needing early diagnosis. Since all women at risk may be identified by their prenatal history screening for presymptomatic carcinoma is possible. We suggest that the best methods are visualising the vagina and cervix, cytology of both vaginal aspirate and cervical scrape, iodine staining, and colposcopy with biopsy of suspicious areas. It is imperative that all physicians treating young women should be aware of these disease processes if the maximum therapeutic benefit is to be achieved.

We thank Dr W Cowan for his assistance, and Mr G Wilkinson for the histological sections; also Miss Margaret Taylor for typing the script.

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# SHORT REPORTS

## Poncet's disease: para-infective tuberculous polyarthropathy

In 1897 Poncet[1] introduced the term tuberculous rheumatism, meaning joint disease in patients with tuberculosis not due to tuberculous invasion of the joints—a para-infective arthropathy. The condition has rarely been referred to in British reports[2–4] and is not mentioned in most current textbooks of medicine. We therefore report a child whom we believe suffered from this disease.

### Case report

A 5-year-old boy, who had been born in this hospital of Pakistani parents, presented with a three-week history of swelling of both knees, which had begun to ache on the day before his hospital admission. No other symptoms were complained of or admitted to on direct questioning. He had returned from Pakistan with his family four weeks previously after a six-month stay, during which he had been generally unwell with some vomiting, but the cause was unknown. He had no other medical history. He had been vaccinated with BCG in 1976, when his mother had been treated for tuberculosis. His father had died from a myocardial infarction in the same year. He had one brother aged 12 years, who was well.

On admission he was afebrile. Findings were as follows: scattered papules and pustules over the anterior surfaces of the thighs, and submandibular lymph node enlargement on the right; effusion into both knee joints, which were slightly warm and tender; and pain on movement of the left elbow, and swollen and slightly painful ankles. There was no other abnormality apart from a convergent squint of long standing. The results of investigations were as follows: Hb 9·0 g/dl; WBC $14{\cdot}8 \times 10^9$/l; ESR 56 mm in first hour; blood urea and electrolyte concentrations normal; throat swab culture sterile; no pyuria in three early morning specimens; tests for serum rheumatoid factor, antinuclear factor, and Widal titres were all negative, and the ASO titre was 100 IU/ml; routine viral antibody tests no significant titres. In three gastric washings *Mycobacterium tuberculosis* was not identified by microscopy or culture; blood culture was sterile; a chest *x*-ray film showed right hilar enlargement, with streaky shadowing of the right upper lobe; *x*-ray films of knees were normal; and a Mantoux test 1 in 1000 was strongly positive with 15-mm induration.

Primary pulmonary tuberculosis was diagnosed and treatment was initiated with rifampicin, 450 mg daily; isoniazid, 125 mg daily; ethambutol, 400 mg daily; and pyridoxine, 10 mg daily. He improved generally and the rash on his thighs resolved, but the arthritis remained static after two weeks. A course of prednisolone, 60 mg/day, was then begun and the dosage gradually reduced as the joint symptoms subsided over the next four weeks. Two months after starting antituberculous treatment he had only slight residual soft tissue swelling at the knees and his other joints were normal. The knees too had returned to normal after three months. The changes on the chest *x*-ray film resolved slowly on treatment.

### Discussion

This child had a polyarthropathy associated with convincing evidence of primary pulmonary tuberculosis. The simultaneous swelling of five joints coincident with the primary complex virtually excludes direct tuberculous infection of the joints. Seronegative rheumatoid arthritis cannot be totally excluded but we believe that it is more likely that the joint features were a manifestation of tuberculous allergy. The rash on the thighs appeared typical of papulonecrotic tuberculid[5] and resolved with antituberculous chemotherapy, giving further evidence of tuberculous allergy. The possibility of tuberculosis should be considered whenever a patient has a polyarthropathy of obscure cause.

We thank Dr B Taylor for introducing us to the earlier reports on Poncet's disease.

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## Tetanus after rubber-band ligation of haemorrhoids

Haemorrhoidectomy is usually regarded as a safe operation. Slack[1] stated that no preoperative bowel sterilisation with antibiotics was necessary; all that was required was an enema followed by a rectal washout. Among complications he listed "infection" as an occasional abscess under a redundant skin fold. This case report shows that tetanus can be a complication of operations on the rectum.

### Case report

A 33-year-old housewife was admitted to hospital because of a two-day history of difficulty in opening her mouth and in swallowing, neck pain, neck and abdominal muscle stiffness, and excessive sweating. Rubber-band ligation of internal haemorrhoids had been performed nine days previously (seven days before symptoms began). She had had no other injury, and had not been immunised against tetanus. On admission she had clinical tetanus. She had a particularly severe spasm of the jaw on trying to remove her dentures. She was treated with penicillin, tetanus immunoglobulin, and originally with intravenous diazepam. The symptoms were not controlled, so endotracheal intubation was performed followed by paralysis, intermittent positive-pressure respiration, and tracheostomy. Progress was complicated by an unstable blood pressure and a lower-lobe pneumonia, but the relaxants could be discontinued after 25 days and progress thereafter was uneventful.

### Comment

The source of clinical tetanus may be difficult to determine. In our experience 10% of patients have no detectable wound. Spores of

*Clostridium tetani*, however, have been found in up to 40% of samples of human faeces.[2] Another of our patients developed postoperative tetanus, which was attributed to self-contamination, after removal of a tumour of the buttock. Since rubber-band ligation of haemorrhoids produces an avascular area, and since this is in contact with a potential source of *Cl tetani*, the tetanus in this case may reasonably be attributed the operation. Certainly the time relationship fits in with the incubation period of tetanus.

