Drainage of multiloculated tuberculous pleural effusion by medical thoracoscopy: When and why should it be considered?

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SUMMARY

Tuberculous pleural effusion (TBE) is a common encounter in our region. Up to 50% of patients with TBE will develop residual pleural thickening (RPT) which can lead to functional impairment. However, the need of drainage remains controversial. We report a case of end-stage renal failure patient who presented with right multiloculated tuberculous pleural effusion which was drained via a medical thoracoscope. Patient reports immediate relief of breathlessness post procedure and one month follow up shown significant improvement of RPT. We also discussed the current perspective on the rationale of TBE drainage and the role of medical thoracoscope in TBE management.

INTRODUCTION

Tuberculous pleural effusion (TBE) is a common encounter in our region. Up to 50% of patients with TBE will develop residual pleural thickening (RPT) and RPT of more than 10mm can lead to functional impairment.[1-3] However, TBE drainage remains controversial as studies have shown that it does not reduce the risk of RPT significantly.[2] On the other hand, growing evidence suggests that early complete drainage of TBE is useful to prevent the development of RPT and also improve dyspnoea and accelerate lung function recovery.[1] We report a case of multiloculated TBE who was drained by medical thoracoscopy and review the current perspective on when and why TBE drainage should be considered.

CASE REPORT

A 43-year-old gentleman with underlying end-stage renal failure on haemodialysis, presented with one week of fever, dyspnoea, productive cough and right pleuritic chest pain. Initial chest x-ray showed bilateral pleural effusion with cardiomegaly consistent with fluid overload state. However, he failed to respond to antibiotics and hemodialysis, a repeated chest x-ray (Figure 1) and ultrasound revealed a multiloculated right pleural effusion. Diagnostic thoracocentesis yielded straw-coloured fluid with pH of 7.720, glucose 12.0mmol/l, pleural fluid/serum (PF/S) protein of 52/76 mmol/l (Ratio 0.68) and PF/S lactate dehydrogenase of 578/420 mmol/l (Ratio 1.38). Sputum and pleural fluid acid-fast bacilli were negative. As the patient was dyspnoeic and diagnosis remains uncertain, medical thoracoscope was decided for both diagnostic and therapeutic purposes.

Under conscious sedation, the right pleural cavity was accessed via blunt dissection followed by the introduction of a 13mm metallic trocar with a cannula (40107CD, Karl Storz, Germany). The semi-rigid thoracoscope (LTF-160, Olympus Medical, Japan) was then introduced into the pleural cavity through the metallic cannula. The pleural fluid was severely loculated due to multiple septations. Adhesiolysis was performed using flexible forcep until the parietal pleural was visualized which was diffusely inflamed and thickened with two caseating nodules that contain yellow cheesy material upon biopsy (Figure 2). A 24 Fr intercostal drain was inserted post procedure for drainage of residual fluid.

The patient reported significant improvement of dyspnoea post procedure and chest x-ray showed good drainage with RPT of only 13mm. Antituberculous therapy was commenced immediately post procedure and pleural biopsy subsequently confirmed chronic caseating granulomatous inflammation that was consistent with the diagnosis of tuberculosis. Chest x-ray one month later showed improvement in RPT with no re-accumulation of pleural effusion.

DISCUSSION

Tuberculous pleural effusion (TBE) is a result of delayed hypersensitivity reaction towards tuberculous bacilli when subpleural caseous foci rupture into the pleural cavity. The resulting rich inflammatory pleural exudates augment pleural inflammation and result in residual pleural thickening (RPT) in up to 50% of patients.[1]

Utilizing pleural fluid parameters to predict RPT is not feasible. While some reports suggest that lower pleural fluid pH, glucose and higher pleural fluid concentration of lysozyme and tumour necrosis factor-α are associated with higher risk[3]; some studies had proven otherwise. Despite this, the recent study had shown that loculated pleural effusion at initial presentation is a promising predictor for RPT in TBE.[1] As there are no firm recommendations at the moment, the decision has to be on the discretion of the physician, taking

