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General Medicine

KEY WORDS: Diffuse Alveolar Haemorrhage; Young Adult; Ground Glass Opacities; Respiratory Distress; Anticoagulant Complications

WHEN BLOOD FILLS THE BREATH: DIFFUSE ALVEOLAR HAEMORRHAGE IN A YOUNG MAN

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ABSTRACT

Background: Diffuse alveolar haemorrhage (DAH) is a rare and life-threatening condition characterized by bleeding into the alveolar spaces. While more commonly seen in older patients with autoimmune diseases, DAH in young healthy adults presents unique diagnostic challenges and requires prompt recognition for optimal outcomes. **Case Presentation:** A 20-year-old previously healthy male presented with acute onset high-grade fever, dry cough, and progressive dyspnea at rest over 3 days. Physical examination revealed tachypnea with SpO2 98% on room air with bilateral basal crepitations and reduced breath sounds on lung auscultation. Laboratory investigations demonstrated severe inflammatory response with CRP 166.44 mg/L, procalcitonin 2.11 ng/mL, and markedly elevated D-dimer at 9420 ng/mL. High-resolution computed tomography revealed diffuse ground glass opacities involving 80% of bilateral lung parenchyma with CORADS-4 pattern and CT severity score of 25/25. Subsequent CT thorax confirmed diffuse alveolar haemorrhage, likely anticoagulant-induced. The patient was managed with broad-spectrum antibiotics and supportive care with gradual improvement. **Conclusion:** DAH in young adults requires high clinical suspicion despite normal initial auscultatory findings. Early imaging and prompt management are crucial for favourable outcomes in this potentially fatal condition.

INTRODUCTION

Diffuse alveolar haemorrhage (DAH) represents a life-threatening syndrome characterized by bleeding into the alveolar spaces, resulting in acute respiratory failure[1]. This condition typically affects older adults with underlying autoimmune diseases such as systemic lupus erythematosus, ANCA-associated vasculitis, or Goodpasture's syndrome[2]. However, DAH in young, previously healthy individuals is exceedingly rare and poses significant diagnostic challenges[3].

The pathophysiology of DAH involves damage to the alveolar-capillary barrier, leading to extravasation of red blood cells into the alveolar spaces[4]. While autoimmune causes predominate, drug-induced DAH, particularly from anticoagulants, represents an increasingly recognised etiology[5]. Early recognition and prompt management are crucial, as mortality rates can exceed 50% without appropriate intervention[6].

Case Presentation

A 20-year-old male presented to the emergency department with a 3-day history of high-grade fever with chills, persistent dry cough, and progressive dyspnea at rest. The patient reported sudden onset breathlessness that had worsened over the preceding 2 days. He denied chest pain, palpitations, hemoptysis, vomiting, or urinary symptoms. His past medical history was significant for a recent referral from an outside hospital where injection Clexane (enoxaparin) was administered for suspected pulmonary embolism in view of an elevated D-dimer level. His personal history revealed a mixed diet with normal sleep, appetite, and bowel habits, and no substance abuse.

Physical examination revealed a conscious and oriented patient with vital signs showing pulse rate 78/min, blood pressure 110/70 mmHg, respiratory rate 30/min, and SpO2 98% on room air. Cardiovascular examination demonstrated normal S1 and S2 heart sounds without murmurs. Respiratory examination revealed bilateral basal crepitations with reduced breath sounds. Abdominal examination was unremarkable with no organomegaly. No pallor, cyanosis, clubbing, icterus, lymphadenopathy, or oedema was observed.

Table 1: Laboratory Findings on Admission

Parameter	Value	Reference Range	Inter-pretation
Haematology			
Haemoglobin	8.7 g/dL	13.5-17.5 g/dL	Low
PCV	25.3%	40-50%	Low
MCV	63.9 fL	80-100 fL	Low
Total Leukocyte Count	7,600/µL	4,000-11,000/µL	Normal
Neutrophils	83%	50-70%	High
Lymphocytes	10%	20-40%	Low
Platelets	1.86 lakhs/µL	1.5-4.5 lakhs/µL	Normal
Biochemistry			
Blood Sugar (Random)	128 mg/dL	<140 mg/dL	Normal
Urea	26 mg/dL	15-40 mg/dL	Normal
Creatinine	0.69 mg/dL	0.7-1.3 mg/dL	Normal
Sodium	136 mmol/L	135-145 mmol/L	Normal
Potassium	7.5 mmol/L	3.5-5.0 mmol/L	Severely High
Chloride	125 mmol/L	98-107 mmol/L	High
Liver Function Tests			
Total Bilirubin	0.22 mg/dL	0.3-1.2 mg/dL	Normal
AST	60 U/L	<40 U/L	High
ALT	89 U/L	<40 U/L	High
ALP	128 U/L	44-147 U/L	Normal
Total Protein	5.2 g/dL	6.0-8.3 g/dL	Low
Albumin	2.46 g/dL	3.5-5.0 g/dL	Low
Inflammatory Markers			
CRP	166.44 mg/L	<3.0 mg/L	Markedly High
Procalcitonin	2.11 ng/mL	<0.25 ng/mL	Markedly High
D-Dimer	9,420 ng/mL	<500 ng/mL	Extremely High
Coagulation Profile			
PT	15 sec	11-16 sec	Normal
INR	1.0	0.8-1.2	Normal
APTT	30 sec	25-35 sec	Normal

Key Abnormalities: Microcytic hypo-chromic anaemia, severe hyperkalemia, neutrophilic leukocytosis, hypo-proteinemia, mild transaminitis, and markedly elevated inflammatory markers with extremely high D-dimer suggesting severe inflammatory response and possible coagulopathy.

