BMC Pulmonary Medicine

CASE REPORT

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Constrictive pericarditis in a patient with fiberglass lung disease: a case report

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Abstract

Background Fiberglass has a larger aerodynamic diameter and is less likely to be inhaled into the lungs. Further, it will be cleared even if it is mechanically broken into smaller pieces and inhaled into the lungs. Fiberglass lung disease has been well documented if long term exposure but was thought reversible and would not cause severe diseases. The diagnosis of fiberglass lung disease depends on exposure history and histopathological findings. However, the exact occupational exposure history is often difficult to identify because mixed substance exposure often occurs and fiberglass disease is not as well-known as asbestosis.

Case presentation A 66-year-old man had unexplained transudative pericardial effusion requiring pleural pericardial window operation twice at another medical center where asbestosis was told because of his self-reported long-term asbestosis exposure and the histopathological finding of a ferruginous body in his lung. Constrictive pericarditis developed two years later and resulted in congestive heart failure. Radical pericardiectomy combined with lung biopsy was performed following chest computed tomography imaging and the transudative nature of pericardial effusion not compatible with asbestosis. However, the histopathologic findings of his lung and pericardium at our hospital only showed chronic fibrosis without any asbestosis body. The patient's lung was found to be extremely fragile during a lung biopsy; histopathologic specimens were reviewed, and various fragments of fiberglass were found in the lung and pericardium. The patient's occupational exposure was carefully reevaluated, and he restated that he was only exposed to asbestosis for 1–2 years but was heavily exposed to fiberglass for more than 40 years. This misleading exposure history was mainly because he was only familiar with the dangers of asbestos. Since most fiberglass lung diseases are reversible and the symptoms of heart failure resolve soon after surgery, only observation was needed. Ten months after radical pericardiectomy, his symptoms, pleural effusion, and impaired pulmonary function eventually resolved.

Conclusion Fiberglass could cause inflammation of the pericardium, resulting in pericardial effusion and constrictive pericarditis, which could be severe and require radical pericardiectomy. Exact exposure history and histopathological examinations are the key to diagnosis.

Keywords Asbestosis body, Constrictive pericarditis, Fiberglass lung disease, Pericardial effusion, Radical pericardiectomy

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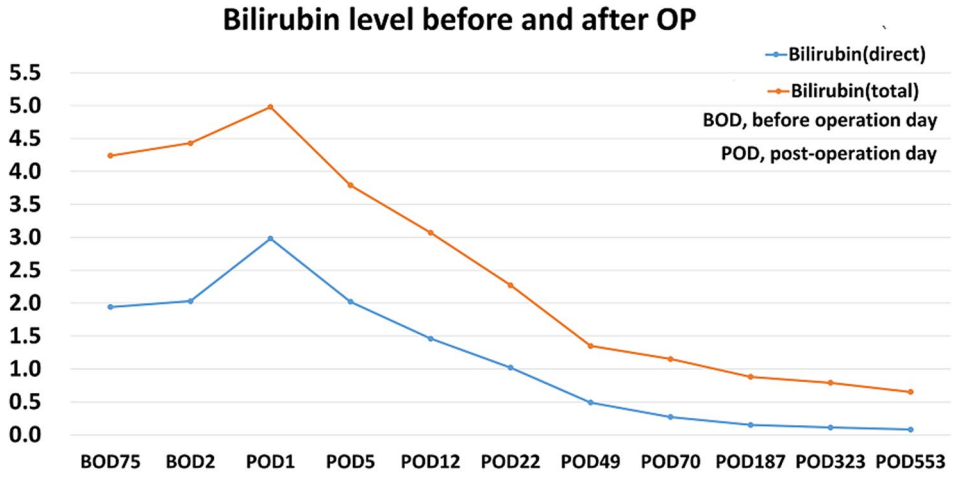

Fig. 1 Liver functional tests. The bilirubin level showed improvement after radical pericardiectomy

