of Walton (1963) this case should probably be described as a spontaneous rupture of a pathological spleen.

Although this patient was debilitated there was no history of trauma, indeed if the time of the first collapse is considered to be the time of rupture, then he was in bed. However, it may be that at the time of his third admission with faintness, the haemorrhage had already commenced and the collapse represented an extension of this process. Although pain was not a prominent feature of his illness, it did not occur at all until after his subsequent collapse.

At the outset, the clinical presentation was of retroperitoneal haemorrhage and in this respect was similar to the case recorded by Thompson in 1969. However, the development of large bowel obstruction is unusual. Ileus is a well recognized complication of retroperitoneal haemorrhage but bowel activity remained throughout this man’s illness and before operation it appeared most likely that a submucosal haemorrhage had caused a local obstruction. At operation no such cause was found although there was a large volume of free blood in the peritoneal cavity and the obstruction appeared to be due to the large haematoma at the hilum of the spleen. For this reason the bowel was not opened. Furthermore, postoperatively there was no suggestion of recurrence of the obstruction.

This case demonstrates the varied clinical presentation with which a ruptured spleen may present and reminds one of its potential hazard in the management of the leukaemias and lymphomas.

References


Case reports

Hepatoma causing a massive tumour embolus

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Summary

A case of hepatoma is reported, with a typical clinical setting but a most unusual and dramatic final episode. A massive tumour embolus weighing 30 g became arrested on the tricuspid valve, resulting in acute circulatory failure.

Introduction

Intravascular growth is an important feature of hepatoma (Edmondson, 1958). Although hepatoma has been reported to extend directly into the right atrium (Gregory, 1939), massive tumour embolism such as encountered in this case is not cited in the world literature.

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Case report

A 66-year-old man was admitted with a history of recurrent leg swelling for the past 3 years. He had suffered increasing abdominal and leg swelling during the last 5 months with the development of icterus. He was found to have hepatosplenomegaly which had been noted 3 years earlier, during which time he was taking diuretics. Myocardial ischaemia with thyrotoxicosis had been treated 10 years earlier. He was a mild cigarette smoker and never a heavy drinker. Chest X-ray showed miliary shadowing.

Examination showed an ill-looking jaundiced man with generalized wasting. Ascites and massive hepatosplenomegaly were present. Liver cirrhosis, portal hypertension and hepatoma were diagnosed. He was treated with frusenide 40 mg daily, and
‘Slow K’ 1200 mg t.d.s. He responded well, as shown by diuresis with lessening of oedema and a loss of 3 kg body-weight 6 days after admission. Five days later, however, he started to deteriorate with abdominal pain and vomiting. The next day he developed tachycardia, a raised jugular venous pulse and basal crepitations. Ampicillin 500 mg 6-hourly was given. Abdominal distension became worse and tapping showed blood-stained ascitic fluid. His temperature remained normal. He died, suddenly, 2 weeks after admission.

Relevant investigations: barium meal showed extensive oesophageal varices; prothrombin time 19 sec (control 14 sec); aspartate aminotransferase 123 i.u. (normal, up to 30 i.u.); alkaline phosphatase 77 i.u. (normal 20–100 i.u.); α-fetoprotein 0·9 mg/ml (normal 1–16 ng/ml according to Rouslahti et al. (1974).

Post-mortem examination showed a deeply jaundiced man with abdominal distension. One litre of bloody ascitic fluid was present. The liver weighed 1600 g, the right lobe showing micronodular cirrhosis and the left almost completely replaced by a haemorrhagic tumour (Fig. 1). This had ulcerated the diaphragm and inferior vena cava just above the openings of the hepatic veins. The spleen was enlarged and weighed 720 g. Oesophageal varices were evident. There was thrombosis of the portal, splenic and superior mesenteric veins, in the latter extending into its small tributaries. Haemorrhagic necrosis of 50 cm of the small intestine was present, with free blood in the lumen. A tumour mass weighing 30 g plugged the tricuspid valve (Fig. 2); it readily fell off the valve cusps and was not attached. The lungs showed miliary carcinomatosis. Histological examination of the tumour showed a trabecular pattern in most areas, with a minor tubular component (Fig. 3). Bile and mucin production were demonstrated. The tumour was classified as a mixed hepatoma–cholangiocarcinoma.

