Cervical subcutaneous emphysema and pneumomediastinum after sneezing

A 20-year-old Chinese man, with a history of chronic rhinitis, was admitted to our hospital in July 2011, with sudden severe non-radiating retrosternal chest discomfort. He had had a recent upper respiratory tract illness with sore throat and running nose that induced forceful sneezing. He denied any accidental trauma to the chest. On admission, the respiratory rate was 18 breaths/min, oxygen saturation was 98% on room air, blood pressure 110/60 mm Hg, pulse rate 80 beats/min, and body temperature 37°C. Physical examination revealed bilateral cervical subcutaneous emphysema, spreading to both supraclavicular fossae, while oro-laryngopharyngeal, chest and cardiovascular examination revealed no abnormality. Findings from blood test (including complete blood picture, and renal and liver function test) were unremarkable. The chest X-ray (CXR) showed subcutaneous emphysema in the supraclavicular fossae, with air tracking into the neck area bilaterally and a strip of para-aortic air on the left side (Fig 1). The trachea was central and the lung fields and cardiac silhouette were normal. Contrast-enhanced neck and thoracic computed tomography revealed extensive cervical surgical emphysema and pneumomediastinum (Fig 2). There was no abnormal soft tissue swelling or mass noted in the oro-nasopharynx. The diagnosis was cervical subcutaneous emphysema and pneumomediastinum was caused by forceful sneezing. The patient was managed conservatively without complications and the follow-up CXR 2 weeks later showed complete resolution of the subcutaneous emphysema and pneumomediastinum.

Pneumomediastinum was first described by Laennec in 1819 as a consequence of traumatic injury. It is classified as either spontaneous pneumomediastinum (SPM) that is not associated with penetrating chest trauma; invasive (after cardiothoracic procedures or interventions); and secondary—caused by trauma, neoplasm, anaerobic infection, or rupture of the oesophageal mucosa (Boerhaave’s syndrome).1

FIG 1. Chest X-ray shows subcutaneous emphysema in the supraclavicular fossae, with air tracking into the neck area bilaterally and a strip of para-aortic air on the left side.

FIG 2. (a) Coronal reformat image of the thorax (lung window) shows extensive surgical emphysema at the lower neck and pneumomediastinum surrounding the trachea and subcarinal region. (b) Coronal reformat, axial and sagittal reformat computed tomographic images (lung window) of the thorax show extensive surgical emphysema and pneumomediastinum. No significant complicated mediastinal collection or infection was associated.
Hamman, after whom the Hamman sign is named, was the first to report SPM in 1939. The generally accepted explanation for its development is the Macklin effect, also described in 1939. The sequence of events is as follows: (1) alveolar rupture, (2) air dissection along the bronchovascular sheath, and (3) free air reaching the mediastinum along the pressure gradient developed during the respiratory cycle. The dissection of free air may not be confined solely to the mediastinum. Zylak et al noted that the mediastinum communicates with the submandibular space, the retropharyngeal space, and vascular sheaths within the neck; pneumomediastinum can therefore cause cervical emphysema, as in our patient. There are two other routes of communication with the retroperitoneum, namely: via a tissue plane extending through the sternocostal attachment to the diaphragm, as well as via periaortic and periesophageal fascial planes. These communications may facilitate development of pneumopericardium, pneumothorax, subcutaneous emphysema, pneumoperitoneum, or pneumoretroperitoneum.

Predisposing factors to SPM are Valsalva manoeuvres: defaecation, childbirth, vomiting, and balloon or glass blowing. The normal imaging appearance of the neck and lung parenchyma in our patient with an underlying rhinitis history suggests that development of SPM gave rise to cervical subcutaneous emphysema due to a high pressure Valsalva manoeuvre generated by forceful sneezing with bilaterally blocked nostrils. In fact, SPM associated with sneezing has already been described in the literature.

Patients with SPM should be closely monitored to anticipate the development of more serious complications, such as tension pneumomediastinum, pneumothorax, or pneumopericardium. In general, most patients have an uneventful clinical course and SPM subsides spontaneously without any residual disability.

References