Simultaneous onset of giant cell arteritis and subacute thyroiditis

Sir: The classical presentation of giant cell arteritis (GCA) with headache, scalp sensitivity, and jaw claudication is caused by inflammation of the external carotid arteries or their branches. Giant cell arteritis is now recognised as a systemic illness that may be associated with generalised vasculitis.1 Subacute or giant cell thyroiditis is a self-limiting inflammation of the thyroid, presenting with painful thyroid swelling, thyrotoxicosis, and low radioactive iodine uptake. We report here on a woman with the simultaneous onset of GCA and subacute thyroiditis.

An 81 year old, previously healthy, white woman had noticed a swollen throat two weeks before admission in July 1990, followed by fever, night sweats, and moderate weight loss. Other complaints were dry cough, continuous awareness of her throat and ears, extreme jaw pain, and the scalp being sensitive to hair combing. She denied headache but had noticed a thickened blood vessel on the left lateral forehead.

Physical examination showed a moderately ill woman with blood pressure 168/85 mmHg, regular pulse rate of 104 beats/min, and temperature of 38.8°C. Both temporal arteries were tender, thickened, and vigorously pulsating. The right thyroid lobe was enlarged threefold, smoothly firm, and tender. Fundoscopy showed no vasculitis. The remainder of the examination was non-contributory. Electrocardiography and chest radiograph were normal. Laboratory data showed an erythrocyte sedimentation rate (ESR) of 120 mm/first hour, 13-8 × 10⁹ leucocytes/l with normal differentiation and 492 × 10⁹ thrombocytes/l. Abnormal biochemical values were: alkaline phosphatase 275 U/l (normal value <100 U/l), aspartate aminotransferase 42 U/l (<20 U/l), alanine aminotransferase 51 U/l (<20 U/l), and γ-glutamyltransferase 100 U/l (<52 U/l).

Thyroid function test showed moderate primary thyrotoxicosis: thyroxine 148 nmol/l (normal value 75-135 nmol/l), triiodothyronine uptake 0-99 (0-05-1-15), free thyroxine index 1-46 (0-75-1-45), and thyroid stimulating hormone 0-2 mU/l (0-3-4-0 mU/l). Antibodies to thyroid could not be demonstrated. Virus serology was positive for anti-hepatitis B surface antigen only. The thyroid scan failed to show uptake of technetium-99m pertechnetate in the thyroid area, consistent with subacute thyroiditis (figure A). Biopsy of the left temporal artery showed characteristic GCA. Similar changes obtained by thyroid aspiration showed an inflammatory infiltrate comprising macrophages, lymphocytes, granulocytes, and multinucleate giant cells. Giant cell arteritis and concomitant subacute thyroiditis were diagnosed. Treatment consisted of prednisone 30 mg twice daily, with rapid improvement of all symptoms. Two months after starting treatment her ESR had fallen to 27 mm/first hour, and the prednisone dose was gradually tapered. Liver biochemistry tests were normalised by four weeks and at eight weeks the thyroid function was normal, with moderately active uptake of isotope on a thyroid scan (figure B). Treatment with prednisone was stopped in 1992. Since then the patient has had no relapse, her ESR is 14 mm/first hour, and the thyroid scan now shows a normal uptake (figure C).

The simultaneous onset of GCA and subacute thyroiditis has to the best of our knowledge not been presented as a case report before. Malimvall et al mention two cases of subacute thyroiditis during a follow up of 68 patients with GCA, without giving detailed information of these cases.2 The combination of GCA with subacute thyroiditis may be a coincidence of two non-related disorders, but the simultaneous onset and, furthermore, the similarities in clinical presentation, histological picture, and striking response to steroids point to a more than chance relation. Either subacute thyroiditis might be a manifestation of GCA or some common causative and pathogenetic pathway might lead to both entities.

Arteries of head and neck are most frequently affected in GCA, with sparing of the intracranial arteries. Involvement of the thyroid arteries in GCA was described in 1939 by Lucien et al.3 In fact this was the first histological evidence that GCA affects arteries other than the temporal vessels. Though thyroid arteritis might have caused ischaemic thyroiditis in our patient, this could not be proved. Giant cells were found in material obtained by aspiration, but this can be a feature in both GCA and classical subacute or giant cell thyroiditis. Biopsy of the thyroid might have helped in the differentiation, but this procedure was not performed because of the significant risk.

Giant cell arteritis and thyroid disease may have manifestations of autoimmunity. Giant cell arteritis is associated with HLA types that are also associated with several autoimmune disorders.4 The association of GCA with autoimmune thyroid disease has been described,5 but its significance has been doubted. In our patient autoimmune thyroid disease was not present.

Subacute or de Quervain’s thyroiditis often follows an upper airways virus infection, but the infectious agent can rarely be identified.6 The association with certain HLA factors suggests a role for immune mechanisms in the pathogenesis.7 Both GCA and subacute thyroiditis might have been caused by virus induced alteration of autologous antigen, respectively, in artery wall and thyroid, with an immune response to these neo-antigens causing the clinical symptoms. As no serological proof of a recent infection was obtained in this patient, this hypothesis remains speculative. We believe that the subacute thyroiditis in the presented patient was caused by thyroid arteritis.

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