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# Primary biliary cirrhosis after long-term practolol administration

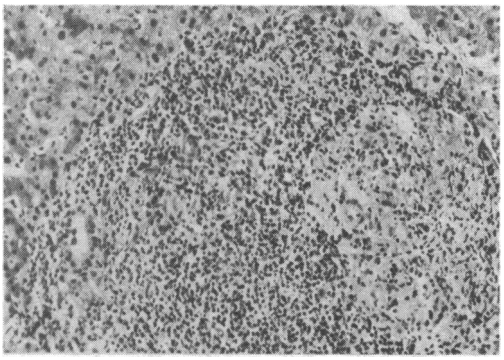
Oculocutaneous syndrome, sclerosing peritonitis, and pulmonary fibrosis may result from practolol treatment[1 2] but liver disease has not been implicated as an adverse effect. We have recently seen two patients with oculocutaneous lesions typical of practolol sensitivity who also had clinical and laboratory features of primary biliary cirrhosis (PBC, primary non-suppurative destructive cholangitis).

## Case reports

A 76-year-old man developed pruritus after four years' continuous treatment with practolol 300 mg daily for angina of effort. He had an eczematous rash with palmar erythema; dryness of the eyes was confirmed by Schirmer filter paper testing. Smooth, non-tender hepatomegaly was noted. The following measurements were made: serum bilirubin 25 μmol/l (1·5 mg/100 ml) (normal 1·7-17 μmol/l (0·1-1·0 mg/100 ml)), alkaline phosphatase (liver isoenzyme) 405 IU/l (normal 20-90), serum aspartate transaminase (SGOT) 12 IU/l (normal 4-20), serum albumin 36 g/l, serum total globulin 40 g/l, serum IgM 2·2 g/l (0·7-2·0), IgG 11·6 g/l (9·5-16·5). Hepatitis B antigen was absent. Antinuclear factor was present (titre 1/80) as was smooth muscle antibody (1/20) and mitochondrial antibody (1/80). Liver scan and histological examination of a needle biopsy specimen confirmed the presence of cirrhosis.

He remained well after practolol was stopped, but liver function tests showed persisting cholestasis. The development of cervical lymphadenopathy two years later, however, led to the diagnosis of nodular sclerosing Hodgkin's lymphoma. Despite radiotherapy he died after six months. Patent bile ducts were found at necropsy, and histology of tissue obtained a few minutes after death confirmed advanced cirrhosis with proliferating bile ducts and lymphocyte aggregates consistent with primary biliary cirrhosis. Staining with orcein and rubeanic acid showed hepatocytic metallo-protein complex as found in PBC.[3] Typical sclerosing peritonitis affecting the jejunum was also present.

A 57-year-old man was treated with practolol 300 mg/day for angina. After seven months he developed a psoriasiform rash and two months later he complained of dryness of the eyes. Liver function was not tested until 16 months after discontinuing the drug. Findings were: serum bilirubin concentration 50 μmol/l (3·0 mg/100 ml), alkaline phosphatase concentration 144 IU/l, and SGOT 15 IU/l. Serum protein and immunoglobulins were normal as were autoantibody tests except mitochondrial antibody, which was positive to a titre of 1/160. A needle biopsy specimen of the liver showed normal lobular architecture, but near bile ducts were large epithelioid granulomata with surrounding lymphocytes (fig).



Epithelioid granulomata and lymphocytic infiltrate in the periportal area. (Haematoxylin and eosin. × 180.)

## Comment

These patients conform to the usual diagnostic criteria for primary biliary cirrhosis,[4] including cholestasis, a high mitochondrial antibody titre, and consistent liver histology. Chronic liver disease after drug administration usually resembles active chronic hepatitis. Only three cases of abnormal liver function after practolol administration have been reported to the Committee on Safety of Medicines in the United Kingdom, but the details are incomplete and difficult to interpret. Autoantibody abnormalities, however, are recognised with practolol rashes. Thus the serum in five out of 18 patients was reported positive for antinuclear factor but none of them had mitochondrial antibody.[1] A relationship between practolol and chronic hepatobiliary disease seemed likely but the presence of PBC-like histology and mitochondrial antibody in patients taking practolol could also have been fortuitous. We have therefore tried to calculate the probability of men with PBC being exposed to practolol. By making several assumptions, an estimate of the figure may be obtained from the number of prescriptions for practolol and the number of certified deaths from PBC a year. Two cases of PBC exposed to practolol in the northern region gives a much greater overall incidence than would be expected from annual prescription and mortality data. We therefore think that the association between PBC and practolol in our two cases was probably not fortuitous.

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# Malignant disease presenting as Addison's disease

Primary adrenal hypofunction—Addison's disease—is uncommon. Autoimmune destruction of the adrenals is now the commonest cause, other causes being due to metastatic carcinoma and tuberculosis. Vieweg *et al*[1] reviewed the published work and found only eight cases of Addison's disease associated with malignancy in which the diagnosis had been confirmed by the results of ACTH and cortisol estimations. It must be unusual for malignant infiltration of the adrenals to produce Addison's disease when there is no other evidence of malignant disease. Two such cases are described.

## Case histories

*Case 1*—A 75-year-old man presented in 1975 with prostatism. His medical history included tuberculosis at the age of 15; partial gastrectomy for duodenal ulcer in 1944; and syncopal attacks due to cardiac arrhythmias in1966. In the six months after prostatectomy for benign prostatic hypertrophy, he