arthritis). Sixty nine patients were diagnosed as having non-IJD, of whom 31 scored 3 or more on the questionnaire and 38 scored less than 3. For a score of 3 or more the questionnaire diagnosed IJD with a sensitivity of 97% (30/31), a specificity of 55% (38/69), a positive predictive value of 49% (30/61) and a negative predictive value of 97% (38/39).

Given both its accuracy in identifying patients with non-IJD and high sensitivity in identifying patients with IJD, the questionnaire appears to offer a means of differentiating urgent referrals from routine cases. Further studies are necessary to establish reproducibility of the tool when used by other medical and nursing staff, in the hope that GPs could use this tool to determine the urgency of referral—using three or more positive answers as criteria for prompt action. This will potentially reduce delay from onset of symptoms to initiation of DMARD treatment in patients with IJD.

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Anti-annexin V antibodies in patients with cerebrovascular disease

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Annexin V (ANXV) is a protein with a high affinity for negatively charged phospholipids and shows in vitro a potent anticoagulant activity. It has been suggested that it has a significant role in the prevention of arteriovenous thromboses or fetal loss, or both.[1] Increased levels of antibodies against ANXV (aANXV) have been reported in patients with different systemic autoimmune disorders[2–4] as well as in women with recurrent fetal loss and pre-eclampsia.[5] The presence of aANXV in patients with thromboembolic cerebrovascular disease (CVD), however, has not yet been described. We report on two patients with CVD who had evidently raised levels of IgG aANXV, whereas all the other tested antiphospholipid antibodies (aPL) were negative.

We examined 37 young patients with no evident systemic autoimmune disease (23 women, 14 men; mean age at CVD 32 years (range 18–40)) 11 months to six years after CVD: seven with transient ischaemic attack (TIA), 25 with ischaemic cerebrovascular insult, and five with venous sinus thrombosis. Diagnoses based on the history and clinical manifestations were objectively verified by computed tomography (CT), magnetic resonance imaging (MRI), and/or angiography at the time of the onset of symptoms. After prospective clinical re-examination, two blood samples were obtained from each patient eight weeks apart.

Serum samples were analysed by enzyme linked immunosorbent assay (ELISA) for the presence of aANXV,[5] anticardiolipin,[6] anti-β_2-glycoprotein,[7] and anti-prothrombin antibodies.[8] Antinuclear antibodies (ANA) were determined by indirect immunofluorescence.

CASE REPORTS

Patient 1

A 36 year old woman with a history of fetal loss in 1982 became pregnant for the second time in 1998. At the 36th gestation week a caesarean section was performed owing to placental abruption. A few days after the delivery, she became somnolent with mild right sided hemiparesis. CT and an MRI scan confirmed superior sagittal sinus thrombosis and therefore treatment with warfarin was started. Three years later, her condition was stable with mild occasional headaches and mild right sided pyramidal symptomatology. Laboratory examinations showed positive ANA (up to 1/320) and persistently raised levels of IgG aANXV, while all the other tested aPL were negative. No clinical manifestations of a systemic autoimmune disease could be found. Except for a short period of smoking, no other thrombotic risk factors were identified.

Patient 2

A 24 year old woman had a TIA in 1996, two months after starting hormonal contraceptives. She experienced paraesthesia over both arms and legs and gait ataxia was found. MRI, echocardiography, and sonography of the neck vessels were normal, suggesting TIA in the vertebrobasilar region. In 2000 she became pregnant for the first time. Generalised oedema and hypertension appeared in the fifth and eighth month, respectively. A healthy child was born one month pre-term. In 2001 she was in good health except for rather frequent headaches. Clinical and special neurological examinations were completely normal. Among the tested aPL only IgG aANXV were found to be positive. Contraceptives were the only risk factor for CVD.

DISCUSSION

ANXV is one of the possible cofactors for aPL. Rand *et al* reported that aPL can disrupt the protective shield of ANXV on procoagulant surfaces,[9] leaving sufficient space for the formation of coagulation complexes. aANXV were shown to induce the apoptosis of endothelial cells, creating a procoagulant environment[10] with increased risk for thrombosis.

