



## Case report

## Diffuse parenchymal lung disease with micro aspirations in presence of hiatal hernia

Rishitha Yeliseti<sup>\*</sup>, Areig Awad, Anand Kaji

Department of Internal Medicine, St. Francis Medical Center, Trenton, NJ, United States

## ARTICLE INFO

## Article history:

Received 25 June 2017

Accepted 9 August 2017

## Keywords:

Micro aspirations

Diffuse parenchymal lung disease

Hiatal hernia

## ABSTRACT

There has been controversy regarding the relationship between gastroesophageal reflux, micro-aspiration, and idiopathic pulmonary fibrosis (IPF). In the last decade, there is increasing evidence supporting a relationship between gastroesophageal reflux, microaspiration, and IPF. The presence of hiatal hernia further propagates microaspirations. Surgical management of hiatal hernia plays a vital role in the treatment.

© 2017 Published by Elsevier Ltd. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

## 1. Introduction

Interstitial lung diseases represent several conditions that chiefly involve the parenchyma of the lung. There are about 200 individual diseases that are classified under this broad category. They are grouped together because of similar clinical, roentgenographic, physiological, and pathological manifestations.

Idiopathic pulmonary fibrosis is a type of diffuse parenchymal lung disease which causes scarring of the lung tissue. Etiology remains mostly unexplained. Recent data has shown the potential role of subclinical microaspirations in the development of this condition. We present a case of diffuse pulmonary fibrosis in a patient with chronic microaspirations promoted by the presence of hiatal hernia. We hope to illustrate the contributory role of microaspirations in the pathophysiology of idiopathic pulmonary fibrosis to provide earlier recognition of the disease and offer possible interventions such as surgical repair of the hiatal hernia.

## 2. Case presentation

A 55-year-old Caucasian female with past medical history of gastroesophageal reflux disease, mild intermittent asthma, anxiety and panic attacks, moderately large hiatal hernia, primary hypothyroidism, and chronic low back pain presented to the emergency department with complaints of increasing dyspnea since the last 6 month period. The dyspnea has started insidiously and gradually

worsened. She noted a decrease in her exercise tolerance. Pt denied any orthopnea and paroxysmal nocturnal dyspnea. She also complained of chronic cough that was productive of moderate amounts of whitish sputum. Pt denied any recent fevers, hemoptysis, and weight loss. She did not have any recent history of travel or sick contacts. She had a 30 pack year of smoking history which she quit about a year before presentation. Her occupational history was noncontributory to the presentation.

At the time of presentation her vitals were: temp of 37.6 C, pulse rate of 64 beats/min regular, respiratory rate of 20 breaths/min, and oxygen saturation of 92% breathing on 2 L nasal cannula. On physical examination, she was an obese Caucasian female who looked her stated age. Notable pertinent positives on the examination included a thick neck and dry crackles with Velcro rales auscultated at the bases of both lungs. Remainder of the examination was unremarkable. Review of the previous records from our institution and other institutions indicated that the patient had multiple admissions with similar complaints in the past. Provisional diagnosis during these admissions were either community acquired pneumonia or hospital acquired pneumonia.

On this admission, the patient was also initially started on antibiotics but she only noted minimal improvement in her symptoms. Further workup was conducted to identify the cause. Human immunodeficiency virus testing was negative. Tests for other infectious processes such as tuberculosis, Streptococcal, and Legionella pneumonia were negative. Angiotensin converting enzyme level was normal and there was no evidence of pulmonary embolism on imaging. Serial computerized axial tomography scans (CAT scans) demonstrated interstitial lung involvement with patchy bilateral ground glass opacities and mediastinal lymphadenopathy.

<sup>\*</sup> Corresponding author.

