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SHORT REPORTS

Pressure Inhibition of the Koebner Reaction by Capillary Occlusion

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Abstract. A 38-year-old male with extensive psoriasis received a linear experimental abrasion. Immediate pressure was applied for 24 hours to the central portion of the traumatized site. During a psoriatic relapse 5 weeks later, Koebnerization occurred in the abraded areas but spared the site where pressure had been previously applied. The mechanism is not known but may be due to a local suppressive effect of pressure on inflammation.

CASE REPORT

A 38-year-old male was admitted to hospital with extensive active psoriasis unresponsive to topical therapy. He had had psoriasis for the last 8 years, initially involving only his scalp but subsequently becoming more extensive. Previous treatment with Methotrexate had not been feasible due to a history of alcoholism and PUVA therapy had been declined by the patient.

On admission to hospital he had extensive psoriasis affecting approximately 65% of his body. In many areas there was pustulation. He had psoriatic arthropathy affecting the small joints of his hands and feet and a severe onychodystrophy.

He commenced treatment with the antimetabolite Razoxane (500 mg twice weekly) (1) and showed considerable improvement during the next 6 weeks in hospital. During a period when his psoriasis was relatively quiescent, clinically uninvolved skin (Fig. 1) was scratched with a No. 19 needle four times in a linear fashion on the volar surface of his left forearm (Fig. 2). At the centre of the abrasion, a circular plastic cap was placed and attached to the arm with adhesive tape such that enough pressure was applied to restrict capillary blood flow to this area (Fig. 2). This was removed after 24 hours (Fig. 3). He was discharged from hospital 1 week later and followed on an out-patient basis. Five weeks after discharge his psoriasis again flared despite continued antimetabolite therapy. During this relapse psoriasis occurred in the scratched site but spared the central area where pressure had previously been applied (Fig. 4).

DISCUSSION

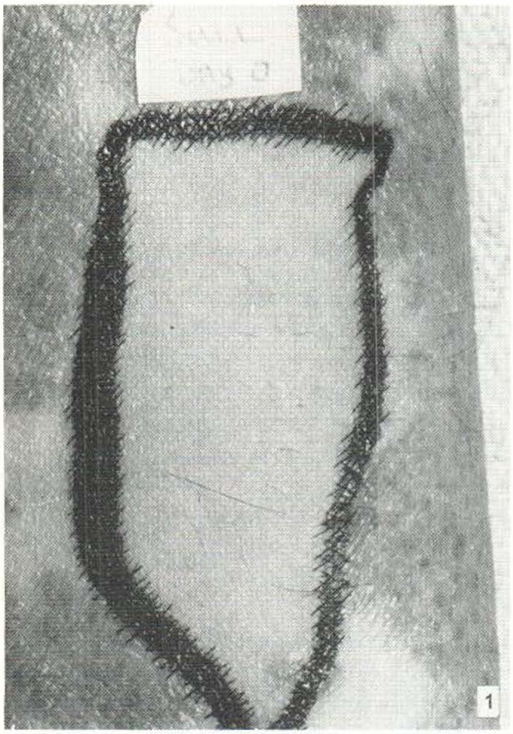
The Koebner phenomenon is a well known reaction seen in patients with active unstable psoriasis (5). It may occur following a wide variety of clinical and experimental stimuli which serve as precipitating factors in the predisposed individual.

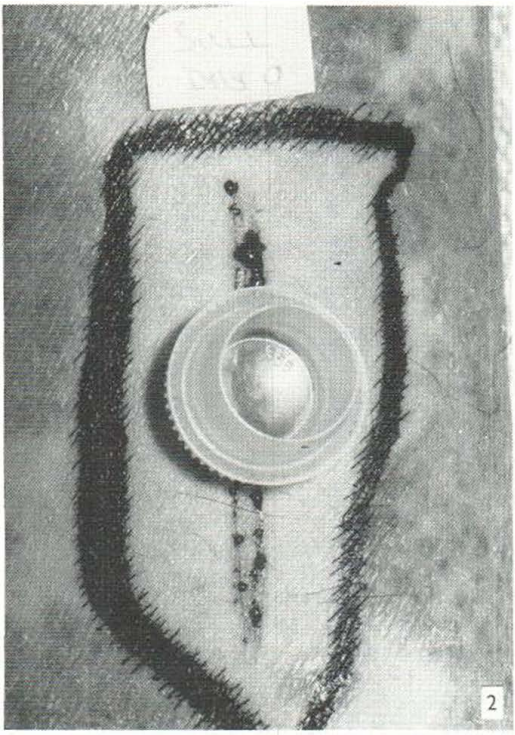
Studies have been made using various drugs and techniques in attempts to inhibit its development. It has been assumed that knowledge of these inhibitory factors might not only offer information to elucidate the pathogenesis but also dictate future therapeutic directions.

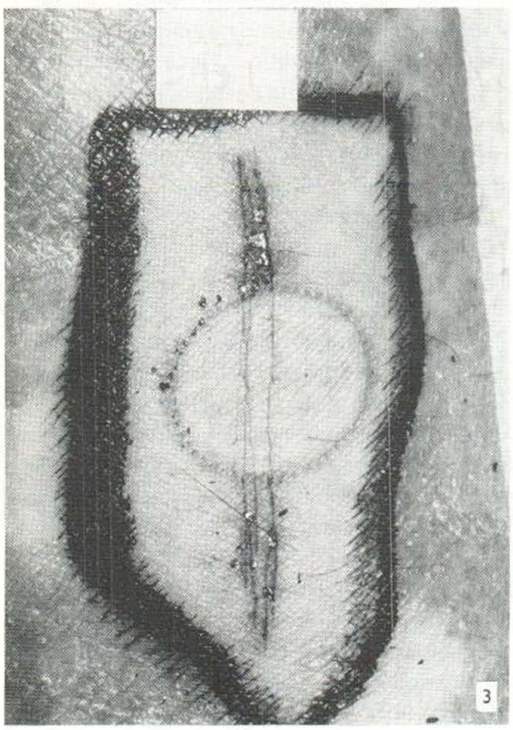
Injection of epinephrine has been shown to delay Koebnerization, possibly due to its vasoconstrictor action (3). White soft paraffin applied topically also has an inhibitory action (2) which may be related to the antimitotic effect of bland ointments (10).

