Vinylchloride induced hepatic angiosarcoma

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The relationship between vinylchloride exposure and human angiosarcoma of the liver (ASL) received attention in 1973 when a case of this rare tumour was diagnosed at autopsy[1].

Case report

A 39-year old man was first seen in July 1979 with pain in the right upper quadrant. The liver was palpated at the right costal margin. Oral cholecystography and barium swallow were normal and liver function tests were in normal limits. From 1965 to 1970 he had cleaned reactors used for the polymerization of the vinylchloride monomer in PVC and was thus exposed to high amounts.

He was admitted 3 months later with persisting right upper quadrant pain, loss of appetite and fatigue. The liver edge was by then hard and 4 cm below the costal margin. The ESR was accelerated (80 mm/h) and alkaline phosphatases and GGTP were elevated. Carcino-embryonic antigen (CEA) and α fetoglobulin were normal. Ultrasound showed a large dense tumour of the right hepatic lobe with areas of necrosis. The ^{99}Tc hepatic scan confirmed the presence of an ill-defined filling defect in the inferior part of the right lobe with two small defects at the level of the hilum.

The liver computerized tomography confirmed the integrity of the left lobe.

A selective angiography of the celiac artery revealed a hypervascularized tumour of the right hepatic lobe (*Figure 1*).

At a right thoracophrenolaparotomy the tumour was found to be confined to the right lobe. An extended right lobectomy was then performed. The postoperative recovery was uneventful and the patient was sent home with a monthly administration of Vincristine (1 mg IV) and Adriamycin (150 mg/m² IV) which was discontinued in June 1981.

Repeated controls up to September 1981 by liver scan and computerized tomography remained normal. The patient is still in good health 38 months after the resection.

Pathology

The resected specimen was 2050 g. On macroscopical examination, the main tumour (13·5 × 8 cm) was yellowish and spongy and contained cystic and haemorrhagic zones. A second smaller haemorrhagic mass was found at the inferior aspect of the right lobe surrounded by numerous purple masses.

Macroscopically, the main tumour showed large areas of necrosis and haemorrhagic pseudocystic spaces (*Figure 2*). These spaces were surrounded by areas of dense vascular proliferation. The sinusoids were lined with variably sized irregular sarcomatous cells with hyperchromatic nuclei. Elsewhere, blood-filled spaces were surrounded by sarcomatous cells. The sarcoma cells encompassed adjacent liver cells and bile ductules and infiltrated the parenchyma. The pathological diagnosis of multicentric angiosarcoma was made. The rest of the liver was normal except for some moderately enlarged portal tracts. Progressive fibrosis separated hepatocytes at the margins of the portal tracts from adjacent hepatic

