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Commentary

J E Harvey

These two case reports illustrate some interesting points in both the aetiology and management of pneumothorax.

Fortunately pneumothorax is rare during pregnancy and usually occurs in patients with normal lungs, apart of course from any associated leaking subpleural bleb or bulla. Lymphangiomyomatosis, neurofibromatosis, and choriocarcinoma are rare conditions that may cause a haemorrhagic or chylous pleural effusion during pregnancy, with or without an associated pneumothorax. The changes in alveolar ventilation mentioned, and particularly the increase in intrathoracic pressures generated by repeated Valsalva manoeuvres during labour, will all tend to increase the chance of rupture of weak areas on the visceral pleural surface. More than one third of pneumothoraces occur within a few weeks of term so that a chest drain can be inserted to cover the increased ventilation and raised intrathoracic pressures associated with labour, followed if necessary by definitive surgery after delivery.

The case reported by Levine is unusual with a pneumothorax occurring at 32 weeks, so that once it had recurred despite adequate intercostal drainage and a well positioned second drain continued to bubble, the patient was faced with either two months of intercostal drainage or immediate thoracotomy. The use of a flutter bag system, however, allowed outpatient management until normal labour could proceed, subsequently followed by elective ligation of the apical bulla. I have always had reservations about the use of flutter bags on an outpatient basis in case of sudden failure of the flap valve system and development of tension pneumothorax. However, the system used in this case is well designed and seems safe and would certainly have advantages over a urinary catheter and a bottle of Evian water!¹

It is reassuring to note the absence of any infection of the skin or pleura despite the use of the flutter bag for eight weeks. Once an intercostal drain has been in position for over a week a tract often develops around the insertion hole allowing air to be sucked in during inspiration which may necessitate reinsertion of the drain or encourage earlier definitive surgery.

This does not, however, appear to have been a problem in the case described.

The length of time air continues to bubble rather than the initial size of a pneumothorax seems to determine the likely need for surgery,² and this case would normally have required much earlier surgical intervention. Because of the high risk of enlarging or tension pneumothorax developing during labour, prior thoracotomy and resection of apical blebs has been advocated and even intrapartum thoracotomy has been safely undertaken in a few cases. There is no evidence of any teratogenic effect of anaesthetic drugs nor of any increased risk of spontaneous abortion or premature labour following general anaesthesia in pregnancy, but there have also been no reports of pneumothorax or pneumomediastinum causing maternal or fetal death since 1908 and 1949, respectively.³ Should observation, simple aspiration, or intercostal tube drainage fail, then within a few weeks of term intercostal drainage – perhaps using the flutter bag system – should be continued until delivery to guard against the development of tension pneumothorax during labour. Pneumothorax occurring during pregnancy is associated with a higher than usual recurrence rate so that surgical management should always be considered, especially in those (about 20%) who have a previous history of pneumothorax. In those cases who appear to resolve spontaneously or following aspiration or drainage, careful monitoring during the subsequent labour is essential.

In the case described by Levine a choice had to be made between ligation of a bulla and pleurectomy through a thoracotomy or thoracoscopic approach eight weeks from term, or leaving a chest drain in position for eight weeks pending definitive surgery postpartum. The risk of surgery and general anaesthesia at 32 weeks into a pregnancy was deemed to be greater than the risk of a further eight weeks of intercostal drainage using the flutter bag system for the rest of the pregnancy and, of course, throughout labour. This was a difficult choice and I suspect opinions would be divided as to the relative risks to mother and baby, though clearly the results in this case are reassuring.

Both pregnancy and labour may be complicated by the development of pneumomediastinum, but Torres-Melero and colleagues remind us of one of the many other unusual causes of this condition which they also describe as iatrogenic mediastinal emphysema. The presence of air in the mediastinum should always alert clinicians to the possibility of

mediastinitis, especially if it is caused by oesophageal rupture or, as in this case, when there may be a communication between the mediastinum and the oropharynx. Pneumomediastinum may simply follow the hyperventilation that occurs in pregnancy, but has also been associated with many other situations associated with hyperventilation from diabetic ketoacidosis to sexual intercourse. The extra pressures generated by valsalva manoeuvres involved in labour, severe asthma, belching, or the inhalation of cocaine or marijuana may also result in air leaking into the mediastinum as, too, can any cause of excessive vomiting such as hyperemesis gravidarum or abuse of "Ecstasy".

