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Silicosis presenting as bilateral hilar lymphadenopathy and lithoptysis

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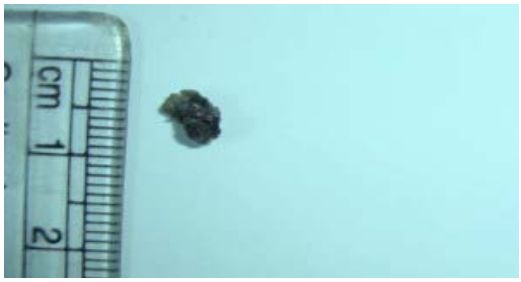
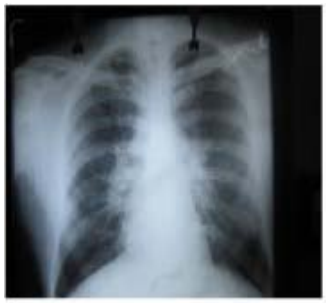
Abstract : Silicosis is one of the most common occupational lung diseases in the world. It has varied clinical presentation . Silicosis presenting as bilateral hilar lymphadenopathy without any pulmonary nodule is very rare.Only few studies are available describing such a presentation despite the known fact that hilar nodes are the first to be involved in pathogenesis of silicosis. Lithoptysis is a rare medical phenomenon commonly caused by tuberculosis and histoplasmosis. Only few cases of lithoptysis have been reported in patients with silicosis in medical literature. We report a case of silicosis who worked as a ledgeman in quarry fields 9 years back and presented to us with lithoptysis . His imaging study showed bilateral hilar lymphadenopathy with egg shell calcification.

Keyword :silicosis, lithoptysis, broncholithiasis, egg shell calcification

Case report:
A forty year old male presented with dyspnea on exertion for past 5 years , now MRC grade 2 without orthopnea or PND. History of cough with mucoid sputum was present for past five years. He gave history of spitting up of stone like particles for past one week which has brought him to the hospital. On the day of admission he spat out a stony hard particle during a out of cough.He has no history of fever, weight loss or loss of appetite. He was not a diabetic or hypertensive. He had no past medical history of tuberculosis . He gave a history of twenty pack years of smoking. He worked in quarry as a ledgeman from 1992-2002 without wearing any personal protective devices. Now he works in a tea shop.**lithoptysis**



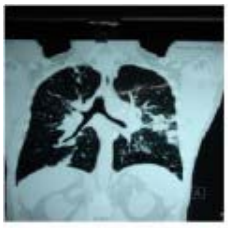
On examination he was afebrile and tachypneic. He had no pallor , cyanosis, clubbing, icterus, pedal edema or lymphadenopathy. His vitals were BP: 130/90 mm Hg, PR: 96/ min, RR: 34/min. On auscultation he had bilateral wheeze. Other systems were normal. Blood sugar: 102 mg%. Blood urea: 34 mg% , serum creatinine: 0.6 mg%. Total bilirubin 1.0mg/dl. AST: 30 IU/L, ALT : 34 IU/L, ALP : 96 IU/L, Total protein: 6 g/L, albumin: 4 g/L, globulin: 2 g/L . Total count : 7600 /mm3 . ESR 46 mm/hr, Hb 10.4 gm/dl.HIV ELISA: negative



hyperinflated lung fields with bilateral hilar lymphadenopathy

egg shell calcification of nodes: typical ring like shadows noted around the nodes

Chest x ray showed hyperinflated chest and bilateral multiple hilar lymphadenopathy with typical egg shell calcification His serum ACE levels were normal. Serum calcium 9.0 mg/dl. Fundus and slit lamp examinations were normal. Sputum smear and culture were negative for Mycobacterium tuberculosis. Mantoux was negative.On fifth day of hospital admission , patient developed fever and cough with purulent sputum.CT thorax showed consolidation of left upper lobe and bilateral peribronchial calcified (egg shell pattern) lymph node



CT Thorax showing left upper lobe consolidation

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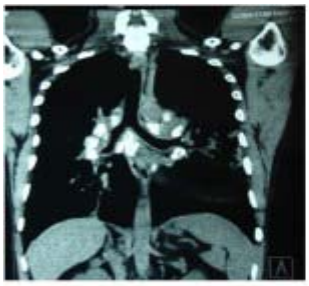

CT Thorax showing bilateral peribronchial lymph nodes distorting both bronchus which is a charecteristic finding of Broncholithiasis

Patient was treated with antibiotics and bronchodilator. He was afebrile and hemodynamically stable at the time of discharge.

