

formed in areas where the endemicity of the reported infections is very high, we have conducted a study to evaluate the prevalence of HBV and HIV infection in leprosy patients in our country, which has a relatively low endemicity of HBV, HIV and leprosy.

Seventy leprosy patients (52 male; mean age 59.8 years, range 37–86 years; 52 lepromatous, 14 tuberculoid, 5 borderline) were randomly selected from the Centres of Treatment and Control of Leprosy of Jaen and Trillo, Spain. None of the patients had any other risk factor for HBV or HIV infection. Sera from all patients were tested for HIV antibodies (ELISA) and HBV markers (HBsAg, anti-HBs and anti-HBc – Ausria, Ausab, Corab; Abbot Labs, North Chicago, Ill, USA). Positivity for HIV antibodies or HBsAg was not found in any case. However, 32 patients (45.7%) had serological evidence of past HBV infection (positivity of anti-HBc with or without anti-HBs). When patients with evidence of past HBV infection were compared with those without it, no differences were found regarding sex, site of origin (rural or urban) nor clinical type of leprosy. However, cases with past HBV infection tended to be older than patients with negative markers (63.1 ± 7.0 vs 57.1 ± 10.9 years, $P < 0.01$, Student's *t*-test); and a history of institutionalization in leprosy centres for more than a year was more frequent among HBV-marker-positive patients (68.7% vs 28.9%, $P < 0.001$, Chi-square test). Furthermore, the time since the initial diagnosis of leprosy was longer in subjects with serological evidence of past HBV infection than in patients without it (35.6 ± 13.0 vs 21.6 ± 12.1 years, $P < 0.001$, Student's *t*-test). These three factors, namely age, institutionalization and time of evolution of leprosy, were independently correlated with HBV-marker-positivity in a multivariate analysis (stepwise logistic regression).

In spite of lacking a control group, these results suggest that there is a high prevalence of markers of past HBV infection in leprosy patients (45.7%), since the same figure in the Spanish general population is 18.2%.⁷ Similar results have been observed in Greece.⁸ This high prevalence may be due to leprosy itself, since it is well known that a history of skin disease is a risk factor for HBV infection.⁹ Besides, other associated factors, such as institutionalization, may be present. Interestingly, a similar high frequency of past HBV infection has been reported in Spanish mentally retarded institutionalized patients.¹⁰ Increasing prevalence of past HBV infection with age has been reported before.¹¹ However, leprosy patients seem to have normal immunological response to HBV since no case of chronic HBV infection was found in our study despite the high frequency of past infection. Moreover, in our experience, there is no relationship between leprosy and HIV infection other than the common and unfortunate comparison of AIDS as 'leprosy of the twentieth century'.

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Benign hepatic tumours of unusual size in two Samoan siblings

Sir,

Most benign hepatic tumours are less than 10 cm in diameter and rarely give rise to symptomatic abdominal masses.^{1–3} Two cases of large benign hepatic tumours in adult Samoan siblings are described. The first case was a hepatic cavernous haemangioma while the second one was a liver cell adenoma.

Case 1 – A 43 year old Samoan woman was admitted to the Western Samoa National Hospital, with a mass in the right upper quadrant of the abdomen for a year. She had previously had 10 deliveries and one miscarriage. Macroscopically the resection specimen was a solid dark brown tumour measuring $15 \times 13 \times 5$ cm adjacent to the left hepatic lobe. The tumour was demarcated from the surrounding liver parenchyma but was not encapsulated. Microscopically it was of 'cavernous haemangioma' of the liver. There was no sign of recurrence 2 years later.

Case 2 – A 35 year old Samoan woman was hospitalized one year after her sister (Case 1) with right upper quadrant pain and nausea. She was multiparous with 5 deliveries. She had a history of long-acting progesterone injections for the purpose of birth control, irregularly for a few years. Examination showed a mass in the right upper quadrant of the abdomen. Explorative laparotomy revealed a tumour in the right lobe of the liver, and

resection was carried out. Postoperative course was uneventful, and the one year follow-up was normal. Macroscopically the tumour was 12 × 10 × 5 cm, well-demarcated from the darker normal hepatic parenchyma and unencapsulated. The tumour showed microscopical features consistent with liver cell adenoma (LCA): bland hepatocytes arranged in irregular cell cords, two or three cells thick, that were separated by sinusoids; and also absence of fibrosis and proliferating bile ducts, which excludes the possibility of focal nodular hyperplasia of the liver (FNH).

