infiltrate consisting of lymphocytes and plasma cells of grade 4 according to Chisholm and Mason [5].

A diagnosis of primary Sjögren’s syndrome was made according to the revised European classification criteria for Sjögren’s syndrome [6]. Prednisolone (1 mg/kg/day) treatment was started with some improvement in the low back pain. The patient was discharged.

Ten days later after his discharge, the patient was referred back to us because of recent onset haematochezia and mild left iliac fossa pain. A repeat abdominal CT-scan with enhancement showed no signs suggestive of aneurismal fistulation, whereas blood tests showed decreased haemoglobin levels (9.2 g/dl) with MCV 86.7 fl.

Blood transfusions were performed to treat the anaemia, however, the patient’s clinical condition rapidly deteriorated and he died soon afterwards. Necropsy demonstrated a left posterior–lateral transmural myocardial infarction, which was judged to be the likely cause of death.

Discussion

CP has already been described in association with various autoimmune disorders, including Hashimoto’s thyroiditis, vasculitis, SLE and primary biliary cirrhosis [2–4]. To our knowledge, this is the first reported case of primary Sjögren’s syndrome associated with CP. Recently, a case-control study, which compared inflammatory and non-inflammatory abdominal aortic aneurysms, showed an autoimmune disease in 19% of patients with IAAA, compared with none of the control subjects [2]. Likewise, in a recent prospective study Vaglio et al. found that in 7 (44%) out of 16 cases CP was associated with other autoimmune conditions, namely ANCA-positive renal disease and autoimmune thyroiditis [3].

The association with other autoimmune disorders suggests that CP is a manifestation of a systemic autoimmune process rather than an exaggerated local reaction to atherosclerosis [4]. In this particular case, the presence of constitutional symptoms, raised acute-phase reactants, and increased vascular uptake in the abdominal aorta and/or iliac artery at FDG-PET scan consistent with large-vessel vasculitis further support the concept that IAAA is a systemic inflammatory process [1, 7].

This case, together with the published literature, suggests that patients with CP should be investigated for the presence of underlying autoimmune disorders, including Sjögren’s syndrome.

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Neonatal antiphospholipid syndrome associated with heterozygous methylenetetrahydrofolate reductase C677T and prothrombin G20210A gene mutations

Sir, Neonatal antiphospholipid syndrome (APS) is a rare clinical entity characterized by neonatal thrombotic disease due to the transplacental passage of maternal antiphospholipid antibodies (aPL) [1]. While women with aPL show a high incidence of obstetric and fetal complications, the aPL-related thrombosis in their offspring seems to be exceedingly rare. The low frequency of neonatal thrombosis has been attributed to the lack of the most known ‘second hit’ risk factors in infants, and to a low transplacental passage of IgG2 subclass of aPL, which are responsible for most clinical pathogenicity [2]. We describe a neonate who developed ischaemic stroke in the left middle cerebral artery and was found to have positive aPL together with two underlying inherited prothrombotic disorders.
The girl was born to healthy, unrelated parents after the first uncomplicated pregnancy at 40 weeks of gestation. Maternal grandfather had leg thrombosis and died of heart infarction and maternal uncle suffered perinatal intracranial haemorrhage. The delivery was uneventful except for meconium in amniotic fluid. The girl weighted 3280 g (P50), was 50 cm long (P50), had head circumference of 35 cm (P50) and Apgar score 9/9. At the age of 13 h she developed convulsions, which were stopped by phenobarbitone. Diagnostic workup revealed normal blood count, glucose and electrolytes. She had no laboratory signs of infection and blood culture remained sterile. An electroencephalogram was focally abnormal. Magnetic resonance imaging of the head showed ischaemic changes in the left hemisphere (Fig. 1A) and magnetic resonance angiography identified occlusion of the left-middle cerebral artery (Fig. 1B). Heart ultrasound demonstrated atrial septal defect type secundum.

The coagulation profile, which included prothrombin time, thrombin time, partial thromboplastin time, platelet count, fibrinogen, antithrombin III, protein S, protein C and lipoprotein, was normal. Molecular genetic testing demonstrated heterozygous methylentetrahydrofolate reductase C677T and prothrombin G20210A gene mutations. The G1691A factor V Leiden mutation was negative. Testing for aPL was performed 14 days after the delivery in the mother and showed very high levels of IgG anti-cardiolipin antibodies (>100 GPL), negative anti-β2 glycoprotein I antibodies and negative lupus anticoagulant. Subsequently, we confirmed positive IgG antcardiolipin antibodies (24 GPL) also in the baby at the age of 7 weeks. She had no recurrence of seizures on therapy with phenobarbitone, but her neurological status remained abnormal with trunk hypotonia. There is an increasing number of case reports describing neonates or infants who have suffered from thromboses associated with transplacently acquired aPL [3–6]. Generally, it is believed that aPL per se are unable to induce thrombotic process in the intact neonatal vessel wall and it has been suggested that another thrombophilic factor, such as indwelling catheter, sepsis or prematurity may act as an initial trigger [3, 6]. Hereditary prothrombotic conditions, in particular methylentetrahydrofolate reductase C677T and prothrombin G20210A gene mutations, had not been routinely investigated in previous studies of neonatal APS. Our case report demonstrated for the first time the concomitant presence of antiphospholipid antibodies and multiple prothrombotic gene mutations in a neonate with thrombosis. This finding further supports the multifactorial pathogenesis of neonatal APS and highlights the importance of thorough laboratory evaluation, including genetic testing of possible inherited prothrombotic abnormalities.

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Psoriatic arthritis patients doing better on infliximab than etanercept

SIR, In July 2006, the National Institute for Health and Clinical Excellence (NICE) issued guidance on the use of anti-TNF therapy in psoriatic arthritis (PsA) [1]. They recommend etanercept as the first-line anti-TNF agent for severe active PsA. Infliximab is only indicated when a patient is intolerant of, or has contraindications to, etanercept, or has difficulties with self-administered injections. The guidance states that failure to respond to etanercept should not be substituted with infliximab. We present two patients with severe longstanding PsA who went onto infliximab with benefit, then changed over to etanercept for the convenience of subcutaneous administration, but when their response was unsatisfactory returned to infliximab.

Patient A

A 49-yr-old female diagnosed with PsA in 1995, had severe symptoms including pain in most joints and prolonged early morning stiffness, despite receiving methotrexate, sulfasalazine, oral prednisolone and leflunomide. She required arthrodesis in both wrists and synovectomies in both knees. Psoriasis skin