10 Spector T D, Ollier W E R, Perry L A, Silman A J, Thompson P W, Edwards A. Free and serum testosterone levels in 276 males: a comparative study of rheumatoid arthritis, ankylosing spondylitis and healthy controls. *Clin Rheumatol* 1989; **8:** 37–41.
11 Tapia-Serrano R, Jimenez-Balderas F J, Murrieta S, Bravo-Gatica C, Guerra R, Mintz G. Testicular function in active ankylosing spondylitis: therapeutic responses to chorionic gonadotropin. *J Rheumatol* 1991; **18:** 841–8.
12 Gordon D, Beastall G H, Thomson J A, Sturrock R D. Androgenic status and sexual function in males with rheumatoid arthritis and ankylosing spondylitis. *Q J Med* 1986; **60:** 671–9.
13 Ollier W, Spector T, Silman A, *et al.* Are certain HLA haplotypes responsible for low testosterone levels in males? *Dis Markers* 1989; **7:** 139–43.
14 Semple C G. Hormonal changes in non-endocrine disease. *BMJ* 1986; **293:** 1049–52.

Asymmetric rheumatoid vasculitis in a hemiplegic patient

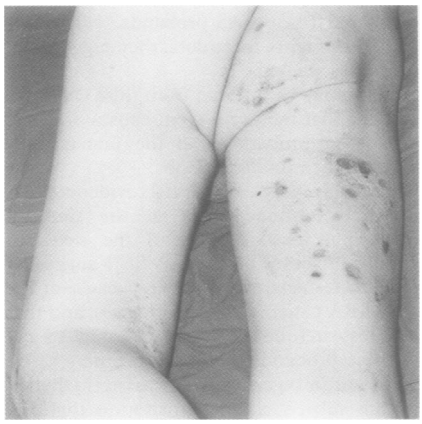
In 1962, Thomson and Bywaters described the phenomenon of a unilateral rheumatoid arthritis occurring in a hemiplegic patient;[1] Oblick subsequently described a similar sparing in a patient in whom the limbs had been paralysed as a result of poliomyelitis.[2] This protection was initially explained by lack of use of the hemiplegic limb, sparing the joints. More recently, a neurogenic mechanism has been proposed.[3]

The case to be described is of rheumatoid vasculitis which largely spared the hemiplegic side—a phenomenon not described previously. It is suggested that the asymmetric distribution supports a neurogenic mechanism.

The patient, who suffered from an arteriopathy, had his first myocardial infarction at the age of 37. A second infarct one year later was followed rapidly by a cerebrovascular accident, thought to arise from embolism of a left ventricular wall thrombus, seen on the echocardiogram. This resulted in a dense left hemiplegia. In 1988, at the age of 48, he developed seropositive rheumatoid arthritis and presented with a predominance of right sided synovitis, involving the right hand, knee and ankle, although there was mild involvement of the left hemiplegic side. He was treated with Voltarol retard, sulphasalazine 1 g twice daily and chloroquine 250 mg three times a week, which produced good disease control and a decrease in the erythrocyte sedimentation rate from 50 to 25 mm/1st h. Radiographs of the hands, taken two years later, showed bilateral erosions; joint space loss was considerably greater on the right side.

In April 1994, at the age of 51, the patient presented with a predominantly right sided vasculitic rash involving his hand, arm, foot, thigh, and buttock, with vasculitic ulcers on his right thigh and buttock (figure). He had nail fold infarcts of the right fingers and toes. Peripheral pulses were present and equal; joints were not active. There was no evidence of peripheral neuropathy, and no fever. There had been no recent change in medication to suggest a drug induced aetiology. Urine analysis was negative. Immunological studies showed him to be negative to antinuclear factor, antineutrophil cytoplasmic antibodies, and lupus anticoagulant, but positive to La(SS-B) antibody. A skin biopsy demonstrated a leucocytoclastic vasculitis. He was treated with intermittent pulsed intravenous cyclophosphamide 15 mg/kg and prednisolone. The rash faded and healed over the first two weeks of treatment, and he made a good subsequent recovery.

Whilst one could explain the unilateral rheumatoid joint involvement in a hemiplegic patient in terms of joint sparing in the paralysed limb, this mechanism could not be evoked to explain the predominantly unilateral distribution of the vasculitis to the non-hemiplegic side as described in this patient. This may provide further support for a neurogenic mechanism.



Asymmetric vasculitic rash on thigh.

Evidence for sensory nerve involvement in chronic arthritis comes from animal studies in which sectioning of the sciatic nerve seven days before the induction of adjuvant arthritis delayed the onset and severity of disease in the operated limb.[4] Levine has suggested that neuromechanisms may also explain why rheumatoid arthritis is bilaterally symmetric.[5] Evidence is growing that these effects are mediated through substance P in the sympathetic nervous system.[6]

The vasculitis in rheumatoid arthritis is predominantly small vessel and is caused by a precipitate of immune complex with subsequent polymorphic leucocyte infiltration.[7] Why the vasculitis should have occurred asymmetrically in this patient is unknown. There was no recent change in his medication to suggest a drug induced aetiology. The cerebrovascular accident predated the vasculitis by 10 years and was embolic; it was thus unlikely to have been the result of a single vascular process such as Sneddon's syndrome. One could speculate that alteration in vascular sympathetic tone in the hemiplegic limb[8 9] could decrease the local precipitation of immune complexes and thus explain the unilateral rheumatoid vasculitis described here.

A L DOLAN
Department of Rheumatology
Greenwich District Hospital
London SE10 9HE, United Kingdom

1 Thomson M, Bywaters E G L. Unilateral rheumatoid arthritis following hemiplegia. *Ann Rheum Dis* 1962; **21:** 370–7.
2 Glick E N. Asymmetrical rheumatoid arthritis after poliomyelitis. *BMJ* 1967; **iii:** 26–9.
3 Kidd B L, Mapp P I, Blake D R, Gibson S J, Polak J M. Neurogenic influences in arthritis. *Ann Rheum Dis* 1990; **49:** 649–52.
4 Courtright L J, Kuzzell W C. Sparing effects on neurological deficit and trauma on the course of adjuvant arthritis in the rat. *Ann Rheum Dis* 1965; **24:** 360–7.
5 Levine J D, Collier D H, Basbaum A I, Moskowitz M A, Helms C A. The nervous system may contribute to the pathophysiology of rheumatoid arthritis. *J Rheumatol* 1985; **12:** 406–11.
6 Garrett N E, Mapp P I, Cruwys S C, Kidd B L, Blake D R. Role of substance P in inflammatory arthritis. *Ann Rheum Dis* 1992; **51:** 1014–8.
7 Scott D G I, Bacon P A, Tribe C R. Systemic rheumatoid vasculitis: a clinical and laboratory study of 50 cases. *Medicine* 1981; **60:** 288–97.
8 Hachinski V C, Oppenheimer S M, Wilson J X, Guiraudon C, Cechetto D F. Asymmetry of sympathetic consequences of experimental stroke. *Arch Neurol* 1992; **49:** 697–702.
9 Uncini A, DiMuzio A, Lugaresi A, Gambi D. Sympathetic skin response in hemispheric lesions. *Neurophysiol Clin* 1992; **22:** 475–81.