Pulmonary hypertension associated with combined fibrosing mediastinitis and bronchial anthracofibrosis: A retrospective analysis in a single Chinese hospital

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Abstract

Introduction: Both fibrosing mediastinitis (FM) and bronchial anthracofibrosis (BAF) are unique diseases. The combined appearance of FM and BAF is extremely rare.

Objectives: The aim of this study was to investigate the clinical features of patients with coexisting FM and BAF.

Method: Between January 2003 and December 2015, a total of eight patients were diagnosed at the Peking Union Medical College Hospital as having combined FM and BAF. The clinical presentations, radiographic features and bronchoscopic findings of the eight patients were reviewed.

Results: The patients were five women and three men with a median age of 64 years (range 56-86 years). Symptoms included dyspnea (eight patients), cough (seven patients), chest pain (two patients), hemoptysis (two patients) and so on. Chest CT of all eight patients showed mediastinal soft-tissue lesions, with multiple narrowed or obliterated lobar or segmental bronchi and arteries. Bronchoscopy showed that all of the patients had multiple stenoses of lobar or segmental bronchi with anthracotic pigmentation on the mucosa. Echocardiography showed that all of the patients had elevated pulmonary arterial systolic pressure (median 81 mm Hg, range 51-107 mm Hg). Each of the eight patients had a history of exposure to, or infection with, tuberculosis, although there was no evidence of active disease. All of the eight patients had long-term exposure to indoor coal or biomass fuel smoke.

Conclusions: FM can coexist with BAF, characterized by prominent pulmonary hypertension. The possible etiological factors are tuberculosis and coal or biomass fuel exposure.

KEYWORDS
biomass fuel, bronchial anthracofibrosis, fibrosing mediastinitis, pulmonary hypertension, tuberculosis

1 | INTRODUCTION

Fibrosing mediastinitis (FM) is a clinical syndrome caused by abnormal proliferation of fibrous tissue in the mediastinum that encases or compresses mediastinal structures. FM is most commonly associated with infectious diseases such as Histoplasma capsulatum infection, tuberculosis (TB), aspergillosis, actinomycosis and mediastinal purulent diseases. Other causes include sarcoidosis, Behçet disease, mediastinal radiotherapy, exposure to asbestos, and...
immunoglobulin G4-related disease. FM is a rare cause of pulmonary hypertension (PH). Secondary to FM is classified as group 5 PH (i.e., PH with unclear and/or multifactorial mechanisms). The possible mechanism of PH in patients with FM might be extrinsic compression of the pulmonary arteries.

Bronchial anthracofibrosis (BAF) is a distinct entity characterized by the bronchoscopic findings of multiple areas of anthracotic pigmentation on airway mucosa with bronchial narrowing or obliteration. Although chronic exposure to biomass fuel smoke is now considered to be the most probable causative factor of BAF, a strong association between BAF and TB has also been observed. Each of these diseases is rare. Combined BAF and FM are extremely rare. To date, the only case in the literature was reported by Boonsarngsuk et al. Their patient had coexisting bronchial anthracotic stenosis and mediastinal fibrosis caused by chronic inhalation of wood smoke.

Here, we report a series of patients with coexisting FM and BAF, with PH as a prominent feature.

2 | MATERIALS AND METHODS

2.1 | Patients

After receiving institutional review board approval, we performed a retrospective search of the medical record system of Peking Union Medical College Hospital using the key words “fibrosing mediastinitis” and “mediastinal fibrosis.” Between January 2003 and December 2015, a total of 12 patients were identified as having FM. Among them, 10 patients also underwent bronchoscopy, and eight of them were identified as having BAF as well.

2.2 | Diagnosis criteria

The diagnostic criterion for FM was that chest computed tomography (CT) showed infiltrative mediastinal lesions and associated vascular, airway and/or esophageal compression, after excluding malignancy as a cause. The diagnostic criterion for BAF was that bronchoscopically revealed anthracotic pigmentation associated with narrowing or obliteration of the bronchi. PH was defined as an estimated pulmonary artery systolic pressure (PASP) > 50 mm Hg and the Peak tricuspid regurgitation velocity > 3.4 m/s, as assessed by transthoracic echocardiography, which suggests high probability of PH according to ESC/ERS PH guideline.

