A labourer in his forties complained of pain and dusky discolouration over his ankles and feet for 6 weeks before admission to hospital. He had also developed patchy and asymmetric numbness and weakness in all four limbs, and a rash on his legs [Fig. 1] accompanied by digital ischaemic lesions affecting his fingers and toes. He used to be an intravenous drug user. Following a prolonged period of drug abstinence, 2 weeks before the onset of his symptoms, he had sniffed cocaine. Neurological examination revealed ulnar and common peroneal palsies [Figs 1 and 2] suggesting mononeuritis multiplex, and nerve conduction studies [Fig. 3] confirmed a very severe active axonal sensory and motor polyneuropathy with multifocal features. The distribution of nerve involvement was non-confluent, and electromyography showed severe active denervation, in keeping with a vasculitic neuropathy.

Skin biopsy showed acute leukocytoclastic vasculitis involving small vessels in the papillary dermis [Fig. 4]. Blood tests revealed cryoglobulinaemia and positive hepatitis C serology that was also demonstrated with PCR. Liver enzymes and other routine blood tests were normal. A diagnosis of acute vasculitic neuropathy complicating type II (mixed) cryoglobulinaemia secondary to chronic hepatitis C (HCV) infection was made.

HCV is spread predominantly by the parenteral route. At highest risk are intravenous drug users and those with multiple parenteral exposures to body fluids (e.g. blood transfusion). Sexual transmission is rare. Other potential sources of HCV are needle stick accidents, and sharing straws during intranasal cocaine use, which made us wonder whether the recent cocaine use, a well-reported risk factor for neurovasculitis (Neiman et al. 2000), had triggered the vasculitic pathology in conjunction with the cryoglobulins.

Around one-half of patients with chronic HCV infection have detectable cryoglobulins that are associated with organ involvement in about one quarter of cases. Cutaneous manifestations and arthralgia are usual, with glomerulonephritis and multiple mononeuropathies less common, the latter often evolving in a stepwise
and asymmetric fashion, and commonly including both wrist drop and foot drop, as in this case (Apartis et al. 1996; Gemignani et al. 2002).

The patient was treated with intravenous, then oral methylprednisolone, tinzaparin and plasma exchange. He had a liver biopsy, which showed liver fibrosis, and was then started on antiviral therapy for HCV. Improvement of his neuropathic deficit has been slow and only partial, but the digital ischaemia has fortunately recovered without requiring surgery and the cryoglobulins are currently undetectable.

REFERENCES


Figure 3 (a) Abnormal ulnar motor nerve study showing a very small compound motor action potential (abductor digiti minimi). (b) Normal nerve conduction study for comparison. (Courtesy of Dr Aline Russell, Department of Neurophysiology, Institute of Neurological Sciences, Glasgow, 2003.)

Figure 4 Skin biopsy from the vasculitic lesions on the legs. Arrows show polymorphonuclear cells infiltrating the capillary wall. (H&E stain, courtesy of Dr Richard Morton, Department of Pathology, Southern General Hospital, Glasgow, 2002.)