

**IMAGES IN THORAX** 

## Ghost of chest drain past

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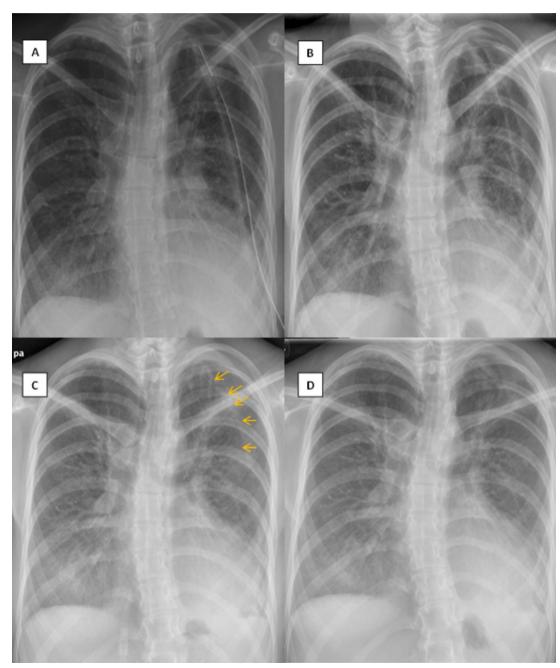
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A 20-year-old woman with recurrent left-sided pneumothoraces underwent video-assisted thoracoscopic pleurectomy and wedge resection of a left apical bulla. Medical history included ABCA3 pulmonary surfactant dysfunction and subsequent interstitial lung disease with diffuse ground glass changes suggestive of a desquamative interstitial pneumonitis (DIP). She was a lifelong non-smoker.

Spirometry revealed forced vital capacity 0.97 L, 32% predicted and carbon monoxide transfer factor (TLCO) 27% predicted. The operation was uneventful but there was a persistent postoperative air leak requiring prolonged pleural drainage but no other intervention. Histology of the resected lung tissue confirmed subpleural fibrotic disease with dilated airspaces including blebs and bullae. After



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Figure 1 Plain chest radiography with chest drain in situ (A), following removal (B), 2 weeks following removal (C) and 8 weeks following removal (D).



## Chest clinic

10 days of pleural drainage, the air leak resolved and the drain was ready for removal (figure 1A). Following uncomplicated chest drain removal, a chest radiograph revealed the presence of a well-defined curvilinear tract in the area vacated by the drain (figure 1B). Initially, there was a concern that this may represent the air reaccumulating in the pleural space or structural damage by the chest drain, but the patient remained well and went home the next day. A follow-up radiograph in clinic 2 weeks later showed partial resolution of the abnormality (figure 1C) and after 2 months there was complete resolution (figure 1D).

Pulmonary surfactants are essential for reducing airway surface tension in the lung. Inherited surfactant deficiencies are rare but can be associated with acute respiratory distress in the neonatal period and progressive interstitial disease later in childhood. DIP has been reported to be the predominant form of ILD associated with an ABCA3 pulmonary surfactant disorder. In this case, we hypothesise that increased lung stiffness secondary to subpleural fibrosis resulted in delayed re-expansion of the lung into the space occupied by the chest drain. The changes persisted for over 2 weeks, in keeping with the severe restrictive lung disease seen in this patient. To our knowledge, this is the first case report to demonstrate a well-defined tract left behind by

an intercostal drain. Other reports of the 'ghost drainage' sign<sup>4</sup> have demonstrated smaller areas of banded atelectasis following chest drain removal. These images serve to highlight the degree of architectural and fibroelastic disturbance associated with the interstitial lung diseases related to pulmonary surfactant dysfunction.

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