weight during the past three months. Physical examination disclosed a moderately ill woman. Skin and mucosae were pale. A 2·5 cm, indurated, slightly tender, not erythematous nodule was palpable in the right supraclavicular region. Temporal arteries were not swollen, but the right one beat weakly. The rest of the physical examination was normal. Laboratory data showed a mild normochromic, normocytic anaemia and an erythrocyte sedimentation rate of 92 mm/h. Biopsy of the nodule was then performed. Its histological examination disclosed an abundant fibroadipose tissue with foci of blood vessels showing inflammatory changes consisting of giant cell granulomatous vasculitis. Biopsy of the right temporal artery was also performed and a segmentary inflammatory infiltrate by aggregates of giant cells was seen. Treatment with prednisolone (60 mg/day) was started. Two days later the patient showed general improvement. Six months later, she is symptom free, with low dose steroid treatment.

Several dermatological manifestations of giant cell arteritis have been described. Most are either inflammatory changes, such as oedema, erythema, and tender nodules overlying inflamed superficial arteries, or ischaemic lesions, such as vesicles, bullae, ulcers, and gangrene due to occlusion of these vessels.[2] These abnormalities are usually located at the scalp. Other uncommon reported cutaneous manifestations include urticaria, hyperpigmentation, purpura, and ecchymoses.[2 3] Recently, Goldberg *et al* described tender nodules in lower extremities simulating erythema nodosum,[4] and Stephenson and Underwood reported mammary masses.[5] In both cases a histological examination of the nodules showed typical giant cell vasculitic lesions within the subcutaneous fat. These lesions were similar to those seen in our case. Our patient had both non-specific symptoms and laboratory findings which were not suggestive of any concrete clinical entity. Thus the main clinical manifestation was the presence of a supraclavicular nodule simulating a lymph node, its biopsy being consistent with giant cell arteritis. In addition, temporal artery biopsy was also consistent with this diagnosis and the response to steroids was dramatic.

We concluded that giant cell arteritis can cause vasculitis in the subcutaneous tissue, presenting clinically as a palpable mass. Furthermore, this mass may show the typical histological features of this disease.

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Occlusive ocular vascular disease and antiphospholipid antibodies

Sir: We read with interest the report by Asherson *et al*, which concluded that 8% of patients with systemic lupus erythematosus (SLE) and raised levels of anticardiolipin antibodies develop occlusive ocular vascular disease.[1] One of their seven patients reported had the primary antiphospholipid syndrome, which includes one or more of recurrent fetal loss, venous and arterial thrombosis, and thrombocytopenia together with raised levels of anticardiolipin antibodies or lupus anticoagulant, or both and no other well defined autoimmune disease, such as SLE.[2]

There have been a few reports linking occlusive ocular vascular disease and anticardiolipin antibodies, most of them in SLE.[1 3–6] Another recent report found no raised levels of anticardiolipin antibodies in any of the 40 patients with retinal vascular occlusion in the absence of autoimmune disease, suggesting that anticardiolipin antibodies are important only in the presence of SLE.[7]

We studied 26 patients with the primary antiphospholipid syndrome and found three with vascular retinopathy—that is, a prevalence of 12%, which is slightly higher than that found by Asherson *et al*.[1] The table records the main clinical and serological features of our patients. An ophthalmic examination of patients 1 and 2 was made because they had complained of loss of vision. Both were found to have inferotemporal branch vein occlusions. Patient 1 was also moderately hypertensive (blood pressure 170/110 mmHg). We then made a prospective ocular evaluation of our other patients with primary antiphospholipid syndrome and found a third case who showed peripheral ischaemic retinopathy. This patient had not complained of visual disturbance at any time. All the patients had normal intraocular pressure.

The higher prevalence of occlusive vascular retinopathy in patients with raised anticardiolipin antibody levels and SLE[1] and the prevalence found in our 26 cases with clinical manifestations related only to raised anticardiolipin antibody levels suggest that the presence of these antibodies, with or without any associated disease, may increase the risk of developing occlusive ocular vascular disease.

This study was supported by Fondo de Investigaciones de la Seguridad Social (Fiss).

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Occlusive ocular vascular disease and primary antiphospholipid syndrome

Patient No	*Sex*	*Age (years)*	*ACA*†*		*LA**	*ANA**	*Manifestations of PAPS**	*Ocular lesions*
			IgG	*IgM*				
1	M	30	Medium	(−)	+	−	Deep vein thrombosis, thrombocytopenia, aortic valve lesion, thrombotic renal microangiopathy	Inferotemporal branch vein occlusion, vitreous haemorrhage
2	M	43	Low	(−)	+	−	Aortic valve lesion	Inferotemporal branch vein occlusion
3	M	49	Medium	Medium	+	+ 1/320	Multi-infarct dementia, thrombocytopenia, livedo reticularis, aortic valve lesion, thrombotic renal microangiopathy	Peripheral ischaemic retinopathy

*ACA=anticardiolipin antibodies; LA=lupus anticoagulant; ANA=antinuclear antibody titre; PAPS=primary antiphospholipid syndrome.
†IgG ACA: high=>80 GPL units; medium=15–80 GPL units; low=5–15 GPL units.
IgM ACA: high=>50 MPL units; medium=6–50 MPL units; low=3–6 MPL units.