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A RARE CASE REPORT OF CRYPTOGENIC ORGANIZING PNEUMONIA WITH HAEMORRHAGIC PLEURAL EFFUSION

Respiratory Medicine

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ABSTRACT

Cryptogenic organizing pneumonia (COP) is a rare idiopathic interstitial lung disease characterized by granulation tissue proliferation within the distal airways and alveolar ducts, often misdiagnosed as community-acquired pneumonia (CAP) due to overlapping clinical and radiological features. We present a case of a 32-year-old male with subacute symptoms of fever, cough, and progressive dyspnea unresponsive to antibiotics. Initial investigations revealed elevated inflammatory markers, consolidation on chest radiograph, and negative infectious and autoimmune screening. Despite empirical antibiotic therapy, the patient showed no clinical improvement. High-resolution computed tomography (HRCT) demonstrated patchy peripheral consolidations and ground-glass opacities. Bronchoalveolar lavage (BAL) showed mixed inflammatory cells, and transbronchial lung biopsy confirmed the diagnosis by revealing intra-alveolar fibroblastic plugs (Masson bodies). Notably, the patient developed hemorrhagic pleural effusion—an uncommon and atypical feature of COP. Corticosteroid therapy with oral prednisolone led to significant clinical and radiological improvement. This case underscores the importance of considering COP in non-resolving pneumonia, particularly when imaging and clinical findings deviate from typical infectious etiologies. Early recognition and corticosteroid therapy are crucial for favorable outcomes. Atypical presentations, such as hemorrhagic pleural effusion, warrant careful evaluation to rule out alternative diagnoses and emphasize the variable spectrum of COP manifestations.

KEYWORDS

Cryptogenic organising pneumonia, haemorrhagic pleural effusion, atypical, steroids

INTRODUCTION:

Cryptogenic organizing pneumonia (COP) is a rare form of idiopathic diffuse interstitial lung disease.[1] Alveolar injury from various causes results in granulation tissue proliferation within the distal airways and alveolar ducts consisting of fibroblasts and myofibroblasts intermixed with loose connective matrix.[2] While COP is typically idiopathic, it can also occur secondary to infections, drug reactions, autoimmune diseases, posttransplant, radiation or environmental exposures.[3] Clinically, OP is associated with a syndrome of acute or subacute onset that may include cough, fever, and progressive dyspnoea, and radiographically, with multiple patchy alveolar opacities not responsive to antibiotics.[4] Diagnosis requires a combination of clinical, radiological and histopathological findings with transbronchial or surgical lung biopsy often confirming the presence of intra-alveolar fibroblastic plugs (Masson bodies) and organizing pneumonia.

Case:

A thirty-two years old male presented in the emergency of Pulmonary Medicine department on December 11, 2024 with chief complaints of fever, minimally productive cough and breathlessness from 15 days. Fever was insidious, low grade, intermittent, not associated with chills and rigors and relieved on taking oral antipyretics. Cough was gradual in onset, progressively increasing in frequency, productive in nature with no diurnal or postural variation. Breathlessness was gradual in onset, progressively increasing in severity, having progressed from modified Medical Research Council grade 1 to 3 over a period of 15 days, was not associated with orthopnea and paroxysmal nocturnal dyspnoea. There was no history of similar episodes in the past. Furthermore, there was no history of Coronavirus Disease 2019 (COVID 19) infection, recent travel, any comorbidities or chest trauma. Patient was a non-smoker, non-alcoholic and driver by occupation.

Patient was febrile with temperature of 101 F. His pulse was 112/min and respiratory rate was 24 breaths per minute and Sp02 of 88% on room air and patient was using accessory muscles of respiration. Chest examination was unremarkable except for fine crepitations in bilateral interscapular and infrascapular areas and rhonchi.

On the basis of clinical findings, differential diagnosis of acute respiratory infection and interstitial lung diseases were considered and patient was investigated accordingly.

The total leucocyte count (TLC) was 13,800 cells/mm^3, haemoglobin (Hb) was 9.8 g/dL, and the red blood cell (RBC) count was 3.64 million/mm^3. Serum glutamic-oxaloacetic transaminase (SGOT) was 57 U/L, and serum glutamic-pyruvic transaminase (SGPT) was 32 U/L. Blood urea was 70 mg/dL, and serum creatinine was 1.1 mg/dL. Serum sodium (Na^+) was 136 mmol/L, and serum potassium (K^+) was 5.2 mmol/L. Total serum protein (TSP) was 5.6 g/dL, and direct serum protein (DSP) was 3.2 g/dL. Screening for human immunodeficiency virus (HIV), hepatitis C virus (HCV), and hepatitis B surface antigen (HBsAg) was non-reactive. D-dimer was measured at 0.4 µg/mL. The erythrocyte sedimentation rate (ESR) was 30 mm/hr. The antinuclear antibody (ANA) test showed a titre of 1:30, and the rheumatoid factor (RA factor) was 10 units/mL. Induced sputum samples revealed no organisms on Gram stain, fungal stain, fungal culture, or cartridge-based nucleic acid amplification test (CBNAAT). A nasopharyngeal swab was sent for influenza A (H1N1), respiratory syncytial virus (RSV), and COVID 19 reverse transcriptase polymerase chain reaction (COVID-19 RT-PCR), all of which were negative. Chest radiograph obtained on Day 1 showed consolidation in right middle and lower zone with air bronchogram and heterogenous opacities in rest of the lung fields. A diagnosis of community acquired pneumonia was made and patient was managed with respiratory fluoroquinolone and macrolide along with other supportive therapy.

Patient did not show significant improvement in respiratory symptoms on Day 3 and hence Computed Tomography (CT) scan and bronchoscopy were done where CT scan showed patchy, peripheral consolidations with ground glass opacites (as shown in Figure I).

