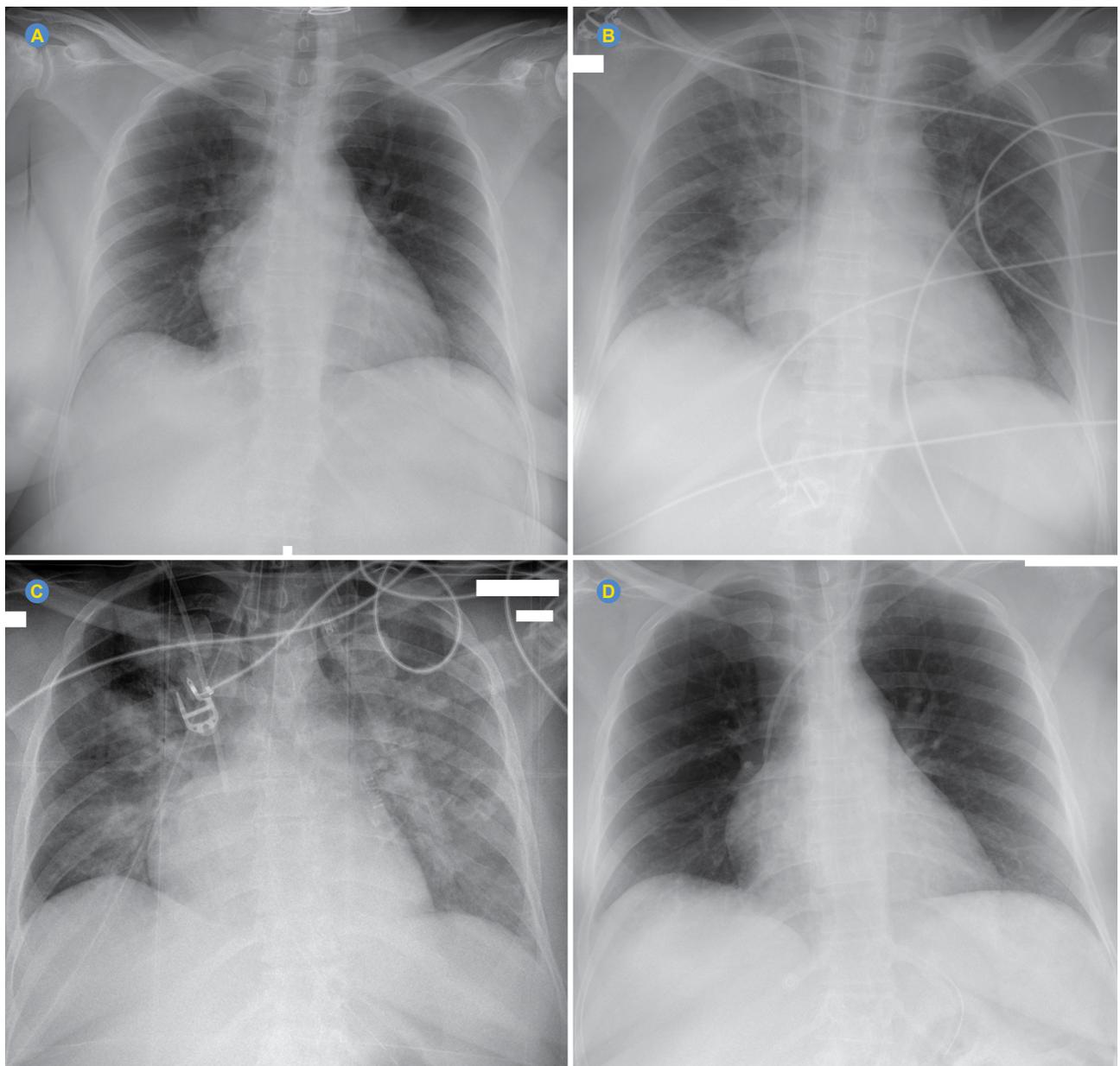
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Table 1 – Laboratory results at admission and after 24 hours

