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An unexplained effusion with indwelling pleural catheter related complications

Leher Gumber, Cheng Hong Lim, Kirk Ramharack, Avinash Aujayeb

ABSTRACT

Indwelling pleural catheter colonization and infection are separate clinical entities, and the management is different. As the use of those catheters increases, clinicians must be aware of those potential complications.

Keywords: Empyema, Indwelling pleural catheter, Pleural catheter colonization, Pleural space infection, Thoracoscopy

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INTRODUCTION

Indwelling pleural catheters (IPCs) are increasingly used in respiratory medicine, as a method of controlling both benign and malignant pleural effusions. They can be placed at the end of a local anesthetic thoracoscopy (LAT), which is a multimodality approach to investigate an unexplained effusion. There are some nuances between colonization of the IPC and true IPC or pleural space infection, which the following case report highlights.

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CASE REPORT

An 83-year-old male presented to the pleural clinic with a few months' long history of progressive dyspnoea. He had no constitutional symptoms or chest pain. He had a 10-pack year smoking history, 40 years ago. He worked with refrigerators, and it was possible that he had been exposed to asbestos during that time.

His past medical history included ischemic heart disease with previous coronary stents, and he was on bisoprolol 5 milligrams (mg) daily, simvastatin 40 mg daily, ramipril 10 mg daily and aspirin 75 mg daily.

On examination, he was comfortable at rest with a normal cardiac examination, with saturations of 96% on air and a respiratory rate of 17 breaths per min. He had no clubbing or palpable lymphadenopathy. He had dullness to percussion at the right base extending to halfway up his hemithorax, with reduced air entry over that same area. There was no peripheral edema.

A chest radiograph showed a large right pleural effusion. A thoracic ultrasound showed a large right sided hyperechoic effusion. The maximal depth was 15 centimeters (cm), there were no septations or locules or pleural thickening.

Hematological investigations revealed hemoglobin of 143 $g{\cdot}L^{-1}$ (normal range 130–180 $g{\cdot}L^{-1}$), white cell count of 4.41 $\times 10^9$/L (normal range 4–11 $\times 10^9$/L), platelet count 144 $\times 10^9$/L (normal range 140–400 $\times 10^9$/L). Plasma lactate dehydrogenase (LDH) levels were 101 $U{\cdot}L^{-1}$. His total protein was 67 $g{\cdot}L^{-1}$ (normal range 60–80 $g{\cdot}L^{-1}$) and albumin 42 $g{\cdot}L^{-1}$ (normal range 25–50 $g{\cdot}L^{-1}$). N-terminal Pro B type natriuretic peptide levels were 417 ng L^{-1} (normal range less than 400). An echocardiogram done a few weeks before was normal.

With the appropriate consent and under asepsis, 1.2 L of straw-colored pleural fluid was removed via thoracocentesis. The pleural fluid was sent for microbiological, cytological and biochemical analysis.

Chest radiograph showing large, right-sided pleural effusion (Figure 1).

Pleural fluid analysis was then available: fluid LDH 77 U·L^{-1}, fluid protein 25 g·L^{-1}, fluid pH 7.46, and fluid glucose 6 mmol·L^{-1}, confirming the fluid as an exudate by applying Light's criteria. Microbiology and cytology were negative. The predominant cell count was lymphocytes.

A venous contrast enhanced computed tomogram (CT) scan of the thorax was performed. Figure 2 and 3 show slices of the chest CT with a large pleural effusion, but no pleural visceral or mediastinal thickening, no pleural nodules, and no pleural plaques.

In the absence of a specific target on the pleura, there is no real role for an image-guided biopsy using CT or ultrasound scan (USS). While CT scans are the accepted next radiological investigative step after a chest radiograph, it is known that CT scans can be normal in up to 40% of patients with pleural effusions. Features such as circumferential pleural thickening, nodular pleural thickening, parietal pleural thickening greater than 1 cm and mediastinal pleural involvement (Leung's criteria) make a malignancy more likely. In the presence of an unexplained exudative effusion, depending on patient fitness and choice and resource availability, thoracoscopic biopsies can be pursued. This can be physician led via LAT, or through cardiothoracic surgery with video-assisted thoracoscopic surgery. Diagnostic sensitivities and complication rates between LAT and the surgical approach are comparable, and the final choice is often resource dependent. LAT is offered locally in our center as a day case procedure with IPC insertion and the patient consented to that.

LAT was performed in theater under strict asepsis and with prophylactic antibiotic cover [a single shot of 2 grams (g) of flucloxacillin intravenously]. 3.1 L of pleural fluid was drained. The pleura appeared normal (pleural plaques were directly seen) and multisite biopsies were taken. An IPC was inserted post-LAT, and the procedure was uneventful with the patient discharged on the day.

