TABLE I

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>
</tr>
<tr>
<td>2†</td>
<td>67/F</td>
<td>Polymyalgia rheumatica</td>
<td>200</td>
<td>18</td>
<td>11</td>
</tr>
<tr>
<td>3‡</td>
<td>74/F</td>
<td>Osteoarthritis of the knee</td>
<td>200</td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td>4</td>
<td>67/F</td>
<td>Osteoarthritis of the knee</td>
<td>200</td>
<td>12</td>
<td>18</td>
</tr>
<tr>
<td>5</td>
<td>50/F</td>
<td>Low back pain</td>
<td>100</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>6‡</td>
<td>72/M</td>
<td>Humeral fracture</td>
<td>200</td>
<td>1</td>
<td>28</td>
</tr>
<tr>
<td>7‡</td>
<td>68/F</td>
<td>Minor trauma of the ankle</td>
<td>200</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>8‡</td>
<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-drug 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.

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

R. Morros, A. FIGUERAS, D. CAPELLÀ, J.-R. LAPORTE
Institut Català de Farmacologia, Universitat Autònoma de Barcelona, Servei de Farmacologia Clínica, CSU Vall d’Hebron, 08035-Barcelona, Spain
Accepted 27 September 1996

Correspondence to: J.-R. Laporte, Institut Català de Farmacologia, Servei de Farmacologia Clínica, CSU Vall d’Hebron, 08035-Barcelona, Spain.


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 discoloration 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,
had fallen to 22 mm/h and CRP was <10 mg/ml. He was seronegative for rheumatoid factor and ANA was negative. A technetium bone scan demonstrated increased uptake of tracer in the right wrist, carpus, MCP and PIP consistent with synovitis.

The right wrist joint was injected with 40 mg of triamcinolone acetonide. No synovial fluid could be aspirated from the joint. There was rapid resolution of the symptoms in the right wrist over 48 h. The range of movement of the fingers improved slightly over the first 48 h and with continued active physical therapy the right hand and wrist were normal after 12 weeks.

This case describes synovitis of the wrist and small joints of a hand following a low-voltage electrical injury. The synovitis was evident clinically and supported by an elevated ESR, the presence of osteopenia on radiographs and bone scintigraphy. The synovitis was associated with cutaneous changes consistent with a resolving thermal injury. The cutaneous features may also be compatible with resolving cellulitis; however, there was no evidence of infection and the patient did not receive antibiotic therapy. Current markings were not present. The synovitis was minimally responsive to non-steroidal anti-inflammatory drug therapy. Intra-articular corticosteroid therapy resulted in rapid clinical improvement. Within 3 months, the synovitis and cutaneous changes had completely resolved.

The cause of the synovitis in this case is unclear. In a review of 182 cases of electrical injury, five cases of acute gout were recorded [1]. It is possible that the synovitis in the case I have described was crystal induced; however, the patient had had no previous episodes of acute synovitis, there were no features of a chronic arthropathy, the synovitis involved the majority of the joints of the hand and wrist, and there were no gouty erosions or chondrocalcinosis on X-ray. Reflex sympathetic dystrophy has been described following low-voltage injuries; however, in this case there were no clinical features of reflex sympathetic dystrophy [2]. Direct electrical injury to the joints is unlikely to have occurred in a low-voltage injury. The low resistance of neural tissue makes it prone to electrical injury. It is possible that neural electrical damage resulted in the release of substance P and subsequent joint inflammation as a result of prostaglandin E2 and collagenase release, and synoviocyte proliferation.

Trauma to a joint, usually in the form of a direct blow or as a result of forced inappropriate motion, may be followed by an acute synovitis [3]. It is possible that forceful muscle contractions at the time of the electrical insult resulted in direct joint trauma. However, the development of finger involvement was delayed by 3 weeks and suggests that this was not responsible. Trauma has also been reported to trigger the development of seronegative spondyloarthropathies and psoriatic arthritis, and the time course of small hand joint involvement is consistent with this [3]. However, only the hand affected by the electrical injury was involved and the synovitis was self-limiting. A ‘deep’ Koebner phenomenon has been suggested as an explanation for the development of psoriatic arthritis after trauma, as has the release of self-antigens from injured joints in the genesis of reactive arthritis [4].

I believe that this is the first case to be described of an acute inflammatory arthropathy occurring as a direct result of an electrical injury. Awareness of this possible complication of electrical injuries may result in its more frequent detection.

A. L. TAYLOR
Department of Rheumatic Diseases, Royal Perth Rehabilitation Hospital, 6 Selby Street, Shenton Park 6008, Western Australia, Australia
Accepted 6 September 1996


Has Hepatitis C Virus a Specific Tropism for the Synovial Membrane?

Sr—Arthritis is not included among the extrahepatic manifestations of hepatitis C virus (HCV) infection [1]. However, three anecdotal reports of polyarthritides associated with HCV infection have recently been reported [2, 3]. Two out of these patients had chronic HCV infection and a rheumatoid arthritis (RA)-like disease; HCV RNA was detected not only in the serum, but also in the synovial fluid (SF). The prevalence of antibodies to HCV is increased in patients with RA [4], a finding that may be related to the infection risk inherent in the number of medical procedures performed on them. However, in the absence of population-based studies, the possibility of a link between HCV infection and RA cannot be discarded. This hypothesis would be supported by a specific tropism of HCV for the synovial membrane (SM). As a first step, we have evaluated HCV RNA in both serum and SF of patients with RA and concomitant HCV infection.

Four RA patients [5] with concomitant HCV infection were studied (Table I). All the patients had antibodies to HCV by ELISA and Western blotting in the serum, but none of them had elevation of alanine or aspartate transferases. Sera and SF effusions were tested on a mean of three different occasions (range 1–5) 1 month apart for HCV nucleic acids. Qualitative analysis of HCV RNA was performed with a nested polymerase chain reaction (PCR) with four primers complementary to the highly conserved 5′ non-coding region of the HCV genome. Quantitative analysis of HCV RNA was performed using the branched-DNA signal amplification assay (Quantiplex, Chiron Corp.,

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**References**