Isolation of Klebsiella pneumoniae from an hepatic inflammatory pseudotumour

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Inflammatory pseudotumours (IPT) are rare solid inflammatory mass lesions which have been described in several organs and given different names, including plasma cell granuloma (1). Hepatic IPT are very rare with only 23 case reports in English language literature (2-9). The etiology and pathogenesis of IPT has not been determined. Janigan and Marrie (10) isolated Coxiella burnetii from an IPT of the lung. However, until the case here presented, no microorganisms have been identified from hepatic IPT. The authors present the case of a patient with hepatic IPT from which Klebsiella pneumoniae was isolated.

CASE PRESENTATION
A 42-year-old male coal miner with chronic obstructive lung disease and controlled hypertension presented to his family physician with left chest pain and fever of several days duration. Clinical and radiological findings confirmed bilateral lower lobe pneumonia. Sputum cultures grew K pneumoniae and blood cultures were negative on three occasions. Despite two weeks of treatment with tetracycline, clinical improvement was slow and the patient...
was referred to hospital for further evaluation. There was no history of diabetes and physical examination on admission was normal.

The following were the abnormal laboratory findings, with the normal ranges (where appropriate) shown between parentheses: white blood cells $11.9 \times 10^9/L$; erythrocyte sedimentation rate 115 mm/h; alkaline phosphatase 562 U/L (30 to 104); alanine aminotransferase 97 U/L (1 to 41); aspartate aminotransferase 48 U/L (8 to 29); lactate dehydrogenase 335 U/L (117 to 259); gamma glutamyltransferase 585 U/L (0 to 40). The rest of the routine biochemical and hematological profiles were normal.

Chest x-ray films demonstrated bilateral lower lobe segmental atelectasis. Ultrasound of abdomen revealed a hypoechoic mass in the left lobe of liver. A computed tomography scan demonstrated the liver's left hepatic lobe to be enhancing along the periphery but not in the centre (Figure 1A). A technetium scan (Figure 1B) identified the lesion as 'cold' while the gallium scan depicted it as 'hot'. Selective left hepatic angiography suggested increased vascularity of the rim with relative hypovascularity. The radiological findings were considered inconclusive. As the lesion was suspected to be malignant, a left hepatic lobectomy was performed. Intraoperative biopsy for cultures taken from the lesion grew K pneumoniae. The patient had an uneventful recovery and is alive seven years later.

The lesion was approximately $6 \times 5 \times 3$ cm, well demarcated, yellowish, rubbery and contained small areas of necrosis and hemorrhage (Figure 2). The surrounding liver parenchyma was normal. Histologically, the lesion was composed of lymphocytes, plasma cells, foamy histiocytes, hemosiderin-laden

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**Figure 1**
- A, Ultrasound showing hypoechoic mass.
- B, Technetium scan demonstrating a cold solitary lesion occupying most of the left lobe.

**Figure 2**
- Cut surface of left hepatic lobectomy specimen showing a well circumscribed $6 \times 5 \times 3$ cm yellowish mass surrounded by partially fixed normal liver parenchyma.

**Figure 3**
- A, Inflammatory pseudotumour composed of a mixture of lymphocytes, plasma cells, macrophages, fibroblasts and capillaries. Hematoxylin and eosin.
- B, Sclerosing phlebitis with partial obliteration of a portal vein. Hematoxylin and eosin.
macrophages and fibroblasts with collagen (Figure 3). Scattered foci of necrosis were noted but there was no cavitating abscess. Some veins within the lesion showed sclerosing phlebitis (Figure 3B).

**DISCUSSION**

The etiology of IPT is unknown. There is, however, strong clinical circumstantial evidence indicating it is infectious in origin. Patients often present with fever, weight loss, leukocytosis, elevated erythrocyte sedimentation rate and positive C-reactive protein (7,9). Microorganisms from the gut may be responsible for hepatic IPT (9) but until the case under discussion, no microorganisms have been isolated from an hepatic IPT. The mechanism of IPT formation remains obscure.

Histologically, the lesion consists of a chronic inflammatory process in which lymphocytes, plasma cells, macrophages and fibroblasts abound while suppuration with liquefactive necrosis is minimal or absent. The isolation of *K pneumoniae* from the patient’s sputum and subsequently from the hepatic IPT suggests a low grade infection with chronic inflammatory tissue response predominating. The concept of chronic low grade infection may explain why previous attempts to isolate microorganisms from hepatic IPT have been unsuccessful.

The histological distinction between hepatic IPT and resolving hepatic abscess may be difficult (9). Accordingly, the term ‘inflammatory pseudotumour’ likely is best used in the radiological and macroscopical context for space-occupying solid inflammatory lesions mimicking a neoplasm. This latter aspect of hepatic IPT has often led to the diagnosis of malignancy and extensive hepatic resection (7,9). Needle biopsy under ultrasonography guidance would be the procedure of choice for identifying the lesion, thus avoiding radical surgery.

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**REFERENCES**
