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A Pleural Effusion Secondary to Unusual Dual Pathology: A Case Report

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Abstract

Pleural effusion is a commonly encountered respiratory disorder. In the majority of cases, a single causative agent is responsible. Pleural effusions resulting from simultaneous involvement of the membranes by two different pathologic processes are unusual.

Keywords

Pleural Effusion, Adenocarcinoma, *Mycobacterium fortuitum*

1. Introduction

Pleural effusions are not uncommon in clinical practice. They can result from multiple disorders. These can be pleuro-pulmonary or systemic. The disorders can be benign or malignant. To narrow the differential diagnosis the patient's history and physical examination are critical. Tuberculosis, cancer, heart failure and para-pneumonic effusions account for most of the cases [1]. Dual pathologies are uncommon and pleural effusions are rarely associated with atypical mycobacteria, in particular the rapidly growing species.

We are reporting an unusual case of a pleural effusion secondary to two different aetiologies occurring simultaneously. The effusion contained cells consistent with a metastatic lung adenocarcinoma. In addition, *Mycobacterium fortuitum* was cultured on the fluid.

2. Case Report

A 48-year-old previously well mother of two was initially evaluated in our medical outpatient clinic for a productive cough of ±2 week's duration, right pleuritic pain, fever, chills and a single episode of minor haemoptysis Her physical examination was thought consistent with a right lower lobe pneumonia and her

chest radiograph (CXR) showed an associated small effusion. The patient was assessed to have community-acquired pneumonia with a small para-pneumonic effusion, a CURB-65 score of zero and discharged home on oral antibiotics. She tested negative for the human immunodeficiency virus.

She represented 8 weeks later with worsening symptoms (more cough, unresolving chest pain and worsening shortness of breath) and a repeat CXR showed the effusion to have increased in size. She was admitted for inpatient management. A chest computerised tomography (CT) and a diagnostic thoracentesis were performed. The chest computed tomography with contrast showed an enhancing ill-defined obstructing mass lesion at the level of the bronchus intermedius causing atelectasis of both the right middle and lower lobes, a right pleural effusion with associated pleural nodules. Bilateral pulmonary nodules of varying sizes and enlarged para-tracheal, cervical and supraclavicular nodes (Figure 1 and Figure 2).

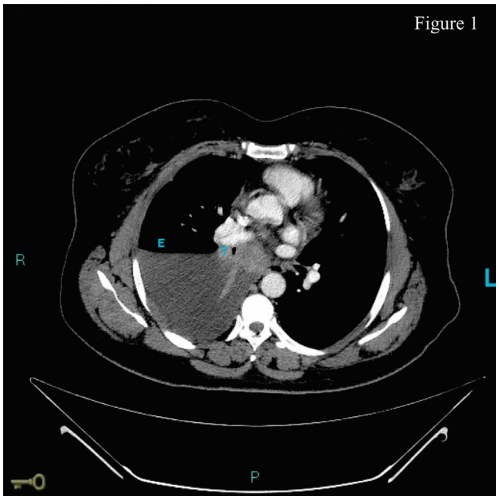

Figure 1. E = pleural effusion; T = tumor mass at bromchus intermedius.

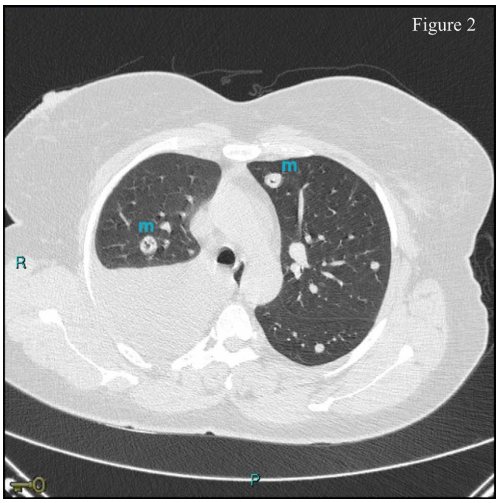

Figure 2. m = metastases.

Biochemical analysis of the effusion showed it to be an exudate with a normal level of adenosine deaminase enzyme. Cytological evaluation of the aspirate showed an adenocarcinoma of undetermined origin. In addition, growth of *Mycobacterium fortuitum* was detected on day 5 of culture of the effusion. Subsequent biopsy of the middle lobe mass seen on her chest CT confirmed it to be an infiltrating adenocarcinoma. The tumour cells were arranged in tubular and nests of discohesive cells (**Figure 3** and **Figure 4**). The cells had eosinophilic cytoplasm with pleomorphic nuclei. The chromatin was coarse and granular with prominent nucleoli. On immune-histochemistry the tumour cells were positive for CK7, CEA and TTF1 and negative for CK20, favouring lung origin of the cancer (**Figure 5** and **Figure 6**).

The patient was referred to medical oncology and infectious disease services for further care. She completed six cycles of platinum-based chemotherapy and received 10 months of antibiotic treatment with clarithromycin, ciprofloxacin and sulfamethoxazole. Her follow-up imaging and clinical review suggested progression of her malignancy; now with osseous and suprarenal gland metastatic deposits. She has recently completed a course of second-line chemotherapy [Docetaxel] and is awaiting review by the medical oncology team.