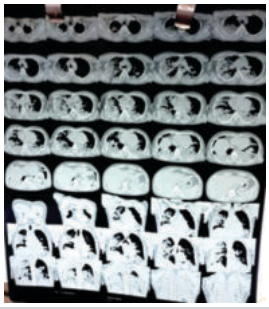

Figure I - CT Chest on Day 3 shows patchy and peripheral consolidations with ground glass opacites.

Bronchoalveolar lavage (BAL) fluid showed significant accumulation of lymphocytes, neutrophils, and eosinophils and was negative for CBNAAT and no organisms were identified on Gram stain and culture and fungal stain and culture. ON day 5 Repeat chest radiograph was performed which showed right synpneumonic effusion which was aspirated which was haemorrhagic and its investigations are as follows-Pleural fluid for gram stain and culture and CBNAAT was negative, Cytology showed 55% lymphocytes, 48% neutrophils and no malignant cell was present. Repeat hemogram showed persistent leucocytosis with neutrophilia and antibiotics were changed to beta lactams with extended gram-negative coverage and aminoglycosides. On day 8 patient did not show clinical improvement and in view of the above findings, a diagnosis of cryptogenic organizing pneumonia was considered and patient was started on corticosteroid therapy with 60 mg prednisolone per day. After one week of initiation of steroid treatment, patient showed significant improvement in respiratory symptoms, hemogram also normalised. Patient was sent for repeat CT Chest which showed patchy areas of consolidation with fibrobronchiectatic changes in bilateral lung fields and interlobular septal thickening with right sided hydropneumothorax and left side pleural effusion. Honeycombing was also seen in right subpleural and basal areas with rest of the lung showing ground glass haziness suggestive of consequences of COP (as shown in Figure II).

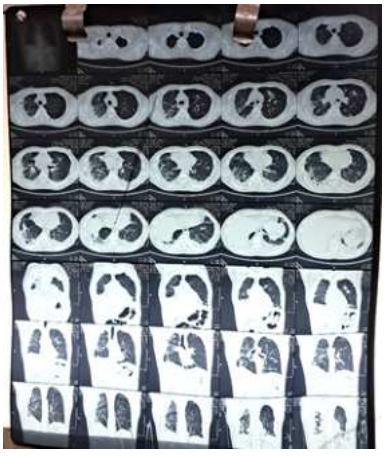

Figure II - Day 15 CT CHEST shows patchy areas of consolidation with fibrobronchiectatic changes in bilateral lung fields and interlobular septal thickening with right sided hydropneumothorax and left side pleural effusion.

Transbronchial lung biopsy of the patient showed intraluminal fibromyxoid plugs composed of fibroblasts and myxoid connective tissue (Masson Bodies) and found within bronchioles, alveolar ducts, and alveoli causing airway obstruction. In view of the above findings, a diagnosis of cryptogenic organizing pneumonia was made and patient was discharged on corticosteroid therapy with 60 mg prednisolone per day along with tab calcium and vitamin D3. Subsequent chest radiograph 1 month after initiation of steroid therapy showed complete resolution of infiltrates (as shown in Figure III) and chest was clear on auscultation.

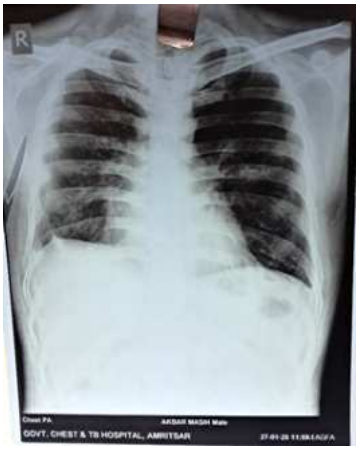

Figure III Chest Xray after one month of steroid therapy shows complete resolution

DISCUSSION:

Cryptogenic organizing pneumonia (COP), formerly known as bronchiolitis obliterans organizing pneumonia (BOOP), is a rare idiopathic interstitial pneumonia characterized by intra-alveolar fibroblastic proliferation and chronic inflammation. While it may be idiopathic, secondary causes include infections, autoimmune disorders, drug reactions, and environmental exposures.[5] COP typically presents with subacute symptoms such as cough, fever, and dyspnea, often mimicking community-acquired pneumonia (CAP), which can lead to misdiagnosis.[6] Radiologically, COP usually demonstrates patchy alveolar opacities and ground-glass changes, contributing to diagnostic challenges.[7]

Pleural effusion is uncommon in COP and is typically mild and non-hemorrhagic. Hemorrhagic pleural effusion, as seen in this case, is atypical and mandates thorough evaluation to exclude tuberculosis, malignancy, vasculitis, or pulmonary embolism. The pleural fluid analysis, negative for malignancy and infections but with elevated red blood cells and inflammatory cells, helped rule out common causes. The hemorrhagic nature may result from capillary inflammation, vascular fragility, or secondary infection.[8]

Diagnosis relies on high-resolution computed tomography (HRCT) and histopathological confirmation. In this case, bronchoscopy with bronchoalveolar lavage (BAL) and transbronchial lung biopsy (TBLB) revealed intraluminal fibroblastic plugs (Masson bodies), confirming COP.[9] The absence of granulomas, malignancy, or infection supported the diagnosis. Treatment with corticosteroids, such as oral prednisolone, typically results in rapid clinical and radiologic improvement, as observed here. However, relapses are common with rapid tapering, emphasizing the need for gradual dose reduction and regular follow-up.[10]

This case illustrates the need to consider COP in non-resolving pneumonia, particularly with atypical findings like hemorrhagic effusion. Early recognition and steroid therapy can significantly improve outcomes. Further research is warranted to explore the mechanisms of pleural involvement and prevent relapses.

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