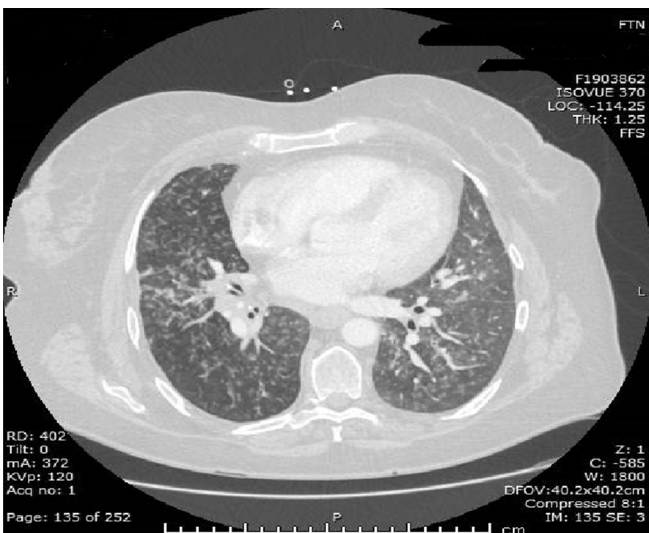
E-mail address: [rishitha.yeliseti@gmail.com](mailto:rishitha.yeliseti@gmail.com) (R. Yeliseti).



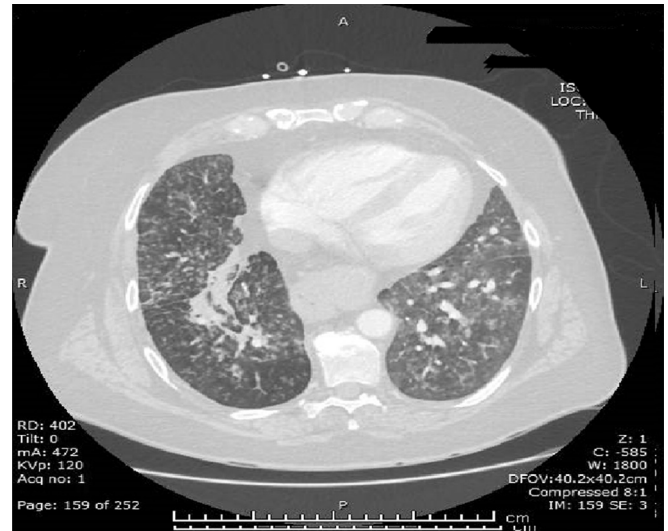
**Fig. 1.** X-ray: shows diffuse bilateral interstitial infiltrates.

The infiltrates were progressively worsening on repeated CAT scans [Fig. 1](#) demonstrating X- ray and [Figs. 2–4](#) - CAT scans.

Patient finally underwent a video-assisted thoracoscopy for biopsy of the lung. Pathology revealed patchy organizing fibrosis with acute on chronic inflammation, microabscess formation, and foreign body granulomas with demonstration of frank vegetable matter in the lungs. Acid-fast stain test and Grocott's methenamine silver stain were negative. Findings were compatible with chronic microaspirations likely propagated by hiatal hernia causing idiopathic pulmonary fibrosis. The patient was referred for surgical repair of the hiatal hernia.



**Fig. 2.** CT: lung windows: diffuse bilateral interstitial infiltrates, with tree in bud configuration. There re patchy ground glass changes seem throughout, with focal consolidation within the right mid to lower lung zone.



**Fig. 3.** CT: lung windows: diffuse bilateral interstitial infiltrates, with tree in bud configuration. There re patchy ground glass changes seem throughout, with focal consolidation within the right mid to lower lung zone.



**Fig. 4.** CT: soft tissue window: shows a moderate to large hiatal hernia.

### 3. Discussion

Aspiration of gastric contents into the lungs is well-described phenomenon known to create an acute inflammatory reaction [9]. However, microaspirations and their consequences have not been described well in the literature. Recently, in a study conducted at the University of Chicago of twenty-five patients, number of associations have been found between chronic microaspirations, gastroesophageal reflux disease [GERD], elevated body mass index, and obstructive sleep apnea [10]. The most plausible explanation is the disruption of the ability to protect the airways.

