Figure 1 Histopathological analysis of thyroid gland (haematoxylin-eosin-saffron, original magnification $\times 250$). Giant cells and granulomatous inflammatory infiltrate near fragmented elastica tunica.

sedimentation rate 21 mm/1st h and C reactive protein 10 mg/l). A search for anti-nuclear, anti-extract cell antigen, and anti-cardiolipin antibodies as well as lupus anticoagulant was negative. Treatment with methotrexate was added (10 mg/week), which allowed progressive tapering of the prednisolone dose.

At her last medical visit in April 2000 the clinical and biological outcome was good without any sign of relapse in the GCA. Treatment with prednisone (2 mg/day), methotrexate (10 mg/week), and L-thyroxine (125 µg/day) was continued.

Associations of thyroid dysfunctions and GCA have included hypothyroidism,^{2,3} thyrotoxicosis,⁴ Graves's disease,⁵ and de Quervain's thyroiditis.⁶ In the series of Bowness *et al* 10% of the 367 patients with PMR or GCA, or both, had antithyroid autoantibodies and 4.9% had clinical and biochemical hypothyroidism requiring replacement treatment with thyroxine, which was significantly greater than in the control group.³ To explain the association between PMR and/or GCA and autoimmune thyroid diseases, the authors suggested a common aetiological agent (perhaps infective) or a common immunological predisposition. However, a recent multicentre case-control study on 285 patients with GCA failed to show a significant relation with thyroid diseases.⁷

The involvement of non-classical organs in GCA with histologically proved vasculitis has been reported for coronary, renal, hepatic, and female genital tract arteries.⁸ As thyroid arteries are branches of the external carotid artery, the occurrence in our observation of GCA and nodular goitre with thyroid arteritis was probably not fortuitous. Moreover, GCA was seen on the same side of the head and neck (respectively left temporal artery and left superior thyroid artery). To our knowledge, only one previous observation of an inflammatory infiltrate of the thyroid gland, comprising multinucleated giant cells, has been reported and concerned subacute thyroiditis occurring simultaneously with GCA.⁹ As the sample was obtained by thyroid aspiration, the author could not conclude that there was GCA involvement.⁹

From a pathophysiological point of view we feel that, in our case, thyroid ischaemic lesions (often seen in goitres¹⁰) and/or inflammatory infiltrate induced by the vasculitis process may have played a part in the formation of the goitre. Thus a possible link between GCA and goitre must be kept in mind.

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Giant cell arteritis associated with demyelinating polyradiculoneuropathy

Peripheral nerve involvement—mononeuritis,¹ mononeuritis multiplex, and polyneuropathies²—has been reported in 14% of patients with giant cell arteritis (GCA).^{3,4} GCA associated with acute inflammatory demyelinating polyneuropathy (AIDP) has exceptionally been described.^{5,6} We report an unusual case of this association.

A 67 year old woman presented with a one month history of difficulty in walking, weakness, and proximal myalgia that worsened progressively. Examination showed proximal weakness and abolition of all deep tendon reflexes; there was no sensory deficit and cranial nerve function was normal. Laboratory tests disclosed normal haematological findings with an erythrocyte sedimentation rate (ESR) of 12 mm/1st h; renal and liver function tests, muscle enzymes, and thyroid stimulating hormone were in the normal range. Serum protein electrophoresis, anti-nuclear antibodies and rheumatoid factors, and serology for Lyme disease, Q fever, mycoplasma, Venereal Disease Research Laboratory test, HIV, and cytomegalovirus were all negative. Chest radiographs and abdominal echography were normal. Electrophysiological investigation disclosed an inflammatory demyelinating polyradiculoneuropathy, with prominence of motor and proximal involvement. The cerebrospinal fluid (CSF) showed four mononuclear cells/ml; protein 0.56 g/l and glucose 3.4 mmol/l.

At this moment, an AIDP or Guillain-Barré syndrome was diagnosed, and plasma exchange was performed, using standard procedures. After the second plasma exchange, the weakness had completely disappeared, but the patient started with fever (40°C) and increasing bitemporal and occipital headache. Physical examination showed that temporal and occipital arteries were nodular and swollen with palpable pulse. Laboratory tests disclosed haemoglobin 78 g/l, platelet count $999 \times 10^9/l$, ESR 94 mm/1st h, alkaline phosphatase 170 U/l, and γ -glutamyltransferase 99 U/l. Temporal artery biopsy disclosed changes typical of GCA. Treatment with prednisone orally 1 mg/kg weight was started. There was an excellent response, the symptoms stopped within days and the ESR and liver function test returned to normal. Prednisone was reduced gradually over time. Repeated nerve conduction studies showed an objective improvement.

