physicians, with little to suggest the underlying disease, so that the diagnosis of choriocarcinoma may not be considered for some time.

We thank Mr T F Redman for gynaecological advice and comments on this report; Dr G W Pennington and Dr F E Neal for chemotherapy and subsequent care of the patient; and the Public Health Laboratory, Leeds, for bacteriological studies.

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# Hypertrophic osteoarthropathy and purgative abuse

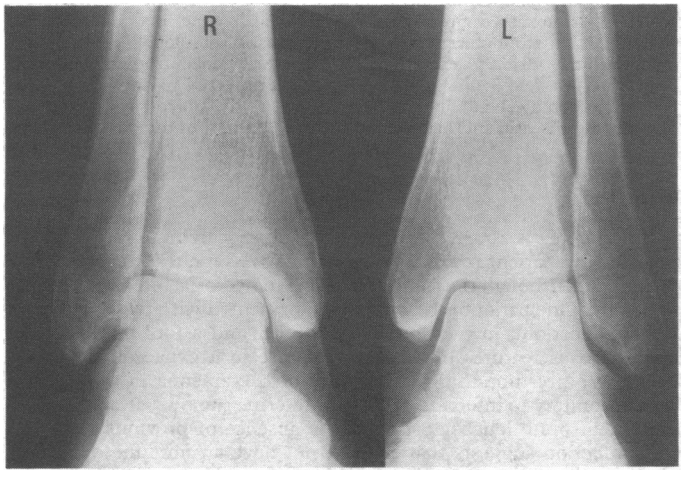
We describe a young woman who presented with clubbing of digits and hypertrophic osteoarthropathy. A history of purgative abuse emerged and it appeared likely that this was more than a chance association. We are unaware of any previous reports linking hypertrophic osteoarthropathy with purgative abuse.

## Case report

A 21-year-old dental nurse presented in mid-1978 with a nine-month history of painful swelling of both ankles followed by painful swelling and morning stiffness affecting proximal and distal interphalangeal joints of both hands. Her only other symptom was intermittent diarrhoea of three years' duration. There was no family history of clubbing or any rheumatic disorder.

On examination she weighed 49·1 kg. There was clubbing of fingers and toes with pronounced periungual erythema. Both ankles were swollen, red, and tender, and there was tenderness of interphalangeal joints of the fingers. General examination showed no other abnormality. Cardiovascular and respiratory systems were normal, as were the findings on abdominal and rectal examination. Results of sigmoidoscopy to 15 cms were similarly completely normal.

Investigations showed an erythrocyte sedimentation rate of 10 mm in first hour, haemoglobin concentration 13·9 g/dl, and white cell count $8·3 \times 10^9$/l (8300/mm$^3$). Serum electrolyte, urea, and protein concentrations were normal, as were iron and folate. Serum calcium concentration was 2·5 mmol/l (10·0 mg/100 ml), phosphate concentration 1·1 mmol/l (3·5 mg/100 ml), and alkaline phosphatase activity 114 u/l (normal range 20-90). Autoantibody screen was negative and thyroid function values normal. Radiographs of chest and hands were normal but those of knees and ankles showed striking symmetrical bilateral periosteal new bone formation, affecting particularly the ends of the long bones (figure). Barium follow-through and enema examinations showed only mild flocculation of contrast material in the small bowel.




Radiograph of ankles showing normal joints but florid periosteal reaction affecting distal tibiae.

The patient's mother disclosed that for three years the patient had been taking at least three senna (Senokot) tablets daily. The patient then confessed to habitually taking this aperient to control her weight. She also admitted to a period of secondary amenorrhoea of several months' duration a year before.

In October 1978 she stopped taking laxatives, and subsequently her weight increased to 57·2 kg. Within six months the clubbing had disappeared, though the periungual erythema persisted. Her rheumatic symptoms were less severe and controlled by non-steroidal anti-inflammatory drugs, though there had been no regression of the radiological bone abnormalities.

## Comment

Although the most familiar association of hypertrophic osteoarthropathy is with intrathoracic disease, it may also be hereditary or idiopathic.[1] In addition, there is a well-recognised association with various extrathoracic disorders, especially a wide range of gastrointestinal diseases whose common feature is diarrhoea.[1 2] Our patient's diarrhoea may be explained solely by purgative abuse, since it disappeared once she stopped the practice and there was no evidence of an underlying gastrointestinal disorder. The abnormality on barium follow-through examination was thought to be consistent with purgative abuse.

