Industrial Health 2011, **49**, 122–125 **Case Report**

# Pericardial Plaque: A Unique Complication of Silicosis

**Iraj MOHEBBI[1]*, Aboulfath LAMEEI[2], Behzad BOOSHEHRI[3], Naser ASLANABADI[4], Reza MAASOMI[5] and Mohammadreza DEHGHANI[6]**

[1]Department of Occupational Medicine, Urmia University of Medical Sciences, Urmia, Iran
[2]Department of Infectious Diseases, Urmia University of Medical Sciences, Urmia, Iran
[3]Department of Anatomy, Urmia University of Medical Sciences, Urmia, Iran
[4]Department of Cardiology, Tabriz University of Medical Sciences, Tabriz, Iran
[5]Martuary Ward, Medicolegal Organization, Urmia, Iran
[6]Department of Cardiology, Urmia University of Medical Sciences, Urmia, Iran

*Received July 28, 2009 and accepted April 6, 2010*
*Published online in J-STAGE September 1, 2010*

**Abstract: The heart and the respiratory system are closely connected in such a way that disorders of one system would influence the function of the other. This unique case of silicosis complicated by corpulmonale associated with pericardial plaque formation in a young adult male is reported here due to an unknown complication of silicosis.**

**Key words:** Silicosis, Lung fibrosis, Plaque, Classification, Oxygen radical

## Case Presentation

We report a young man who died of cardiogenic shock secondary to cardiopulmonary complication of silicosis. 19 yr-old man was admitted in the Occupational Medical Center of Urmia University of Medical Sciences with loss of appetite, weight loss and exertional dyspnea. The patient had worked as a packer in a stone grinding workplace from age 17 for eighteen months. He had immigrated as seasonal workers from West Azarbaijan province (in the west of Iran) to the Azandarian area, a suburb of Hamadan province in the center of Iran. This area is a base for stone-grinding workplaces in Hamadan province. In accordance with the regional Industrial & Mining Organization report, White Quartz is mineralogical compound of the stone which are processed in these workplaces.

On admission, he said that the workplace had poor ventilation. He was non-smoker and had no known exposure to fibers or to asbestos. The patient had no previous history of respiratory symptoms before occupational silica dust exposure. His complaints started six months after he had discontinued -dust exposure. The patient's symptoms started with breathlessness on exertion, malaise, and dry cough. Auscultation of the lungs at first admission revealed bilaterally fine crackles at the end of inspiration. His chest x-ray showed small opacities (p/q) with radiological profusion category 1/2 using the ILO classification system. After a full work-up, and based on findings of history and physical examination acute silicosis was diagnosed. During the next three years he developed orthopnea, paroxysmal nocturnal dyspnea, resting dyspnea, episodic abdominal pain, distended neck veins during inspiration as well as expiration, prominent Jugular vein pulses, pre-systolic gallop sound (S4), significant right ventricular S3 gallop, hepatomegaly, peripheral edema, and ascites. For investigation of pulmonary tuberculosis, at least three sputum specimens were taken annually. All of the samples were negative for acid fast bacilli (AFB –). Latest chest x-ray showed small opacities (r/u) with radiological profusion category 3/3, and large opacity with B scale (Fig. 1) which confirmed by lung HRCT (Fig. 2). Laboratory investiga-

