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CASE REPORT

An Unusual Complication of Systemic Lupus Erythematosus(SLE) Presenting with Features of Acute Heart Failure

Arya R.P. Anand

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ABSTRACT

Background: Diffuse alveolar haemorrhage (DAH) is a rare but potentially fatal pulmonary complication of SLE.

Aims: To describe an unusual presentation of DAH mimicking acute heart failure.

Settings and Design: Emergency department case presentation.

Methods and Material: Clinical assessment, laboratory testing, imaging (CXR, CT), echocardiography, and renal biopsy.

Results: A 34-year-old woman with longstanding SLE presented with acute dyspnoea and hypoxia. CT chest revealed diffuse alveolar haemorrhage. She responded to corticosteroids and supportive therapy.

Conclusions: DAH can mimic acute heart failure in SLE and requires rapid recognition for survival.

KEYWORDS

• Systemic lupus erythematosus • Diffuse alveolar haemorrhage • Heart failure mimicry • Corticosteroids

INTRODUCTION

Systemic lupus erythematosus (SLE) is an autoimmune condition affecting body systems, where immune complexes build up and multiple organs can become involved. Although lung problems happen often, diffuse alveolar haemorrhage (DAH) occurs rarely yet it's one of the most dangerous complication. Instead of typical signs like sudden breathing difficulty, dropping haemoglobin levels, and

AUTHOR'S AFFILIATION:

Department of Emergency Medicine, Medical College Thiruvananthapuram, Thiruvananthapuram, Kerala, India.

CORRESPONDING AUTHOR:

Arya R.P. Anand, Department of Emergency Medicine, Medical College Thiruvananthapuram, Thiruvananthapuram, Kerala, India.

E-mail: aryaanand317@gmail.com

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widespread shadows on imaging, symptoms sometimes mimic those of fluid buildup in lungs due to heart issues - which may confuse diagnosis at first. Because timing matters greatly when treating this complication, spotting it early improves survival chances significantly.

METHODS

Diagnostic tests covered blood counts, basic metabolic profile, signs of inflammation like CRP or ESR, complement activity, clotting function, chest X-ray, lung CT scans, heart ultrasound, urine analysis - alongside kidney tissue sampling with electron imaging if needed. Treatment aligned with hospital guidelines as well as ethical standards from the updated Helsinki Declaration dated 2013.

RESULTS

A 34-year-old female, diagnosed with SLE two decades ago along with ITP and a past brain bleed, came to the ER due to increasing breathlessness over two days - now worse within one day - plus trouble breathing while lying flat, belly bloating, swollen legs on both sides, and puffy face. Her pulse was fast at 112 beats per minute upon admission; blood pressure stayed normal at 120/80 mmHg despite low oxygen levels (only 77% without extra O_2). She looked pale during assessment, had widespread fluid buildup throughout the body, and crackling sounds were heard at the base of both lungs.

Lab tests showed low red blood cell count with normal-sized cells (Hb 5.6 g/dL), alongside a drop in platelets (50 × 10^9/L). White blood cell count was high at 13 × 10^9/L, while protein levels in blood were reduced (albumin 2.4 g/dL). Inflammation signs included CRP rising to 100 mg/dL, ESR reaching 40 mm/hr, plus LDH spiking up to 1286 U/L. C3 and C4 complement proteins fell slightly - measured at 68 and 18 mg/dL respectively. Kidney values stayed stable: BUN stood at 38 mg/dL, creatinine remained near baseline (0.7 mg/dL). A urine check pointed to moderate protein leakage (2+) together with many red blood cells. Chest X-ray displayed patchy white areas on both sides. Echo test found normal heart pumping function - about 65% - along with slight leakage in the tricuspid valve while showing a slightly oval-shaped left chamber.

The patient presented with coughing up blood along with blood in urine at hospital entry. A contrast-based chest CT scan showed solid areas around bronchi and vessels, plus hazy patches nearby, affecting every lobe on both sides - signs pointing to widespread lung bleeding. Possible conditions explored were diffuse alveolar hemorrhage, anemia-linked fluid buildup in lungs, blocked lung arteries, or anti-GBM antibody disease (also known as Goodpasture's). Blood tests for immune markers and clotting function came back normal. Renal biopsy by day 5 revealed focal lupus nephritis with <50% involvement; findings from immunofluorescence and electron microscopy lacked anti-glomerular basement membrane IgG, ruling out anti-GBM disease. The peripheral blood smear indicated normochromic, normocytic anemia along with slight thrombocytopenia.

The patient got non-invasive breathing support, whole blood transfusions, platelet infusions, followed by short bursts of low-dose steroids, together with general supportive measures. Antibiotics weren't given right away because there were no signs of infection. In the next few days, her oxygen levels and overall condition got better, blood values stopped fluctuating, while coughing up blood disappeared completely. Later, she moved from ICU to a regular ward then went home on day 15 taking oral prednisolone combined with mycophenolate mofetil (MMF), continuing treatment for lupus-related kidney disease.