Parameter	Reference range	On admission	24 hours later
Hemoglobin (g/dL)	12 - 16	8.6	4.3
Hemoglobin S (%)	--	44	--
Reticulocytes ($\times 10^6/\mu\text{L}$, %)	0.8 - 4	0.06 (1.86%)	0.0036 (0.22%)
Haptoglobin (mg/dL)	30 - 200	< 10	< 10
LDH (U/L)	135 - 214	367	938
Total bilirubin (mg/dL)	0.1 - 1.1	0.73	0.91
Leucocytes ($\times 10^3/\mu\text{L}$)	3.6 - 11	8.3	12.24
Platelets ($\times 10^3/\mu\text{L}$)	150 - 450	241	78
CRP (mg/dL)	< 0.5	0.15	22

**Figure 1** – Chest radiography images (A) at admission, (B) 24 hours after admission demonstrating bilateral opacities, (C) worsening after 48 hours, and (D) normal at discharge

crisis, she was admitted for pain management with intravenous lidocaine and tramadol, alongside intensive hydration.

Within 24 hours, her condition deteriorated significantly, presenting with an impaired consciousness, fever, productive cough with gold sputum and respiratory failure ($\text{PaO}_2/\text{FiO}_2$ ratio of 118). Chest radiography showed extensive bilateral pulmonary infiltrates (Fig. 1). Repeat laboratory tests (Table 1) revealed a marked hemoglobin drop, thrombocytopenia and elevated inflammatory markers. Chest computed tomography (CT) showed extensive consolidations in the lower lobes, contiguous posterior consolidations in the middle and upper lobes, scattered ground-glass opacities, and signs of right humeral head necrosis, with no signs of pulmonary embolism (Fig. 2). Echocardiography revealed mild signs of right ventricular dysfunction suggestive of acute pulmonary hypertension. The patient was intubated for mechanical ventilation and treated with oxygen, ceftriaxone and azithromycin. She performed two exchange transfusions (targeting $\text{HbS} < 15\%$), which significantly reduced HbS from 44% at admission to 12%, leading to an immediate benefit on microcirculation and regression of hypoxia. Parvovirus B19 infection was confirmed (positive IgM and

viral load), while blood cultures and bronchial lavage (BL) cultures and a multiplex polymerase chain reaction panel for pneumonia from BL, were negative.

Clinical improvement was observed after nine days, allowing sedation withdrawal and extubation. However, the patient remained minimally responsive, opening her eyes spontaneously, localizing pain but not vocalizing (GCS 10), responding to voice but not following commands. Brain CT and electroencephalogram were unremarkable. Magnetic resonance imaging (MRI) revealed numerous cerebral microhemorrhages on T2*, symmetrically involving the juxtacortical white matter, corpus callosum, internal and external capsules, and cerebellar peduncles, without restricted diffusion (Fig. 3). A lumbar puncture was not performed because the clinical presentation was not suggestive of central nervous system infection—there were no meningeal signs, seizures, or sudden focal neurological deficits. In addition, non-invasive diagnostic tools were prioritized, and the MRI findings adequately explained the neurological status.

Three days after extubation, the patient was transferred to the general ward. Throughout the 34-day hospitalization, she exhibited a gradual recovery of consciousness.