Two of 37 young patients after CVD had significantly raised IgG aANXV only. Besides some CVD risk factors (smoking, delivery and bleeding, oral contraceptives) both patients had pregnancy complications, which might be associated with aANXV.[5] Our results did not show a statistically significant association between aANXV and CVD. Nevertheless it is possible that aANXV represented an additional risk factor, and together with other factors might have led to thrombosis. A study of larger groups of patients will enable firm conclusions to be drawn about the clinical significance of aANXV in CVD.

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Intra-alveolar haemorrhage in temporal arteritis

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Temporal arteritis (TA) is the most common systemic vasculitis. We report herein a case of TA complicated with intra-alveolar haemorrhage. To our knowledge, this manifestation has not previously been reported.

CASE REPORT

A woman born in 1926 presented in 1999 with persistent dry cough and raised erythrocyte sedimentation rate at 60 mm at the first hour. C reactive protein was 14 mg/l. She had a history of pulmonary tuberculosis treated in 1951 with streptomycin and *p*-aminosalicylic acid. She complained of headache without fever, jaw claudication, scalp tenderness, and visual or musculoskeletal manifestations. She denied any other upper airways symptoms. Physical examination was normal. Arterial pressure was 140/70 mm Hg. Leucocyte count was 6.8×10^9/l with 4.2×10^9/l polynuclear neutrophils and 0.2×10^9/l eosinophils, haemoglobin 130 g/l, and platelets 310×10^9/l. A dipstick urinary test showed no proteinuria and no haematuria. Chest radiography disclosed calcified nodular density in the upper right lobe, which was confirmed by computed tomography. An electrocardiogram and echocardiogram were normal. Fibre optic bronchoscopy was normal. Bronchoalveolar lavage fluid examination showed 120×10^9 cells/l, comprising macrophages 56% with siderophages 30%, lymphocytes 39%, polynuclear neutrophils 1%, and polynuclear eosinophils 4%. Bronchoalveolar lavage was sterile on cultures for bacterial infection, *Mycobacterium tuberculosis*, cytomegalovirus, parasites, and fungi. A search for antinuclear antibodies and cryoglobulin was negative. Histological study of the temporal artery disclosed a granulomatous inflammation of the vessel wall containing mononuclear cells and histiocytes without giant cells, leading to fragmentation of the elastic lamina.

Prednisone 30 mg daily was started. The headache and cough disappeared rapidly. At the two year follow up, the patient is asymptomatic with 7 mg daily prednisone. C reactive protein is negative.

DISCUSSION

This patient had four of five American College of Rheumatology criteria for the classification of TA, which have a sensitivity of 93.5% and a specificity of 91.2%.[1] Chronic dry cough without any chest radiograph anomaly is a classical symptom of TA. Machado in Minnesota reported respiratory symptoms in up to 25% of cases of TA.[2] Cough may be the sole manifestation of the disease for a long time—up to one year.[3] A three year course of cough associated with upper limb arteritis was also reported as an initial symptom of TA.[4] The origin of the cough is not clear. In rare cases, cough might have been attributed to pleural effusion, interstitial pneumonitis, pulmonary vasculitis, or hyperreactive airways.[5]

Blockmans *et al* found T4 lymphocytic alveolitis in three patients with histologically proved TA.[6] Only one patient complained of dry cough and dyspnoea. Chest radiographs and lung function tests were always normal. Cell count was normal on bronchoalveolar lavage fluid examination, but the number of lymphocytes was increased, as in our case, with a percentage varying between 16 and 61%. Discovery of nodular parenchymal densities raises the question of a borderline systemic vasculitis with Wegener granulomatosis.[4]

In our case, there was no obvious cause for intra-alveolar haemorrhage besides TA. Congestive cardiac failure and exogenous agents were excluded. Bacteriological, viral, fungal, and parasitic studies of bronchoalveolar lavage fluid were negative. There was no manifestation suggesting another systemic disease such as microscopic polyangiitis, Wegener's granulomatosis or vasculitis secondary to systemic lupus erythematosus. The favourable outcome with medium dose prednisone in the absence of any other immunosuppressive agent also suggests TA. Cytomegalovirus infection was ruled out, but not other viral infections. Several viruses such as cytomegalovirus,[7] coxsackievirus,[8] adenovirus,[9] enterovirus,[10] mumps,[11] and hepatitis C virus[12] can cause pulmonary haemorrhage. Some authors have suggested that TA has a viral cause, and various agents were incriminated. Varicella zoster[13] and parvovirus B19 DNA[14] were found in temporal biopsy specimens of