

Pulmonary-Renal Syndrome due to Fat Embolism: Following Intravenous Administration of an Oil based Anabolic Steroid Injection in Young Male

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ABSTRACT

Introduction: Presenting a case of Pulmonary-renal syndrome following intravenous administration of an oil based steroid injection in a young male.

Case Report: 27 years old male presented with breathlessness. On evaluation, he was detected to have bilateral diffuse lung infiltrates on CXR PA, azotemia, deranged LFTs. Computed tomography revealed predominantly peripheral ground-glass opacifications. Renal biopsies revealed acute tubular necrosis. The patient's symptoms gradually improved over 72 hours and imaging of the chest was unremarkable one week later. The pathophysiology, diagnosis and treatment of this rare but potentially life-threatening complication of intravenous oil injection are discussed.

Conclusion: The present case illustrates the need for timely diagnosis of pulmonary oil embolism and institution of supportive care measures to limit the morbidity and mortality associated with this rare, but potentially life-threatening condition. A high index of suspicion is necessary to make an accurate diagnosis given the variability of clinical presentation and the relative paucity of specific findings on diagnostic imaging and laboratory testing results.

Keywords: Pulmonary Oil Embolism, Steroid-Oil Injection, Pulmonary-Renal Syndrome

INTRODUCTION

Presenting a case of Pulmonary-renal syndrome following intravenous administration of an oil based steroid injection in a young male. 27 years old male presented with breathlessness. On evaluation, he was detected to have bilateral diffuse lung infiltrates on CXR PA, azotemia, deranged LFTs. Computed tomography revealed predominantly peripheral ground-glass opacifications. Renal biopsies revealed acute tubular necrosis. The patient's symptoms gradually improved over 72 hours and HRCT chest plain done later showed significant improvement. Pathophysiology of this rare disorder of fat embolism causing multiorgan dysfunction is discussed.¹

CASE REPORT

27 year old male with no prior comorbidities came with complaints of low grade Fever and cough since 3-4 days, hemoptysis with bright red blood 2 episodes. Later he developed progressively worsening Dysnoea, No any history of chest pain, palpitations, orthopnea or PND, pedal edema. No history of loss of weight or loss of appetite, No history of any other bleeding manifestations. No any significant family and personal history. On examination there was tachycardia,

tachypnea, desaturation on room air, bilateral diffuse coarse creptations. Patient was admitted in ICU. Started on oxygen. Initially patient hide history of intravenous administration of oil based steroid injection. His routine labs on admission as shown in chart.

Table-1 shows HB-13 gm/dl Total Bilirubin-2.85mg/dl TLC-12000 Direct Bilirubin-2.0mg/dl DLC- N 68%, L 28%, E 2%, M 2% Indirect Bilirubin -0.85mg/dl.

Platelet counts -70,000/cmm PCV-36.6 SGOT-380.6IU/ml Blood urea - 64mg/dl SGPT-164.5IU/ml Serum creatinine -2.18mg/dl ALP-100.1IU/ml Serum sodium - 135.6meq/l Serum Albumin -2.14g/dl Serum potassium -3.16meq/l Serum Globulin-2.11g/dl INR-3.16 APTT-45.4

Malaria, dengue, leptospirosis, Weil Felix, widal, brucella all were negative by ELISA method. Urine routine shown albumin 2+, plenty of pus cells, Urine protein creatinine ratio -1.27, Peripheral smear showed normocytic normochromic, no schistocytes, Procalcitonin -2.1, ESR 47, CRP 202, HIV, HBsAg, HCV negative, Urine and blood cultures were sterile. Sputum analysis does not revealed any other infective pathology, USG abdomen - splenomegaly with bilateral altered renal echogenicity, Echocardiography and electrocardiogram were unremarkable. D-dimer was elevated and fibrinogen was low.

Chest x-ray on admission (Fig-1)

HRCT chest (Fig -2)

In view of suspected Pulmonary-renal syndrome, vasculitis workup was sent and patient was initiated on intravenous methylprednisolone and intravenous cyclophosphamide. C-ANCA, P-ANCA, Anti-GBM antibody were negative by ELISA method. Renal parameters and hematological parameters monitored regularly.