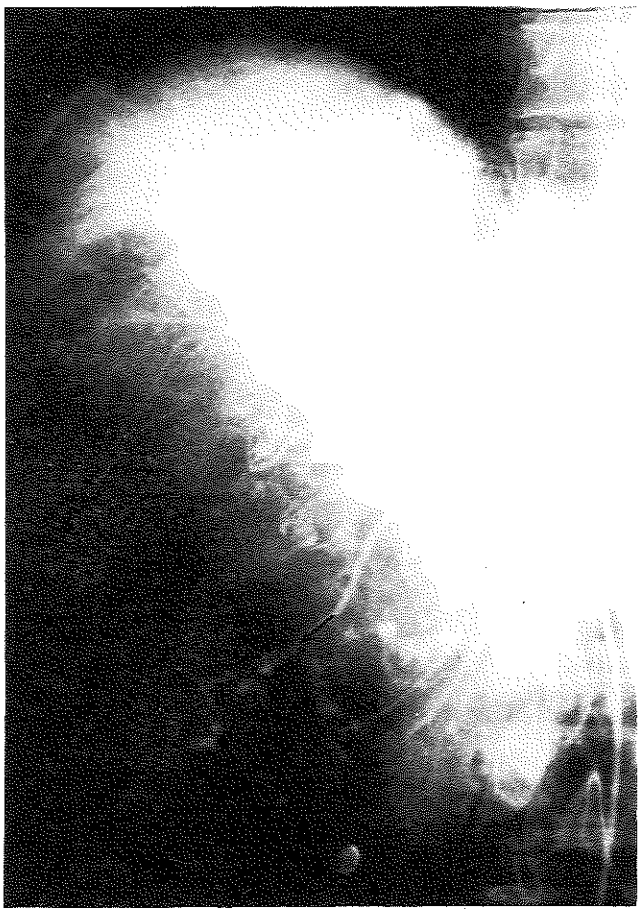

Figure 1 *Selective angiography of the coeliac artery. The hypervascularized tumour is supplied by an anterior branch (arrow) of the right hepatic artery*

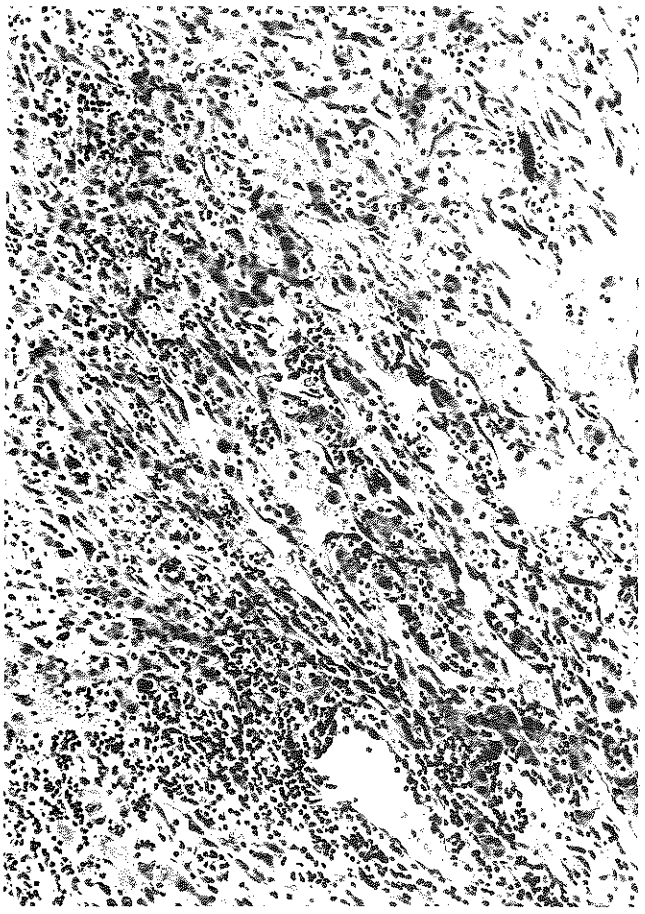

Figure 2 *Photomicrography of the main tumour showing blood filled spaces surrounded by sarcomatous cells. (Haematoxylin eosin, × 160)*

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cord cells. Anisocaryosis and anisocytosis were frequent. A cross-section biopsy of the left lobe showed normal tissue with slight hepatocytic anisocaryosis. The lymph nodes taken from the liver hilum were hyperplastic. The main features of this tumour were its multicentricity and the presence of mild fibrosis.

Discussion

The occurrence of liver angiosarcoma in vinylchloride polymerization workers was reported in 1974[1,2]. Prolonged exposure and long interval from initial exposure is required before liver disease becomes apparent. The average interval is 12 years (range 6–29)[3]. Our patient was exposed for 5 years and became symptomatic 14 years later.

It is a rapidly progressing fatal disease, especially in adults. The clinical features include rapid liver enlargement with haemorrhagic ascites, fast deterioration and cachexia usually with death within 6 months.

The treatment is disappointing and chemotherapy and radiation of palliative value only. If the diagnosis is made early, the disease is localized and there is no associated liver fibrosis or portal hypertension, resective operation might prove to be curative. However, there are few reported cases of successful operative removal of hepatic angiosarcoma and the longest survival has been 16 months[4].

In our case the tumour was confined to the right lobe and there was no sign of the extensive fibrosis. So far the patient is apparently free of disease after 38 months. This is, to our knowledge, the longest published survival.

References

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