Background

Fiberglass with a larger aerodynamic diameter is less likely to be inhaled into the lungs [1]. Even if it mechanically breaks to a smaller fraction to be inhaled into the lungs, it will be resolved and cleared rapidly and was previously thought not to cause chronic lung disease [1, 2]. However, fiberglass lung disease occurs in cases of long-term exposure but is considered reversible and not causative of severe diseases [3]. The diagnosis of fiberglass lung disease depends on the exposure history to fiberglass and the histopathological findings. However, occupational material exposure and inhalation history are often difficult to identify, and exposure to mixed substances is often observed. Fiberglass is not well known for causing occupational lung disease. Herein, we report a case of fiberglass lung causing lung fibrosis and constrictive pericarditis after 40 years of exposure.

Case presentation

On July 30, 2021, a 66-year-old male ex-smoker who worked as a medical equipment operator and self-reported heavy exposure to asbestos for 40 years presented at our hospital with massive transudative pericardial effusion, which was treated with two pleural–pericardial windows at another hospital in March 2020. During the second pleural–pericardial window, a lung biopsy was performed, and his final histopathological diagnosis was considered asbestosis. However, his dyspnea and leg edema gradually worsened over the subsequent 18 months.

Cardiac catheterization confirmed constrictive pericarditis on September 15, 2021. Given the poor response to diuretics and progressive impairment of liver function due to congestive liver (Fig. 1), the patient was admitted on October 11, 2021.

Upon admission, chest computed tomography (CT) scan revealed bilateral pleural effusion, pericardial effusion, pericardial thickening, thickened intra- and inter-lobular lines, subpleural fibrosis, and parenchymal fibrosis bands. However, no obvious pleural calcification or plaque, which are typical signs of asbestosis, was observed (Fig. 2A). Thoracentesis revealed a transudative pleural effusion without evidence of mesothelioma. Pulmonary function tests revealed a restrictive pattern. Accordingly, the patient underwent radical pericardiectomy combined with an open lung biopsy on October 13, 2021. The surgeons noted that the pericardium was as hard as corrugated paper and was firmly attached to the epicardium; additionally, his lungs were very fragile. Lung pathology revealed mild interstitial fibrosis of the lung and pleura; however, there were no asbestos or

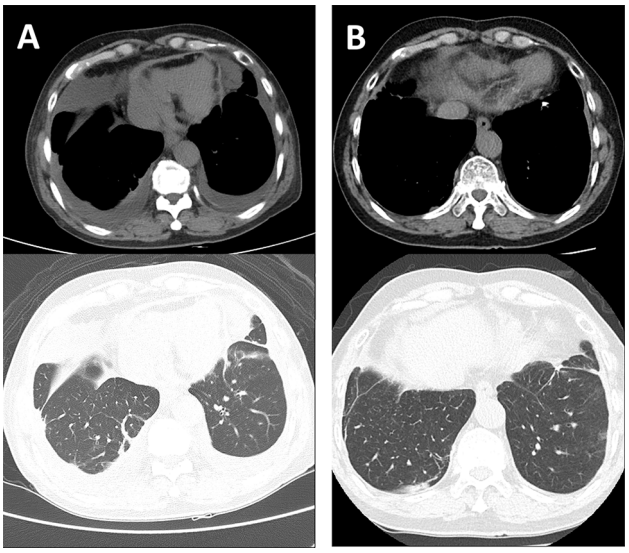

Fig. 2 HRCT imaging. Chest computed tomography showed some calcified granuloma within the pleura in the left lower lobe, as well as bilateral pleural effusion with some linear consolidation and a small amount of pericardial effusion (**A**). Pleural effusion, pericardial effusion, and linear consolidation improved after surgery (**B**)

