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CASE REPORT **OPEN ACCESS**

Silicosis, fluorosis, tobacco smoking and limited cutaneous systemic sclerosis: A case report

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ABSTRACT
We report a case of a young chronic tobacco smoker having pneumoconiosis due to silica and developed limited scleroderma. Additionally, he had fluorosis and received treatment for tuberculosis.
Key words: Systemic sclerosis, Limited cutaneous systemic sclerosis, CREST, Scleroderma, Flourosis, Silicosis, Tobacco smoking

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INTRODUCTION

Silicosis is an irreversible but preventable occupational lung disease caused by the inhalation of respirable crystalline silica (RCS).[1] Silica, a compound composed of silicon and oxygen. Two forms of silica are found in the earth crust, an abundant crystalline and rare amorphous. The crystalline variety is found mainly as quartz while the other two namely cristobalite and tridymite are less abundant.[2] The disease occurs due to its lengthy exposure in occupations such as mining, concrete manufacturing, sand blasting, glass manufacturing and foundry work.[1,3] The disease may manifest several weeks to years of exposure to silica dust.[1] Only respirable silica particles (size <5µm) are capable of causing the disease as they are able to reach distal airways and alveoli. These particles known as respirable crystalline particles are produced during sand blasting and cutting of rocks.[3]

Systemic sclerosis is a connective tissue disorder due to excessive deposition of extracellular matrix components in different tissues and organs, inflammation and vasculopathy. The causes of systemic sclerosis are not fully understood. Silica, vinyl chloride, epoxy benzene, organic solvents has been implicated in systemic sclerosis. Silica-associated systemic sclerosis also known as 'Erasmus syndrome' is a rare entity in which systemic sclerosis occcurs due to exposure of silica dust in the presence or absence of silicosis.[4] Chronic fluorosis is a condition which mainly is due to prolonged ingestion of fluoride (>1.5 mg/l) in water. The incorporation of fluorine as fluorapatite in calcified tissue is the main cause of skeletal fluorosis and the earliest manifestation being dental mottling.[5]

CASE REPORT

A 28-year-old male patient, a resident of Rajasthan, worked in stone quarries for 15 years without protective gears and drinking ground water presented with progressive increase in breathlessness for six years. He also complained of tightening and discoloration of skin for the last 4 years. At present the patient gets breathless even after walking few steps. There is no dyspnea at rest or while lying down. It is not associated with wheezing. The tightening of the skin was first noticed in fingers of both hands followed by forearms, feet and legs. Simultaneously, he also noticed painful bluish discoloration of fingers of both hands when exposed to cold. Terminal phalanx of index, middle, little finger of right hand and middle finger of left hand were amputated as they turned black. He also has difficulty in swallowing of solid foods and abdominal bloating for 3 years. A gradual weight loss for last 2 years was noticed by him.

He gave history of stiffness, rigidity and restricted movement of the whole spine and joints. He is a chronic smoker (10 bidi/day) but non-alcoholic. There is no history suggestive of photosensitivity, hemoptysis and chest pain.

He received antitubercular treatment for pulmonary tuberculosis 7 years back. He also received 6 cycles of dexamethasone-cyclophosphamide pulse in a tertiary care center 3 years back after being diagnosed as systemic sclerosis without any relief.

The patient at the time of presentation was emaciated and had stiff neck and back. He was unable to bend his neck and back [Figure 1]. The pulse was 70 beats/minute, regular rhythm; blood pressure 90/60 mm of Hg; respiration 18/min and temperature 36.8˚C. He was pale having no icterus, cyanosis, clubbing, lymphadenopathy and organomegaly. Cutaneous examination revealed that skin of face, both forearms, dorsum of hands, both legs, feet was severely bound down. There was microstomia and pinched nose [Figure 2]. Calcinosis cutis were present on dorsum of hands and elbows. Modified Rodnan skin score was 11. Three finger trismus and dental fluorosis was present on oral examination [Figure 3]. The terminal phalanx of index, middle and little finger of right hand and middle finger of left hand were amputated [Figure 4]. Chest was bilaterally symmetrical, with prominent barrel shaped cage. Accessory muscles of respiration were active with intercostal recession. Chest expansion at the level of nipples was 1.8 cm. The vesicular breath sounds and air entry was reduced on both sides without any added sounds. Heart sounds S1 and S2 were normal.

