



Journal Homepage: - www.journalijar.com
**INTERNATIONAL JOURNAL OF
 ADVANCED RESEARCH (IJAR)**

Article DOI: 10.21474/IJAR01/1432
 DOI URL: <http://dx.doi.org/10.21474/IJAR01/1432>



RESEARCH ARTICLE

A CASE REPORT ON PULMONARY ALVEOLAR MICROLITHIASIS.

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Manuscript Info

Manuscript History

Received: 12 June 2016
 Final Accepted: 19 July 2016
 Published: August 2016

Key words:-

Abstract

Background: Pulmonary alveolar microlithiasis(PAM) is a rare inborn error of calcium metabolism with unknown etiology and familial association in which concretions composed of calcium and phosphorus collect in alveolar spaces¹. It was first named in 1933 by Puhr.

Case Report: A 42 year old male non smoker, weaver presenting with 6 months history of fever, cough with mild expectoration and dyspnea on exertion was diagnosed as miliary tuberculosis by a local physician for which he was given antitubercular treatment for 3 months but his symptoms was not improving for which he came to outdoor. He had no past history of any chronic diseases or any chronic drug use. His general physical examination was unremarkable with vitals being normal. Respiratory system examination revealed bilateral vesicular breath sound with fine inspiratory crackles in axillary, infraaxillary infrascapular area.

Investigation: His laboratory parameters and Pulmonary function test were normal and sputum was negative for acid fast bacilli. His ABG parameter and Echocardiography report were normal. Chest X-ray shows bilateral diffuse micronodular shadows (sand-storm pattern) predominant in middle and lower zones and disease free pleura as black pleura line. HRCT thorax shows diffuse calcified micronodular opacities with lower lobe and subpleural predominance and septal thickening. BAL Fluid shows characteristic calcified spherules.

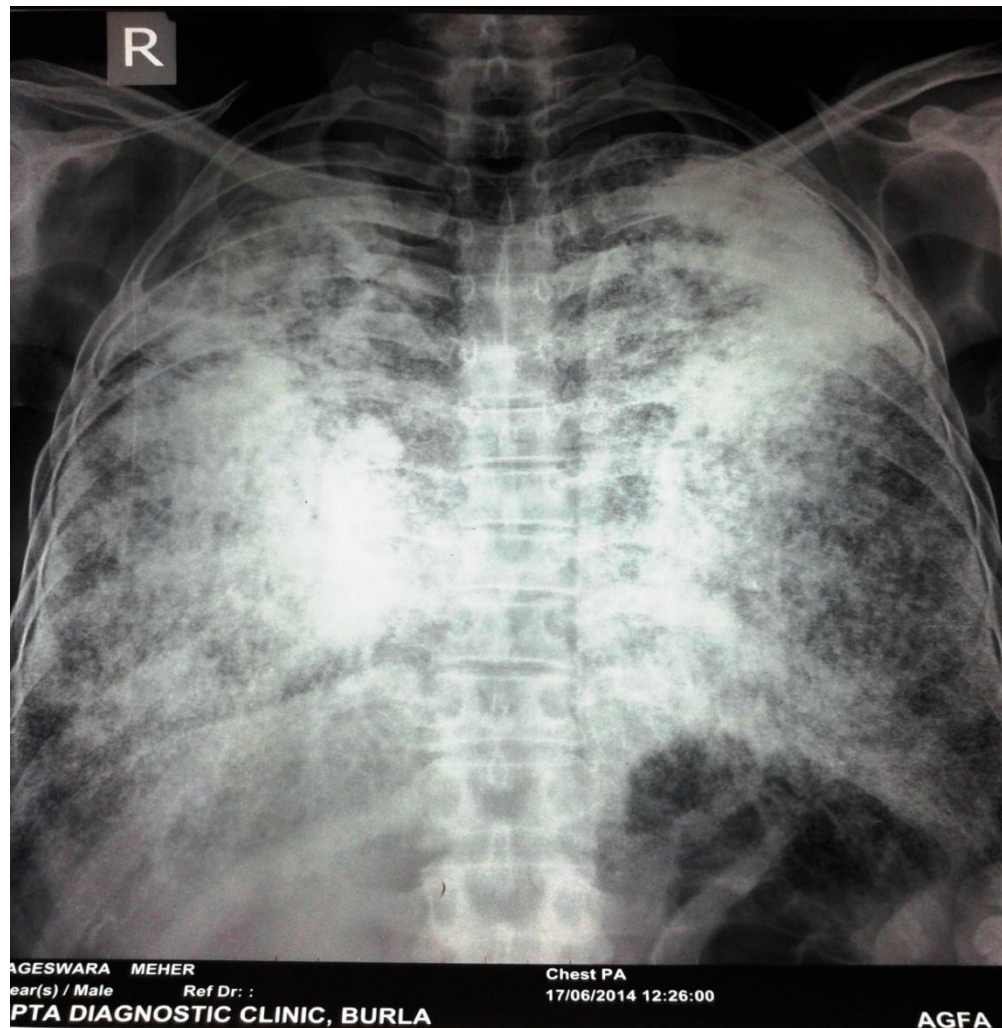
Discussion: PAM is due to inborn error of calcium metabolism confined to lung and leading to deposition of calcified spherules in alveolar spaces². It is inherited as autosomal recessive condition diagnosed during third through fifth decades³. Most patients are asymptomatic. Some may present with cough, progressive dyspnea, haemoptysis. Inspiratory crackles, clubbing and signs of cor pulmonale seen in advanced cases. Chest X-ray may show diffuse micronodular shadows (sand storm pattern) and black pleura line (disease free pleura appears as black line). HRCT may show micronodular opacities and subpleural predominance. PFT may show restrictive defect⁴. Transbronchial lung biopsy is confirmative⁵. Currently there is no effective treatment is available except lung transplantation.

