Whilst our experience is limited in this type of presentation, of importance was that the patient survived 17 months from the time of diagnosis, and enjoyed several intervals of remission of pain and periods at home. The repeated 1% phenol irrigations were carried out with ease, sepsis was controlled, pain markedly reduced, and fluid production by the developing tumour was retarded.

It is possible that in this application the use of a phenol irrigation system in clinical management may play a useful role in reducing morbidity.

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Bilothorax - an unusual problem

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Keywords: bilothorax; pleural effusion; bile

Since the classic paper by Meigs and Cass[1] relating the presence of ascites with pleural effusion to fibromas of the ovary there have been reports that large volumes of intra-abdominal air[2], ascitic fluid[3] or blood[4] are capable of directly traversing the diaphragm to enter the pleural space. I present a case of biliary peritonitis secondary to the perforation of the afferent limb of a gastrojejunostomy causing bilateral pleural effusions containing litres of bile (bilothorax).

Case report

A 63-year-old man presented with sudden onset of acute epigastric pain radiating to the back, nausea and vomiting. There was no history of heavy alcohol intake or drug ingestion. In 1945 partial gastrectomy was performed for perforated duodenal ulcer. On examination he was distressed, in severe pain but apyrexial, and not icteric. Pulse was 70/min and blood pressure was 180/90 mmHg.

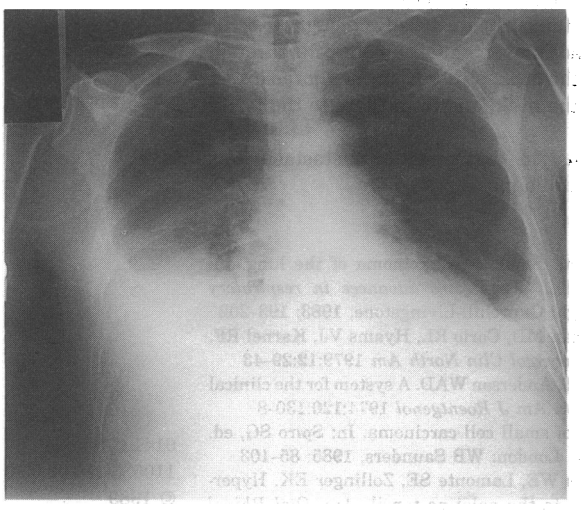

Figure 1. Chest radiograph revealing bilateral pleural effusions containing bile - a bilateral bilothorax

Generalized abdominal tenderness and guarding were present. Serum amylase was greater than 4000 (normal <220 units/l). Full blood picture, electrolytes, urea, calcium glucose and blood gasses were all normal. X-rays showed no subdiaphragmatic air.

A provisional diagnosis of acute pancreatitis was made and resuscitation commenced. No improvement occurred over the next 48 h and his serum amylase remained elevated. An ultrasound revealed a normal biliary tree, and a distended thickened loop of bowel in the epigastric region. Barium meal showed no recurrent ulceration and no filling of the afferent loop. Over the next 12 h the patient developed bilateral pleural effusions (Figure 1), deteriorating respiratory function and his serum amylase suddenly fell to normal. Needle aspiration revealed bile in both pleural cavities. Bilateral chest drains were inserted and over a litre of bile drained from each pleural cavity.

Emergency laparotomy revealed a perforation of the afferent loop 4 cm distal to the gastrojejunostomy. There was no evidence of recurrent ulceration. The ischaemic segment was resected and a Roux-en-Y anastomosis constructed. Postoperative recovery was uneventful.

Discussion

Although the original report of Meig's syndrome concluded that the peritoneal and the pleural fluid had the same aetiology, no logical cause was given for the pleural effusion. Direct extension seemed the most likely answer, but transfer via lymphatic channels was also suggested[5].

The finding of spontaneous pneumothorax secondary to therapeutic pneumoperitoneum supports the theory of direct transdiaphragmatic passage either by following the connective tissue sheaths of the oesophagus and great vessels into the mediastinum and then rupturing into the pleural space or by direct communication through a diaphragmatic defect. Defects of the diaphragm maybe congenital, Bochdalek's defect being the most common. In addition wide spaced openings between diaphragmatic muscle bundles have also been demonstrated[6]. A congenital diaphragmatic defect was not demonstrated in this case and the most plausible explanation for the presence of bile in the pleural cavities is transdiaphragmatic passage from the peritoneal cavity either via defects between muscle bundles or via the lymphatics. The one significant contributing factor may have been his previous gastrectomy which may have facilitated passage of bile via the oesophageal hiatus. It is important to recognize that large volumes of free intra-abdominal contents are capable of directly traversing the diaphragm to enter the pleural space and compromise the respiratory status of the patient. I present a case involving bile as the culprit fluid producing a 'bilothorax'. I can find no previous reports of such a case.

