concentration was 2·35 mmol/l (9·4 mg/100 ml), phosphate concentration 0·82 mmol/l (2·5 mg/100 ml), and alkaline phosphatase activity 115 KA units. Liver and renal function were normal and there was mild generalised aminoaciduria.

Case 2—A 13-month-old girl underwent a routine *x*-ray examination of the hips as part of follow-up for suspected congenital dislocation of the hip detected in the neonatal period. Typical changes of rickets were seen and were also present at the wrists and knees. Serum calcium concentration was 2·24 mmol/l (9·0 mg/100 ml) phosphate concentration 0·73 mmol/l (2·3 mg/100 ml), and alkaline phosphatase activity 148 KA units.

Case 3—A 13-month-old girl was screened for rickets after the disease had been diagnosed in the first two children. Florid radiological changes were present at the wrists, knees, and ribs. Serum calcium concentration was 2·24 mmol/l (9·0 mg/100 ml), phosphate concentration 0·66 mmol/l (2·0 mg/100 ml), and alkaline phosphatase >40 KA units.

Case 4—The 20-month-old stepsister of case 1 was brought for screening by her step-mother. Radiological changes were present at knees and wrists. Serum calcium was 2·14 mmol/l (8·6 mg/100 ml), phosphate concentration 1·07 mmol/l (3·3 mg/100 ml), and alkaline phosphatase 145 KA units.

Comment

All four children were born in the United Kingdom of black Rastafarian parents of West Indian origin. They were breast fed until the second half of the first year of life, when they were weaned onto an essentially vegetarian diet known as I-tal. None of the children received vitamin supplements during infancy and none completed a full course of immunisations.

The Rastafarians do not constitute a homogeneous group and details of the diet vary. They eat very little, if any, meat, milk, fats, or oils, most of the food being vegetable in origin. For Rastafarians in Jamaica fish is an important part of the diet, providing they have scales and are less than 12 in (30 cm) long.[1] In our patients, however, fish seemed to be forbidden. Full dietary assessments were performed in cases 1 and 3. The mean daily intakes of vitamin D were estimated to be 2·2 μg in case 1 and 2·78 μg in case 3 compared with a daily recommended intake of 10 μg.[2] Daily calcium intakes were 660 mg in case 1 and 696 mg in case 3, recommended daily intake 600 mg.[2] The diets were also low in iron; indeed, all our patients had low serum iron concentrations, though their haemoglobin concentrations were within normal ranges.

Risk factors for rickets in our patients include prolonged breast feeding without vitamin supplementation and a vegetarian diet low in vitamin D. The families live in a depressed inner city area where opportunities for outdoor play are few and exposure to sunlight is likely to be limited.

This series is similar to that reported by Edidin *et al*,[3] many of whom were black and had had prolonged breast feeding, no vitamin supplementation, and few, if any, immunisations. None of their patients were described as Rastafarian.

Infantile rickets has also been reported in Britain in children of West Indian, Cypriot, and other immigrant groups who did not receive vitamin D supplements or welfare foods in London in the early 1960s.[4] Children of Rastafarian families living in the United Kingdom appear to be at risk of nutritional disorders, particularly rickets, and further studies are indicated to assess the magnitude of the problem.

We thank Dr J H Baumer for detecting case 2 and Drs M G Mott and P M Dunn for permission to describe their patients.

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Cholestatic jaundice in fascioliasis treated with niclofolan

Fascioliasis, which occurs widely throughout the world, can produce the symptoms of urticaria, jaundice, tender hepatomegaly, and eosinophilia and abnormal results of liver function tests. The usual treatment is with emetine hydrochloride, bithionol, and now praziquantel.[1] Niclofolan has been extensively used in the treatment of fascioliasis in sheep and cattle and recently in man.[2] We describe a patient with fascioliasis treated with niclofolan who developed cholestasis that was probably related to the drug.

Case report

A 37-year-old Yemen woman was investigated in Sanaa, Yemen, for occasional loose stools and dull epigastric discomfort. Ova of *Fasciola hepatica* were found in the stool, but no treatment was given and the diarrhoea subsided. One year later she was found to have a few eggs of *F hepatica* still present in the stool.