*To whom correspondence should be addressed.
E-mail: irajmohebbi@umsu.ac.ir

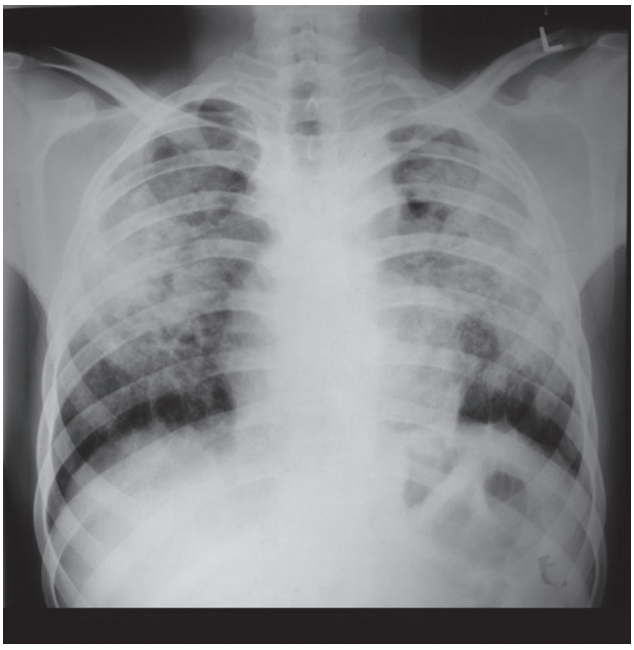

**Fig. 1. Chest radiograph illustrates.**
1) r/u shape size of small opacities. 2) 3/3 scale of small opacities profusion in both lungs, and. 3) large opacity with B scale. 4) Corpulmonale.

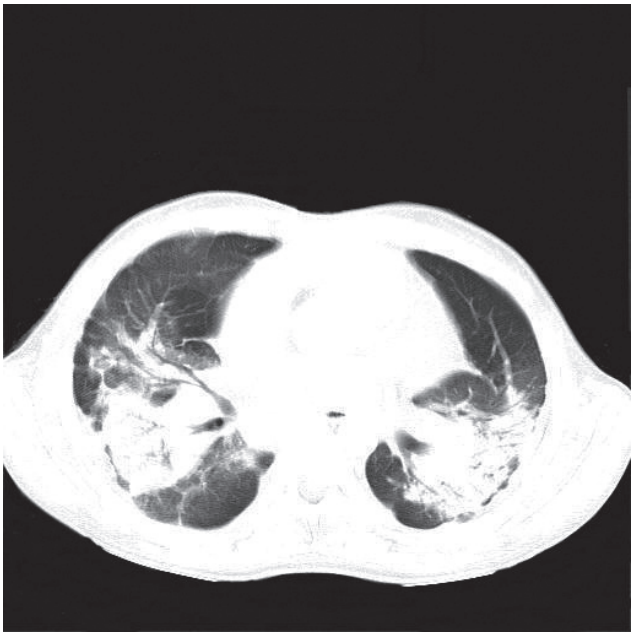

**Fig. 2. HRCT illustrates.**
1) Alveolar pattern with airbronchogram in upper segment of lower lobes. 2) Peripheral bullae. 3) Honey combing. 4) Paraseptal emphysema.

tions for full blood count and basic biochemistry profile were within normal limits. IgG raised to 4,692 mg/dl (normal range=1,034–1,499 mg/dl), C- reactive protein 9.3 mg/l (upper normal value <0.5 mg/ml) and creatine phosphokinase to 474 IU/l (normal=20–190). Parameters of spirometry included: FEV1=1.07 l (28% of predicted), FVC=1.56 l (35% of predicted), FEV1/FVC ratio 83%, and FEF25–75=0.72 l/s (15% of predicted). Diffusing capacity for carbon monoxide (DLCO) was 4.82 mmol/kp/m (46% of predicted). TLC was 3.02 l (51% of predicted) and RV 1.54 (106% of predicted). According to AMA classes of respiratory impairment, spirometry demonstrated very severe functional impairment or class 4.

Transthoracic echocardiography report included: left ventricular ejection fraction=50%, impaired relaxation, trivial mitral regurgitation, both left and right ventricular hypertrophy, severe right ventricular dysfunction, global hypokinesia, severe tricuspid regurgitation, severe pulmonary artery hypertension, tricuspid regurgitation gradient=70 mmHg, pulmonary artery pressure=86 mmHg, right atrial enlargement, inferior vena cava without significant collapse and a moderate degree of pericardial effusion located at the anterior portion of right ventricle.

