lymphocytes, polymorphonuclear leucocytes, plasma cells, and occasional eosinophils. The cortical infiltrations were often around the glomerular vascular pole and one or two aggregations of macrophages were noted in the adventitia of small interlobular arteries. Early diffuse diabetic glomerulosclerosis was present. Immunofluorescence to IgG, IgA, fibrin, and C3 was found in glomerular capillary walls and mesangial regions. The appearances suggested that a granulomatous arteritis was developing in addition to interstitial nephritis.

Clofibrate was discontinued and the rash faded over two days. The urine output was always greater than 800 ml/24 h, but despite appropriate fluid, electrolyte, and insulin treatment the blood urea concentration rose over five days to 56 mmol/l (337 mg/100 ml). It then fell, without dialysis, to 6·5 mmol/l (39 mg/100 ml) nine days later, with a daily diuresis of 5-7 l. Three weeks after admission creatinine clearance was 85 ml/min and the urine was protein free.

Comment

All described cases of an adverse effect of clofibrate on renal function[1 4] had pre-existing chronic renal failure and were associated either with myalgia and muscle damage[1] or with severe vomiting causing salt and water depletion.[4] In none of them were renal biopsy findings reported. In the present case renal function returned almost to normal on withdrawal of clofibrate and there was no evidence of muscle damage or of vomiting and hypovolaemia. The principal feature of the renal biopsy specimen was interstitial nephritis although there was also evidence of granulomatous arteritis, presumably reflecting a general hypersensitivity to the drug. There is an association between adverse reactions to clofibrate and renal disease[1 4 5] and the drug should be avoided in patients with renal impairment.

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Liver damage induced by oxyphenisatin

Oxyphenisatin, a constituent of laxatives, has been associated with hepatocellular damage.[1-4] We describe a patient who developed prolonged hepatitis after continual ingestion of a laxative (Silklax) containing oxyphenisatin.

Case report

The patient, a 21-year-old woman, had been unwell during December 1979 with anorexia, tiredness, nausea, and an inability to drink alcohol. During January 1980 she developed jaundice. Infective hepatitis was diagnosed, although no investigations were done, and she was advised to rest. The jaundice began to disappear after three weeks but had worsened again by late April. She was admitted to hospital in June 1980 with increasing jaundice and nausea. There was no history of contact, infection, or blood transfusion, and no abdominal pain or pruritus. A detailed drug history disclosed that she had taken Silklax, purchased from a health food shop, in a dose of four tablets a day for five days each week for the past year. Silklax contains oxyphenisatin acetate 5 mg, methylcellulose 100 mg, extract of cascara 50 mg, and extract of prunes 25 mg.

On examination she was jaundiced with no signs of chronic liver disease although the liver was enlarged. Haematology was normal (erythrocyte sedimentation rate 44 mm in the first hour). Liver function tests showed gross abnormalities (table). Tests for hepatitis-associated surface antigen and lupus erythematosus cells were negative, and for antinuclear factor and smooth-muscle antibody strongly positive; non-organ-specific mitochondrial antibody and other organ-specific antibodies were not detected. Liver biopsy showed a prominent chronic inflammatory infiltrate in the portal areas, some destruction of lobular architecture, and mild patchy, piecemeal necrosis. The chronic inflammatory cells were mainly lymphocytes with a few plasma cells and eosinophils. Reticulin staining showed increased fibrosis. The picture was consistent with chronic active hepatitis.

Repeat liver function tests 10 days later (table) showed a definite improvement. Although withdrawing the drug caused some improvement, in view of the severity of changes shown by liver biopsy she was started on prednisolone 10 mg by mouth three times daily on day 11 before being discharged home. One month after admission she was feeling well, the jaundice had disappeared, and no other abnormality was detected. Liver function tests showed continuing improvement, and the prednisolone dosage was gradually reduced.

Results of liver function tests

	On admission	After 10 days	After 1 month
Serum glutamate oxaloacetate transaminase (IU/l) (normal 0–30)	1325	895	103
Serum glutamate pyruvate transaminase (IU/l) (normal 0–24)	1220	780	Not done
Bilirubin (μmol/l) (normal 3–17)	132	111	19
Alkaline phosphatase (IU/l) (normal 25–100)	204	154	93
Total proteins (g/l) (normal 62–80)	80	89	71
Albumin (g/l) (normal 35–50)	38	45	40
γ-Glutamyl transaminase (IU/l) (normal 10–55)	136	137	Not done
Immunoglobulin G (g/l) (normal 8–18)	20·0		11·91
Immunoglobulin A (g/l) (normal 0·8–4·5)	2·27		1·77
Immunoglobulin M (g/l) (normal 0·6–2·8 l)	2·52		2·27

Conversion: SI to traditional units—Bilirubin: 1 μmol/l ≈ 58 μg/100 ml.

Comment

Oxyphenisatin-induced liver damage usually occurs when the drug has been taken for at least six months and usually two years.[5] Patients may present with an acute episode of jaundice, fatigue, or abdominal discomfort, or with a more chronic course,[2] and recovery usually follows withdrawal of the drug.[5] In severe or prolonged reactions benefit has been shown with corticosteroids.

Contrary to the widely held belief that oxyphenisatin is not available in Britain, the case reported here shows that oxyphenisatin is still available over the counter in so-called health food shops. This case emphasises the importance of taking a particularly careful drug history, noting any laxatives, however benign they may appear. A wide variety of proprietary laxative preparations are available over the counter, and reluctance by the public to consider them to be drugs suggests that increased diligence is required and particular attention should be paid to their formula.

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Correction

Effects of oral propranolol on parathyroid hormone secretion in chronic renal failure

An error occurred in this paper by Dr K Farrington and others (15 November, p 1320). The top curve in the figure (▲) should have been labelled phosphate and the middle curve (■) calcium.