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Correspondence

C4 deficiency, SLE, and Whipple's disease

SIR, We read with interest the report of a patient with C4 deficiency, SLE, and Whipple's disease.[1] We would like to propose an alternative to the diagnosis of Whipple's disease in this case.

There have been several recent publications highlighting the striking resemblance of the lesions of *Mycobacterium avium-intracellulare* infection to those of Whipple's disease.[2–4] PAS-positive macrophages are found in tissue infected by *M. avium-intracellulare* as well as in Whipple's disease; the bacillary bodies seen by electron microscopy in each condition are indistinguishable.[3 4]

The authors postulate that complement deficiency, SLE itself, and steroid therapy all rendered their patient more susceptible to Whipple's disease. The same argument suggests *M. avium-intracellulare* infection. Since the special procedures required to culture this organism[5] were not carried out, the diagnosis of Whipple's disease remains uncertain.

An acid-fast stain, said to be negative in Whipple's disease and positive in *M. avium-intracellulare* infection,[2 3] could be performed on tissues obtained at necropsy in this case. The results would be of great interest.

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Tachyarrhythmia in yersinia arthritis

SIR, Heart disease with abnormalities of conduction and rhythm as well as valvular dysfunction is well known to be an acute or even late manifestation of reactive postinfectious arthritis.[1 2] Involvement of the heart in yersinia arthritis is reported in 7–14% of cases, where the features are murmurs, friction rub, heart enlargement, premature ventricular beats, ST segment elevation, and negative T waves.[2–4] We wish to report a case with tachyarrhythmia and atrial fibrillation during the acute course of yersinia arthritis.

A 43-year-old male patient (height 186 cm, weight 114 kg) was admitted to hospital because of tachyarrhythmia, atrial fibrillation, and fever (40°C). Initially the radiological examination of the thorax showed discrete heart enlargement. The electrocardiogram revealed tachyarrhythmia 140/min, atrial fibrillation, and slight ST segment elevation in leads V2 to V4. The muscle enzyme creatine phosphokinase was maximal 90 U/l and subsequently normal. The year before, during treatment for nephrolithiasis (calcium oxalate), the electrocardiogram had revealed a normal sinus rhythm. The day after admission to hospital the patient developed polyarthritis with symmetrical involvement of the large joints (ankles, knees, elbows, and hands) except the hip. He had no diarrhoea and no abdominal pain, blood cultures were negative, and stool cultures were negative for yersiniae, salmonellae, shigellae, and campylobacter. Blood titres to *Yersinia enterocolitica* I rose (1:1200 to 1:2600) and subsequently decreased (to 1:360) within eight weeks. The HLA antigen B27 was positive, whereas tests for rheumatoid factor and antistreptolysin titres were negative. The patient had no urethritis, no iritis, no radiological signs of spondylitis, and no skin manifestations.

The echocardiogram revealed no valvular dysfunction and a normal-sized left atrium. Hyperthyroidism was excluded by laboratory investigation. Blood cultures for streptococci and other causes of endocarditis were negative. Atrial fibrillation was treated for eight weeks with digoxin 0·2 mg and quinidine 1200 mg daily, and a stable sinus rhythm was restored. No further antiarrhythmic medication was required, and electrocardiogram and heart radiology were normal 12 weeks later, even after withdrawal of digoxin and quinidine. We therefore conclude that tachyarrhythmia due to atrial fibrillation should be considered a manifestation of heart involvement during the acute phase in postinfectious yersinia arthritis.

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