Discussion

In this patient, hepatoma developed on pre-existing cirrhosis. In three British surveys, the incidence of hepatoma in cirrhosis was 12·3%, 14·2% and 16·3% respectively, with predominance in males (McSween and Scott, 1973; Stone, Islam and Paton, 1968; Parker, 1957). Conversely, approximately 66% of hepatoma patients have pre-existing cirrhosis (Dawson and Winch, 1973). In Africa,
Case reports

FIG. 3. A superficial portion of the tumour embolus. Necrosis is evident in deeper parts deprived of nutrition from the overlying stream of blood.

where the population has a high incidence of hepatoma (Higginson and Svoboda, 1970), the frequency of underlying cirrhosis is difficult to determine in the absence of post-mortem data (Leading Article, 1975).

The post-mortem findings in this case show that the liver tumour had extended into the inferior vena cava and right atrium, so that a large embolus was detached to become arrested at the tricuspid valve, thereby causing sudden death owing to acute circulatory failure. Extension of hepatoma into the inferior vena cava is uncommon. In a review of twelve cases of tumour thrombosis of the inferior vena cava or the right atrium resulting from primary carcinoma of the liver, the constant symptoms and signs were swelling of the lower extremities (usually progressive) and swelling of the abdomen with ascites (Gregory, 1939). Those features were present in this patient during his final illness. On the other hand, massive tumour embolism causing sudden death is not found in previous reviews of hepatoma, whereas it had been reported in hypernephroma (Judd and Scholl, 1924; Masson and Branwood, 1955).

The primary tumour in this patient was in the left lobe and did not cross to the right. Hepatoma is reported to involve the liver diffusely in about 66% of cases. In the remaining 33%, the right lobe is involved two to three times more frequently than the left (Curuchet et al., 1971; El-Domeiri et al., 1971; Linder, Crook and Cohn, 1974). Histology of the tumour showed a mixed hepatoma–cholangiocarcinoma, which cannot be distinguished macroscopically from a purely hepatocellular carcinoma (Allen and Lisa, 1949). The mixed type is an infrequent form of primary liver cancer, accounting for about 0·5–0·8% of reported cases. It has been regarded as a variant of the hepatocellular type (Ewing, 1940).

Prognosis for hepatoma patients is very poor, as illustrated in this case. It is generally fatal within 1 year of onset of symptoms and 6 months from diagnosis (Becker, 1974; Robbins, 1974). However, there are many reports of cases surviving several years (Curuchet et al., 1971; El-Domeiri et al., 1971).

The elevation of ς-fetoprotein to 0·9 mg/ml in this case might have been related to a high tumour turnover (Parkes et al., 1974). A rise in this tumour marker is reported in approximately 25–80% of hepatoma patients in different series.

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**Case reports**


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**Carcinosarcoma of the adult kidney**

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**Summary**

Carcinosarcoma of the adult kidney is a very rare tumour and there are only a few well documented cases in the literature. In this report such a tumour is described from a 50-year-old white male, which progressed very rapidly with widespread metastases. Histologically, the tumour consisted of renal cell carcinoma and fibrosarcomatous components. The interesting features in this case were that both the carcinomatous and sarcomatous elements of the tumour exhibited metastases separately to various organs.

**Introduction**

Carcinosarcomas are rare tumours and are known to occur in a wide variety of organs, such as uterus, breast, oesophagus, larynx, lungs, urinary bladder, prostate, oviducts and kidneys with a variable frequency. The term carcinosarcoma implies a mixed neoplasm containing both epithelial and mesenchymal elements, each of which displays a different morphological and biological criterion of malignancy (Batsakis, 1974). Since the original description of these tumours by Virchow (1864), the histopathology of carcinosarcomas has become a controversial subject. Many cases of carcinosarcoma reported previously in the literature were repudiaded by Saphir and Vass (1938) who claimed that the sarcomatous elements are either variants of epithelial cells or stromal reaction to an epithelial cancer. Later on, Lane (1957) suggested the metastatic ability of the sarcomatous component as a criterion for the diagnosis of carcinosarcoma, in the absence of which he preferred the term 'pseudosarcoma'.

In this report a carcinosarcoma of the adult
Hepatoma causing a massive tumour embolus.

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