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

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[1] Rollo IM. Chemotherapy of parasitic diseases. In: Gilman AG, Goodman LS, Gilman A, eds. *Goodman and Gilman's the pharmacological basis of therapeutics*. New York: McMillan, 1980:1024-35.
[2] Eckhard T, Heckers H. Treatment of human fascioliasis with niclofolan. *Gastroenterology* 1981;**81**:795-8.
[3] Rim MH. Studies on the chemotherapy of trematode infection in Korea. *Korea Medical Journal* 1975;**12**:425-57.
[4] Nwokolo C, Voikmer KH. Single dose therapy of paragonimiasis with menichlopholan. *Am J Trop Med Hyg* 1977;**26**:688-92.
[5] Soh CT, Im KG. Therapeutic trial of niclofolan against clonorchis sinensis infection. *Yonsei Reports on Tropical Medicine* 1977;**8**:72-8.

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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.

[1] Derrick VH. Q fever. A new fever entity: clinical features, diagnosis and laboratory investigations. *Med J Aust* 1937;**2**:281.
[2] Varma MPS, Adgey AAJ, Connolly JH. Chronic Q fever endocarditis. *Br Heart J* 1980;**43**:695-9.
[3] Turck WPG, Howitt G, Turnberg LA, *et al.* Chronic Q fever. *Q J Med* 1976;**45**:193-217.
[4] Dacie JV, Brain MC, Harrison CV, Lewis SM, Worlledge SM. "Non-tropical idiopathic splenomegaly" ("primary hypersplenism"). A review of ten cases and their relationship to malignant lymphomas. *Br J Haematol* 1969;**17**:317-33.

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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.