White blood count showed an eosinophilia of 10%. Her body weight was 58 kg, and she was treated with niclofolan 0·5 mg/kg twice daily for three days. She had considerable vomiting and abdominal pain during treatment but these stopped when the treatment was stopped. A week later she complained of generalised itching, and four days later she was jaundiced and passing dark urine. She did not feel tired or anorexic. She was not taking any other drug or a contraceptive pill. On examination she was jaundiced with an erythematous rash and scratch marks on her arms and legs. The liver was not enlarged or tender. Examination was otherwise normal. Investigations showed a mild rise in aspartate transaminase and alkaline phosphatase activities with a considerable increase in bilirubin concentration. Severe eosinophilia was noted (table).

*Clinical findings**

	Normal range	18 June	28 June	6 July	23 July	29 July
Aspartate transaminase (IU/l)	5–40	37	63	88	25	23
Alkaline phosphatase (IU/l)	30–110	113	154	165	81	72
Total bilirubin (μmol/l)	5–17	184	152	127	28	14
Albumin (g/l)		41	40	39	39	41
White cell count ($\times 10^9$/l)		7·4			5·1	6·5
% Eosinophils		28			13	11

*Testing for hepatitis B surface antigen yielded negative results.
Conversion: SI to traditional units—Bilirubin: 1 μmol/l ≈ 58·5 μg/100 ml.

Three concentrated stool specimens were analysed, but ova of *F hepatica* were not found. Ultrasonography of the biliary tract and gall bladder showed no abnormality. Liver biopsy showed a normal architecture with pronounced centrilobular canalicular cholestasis; there was mild inflammatory infiltrate in the portal spaces, in which eosinophils were prominent. No eggs were seen. Percutaneous transhepatic cholangiography showed normal intrahepatic and extrahepatic bile ducts.

After three weeks of persistent itching and jaundice the symptoms subsided gradually and results of liver function tests became normal; mild eosinophilia persisted.

Comment

This patient suffered from *F hepatica* infestation presenting as diarrhoea and confirmed by the finding of *F hepatica* in the stools. She never suffered from jaundice or pruritus and had been asymptomatic for one year. One week after starting niclofolan she developed cholestasis, which lasted for five weeks. Percutaneous cholangiography showed patent intrahepatic and extrahepatic bile ducts. The histological picture showed normal liver architecture with centrilobular canalicular cholestasis, mild inflammatory infiltrate, and eosinophils compatible with drug-related liver injury.

Niclofolan has been used to treat fascioliasis in sheep and cattle and paragonimiasis, clonorchiasis, and fascioliasis in man.[2–5] Side effects include transitory muscle and abdominal pain, nausea, sweating, and transient mild rash. Slight increases in serum aspartate transaminase and alkaline phosphatase activities have been noted,[5] but jaundice has not been described. Severe infections with *F hepatica* and other trematodes such as opisthorchiasis and clonorchiasis cause cholangitis and subsequent cholestasis. Niclofolan can also cause cholestasis, and this may lead to diagnostic confusion. Establishing the temporal relation of the jaundice to administration of the drug

and if necessary performing liver biopsy and endoscopic or percutaneous cholangiography should enable the correct diagnosis to be made.

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Chronic Q fever endocarditis causing massive splenomegaly and hypersplenism

Q fever endocarditis is an uncommon condition in which splenomegaly almost invariably occurs; massive splenomegaly, however, is extremely rare. We report on a patient with aortic and mitral valve prostheses who developed massive splenomegaly with hypersplenism. Q fever endocarditis was diagnosed four months later when for the first time he developed systemic symptoms.

Case report

A 50-year-old man presented with dyspnoea and angina in 1973. Investigations confirmed aortic and mitral stenosis and regurgitation, and in February 1974 both valves were replaced with Starr-Edwards prostheses. He made an excellent postoperative recovery. In December 1978 a palpable splenic tip was noted but he was asymptomatic. Investigations showed a raised erythrocyte sedimentation rate, leucopenia, mild thrombocytopenia, and mildly abnormal results of liver function tests (table), all of which had previously been normal. Multiple blood cultures were sterile. He remained asymptomatic, but the spleen gradually enlarged to 18 cm below the left costal margin. In February 1980 the liver edge was found to be 2 cm below the right costal margin. Normal results were obtained for hepatitis serology and antinuclear factor and other autoantibodies. A bone-marrow biopsy specimen was normal. Liver biopsy showed lymphocytic infiltration of the portal tracts, which was non-diagnostic.