Upon review in clinic two weeks later, the IPC was being drained twice a week (approximately 300 milliliters at a time). The patient was complaining of pain at the site of the IPC. This looked red and inflamed. A thoracic USS showed a small remaining free flowing hyperechoic pleural effusion. The patient was prescribed 14 days of 2 g of oral flucloxacillin four times a day. A swab taken from the site was negative. Upon review 10 days later, there was no improvement to the site, with now a small amount of pus seen. The patient had lost weight, had increased pain, and was quite lethargic. A thoracic USS showed a moderate effusion. Pulse, blood pressure, and pulse were all normal. C-reactive protein (CRP) levels were 45, with a normal white cell count. The IPC site was still inflamed, and the fibrotic cuff was migrating out (Figure 4).

The pleural biopsy result was now back and showed focal fibrous thickening of pleura with mild to moderate chronic inflammation. There was no evidence of atypical mesothelial proliferation. This was felt to be a subset of fibrinous pleuritis, and the biopsies were felt to be representative of the CT scan and the macroscopic appearance at LAT.

Often, IPC site infections remain superficial and can be treated with oral antibiotics. A swab of the superficial infection can be taken to see if any specific antibiotics can be given (such as either anti-staphylococcal or anti-pseudomonal ones for example). This is well-established practice. However, in the case of non-resolution of that infection, intravenous antibiotics can be used to salvage the IPC, but removal is often required. Some fluid was aspirated via the IPC for further analysis. Under local anaesthetic, the IPC cuff was easily dissected and the IPC removed. A small amount of pus was washed out and the wound loosely closed with nylon sutures. A 12 French gauge seldinger chest drain was inserted in the pleural effusion. Further fluid from the new puncture site for the chest drain and further site swab were sent for analysis. Intravenous antibiotics (piperacillin-tazobactam) were prescribed. There was rapid biochemical and clinical improvement. 1.2 L of straw-colored fluid was drained and the pleural space was dry on day 3. A chest radiograph showed full lung expansion. The swab was negative, but the pleural fluid aspirated from the IPC line grew a fully sensitive *Staphylococcus aureus*. The pleural fluid from new puncture site for the chest drain did not grow anything. Also, pleural fluid analysis from the new puncture site was as follows: fluid LDH 185 U L^{-1}, fluid protein 42 g L^{-1}, fluid pH 7.5, and fluid glucose 5.7 mmol·L^{-1}, cytology was again negative with predominant lymphocytes.

The patient was consented for talc pleurodesis, and 4 g

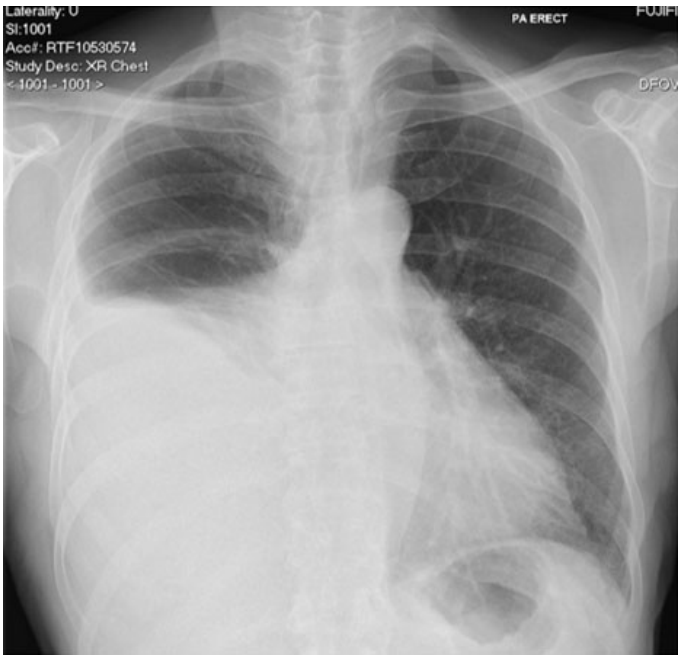

Figure 1: Showing large right-sided pleural effusion 186 × 178 mm (120 × 120 DPI).

Figure 2: Showing CT scan with large right effusion but no pleural nodules or thickening 179 × 114 mm (120 × 120 DPI).