ferruginous bodies (Fig. 3A and B). Polarized microscopy revealed various translucent particles in the lung parenchyma (Fig. 3C). Pathology examination of the pericardium revealed an extremely thickened pericardium and epicardium with a few tiny translucent particles (Fig. 4). Re-examination of pathological specimens from the other hospital only revealed one ferruginous body but no asbestos bodies (Fig. 5A). Polarized microscopy revealed translucent particles of various sizes (Fig. 5B). All tests for autoimmune diseases, tuberculosis, and fungi were negative. After discussion, the translucent particles were hypothesized to be fiberglass. Therefore, the patient's occupational exposure history was re-examined in detail. He reported that he was only slightly exposed to asbestos during the first 3 years, but was heavily exposed to fiberglass in the past 40 years. Finally, the patient was diagnosed with fiberglass lung disease but not asbestosis. Ten months after radical pericardiectomy, his symptoms, pleural effusion, and pulmonary function eventually resolved (Fig. 2B).

Discussion and conclusion

Compared with asbestos, fiberglass has a larger aerodynamic diameter and is less likely to be inhaled into the lungs [1]. Even if it is mechanically broken down into smaller fractions and inhaled into the lungs, it is usually resolved and cleared rapidly [1]. Fiberglass does not cause severe diseases unless there is long-term exposure [3]. To our knowledge, the present case is the first reported case of fiberglass-associated constrictive pericarditis. The underlying mechanism may involve fiberglass translocation to the distal airways and subsequent migration to interstitial storage sites or along lymphatic drainage pathways [1]. The diagnosis of fiberglass lung disease was delayed owing to a misleading self-reported history of asbestos exposure. The patient's failure to report his exposure to fiberglass may be attributed to the lack of awareness regarding fiberglass lung disease compared with asbestosis. Government public health departments should broadly target these workers and instrument manufacturers to manage and prevent occupational lung diseases.

The diagnosis of asbestosis is based on an exposure history of >20 years and histologic evidence of ≥2 asbestos bodies/cm^2 within a 5-μm-thick lung section either lying freely in the air spaces or embedded in the fibrotic interstitium [4]. Asbestos bodies usually appear in droves in asbestosis; however, fewer asbestos bodies do not exclude asbestosis, and subsequent mineral analysis is warranted to confirm or refute the diagnosis of asbestosis [4]. Ferruginous bodies, which are different from asbestos bodies, do not always indicate asbestosis [4]; however, they are not observed in fiberglass lung disease [5]. The observed single ferruginous body in our patient's lungs might have been a pseudo-asbestos body resulting from very little mixed exposure to other materials in the past 40 years

