size. The nodules can cause haemoptysis, pneumothorax or bronchopleural fistula depending on their location. Pneumothorax in RA may be associated with eosinophilia, high ESR and other pulmonary manifestations of RA, such as pulmonary fibrosis and vasculitis [4]. Our patient also had eosinophilia at the time of presentation.

Anecdotal reports suggest that methotrexate might exacerbate subcutaneous nodulosis in RA [1]. Gotsman et al. [1] has described a case of spontaneous pneumothorax developing in an RA patient treated with methotrexate. As in other cases of pneumothorax secondary to pulmonary rheumatoid nodules, their patient was a male with longstanding RA. In view of these reports, we avoided disease-modifying treatment with methotrexate in our patient because of concern that this might aggravate pulmonary nodulosis.

To our knowledge this is the first described case of a patient with spontaneous pneumothorax due to a pulmonary rheumatoid nodule which preceded the development of RA.

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Renal microinfarction in Behc¸et’s disease

SIR, We describe a patient with Behc¸et’s disease and renal cortical microinfarction whose renal impairment stabilized following anticoagulation. Behc¸et’ disease is a chronic inflammatory vascular disorder of unknown aetiology. Vasculitis, thrombosis and infarction have been reported in a variety of organs with varying frequency. Renal involvement in Behc¸et’s disease is uncommon and includes amyloidosis, crescentic glomerulonephritis and IgA nephropathy. In the literature there is only one report of acute renal infarction due to Behc¸et’s disease [1]. We describe a patient with Behc¸et’s disease with bilateral renal microinfaracts confirmed by computed tomography (CT) and DMSA renal perfusion scans. The patient gave his informed consent for publication.

A 38-yr-old West African man developed sore throats, headaches, swinging fevers and joint pains mainly in the right wrist, which was hot, tender and swollen. During the pyrexial episodes he noted macroscopic haematuria. He then developed oral and genital ulcers, sterile pustules and a symmetrical peripheral neuropathy. Investigations showed negativity for antinuclear antibodies, anti-extractable nuclear antigen, anti-DNA and anticardiolipin antibodies, lupus anticoagulant and antineutrophil cytoplasmic antibodies, and normal serum complement levels. Renal function was abnormal: urea 12.4 mmol/l, serum creatinine 155 mol/l. The erythrocyte sedimentation rate (ESR) was 96 mm in the 1st hour and C-reactive protein (CRP) was 70 mg/l. Radiographs of the chest, hands and feet were normal. A formal pathergy test was negative but he frequently noted pustules developing at venepuncture sites. Abdominal CT scan and an intravenous urogram showed bilateral kidney scarring. A renal biopsy showed no evidence of a thrombotic microangiopathy and skin biopsy showed a neutrophil-rich perivascular infiltrate in the mid-dermis. He was diagnosed with Behc¸et’s disease complicated by renal microinfarction. HLA B51 was negative. He responded well to three 500 mg methylprednisolone pulses and 20 mg oral prednisolone reducing to 12.5 mg daily and colchicine 0.5 mg twice daily. However, he continued to have flares of Behc¸et’s disease every 6 weeks with episodes of fever, oral, nasal and genital ulcers, skin lesions and arthralgia. Azathioprine was commenced but was stopped after 6 months because of neutropenia. On methotrexate 10 mg weekly he improved, with normalization of his ESR and CRP levels. Five years after presentation, he developed hypertension. His creatinine clearance was 74 ml/min and there were no casts in his urine. He responded to lisinopril and nifedipine.

Two years later, whilst his disease was still in remission, he was investigated following an episode of macroscopic haematuria. His radioisotope glomerular filtration rate was 64 ml/min. Cystoscopy was normal and a DMSA scan showed bilateral shrunken scarred kidneys with an equal division of glomerular filtration rate. Antiphospholipid antibodies remained persistently negative and a full thrombophilia screen was negative. He had never had any urinary tract infections and the most likely diagnosis was bilateral renal cortical
scarring as a consequence of microinfarcts complicating his Behçet’s disease. He was commenced on warfarin (target INR 2–3), he had no further episodes of macroscopic haematuria, and his creatinine concentration was stable at 119 mol/l. A repeat DMSA scan 1 yr later showed no progression of the renal scars (Fig. 1). Currently, his renal function remains stable with a creatinine concentration of 110 mol/l and his INR has been very stable within the target range.

Behçet’s disease is a multisystem disease characterized by recurrent oral and genital ulceration. The disease may also involve the eyes, skin, joints, nervous system, gastrointestinal tract, kidney and major vessels, including large arteries and veins. Large-vessel involvement of the arterial and venous systems is a major cause of morbidity and mortality [2]. Arterial lesions may occur in the systemic circulation or the pulmonary arterial bed: stenoses, occlusions and aneurysms frequently coexist.

Renal involvement was previously regarded as virtually unknown [3]. During the last two decades, reports have documented a wide spectrum of renal manifestations in Behçet’s disease, although these complications remain very uncommon. A review of the literature reveals a multitude of glomerular abnormalities in Behçet’s disease, varying from minimal-change disease [4] to proliferative glomerulonephritis (focal or diffuse) and rapidly progressive crescentic glomerulonephritis [5]. Occasional cases of mesangial proliferative glomerulonephritis and membranous nephropathy have also been reported [6]. Mesangiocapillary glomerulonephritis with granular deposits of IgA along the capillary walls was described in a patient with Behçet’s syndrome [7] and several cases of nephrotic syndrome secondary to amyloidosis have been documented. Behçet’s syndrome is one of the few vasculitides that can involve large and small veins.

Thrombophlebitis occurs in 25% of all patients and is most frequent in the legs, though upper limb veins are not spared. Major vessel thrombosis is less common but is more severe in males. Thrombosis may lead to vena cava obstruction and occlusion of the suprahepatic veins or renal veins, as well as calf vein thrombosis and pulmonary emboli. Thrombosis has been attributed to underlying vasculitis and is associated with a high risk of mortality. Conventional treatment includes immunosuppression, though the role of anticoagulation remains controversial [8]. Renal vein thrombosis is rare and there are only a few reports in the literature in relation to Behçet’s syndrome [4].

In conclusion, we describe a patient with Behçet’s disease characterized by oral and genital ulcers, peripheral neuropathy, arthritis and microinfarcts of both kidneys, revealed by DMSA scan, who responded to immunosuppression and anticoagulation.

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