2.3 | Methods

The patients’ medical records were then reviewed for clinical symptoms and signs, exposures, laboratory findings and outcomes. The CT and bronchoscopic data were reviewed. Data were expressed as the median (range).

This study was approved by Peking Union Medical College Hospital Institutional Review Board. Informed consent for publication of the clinical information was obtained from the patient at the time of diagnosis or follow-up.

3 | RESULTS

3.1 | Clinical characteristics

The clinical characteristics of the eight patients (five women, three men) were shown in Table 1. The patients’ median age was 64 years (56-86 years). Only one patient had a history of smoking (45 pack-years). Symptoms included dyspnea in eight patients, cough in seven patients, chest pain in two patients, hemoptysis in two patients, hoarseness in one patient, and peripheral edema in one patient. One patient presented with dry mouth and eyes secondary to Sjögren syndrome.

3.2 | Radiological features

Chest CT and CT pulmonary angiography (CTPA) were performed on all eight patients at the initial evaluation. The radiological features of the patients were summarized in Table 2.

| Variable | Results |
|----------|---------|
| Age (years) | 64 (56-86) |
| Sex (M/F) | 3/5 |
| Non-smoker | 7 |
| Dyspnea | 8 |
| Cough | 7 |
| Chest pain | 2 |
| Hemoptysis | 2 |
| Hoarseness | 1 |
| Peripheral edema | 1 |
| PASP (mm Hg) | 81 (51-107) |
| PaO₂ (mm Hg) | 65.1 (52.7-84.0) |
| PaCO₂ (mm Hg) | 36.5 (34.6-58.0) |
| Follow-up (months) | 39.1 (11.7-96.0) |

Results are given as the median (range) or the number of patients unless otherwise specified.

Pulmonary artery systolic pressure (PASP) was estimated by echocardiography.
and multiple distributions. Calcifications were detected in six patients. Commonly involved sites included the following: All eight patients had involvement of the subcarinal region (station 7), right hilar region (station 10R), left hilar region (station 10L) and right interlobar region (station 11R). Seven patients had involvement of the aortopulmonary window region (station 5) and left interlobar region (station 11L). Six patients had involvement of the right lower paratracheal region (station 4R); three patients had involvement of the left lower paratracheal region (station 4L); and two patients had involvement of right lobe lymph node region (station 12R).

### Imaging findings for the eight patients studied

| Variable                                           | No. of patients |
|----------------------------------------------------|-----------------|
| Mediastinal mass                                   | 8               |
| Right/left lower paratracheal region (station 4R/4L)| 6/3             |
| Aortopulmonary window region (station 5)           | 7               |
| Subcarinal region (station 7)                      | 8               |
| Right/left hilar region (station 10R/10L)         | 8/8             |
| Right/left interlobar region (station 11R/11L)     | 8/7             |
| Right lobe lymph node region (station 12R)         | 2               |
| Calcifications                                     | 6               |
| Airway stenosis                                    | 8               |
| Arterial stenosis                                  | 8               |
| Right/left upper lobe artery                       | 5/3             |
| Right middle lobe/lingual artery                   | 4/5             |
| Right/left interlobar artery                       | 4/5             |
| Right/left lower lobe artery                       | 7/4             |
| Parenchymal findings                               |                 |
| Irregular linear opacities                         | 7               |
| Ground-glass opacities                             | 5               |
| Atelectasis                                        | 2               |
| Thickening of interlobular septa                   | 2               |
| Subpleural scar opacities                          | 2               |
| Consolidations                                     | 2               |
| Nodules with calcification                         | 5               |

3.3 | Bronchoscopic features

Bronchoscopy revealed that all eight patients had anthracotic pigmentation of the bronchial mucosa and luminal narrowing of lobar or segmental bronchi (Figure 2). The lesions were distributed bilaterally (Table 3). The bronchial mucosa was biopsied in one patient. The specimen contained fibrotic collagenous material, chronic inflammation and anthracotic pigmentation in the bronchial submucosa. The washing and brushing specimens, however, did not show any evidence of active TB.

