Letters to the Editor

Diagnosis of anaplastic pancreatic cancer with multiple liver metastases

Sir,
Pyogenic liver abscess is a rare but highly lethal disorder. The aetiology of liver abscess has undergone a significant change over the past 50 years; nowadays, benign and malignant biliary tract obstructions such as cholecystolithiasis and cholangiocarcinoma are the main causal factors leading to liver abscesses.^{1,2} Since computed tomography (CT) and magnetic resonance imaging (MRI) became available, we have been able to diagnose this life-threatening disease accurately. However, differential diagnosis between liver abscesses and other disorders such as biliary cystadenocarcinoma and metastatic liver tumours is still problematic. In this paper we report a case of anaplastic pancreatic cancer with multiple liver metastases, which is difficult to differentiate from liver abscesses.

A 62-year-old woman was admitted to our hospital with anorexia and high-grade fever lasting for 3 weeks. On physical examination, she appeared to be cachectic with high body temperature (39.5°C), and a firm liver was palpable 5 cm below the costal margin. Laboratory examinations revealed normocytic anaemia (haemoglobin 9.0 g/dl), severe leucocytosis (white blood cell count $27.7 \times 10^9/l$ and elevated serum C-reactive protein level (26.5 mg/dl), but aspartate transaminase, alanine transaminase and alkaline

phosphatase levels were within normal ranges. CT of the abdomen showed multiple low-density areas with contrast-enhanced rims in liver and spleen (figure), which were also detected as hypo- and markedly hyperintense signal lesions in T1- and T2-weighted MRI, respectively.

These clinical and radiographical findings yielded a tentative diagnosis of pyogenic liver and spleen abscesses. However, wide-spectrum antibiotics proved to be ineffective and ultrasound-guided percutaneous drainage of the abscesses was unsuccessful. Later, tumour marker levels were found to be extremely elevated (CA19-9 28 000 U/ml and CEA 18.6 ng/ml), and anaplastic cells were detected in the fluids aspirated from the splenic lesion, suggesting the presence of pancreatic tumours. Two weeks later, massive haematemesis and melaena due to a gastric ulcer were observed, and eventually she died of uncontrollable bleeding. Pathological dissection confirmed that she was suffering from anaplastic pancreatic cancer with multiple liver metastases and direct splenic and gastric invasion.

In conclusion, we should always remember anaplastic pancreatic cancer when considering the differential diagnosis of pyogenic liver abscesses.

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Systemic capillary leak syndrome: where does the fluid go?

Sir,
Systemic capillary leak syndrome is a rare condition characterised by capillary hyperpermeability and clinically by abdominal pain, vomiting, hypotension and angio-oedematous swellings. Here we report on the post-mortem findings of a 53-year-old man who had been treated with epoprostenol in the past,¹ but who eventually succumbed during a particularly severe attack.

After his death, external examination showed that he had a distended abdomen and mild pitting oedema of both lower limbs below the level of the knees; the subcutaneous fat of the abdominal wall and legs was soft and oedematous. Internal examination of the thorax revealed a heart with mild left ventricular hypertrophy (15 mm wall thick-

ness), possibly a reflection of the additional strain placed on the myocardium during hypotensive episodes.² There was marked congestion of the lungs which, on histological examination, was found to be due to pulmonary oedema rather than bronchopneumonia. The literature suggests that this is more likely to be due to fluid overload incurred by resuscitation with fluids than to capillary hyperpermeability, which seems to spare the pulmonary vasculature.^{3,4}

Of particular interest was the macroscopic appearance of the small and large bowel, ie, marked distension with thickening of their walls. This has never been reported before, and is likely to account for the colicky abdominal pain that so often accompanies attacks of systemic capillary leak syndrome. It is also interesting to note that there was no free fluid within the peritoneal, pleural or pericardial cavities which suggests that the hypotension is the result of extravasation from capillaries within viscera rather than from serosal surfaces. An unexpected finding of this post-mortem was the presence of a gallstone impacted at the ampulla of Vater associated with marked dilatation (up to 1 cm) of the hepatic and common bile ducts. There was also evidence of mild ascending cholangitis. It is possible that this could have precipitated the final hypotensive episode and I note the report of Atkinson *et al*⁵ of 'periportal hepatitis' and cholelithiasis in a similar patient. We realise that gallstones alone could cause the aforementioned abdominal pain but against this there are several other reported cases of systemic capillary leak syndrome with abdominal pain in whom stones have not been identified.

Given the above findings, and in order to further our understanding of this curious condition, we suggest that future cases undergo imaging during hypotensive episodes. Computed tomography scanning, which is generally accepted to be a sensitive method for the detection of thickened bowel loops, could be used to establish the pattern and frequency of bowel involvement in living subjects with systemic capillary leak syndrome.

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Figure Plain (A) and contrast-enhanced (B) CT of the abdomen.