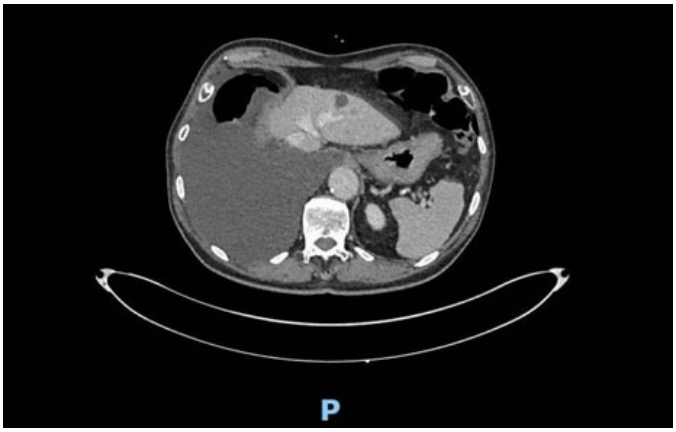

Figure 3: Showing CT scan with large right effusion but no pleural nodules or thickening 178 × 117 mm (120 × 120 DPI).

Figure 4: Showing infected IPC site with cuff migrating out 239 × 239 mm (300 × 300 DPI).

of sterile talc was inserted into the chest tube with consent. There were no immediate complications. The patient was switched to oral flucloxacillin 2 g four times a day again for a further five days. A review in clinic five days later showed improving clinical condition and no fluid build-up. Six months later, there is clinical stability. Follow-up will continue for two years in total as per standard for fibrinous pleuritis, which is the final diagnosis.

DISCUSSION

Many aspects of the case can be discussed. However, the role of medical thoracoscopy in unexplained exudative pleural effusions has been discussed before, as has the fact that CT scan appearances can be normal in pleural malignancies [1]. Computed tomogram cannot also differentiate between types of effusions (exudative versus transudative for example), which is why fluid analysis and biopsies are usually required [1]. We have previously published on day case thoracoscopy and its feasibility [2]. The pathogenesis and accepted follow-up protocols of fibrinous pleuritis are also beyond the scope of this article. What is important here is the fact that there was a localized IPC infection, and that the pleural fluid aspirated from the IPC grew a skin pathogen (*Staphylococcus aureus*), but that a concomitant pleural fluid sample was negative for any organisms. This signified that the IPC was colonized, and that the colonization did not cause a pleural space infection. The localized cellulitis and inflammatory reaction were responsible for the overall clinical picture.

LAT was related infections can be divided into 2 categories. Superficial infections are cellulitis, exit site infections, and tunnel tract infections. Deep infections are pleural space infections. It is important to differentiate IPC colonization from deep infection, as the management will differ. Little is known about IPC colonization. Preliminary data from the recent "Bacteria Responsible for IPC Infection and Colonization" (BRICC) study and other meta-analyses have shown that *S. aureus* and coagulase-negative staphylococci (CoNS) are commonly cultured in deep infection and colonization respectively [3, 4]. It is thought that biofilm formation on the IPC surface causes the subsequent colonization. It is not known when biofilms form after IPC insertion and their role in deep infection. However, it is known that bacteria within a biofilm differ from their other forms for many reasons including their ability to slow down their metabolism, thus limiting the efficacy of antibiotics. Hence, once a biofilm has formed on a foreign body, removal is usually required [4].

Our patient thus had a superficial infection and IPC colonization. He did not have a deep pleural infection. Removal of the IPC was the correct decision and as there was no deep pleural infection, we assumed that talc pleurodesis was safe, and would not cause any issues. There has been no recurrence of the effusion so far.

Due to the COVID-19 pandemic, we lost a large number of inpatient respiratory beds and had to convert to day case thoracoscopy very quickly by inserting an IPC and allowing same day discharge, instead of inserting a large bore drain, performing talc pleurodesis and admitting post-operative patients afterwards. Our service has not recovered its bed base, and so IPCs are inserted into everyone post-operatively. This is a current limitation of our otherwise well recognized pleural service, and one that we are actively working to change. As such, in the future, if there is a presumed benign effusions, perhaps IPCs should not be placed in those at the time of LAT as it is known that the infection risk is higher [5].

CONCLUSION

The above case highlights the issue around taking pleural fluid samples from existing IPCs, in that they become quickly colonized. Interpretation of any positive microbiological results requires careful clinical and radiological correlation. Further research such as from the BRICC study described above should shed light on mechanisms underlying IPC colonization.

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Author Contributions

Leher Gumber – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree 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

Cheng Hong Lim – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree 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

Kirk Ramharack – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree 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

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Guarantor of Submission

The corresponding author is the guarantor of submission.

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Consent Statement

Written informed consent was obtained from the patient for publication of this article.

Conflict of Interest

Authors declare no conflict of interest.

Data Availability

All relevant data are within the paper and its Supporting Information files.