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Case Report

Evaluation of massive hemoptysis with high suspicion for alveolar hemorrhage in the emergency room

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ABSTRACT

Diffuse alveolar hemorrhage is a serious life threatening condition that has to be promptly addressed during early period of presentation to emergency room. It is usually associated with some underlying causative factor. Early identification of causative factors will help in early initiation of definitive management, thereby reducing the mortality and improving the outcome. We report a case of 64-year-old male with hemoptysis who has been identified as alveolar hemorrhage in emergency room. We also evaluated its possible causative factor by clinical suspicion, laboratory investigations and contrast enhanced computed tomography imaging. Patient started showing improvement with the initiation of face mask oxygenation, empirical IV antibiotics and IV steroid which was further augmented with bronchoscopic intervention by the pulmonology team. Flexible bronchoscopy played both a diagnostic and therapeutic role in this patient. A patient presenting to emergency room with clinical triad of cough, dyspnea and massive hemoptysis has to be approached with high suspicion for diffuse alveolar hemorrhage. Rapid evaluation for its etiology and initiating empirical treatment can prevent morbidity and mortality, thereby improving survival outcome.

1. Introduction

Dyspnea is the most prevalent symptom among patients with cardiac and respiratory diseases.[1] Some manifestations of this symptom are less known and pose a challenge, either in identifying the underlying mechanisms or its impact on patients.[2] Dyspnea associated with hemoptysis help us to narrow down the provisional diagnosis. Its occurrence often indicates sinister thoracic pathology.[3] Determining the causes and management in such symptomatology requires a multidisciplinary approach. Expectoration of even a relatively small amount of blood is an alarming symptom, and massive hemoptysis can be a life-threatening event. Although pulmonary tuberculosis is the most important cause of hemoptysis in India, hemoptysis may also occur due to a variety of other causes.[4] Some of the causes require a high degree of suspicion to avoid the mortality that might occur in an emergency room (ER). Several critical diagnoses should be promptly considered to determine the best treatment option to stabilize the patient and prevent mortality. We describe a case of a 64 years old male who presented to an ER with complaints of acute onset of dyspnea, cough and hemoptysis.[5] We present the following case in accordance with the CARE reporting checklist.

2. Case Presentation

A 64 years old male presented to our ER complaining of acute onset dyspnea (NYHA class III), cough and fever for 2 days. Six hours prior to the arrival, he had a history

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of continuous cough associated with hemoptysis of around 500ml. He is a known Hypertensive on oral medications for 6 years. He had no past history of tuberculosis, chronic lung diseases, drug allergy, sick contact and smoking.

On presentation, general examination revealed cold clammy peripheries. His vital signs were saturation of 86% at room air, pulse rate of 108bpm, blood pressure of 80/50mmHg, respiratory rate of 26 cycles per minute and temperature of 101.4°f. On auscultation of the chest, bilateral crackles were heard. His Electrocardiography was suggestive of sinus tachycardia with the right bundle branch block. Initial Blood gas showed hypoxemic respiratory failure with pH (7.423), PO2 (64.9mmHg), PCO2(31.5mmHg), cHCO3(20.1mmol/L) & SO2(92.7%). Routine laboratory blood investigations were sent along with the sputum acid fast bacilli & gram stain tests. Initial Chest X-ray (Figure 1) revealed bilateral lower zone haziness. Transthoracic echocardiography revealed mild left ventricular hypertrophy with normal cardiac function.

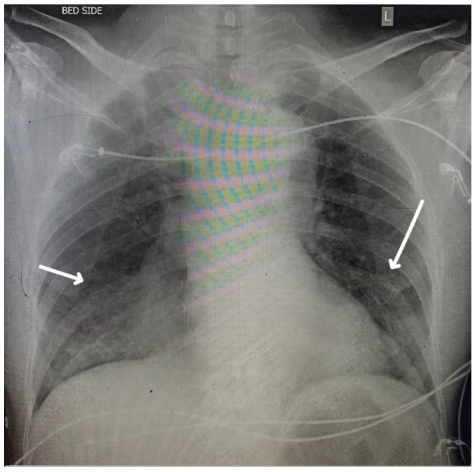

Fig. 1: Chest X-ray AP view revealed bilateral lower zone haziness (right>left) indicated by white arrow (Image taken from the department of radiology, KMCH)

