Re-expansion pulmonary oedema in pneumothorax

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DESCRIPTION
Re-expansion pulmonary oedema (REPE) is described in the literature, mostly after drainage of more than approximately 1 L of fluid from the pleural space. REPE can occur after a pneumothorax is drained. This is under-recognised and under-reported.

An 18-year-old male patient presented 10 days after sudden onset pleuritic chest pain and breathlessness. Figure 1 illustrates his chest X-ray that shows a large right primary spontaneous pneumothorax and a small pleural effusion. He was a never smoker with no medical history. He had normal observations that included saturations of 99% on room air. He was a suitable candidate for ambulatory management with an 8 French Gauge (FG) Rocket Pleural Vent, which was inserted in the second intercostal space, midclavicular line. Air audibly exited the pleural space and the patient’s symptoms improved.

A repeat chest X-ray (figure 2) showed good lung re-expansion but new infiltrates in the right upper lobe. Differential diagnoses included re-expansion pulmonary oedema (REPE), pneumonia or pulmonary haemorrhage. He was admitted and observed for 12 hours. Full blood counts and coagulation screens were normal. He coughed red-streaked sputum for the first few hours. These symptoms spontaneously settled, his observations remained stable and he required no supplemental oxygen. He was thus discharged with outpatient follow-up. Further chest X-rays confirmed resolution of the infiltrates and of the pneumothorax over 7 days (figure 3). He is under active surveillance.

Patients with REPE had a longer duration of symptoms (12.8 vs 4.0 days) and larger pneumothoraces (63.5% vs 47.1%) than patients without REPE. However, the risk of REPE is unrelated to the performance of pleural procedure with no statistically significant differences between large bore drains (size greater than 18FG), small bore drains (8–12FG) or needle aspiration if suction is applied.
At first, I was very apprehensive as I wasn’t sure what to expect and also knowing that this process was relatively new. However, the implanting of the device onto my chest was relatively painless and very quick. I was scared after that because I spent the next few hours coughing up yellow and red liquid but the staffs on the ward were very helpful doing frequent check-up and syringing out the liquid that collected in the device. The consultant in the morning explained the process of REPE which I was suffering from by showing me the relevant chest X-rays. I didn’t exactly know what had happened but was relieved to know that I would fully recover. To start off with, I was very sore down my right-hand side and found movement hard but with pain-relief medicines I found movement much easier. The pleural vent quickly helped to inflate my lung; unfortunately when I was capped it off, my lung then deflated a bit again. Overall I’ve been satisfied with the device as it allowed me to go home and get on with life, without being stuck in hospital with a traditional drain. The only thing I’ve found hard is sleeping as it can be bit uncomfortable to sleep however than expected.

The mechanism of REPE is thought to be a combination of damage to the pulmonary interstitium combined with an imbalance of hydrostatic forces. The pulmonary interstitium is a space bordered by visceral pleura and contains a barrier between the alveoli and the capillaries. Changes in intrapleural pressure directly affect that space. Rapid re-expansion of the underlying collapsed lung causes pressure-related mechanical damage to the pulmonary blood vessels leading to increased permeability. Sudden reversal of hypoxic vasoconstriction is a proinflammatory process precipitating oxidative stress and fluid production. The foregoing is combined with increased negative intrapleural pressures that occur when large volumes of air or fluid are removed, reducing the pressure in the pulmonary interstitium. This creates an increased gradient for fluid movement across the alveolar-capillary barrier.

Radiographical changes such as ipsilateral infiltrates after drainage of pleural fluid or air should aid diagnosis. Management is supportive until symptoms such as cough or breathlessness settle, if they arise at all. Cases requiring supplemental oxygenation, non-invasive or invasive ventilation and a death due to progressive cardiorespiratory compromise have been described.1–5

Learning points

- Re-expansion lung injury/pulmonary oedema can occur after drainage of pneumothorax.
- It is more common in large pneumothoraces that have been present for a number of days.
- The pathophysiology is complex and poorly understood.
- It is usually self-limiting but some patients may require supportive treatment.

REFERENCES


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