Large symptomatic hepatic haemangiomas and liver cell adenomas, are rarely seen.^{2,3} Recent evidence indicates that LCA is associated with the use of oestrogenic hormones.^{4,5} On the other hand, FNH is considered to be of developmental origin like haemangioma. It has been reported that, in women, gonadal contraceptives and steroids may exert a trophic effect on preexisting focal hyperplasia to gradually transform to liver cell adenoma.⁵ Our adenoma case had a history of the irregular use of long acting progesterone, which is a not uncommon practice for birth control purposes in the rural areas of the South Pacific islands.

We believe that the lesion in Case 2 may have started as FNH and gradually transferred to LCA with progesterone. This seems likely because her sister had haemangioma, also a developmental lesion of the liver. The second possibility is that the patient had LCA, and that the use of progesterone contributed to the growth of the lesion. On the other hand, it is not possible to comment as to why the haemangioma reached such an unusual size. Clinical screening of other members of the family of our patients was not carried out, but family history revealed no additional information.

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Myelofibrosis with bleeding jejunoileal angiodysplasia diagnosed by intraoperative endoscopy

Sir,

Massive haemorrhage from diffuse angiodysplasia (AD) of the jejunum and ileum is a rare condition which may be difficult to diagnose. We describe a patient with myelofibrosis and recurrent melaena due to jejunoileal angiodysplasia where the diagnosis was made by intraoperative endoscopy (IOE).

A 73 year old male, known to have myelofibrosis, was admitted to the Rambam Medical Center with melaena. The haemoglobin level was 5.1 g/dl and platelet count 20,000 per litre. Prothrombin time and partial thromboplastin time were normal. Blood transfusions were given. Upper and lower gastrointestinal tract barium series and endoscopy did not show any pathology and the patient was discharged. A few days later the patient was readmitted because of syncope, melaena and a haemoglobin level of 4.6 g/10 ml. Radioisotopic examination failed to show Meckel's diverticulum. Enteroclysis and repeated upper and lower endoscopy were normal. A week later, the melaena recurred. Scanning with technetium 99-labelled autologous red blood cells suggested the presence of active bleeding in the regions of the ascending colon, right and left flexures. However, coeliac angiography following this scanning was normal.

After 4 weeks of continuous bleeding, that required a transfusion of 65 pints of blood to keep the haemoglobin stable, an exploratory laparotomy was performed. Intraoperatively, a colonoscope was introduced through the mouth, and passed to the small intestine. The scope was passed along the small intestine by invaginating the intestine over the colonoscope. The IOE with transillumination disclosed multiple jejunoileal angiodysplastic lesions. Some of the lesions were actively bleeding and were sutured. Histological examination of the biopsy from one of such lesions confirmed the endoscopic diagnosis of AD. A week later the patient died from massive gastrointestinal bleeding.

Angiodysplasia (a focal submucosal vascular ectasia) of the bowel is an uncommon source of massive gastrointestinal bleeding. The majority of cases occurring in the right colon in elderly patients. Jejunoileal AD is more rare^{1–3} and has a high rate of recurrent bleeding, presumably from the multiplicity and multifocality of lesions. Bleeding jejunoileal AD is difficult to diagnose by an invasive procedure such as endoscopy or contrast angiography because haemorrhage is unexpected with a slow rate of bleeding and the source is the 'blind area' of the gastrointestinal endoscopy. Occasionally even exploratory laparotomy may fail to diagnose jejunoileal AD. In such cases, IOE enhances the surgeon's ability to find the bleeding lesions.^{4,5} Increased awareness and exploratory laparotomy with IOE may allow more frequent recognition of jejunoileal AD. Because small intestinal AD may sometimes by localized and treatable by segmental resection, a careful use of this procedure is recommended.