varices to be the source of haemorrhage, and these were treated by injection sclerotherapy. The serum alkaline phosphatase and γ-glutamyl transpeptidase levels rose over the next three months to 334 IU/l and 106 IU/l respectively. Radioisotope liver scan showed multiple filling defects consistent with tumour deposits. Ascites and ankle oedema developed, and he died suddenly in January 1980 from an intraperitoneal bleed after a liver biopsy. Necropsy showed that a large haemorrhagic tumour, occupying most of the right lobe of the liver, had ruptured into the peritoneum. Several smaller haemorrhagic tumours were found elsewhere in the liver. An enlarged spleen (wt 760 g) and thrombosed oesphageal varices were also noted. Histology showed the tumours to be angiosarcomas, with areas of sinusoidal dilatation and portal fibrosis in unaffected areas of the liver.

Discussion

Vinyl chloride monomer is transformed by hepatic microsomal enzymes to toxic metabolites that covalently bind to DNA.[7] After exposure to VCM hepatocytic proliferation, sinusoidal lining cell proliferation, and focal sinusoidal dilatation occur[4] and angiosarcoma may later develop from the sinusoidal lining cells. Enlarged lipocytes may be seen in the space of Disse[8]; these cells are fibroblast precursors and can lay down collagen.[9] Hepatic fibrosis results and may be associated with presinusoidal portal hypertension.[10] It is commoner than angiosarcoma.[2] Although fibrosis was found in tumour-free portions of the liver tissue from VCM workers dying of angiosarcoma,[3] transition of hepatic fibrosis to angiosarcoma, although postulated,[3] has not been observed.

The two patients described here were originally included in a series of seven VCM workers with portal hypertension.[2] They were followed for five and 10 years respectively from the time of diagnosis of portal hypertension to death from angiosarcoma. During this period they were not further exposed to VCM. Their case histories indicate that hepatic fibrosis in a VCM worker may be a precursor of future malignant change and life-long follow-up is necessary. We know of only one other similar patient who died seven years after a portacaval shunt from an angiosarcoma.[6]

Non-cirrhotic fibrosis can also occur in the absence of portal hypertension, and may then be difficult to detect. It does not lead to a disturbance of liver function tests and may even be missed on needle biopsy of the liver.[2] Greyscale ultrasonography is a useful diagnostic aid,[11] but it has yet to be used in a large industrial survey, and there are probably several unrecognised affected VCM workers. Stringent measures to control levels of exposure should lead to eventual disappearance of non-cirrhotic fibrosis. Those patients who already have the condition, however, are still at risk of developing hepatic angiosarcoma in the future.

A problem highlighted by the second case is that angiosarcoma is, by definition, a vascular lesion, and there is a risk of uncontrolled haemorrhage after blind liver biopsy. We believe, therefore, that the diagnosis should be sought either by laparoscopic biopsy or by hepatic angiography.

We thank Dr D M D Evans and Professor Peter Scheuer for their help in interpreting the biopsy material.

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