Radiologic findings along with clinical signs confirmed DAH caused acute respiratory failure. Left ventricular function remained normal, which made heart-related edema unlikely. No anti-GBM antibodies were found in kidney tissue, ruling out Goodpasture's syndrome. Improvement after steroid treatment plus blood transfusions supported the diagnosis, resulting in full recovery - mechanical breathing support or cyclophosphamide weren't required.

DISCUSSION

DAH in SLE occurs rarely, yet it often leads to serious health issues or death.[1,2] A strong level of alertness is needed if a patient shows fresh lung shadows on imaging along with a sudden fall in haemoglobin levels - especially

without coughing up blood. Because DAH may resemble acute heart failure through shared features like breathlessness, difficulty breathing while lying down, abnormal lung sounds, and similar chest X-ray findings, distinguishing between them requires combining information from symptoms, lab tests, and echo results thoughtfully.

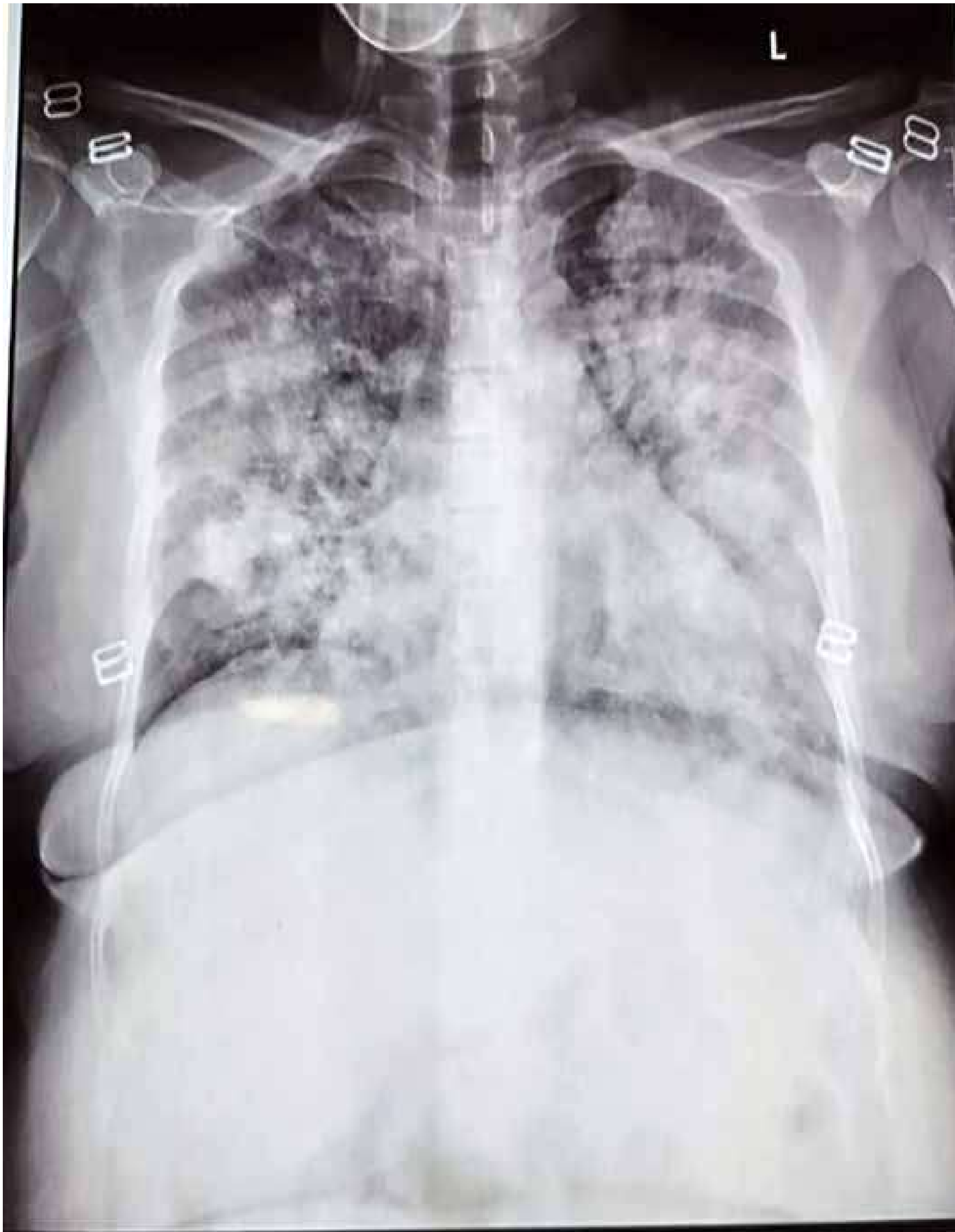

Figure 1: Chest X-ray showing bilateral dense heterogeneous infiltrates

Chest CT showing widespread ground-glass areas along with denser patches around airways points to DAH. Kidney issues commonly appear in lupus patients who have DAH; tissue sampling helps rule out conditions like anti-GBM disease. Treatment mainly relies on strong steroid doses; extra therapies - such as cyclophosphamide or rituximab - alongside plasma exchange or ECMO might be needed when symptoms are severe or don't respond. Spotting the condition early and starting care quickly leads to better results, much like seen here.

