T-lymphocyte abnormalities [5], and their subsequent clinical pictures and profound disability.

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Accepted 11 October 1996
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Re: Three Year Follow-up of a Case of Giant Cell Arteritis Presenting with a Chronic Cough and Upper Limb Ischaemic Symptoms
Sir—The association of giant cell arteritis and pulmonary disease, exemplified by a recent case report [1], has its corollary in the rare association of allergic asthma and cryptogenic fibrosing alveolitis (CFA) [2] in a patient with a previous history of polymyalgia rheumatica (PMR). At the age of 60, this patient had a clinical diagnosis of PMR characterized by weight loss and a lumbosacral distribution of myalgia [3], and concurrent investigations (with reference ranges) showing an erythrocyte sedimentation rate of 95–124 mm/h (Westergren), positive test for C-reactive protein, serum alkaline phosphatase 160–212 IU/l (25–125), γ-glutamyl transerase 106–248 IU/l (0–45), and an elevated serum immunoglobulin G level of 314 IU/ml (80–220). Rheumatoid factor was absent, but the lupus erythematosus test was weakly positive. His symptoms and hepatic biochemical derangements resolved (with the exception of immunoglobulin G, which remained elevated, and immunoglobulins A and M, which later increased, despite the absence of alcohol abuse), and the ESR fell, after treatment with corticosteroids, which were discontinued after 12 months. At the age of 73, he presented with acute asthma, characterized by sputum eosinophilia, and a striking improvement in peak expiratory flow rate (from zero to 380 l/min) after corticosteroids and bronchodilators. Although he had marked finger clubbing, his chest X-ray showed no focal abnormality. Two years later, however, bilateral basal mottling was noted on chest radiography, and computerized axial tomography displayed the typical peripheral crescentic pattern of honeycomb shadowing attributable to CFA. As expected, lung function tests showed an obstructive profile, compatible with obstructive airways disease, and an impairment of pulmonary diffusing capacity consistent with interstitial lung disease. The lupus erythematosus test was again weakly positive, but the relevant tests were negative for antinuclear factor and rheumatoid factor. His histocompatibility status was HLA B8.

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Accepted 5 September 1996


Ehlers–Danlos Support Group
Sir—I was pleased to see Dr D. L. Scott’s review of our booklets in British Journal of Rheumatology 1996;35:913–4.

In his review, Dr Scott has suggested that a ‘telephone helpline would be useful for patients to ask difficult questions when necessary’. I am sure your members would be interested to know that such a helpline does exist as anyone phoning the support group phone number (01748 823867) will be given all the help they require.

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Accepted 17 September 1996

Hypersensitivity Vasculitis Related to Aceclofenac
Sir—Hypersensitivity vasculitis (HV), also referred to as leucocytoclastic vasculitis, is thought to be an immunopathogenic disease resulting from the deposition of immunocomplexes, mainly in the small vessels. The precipitating antigen can be endogenous (in the context of connective tissue disorders or other chronic diseases) or exogenous (drugs or infections) [1]. The proportion of idiopathic cases ranges from 20 to 60% [2]. In its most benign form, the skin is the most commonly involved organ, mainly as a palpable purpura, usually in the lower limbs. However, any organ can be affected. Drugs have been implicated as the only possible precipitating factor in 12–18% of two series of non-selected consecutive cases, and as a cofactor in an additional 16–22% [2, 3]. Antibiotics and non-steroidal anti-inflammatory drugs (NSAIDs) are among the drugs most commonly associated with HV [2, 3].

Aceclofenac, a new NSAID marketed in Spain and Portugal since 1992, which has been submitted for approval in most European countries, is a phenylacetic acid derivative. As far as we know, only three reports of HV associated with the use of this new NSAID have been published [4–6]. We report on five additional cases of HV in patients treated
Cases of hypersensitivity vasculitis associated with aceclofenac