He died of cardiogenic shock. Autopsy revealed advanced pigmented silicotic masses associated with enlargement of hilar lymph nodes in both lungs (Fig. 3), pericardial effusion of nearly 450 ml, cardiomegaly associated with a smooth irregular plaque on visceral layer of pericardium (Fig. 4), and significant hepatomegaly.

Pathological investigation showed well-developed silicotic nodules in both lungs and histiocytes containing abundant silica particles as revealed under polarized light examination (Figs. 5, 6). Microscopic appearance of the plaque showed the typical basket-weave pattern of collagen (Fig. 7). In autopsy specimens, there was no histopathological evidence of tuberculosis. The PCR test of specimens for tuberculosis was negative.

## Discussion

Silicosis is characterized pathologically by the identification of circumscribed nodules, which consist of whorled zones surrounded by a fibrous tissue. The pathological expressions and clinical forms of silicosis reflect both different exposure intensities and latency periods. Silica particles may be revealed under polarized light microscopic examination[1]. The hypothesis that exposure to silica particles presumably is linked to a wide variety of diseases, has been discussed in the last decade but a few cases of its extra pulmonary complications has been reported[2, 3]. Silica deposition, with or without nodule formation has been reported in the extra pulmonary organs. The nodule formation had been considered in the peritoneum, bone marrow, extrathoracic lymph nodes, liver, and spleen[2]. Haustein *et al*. have declared that silica particles may deposit in the