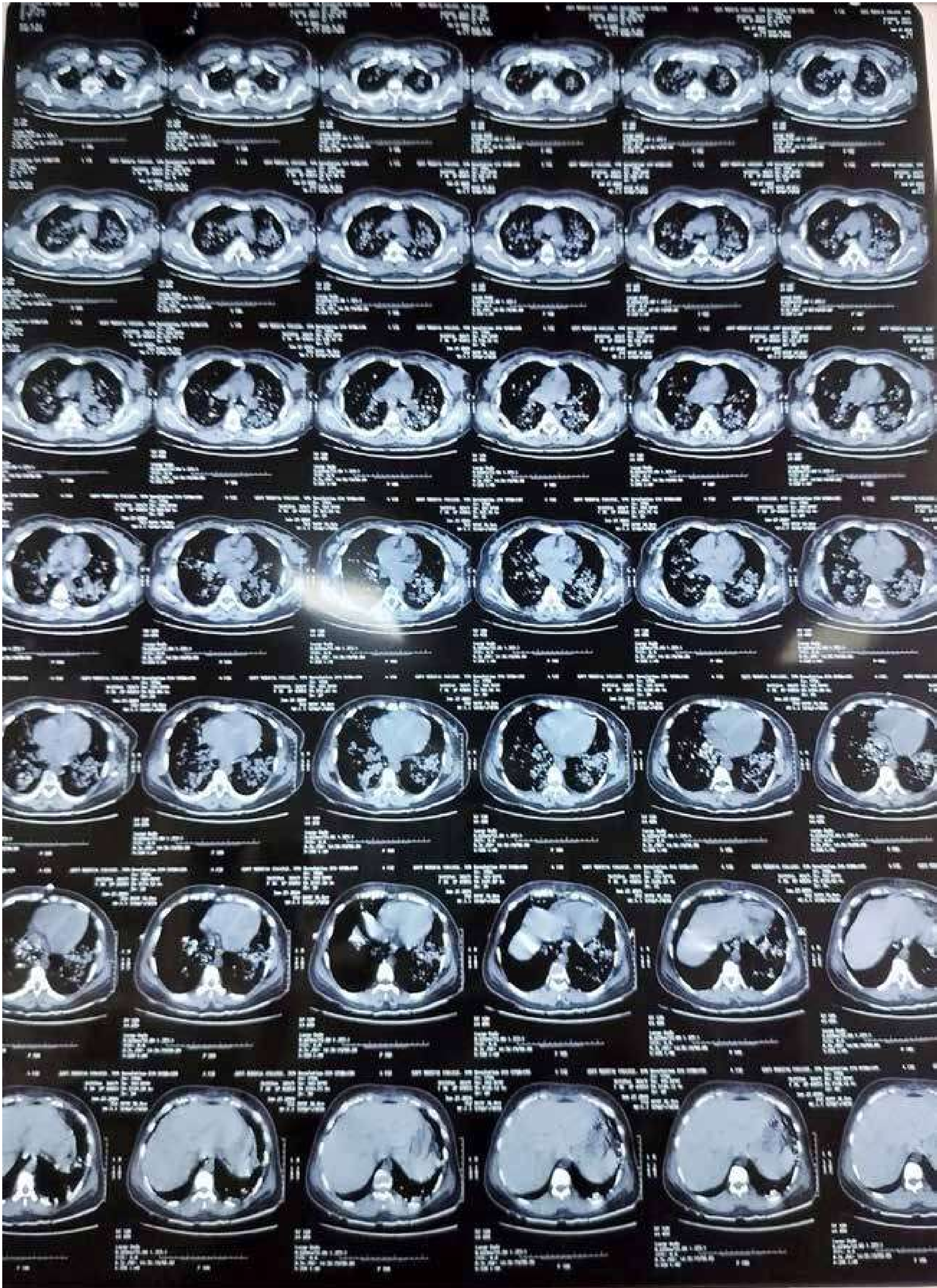

Figure 2: CT Chest demonstrating peribronchovascular consolidation with ground-glass opacities across all lobes

Statistical Analysis: Not applicable

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