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Acknowledgment: I thank Mr J D Maynard, Consultant Surgeon, for permission to report this case.

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Tonsillar metastasis at presentation of small cell carcinoma of the lung

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Keywords: tonsillar metastasis; small cell carcinoma

Small cell carcinoma of the lung is a common, highly malignant neoplasm and accounts for approximately 25% of all bronchial carcinomas. In the majority of cases, the tumour has spread beyond the lung before the diagnosis is made. In spite of significant advances in treatment, a cure for this disease has yet to be found[1].

Secondary deposits from a primary neoplasm are responsible for only a small number of all tonsillar tumours. In a series of 1547 tonsillar tumours, only 12 were due to metastasis, the rest resulting from either primary carcinoma or lymphoma of the tonsil[2].

Case report

An 83-year-old woman presented with a one month history of increasing cough, shortness of breath and weight loss. She had occasional haemoptysis but no chest pain. There was no dysphagia. She smoked 10 cigarettes a day.

Physical examination of the thorax revealed slight shift of the mediastinum to the right and diminished air entry over the right hemithorax. Examination of the head and neck disclosed a large lobulated mass arising from the left tonsil, displacing the soft palate and extending across the midline of the oropharynx (Figure 1). An enlarged firm lymph node was palpable at the angle of the left mandible. There were no other physical signs.

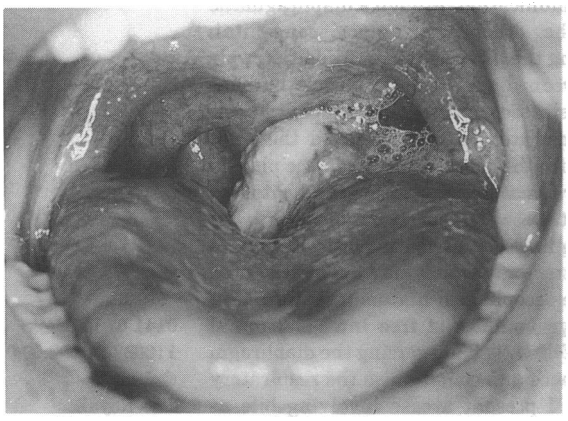

Figure 1. Tumour deposit arising from left tonsil

A chest radiograph confirmed the shift of the mediastinum and demonstrated a large mass arising from the lower lobe of the right lung.

In view of its large size, the tonsillar mass was excised under general anaesthesia. Histological examination of the tissue removed demonstrated invasion by small cell carcinoma.

In view of her age and general condition, fibreoptic bronchoscopy was not performed.

The tonsillar area was treated with a palliative course of external radiotherapy and the patient was admitted to a hospice unit where she subsequently died. Postmortem examination confirmed the presence of a small cell carcinoma of the right lung, the histology of which was similar to that of the tonsillar metastasis.

Discussion

Small cell carcinoma is renowned for its propensity to disseminate widely throughout the body at an early stage in its clinical course[3]. Tissues that are particularly susceptible include liver, abdominal lymph nodes, bone, brain, the adrenal glands, skin, the kidneys and the pancreas[4].

The palatine tonsil is a rare site in which to find metastatic tumour deposits, and as such is not discussed in standard textbooks of pathology. In a review of 76 cases of primary neoplasms complicated by tonsillar metastasis, 12 were found to be due to carcinoma of the bronchus. Small cell carcinoma was the predominant histological type. In 10 of these 12 patients, the metastasis was unilateral and in 2 bilateral. In 10 of the 12 cases there was evidence of metastasis to other tissues, and in all cases the tonsillar metastasis developed following presentation[5].

Other tumours recorded as having been complicated by tonsillar metastasis include carcinoma of the breast, carcinoma of the stomach, hypernephroma, seminoma, melanoma and carcinoma of the rectum[5].

Spread of secondary tumour to the tonsil is thought to occur as a result of retrograde movement of tumour cells through lymphatic vessels of the neck, either from the thoracic duct or from the veins of the neck, and from there to the tonsil itself.

This case is unusual in that tonsillar metastasis was present at the time of diagnosis.

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