Silk *et al*[3] first described reversible finger-clubbing in a patient who ingested excessive quantities of purgatives, and the association has been noted by others,[4 5] though in these instances the reversibility of clubbing could not be established because the patients persisted in taking purgatives. Interestingly these patients suffered from anorexia nervosa, and our patient's history suggested some features of this condition. We think that the presentation of clubbing with hypertrophic osteoarthropathy and purgative abuse is more than coincidental: there was no evidence of any other disease and, significantly, the patient's clubbing regressed and her symptoms improved after stopping the purgatives.

We thank Mr K M N Kunzru for referring the patient, and Dr G Sladen for helpful advice.

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# Primary biliary cirrhosis and focal glomerulonephritis

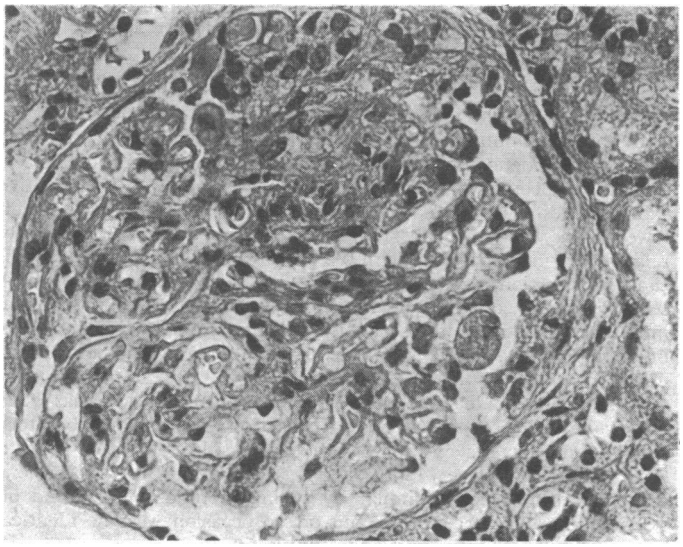
Primary biliary cirrhosis is a progressive liver disease, and, though the aetiology is unknown, autoimmune mechanisms are probably implicated. Thomas *et al*[1] suggested that the liver damage may be mediated by immune complex deposition, but evidence for this is not conclusive. Although immune complex deposition may mediate some of the diseases with which primary biliary cirrhosis is associated, to our knowledge only one case has been reported[2] in which this has been clearly shown. We report a further case of primary biliary cirrhosis and immune complex disease and discuss briefly the importance of our findings.

### Case report

A 66-year-old man presented in April 1979 with a flu-like illness and pleuritic chest pain. He was referred for investigation four months later because of persisting fatigue and lassitude. He had developed pain and stiffness of the neck and shoulders but no other symptoms.

Examination showed a blood pressure of 220/110 mm Hg, an exudative retinopathy, and albuminuria. The liver was moderately enlarged. Haemoglobin concentration and white cell and platelet counts were normal. Erythrocyte sedimentation rate was 106 mm in first hour. Blood urea concentration was 6·8 mmol/l (41 mg/100 ml), plasma sodium 141 mmol (mEq)/l, potassium 4·1 mmol(mEq)/l, and creatinine 73 μmol/l (0·82 mg/100 ml). Creatinine clearance was 68 ml/min and urinary protein 6 g/24 h, and red and white cell casts were seen in the urinary deposit. An intravenous pyelogram was normal.

A renal biopsy specimen showed focal glomerulonephritis (figure). There



Single glomerulus showing focal glomerulonephritis with capsular epithelial proliferation. (Haematoxylin and eosin × 480 (original magnification).)

was focal lobular obliteration and fibrinoid deposition, with focal thickening of the peripheral capillary loops, occasional epithelial crescent formation, and a diffuse increase in mesangium. Immunofluorescence staining of fresh material showed heavy granular deposition of IgG, IgA, IgM, C3, and C1q in both the capillary loops and mesangium. Ultrastructural examination of glutaraldehyde-fixed material showed electron-dense immune deposits in the mesangium and beneath the epithelial cells in the capillary loops. There were no subendothelial deposits. The morphological features, except for the absence of subendothelial deposits, were suggestive of systemic lupus erythematosus. Antinuclear factor was subsequently detected in the serum at a dilution of 1 in 128, but DNA binding was only 5% (thus he had four of the American Rheumatism Association criteria for systemic lupus erythematosus).