Conclusion: Pulmonary alveolar microlithiasis being a rare disease with unknown etiology has burdened the patient with unnecessary doses of anti-tubercular treatment because of its resemblance to miliary

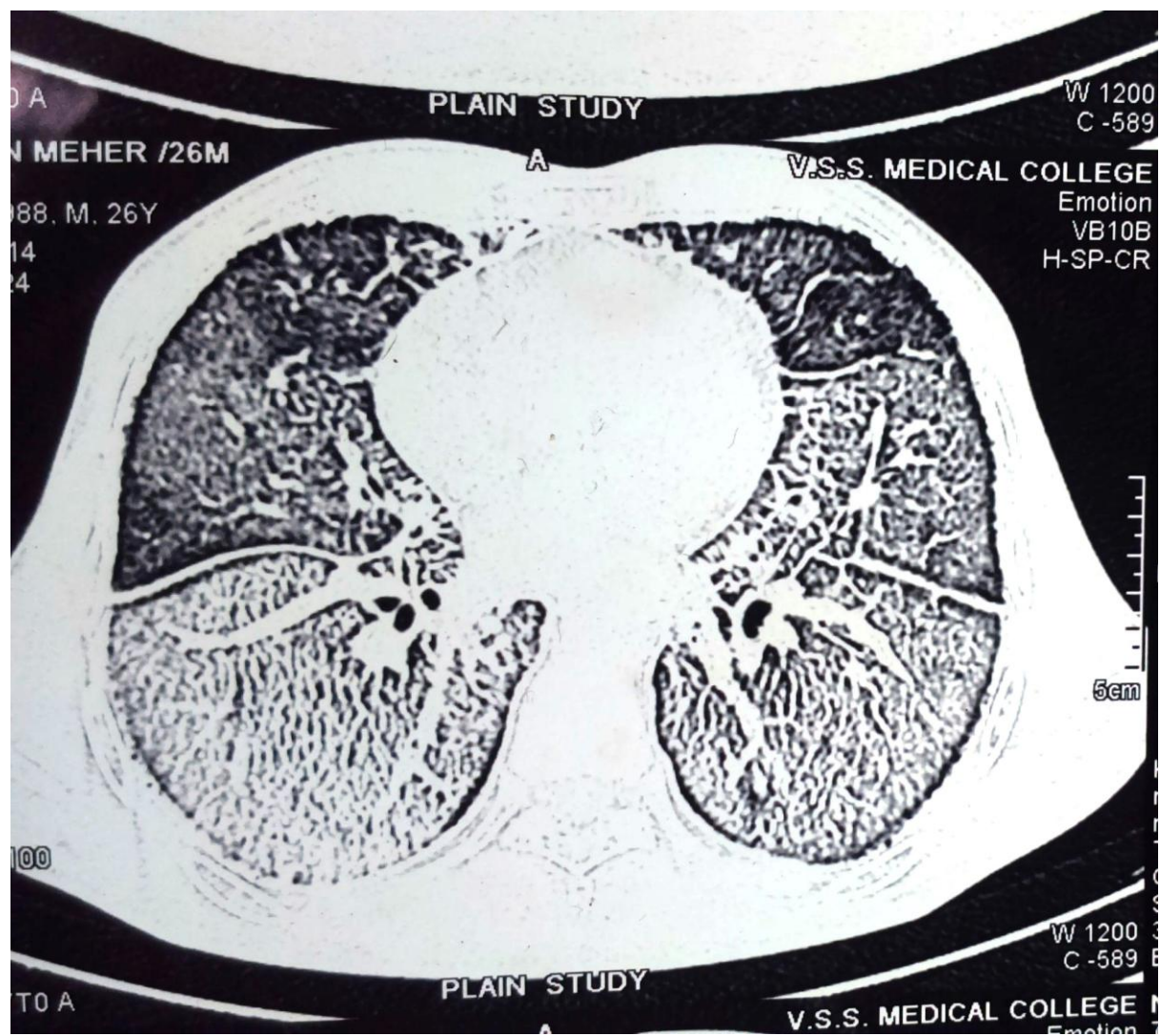
tuberculosis on chest X-ray. Patients due to delayed diagnosis ultimately progress to end stage lung disease necessitating lung transplantation. So attempt for earlier diagnosis and finding the etiology is all that needed.