In patients with esophageal dysfunction, ineffective passage of oral contents puts the patient at increased risk of chronic aspiration. GERD previously had been associated with interstitial lung disease in number of studies [4,5]. Elevated body mass index is postulated to create a higher intra-abdominal pressure which leads to reduced pressure gradient across the lower esophageal sphincter

promoting aspiration [6–8]. Anatomical or functional disorders of the lower esophagus also create a similar pathophysiological picture. Studies have shown that the hiatal hernias contribute to microaspiration as well. Clinically, these patients are more likely to be obese and often present with gradually progressive shortness of breath and chronic cough.

Chest radiography and CAT scans demonstrate bronchiolitis with centrilobular wall thickening, tree-in-bud opacities, and ground glass infiltrates. Pathologically, the spectrum ranges from foreign body giant cells and peribronchiolar granulomas to the terminal stage of fibrosis. Of note, the granulomas are more loosely arranged than those of sarcoidosis. Visualizing vegetable matter in the lung biopsy specimens is a rare occurrence and diagnosis is one of exclusion. There are no gold standard diagnostic tests. Imaging findings of hiatal hernia and the swallowing function studies can be supportive. Pulmonary function tests reveal a restrictive pattern of involvement.

Treatment is mainly focused on addressing aggravating factors like GERD [1], underlying sleep apnea, and weight reduction which helps in reducing the intra-abdominal pressure. Prompt surgical evaluation and correction of anatomical abnormalities of the lower esophagus such as addressing the hiatal hernia in our case can play a contributory and vital role [2].

## References

- [1] American Thoracic Society, Idiopathic pulmonary fibrosis: diagnosis and treatment. International consensus statement. American thoracic society (ATS), and the European respiratory society (ERS), *Am. J. Respir. Crit. Care Med.* 161 (2000) 646–664.
- [2] J.E. Pearson, R.S. Wilson, Diffuse pulmonary fibrosis and hiatus hernia, *Thorax* 26 (1971) 300–305.
- [4] R.W. Tobin, C.E. Pope II, C.A. Pellegrini, M.J. Emond, J. Sillery, G. Raghu, Increased prevalence of gastroesophageal reflux in patients with idiopathic pulmonary fibrosis, *Am. J. Respir. Crit. Care Med.* 158 (1998) 1804–1808.
- [5] G. Raghu, T.D. Freudemberger, S. Yang, J.R. Curtis, C. Spada, J. Hayes, J.K. Sillery, C.E. Pope 2nd, C.A. Pellegrini, High prevalence of abnormal acid gastroesophageal reflux in idiopathic pulmonary fibrosis, *Eur. Respir. J.* 27 (2006) 136–142.
- [6] L.J. Wilson, W. Ma, B.I. Hirschowitz, Association of obesity with hiatal hernia and esophagitis, *Am. J. Gastroenterol.* 94 (1999) 2840–2844.
- [7] R. Anggiansah, R. Sweis, A. Anggiansah, T. Wong, V. Cooper, M. Fox, The effects of obesity on oesophageal function, acid exposure and the symptoms of gastroesophageal reflux disease, *Aliment. Pharmacol. Ther.* 37 (2013) 555–563.
- [8] G.R. Locke III, N.J. Talley, S.L. Fett, A.R. Zinsmeister, L.J. Melton III, Risk factors associated with symptoms of gastroesophageal reflux, *Am. J. Med.* 106 (1999) 642–649.
- [9] P.E. Marik, Aspiration pneumonitis and aspiration pneumonia, *N. Engl. J. Med.* 344 (9) (2001) 665–671.
- [10] C.L. Ogden, M.D. Carroll, B.K. Kit, K.M. Flegal, Prevalence of Obesity in the United States, 2009–2010, 82, *NCHS Data Brief*, 2012, pp. 1–8.