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age/sex</th>
<th>Indication for use of aceclofenac</th>
<th>Daily dose (mg)</th>
<th>Induction period (days)</th>
<th>Recovery period (days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1*</td>
<td>66/F</td>
<td>Sciatica</td>
<td>200</td>
<td>50</td>
<td>3</td>
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<tr>
<td>2†</td>
<td>67/F</td>
<td>Polymyalgia rheumatica</td>
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<td>11</td>
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<tr>
<td>3‡</td>
<td>74/F</td>
<td>Osteoarthritis of the knee</td>
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<tr>
<td>4</td>
<td>67/F</td>
<td>Osteoarthritis of the knee</td>
<td>100</td>
<td>12</td>
<td>18</td>
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<tr>
<td>5</td>
<td>50/F</td>
<td>Low back pain</td>
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<td>3</td>
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<tr>
<td>6‡</td>
<td>72/M</td>
<td>Humeral fracture</td>
<td>200</td>
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<td>28</td>
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<tr>
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<td>68/F</td>
<td>Minor trauma of the ankle</td>
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<td>3</td>
</tr>
<tr>
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<td>52/M</td>
<td>Ankylosing spondylitis</td>
<td>200</td>
<td>5</td>
<td>10</td>
</tr>
</tbody>
</table>

F, female; M, male.
*The patient had also taken Nervobion® (a fixed-dose combination of cyanocobalamin + carboxylase + pyridoxal-5-phosphate).
†Diagnosis confirmed by cutaneous biopsy.
‡Positive rechallenge.

Case 6 was published by Gómez Rodríguez et al. [4]. The patient also presented with microscopic haematuria.
Case 7 was published by Epelde and Boada [5]. The patient also presented with haemoptysis.
Case 8 was published by Núñez et al. [6]. The patient also presented with microscopic haematuria.

with aceclofenac which have been reported to the Catalan Centre of the Spanish System of Pharmacovigilance.

Between 1 and 50 days after starting treatment with 100–200 mg/day of aceclofenac, palpable purpura developed in all patients (Table I). All patients recovered completely 3–18 days after withdrawing aceclofenac. Except for one patient (patient no. 1), who had also taken a fixed-dose combination of cyanocobalamin + carboxylase + pyridoxal-5-phosphate 1 month before purpura developed, other known causes of HV had been ruled out. Patient no. 2 had polymyalgia rheumatica and although this syndrome is not a connective disease related to HV, its possible predisposing role cannot be completely ruled out; this patient had biopsy-proven leucocytoclastic vasculitis. No patient had systemic involvement and, except for case no. 2, diagnoses were made by clinical inspection of the cutaneous lesions. The positive rechallenge experienced by patient no. 3 strengthens the causal association between aceclofenac and HV.

Although HV is commonly a benign disease, in some instances its outcome may be harsh. Alclofenac and fenclclofenac, two NSAIDs structurally similar to aceclofenac, were withdrawn in the late 1970s and 1980s because of severe cutaneous adverse reactions, including several cases of generalized vasculitis associated with alclofenac [7]. Unfortunately, our data do not allow estimation of the risk of this previously undescribed adverse effect of aceclofenac.

We thank the reporting physicians. This work was supported by Servei Català de la Salut.

Re: Synovitis Associated with an Electrical Injury

Sir—Electrical injuries may be due to several mechanisms: (1) direct contact; (2) arcing of electricity; (3) exposure to the intense heat of an arc flash; (4) fires ignited by the heat; (5) mechanical injuries associated with the electrical accident. Musculoskeletal complications are frequently encountered and are most commonly fractures due either to falls or sustained tetanic contractions [1]. Acute synovitis is not a recognized complication of electrical injury. I wish to report a case of acute synovitis of the wrist and hand following a domestic electrical injury.

A 72-yr-old Asian man developed acute painful swelling of the right wrist associated with stiffness and erythema. The symptoms came on 3 h after touching a live domestic electrical wire (240 V, AC) with the right hand. Three weeks later, he developed increasing pain and stiffness of the second and third digits of the right hand. Tenoxicam 20 mg/day was commenced with slight improvement of wrist swelling and pain.

Six weeks after the electrical injury, his right hand showed diffuse swelling of the dorsum of the hand as well as firm swelling of the second and third fingers. Mild, tender synovial swelling was evident of the second and third PIP and DIP joints, all MCP joints and the wrist. Only 5° flexion and extension were possible in the wrist. A blue–brown discolouration of the skin was present, overlying the dorsal and ventral surfaces of the wrist with desquamation of the wrist, fingers and thumb. The left hand and wrist were normal.

An X-ray of the right hand demonstrated carpal osteopenia and soft-tissue swelling about the wrist and second and third fingers. There was no chondrocalcinosis. The ESR, 43 mm/h 2 weeks earlier,