tions is unclear. The groups compared may have been too small to obtain statistical significance. These groups were similar in size to those of others,[1 2] however, and since the trends were different in men and women this would seem to be an unlikely explanation. Alprenolol-induced increases in urate concentration have been shown to return baseline levels within two years despite continuous treatment.[2] If this decreasing effect of alprenolol on urate is also true for other beta-blockers, no difference in urate values should be expected between patients treated long term with beta-blockers and those not treated with beta-blockers. In fact, no differences were found in this study, where the patients taking beta-blockers had been treated with such agents for an average of more than four years before the study. Moreover, this study confirmed the positive correlation between urate and triglyceride concentrations, and since triglyceride values correlate negatively with HDL-cholesterol concentrations, the negative correlation between urate and HDL-cholesterol concentrations is not surprising, although the underlying mechanism is unknown. Thus the lack of a difference in the plasma lipid and lipoprotein concentrations between patients on long-term beta-blocker treatment and those not on such treatment may be explained by the observed interrelationships between urate and these lipids and by assuming that propranolol, like alprenolol, has a decreasing effect on triglyceride and HDL-cholesterol concentrations, as well as on urate values.

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Arthritis and arthralgia associated with toxocaral infestation

Arthritis and arthralgia have been associated with various worm infestations—for example, filariasis[1] and dracontiasis.[2] Commonly retinal lesions have been associated with toxocaral infestation,[3] but encephalitis and disease of the liver, lung, and heart may occur. We report a case of arthritis and arthralgia associated with toxocaral infestation.

Case report

In April 1977 an 18-year-old woman presented with a seven-day history of blurring of vision in the left eye. She had kept dogs and rabbits as pets for many years. On examination visual acuity was 6/6 on the right and 6/24 on the left. Examination of the left fundus showed an area of new choroiditis and a pigmented scar near the macula. The vitreous was noted to be cloudy. White cell count was $8{\cdot}3 \times 10^9$/l and erythrocyte sedimentation rate 3 mm in first hour. No eosinophil count was carried out. A toxoplasma dye test gave negative results. Her symptoms settled with oral prednisolone 30 mg daily and betamethasone eye drops.

In November 1977 she presented with transient swelling and stiffness of the right elbow and left wrist and ankle. On examination limitation of movement and slight swelling of the affected joints were noted. White cell count, erythrocyte sedimentation rate, liver function tests, antistreptolysin O titres, autoantibodies, rheumatoid factor, Wassermann reaction, and toxoplasma dye and haemagglutination tests were all normal. HLA typing was A2, A28, B12, and B14. Her symptoms improved on treatment with benorylate.

Between January 1978 and January 1979 she suffered repeated episodes of choroiditis in the left eye and arthralgia. A toxocaral fluorescence antibody test was performed, which was positive. The eosinophil count was 2×10^8/l. She was treated with a 21-day course of diethylcarbamazine citrate (3 mg/kg). After this her ocular and joint symptoms settled rapidly and oral steroids and non-steroidal anti-inflammatory drugs were tailed off.

Comment

We are unaware of any other reports of arthritis and arthralgia associated with toxocaral infestation. In visceral larva migrans larvae of *Toxocara canis* migrate through lymphatic and vascular channels throughout the body. Commonly the liver, lung, heart, and eyes are affected. Presumably joint symptoms might occur from direct joint disease as in dracontiasis. In this case joint aspiration was not possible owing to the transient, flitting nature of her arthritis.

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Polycythaemia rubra vera and congenital deafness in monozygotic twins

Polycythaemia rubra vera occurring in families is rare, and we can find no previous report of it in monozygotic twins. The cases we describe provide an interesting parallel with the recent report of primary thrombocythaemia in uniovular twins.[1]

Case reports

CASE 1

A 49-year-old woman who had been deaf since birth presented in 1954 after an episode of haematemesis and melaena. On examination splenomegaly was noted, and investigation showed haemoglobin concentration 14·4 g/dl; white cell count $37{\cdot}2 \times 10^9$/l with a normal differential; platelet count 319×10^9/l, and hypercellular bone marrow. Polycythaemia rubra vera was diagnosed, and she was treated with phosphorus-32 and splenic irradiation.

Over subsequent years she suffered from chronic gastrointestinal blood loss from an undetermined site, with asymptomatic hypochromic microcytic anaemia (haemoglobin concentration 6·4-10·0 g/dl) that showed rapid rises to 17·0 g/dl and 20·4 g/dl after courses of oral iron. In March 1968, with a haemoglobin concentration of 21·3 g/dl, she suffered a fatal mesenteric vein thrombosis. Her white cell count had remained raised during the whole of the illness.

CASE 2

The twin of the first patient, who was physically identical to her and shared the same blood group (B rhesus positive), presented in January 1980, aged 75 years, with a rash of some months' duration. Skin biopsy showed the histology of eczema, which responded satisfactorily to topical steroids. The tip of the spleen was palpable, and splenomegaly was confirmed by technetium scan.

Investigations showed: haemoglobin 19·3 g/dl; red cell count 6780×10^9/l; packed cell volume 0·614; white cell count $21{\cdot}0 \times 10^9$/l (54% neutrophils, 39% lymphocytes, 5% monocytes, 2% eosinophils); platelet count 235×10^9/l; red cell volume 2020 ml (48 ml/kg); arterial oxygen pressure 13·1 kPa; and neutrophil alkaline phosphatase score 225 (normal range 10-110). Haemoglobin electrophoresis showed a normal pattern and an intravenous pyelogram no abnormality. A bone-marrow aspirate was hypercellular with no other abnormality. The bone-marrow karyotype was 46xx with no Ph^1 chromosome. Like her twin sister, she had been deaf since birth. Audiometry showed high-tone loss of inner-ear origin compatible with congenital deafness. She was treated by regular venesection to maintain a packed cell volume below 0·45. White cell and platelet counts remained unchanged.