In ER, the patient was managed with face mask oxygen, nebulisation, IV fluids, IV antipyretic and IV tranexamic acid. Blood and Urine samples were sent for culture/sensitivity. He was started on IV Piperacillin tazobactam and Doxycycline. In view of hypotension, fluid challenge of 500ml 0.9% normal saline was given. Since hypotension persisted despite fluid challenge, norepinephrine infusion started and titrated to maintain mean arterial pressure ≥65mm Hg. Initial blood investigations (Table 1) revealed normal hemoglobin and creatinine with elevated total WBC counts.

Diagnostic criteria were followed based on algorithms derived from the standard article that was published by Kathuria et al., and patients were evaluated further.[6] Patient CECT thorax (Figure 2) suggestive of diffuse alveolar hemorrhage (DAH) and bilateral tortuous bronchial arteries

Table 1: Initial routine laboratory investigations

Lab parameters	Patient values	Normal values
Hemoglobin	12.6 g/dL	13 to 17.5 g/dL
Total WBC Count	25,500 Cells/cumm	4000 to 11300 Cells/cumm
Hematocrit	37.8%	40 to 52%
Urea	31 mg/dL	10 to 50 mg/dL
Creatinine	1.2 mg/dL	0.6 to 1.2 mg/dL

WBC- White blood corpuscles, g-gram, dL-decilitre, cumm-cubic millimetre, mg-milligram

with no aneurysm / active extravasation. He was managed with high dose IV corticosteroids (1 g/day) in the form of pulse therapy. His autoimmune profile sent and results awaited. He remained stable throughout his ER observation and shown improvement with above management.

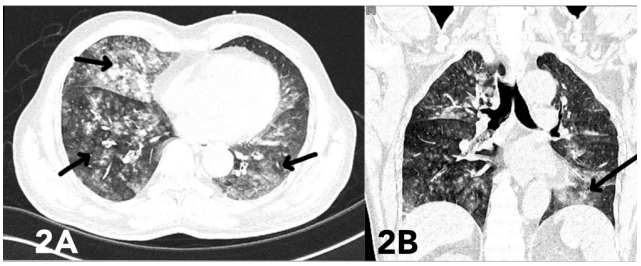

Fig. 2: CECT Thorax showed diffuse centrilobular ground glass nodular opacities with few patchy areas of consolidation in right middle lobe suggestive of alveolar hemorhage (**2A**) and dependant ground glass opacities in left lower lobe basal segments suggestive of post aspiration changes (**2B**). (Images taken from the department of radiology, KMCH)

On the following days in the acute care unit, the patient had respiratory distress requiring non invasive ventilation (NIV) support. Follow up CT thorax (Figure 3) after 24 hours revealed an evolving right middle lobe consolidation & mildly decreased density of alveolar hemorrhage. His autoimmune profile (Table 2) was negative for all diseases.

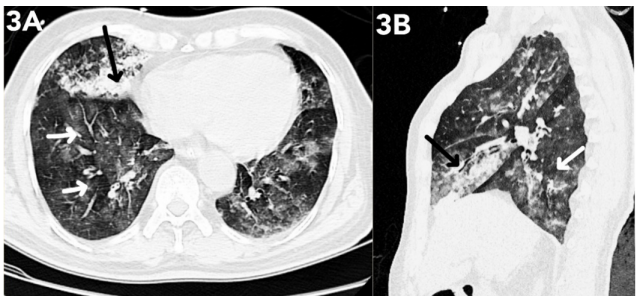

Fig. 3: CT Thorax follow up (both **3A & 3B**) revealed mildly decreased density of alveolar hemorrhage (white arrows) and evolving right middle lobe consolidation (black arrows). (Images taken from the department of radiology, KMCH)

