Bilateral pseudothrombophlebitis

Sir: Katz *et al* defined the pseudothrombophlebitis syndrome as the presence of signs and symptoms of thrombophlebitis secondary to intact, dissected, or ruptured popliteal cysts demonstrated by arthrography.¹ Subsequently, many other causes of pseudothrombophlebitis have been described, but Baker's cyst remains the most common cause of the syndrome. We report a case in which a bilateral pseudothrombophlebitis was secondary to ruptured Baker's cysts of both knees. To the best of our knowledge this clinical situation has not been previously reported.

A 59 year old man was admitted to hospital because of pain, swelling, and erythema of both calves. Over the preceding 10 years he had had recurrent attacks of transient migratory monoarthritis of the larger joints, which cleared up within a few days either spontaneously or with anti-inflammatory agents. Two weeks before the current admission he developed a synovial effusion of the left knee joint without evidence of previous local trauma. An arthrocentesis ruled out the presence of crystals or micro-organisms, and he was treated with anti-inflammatory drugs. One week later he developed a synovial effusion of the contralateral knee joint with pain, tumefaction, and erythema of both calves.

On admission, blood pressure was 140/80 mmHg and temperature 37°C. There was difficulty in walking and both Homans' sign and Löwenberg's test were positive. The remainder of the physical examination was normal. The laboratory results showed a sedimentation rate of 121 mm/h and an increase of other acute phase reactants. The complete blood count, muscle enzyme activity, rheumatoid factor, antinuclear antibody test, D-xylose test, and the upper gastrointestinal series were either normal or negative. A deep vein thrombosis was ruled out by a phlebogram and Doppler ultrasound study. An ultrasound examination of the limbs showed the presence of fluid in the popliteal regions of both knees, extending along the fascial planes as far as the ankle. A culture of synovial fluid obtained by fine needle aspiration was negative. A bilateral nuclear magnetic resonance scan (figure) showed liquid collection extending down from the popliteal space to the lower third of both legs. These findings were considered compatible with a diagnosis of complicated popliteal cyst. An operation was performed, initially on the left knee, owing to the persistence of the symptoms after one month of conservative treatment with rest and

anti-inflammatory agents. During the operation a large Baker's cyst dissecting through the fascial planes of the gastrocnemius and soleus muscles of the calf was resected. Three months later the other knee was operated on.

Both intact Baker's cysts and those complicated by rupture or dissection may manifest clinically as thrombophlebitis. The differential diagnosis may be at times impossible.² Moreover, popliteal cysts may develop in patients without prior arthropathy,³ so that the absence of a previous history of joint involvement does not exclude the possibility of this diagnosis. A phlebogram will exclude thrombophlebitis, but the diagnosis of Baker's cyst is best shown by arthrography, which is currently considered the most sensitive diagnostic method available. Computed tomographic scans and ultrasonography are other useful diagnostic examinations, but nuclear magnetic resonance scans have been seldom used. A Baker's cyst might also coexist with a thrombophlebitis, which is probably secondary to the cyst itself (pseudo-pseudothrombophlebitis).^{4,5}

We were prompted to report this case as the existence of bilateral Baker's cyst is uncommon and their simultaneous complication producing a clinical picture of bilateral pseudothrombophlebitis is extremely unusual. In our patient an arthrography was not deemed necessary before the operation because of the precise demonstration of complicated popliteal cyst by both ultrasonography and nuclear magnetic resonance examinations. We feel that nuclear magnetic resonance is an equally useful method for confirming the presence of a Baker's cyst in patients with a clinical diagnosis of pseudothrombophlebitis.

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Cytomegalovirus pneumonia in a patient with rheumatoid arthritis treated with low dose methotrexate and prednisone

Sir: In a recent issue of the *Annals* Wallis *et al* emphasised the possibility of opportunistic infection in patients treated with low dose methotrexate.¹ We report the case of a patient with rheumatoid arthritis receiving weekly low dose oral methotrexate in conventional dosage and prednisolone (10 mg daily) who presented with cytomegalovirus pneumonia.

The patient, a 51 year old man, had an eight year history of seropositive rheumatoid arthritis. He was treated for two years with oral methotrexate, 7.5 mg weekly. He also received prednisolone (10 mg daily) and indomethacin (100 mg daily). He was admitted with cough and fever (39-41°C) without breathlessness. There were no abnormal breath sounds, no signs of heart failure, and no lymphadenopathy or hepatosplenomegaly. Two days after admission a non-palpable purpuric eruption appeared on both legs. Investigations disclosed: erythrocyte sedimentation rate 16 mm/1st hour; haemoglobin 125 g/l; leucocytes $12 \times 10^9/l$, 38% neutrophils, 3% eosinophils, 52% lymphocytes, 7% monocytes with atypical blue lymphocytes; platelets $328 \times 10^9/l$; serum aspartate transaminase 70 IU (normal <50); serum alanine transaminase 72 IU (normal <60); alkaline phosphatase 260 IU (normal <110); γ -glutamyltransferase 357 IU (normal <50). Renal function was normal. Abdominal ultrasound examination showed no abnormalities. Chest radiograph showed mild interstitial pulmonary infiltration. Arterial oxygen and CO₂ pressures were respectively 5.7 kPa and 3.8 kPa. Bacterial blood cultures were negative. Bronchoalveolar lavage showed 990×10^9 cells/l with 68% lymphocytes while bacterial and fungal cultures remained negative. As assessed by monoclonal antibodies, cytomegalovirus antigen was absent in blood and urine but present in bronchoalveolar lavage after 48 hours of culture. Cytomegalovirus serology was positive (IgG >1/3200, IgM >1/400). Methotrexate treatment was stopped and antibiotics were prescribed before the diagnosis of cytomegalovirus pneumonia was established. Cough, fever, and purpuric eruption disappeared in three weeks. Haematological values and hepatic tests returned to normal.

Our patient clearly did have cytomegalovirus pneumonia. The incidence of cytomegalovirus infection in the immunocompromised host is well reported.² Methotrexate may be more immunosuppressive than previously suspected. To our knowledge cytomegalovirus pneumonia has not been described during treatment of rheumatoid arthritis with low doses of methotrexate. As in the case of Wallis *et al*¹ corticosteroid treatment and interaction between methotrexate and indomethacin might have contributed to the development of cytomegalovirus infection. In any case, acute pulmonary symptoms during low dose methotrexate treatment must not only suggest hypersensitivity pneumonitis³ but also opportunistic infection.

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Nuclear magnetic resonance scan of the limbs. Bilateral Baker's cyst dissecting down through the fascial planes.