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Chronic Silicosis Presenting With Severe Pulmonary Hypertension And Right Heart Failure: A Case Report Highlighting Late Vascular Complications And Diagnostic Delay.

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Abstract
Chronic silicosis is a progressive fibrotic lung disease caused by prolonged inhalation of crystalline silica dust, commonly seen in individuals with occupational exposure such as stone-cutters and miners[1]. While pulmonary fibrosis is well recognized, its vascular complications–especially pulmonary hypertension (PH)–are underappreciated and often misdiagnosed as chronic obstructive pulmonary disease (COPD) or idiopathic pulmonary arterial hypertension (IPAH)[2,3]. We report a case of a 56-year-old male with a significant occupational history of silica exposure who presented with progressive dyspnea, peripheral edema, and right heart failure. He had been misdiagnosed with COPD for over a year and showed minimal response to inhaled therapies. High-resolution CT of the chest revealed bilateral upper lobe nodular opacities and progressive massive fibrosis, consistent with silicosis[2]. Right heart catheterization confirmed pre-capillary pulmonary hypertension [4,5]. The case underscores the importance of occupational history, imaging, and early screening for vascular complications in fibrotic lung diseases [6]. It also highlights how misdiagnosis may lead to delayed treatment and worsened outcomes. In regions with high silica exposure, clinicians must consider silicosis as a differential diagnosis in patients with unexplained PH or right heart failure [9]. Early recognition and management may improve quality of life and prognosis in such patients.
Keywords: Silicosis, pulmonary hypertension, right heart failure, occupational lung disease, diagnostic delay, COPD mimic

INTRODUCTION

Silicosis is a chronic, irreversible fibrotic pneumoconiosis caused by prolonged inhalation of crystalline silica particles, commonly encountered in occupations such as mining, quarrying, construction, and stone masonry[1,9]. Chronic silicosis typically develops after 10–30 years of exposure and manifests radiologically as upper lobe predominant nodular fibrosis, often progressing to progressive massive fibrosis (PMF)[2]. Emerging evidence suggests that pulmonary vascular involvement, particularly pulmonary hypertension (PH), represents a significant yet underrecognized complication of advanced silicosis[3,6]. PH may result from multifactorial mechanisms, including fibrotic obliteration of the capillary bed, hypoxic vasoconstriction, and inflammatory vascular remodeling[3,6]. Despite these findings, PH in silicosis remains underdiagnosed due to symptom overlap with other respiratory disorders such as chronic obstructive pulmonary disease (COPD) or idiopathic pulmonary arterial hypertension (IPAH)[3,7]. This case report highlights a diagnostically challenging presentation of silicosis-associated PH and right heart failure that was initially misattributed to COPD, underscoring the importance of occupational history and advanced cardiopulmonary assessment[9].

CaseReport

A 56-year-old male, lifetime non-smoker, presented with progressive exertional dyspnea (mMRC Grade

III), orthopnea, paroxysmal nocturnal dyspnea, fatigue, and bilateral pedal edema for the past year. His symptoms had gradually worsened despite adherence to inhaled bronchodilator therapy prescribed under the presumptive diagnosis of COPD.Occupational history revealed 28 years of silica dust exposure while employed as a stone cutter without use of personal protective equipment. There was no history of connective tissue disease, chronic thromboembolic events, or family history of pulmonary hypertension.

On examination:

*Vital signs : HR 98/min, BP 122/76 mmHg, RR 22/min, SpO_2 92% on room air
*Neck veins : Elevated jugular venous pressure (5 cm above sternal angle)
*Cardiac : Loud and palpable P2 component of the second heart sound
*Abdominal : Hepatomegaly (4 cm below costal margin), ascites
*Extremities : Bilateral pitting pedal edema
* Respiratory : Fine late inspiratory bibasal crackles, no wheeze

Differential diagnoses considered included: COPD, idiopathic pulmonary fibrosis with secondary PH, connective tissue disease-related interstitial lung disease (CTD-ILD), and chronic thromboembolic pulmonary hypertension (CTEPH).

Investigations

Chest X-ray :

* Bilateral reticulonodular opacities predominantly in upper lobes
* Volume loss and traction bronchiectasis

High-Resolution CT (HRCT) Thorax :

* Numerous centrilobular and perilymphatic nodules in upper lobes
* Progressive massive fibrosis with upper lobe conglomerate masses
* Calcified mediastinal and hilar lymph nodes (eggshell pattern)
* No evidence of pulmonary embolism

Pulmonary Function Tests (PFTs) :

* FVC: 52% predicted
* FEV_1: 61% predicted
* FEV_1/FVC: 88% (restrictive pattern)
* DLCO: 38% predicted (markedly reduced gas exchange)

Echocardiography :

