RON VERHEIJEN
WALTHER J VAN VENROOIJ
Department of Biochemistry
University of Nijmegen
Nijmegen
The Netherlands

ALEXANDRA J CROCKEWIT
Department of Haematology
University Hospital
Nijmegen
The Netherlands

Correspondence to: Frank H J van den Hoogen,
Department of Rheumatology, University Hospital,
PO Box 9101, 6500 HB Nijmegen, The
Netherlands.

- 1 Graf M W, Uhr J W. Regulation of antibody formation by serum antibody. I. Removal of specific antibody by means of immunoadsorption. *J Exp Med* 1969; **130**: 1175-86.
- 2 Bystryn J C, Graf M W, Uhr J W. Regulation of antibody formation by serum antibody. II. Removal of specific antibody by means of exchange transfusion. *J Exp Med* 1970; **132**: 1279-87.
- 3 Verrier Jones J, Robinson M F, Parcianny R K, Layfer L F, McLeod B. Therapeutic plasmapheresis in systemic lupus erythematosus. Effect on immune complexes and antibodies to DNA. *Arthritis Rheum* 1981; **24**: 1113-20.
- 4 Dau P C, Kahaleh M B, Sagebiel R W. Plasmapheresis and immunosuppressive drug therapy in scleroderma. *Arthritis Rheum* 1981; **24**: 1128-36.
- 5 Ferri C, Bernini L, Gremignai G, et al. Plasmaexchange in the treatment of progressive systemic sclerosis. *Plasma Therapy and Transfusion Technology* 1987; **8**: 169-76.
- 6 Wollheim F A, Akesson A. Treatment of systemic sclerosis in 1988. *Semin Arthritis Rheum* 1989; **18**: 181-8.
- 7 Guillevin L, Amoura Z, Merviel PH, et al. Treatment of progressive systemic sclerosis by plasma exchange: long-term results in 40 patients. *Int J Artif Organs* 1990; **13**: 125-8.
- 8 Verheijen R, van den Hoogen F, Beijer R, et al. A recombinant topoisomerase I used for autoantibody detection in sera from patients with systemic sclerosis. *Clin Exp Immunol* 1990; **80**: 38-43.
- 9 Schroeder J O, Euler H H, Löffler H. Synchronization of plasmapheresis and pulse cyclophosphamide in severe systemic lupus erythematosus. *Ann Intern Med* 1987; **107**: 344-6.

Polyarteritis nodosa following angioplasty

Sir: Polyarteritis nodosa is a rare condition characterised by necrotising arteritis of small and medium sized arteries.¹ Infection may act as a trigger, with from 10%² to 54%³ of patients being hepatitis B surface antigen positive, but evidence of infection cannot be found in all patients. We report a case of PAN which occurred after femoral angioplasty.

A 54 year old man with a one year history of bilateral claudication was admitted for angiography and angioplasty. A heavy smoker from the age of 20, he otherwise had no significant past medical history. There was no relevant family history of vascular disease.

A preoperative full blood count, urea and electrolytes, fasting lipids, electrocardiogram, and results of a chest x ray examination were normal. Femoral angiography confirmed the presence of short segment bilateral atheromatous stenoses of the femoral arteries at the level of the adductor hiatus. As symptoms were more marked on the left he underwent angioplasty of that side. The immediate postoperative period was uneventful.

One week after discharge he developed pain and swelling of the small joints of the hands, wrists, elbows, shoulders, and ankles. His condition was managed by his general

practitioner. An initial full blood count was unremarkable, erythrocyte sedimentation rate (ESR) was 6 mm/h, and rheumatoid latex was negative.

His condition continued to deteriorate. Six weeks after the onset of his symptoms he was admitted for investigation of a 24 hour history of pain and swelling of the left calf.

On admission he was feverish (38°C, blood pressure 120/70 mmHg). There was an acute polyarthritis of proximal interphalangeal and metacarpophalangeal joints, wrists, shoulder, knees, and ankles. Proximal muscle groups were tender, the left calf was painful and swollen. There was no rash. Investigation showed haemoglobin 139 g/l, neutrophils $8.73 \times 10^9/l$, ESR 68 mm/h, urea and electrolytes normal, alkaline phosphatase 281 IU/l, alanine transaminase 46 U/l, creatine kinase 400 mg/l. Autoantibodies, including rheumatoid arthritis latex, antinuclear antibodies, and antineutrophil cytoplasmic antibodies, were negative. Blood cultures and hepatitis B surface antigen were negative. Midstream urine showed 3+ red blood cells. Doppler studies of the calf showed no evidence of deep venous thrombosis.

An electromyogram was consistent with myositis. Muscle biopsy of the left calf disclosed necrotising arteritis, with no muscle fibres affected. Mesenteric angiography showed numerous small aneurysms of the hepatic, intrahepatic, splenic, and renal arteries, consistent with the diagnosis of polyarteritis nodosa.

He was treated with prednisolone and cyclophosphamide and is currently well.

The temporal relation between the angioplasty and the development of polyarteritis nodosa in this case strongly suggests a link. Vascular damage certainly occurs during angioplasty and up to 2% of all femoral angioplasties require surgical intervention.⁴ Thus several mechanisms might have stimulated the immune response, leading to the development of vasculitis. The simplest of these involves the exposure of previously hidden epitopes to which the patient was not tolerant. Besides simple traumatic damage to the vascular endothelium, however, angioplasty may also render the area temporarily ischaemic. In coronary angioplasty this is known to result in the generation of superoxides.⁵ In this case it is interesting to note that the left calf was the worst affected area. Could ischaemically generated superoxides have altered endothelial constituents, rendering them antigenic? This is the first reported case of vasculitis after angioplasty. With the increasing use of such techniques, more can presumably be expected. The procedure provides an interesting opportunity for further research.

