Editorial

Patient centred care for spontaneous pneumomediastinum: a step in the right direction

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René Laennec, the 19th century French physician, invented the stethoscope in 1816 and is widely credited for naming the various clinical characteristics such as 'rales, rhonchi, crepitance, and egophony' associated with respiratory pathophysiology (1). He also described pneumomediastinum (PNM) as an abnormal presence of air or gas within the mediastinum in 1827 (2). Spontaneous PNM occurs with no definite cause (trauma, infection, perforation in the respiratory system or gastro-intestinal tract) and the first case series was described by Hamman in 1939 (3).

Hamman's sign or crunch is associated with approximately 20% of cases—the sign constitutes of precordial systolic crepitations synchronous with the cardiac cycle and are increased during inspiration (3). The mechanism of PNM was described by Macklin and Macklin (4). Rupture of peripheral alveoli follow from an increased pressure gradient between the alveolus and the interstitial space. Air then dissects along the sheaths of the bronchovascular bundles into the mediastinum. Air can move along various fascial planes into the chest wall, neck or face. Air can also tear the relatively thin mediastinal pleura to cause pneumothoraces, pneumopericardium pneumoperitoneum. This is often a benign process in spontaneous PNM but could be devastating if pericardial compression impaired venous return or a tension pneumothorax developed. Numerous cases series attest to this, although care should be taken to exclude cases associated with Covid-19 as the pathophysiology and outcomes are probably different (5).

Compression of the bronchovascular bundles could also lead to a splinting effect leading to hyperinflation and poor ventilation/perfusion mismatch. Discovery of PNM usually prompts the exclusion of other causes, namely gastrointestinal (oesophageal perforation), due to the associated increased morbidity and mortality. Cases of PNM thus usually necessitate admission, observation and often further costly diagnostics (6).

Previous case series have spanned decades but have been limited to small numbers. For example, Carceres et al in 2007 described 28 patients, presenting mostly with chest pain, dyspnoea and subcutaneous emphysema and having main triggers as vomiting or asthma (7). All investigations such as bronchoscopies or oesophageal studies were negative. Koullias *et al.* described PNM in a predominantly young male cohort with the main culprit as illegal drug use but also mentioned asthma and vomiting (8). Mean length of hospital stay was 2 days and all investigations were again negative. Campillo-Soto *et al.* (9), Macia *et al.* (10) and Mondello *et al.* (11) reported similar findings in 36, 41 and 18 cases respectively, further underlying the dramatic presentation but ultimately benign nature of this condition. The largest case series is from Japan, Yamairi *et al.* with 71 cases and has similar results (12).

This is where the study by Morgan *et al.* comes in (13). One of the larger case series of PNM in the literature, compiled over nearly 20 years. As the patient characteristics

are similar to previously described cohort studies, this study can be generalised. The findings of tachycardia and leucocytosis are not out of keeping with common knowledge but it is the finding that those patients with PNM with no history of retching or vomiting, under the age of 40, with a known risk factor such as asthma or smoking, can simply be observed with no detrimental effect. This is a simple observational retrospective study using a descriptive methodology (and the authors take care not to infer significant practice changing conclusions) and thus has some significant limitations, namely the reliance on case note review and the lack of standardised approach on who to investigate further for oesophageal perforation. Healthcare utilisation was alluded to but a formal cost analysis was not performed, and internal correspondence suggests that this was beyond the ability of the study authors. Nevertheless, this is an important study in a relatively under-studied field which aims to stratify those who could be observed versus those who need further investigations. In this era of personalised healthcare, Morgan *et al.* pave the way for further work on this topic. What is required is a large multicentre, international trial looking at all comers with PNM, and applying those characteristics that they found in their study to have 2 groups of patients (those being observed and those being investigated further) and eventually collate outcomes. A simple derivation score could be produced then validated, both internally and externally. Associated healthcare costs would be difficult to measure across various health systems, but could be individually worked out. Societies such as the British Thoracic Society, European Respiratory Society and the Interventional Pulmonary Outcomes Group (based in the United States), could help with sponsorship and allowing various centres to link in together. Only then can these findings be widely adopted.

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Footnote

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