Among physical modalities, Fleck (4) found that the Koebner reaction could be inhibited by pressure to the skin. With reference to this observation, he described a negative Koebner phenomenon in which psoriasis did not occur under an area of pressure from a wristwatch strap. Ryan (8) demonstrated that it was possible to prevent psoriasis from developing in a traumatized state by obliterating the vessels with external pressure during the first 24 hours after injury. Transient applications of heat or cold did not prevent or adversely affect the development of the Koebner reaction (6).

The mechanism by which pressure may inhibit the Koebner reaction is not known. The immediate biochemical and pathological events occurring after skin injury are illustrated in Fig. 5. Occlusion of capillary blood flow by pressure during the immediate 24 hours after injury may have a profound effect on the normal sequence of inflammation. Vascular blockage may not only impede distribution of inflammatory mediators from this site but also prevent influx of neutrophils, mast cells, and eosinophils. Normally the accumulation of neutrophils reaches a peak some 2–3 hours after injury and the majority are degenerate by 12–24 hours (9). The effect of impairment of vascular perfusion on







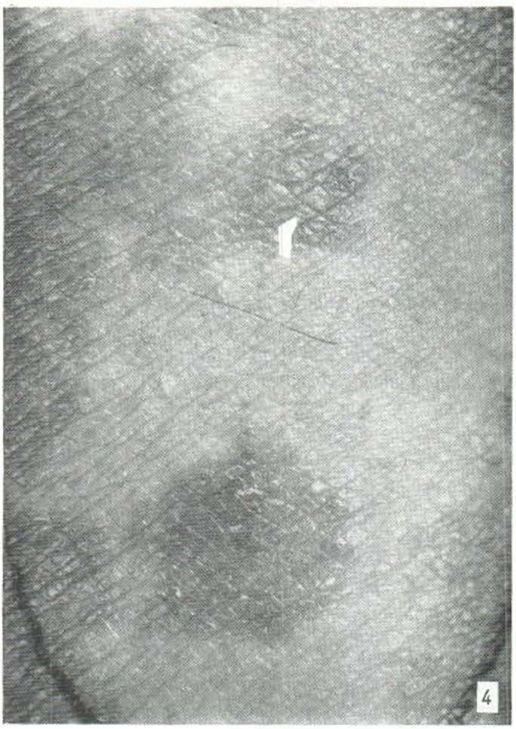


Fig. 1. Day 0. Clinically uninvolved skin of left forearm.

Fig. 2. Day 0. Immediately after needle abrasion. Pressure in process of application.

Fig. 3. Day 1. 24 hours later. Pressure cap has just been removed.

Fig. 4. Week 6. Psoriasis has occurred in abrasion sites but has spared central area where pressure had been applied.

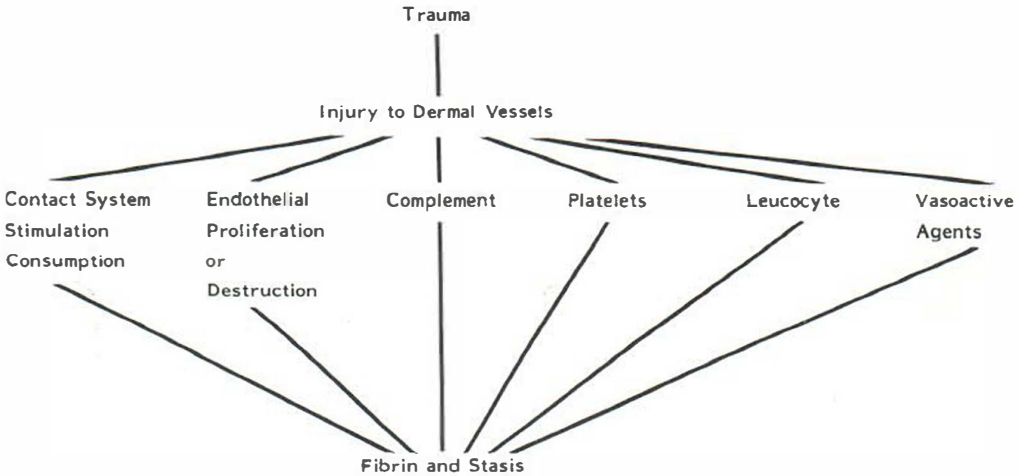


Fig. 5. Changes in microvascular system in response to injury (Ryan, 1976).

their early recruitment may affect their later performance once the vascular channels are re-opened.

The ultimate result of inflammation is fibrin deposition. In inflammatory events fibrin is found where the cellular necrosis is most intense. It attracts neutrophils and helps prolong the inflammatory response in addition to acting as a scaffold for vessel growth. Fibrin is, therefore, an intrinsic part of delayed or prolonged inflammation. Pressure may reduce the amount of fibrin formed and thus interfere with the normal inflammatory response.

As tissue injury—and hence inflammation—are a necessary prelude for Koebnerization, sites deprived of the full consequences of this stimulus may not manifest the potential of developing psoriasis.

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