Table 2: Autoimmune profile

Autoimmune profiles	Patients results	Normal range
Anti-Sjögren Syndrome A	Negative	(0) Negative (+) Borderline (+,++) Positive (+++) Strong positive
Anti-Sjögren Syndrome B	Negative	(0) Negative (+) Borderline (+,++) Positive (+++) Strong positive
Anti-Ribonucleoprotein-sm	Negative	(0) Negative (+) Borderline (+,++) Positive (+++) Strong positive
Anti-Scl-70(Scleroderma antibodies;anti-topoisomerase)	Negative	(0) Negative (+) Borderline (+,++) Positive (+++) Strong positive
Anti-Jo-1(Anti-Histidyl transfer RNA synthetase antibodies)	Negative	(0) Negative (+) Borderline (+,++) Positive (+++) Strong positive
Anti-smith antibody	Negative	(0) Negative (+) Borderline (+,++) Positive (+++) Strong positive
Anti-nuclear antibodies	Negative	(0) Negative (+) Borderline (+,++) Positive (+++) Strong positive
Anti-Glomerular basement membrane IGG antibodies	<0.80	Negative : <7 Equivocal : 7 to 10 Positive : >10
Anti-neutrophil cytoplasmic antibodies	Negative	(0) Negative (+) Borderline (+,++) Positive (+++) Strong positive
Anti-dsDNA antibodies	Negative	(0) Negative (+) Borderline (+,++) Positive (+++) Strong positive
Anticardiolipin IGG & IGM antibodies	<10.0	Negative : <10.0 U/mL Weak positive : 10.0 to 40.0 Positive : >40.0
Antiphospholipid IGG & IGM antibodies	<1.00	Normal : <12 U/mL Equivocal : 12 to 18 Positive : >18

Repeat laboratory investigations suggestive of decreased hemoglobin (9.6 g/dL), decreased hematocrit (28.4%) and preserved renal functions. C3 and C4 complement levels were found to be in the lower range. Blood culture was negative, urine culture showed Enterobacter cloacae.

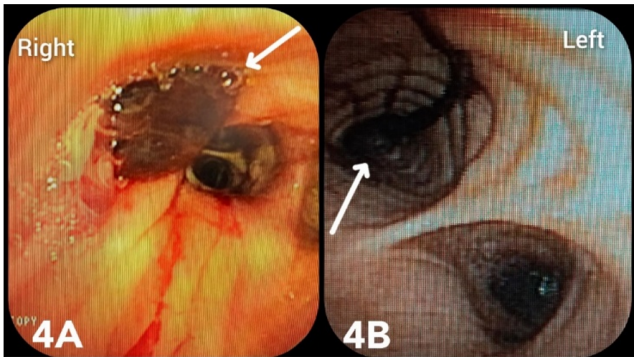

Fig. 4: Bronchoscopic images showing blood clots (white arrows) in right middle lobe bronchus (**4A**) and left lower lobe bronchus (**4B**). (Images taken from the department of pulmonology, KMCH)

Flexible bronchoscopy was done by pulmonology team revealed blood clots causing partial obstructions in secondary & tertiary bronchi of right middle lobe, right lower lobe and left lower lobe (Figure 4). Few obstructive clots were removed and Broncho alveolar lavage(BAL) done.

BAL revealed no Acid fast bacilli, cytology revealed neutrophil predominant smear and culture revealed growth of Enterobacter cloacae which was sensitive to Meropenem, Imepenam, Colistin, Amikacin and Fosfomycin. Patient remained persistently hypoxemic, requiring overnight NIV. Fever spikes persisted and antibiotic therapy escalated to Meropenem and Amikacin. In view of worsening infiltrates in chest X-ray (Figure 5), repeat flexible bronchoscopy was done by a pulmonology team.

Bronchoscopy revealed complete occlusion of the right lower lobe bronchus by organised blood clots extending from right bronchus intermedius to right middle bronchi, lower lobe bronchi & their segmental branches. The entire clots were removed by a pulmonologist and airway patency was established. Patient clinically improved following the procedure and follow up chest x-ray (Figure 6) revealed reduced infiltrates.