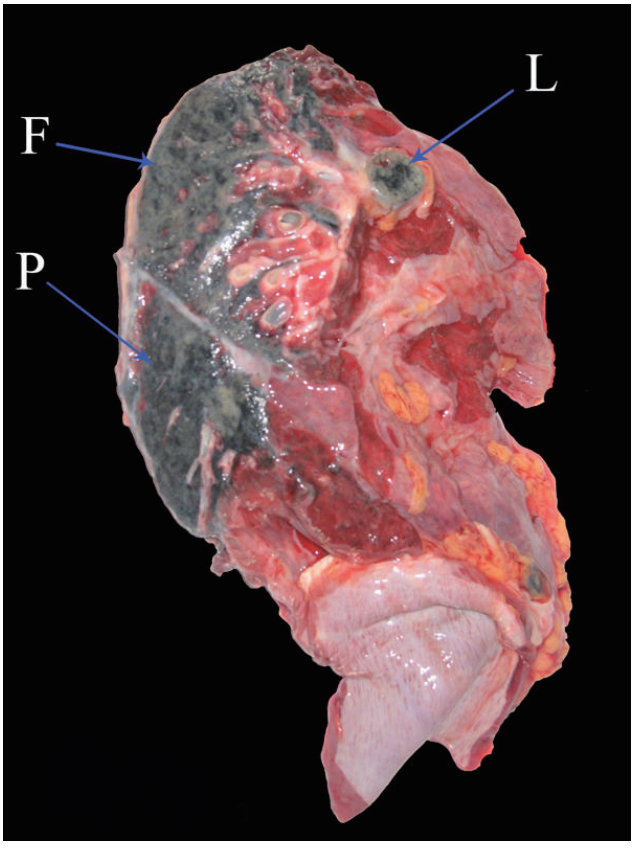


**Fig. 3. There are pigmented masses (P), advanced fibrosis (F), and enlargement of hilar lymph node (L).**

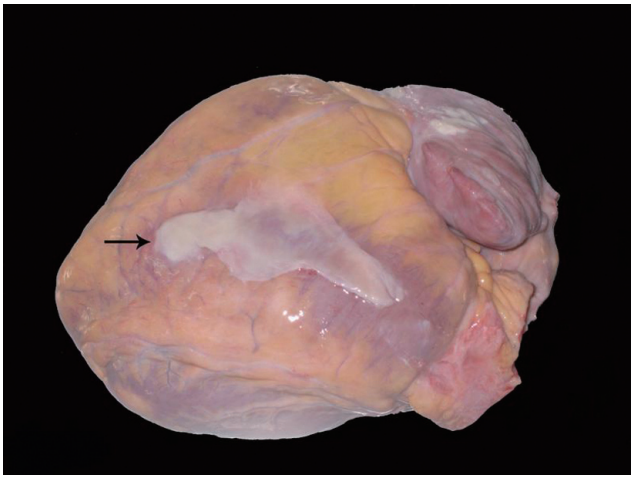

**Fig. 4. Autopsy shows cardiomegaly associated with a pericardial plaque.**

brain, kidney, and skin without nodule formation[4]. On the other hand, autoimmune diseases such as rheumatoid arthritis, lupus erythematosus, scleroderma, and glomerulonephritis are reported to occur more frequently in individuals with silica exposure[5].