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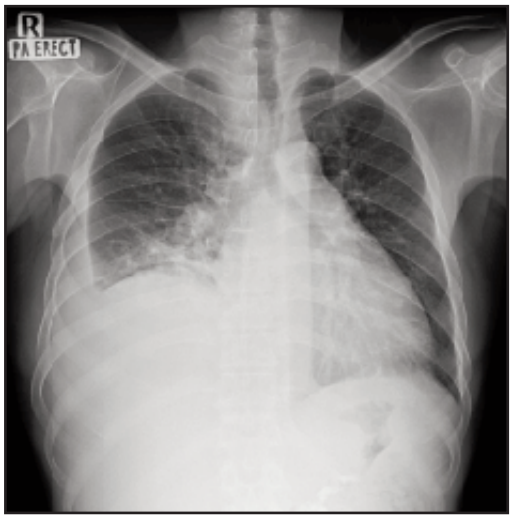

Fig. 1: Chest x-ray demonstrates a loculated right pleural effusion.

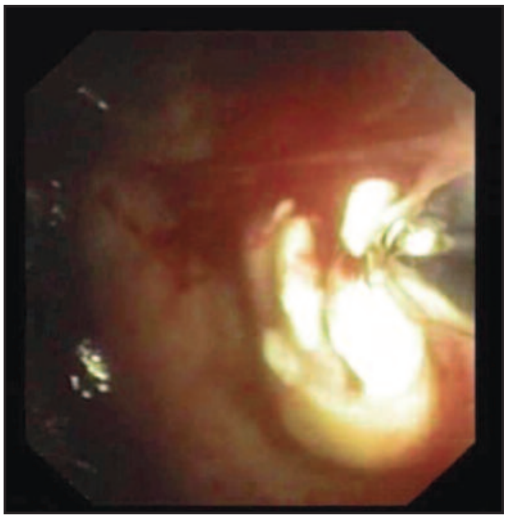

Fig. 2: Caseating nodule at the parietal pleural surface.

account into the risks and benefits of all the potential parameters. Nevertheless, precise risk prediction models need to be developed in future for assessing the risk of RPT development to aid in clinical decision for drainage.

TBE patients commonly present with an acute febrile illness and thus it is challenging to differentiate TBE from complicated parapneumonic effusion. A decision for drainage needs to be considered when bacterial infection cannot be ruled out during initial presentation, as complete drainage is the cornerstone of treatment in thoracic empyema and complicated parapneumonic effusion. Although pleural fluid adenosine deaminase (ADA) has a high positive predictive for tuberculosis in high tuberculosis burden regions and can be helpful in guiding management during initial presentation, this test is not widely available in our country. Moreover, it can be falsely high in empyema, and this further limit its usage in our case as suspicion for bacterial infection remains high as he presented with acute febrile illness with a multiloculated pleural effusion.

The need of TBE drainage remains a controversial topic as reports had shown that drainage did not influence the development of RPT, nor impact on lung function significantly.[2] However, growing studies had shown that earlier drainage of TBE not only reduces the occurrence of RPT but also accelerates pulmonary function recovery.[1] Furthermore, early drainage also significantly reduces dyspnea[1], allowing our patients to return to their daily activities earlier. Besides, TBE is known to reabsorb completely in around four months with antituberculous therapy alone without drainage, and this will definitely trouble our patient with longer duration of dyspnea.

Medical thoracoscopy is both diagnostic and therapeutic when dealing with multiloculated pleural effusion during initial presentation. Other than providing an excellent diagnostic yield in TBE, visual findings of medical thoracoscope provide reasonable diagnostic certainty, which allows instant implementation of antituberculous therapy.[4] Moreover, it also increases the percentage of positive tuberculosis cultures from biopsy. Antituberculous therapy was started immediately for our patient in view of characteristic TBE findings which was proven accurate by histological examination. Furthermore, medical thoracoscopy is minimally invasive and able to achieve shorter drainage duration and hospital stay with no further additional procedures required during the follow up period, compared to intrapleural fibrinolytic administered via conventional chest tube or pleural catheter drainage in the treatment of TBE.[5]

In conclusion, the decision for drainage can be considered when one encounters with a loculated TBE and facing with diagnostic uncertainty when empyema or complicated parapneumonic effusion cannot be safely ruled out. Drainage of TBE has been shown to improve dyspnoea and accelerate lung function recovery, at the same time reducing the risk of RPT development.[1] Medical thoracoscopy has shown promising results in the management of thoracic empyema, but the literature on its role in the management of TBE remains scarce[4], more studies are needed to evaluate its role in the management of TBE in future.

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