3.4 | Echocardiography and electrocardiogram

Doppler echocardiography showed elevated pulmonary arterial pressure in all eight patients. The median Peak tricuspid regurgitation velocity was 4.35 m/s (range 3.5-4.6 m/s). The median PASP was 81 mm Hg (range 51-107 mm Hg). The right ventricle was dilated in three patients.

Electrocardiograms (ECG) of six patients were reviewed. ECG revealed RV hypertrophy in five patients, right axis deviation in two patients, and QTc prolongation in one patient. No P pulmonale or right bundle branch block was identified.

3.5 | Etiological factors

Table 4 shows the possible etiological factors. All eight patients had been using coal or biomass fuels (i.e., wood, leaves) indoors for cooking or heating. None of the patients had occupational exposure to coal dust. All of the patients had evidence of conclusive or suggestive TB. According to their past history, four patients had been diagnosed with TB, and two had been treated with an anti-TB therapy. Chest CT showed old TB lesions in five patients. Five patients also showed increased levels of interferon-γ release assays for TB (T-SPOT.TB; Oxford Immunotec, Oxford, UK) (180-880 spot-forming cells/10⁶ peripheral blood mononuclear cells). Five patients underwent empirical anti-TB therapy after diagnosis, although the smears and cultures for acid-fast bacilli were negative (Table 4).

Screening tests for rheumatological disease revealed that the anti-nuclear antibody assay was positive in two patients (1:80 and 1:160, respectively). One patient had Sjögren syndrome. Serum levels of immunoglobulin G4 were measured in six patients and were within the normal range in all cases.
Other laboratory tests

Blood gas analysis revealed that the arterial partial pressure of oxygen was 65.1 mm Hg (range 52.7-84.0 mm Hg), and the partial pressure of carbon dioxide was 36.5 mm Hg (34.6-58.0 mm Hg). Lung function tests, which were performed in five patients, revealed that four of the five patients had an obstructive ventilation defect, with the forced expiratory volume at 1 s/forced vital capacity ratio at 55.9%-69.0%.

Treatment and follow-up

Five patients underwent empirical anti-TB therapy after diagnosis. Four patients were given anticoagulant therapy. Three patients were treated with vardenafl or sildenafil.

TABLE 3 Bronchoscopic findings in the eight patients studied

| Variable                        | No. of patients |
|---------------------------------|-----------------|
| Anthracotic pigmentation        | 8               |
| Luminal narrowing of bronchus   | 8               |
| Left main bronchus              | 1               |
| Left upper lobar bronchus       | 5               |
| Left lingular bronchus          | 1               |
| Left lower lobar bronchus       | 6               |
| Right upper lobar bronchus      | 5               |
| Bronchus intermedius            | 3               |
| Right middle lobar bronchus     | 3               |
| Right lower lobar bronchus      | 5               |
Table 4: Possible etiologies for fibrosing mediastinitis and bronchial anthracofibrosis in the eight patients studied.

| Etiology                                | No. of patients |
|-----------------------------------------|-----------------|
| Exposure to, or infection with, TB      | 8               |
| History of TB                           | 4               |
| Old TB on CT scans                      | 5               |
| Elevated TB-SPTOT.TB*                   | 5               |
| Positive smear/culture of sputum or bronchoscopic samples for acid-fast bacilli | 0               |
| Anti-TB therapy after diagnosis         | 5               |
| Exposure to indoor biomass fuel or coal smoke | 8               |
| Elevated serum immunoglobulin G4       | 0               |
| Positive ANA test                       | 2               |
| Sjögren syndrome                        | 1               |

ANA, antinuclear antibody; CT, computed tomography; TB, tuberculosis.

*Increased levels of interferon-γ release assays for TB (T-SPTOT.TB; Oxford Immunotec, Oxford, UK).

