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# Skin Blood Flow in Necrobiosis Lipoidica during Treatment with Low-dose Acetylsalicylic Acid

HANS-IVER BECK and PETER BJERRING

Department of Dermatology and Venerology, Marselisborg Hospital, Arhus, Denmark

Beck H-I, Bjerring P. Skin blood flow in necrobiosis lipoidica during treatment with low-dose acetylsalicylic acid. Acta Derm Venereol (Stockh) 1988; 68: 364–365.

Skin blood flow was measured by the laser Doppler technique in lesional and clinically normal skin of 10 diabetic patients with necrobiosis lipoidica during and after treatment with 40 mg acetylsalicylic acid (ASA) daily. The measurements showed that the blood flow during ASA treatment was significantly decreased in the central lesional skin without changes in the peripheral part of the lesions and normal skin. In view of these findings we suggest that low-dose ASA may not be the best treatment for necrobiosis lipoidica. Key words: Laser Doppler flowmetry. (Received January 28, 1988.)

H.-I. Beck, The Department of Dermatology and Venereology, Marselisborg Hospital, DK-8000 Århus C, Denmark.

In a previous study (1) concerning treatment of necrobiosis lipoidica (NL) with 40 mg acetylsalicylic acid (ASA) daily we found that the lesions became significantly larger in spite of inhibited aggregation of the platelets. In this context we measured skin blood flow (SBF) in the NL lesions.

## MATERIAL AND METHODS

Ten diabetic patients (9 female, 1 male) with NL on the front of the lower legs were examined. All patients, mean age 36 (18–54), had been treated with ASA 40 mg daily for 3–28 months (mean 16). No other ASA-containing drugs or topical steroids were allowed. An inhibition of the aggregation of the platelets was found in 7 patients during the ASA treatment (1); 3 were not tested. SBF was measured by means of a laser Doppler flowmeter (Periflux, Perimed, Sweden) (2).

The patients were examined after 30 min of rest with the legs horizontal at constant room temperature (21–24°C). In each patient, SBF was measured in the central and peripheral part of the NL lesion. For

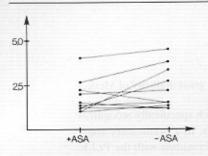


Fig. 1. Skin blood flow in the central part of atrophic necrobiosis lipoidica skin during (+) and after (-) treatment with ASA (Wilcoxon matched-pair test, p<0.02). ASA: Acetylsalicylic acid.

control purposes, two sites of clinically normal skin were selected at lateral sites on both legs. SBF was measured during the ASA treatment and 3 months after the treatment had been stopped. For each skin site the SBF was as the mean value of three measurements.

Wilcoxon's test was used for statistical evaluation. A p>0.05 was regarded as non-significant.

#### RESULTS

The SBF values (Fig. 1) were significantly increased, from a mean value of 1.81 (1.0–2.7) to 2.33 (1.2–4.5) (arbitrary units) after the ASA treatment had been stopped (p<0.02). No significant change in the SBF was found in the peripheral part of NL (mean 1.6–1.7) and the clinically normal skin of both legs (mean 1.0–1.3).

#### DISCUSSION

Our results show reduced bloow flow in the NL lesions during low-dose ASA treatment, in spite of an inhibition of platelet aggregation.

ASA inhibits the cyclo-oxygenase pathway and might in this way eliminate a vasodilatating factor of the prostaglandins. A current shunting towards SRS-A leucotrienes (LTC-4, LTD-4) is also a possibility, as quantitative differences in the effects (vasoconstriction, vasodilatation) have been shown in guinea pig skin and hamster cheek pouch (3). A real vasoconstriction due to release of small amounts of SRS-A may be an explanation in the present cases.

The present data indicate that low-dose ASA may not be the best treatment for NL. This is in agreement with a previous clinical evaluation (1).

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