LETTER TO THE EDITOR

Aspirin in the Management of Necrobiosis lipoidica

We were interested to read the results of the effect of aspirin and dipyridamole in necrobiosis lipoidica recently published by Statham et al. (7). The previously published reports of the effectiveness of aspirin in this condition (2, 3, 8), together with evidence for depressed endothelial cell prostacyclin production (1) and increased platelet aggregation in some cases of diabetes (4) also prompted us to try aspirin therapy. However, we decided to employ a low-dose aspirin regime.

Aspirin will reduce both thromboxane A_2 and prostacyclin production by inhibiting platelet and endothelial cell cyclo-oxygenase respectively. Prostacyclin reduces platelet aggregation and enhances vasodilation and these effects will be antagonized by aspirin LE, there may not be any beneficial effect on the prostacyclin/thromboxane balance. This may explain the failure of Statham et al. to obtain any significant clinical response. Recent evidence (6) has suggested that there may be a difference in the sensitivity of platelet and vessel wall cyclo-oxygenase to aspirin, and that a lower dose given less frequently may well have a beneficial effect on the prostacyclin/thromboxane balance.

In our series of patients we used the regime of Masotti et al. (6) (of 3.5 mg/kg of aspirin given initially at 48-hourly intervals and later in some patients at 72-hourly intervals) in 7 patients with necrobiosis lipoidica which had been present for 6 months to 6 years prior to commencement of this treatment. All were women aged between 21 and 79. Four were diabetic and all had typical necrobiosis lipoidica on the lower legs; 2 had had ulcerated lesions for 4 months and 2 years.

Three of the patients were judged to have made a good clinical response and 3 a reasonable one. Improvement was noted in all cases within 4 months of the start of treatment and in some was noted as early as one month. Interestingly, both patients with ulcerated lesions healed, in 2 months and 6 months respectively. There was no obvious relationship between clinical response and the presence or absence of diabetes mellitus. We studied platelet aggregation in one patient and found a reduction in platelet function after one month's therapy. We would agree with Statham et al. that uncontrolled, anecdotal reports such as this need to be treated with caution but we feel that there has been a sufficiently good response in our patients, with a condition that is usually very persistent, to warrant further trials. Work by Hanley et al. (5) has suggested that even lower individual doses of aspirin, e.g. 40 mg, may be more beneficial on the prostacyclin/thromboxane balance, and that perhaps our regime was not the most advantageous.

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