One patient was treated with glucocorticoid and tamoxifen. Two patients were treated with a glucocorticoid in combination with cyclophosphamide. None of the eight patients underwent interventional therapy. The median follow-up time was 39.1 months (11.7–96.0 months). Six patients were stable after treatment. One patient died of severe community-acquired pneumonia, and one patient was lost to follow-up.

**4 | DISCUSSION**

Both FM and BAF are rare diseases, and the combined appearance of FM and BAF is extremely rare. A search of the literature revealed only one case of coexisting FM and BAF.16 Here we reported eight patients with combined FM and BAF, who had the following features: (1) All were older people, (2) All had irregular soft-tissue attenuation lesions in the mediastinum, with bronchial and pulmonary arterial stenoses—a combination consistent with FM, (3) Bronchoscopy revealed multiple areas of anthracotic pigmentation and narrowing bronchi, suggesting BAF, (4) All had prominent PH, (5) Each of the eight patients had a history of TB exposure and long-term exposure to coal or biofuel smoke.

The literature indicates that FM and BAF are different disease entities and have different diagnostic criteria. The diagnosis of FM is based on CT findings, whereas that of BAF is based on bronchoscopic manifestations. FM and BAF, however, do share some common clinical symptoms, radiological features and etiologies.

The most common symptoms of both diseases are dyspnea and cough.1,12 FM is different from BAF, however, in that it has more clinical manifestations because of the compression of other mediastinal structures, such as PH, dysphagia caused by esophageal compression, swelling of head and face caused by superior vena cava syndrome,18 acute myocardial infarction,19,20 and chylothorax.21 In our study, the clinical manifestations of the eight patients with FM and BAF were dyspnea, cough, hemoptysis and chest pain, which were similar to those in previous reports.1,12 The most prominent symptoms were dyspnea, which was related to the obstructive ventilation defects, and PH, which was due to compression of the bronchovascular bundles.

The typical imaging features of FM included soft-tissue-density mediastinal masses and pulmonary opacities secondary to occlusion of airways or vessels, such as obstructive pneumonia, atelectasis, infarction, or thickening of interlobular septa. The typical imaging features of BAF also showed mediastinal lymph node enlargement, with or without calcification, in both TB-associated11 and biofuel-associated BAF. Other imaging features included peribronchial soft-tissue opacities, bronchial stenosis or occlusion, and secondary pulmonary atelectasis.11,24 Thus, FM and BAF have imaging features in common, but FM could have more compressed mediastinal structures and soft-tissue opacities in the mediastinum, whereas BAF may also involve the lymph nodes outside the mediastinum such as in the interlobar and lobar lymph node regions. Therefore, from the viewpoint of imaging features, BAF might be a subtype of FM.

The etiology and pathogenesis of combined FM and BAF remain unknown. All eight of our patients had a history of conclusive or suggestive TB, although there was no evidence of active TB, which is different from the results reported by Chung et al.11 It is worth noticing that both FM2,25 and BAF11,12 were strongly associated with TB. In fact, TB infection of lymph nodes could result in chronic inflammatory and fibrous changes, causing structural compression in the mediastinum, ultimately resulting in FM syndrome. Perforation of intrathoracic tuberculous lymph nodes into the bronchi could result in endobronchial pigmentation with airway narrowing, or BAF.11,26,27 In addition, all of the eight patients had a history of exposure to indoor coal or biofuel smoke. Chronic exposure to biofuel smoke is most probable causative factor of BAF.12,13 BAF was predominantly observed in elderly housewives in rural areas with prolonged exposure to biomass fuel.27 The inhaled particles of biomass fuel were accumulated at the branching portion of bronchus, while some were taken up by macrophages or bronchial epithelial cells. Anthracotic carbonaceous pigments can deposit in the bronchi subepithelium and interalveolar septa,29–31 increase airway inflammation,30–32 and might cause fibrosis or hypertrophy of the bronchial wall and luminal narrowing,28,33 ultimately resulting in BAF. There’s
no evidence that biofuel smoke can cause FM directly, but biofuel-associated BAF is frequently coexisting with TB (33.9%) and mediastinal lymphadenopathy (66.0%), so that biofuel-associated BAF might be indirectly correlated with FM. As TB had been prevalent and biomass fuel had been used extensively for cooking and heating in China and other developing countries several decades ago, it may be speculated that dual exposure to TB infection and indoor biofuels or coal smoke could be important causes of combined BAF and FM in a developing country.