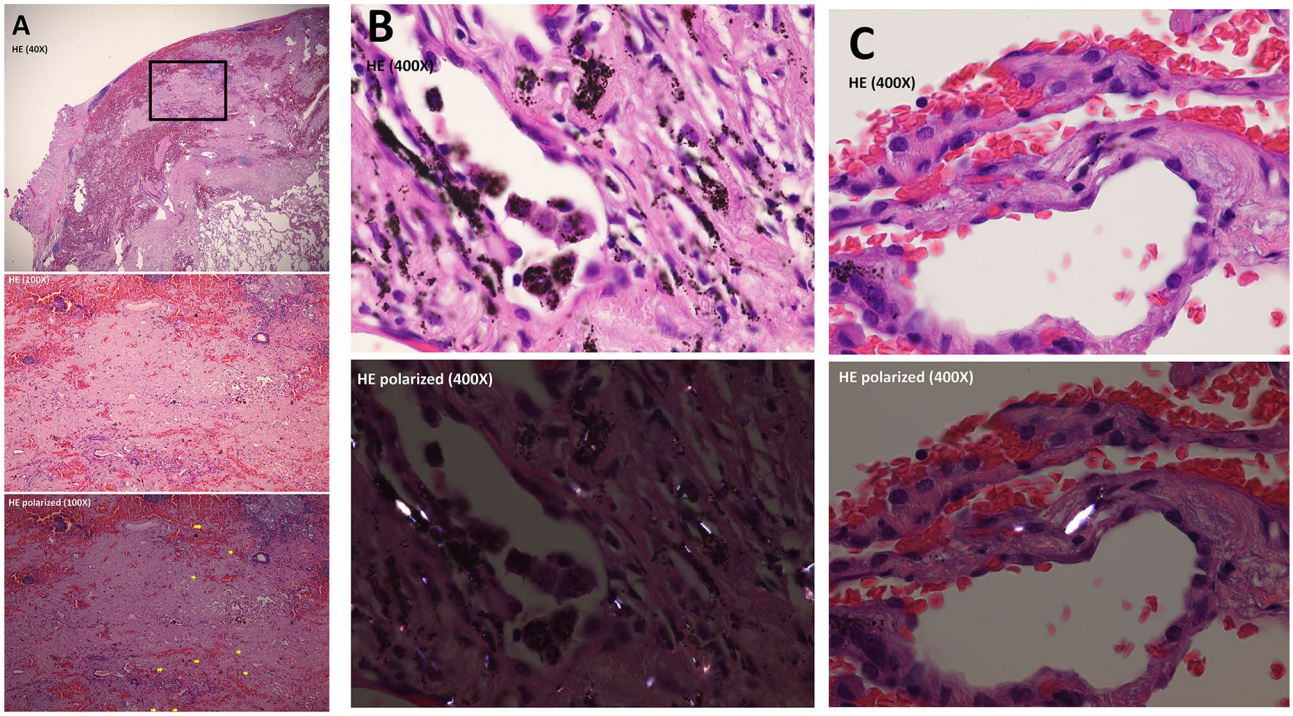

Fig. 3 Pathological examination of the lung and pleura. Lung pathology showed mild interstitial fibrosis of the lung and pleura; however, no asbestos or ferruginous bodies were found (**A**). Some fragments of fiberglass were observed in the lung parenchyma under polarized microscopy (**A**, **B**, and **C**)

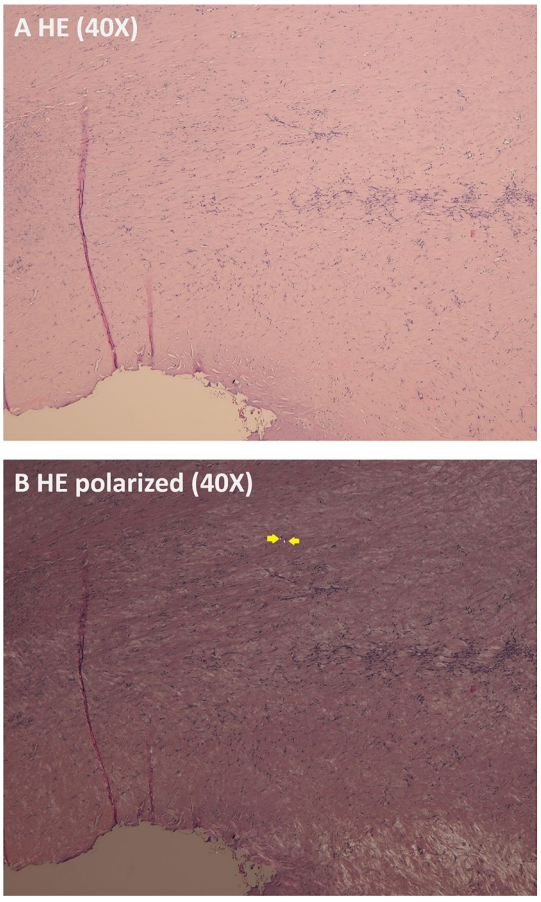

Fig. 4 Pathology of pericardium. Pathology examination of the pericardium showed an extremely thickened pericardium (**A**) and an epicardium with a few tiny fragments of fiberglass (**B**)

[6]. The diagnosis of asbestosis in our case was refuted by the very short history of asbestos exposure and the presence of only one ferruginous body. Asbestosis usually progresses over time [4]; contrastingly, fiberglass lung disease usually shows recovery, which is consistent with the clinical course of our patient.