In August 1980 he developed dragging left hypochondrial pain that increased in severity until he could not walk without discomfort. The only other complaint was mild fatigue. The prosthetic heart sounds were normal, but haematological results remained abnormal (table). In January 1981 splenectomy was performed. The spleen weighed 1500 g, and microscopy showed congested splenic sinuses with reactive hilar lymph glands. Liver biopsy again showed lymphocytic infiltrate in the portal tracts. He made an excellent clinical and haematological recovery, but results of liver function tests remained abnormal (table).

Thee months later he developed malaise and fever. Examination showed a temperature of 37·6°C, normal prosthetic heart sounds, a liver edge 2 cm below the right costal margin, and no peripheral evidence of endocarditis. Urine examination showed only protein. Results of liver function tests deteriorated further (table); multiple blood cultures, brucella agglutinin and chlamydia B (psittacosis) titres, and results of viral studies were negative. Titres of phase I and phase II *Coxiella burneti* agglutinins, however, were 1/256 and greater than 1/512 respectively, giving unequivocal evidence of chronic Q fever infection. He was started on tetracycline 2 g daily by mouth and after four days felt well. His temperature returned to normal and there was some improvement in the results of liver function tests (table). Three weeks later he developed a staphylococcal septicaemia, from which he made a good recovery after treatment with cloxacillin and gentamicin, although at one stage he showed signs of heart failure. At about this time the tetracycline was changed to co-trimoxazole 960 mg twice daily. He continued to take this drug and at follow-up after about one year was reasonably well; valve replacement did not appear to be indicated.

Comment

Q fever, first described in 1937,[1] is usually seen in an acute form as a febrile illness or an atypical pneumonia, but also in a chronic form as Q fever endocarditis. Prosthetic valve endocarditis has been well described.[2] The only clinical sign that suggests Q fever endocarditis rather than other causes of endocarditis is said to be hepatomegaly.[2]

Results of investigations in our patient were typical, with sterile blood cultures, abnormal results of liver function tests, mild thrombocytopenia, and raised immunoglobulin concentrations.[3] The unusual feature was the development of massive splenomegaly with hypersplenism. The findings on splenectomy suggested the diagnosis of "non-tropical idiopathic splenomegaly" as proposed by Dacie *et al.*[4] This case emphasises the need for Q fever endocarditis to be considered as a cause of massive splenomegaly in a patient with heart valve abnormality.

Requests for reprints should be sent to JH.

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Correction

Residual amblyopia in recruits to the British Army

We regret that in this paper by Bryan Hopkisson *et al* (2 October, p 940) the prevalences of amblyopia were expressed incorrectly. In the Subjects, methods, and results the penultimate sentence should have read: "Combining the two years' results gave a mean prevalence of 4·4% (with 95% confidence limits 3·5% and 5·3%) in the men and 4·6% (confidence limits 3·1% and 6·1%) in the women."

Results of clinical investigations throughout course of disease (normal ranges given in parentheses)

Date	Haemoglobin (g/dl) (13–18)	White cell count (× 10⁹/l) (4000–11 000)	Platelets (× 10¹²/l) (130–450)	Erythrocyte sedimentation rate (mm in first h) (3–15)	Bilirubin (μmol/l) (0–17)	Aspartate transaminase (IU/l) (10–30)	Alanine transaminase (IU/l) (6–37)	Alkaline phosphatase (U/l) (36–92)	Globulin (g/l) (15–30)
Dec 1978	12·8	2 600	140	57	18	51	24	68	36
Feb 1980	13·0	2 800	80	45	16	95	52	89	29
Feb 1981	14·6	11 700	360	38	19	126	111	720	36
May 1981 (at diagnosis)	11·0	10 200	320	47	46	229	131	791	39
June 1981 (after two weeks' treatment)	10·9	9 800	320	70	29	118	62	538	36
June 1982 (after one year's treatment)	11·9	6 900	305	21	10	54	38	133	32

Conversion: SI to traditional units—Bilirubin: 1 μmol/l ≈ 58·5 μg/100 ml.