Several tests of liver function were abnormal. Serum alkaline phosphatase activity was 560 U/l (normal 21-94 U/l), aspartate transferase activity 52 U/l (normal 8-40 U/l), and albumin concentration 31 g/l (normal 35-53 g/l). A liver biopsy specimen showed moderate infiltration of mononuclear cells in the portal tracts with considerable fibrosis and focal mild piecemeal necrosis. There were no granulomas, but the bile duct showed considerable periductal inflammation and epithelial damage. The features were consistent with primary biliary cirrhosis. Tests for antimitochondrial antibody were positive and serum IgM was 360 IU/ml (normal 69-322 IU/ml).

### Comment

Thomas *et al*[1] suggested that the liver damage in primary biliary cirrhosis may be mediated by immune complex deposition. Immune complexes certainly occur in the serum of patients with the disease,[3] and the deposition of these complexes in the vicinity of bile ducts might account for the observed histological changes. Furthermore, primary biliary cirrhosis has been reported in association with systemic lupus erythematosus,[4] rheumatoid arthritis,[5] and glomerulonephritis,[2] diseases in which immune complex deposition is thought to have an important pathogenic role. In only one case, however, has immune complex deposition been clearly shown: Rai *et al*[2] reported the association of primary biliary cirrhosis and diffuse membranous glomerulonephritis and showed, by immunofluorescence, IgG and IgM contained in immune deposits in the glomeruli. In our case immunofluorescence and electron microscopy showed complexes containing IgG, IgA, and IgM, which had induced a focal glomerulonephritis.

Thus, with this second case, evidence linking primary biliary cirrhosis and immune complex disease is slowly accumulating, but more case reports are needed before the association is established.

We thank Professor P J Scheuer, of the Royal Free Hospital, London, for his help in interpreting the liver biopsy.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# Addiction to temazepam

Growing awareness of the addictive properties of diazepam and nitrazepam has led doctors to switch to the shorter-acting benzodiazepine hypnotics, such as temazepam, in the belief that they are safer.[1] Although cases of dependence to lorazepam have been reported it is widely assumed that this group of benzodiazepine hypnotics are not as addictive as nitrazepam or flurazepam. Because the short-acting benzodiazepine hypnotics are being prescribed more often, doctors should be aware of the risks.

### Case history

A 22-year-old unmarried factory worker presented with a direct request for help in withdrawing from the sleeping pills on which he had become dependent. He gave a two-year history of taking temazepam (Normison, Euhypnos) in association with alcohol. He had a history of depressive episodes, beginning at the age of 16, when his parents separated. Depression had been treated by his general practitioner with tricyclic drugs. He had had four courses of treatment lasting from three to six months in the last six years. Temazepam was prescribed initially for insomnia during a depressive episode and continued as a repeat prescription. He also drew on a repeat prescription made out for his mother. His job record was steady, but he had on three occasions abruptly abandoned work, usually after a domestic crisis. He was also addicted to nicotine, smoking 20-25 cigarettes a day, and alcohol, with an average intake of 2-4 pints of beer a day. His addiction to temazepam began as a means of augmenting the psychic effects of alcohol. He appears to have achieved optimal effects by taking the drug one hour before going out drinking. His initial dose was 10-20 mg but within a period of eight months he had on occasions increased his intake to as much as 100 mg. He had also found it necessary after the first six months occasionally to take a tablet during the day to abolish withdrawal symptoms, mainly a tremor in his hands. He had read a great deal of the published information on barbiturates but, interestingly, he considered barbiturates too dangerous. There was no other evidence of drug abuse.

At the time of the interview he had been taking 8-10 tablets a day for six months but had stopped them three days before the interview in an attempt to "kick the habit." He showed moderate withdrawal symptoms, mainly subjective anxiety and tension, restlessness, inability to sleep, and loss of appetite. Physical examination showed nothing abnormal other than a tremor of the outstretched hands. He refused admission because he feared loss of his job, and withdrawal was done on an outpatient basis, under cover of chlormethiazole, which was continued for 12 weeks. His continued presence at work in the early days was facilitated by short-time working owing to the recession. Withdrawal was relatively uneventful, the main complaint being difficulty in sleeping and vivid dreams. When the chlormethiazole was stopped he began having fluctuations of mood which stabilised on 25 mg of amitriptyline at night. Six months later he showed some depression of mood consistent with his life situation but was taking no drugs.