Characteristic chest CT findings of asbestosis-related constrictive pericarditis usually reveal dense calcification in the pericardium associated with lung fibrosis. Asbestosis-associated pleural or pericardial effusion can be exudative or bloody. Our patient did not present dense calcification in the pericardium or exudative/bloody pleural effusion; therefore, we could reasonably rule out a diagnosis of asbestosis and avoid lung biopsy. However, given the patient's misleading self-reported history of extended asbestos exposure, it was initially difficult to rule out asbestosis. Clinicians should be familiar with the radiological and clinical characteristics of this condition.

In conclusion, fiberglass can cause inflammation of the pericardium, causing pericardial effusion and subsequently constrictive pericarditis, which is severe, requiring radical pericardiectomy. Careful review of exposure history and histopathological examinations are important for clinicians for exact diagnosis.

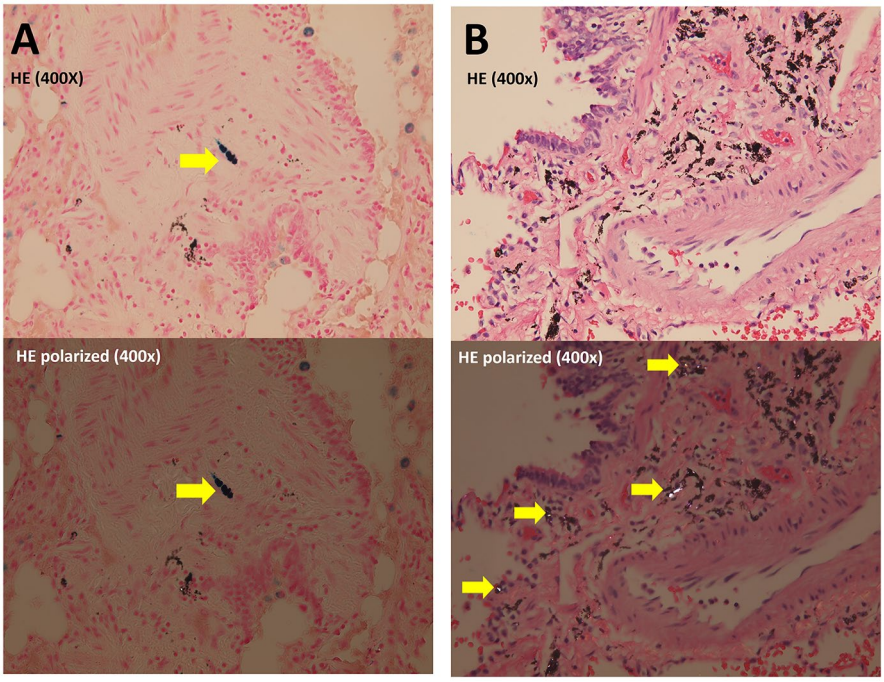

Fig. 5 Pathology of the lung specimen obtained from the other hospital. Pathological specimens from the other hospital showed only one ferruginous body and no asbestos bodies (**A**). We found various fragments of fiberglass by polarized microscopy analysis (**B**)

Acknowledgements
We would like to thank Editage (https://app.wk.editage.com/) for English language editing.

Author contributions
CYH and CCL designed and conducted the work. CYH, HYC and MSH collected the data. YHL and HYC obtained and prepared the histopathological figures. CYH and CCL prepared the draft, and MCY critically reviewed for important intellectual content. All authors read and final approved the manuscript and agreed to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Funding
This report was supported by the Taipei Tzu Chi Hospital (TCRD-TPE-108-RT-3) for the English language editing and the publication fee. The funding body did not have a role in study design, data collection and reporting.

Data availability
The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to publication
The Institutional Review Board of Taipei Tzu Chi Hospital approved this report (IRB No: 12-CR-094) on October 16, 2023. The patient provided written informed consent for the publication of his personal and clinical details, along with de-identified images.

Competing interests
The authors declare no competing interests.

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Received: 21 May 2024 / Accepted: 30 July 2024
Published online: 05 August 2024

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