The high pressures generated by the dental drill during tooth extraction forced air into the neck and mediastinum – a complication that has also been described following oeso-

phagoscopy, both with and without actual rupture of the oesophagus (Boerhaave's syndrome). Pneumomediastinum may also occur following laparoscopic surgery, transbronchial biopsy, mechanical ventilation, and even arthroscopy of the shoulder! In all cases it is wise to consider prophylactic antibiotic therapy with good Gram negative cover and to consider a radiological search for an oesophageal leak as was undertaken in this patient. Fortunately most cases will resolve spontaneously within a week and although, as the authors comment, surgical intervention may be required, this is rarely necessary.

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Chemodectoma of the trachea

Department of Radiology
T M Y S Sing
K P Wong
N Young

Tom M Y S Sing, Kai Ping Wong,
Noel Young, Paul Despas

Department of Respiratory Medicine
P Despas

Westmead Hospital,
Westmead, NSW 2145,
Australia

Correspondence to:
Dr T M Y S Sing.
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Abstract

The case history is described of a patient with lower tracheal chemodectoma who presented with haemoptysis. After conservative management for eight years she represented with airways obstruction. Preoperative tumour embolisation was followed by laser ablation, stenting, and radiotherapy.

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Chemodectomas are rare tumours that arise from chemoreceptor cells of the paraganglia.¹ We believe this is the first case report of tracheal chemodectoma in the English literature.

Case report

A 77 year old woman presented in 1983 with haemoptysis. Bronchoscopic examination revealed a tumour arising from the posterior wall of the trachea at the level of the carina, extending into both main bronchi. A biopsy specimen showed a chemodectoma. Surgery was considered impossible and she was conservatively managed. She had occasional haemoptysis but little change in bronchoscopic appearances.

In June 1991 she represented with progressive dyspnoea and worsening haemoptysis. A computed tomographic (CT) scan showed a carinal mass extending into the proximal 1 cm of both main bronchi with spread into the subcarinal space, intimately related to the oesophagus and thoracic aorta with no mediastinum lymphadenopathy (fig 1). Spirometric tests showed marked impairment (forced expiratory volume in one second (FEV₁)/forced vital capacity (FVC) = 1.0/1.9 l). Bronchoscopic examination again revealed a tumour arising from the lower trachea, extending into both main bronchi. The more distal bronchi were normal.

Bronchial arteriography showed a hypertrophied upper right bronchial artery supplying a 2 cm vascular tumour (fig 2). The artery was embolised with a mixture of Gelfoam and Ivalon (polyvinyl alcohol) particles to control haemoptysis and to reduce the risk of bleeding during laser ablation.

Six days later rigid bronchoscopy and laser ablation of the endotracheal component of the tumour in the right main bronchus and lower trachea was performed. The left main bronchus was also partially obstructed but was not treated as the mucosa was soft and fleshy and the bronchial wall appeared thin with most of the obstruction due to extrinsic compression.

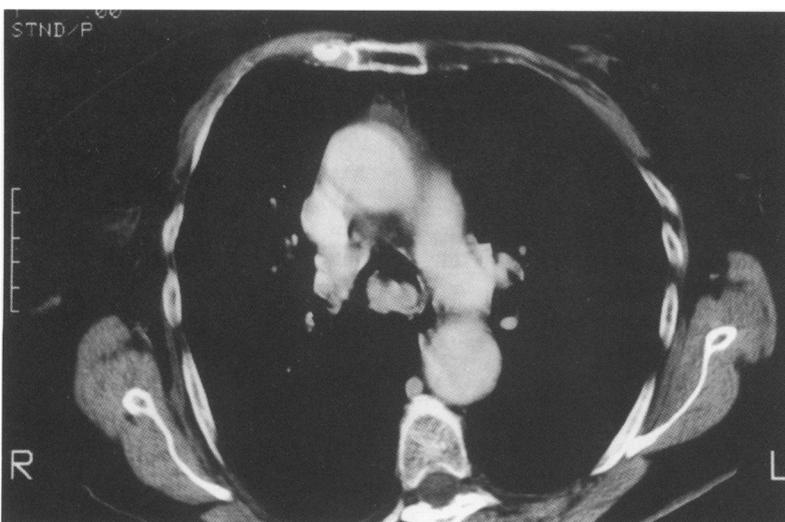


Figure 1 Thoracic computed tomographic scan (mediastinal window) showing tumour spread into the subcarinal space.