It has been observed that silica particles in the select-

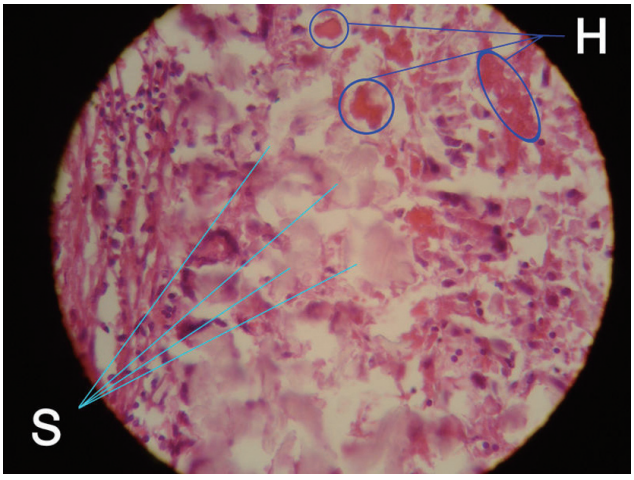


**Fig. 5. Autopsy lung specimen shows hyalinization (H) and silica particles (S).**

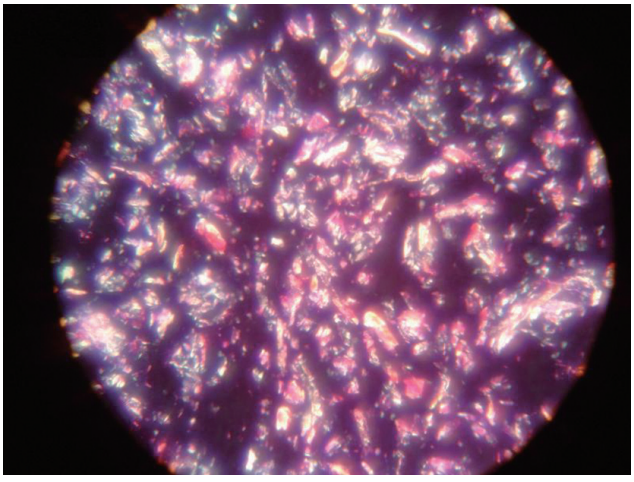

**Fig. 6. Autopsy lung specimen show silica particles as revealed under polarized light.**

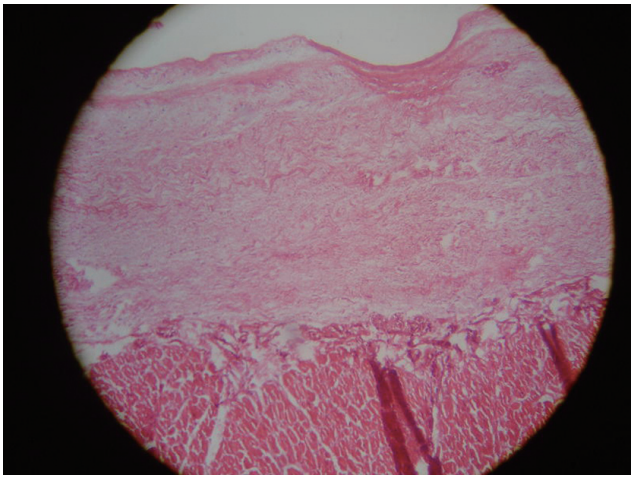

**Fig. 7. Autopsy pericardium specimen shows the typical basket-weave pattern of collagen.**

ed workplaces in the stone-grinding factories had very high concentrations of respirable quartz. The airborne quartz concentration was often much more than maximal permissible exposure level to quartz (0.05 mg/m$^3$)[6–8]. In our case, according to the previous investigation in the stone-grinding factory where the study subject had been employed, the concentration of respirable quartz in the ambient air was 44.80 mg/m[8]. The determination was carried out according to the NIOSH Manual of Analytical Methods, method number 7,500[7]. There have been many workers suffered from silicosis and its complications in this area[9, 10].

Our patient had been employed in stone-grinding factory for 18 months until 2 yr before admission. Considering the clinical findings and exposure period, this case is a rapidly progressive form of silicosis complicated by cor pulmonale. It is not clear how much of the patient's problem was due to right heart failure caused by silicosis and what amount of the problem was related to pericardial tamponade. However the ultimate cause of deterioration of the condition can be due to pericardial involvement leading to tamponade.

Although all of the pulmonary pathological changes of silicosis reported in the literature were observed in our case but to the best of our knowledge, no pathological finding of pericardial plaque had been discussed in the past. The pathological change of pericardium suggests that various pathologic features of extrapulmonary silicosis have still been imperfectly discovered and also the development of extrapulmonary silicosis may be an outcome of the exposure concentration rather than the exposure period.

## Conclusions

This unique case reveals that heavy exposure to silica may lead to further extra pulmonary damages during the development of silicosis. Two main issues are connected with uncertainties in accurately predicting the impact of silica on pericardium.

The first is the lack of understanding of how silica is released to pericardium, and the second is the lack of epidemiological data on the health impact of silica on pericardium. However the exact mechanism underlying the toxicity of silica on pericardium is not clear, plaque formation can be possibly related to accelerate and exacerbate generation of autoantibodies, oxidants, and of inhaled silica translocation via pulmonary capillaries and other mechanisms to systemic sites leading to autoimmune phenomena in pericardium.

## References

1) Banks DE (2005) Silicosis. In: Text book of Clinical Occupational and Environmental Medicine, Rosenstock L, Cullen MR, Brodkin CA and Redlich CA (Eds.), 380–92, Elsevier, Saunders.
2) Miranda RN, McMillan PN, Pricolo VE, Finkelstein SD (1996) Peritoneal silicosis. Arch Pathol Lab Med **120**, 300–2.
3) Slavin RE, Swedo JL, Brandes D, Gonzalez JC, Osorino-Vagas A (1985) Extrapulmonary silicosis: a clinical, morphologic, and ultrastructural. Study Hum Pathol **16**, 393–412.
4) Haustein VF, Ziegler V, Hermann K, Melhorn J, Schmidt C (1990) Silica-induced scleroderma. J Am Acad Dermatol **22**, 444–8.
5) Cooper GS, Miller FW, Germolec DR (2002) Occupational exposures and autoimmune diseases. Int Immunopharmacol **2**, 303–13.
6) Mohebbi I, Abdi Rad I (2007) Secondary spontaneous pneumothorax in rapidly progressive forms of silicosis: characterization of pulmonary function measurements and clinical patterns. Toxicol Ind Health **23**, 125–32.
7) American Conference Governmental Industrial Hygienists (2000) Threshold limit value for chemical substances and physical agents and biological exposure indices. ACGIH, Cincinatti.
8) Mohebbi I, Hassani E, Salarilak S, Bahrami AR (2007) Do bullae and emphysema increase risk of pneumothorax in silicosis? J Occup Med Toxicol **2**, 8.
9) Mohebbi I, Zubeyri T (2007) Radiological progression and mortality among silica flour packers: a longitudinal study. Inhal Toxicol **12**, 1011–7.
10) Mohebbi I, Aslanabadi N, Booshehri B, Zubeyri T, Ghavam F (2007) Rapidly progressive silicosis. Tanaffos **2**, 73–6.