It is worth noting that all of the eight patients with combined FM and BAF had elevated pulmonary artery pressure. PH secondary to FM is classified as group 5 PH (i.e., PH due to unknown or multiple causes), but the exact incidence of PH in patients with FM is still unknown. Peikert et al., who reported the largest case series of FM, revealed that 38 of 80 patients had compression of the pulmonary vessels. The authors, however, did not show data for the pulmonary arterial pressure. The relation between BAF and PH is much more unclear. Sandoval et al. reported that 22 Mexican patients had pulmonary arterial hypertension and cor pulmonale caused by long-term contact with wood smoke. Among them, 14 patients also had anthracotic staining of bronchial mucosa, suggesting BAF. The mechanism of severe pulmonary arterial hypertension was unclear because the pulmonary function impairment was relatively mild. Combined BAF and FM might be an explanation, but there were no CT scans to be evaluated as to whether these patients had mediastinal lesions and a stenotic pulmonary artery.

Our data showed that patients with combined FM and BAF may be susceptible to developing PH. A possible reason for this connection could be that bronchi are anatomically associated with pulmonary arteries. Hence, mediastinal lesions surrounding bronchovascular bundles may simultaneously compress bronchial and pulmonary arteries. PH secondary to FM and BAF was different from other subtypes of PH. The PH was caused by mechanical compression of lobar or segmental arteries, thus the patients with FM and BAF will not respond well to specific drug therapy for PH as other subtypes of PH. Patients with FM and/or BAF should be screened for PH in order to identify those who need specific therapy (e.g., vascular interventional therapy) to relieve their symptoms. It is very important for clinicians to pay special attention to mediastinal lesions in addition to parenchymal abnormalities on CT scan when they make differential diagnosis of PH. Combined FM and BAF should be suspected in patients with PH and the following characteristics: older age; residing in a developing county; a history of TB infection or long-term exposure to biofuels; irregular soft-tissue opacities of the mediastinum with or without calcification.

It is unclear whether FM and BAF simply coexist occasionally or together comprise a distinct pulmonary disorder. Considering that FM and BAF have features in common, including clinical manifestations, radiological findings and etiological factors, BAF could be a subtype of FM. Alternatively, combined FM and BAF could be called a mediastinal and peribronchial fibrotic syndrome.

There are several limitations in our study. First, it was a retrospective study, and there were a limited number of cases. The exact incidence of combined FM and BAF and the incidence of PH in patients with FM and/or BAF are unknown, which should be confirmed in future studies. Second, right ventricular catheter, which is the gold standard for diagnosis of PH, was not performed, since there was no specific drug therapy for the PH associated with FM and BAF so that the patients cannot benefit from this invasive procedure.

In conclusion, FM and BAF can coexist. Our patients with combined FM and BAF characteristically had CT-diagnosed mediastinal masses and multiple stenoses of the bronchi and pulmonary artery, narrowing bronchi with areas of anthracotic pigmentation on bronchoscopy, and prominent PH. Combined FM and BAF could be associated with a TB infection and/or exposure to coal or biomass fuel smoke.

CONFLICT OF INTEREST
The authors declare that they have no conflict of interests.

AUTHORS’ CONTRIBUTIONS
YX collected the data and participated in writing the manuscript. MP conceived of and designed the study and wrote the manuscript. WX conceived of the study and revised the manuscript. YL, XT, KX, JS, MW and BC participated in diagnosing, treating and following up the patients, and helped to revise the manuscript. All authors read and approved the final manuscript.

ETHICS
This study was approved by Peking Union Medical College Hospital Institutional Review Board. Informed consent for publication of the clinical information was obtained from the patient at the time of diagnosis or follow-up.

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