The laboratory tests revealed hemoglobin 9 gm/dl; hematocrit: 34%; leucocyte count 7000/mm^3; erythrocyte sedimentation rate 12 mm/1^{st} hour; antinuclear antibody (ANA) by the method of indirect immunofluorescence on HEp-2 was 1:640 with mixed pattern, Scl-70 was negative. Sputum for *Mycobacterium tuberculosis* and T-spot TB test were negative. Forced vital capacity, total lung capacity was <80% of predicted value and FEV1/FVC was normal. Liver function tests, muscle enzymes and renal function tests were normal. ECG was normal. Chest X- ray postero-anterior view showed loss of thoracic curvature, marginal osteophytes in T9-T12 and interspinous ligament calcification but no changes in lumbar spine [Figure-5]. HRCT showed multiple small nodules, 2-5 mm in diameter predominantly in upper lobe and posterior zone, fibrosis and volume loss in right upper lobe. CT also showed fibrotic patches in left lung apex, additionally had multiple mediastinal, hilar and paratracheal enlarged lymph nodes with peripheral egg shell calcification and mosaic attenuation with interstitial thickening. Barium swallow showed dilated distal esophagus with loss of peristalsis contraction. Skin biopsy showed lymphoplasmacytic infiltrate in dermal collagen bundles, around eccrine glands and paucity of adipocytes around eccrine apparatus. Patient was diagnosed as a case of limited cutaneous systemic sclerosis (CREST).

DISCUSSION

Diagnosis of limited cutaneous systemic sclerosis in this 28-year-old of male was based on typical hide bound skin distal to elbow and knee besides chest and back, presence of Raynaud's phenomena, calcinosis cutis, sclerodactyly, involvement of esophagus, positive ANA and negative ScL 70.

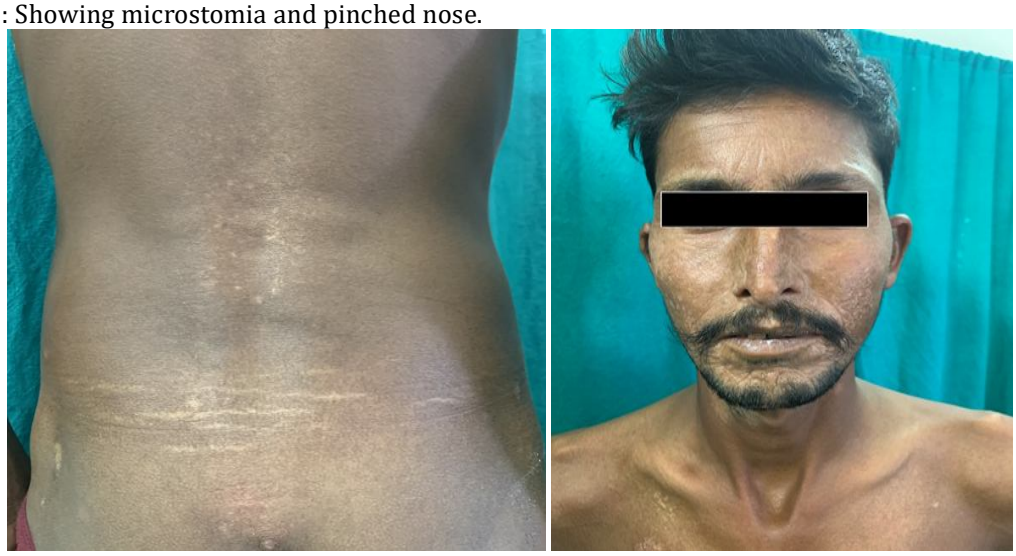
Silicosis was diagnosed on the basis of occupational history of exposure to silica in the stone quarry associated with blasting, cutting, grinding and drilling of stones for over 15 years since early childhood and clinical and radiological finding indicative of the disease, namely the characteristic HRCT finding of multiple small nodules, 2-5 mm in diameter particularly in upper lobe and posterior zone, interstitial thickening and hilar and mediastinal lymphadenopathy with eggshell pattern of calcification. Associated fluorosis was based on history of ground water consumption since childhood, restricted spinal mobility and pain and stiffness and straightening of back and dental mottling.

