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
		AIDP	
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 AIDP[7]; 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 inmunological 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]

M GARCÍA GASALLA
M YEBRA BANGO
J A VARGAS NÚÑEZ
S MELLOR PITA
M GARCÍA ALVARADO
A DE ABAJO ALONSO
Department of Internal Medicine, Clínica Puerta de Hierro, Madrid, Spain

G GOIZUETA
Department of Neurophysiology

Correspondence to: Dr M Yebra Bango, Servicio de Medicina Interna I, Clínica Puerta de Hierro, C/ San Martín de Porres No 4, 28035 Madrid, Spain

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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 mellites 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

Table 1 Patient characteristics

	Placebo	Steroids
Patient numbers	18	18
Age, years (mean (SD))	44.9 (10.0)	42.9 (7.2)
Sex (female/male)	17/1	17/1
Baseline GSS*, (median, IR*)	24 (16–26)	20 (18–26)
CMAP amplitude, mV (mean (SD))	12.95(3.40)	14.49 (3.01)
Distal motor latencies, ms (mean (SD))	5.08(1.20)	4.97 (0.84)
Location of CTS*		
Right	10	9
Left	2	3
Bilateral	6	6

*GSS = global synptom score; IR = interquartile range; CTS = carpal tunnel syndrome.

the response rate in the oral steroid group to be 80% and that of the placebo group 30%.

Thirty six patients were recruited, of whom half were randomly allocated to receive oral steroids and half to placebo. There was no significant difference in demographics such as age and in the severity of electrophysiological parameters as shown in table 1. As compared with baseline, patients receiving steroid had a median (IR) change of −12.5 (−15 to −7) at two weeks, whereas the placebo group was −4.5 (−14 to 0), p=0.027 as shown in fig 1A . After eight weeks, the median (IR) reduction of GSS in the steroid group was −9 (−14 to −6) and in the placebo group −2 (−10 to 0), p=0.034 as shown in fig 1B. The median differences between the two groups at two and eight weeks were −6 (−11 to −1) and –6 (−11 to 0) respectively. All patients completed the short course of treatment.

This study shows a small but statistically significant reduction in GSS in the group prescribed a short course of prednisolone as compared with placebo. Steroid may have a role in the treatment of mild to moderate CTS in patients who decline or who are awaiting surgical decompression. Further trials with larger sample size and longer follow up, using low dose oral steroid in direct comparison with injected steroid, would further clarify the effect of this treatment.

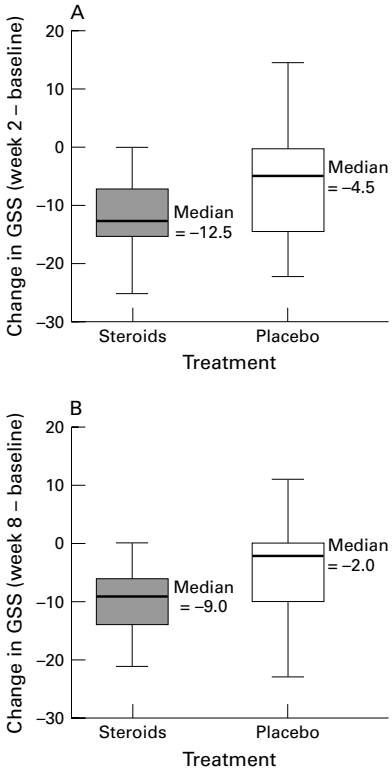

Figure 1 Box plots of changes in global symptom score (GSS) at (A) two weeks and (B) eight weeks.

A C F HUI
S M WONG
K S WONG
E LI
R KAY
Department of Medicine

P YUNG
L K HUNG
Department of Orthopaedics, Prince of Wales Hospital, The Chinese University of Hong Kong

L M YU
Centre for Clinical Trials and Epidemiological Research, The Chinese University of Hong Kong

Correspondence to: Dr S M Wong, Department of Medicine, Prince of Wales Hospital, 30–32 Ngan Shing Street, Shatin, New Territories, Hong Kong, Special Administrative Region China

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Parvovirus B19 infection in Behçet's disease

We read with great interest the article by Kerr which reviewed present knowledge about the possible association of parvovirus B19 infection with various connective tissue and autoimmune disorders.[1] The author concluded that data implicating B19 virus infection in the aetiopathogenesis of rheumatic diseases are insufficient and conflicting. Although a significant number of studies support a possible role for the virus in the pathogenesis of rheumatoid arthritis, juvenile idiopathic arthritis, systemic lupus erythematosus, and vasculitis, the author believes that B19 infection is only one of a number of triggers.

Behçet's disease (BD) is a multisystem disorder originally described by the Turkish dermatologist Hulusi Behçet.[2] Although its cause is unknown, vasculitis is widely accepted as the underlying pathological process.[3] As stated by the author, various case reports have been published demonstrating the presence of B19 virus in patients with vasculitic syndromes.[4 5] Viral infections have also been postulated as triggering factors in BD. Therefore, in a previous study that was not cited in the report by Kerr, we investigated a possible role of B19 virus in BD.[6] We assessed antibodies against parvovirus B19 in serum samples from 41 patients with BD and from 40 age and sex matched controls. Six patients with BD (15%) had anti-B19 IgM antibodies while no IgM antibodies were detected in the control group (p=0.03). However, anti-B19 IgG antibodies were present in 23 patients with BD and 25 controls. There was also no correlation between the presence of anti-B19 IgM antibodies and articular and vascular manifestations of BD (p=0.9 and p=0.5, respectively). Therefore, we concluded that our findings did not strongly support the involvement of B19 in the pathogenesis of BD, and we also concluded that serological evidence of acute B19 infection in six patients with BD might have been coincidental. However, the presence of anti-B19 IgM antibodies in patients with BD might provide evidence for the place of B19 infection in the pathobiology of BD. As far as we know our previous report is the only published study investigating the association of B19 virus infection and BD. Therefore, further studies will be clearly needed to clarify this unresolved issue.

S KIRAZ
M A ÖZTÜRK
I ERTENLI
M ÇALGÜNERI
Hacettepe University School of Medicine, Department of Rheumatology, Ankara, Turkey

Correspondence to: Dr M A Öztürk, Ostim mahallesi 89. sokak AK-84 sitesi A-2 blok no: 8, TR-06170, Yenimahalle, Ankara-Turkey
makifozturk@yahoo.com

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