Though uncommon, the cause for alveolar hemorrhage in this patient was infectious in origin based on BAL culture report, and he was managed with appropriate antibiotics. Later he was taken up for definitive treatment and performed bronchial artery embolization. He improved clinically and oxygen requirement decreased. Antibiotics were continued for 2 weeks, and he was discharged in stable condition.

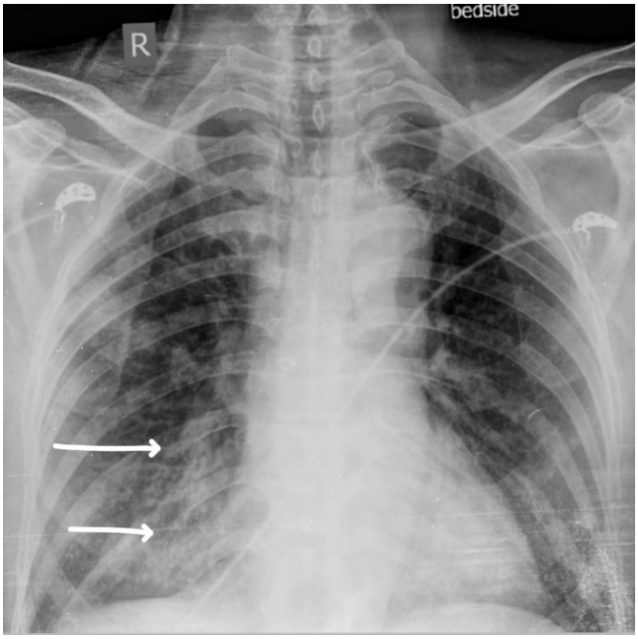

Fig. 5: Chest X-ray follow up revealed significant infiltrates over right lower zone (white arrows). (Images taken from the department of radiology, KMCH)

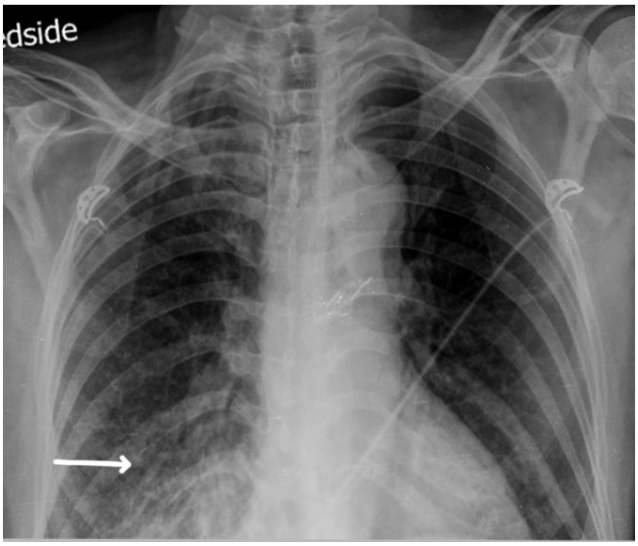

Fig. 6: Post Bronchoscopic chest x-ray revealed reduced infiltrates over right lower zone (white arrow). (Images taken from the department of radiology, KMCH)

3. Discussion

Dyspnea associated with massive hemoptysis is always a serious symptomatology that has to be addressed promptly and evaluated thoroughly. Evaluating for cause would be challenging in the ER and requires a high degree of suspicion for DAH as it can have dreadful consequences.

Proper history taking involving hemoptysis might help in suspecting DAH but modalities like chest imaging or blood studies are required to confirm its existence. Modalities like CT thorax and Bronchoscopy are found to be superior to chest X-ray.[7]

DAH is a rare condition which has to be suspected in all patients with hemoptysis especially when their blood studies and x-ray imaging shows anemia and new onset infiltrates respectively. DAH often pose a challenge in both diagnostic and therapeutic aspects with poor prognosis and can quickly lead to death if there is delayed suspicion or diagnosis.[8] DAH usually expresses unexplained alveolar infiltrates with resultant hypoxemic respiratory failure that should raise suspicion for its existence in the ER.[9]