G R STRUTHERS
M T PUGH
Department of Rheumatology
Coventry and Warwickshire Hospital
Stoney Stanton Road
Coventry CV1 4FH
United Kingdom

D A K WOODWARD
Department of Vascular Surgery
Walsgrave Hospital
Clifford Bridge Road
Coventry CV2 2DX
United Kingdom

Correspondence to: Dr Struthers.

- 1 Cohen R D, Conn D L, Ilstrup D M. Clinical features, prognosis and response to treatment in polyarteritis. *Mayo Clin Proc* 1980; **55**: 146.

- 2 Conn D L, McDuffie F C, Holley K E, Schroeter A L. Immunologic mechanisms in systemic vasculitis. *Mayo Clin Proc* 1976; **51**: 511.
- 3 Trepo C G, Zukerman A J, Bird R C, Prince A M. The role of circulating hepatitis B antigen/antibody immune complexes in the pathogenesis of vascular and hepatic manifestations in polyarteritis nodosa. *J Clin Pathol* 1974; **27**: 863-5.
- 4 Katzen B T. Percutaneous transluminal angioplasty in the treatment of peripheral vascular disease. In: Dudley H, Carter D, eds. *Rob and Smiths operative surgery*. 4th ed. London: Butterworth, 1985.
- 5 Roberts M J D, Young I S, Troughton T G, et al. Transient release of lipid peroxides after coronary artery balloon angioplasty. *Lancet* 1990; **336**: 143-5.

Buerger's disease and antiphospholipid antibodies in pregnancy

Sir: Buerger's disease is a vasculitis of unknown cause, in which several factors have been implicated, one of the most important of which is smoking.¹ We present a case in which Buerger's disease was associated with a primary antiphospholipid syndrome in a pregnant woman.

A patient aged 38 was sent to our centre in her 32nd week of gestation with a suspected diagnosis of intrauterine growth retardation. Her obstetric history included two intrauterine fetal deaths at 30 and 32 weeks, and between them a male child born alive at term but who was small for the gestational age. The patient had smoked more than 30 cigarettes daily for the previous three years and had had a history of Raynaud's disease in the fingers and toes for nine years, with distal necrosis of the middle finger of the right hand. On examination, the absence of left radial and pedal pulse and signs of necrosis of the middle finger of the right hand were noteworthy. Uterine height was 22 cm, which clearly was low for the gestational age. Blood analysis showed persistent thrombocytopenia, with a platelet count between 100 and $140 \times 10^9/l$. Tests for antinuclear antibody, complement, and rheumatoid factor were negative. Anticardiolipin antibodies (by enzyme linked immunosorbent assay (ELISA)) were positive at 40 GPL units in two tests done eight weeks apart. (In our laboratory we consider positive levels of IgM or IgG anticardiolipin antibodies to be values greater than 20 MPL or GPL units respectively.) Obstetric sonography showed a live fetus with biometers corresponding to 26 weeks of gestation. The diagnosis was severe symmetrical intrauterine growth retardation.

As we suspected Buerger's disease a Doppler study of proximal arteries was done, and the results were normal. Non-stress tests showed an absence of fetal reactivity, with decrease of variability and presence of late decelerations, and as a result a caesarean section was performed. A live newborn weighing 1000 g was delivered, with an Apgar score of 6-8. His postnatal evolution was favourable. Anatomopathological examination of the placenta indicated multiple infarcts and calcifications, and no signs of vasculitis were noted. An arteriograph of the hands, carried out after delivery, indicated multiple stenoses and screwdriver lesions in radial, cubital, and palmar arches, which are all compatible with Buerger's disease. The lesions were bilateral.

Buerger's disease and primary antiphospholipid syndrome have in common the existence of arteriovenous thrombosis, though their cause is different. We have not found any report of their coexistence. Although our patient presents diagnostic criteria of Buerger's disease,^{2,3} arterial thrombosis is a clinical diagnostic criterion of primary antiphospholipid syndrome, so in this case the possibility that all the abnormalities noted may be due exclusively to this syndrome cannot be ruled out. We believe this may be a coincidence, but more studies are needed to rule out any possible relation.

M CASELLAS
A PÉREZ
L CABERO
Department of Obstetrics and Gynaecology
Hospital Materno infantil. Ciudad Sanitaria Vall
d'Hebrón
Universitat Autònoma de Barcelona
P^a Vall D'Hebrón s/n
Barcelona, Spain

A SEGURA
E PUIG DE MORALES
Department of Internal Medicine
Hospital Materno infantil. Ciudad Sanitaria Vall
d'Hebrón
Universitat Autònoma de Barcelona
P^a Vall D'Hebrón s/n
Barcelona, Spain

R SELVA O'CALLAGHAN
Department of Internal Medicine
General Residence, Ciudad Sanitaria Vall
Universitat Autònoma de Barcelona
P^a Vall D'Hebrón s/n
Barcelona, Spain

- 1 Buerger L. Thromboangiitis obliterans: a study of the vascular lesions leading to presenile spontaneous gangrene. *Am J Med Sci* 1908; **136**: 567-80.
- 2 Rivera R. Roentgenographic diagnosis of Buerger's disease. *J Cardiovasc Surg* 1973; **14**: 40-51.
- 3 McPherson J F, Juergens J L, Giftord R W J. Thromboangiitis obliterans and arteriosclerosis obliterans: clinical and prognostic differences. *Ann Intern Med* 1963; **59**: 288-96.
- 4 Harris E N. Syndrome of the "black swan". *Br J Rheumatol* 1987; **26**: 324-6.

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