clinical findings, but this did not alter management. Of the 60 patients in category 2 with abnormal radiographs, 57 had clinical evidence of cardiorespiratory disease.

Comment

Radiological abnormalities were seen in virtually half of the patients in the clinical survey. More important, however, the chest film affected the clinical management in 19% of cases, sometimes because of abnormality and sometimes because of normality.[1] This was a high proportion when compared with other, similar surveys.[2 3] In the remaining patients the chest film did not affect management, and most of these patients had diseases clearly distinct from the others—for example, parkinsonism, anaemia, depression, dementia, falls, leg ulceration, cerebrovascular disease (table). In this large miscellaneous group the routine chest radiograph was of management value in only 5% of patients. When the notes of these patients were rescrutinised it was apparent that radiography would in any case have been used selectively. For example, in one patient a suspected perforation was made unlikely, and in another clear lungs were seen when investigating weight loss.

We suggest that routine chest radiography of all patients (policy in two thirds of the hospitals surveyed) could usefully be abandoned and the examination confined to those with cardiorespiratory symptoms or signs, toxic confusional state, and bone (thoracic) pain. This should reduce requests for radiography by about half. Though this would result in a relatively small financial saving in any one hospital, chest radiography of an elderly patient may occupy a radiographer for 15-20 minutes. Moreover, it would be kinder to many frail patients by eliminating an unnecessary ritual.

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Does delta infection play a part in the pathogenesis of hepatitis B virus related hepatocellular carcinoma?

The delta (δ) agent is a defective RNA viral agent which requires helper functions from the hepatitis B virus for its replication and transmission.[1] The agent plays an important part in the pathogenesis of severe acute hepatitis and chronic active hepatitis (and perhaps cirrhosis) in people who have or have had hepatitis B infection.[1] Because about 80% of cases of hepatocellular carcinoma world wide are related to hepatitis B virus and in most there is coexisting cirrhosis, we investigated the possible role of coinfection with the δ agent in these patients.

Patients, methods, and results

Serum from the following groups was examined for δ antigen and anti-δ: 107 South African blacks who were positive for hepatitis B surface antigen (HBsAg) and had hepatocellular carcinoma; 144 black chronic carriers of HBsAg; and 17 multiply transfused renal transplant recipients who were positive for HBsAg. The examinations, using radioimmunoassay, were performed in the laboratory of Dr M Rizzetto in Turin, Italy. The patients with cancer were aged 13-74 years (mean 38 years); there were 101 men and six women. Of the chronic HBsAg carriers 137 were asymptomatic and seven were known to have cirrhosis or chronic active hepatitis. They were aged 3-57 years; there were 132 men and 12 women.

HBsAg, anti-HBs, antibody to the core antigen (anti-HBc), e antigen (HBeAg), and antibody to the e antigen (anti-HBe) were measured by radioimmunoassay (Ausria II, Ausab, Corab, and HBe/anti HBe, respectively, Abbott Laboratories).

Liver and tumour tissue from a further 80 patients with hepatocellular carcinoma was examined for δ antigen by the direct immunoperoxidase technique using a peroxidase conjugated anti-δ antibody.[2] Serum samples were also taken from 55 of these patients. The mean age of the 80 patients was 53·2 years (range 12-81 years) and there were 66 men and 14 women. In all of these patients we performed histochemical staining of the tissues to detect HBsAg.

Serum studies—The serum of all of the 107 patients with hepatocellular carcinoma was positive for HBsAg and anti-HBc; HBeAg was present in 34 and anti-HBe in 66. HBeAg was present in 19 (13%) of the chronic HBsAg carriers and in 15 (88%) of the transplant recipients. Neither δ antigen nor anti-δ was detected in the serum of any of the patients with hepatocellular carcinoma, the chronic carriers of HBsAg, or the renal transplant recipients.

Tissue studies—HBsAg and anti-HBc were present in the serum of 19 of the 55 patients with hepatocellular carcinoma in whom both tissue and serum were studied. Three of these patients were positive for HBeAg and 15 for anti-HBe. A further three patients were positive for anti-HBc in the absence of HBsAg and anti-HBs. Anti-HBs with or without anti-HBc was present in 24 patients and anti-HBe in six of these. Six patients had no markers of hepatitis B infection. Of the 19 patients with HBs antigenaemia tissue HBsAg was detected in 14. One patient with anti-HBc and anti-HBs alone was positive for tissue HBsAg. Of the 25 patients in whom tissue alone was studied six showed HBsAg. In all, 26 patients showed either serological or tissue evidence of HBsAg and one further patient had anti-HBc alone in the serum. δ Antigen could not be shown in either non-neoplastic liver tissue or in tumour tissue in any of the patients.

Comment

δ Infection does not play a part in the pathogenesis of hepatocellular carcinoma in South African blacks. Serological evidence of δ infection was not found in a substantial number of chronic carriers of HBsAg who lived in several areas of South Africa, and thus the agent could not be expected to have a causal role in chronic liver disease, including hepatocellular carcinoma, in this region. We cannot, of course, exclude the possibility that the δ agent has a pathogenetic role in the hepatocellular carcinoma that occurs in other populations with a high prevalence of infection with this agent. Nevertheless, δ antigen was shown immunohistochemically in only 14% of HBsAg positive Greek patients with hepatocellular carcinoma (compared with 29% of patients with HBsAg positive chronic active hepatitis and 28% of those with HBsAg positive cirrhosis)[3] and infrequently in Italian[4] and North American patients.[5]

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