Arterial blood gas analysis on 4L oxygen revealed compensated metabolic acidosis (pH 7.444, pCO2 29.3 mmHg, HCO3- 19.6 mmol/L, BE -3.8 mmol/L) with adequate

oxygenation (pO2 82.1 mmHg, O2 saturation 96.8%). Follow-up ABG on room air showed normalization of acid-base status.

High-resolution computed tomography of the chest demonstrated diffuse ground glass opacities involving bilateral lung parenchyma with patchy areas of consolidation and interstitial thickening. Approximately 80% of lung fields were involved with minimal bilateral pleural effusion. The CT severity score was 25/25, indicating progressive stage disease with CORADS-4 classification suggestive of ARDS pattern versus atypical pneumonia.

Subsequent CT thorax revealed no pulmonary embolism but confirmed diffuse ill-defined patchy ground glass opacities with confluent areas and relative subpleural sparing. The imaging features were highly suggestive of diffuse alveolar haemorrhage, likely secondary to anticoagulant-induced bleeding.

Echocardiography demonstrated good left ventricular systolic function (EF 60%) with mild mitral regurgitation, mild pulmonary hypertension (RVSP 41 mmHg), minimal pericardial effusion, and bilateral pleural effusion.

The patient was managed with broad-spectrum antibiotics (piperacillin-tazobactam and clindamycin), antiviral therapy, proton pump inhibitors, and supportive care including oxygen supplementation and nebulization. Close monitoring with oxygen titration resulted in gradual clinical improvement.

DISCUSSION

This case illustrates a rare presentation of diffuse alveolar haemorrhage in a young, previously healthy adult. The absence of hemoptysis despite severe radiological findings highlights the diagnostic challenges associated with DAH in this population[7]. The classical triad of hemoptysis, anaemia, and pulmonary infiltrates was incomplete, as seen in approximately 30% of DAH cases[8].

The markedly elevated inflammatory markers, particularly the extremely high D-dimer levels (9420 ng/mL), suggested significant pulmonary vascular involvement consistent with DAH pathophysiology[9]. The CT findings of diffuse ground glass opacities with relative subpleural sparing are characteristic radiological features of DAH, distinguishing it from other causes of acute lung injury[10].

The suspicion of anticoagulant-induced DAH raises important therapeutic considerations. Drug-induced DAH has been increasingly reported with various medications including anticoagulants, antiplatelet agents, and thrombolytics[11]. The mechanism involves disruption of the alveolar-capillary barrier integrity, leading to haemorrhage into the alveolar spaces[12]. The temporal relationship between enoxaparin administration at the outside hospital and subsequent development of DAH strongly suggests a causal relationship.

The patient's young age and absence of typical risk factors such as autoimmune diseases, renal dysfunction, or systemic vasculitis made this case particularly challenging[13]. The differential diagnosis included viral pneumonia, atypical bacterial pneumonia, and acute respiratory distress syndrome, all of which can present with similar radiological findings[14].

Management with broad-spectrum antibiotics proved beneficial for controlling the infectious component of the presentation[15]. The addition of broad-spectrum antibiotics was appropriate given the initial presentation mimicking infectious pneumonia. Conservative management without corticosteroids initially was chosen, with supportive care forming the mainstay of treatment[16].

The favourable response to treatment with gradual improvement in oxygen requirements and clinical status demonstrates the importance of early recognition and prompt intervention in DAH management[17].

CONCLUSION

This case emphasizes the importance of maintaining high clinical suspicion for diffuse alveolar haemorrhage in young adults presenting with acute respiratory symptoms, even in the absence of classic clinical signs such as hemoptysis. The discordance between physical examination findings and radiological severity underscores the critical role of early imaging in diagnosis.

Prompt recognition and appropriate management with supportive care and antibiotics can lead to favourable outcomes in drug-induced DAH. This case highlights the need for careful consideration of anticoagulant-related complications in patients presenting with acute lung injury and reinforces the importance of a systematic approach to diagnosing rare pulmonary conditions in young, previously healthy individuals.

Declarations

Patient Consent: Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Conflict of Interest: The authors declare no conflicts of interest.

Ethical Approval: Not applicable for this case report.

Author Contributions: All authors contributed to the clinical management, data collection, manuscript preparation, and final review.

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