

# Decompressive talc pleurodesis for hepatic hydrothorax

## Introduction

A 49-year-old man with myelofibrosis had a severe right-sided hepatic hydrothorax requiring repeated pleural drainage. Up to 10 litres of fluid was drained on each admission by intercostal drainage. As talc pleurodesis is generally ineffective with daily pleural fluid outputs of over 500 ml he was treated with surgical placement of a large bore peritoneal drain at the same time as a surgical talc pleurodesis was performed. This resulted in complete and permanent resolution of the hepatic hydrothorax.

## Discussion

Morrow et al (1958) considered the presence of liver cirrhosis essential to the diagnosis of hepatic hydrothorax. Modern authors rightly consider portal hypertension-driven ascites to be the critical factor in causation (Krok, 2014), as it will only develop in patients with pores connecting the peritoneal and pleural compartments. While most cases will still be associated with liver cirrhosis, a small percentage is the result of pre-hepatic portal hypertension (Abrams et al, 2018).

In thoracic surgical practice, talc pleurodesis is carried out for spontaneous pneumothoraces; in pleural effusions it is generally considered ineffective when the daily fluid output exceeds 500 ml. In the current case the intra-abdominal drain artificially decreased this and prevented the effusion re-occurring while pleurodesis was taking effect. No previous report has suggested this addition.

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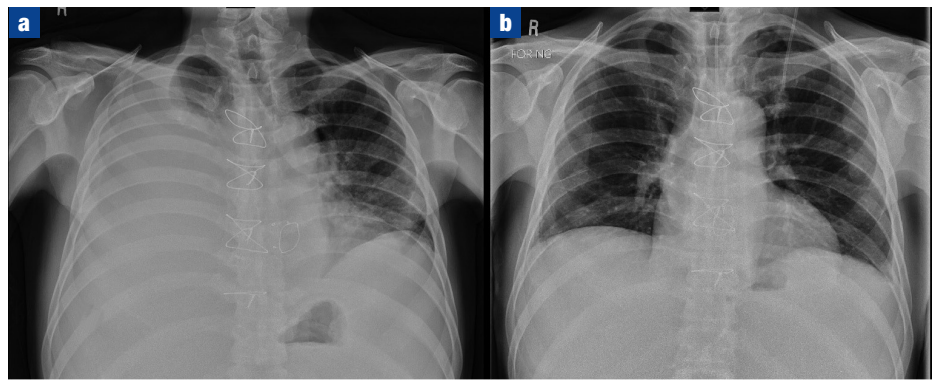
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Indwelling pleural drains are commonly used in managing hepatic hydrothorax where chemical pleurodesis is not considered appropriate, as demonstrated by Shojaee et

**Figure 1. a.** Chest X-ray before relief of right-sided hepatic hydrothorax. **b.** Chest X-ray 3 months after decompressive talc pleurodesis.



## CASE REPORT

A 49-year-old man with myelofibrosis complicated by portal vein thrombosis was admitted to hospital with infective endocarditis of the aortic valve caused by penicillin-sensitive *Streptococcus mitis*. After completing 6 weeks of intravenous antibiotics he was left with severe aortic regurgitation which exacerbated his pre-existing ascites and New York Heart Association grade III dyspnoea. He was known to have portal vein thrombosis and had massive splenomegaly. He underwent surgical aortic valve replacement with a porcine bio-prosthesis.

His postoperative recovery was complicated by a high output right-sided pleural effusion. There had been a moderate-sized right pleural effusion preoperatively. Following surgery he had a persistent output of 2 litres of fluid per day from the right-sided chest drain. This required intravenous fluid replacement with dietary supplementation to make up for ongoing protein and fluid losses. He was discharged 3 weeks following surgery. His subsequent course was one of recurrent massive right pleural effusions requiring admissions to hospital for intercostal drain insertion and fluid resuscitation.

On the first admission 5 litres of ascitic fluid was drained immediately after chest drain insertion and on the second occasion 10 litres was drained (Figure 1a). He was losing weight and his dyspnoea was extreme by the time drain insertion was required. After considering

the options the decision was made to insert an intraperitoneal drain via an appendicectomy incision and simultaneously carry out a right thoracoscopic talc pleurodesis. Thoracoscopy at this time confirmed the presence of a free lung with no evidence of adhesions or trapped lung. The pleural and peritoneal drains were left in place for 2 weeks to permit a sound pleurodesis before removing them and allowing the ascitic fluid to build up again. A wide bore drain was placed in the abdomen rather than an indwelling intrabdominal catheter because of the length of time the drain was to remain in situ and to avoid the increased potential for a small bore catheter to become obstructed during this time. In the first few days following surgery he required careful monitoring of urea and electrolytes and vigorous fluid resuscitation. The peritoneal drain was yielding a steady 2 litres of ascitic fluid daily.

