Cutaneous leucocytoclastic vasculitis caused by cyclosporin A (Sandimmun)

Cutaneous leucocytoclastic vasculitis to cyclosporin A (Sandimmun) has not been previously described. We report the case of a 37 year old man who presented with a 10 day history of a purpuric eruption affecting both legs. He had been diagnosed as having psoriasis and psoriatic arthropathy 12 years previously, on the basis of joint symptoms, sacroiliitis, erosive arthropathy, increased acute phase reactants and positive HLA B27 status. His initial treatments had included oral gold (Auranofin) 3 mg three times per day and salazopyrine 3 g/day. Both were discontinued because of poor control of his joint disease. In July 1997 he was given oral cyclosporin A (Neoral) 50 mg/day, which was increased to 100 mg/day. This was discontinued in January 1998 because of symptoms of fatigue. In April 1998, after further flares of his arthropathy, he was given Sandimmun 50 mg/day by his general practitioner. Within one month of this he developed an acute skin eruption. He was otherwise well and receiving no other medication except indomethacin 150 mg/day, which he had been taking since 1986.

On examination, there was a palpable, non-tender, purpuric eruption with necrosis of the base of Sandimmun rather than the active drug. Routine haematology, biochemistry and urine analysis were normal. Skin biopsy showed a leucocytoclastic vasculitis with heavy dermal neutrophilic infiltrate and few eosinophils (fig 1). Gram stain for organisms was negative.

The Sandimmun was discontinued and routine haematology, biochemistry and urine analysis were normal. Skin biopsy showed a leucocytoclastic vasculitis with heavy dermal neutrophilic infiltrate and few eosinophils (fig 1). Gram stain for organisms was negative. At nine month follow up, the patient was not taking cyclosporin A with no recurrence of his vasculitis.

Leucocytoclastic vasculitis may be secondary to a variety of drugs or infections. Although numerous cutaneous eruptions have been reported with Sandimmun, there is only one report of it causing a vasculitic-type of rash. This report reviewed the cutaneous findings in 67 patients treated with cyclosporin A. Three patients (4.4%) were found to have Bateman’s or senile purpura, although skin biopsies were not performed. Our patient developed a cutaneous leucocytoclastic vasculitis to Sandimmun but not to Neoral. Apart from the active drug, Sandimmun has corn oil and polyoxyethylated glycolysed glycerides as the base, while Neoral has corn oil and a polyoxyl 40 hydrogenated castor oil.

It is possible that the leucocytoclastic vasculitis seen in our patient occurred by chance. However, as a reaction to the base of Neoral has been reported previously and because of the temporal relation between the use of the drug and the onset of the skin eruption, we postulate that the vasculitis was more likely to be attributable to the constituents of the base of Sandimmun rather than the active drug.

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Figure 1  Histological examination showing endothelial cell swelling, thrombosed dermal vessels, dermal neutrophilic infiltrate with scanty eosinophils, nuclear dust and red cell extravasation consistent with leucocytoclastic vasculitis (haematoyxl and eosin stain × 250).

An evidence based EULAR meeting?

One of the main themes of the very successful EULAR Meeting in Glasgow was “Evidence Based Rheumatology”. Also prominent at the meeting were pharmaceutical companies promoting COX 2 inhibitors. Much of this promotional activity, some of which was supported by academic speakers, was for the use of these agents in osteoarthritis. However, the evidence base does not support the use of anti-inflammatory agents in osteoarthritis and current guidelines on the management of this disease suggest the use of simple analgesics rather than non-steroidal anti-inflammatory drugs (NSAIDs). Some years ago, we showed that publications on NSAIDs were dominated by comparative studies of different NSAIDs, rather than more necessary studies, such as comparisons between NSAIDs and simple analgesics; sadly, the appearance of a new class of NSAIDs does not seem to have led to any change in the type of trial being sponsored by the industry. How can we justify holding a large international meeting with an evidence based theme, while including a large amount of information that runs counter to the available evidence?

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MATTERS ARISING

Psychosocial risks for low back pain: are these related to work?

In an interesting prospective study, Papa-georgiou et al reported that in a low back pain free population dissatisfaction with work status doubled the risk of reporting a new low back pain episode in the employed and non-employed. This is an interesting finding that adds to the literature on the importance of worker job perceptions to
the development of low back pain episodes. In this report, however, Papageorgiou et al neglected to discuss previous research on the importance of pre-injury job perceptions to chronic low back pain patients’ intent to return to work after treatment.

In a series of four papers, Fishbain and colleagues have attempted to determine if pre-injury job satisfaction impacts on “intent” to return to work to the pre-injury job after pain facility treatment. In the first report Fishbain et al demonstrated that chronic pain patients not intending to return to work after pain facility treatment were more likely to complain of job dissatisfaction. In the second report from this group, Rosomoff et al demonstrated that an association between non-intent to return to work after pain facility treatment and pre-injury job dissatisfaction was similarly found across Workers’ Compensation and non-Workers’ Compensation chronic pain patients. In the third report, Fishbain et al looked at actual return to work after pain facility treatment in relation to these variables. They found that actual return to work was predicted at one month “by intent”, perceived job stress and job like (job dissatisfaction) plus other variables. At 36 months, return to work was predicted by “intent” and by perceived job stress plus other variables. In the final study, Fishbain et al attempted to predict “intent” to return after pain facility treatment in relation to actual return to work. “Intent” was predicted by perceived pre-injury job stress plus other variables. In addition, those chronic pain patients who intended to return and did not were predicted by whether there was a job to go back to. Also chronic pain patients not intending to go back to work at the pre-injury job, but doing so, were predicted by having a job to go back. Overall, this series of studies points to a strong relation between pre-injury work variables such as job dissatisfaction and “intent” to return to that job after treatment. In addition, these studies indirectly support the findings of Papageorgiou et al. It seems that in trying to understand the low back pain injury and recovery process, it is important to take into account work related perceptions such as those of perceived job dissatisfaction and job stress.

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