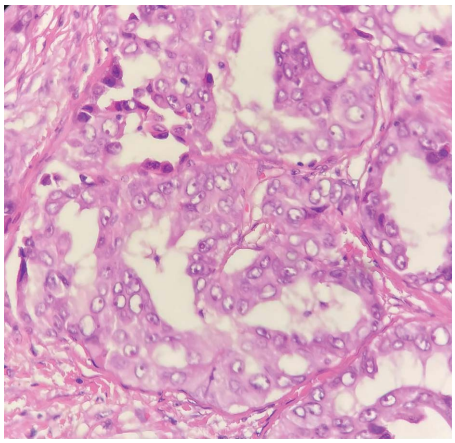

Figure 3. Tumor cells.

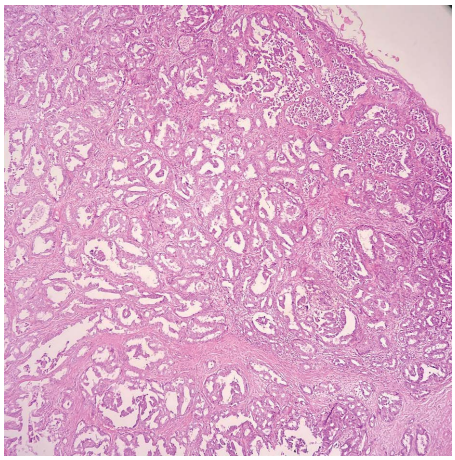

Figure 4. Nests of discohesive cells.

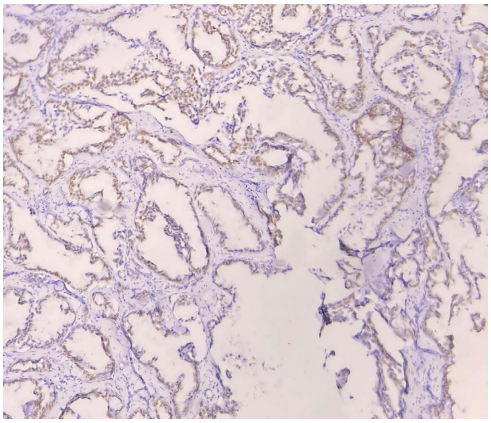

Figure 5. TTF-1 positivity.

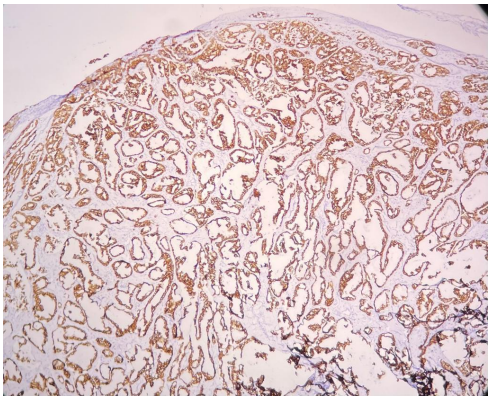

Figure 6. CK 7 positivity.

3. Discussion

Pleural effusions develop secondary to direct involvement of the pleural membranes by disease, which leads to either increased formation of the liquid or interferes with the functioning of lymphatics of the parietal pleura [2]. Elevations in hydrostatic pressure such as occurs in congestive heart failure cause effusions by increasing filtration from the micro-vessels of the pleural membrane. Pleural effusion can also be of extra-vascular origin [3]. This occurs when fluid migrates from an extra-pleural space such as the abdomen, the genitourinary system, the biliary system, or the central nervous system.

For patients with pleural malignancy or pleural infections, it is the combination of increased entry and decreased fluid exit that is thought to operate synergistically to cause fluid accumulation. [4]. Effusions secondary to malignancy are not unusual. Cases of pleural effusions caused by *Mycobacterium fortuitum* have only rarely been described [5] [6] [7].

Mycobacterium fortuitum is a member of the rapid-growing mycobacteria (RGM). It is found worldwide in soil and tap water. It is thought increasing in prevalence in patients with and without HIV but remains an infrequent human pathogen. [5] [8] It is the most common of the RGMs, has low virulence and usually causes colonization or minor infection in patients with pre-existing lung

disease such as bronchiectasis or prior tuberculosis. [8]

Two possible pathogenic mechanisms for effusions developing in patients with *Mycobacterium fortuitum* infection have been suggested: direct extension from lung lesions into the pleura or haematogenous spread.

It is unclear what the most appropriate treatment strategy should be for pleural infection by *Mycobacterium fortuitum* as literature on the topic is sparse. *Mycobacterium fortuitum* is resistant to anti-mycobacterials. [9] It is usually susceptible to multiple oral anti-microbial agents such as the newer macrolides, fluoroquinolones, doxycycline, aminoglycosides, carbapenems and sulphonamides. [9] A treatment period of at least 12 months with at least two anti-microbial agents with in vitro activity against the isolate are recommended for pulmonary involvement [9]

4. Conclusion

It is not possible to determine how much of the effusion was secondary to the mycobacterial infection in this patient with metastatic malignant pleural disease. The pleural space is sterile and *Mycobacterium fortuitum* isolation from this site is unlikely the result of contamination. *Mycobacterium fortuitum* has previously been isolated from pleural fluid and successfully treated; indicating it could be pathogenic to the pleura [6] [7]. It would seem prudent not to discount it when isolated from this site and to consider it in the differential diagnosis of a patient with an exudative pleural effusion.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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