On removing the sutures used to oversew the drain sites 2 weeks later there was a leak of ascitic fluid from the abdominal drain site. A colostomy bag was placed over it and the output from the ascitic fistula was 2 litres per day. He was treated with fluid resuscitation and enteral hyperalimentation via nasogastric tube. After 2 weeks the ascitic fistula closed off spontaneously and he was allowed home. Two months later he felt perfectly well, with a normal chest X-ray (Figure 1b), and was planning on returning to work as a car mechanic.

al (2019). In their paper 28% of patients achieved spontaneous pleurodesis sufficient to allow drain removal. These drains had a 10% risk of infection and 2.5% risk of infection-related mortality, so in this case the authors decided against using small bore catheters. The patient required general anaesthetic; the appendicectomy incision was used because it is renowned for strong wound healing.

This technique proved successful despite difficulties relating to perioperative fluid resuscitation, nutritional support and a temporary ascitic fistula. Ferrante et al (2002) and Papakonstantinou et al (2012) have recommended talc pleurodesis with an emphasis on repeated treatments. Lv et al (2018) found that the success rate was at best around 72–73%.

The traditional surgical approach (thoracotomy, oversewing of diaphragmatic pore, pleural abrasion) promoted pleurodesis but Shields (1994) noted a significant mortality risk. Thoracoscopic oversewing of diaphragmatic pores with talc pleurodesis was recommended by Temes et al (1997) and Cerfolio and Bryant (2006).

Decompressive talc pleurodesis, a simple technique, achieved complete

resolution of hepatic hydrothorax after one treatment. **BJHM**

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**LEARNING POINTS**

- Hepatic hydrothorax is traditionally considered difficult to treat.
- Hepatic hydrothorax is the appropriate diagnostic term in cases of non-cirrhotic portal hypertension where patients have ascites with a communicating pleural effusion.
- Surgical talc pleurodesis can be rendered effective by the addition of a peritoneal drain at the time of surgery. This technique has not been described before.
- Consider decompressive talc pleurodesis for hepatic hydrothorax.

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# Forthcoming case reports

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**Epididymo-orchitis masquerading as an irreducible inguinal hernia**

**Case Report**

## A feverish junior doctor with a diagnosis not to be missed

**Introduction**

*P*neumonia is a clinical presentation often after returning from the endemic setting. This has been established in numerous forms of the parasite residing in the liver and spleen, and acute infection was first described in 1945. In light of this, the authors advise exposing those of recent travel within the last 3–5 years, rather than just the 12 months as is currently taught, to prevent acute pneumonias being overlooked.

During 2016, there were just 136 cases of imported malaria in the UK (Health Protection Agency, 2015), and the clinical spectrum of imported malaria is broad, which makes recognition of the new illness of people travelling to malaria-endemic countries continues to increase (Health Protection Agency, 2015).

**Conclusions**

Given the authors' experience and the changing medical epidemiology, the value of reporting blood film examination of febrile travellers is highlighted by this (2016).

**Case Report**

## Acute interstitial nephritis caused by two different proton pump inhibitors

**Introduction**

Acute interstitial nephritis is an important cause of acute kidney injury and drug account for about 85% of cases. Since 1992 it has been established that proton pump inhibitors can cause acute interstitial nephritis.

This case report describes a 47-year-old woman who presented with acute kidney injury secondary to proton pump inhibitor (esomeprazole) induced acute interstitial nephritis, with a further episode of acute interstitial nephritis when switched with a different proton pump inhibitor (pantoprazole). Previous case studies have shown that coadministration of the same proton pump inhibitor caused recurrence of renal dysfunction.

This is the first report of acute interstitial nephritis occurring secondary to two different proton pump inhibitors in the same individual. This strengthens the hypothesis that proton pump inhibitor-induced acute interstitial nephritis is a class effect and should raise caution when clinicians consider switching another proton pump inhibitor in a patient who has previously been diagnosed with proton pump inhibitor-induced acute interstitial nephritis.

**Discussion**

Proton pump inhibitors are one of the most commonly prescribed drug classes in the United States, the Food and Drug Administration approved the sale of proton pump inhibitors 'over the counter'. The increased availability of these medications has had an important consequence and dissemination of the side-effect profile. While proton pump inhibitors induced acute interstitial nephritis is a rare idiosyncratic reaction, the prevalence of proton pump inhibitors prescribing makes it an increasing problem (Saxena et al., 2007).

Proton pump inhibitor-induced acute interstitial nephritis has been well described in many series and four case reports have shown that the same proton pump inhibitor (esomeprazole) on re-introduction of the same proton pump inhibitor (Chakrasena et al., 1995; Balak et al., 1999; Landry et al., 1996; Balak et al., 2006). This article presents the first case of proton pump inhibitor-induced acute interstitial nephritis caused by two different proton pump inhibitors.

This is important because it demonstrates that proton pump inhibitor-induced acute interstitial nephritis is a class effect, despite