Diagnosing the etiology for DAH depends on clinical features, detailed laboratory studies and relevant imaging. DAH often presents to ER as a complication of another ongoing pathology requiring a multidisciplinary approach. Occasionally, multilobar pneumonia present along with DAH as either associative or simulative factor.[10]

One clinicopathological study by autopsy of 34 patients described the association of DAH with certain conditions apart from pneumonia. Those conditions are capillaritis associated systemic autoimmune disease [30 cases] and disease of unknown origin [4 cases].[11] The most prevailing causes for massive hemoptysis are cardiopulmonary origin [pneumonia, tuberculosis, mycetomas, bronchitis, bronchiectasis, neoplasm, pulmonary embolism, mitral stenosis, heart failure] or autoimmune origin [Goodpasture syndrome, Behcet's disease, Wegener's granulomatosis].[7,12]

Opting for conservative therapy depends on the etiology and severity of the hemoptysis. In case of severe hemoptysis, conservative therapy should be accompanied by significant diagnostic and therapeutic modalities.[7] Evaluation of massive hemoptysis is always challenging in ER and patient's hemodynamic stability should be prioritised before proceeding for any diagnostic or therapeutic modalities. Most importantly, resuscitation of airway, breathing & circulation takes priority in any hemodynamically unstable patients.

In stable patients, CT thorax is the imaging of choice which localises the bleeding site in 70 to 88.5% of cases. In case of unstable patients who require endovascular intervention, Flexible bronchoscopy (FB) is superior to CT imaging. It helps to overcome the difficult radiographic localisation of bleeding in those with extensive lung infiltration. It also contributes in hemorrhage control in case of visible endoluminal lesions but make sure the airway and circulation has been adequately resuscitated.[13]

FB is indicated in all cases of hemoptysis and bronchopulmonary infections. Along with BAL, it aids in identifying the culprit pathogens. Hence, preferred method for excluding infections even in immunocompromised individuals.[14] BAL procedure usually guided by CT imaging done prior to the procedure and samples to be collected from serial of BAL. Alveolar hemorrhage is confirmed when analysis of lavage fluid reveals increasing red blood cell counts in subsequent portions of the sample collected.[15]

DAH patients with hypoxemia often require supplemental oxygen ranging from low flow oxygen to high flow oxygen. They might requires non invasive or invasive ventilation in case of severe hypoxemia and respiratory failure.[10] In case of suspected infection to be the causative factor, empirical antibiotics have to be started in ER. Once DAH has been confirmed and its causative factor has been confirmed as infections through BAL procedure, Specific antibiotic regimen should be initiated.[10]

Enterobacter species are opportunistic pathogens typically found in intensive care units, with varying incubation times from a few hours up to 20 days.[16] In healthy individuals, the gastrointestinal tract represents the most important reservoir. Enterobacter cloacae pathogen which was identified in our patient's BAL sample was a rare pathogen and has the highest pathogenic potential corresponding to approximately 60–75% of all Enterobacter infections.[16] DAH with capillaritis can be life threatening. Early, systemic glucocorticoid therapy is the mainstay of treatment for capillaritis. Corticosteroids and immunosuppressive agents remain the gold standard in such cases.[9]

Mortality rate found to be high in case of massive bleeding (>1000ml/24hrs) and malignancy while other causes like bronchopulmonary infection has low mortality rate.[7] DAH is a one rare condition with in-hospital mortality ranging between 20-50%.[8] Despite advances in the identification and management of DAH, it remains a condition with high morbidity and mortality.[17] Hence high degree of suspicion is necessary in ER for alveolar hemorrhage.

4. Conclusions

Cough with dyspnea and hemoptysis are the symptoms of concerns in the ER. Approaching massive hemoptysis is always a challenge in patients with hemodynamic instability. High index of suspicion for DAH can be life saving. Most often, DAH occurs due to underlying systemic diseases. Infective causes for DAH is salvageable if managed empirically in ER as we did with our patient. Ultimately, early recognition and rapid evaluation of causative factor of DAH is necessary to improve morbidity

& mortality.

5. Conflicts of Interest

The authors have no conflicts of interest to declare.

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None

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