Ten months later, when she was taking prednisone 10 mg/day, the proximal weakness started again, worsening over the following weeks. Electrophysiological study showed

Table 1

	Case 1 ⁵	Case 2 ⁶	This study
Age/sex	71/female	73/male	67/female
Headache	Yes	Yes	Yes
Fever	No	No	Yes
Visual loss	Yes	No	No
Facial diplegia	Yes	Yes	No
PMR* symptoms	No	No	No
Latency between GCA* and PN*	AIDP* occurred 2 weeks before GCA	GCA preceded AIDP by 4 months	First episode of AIDP occurred 2 weeks before GCA
Laboratory tests			
ESR* (mm/1st h)	125	125	94
CSF*	Protein 0.1 g/l; acellular	Protein 3 g/l; acellular	Protein 0.56 g/l; cells 4/ml
Temporal artery biopsy	Not done	GCA	GCA
Nerve conduction study	Normal	AIDP	AIDP
Treatment	Prednisolone, 80 mg/day	Prednisone 75 mg/day, PEX*	Prednisone 60 mg/day, PEX, IVIg*
Clinical outcome	Spontaneous improvement of the AIDP, symptoms of GCA were controlled with steroids	TIA* during GCA prednisone treatment	Cure of GCA with prednisone; improvement of the AIDP with PEX and relapse 8 months later, clinically controlled with prednisone and IVIg
		Cure of the AIDP with PEX	

*PMR = polymyalgia rheumatica; GCA = giant cell arteritis; PN = polyneuropathy; ESR = erythrocyte sedimentation rate; CSF = cerebrospinal fluid; AIDP = acute inflammatory demyelinating polyradiculoneuropathy; PEX = plasma exchange; TIA = transient ischaemic attack; IVIg = intravenous immunoglobulin.

progression of the demyelinating polyradiculoneuropathy. She had no other symptoms. Haematological and blood chemical findings were all normal, as was the ESR. Plasma exchange sessions were restarted. The diagnosis at this time was an AIDP relapsing form. Prednisone was given at 1 mg/kg weight. She improved rapidly. Because of the serious side effects she had previously had with the steroid treatment (30 kg weight gain, hyperglycaemia, mental changes) and the poor vascular access she had for the plasma exchange, we decided to start treatment with intravenous immunoglobulin pulses, 0.4 g/kg weight every four weeks. Steroids were tapered and the patient remained clinically well during the following year.

To our knowledge, only two cases of GCA associated with AIDP have been previously reported (table 1), and no association of GCA with the AIDP relapsing form has been previously described. The first case refers to a patient who presented a clinical picture of AIDP and in whom temporal arteritis was diagnosed two weeks later.⁵ In the second case the patient was diagnosed as temporal arteritis,⁶ with a compatible biopsy; he was treated with prednisone and four weeks later he presented a generalised weakness: CSF and electrophysiological study were concordant with AIDP. A sural nerve biopsy was not done in these cases.

The neuropathies associated with temporal arteritis and other vasculitis have been attributed to ischaemic lesions of the nerves due to an arteritis of the vasa nervorum. A vasculitic neuropathy was excluded in our case because the clinical course and the electrophysiological study were characteristic of AIDP.

The underlying cause and pathogenic mechanisms of AIDP and GCA are not well understood. Immune cellular mechanisms have an important role: an increasing number of T CD4+ cells and macrophages in demyelinated regions of the nerve are seen in AIDP⁷; the histopathology of GCA shows a mixed inflammatory infiltrate with T CD4+ lymphocytes and macrophages secreting proinflammatory cytokines.⁸

Temporal arteritis associated with inflammatory demyelinating polyradiculoneuropathy may be a coincidence, but a common immunological pathogenesis, probably based on a cellular T cell dependent mechanism and related to a cytokine deregulation, cannot be ruled out. Infectious agents, such as a

virus, may be the cause of the onset of both diseases in a subset of patients.⁷⁻⁹

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Oral steroid in the treatment of carpal tunnel syndrome

A range of options are available for the conservative treatment of carpal tunnel syndrome (CTS).¹⁻⁴ Non-operative methods include immobilisation of the affected hand with wrist splint; local injection of steroids and drugs such as diuretics and non-steroidal

anti-inflammatory drugs.⁶⁻¹¹ These oral drugs are thought to decrease the volume of swollen tissue within the CTS and are widely used, but there is limited clinical evidence for their role.

This prospective randomised, double blind, placebo controlled study aimed at evaluating the effect of oral steroids in the symptomatic treatment of CTS. We recruited patients with newly diagnosed CTS of more than three months' duration with confirmatory electrophysiological results (prolonged median nerve distal motor latencies >4 ms or median ulnar palmar sensory latency difference >0.5 ms) including electromyographic recordings of the abductor pollicis brevis (APB)⁵; co-interventions such as drug or injection treatment were withheld during the study. Exclusion criteria included (a) patients with evidence of severe CTS: fibrillation potentials or reinnervation on needle examination of the APB; (b) coexisting disorders or conditions which may mimic CTS, such as cervical radiculopathy or peripheral neuropathy; (c) contraindication to steroid use; and (d) history of underlying disorders associated with CTS, such as diabetes mellitus or rheumatoid arthritis.

Patients who fulfilled the criteria were treated conservatively for two months with splinting. If symptomatic after this period, patients were allocated, using a random computer generated code, to a 10 day course of prednisolone 25 mg/day or a 10 day course of placebo. Both were given as single tablets which were identical in appearance. A physician (SMW) unaware of the treatment allocation assessed the mean global symptom score (GSS) of all patients at two and eight weeks. This is a scoring system first devised by Herskovitz which rates symptoms on a scale of 0 (no symptoms) to 10 (severe) in five categories: pain, numbness, paraesthesia, weakness/clumsiness, and nocturnal awakening. The sum of the scores in each category was the GSS.¹⁰ Median (interquartile range (IR)) changes in GSS at two and eight weeks from baseline were analysed using the Mann-Whitney test. The null hypothesis was that there was no difference in symptom score between the treatment and placebo groups. Results were considered significant at $p < 0.05$ (two sided). The sample population of 36 was planned to enable achievement of 80% power with an $\alpha = 0.05$ for detecting a 50% difference in GSS between the treatment groups, assuming