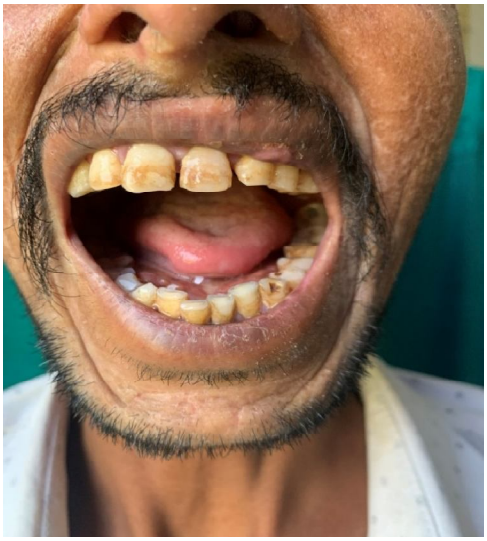
This young man worked in the stone quarries in an atmosphere full of dust arising from blasting and crushing of stones since his early childhood and additionally drank ground water since then.

The atmospheric stone dust full of respirable crystalline silica (size less than 5mm) reaches the respiratory bronchioles and alveoli through inhalation. Then the macrophages phagocytose these particles in order to clear them out but are unable to perform this function. Instead silicotic nodules develop from alveolar macrophages aggregates and due to oxidative stress induced release of liposomal enzymes into the surrounding tissue from their own damage leads to deposition of collagen in concentric pattern at the middle of the lesion. The birefringent silica laden macrophages form cuff around the increasing collage center.[6,7,8]

Further, fluoride has also been reported to have an effect on cardiovascular, respiratory and gastrointestinal system through oxidative stress.[5] Moreover, clinical silicosis has been found to occur more often in smokers than non-smokers inhaling similar quantity of RCS.[2] Thus, the toxic effect of these xenobiotic, SiO2, fluoride and tobacco smoke maybe acting in unison by causing cellular injury via reactive oxygen species and hapten formation. This is resulting in obvious skin and systemic pathology.

Figure 1: Demonstrating emaciation and stiff back along with an inability to bend his neck and back.
Figure 2: Showing microstomia and pinched nose.





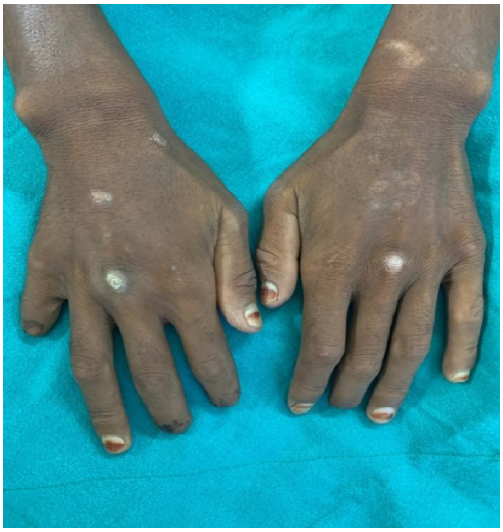


Figure 3: Showing three finger trismus and dental fluorosis on oral examination.
Figure 4: Showing the amputation of terminal phalanx of index, middle and little finger of right hand and middle finger of left hand.

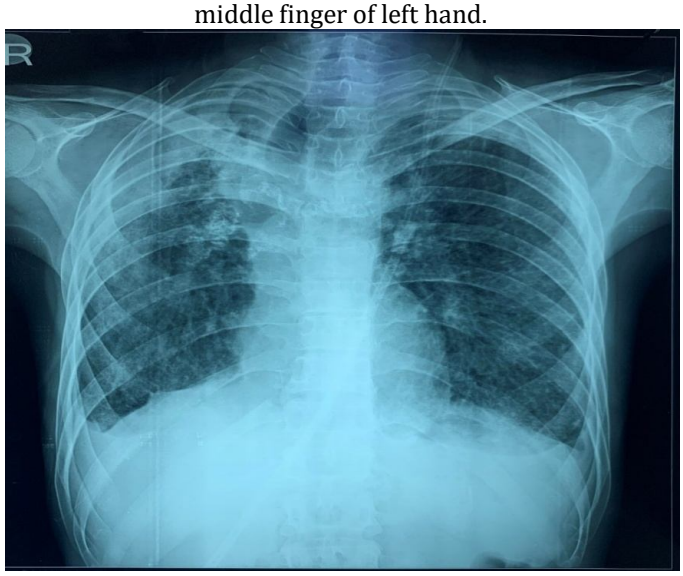


Figure 5: Chest posteroanterior view (PA View) demonstrating loss of thoracic curvature, marginal osteophytes in T9-T12 and interspinous ligament calcification but no changes in lumbar spine.

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