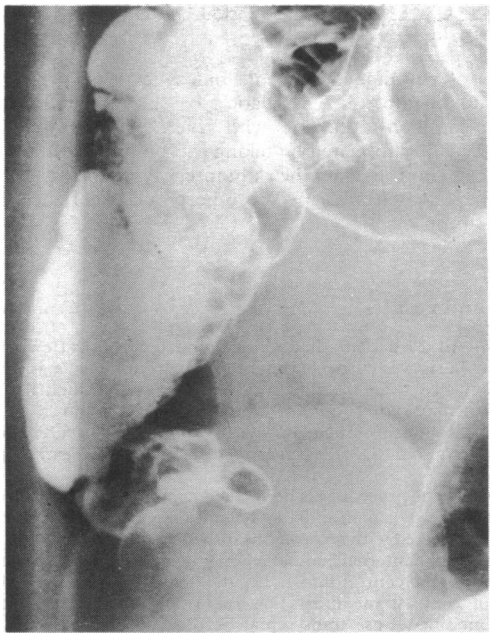

Filling defect in lower pole of caecum.

laparotomy mandatory. A caecal diaphragm must therefore become part of the differential diagnosis of a space-occupying lesion of the lower pole of the caecum found by radiology. The chance of diagnosing correctly a caecal diaphragm by radiology alone must be remote, but if at laparotomy for suspected pathology of the pole of the caecum faeces are palpated in the caecal pole and attempts to move them into the ascending colon fail then the presence of a caecal diaphragm must be seriously considered.

(*Accepted 18 March 1977*)

Bradford Royal Infirmary, West Yorkshire
A D GAYNER, FRCS, registrar
J J PRICE, FRCS, consultant surgeon
O J FOLLOWS, MRCP, FRCR, consultant radiologist

Giant-cell arteritis and hypothyroidism

The aetiology of giant-cell arteritis is unknown, though there is increasing evidence to suggest an immunological basis in which the arterial elastic tissue acts as an antigen.[1 2] An association of giant-cell arteritis with currently well-recognised autoimmune disorders would therefore be of interest and might provide indirect supporting evidence of an underlying abnormal immune host response. The simultaneous presentation of giant-cell arteritis and autoimmune thyroiditis with hypothyroidism has not previously been reported.

Case report

A 73-year-old woman was admitted to hospital with a 12-month history of lethargy and depression for which antidepressants had been prescribed. During the previous six weeks she had also complained of intolerance to cold, acroparaesthesiae, and bitemporal headaches. Relatives had noticed her to be slow and reported the recent appearance of puffiness around the eyes and hoarseness. She denied any visual disturbance, muscle pain, or joint stiffness and there was no history of endocrine disease.

On examination she looked myxoedematous with dry skin and periorbital puffiness. The thyroid was moderately enlarged and the relaxation phase of ankle jerks was slow. Both superficial temporal arteries were clinically normal, pulsatile, and non-tender. There were no abnormal vascular bruits, and all peripheral pulses were present. The locomotor system showed no abnormality. Results of investigations were: haemoglobin 9·9 g/dl, erythrocyte sedimentation rate (ESR) 115 mm in first hour, protein-bound iodine (PBI) 250 nmol/l (3·2 μg/100 ml) (normal 315–630 nmol/l (4·0–8·0 μg/100 ml)), and four-hour thyroidal uptake of ^{132}I 5·4% (normal 10–35%). Anticytoplasmic thyroid antibody titre was strongly positive and antithyroglobulin antibody titre positive at $1/2{\cdot}5 \times 10$.[6] Antigastric antibody titre was negative, and serum vitamin B_{12} and folate concentrations were within the normal ranges. Biopsy of a right superficial temporal artery showed the characteristic changes of giant-cell arteritis.

The patient was treated with oral prednisolone 40 mg daily and at the same time began thyroxine replacement therapy 50 μg daily, later gradually increased to 200 μg daily. Within one month she was entirely asymptomatic; the haemoglobin had risen to 12·1 g/dl and the ESR fallen to 33 mm in the first hour. We later reduced the dose of prednisolone with no untoward effects. At 18 months follow-up she remained well and euthyroid and was taking 2·5 mg prednisolone and 200 μg thyroxine daily.

Comment

This patient presented with the clinical features of hypothyroidism, and the diagnosis was confirmed by the findings of a goitre, strongly positive antithyroid antibody titres, low PBI, and subnormal thyroidal radioiodine uptake. The coexistence of giant-cell arteritis was suspected from the history of temporal headaches of recent onset, and, although the temporal arteries were clinically normal, the histological features on biopsy were typical of giant-cell arteritis. The protean manifestations of hypothyroidism and giant-cell arteritis are well known, and this case illustrates the importance of recognising their possible coexistence in elderly patients, as both conditions are eminently treatable.

The simultaneous presentation of giant-cell arteritis and hypothyroidism has not previously been reported. Polymyalgia rheumatica, however, which is closely related to giant-cell arteritis, was found to co-exist with thyroiditis and hypothyroidism in an elderly woman.[3] In addition, Niarchos and Finn[4] described an association between hypothyroidism and abdominal aneurysm and speculated that the arterial lesions were related to an underlying immunological mechanism. In a retrospective survey, Thomas and Croft[5] reported a higher incidence of thyrotoxicosis but not of hypothyroidism in a group of women with giant-cell arteritis than in a control group.

The coexistence of giant-cell arteritis and autoimmune thyroiditis with hypothyroidism suggests a common cause, and further studies may contribute to our understanding of the pathogenesis of giant-cell arteritis.

[1] Mowat, A G, and Hazleman, B L, *Journal of Rheumatology*, 1974, **1**, 190.
[2] Liang, G C, Simkin, P A, and Mannik, M, *Annals of Internal Medicine*, 1974, **81**, 19.
[3] Fauchald, P, Rygvold, O, and Øystese, B, *Annals of Internal Medicine*, 1972, **77**, 845.
[4] Niarchos, A P, and Finn, R, *British Medical Journal*, 1973, **4**, 110.
[5] Thomas, R D, and Croft, D N, *British Medical Journal*, 1974, **2**, 408.

(*Accepted 18 March 1977*)

University Department of Therapeutics and Clinical Pharmacology, Aberdeen AB9 2ZD
J HOW, MB, MRCP, lecturer
P D BEWSHER, MD, FRCP, senior lecturer
W WALKER, MA, FRCP, regius professor

Benign normolipoproteinaemic acanthocytosis

Acanthocytes, or spiky red cells, in neurological disorders have been described in association with normal serum lipoproteins,[1-3] hypobetalipoproteinaemia,[4] and abetalipoproteinaemia.[5] These cells are morphologically indistinguishable from "burr" cells, or "spur" cells in American reports (*acanthos*, thorn).[5] The third family in the United Kingdom with normolipoproteinaemic acanthocytosis is described here.