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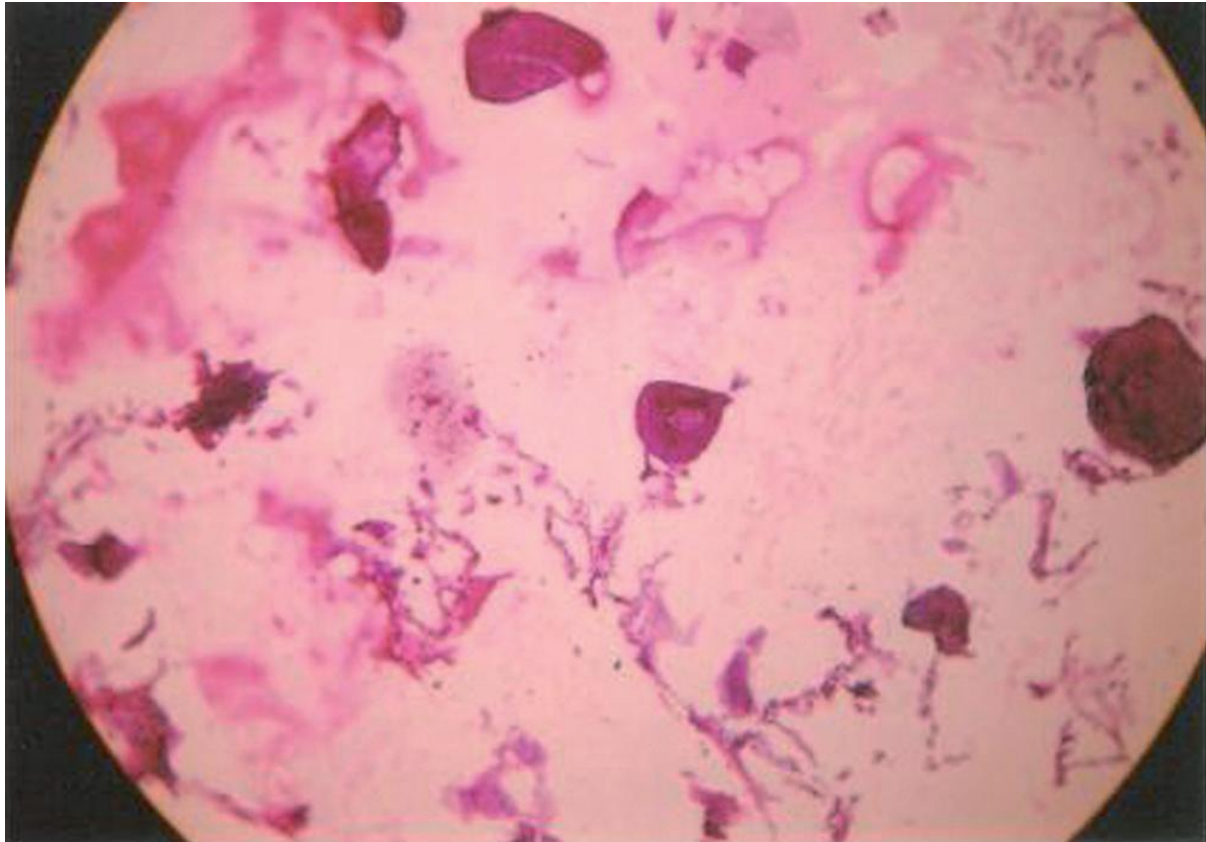
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Chest X-ray showing B/L diffuse micronodular calcific shadows (sandstorm appearance)



HRCT of thorax showing B/L diffuse calcified micronodular opacities with lower lobe and subpleural predominance and septal thickening



Bronchoscopic biopsy demonstrating intraalveolar and intraparenchymal concentric lamellar bodies.