tal pulp ulcers (3, 4).
In contrast, patients with pSS harbor a variety of serological traits which are associated with extraglandular involvements. Let us mention non organ-specific autoantibodies, monoclonal immunoglobulin (5), IgA-rheumatoid factor, IgA-containing immune complexes (6), and, above all, mixed cryoglobulinemia (7). The authors are right in assigning leg ulceration to the latest abnormality, and highlighting its predictive value in pSS for adverse outcomes, most notably non Hodgkin lymphoma (8).
To conclude, we might (should ?) indeed have quoted leg ulceration as a cutaneous manifestations in our review. We acknowledge the interesting contribution of Perry, Gordon and Porter, and agree that the occurrence of vasculitic ulceration should alert to a diagnostic of mixed cryoglobulin.

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Bilateral vertebral artery occlusion in giant cell arteritis

Sirs,
Cerebrovascular accidents are one of the leading causes of GCA-related morbidity and mortality, and they are probably the most common cause of early death after diagnosis in GCA (1, 2). Involvement of the vertebrobasilar system results in strokes of the cerebellum, occipital lobe and brain stem. We report a case of bilateral vertebral artery occlusion (BVAO) due to GCA with acute bilateral cerebellar stroke without development of irreversible neurological complications.
A 78-year-old man was sent to our hospital because of dizziness. One year before, he had been diagnosed with polymyalgia rheumatica in another center. After that diagnosis he was started on treatment with prednisone 10 mg/day. However, to our surprise, the prednisone dosage had been maintained without progressive reduction since the time of diagnosis. Twenty-four hours before admission he began to complain of vertigo and headache. Twelve hours before admission he suffered an episode of right amaurosis fugax. Neurological examination showed gait unsteadiness and ataxia. Laboratory data, including coagulation tests, anticardiolipin antibodies, full blood cell count, and hepatic and renal function parameters, were negative or normal. The ESR was 84 mm/1h, and CRP was 63 mg/L (normal: < 5 mg/L). Plain chest radiograph and electrocardiogram were normal. A T2-weighted and Flair MRI sequences showed several hyperintense foci at both cerebellar hemispheres. Angio-MRI showed BVAO (Fig. 1).
A biopsy of the right temporal artery performed 24 hours after admission showed interruption of the internal elastic laminae with infiltration of mononuclear cells into the arterial wall. Treatment with acetylsalicylic acid (300 mg/day) was started and prednisone dose was increased up to 60 mg/day. Dramatic improvement of symptoms was achieved. The patient was discharged from hospital two weeks later without neurological sequelae.
Atherosclerosis, GCA itself, and trauma are the main causes of BVAO. Headache and elevation of inflammatory laboratory markers may be of some help to consider a potential diagnosis of GCA in patients with BVAO (3). The much more accelerated BVAO related to GCA may contribute to the far higher mortality observed in GCA patients within the first month after the diagnosis of the disease (4).
The reason why our patient did not follow a catastrophic course is uncertain. Compensatory neovascularization phenomena, related to the persistence of a chronic and maintained inflammatory response over the disease course due to the prolonged steroid therapy for a previous polymyalgia rheumatica diagnosis, might have been responsible for compensatory mechanism which prevented the patient from the development of irreversible neurological complications (5-7).

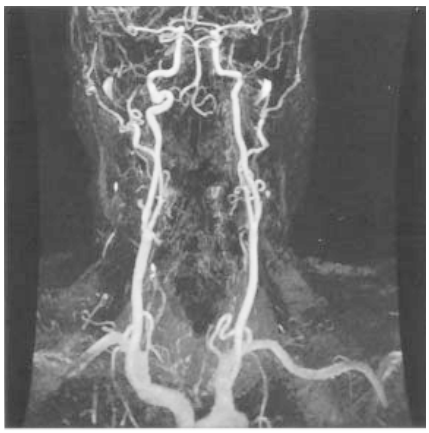

Fig. 1. Angio-MRI showing bilateral vertebral artery occlusion.

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