* Right atrial and right ventricular dilatation
* Interventricular septal flattening ("D-shaped" left ventricle)
* Moderate tricuspid regurgitation
* Estimated pulmonary artery systolic pressure (PASP): 75 mmHg

NT-proBNP : 2580 pg/mL (elevated)

Right Heart Catheterization :

* Mean pulmonary artery pressure (mPAP): 48 mmHg
* Pulmonary capillary wedge pressure (PCWP): 12 mmHg
* Pulmonary vascular resistance (PVR): 6.2 Wood units

6-Minute Walk Test :

* Distance: 230 meters
* Minimum oxygen saturation: 84%

Autoimmune Work-up : ANA, RF, ANCA all negative

Final Diagnosis : Chronic fibrotic silicosis complicated by severe pre-capillary pulmonary hypertension (Group 3 PH) and right heart failure (cor pulmonale).

Management

A multidisciplinary strategy was implemented:

Pharmacotherapy

* Tadalafil 40 mg once daily (PDE-5 inhibitor for pulmonary vasodilation)

* Furosemide 40 mg and Spironolactone 25 mg once daily (volume management)
* Long-term oxygen therapy : Prescribed 15+ hours/day
* Inhaled bronchodilators were tapered as no obstructive component was evident
* Calcium channel blockers were avoided due to risk of worsening ventilation/perfusion (V/Q) mismatch

Supportive Measures

* Sodium restriction and daily weight monitoring
* Pulmonary rehabilitation program with aerobic conditioning and breathing exercises
* Influenza and pneumococcal vaccinations administered
* Counseling on dust exposure avoidance and occupational safety

Follow-up Plan

* Serial echocardiography and 6-minute walk tests every 3 months
* Reassessment of NT-proBNP and DLCO
* Referral to a pulmonary hypertension specialty center for consideration of advanced therapy or lung transplantation if indicated

DISCUSSION

Silicosis-related pulmonary hypertension is increasingly recognized, particularly in patients with progressive massive fibrosis (PMF). Pathophysiologic mechanisms include destruction of the pulmonary capillary bed, hypoxic vasoconstriction, and inflammatory vascular remodeling. Studies suggest that 20–40% of patients with advanced pneumoconiosis may develop PH, which significantly worsens prognosis. The lack of correlation between spirometry and PH severity—as seen in this patient—emphasizes the importance of right heart catheterization in unexplained dyspnea. Unlike PH in COPD, PH in silicosis is more closely linked with vascular remodeling than bronchial obstruction. Current management focuses on symptom control, oxygen therapy, and judicious use of vasodilators. Evidence on long-term efficacy of targeted PH therapy in silicosis is limited, although select patients with severe pre-capillary PH and preserved ventilation-perfusion matching may benefit. Preventive strategies, early detection, and occupational surveillance are critical. This case also highlights the consequences of misdiagnosis, especially in areas with limited resources and low awareness of occupational lung diseases. A detailed occupational history and high-resolution imaging are vital in such scenarios.

CONCLUSION

This case underscores the severe vascular complications that can arise in chronic silicosis, particularly pre-capillary pulmonary hypertension and right heart failure. A high index of suspicion is needed to distinguish it from more common respiratory diagnoses like COPD. Integration of clinical, radiologic, and hemodynamic data is key to early diagnosis and management. Increased awareness and occupational health vigilance are essential in preventing and addressing such complications.

Patient Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying clinical images.

Conflicts of Interest

The authors declare no conflict of interest.

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REFERENCES

1. Balakrishnan K, et al. Silicosis and its vascular complications. Lung India. 2021;38(5):395–400.
2. Leung CC, Yu IT, Chen W. Silicosis. Lancet. 2012;379(9830):2008–18.
3. Chan MH, et al. Pulmonary hypertension complicating pneumoconiosis. Chest. 2002;121(4):1269–73.

4. Simonneau G, et al. Haemodynamic definitions and updated classification of pulmonary hypertension. Eur Respir J. 2019;53(1):1801913.
5. ATS/ERS Task Force. Guidelines for the diagnosis and treatment of pulmonary hypertension. Am J Respir Crit Care Med. 2015;192(3)\:e18–e63.
6. Huerta C, et al. Pulmonary hypertension due to chronic fibrotic lung disease. Respiration. 2016;91(2):135–140.
7. Goh NS, et al. Pulmonary hypertension in interstitial lung diseases: Role of HRCT. Thorax. 2008;63(3):273–278.
8. Rosenberg DM, et al. Pneumoconiosis and occupational lung disease. Med Clin North Am. 1996;80(4):839–857.
9. Pinto LM, et al. Occupational lung diseases: Diagnosis and prevention. Indian J Occup Environ Med. 2018;22(1):1–6.
10. Mandal AK, et al. Silica exposure and silicosis among Indian stone cutters. J Occup Health. 2014;56(6):467–472.