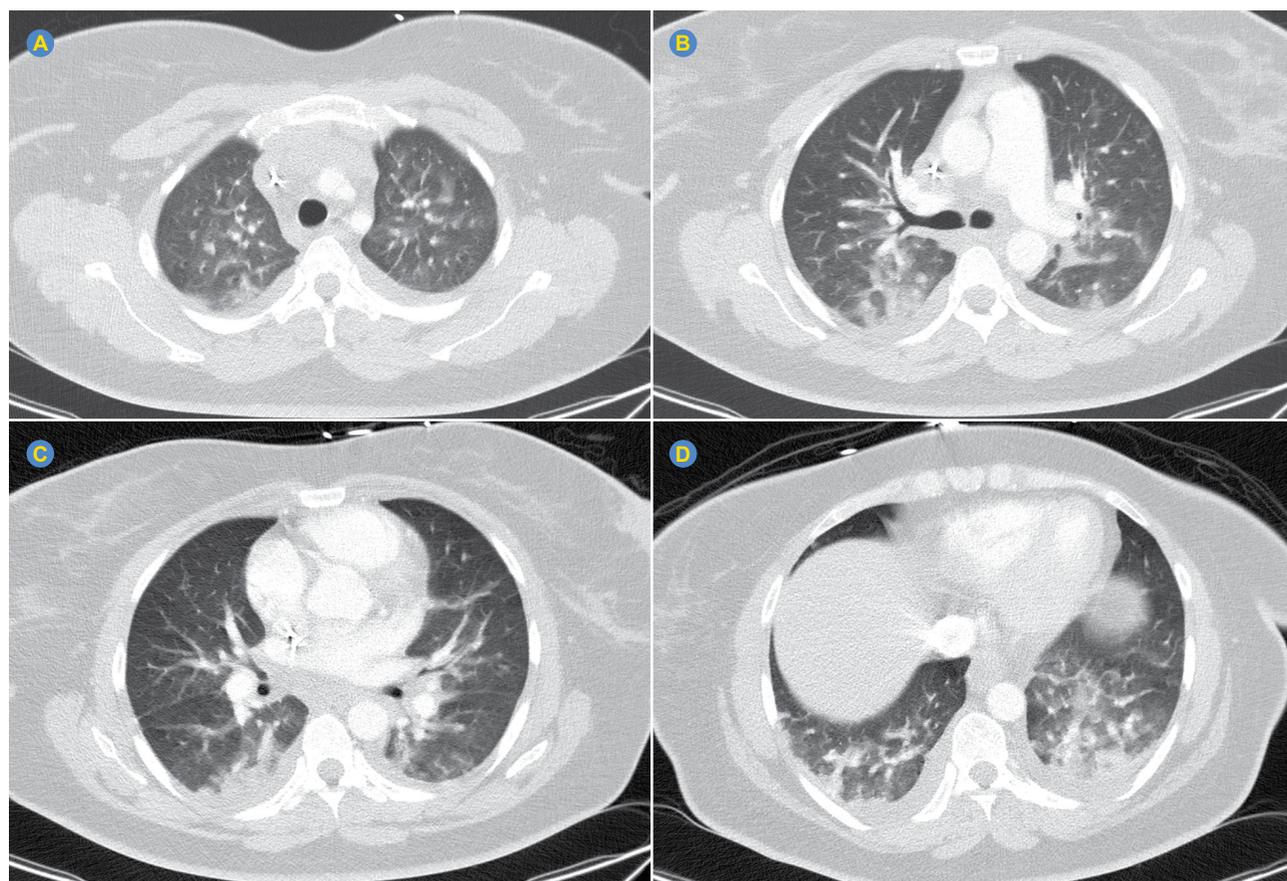


Figure 2 – Chest CT image obtained with the lung window setting shows extensive consolidations in the lower lobes, contiguous posterior consolidations in the middle and upper lobes and scattered ground-glass opacities

logic deficit in 71% of survivors.¹⁰

This case underscores two severe SCD complications: ACS and CICM, both diagnostic and therapeutic challenges, especially when concomitant. Early recognition, aggressive multidisciplinary management, and prompt intervention are crucial to improving patient outcomes. While CICM remains an underdiagnosed phenomenon in critically ill patients, requiring further research to clarify its pathogenesis and long-term consequences, increasing clinician awareness may facilitate earlier detection and better care strategies.

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AUTHOR CONTRIBUTIONS

MJM: Study design, data collection, writing and critical review of the manuscript.

EC: Data collection, critical review of the manuscript.

MAC, SR: Critical review of the manuscript.

RM: Image acquisition, critical review of the manuscript.

All authors approved the final version to be published.

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PROTECTION OF HUMANS AND ANIMALS

The authors declare that the procedures were followed according to the regulations established by the Clinical Research and Ethics Committee and to the Helsinki Declaration of the World Medical Association updated in October 2024.

DATA CONFIDENTIALITY

The authors declare having followed the protocols in use at their working center regarding patients' data publication.

PATIENT CONSENT

Obtained.

CONFLICTS OF INTEREST

The authors have no conflicts of interest to declare.

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