Discussion:

Silicosis can present as acute, accelerated and chronic silicosis. Chronic simple silicosis commonly present as a small nodule in mid or upper zone that may appear even many years after termination of exposure to silica. The time of onset of radiological findings and rate of progression of disease depends on cumulative dose of exposure to silica. Inhaled silica reaches lung aleveoli where it is engulfed by acinar macrophages which are then carried via pulmonary lymphatics to hilar nodes. Hilar nodes get enlarged , fibrosed and sometimes calcify. Once pulmonary lymphatics are blocked parenchymal silicosis begins.[1,2]

Although animal studies suggest hilar nodes are first to be involved in silicosis, only few case reports are avialable in which silicosis present as bilateral hilar lymphadenopathy without parenchymal involvement.A study by Cox Ganser JM et al showed that lymph node silicosis actually precedes parenchymal silicosis.[2] Baldwin et al published a case series of five patients with silicosis who presented with isolated hilar lymphadenopathy.[3]

Egg shell calcification commonly occurs in silicosis and coal workers pneumoconiosis. Other rare causes are sarcoidosis, post irradiation Hodgkins disease, scleroderma, blastomyces and very rarely amyloidosis and histoplasmosis.[4] Our patient did not have any features suggestive of other known causes of egg shell calcification.

The term broncholithiasis denotes the presence of calcified or ossified material within the lumen of the bronchus. But broncholiths may not be visualised by bronchoscopy in all cases of broncholithiasis. [5]It is now defined as a disorder charecterised by presence of peribronchial calcified lymph nodes which may erode into an adjacent bronchi or distort the bronchi which can lead to symptoms due to bronchial obstruction. Broncholithiasis may cause atelectasis, mucoid impaction, bronchiectasis, expiratory air trapping and recurrent pnuemonia.[5,6] Although very rare even in cases of broncholithiasis, history of lithoptysis must be asked for in any patient with respiratory complaints as it gives an important clue for diagnosing underlying broncholithiasis and thereby helps in reducing fatal complications.

Lithopytsis was first described by Aristotle in 300 BC and later confirmed by Galen and Aretaues. Most of the time it is ignored by patients as well as physicians .IM Samson et al reported a psychiatric patient with lithoptysis and bilateral broncholithiasis who was undiagnosed for twenty years because of disregard of patients symptom by treating physician.[7] Tuberculosis is the commonest cause of broncholithiasis followed by Histoplasmosis and other fungal infections. Other less common causes are calcification of aspirated foreign material and erosion by and extrusion of calcified bronchial cartilage plates. [5,7].Sertolli was the first to describe broncholithiasis in patients with silicosis in 1957. [8]. Since then only a few case reports of silicosis with broncholithiasis have been published .VCS Antao reported a case of silicosis in whom broncholiths were generated from parenchymal nodules rather than from lymph nodes.[9] Broncholith is usually composed of 85-90% calcium phosphate and 10-15% calcium carbonate.[7]Broncholiths that were reported in VCS Antao et al case were composed of 78.92% calcium and 21.09% phosphorus.

Broncholithiasis may cause serious complications like fatal hemoptysis, recurent pneumonia, bronchoesophageal fistula, aortotracheal fistula, pleural fistula and mediastinal abscess. [10,11,12,13,14] Bronchoscopy was not done in our patient as it is a relatively less sensitive technique compared to CT for identification of broncholiths . [6]

Most of the broncholiths can be followed up conservatively. Treatment is indicated only in patients with severe hemoptysis, traction diverticula, carcinoma, recurrent pneumonia or fistula formation. Bronchoscopy is a safe and effective option for loose broncholiths that are not fixed to the airway. Any attempt to remove broncholith that are attached to bronchial wall may result in catastrophic hemorrhage. [5] Lobectomy or segmentectomy is usually required, since removal of the calcied mass will almost certainly result in loss of a portion of the bronchial wall. [16,17,18] Lung function study in our patient showed obstructive pattern which is an expected finding in silicosis. Studies showed that even nonsmokers who are exposed to silica had lung functionimpair ment as severe as that of heaviest smokers.[19] Although there was no parenchymal involvement in our patient , he needed further follow up for progression of disease

Conclusion:

We are reporting this case because of two rare presentations of silicosis in same patient. Silicosis presenting as lithoptysis and broncholithiasis is very rare and only few cases have been reported in literature. Silicosis presenting as bilateral hilar lymphadenopathy without any pulmonary nodules is also very rare. Although there was no parenchymal involvement in our patient , he needed further follow up for to look for further progression of disease and development of nodules.

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