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INR-3.16	APTT-45.4

Table-1:

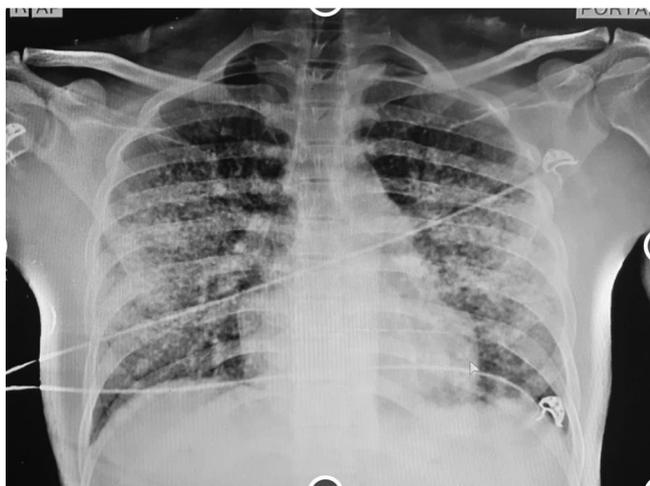


Figure-1: Chest x-ray on admission



Figure-3: CX ray at discharge

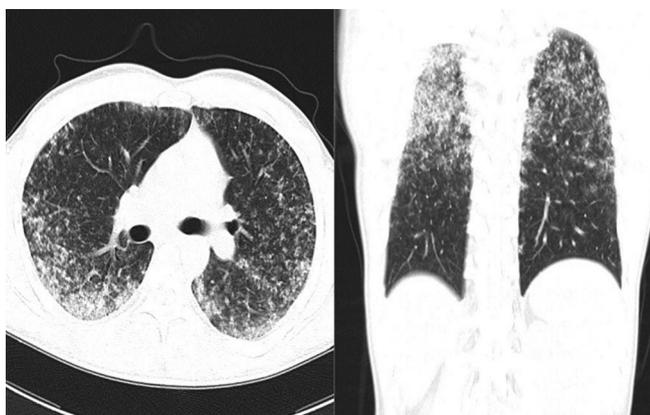


Figure-2: HRCT chest



Figure-4:

Azotemia, thrombocytopenia, coagulopathy and hepatic functions showed improvement over 3-4 days. CXR PA showed gradual improvement. Patient became symptomatically better. CX x ray at discharge- Fig-3)

After correcting coagulatory parameters, renal biopsy was done to rule out vasculitis (Fig-4).

Renal biopsy showed mild acute tubular injury with normal glomeruli.

Patient was started on oral steroids after giving pulse steroid. After discharge patient disclosed that he had taken six oil based injections of nandrolone intravenously by himself without consulting any doctor to increase muscle mass 4 days before hospital admission. (Inj Nandrolone are easily available in indian pharmaceutical shops). Oral steroids were

tapered and stopped. He achieved normal renal functions within 2 weeks of hospital admission. So diagnosis was made of Pulmonary-Renal syndrome due to fat embolism. Renal biopsy showed mild acute tubular injury with normal glomeruli. Patient was started on oral steroids after giving pulse steroid.

DISCUSSION

Fat embolism causing pulmonary renal syndrome has only been described in a small number of case reports.¹⁻⁶ Fat embolism syndrome is characterized by introduction of lipid emboli into the systemic circulation, so damage of pneumocytes and capillary endothelial cells of liver and kidney, this is only after initial asymptomatic latent period.⁷

The clinical presentation of FES is diverse, from mild respiratory distress, non-productive cough, to the life threatening triad of FES consisting of acute respiratory distress, petechial hemorrhages and delirium. Classic triad is very rare 3-4 %, inspite of presence of high incidences of long bone fractures.⁸⁻¹⁰ Majority of cases of fat embolism syndrome go undetected due to clinical asymptomatic period. Diagnosis is also challenging due to initial asymptomatic period and no specific diagnostic tests. Bronchoalveolar lavage to detect fat droplets in alveolar macrophages is also not useful.¹¹ Radiological imaging is also not specific for fat embolism causing acute respiratory syndrome.

In contrast to classic presentation of fat embolism syndrome, patient described in the present report experienced an acute dyspneic reaction, renal and hepatic insufficiency following 4 to 5 days after introduction of lipid injections into the blood stream. Similar reactions preceding the development of acute respiratory syndrome have been described within 1 hour of arterial chemoembolization for hepatocellular carcinoma¹⁴, that means introduction of lipid droplets into blood can give rise to rapid onset of lung damage.

The injection of nandrolone was unwitnessed in the present case and it was, therefore, difficult to discern whether the initial shortness of breath was truly respiratory difficulty due to intravenous injection of oil or more a subjective experience related to the injection event itself.

The patient's condition improved dramatically over the 72 hours following initial presentation, and he was subsequently discharged home with a resting oxygen saturation of 99%. Chest x-rays at the time of discharge and one week later (Figure 1C) were both unremarkable.

CONCLUSION

The present case illustrates the need for timely diagnosis of pulmonary fat embolism, for betterment of patients. In view of lack of specific diagnostic tests for fat embolism syndrome and variable presentation, high index of suspicion is necessary. Chest Xray and CT chest can be useful in prompting physicians to consider the possibility of pulmonary oil embolus as a diagnosis, in clinical setting of acute respiratory distress following intravenous lipid injections.

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