HEMODIALYSIS IN PSORIASIS
(Preliminary communication)

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Summary

Hemodialysis has proved to be a welcome addition to the various already known therapeutic modalities in the treatment of psoriasis. The procedure was attempted in 12 patients with chronic plaque psoriasis recalcitrant to conventional treatment. There was a dramatic improvement in 10 of the 12 patients. The lesions started clearing as early as 2–4 days following first hemodialysis and most patients required 3–4 hemodialysis to be free of lesions. The remission could be maintained for as long as 9 months after the last dialysis. The possible mechanism of induction of remission due to hemodialysis is discussed.

Dialysis has proved to be a welcome addition to the various already known therapeutic modalities in the treatment of psoriasis. The interest was recently initiated following clinical observation of regression of psoriatic lesions in a patient with chronic renal failure on haemodialysis[1]. Subsequently a number of reports (2–4) poured in indicating the beneficial effect of both haemodialysis and peritoneal dialysis on psoriatic lesions. Even the arthritis associated with psoriasis has been reported to improve after dialysis[5]. Since our preliminary trial[6] showed encouraging therapeutic results with haemodialysis in 3 patients with psoriasis, we carried the procedure in further 9 patients. All patients were seen to have chronic plaque psoriasis throughout the year, recalcitrant to conventional tar treatment. Hemodialysis with Keil dialyser employing A-V shunt was carried out twice weekly for 6 hours each time. There was a dramatic subsidence of itching and erythema with gradual clearance of psoriatic lesions in 10 of the total 12 patients. The lesions started clearing with shedding of scales as early as 2–4 days following first hemodialysis and most patients required 3–4 hemodialysis to be free of lesions. The state of remission could be maintained for as long as 9 months after the last dialysis. One patient observed complete clearance for more than 6 months for the first time during the last 20 years of persisting lesions.

The therapeutic success in psoriasis has remained limited due to our poor understanding about its etiopathogenesis and the nature of underlying defect. The accelerated epidermopoiesis is still the most dominating pathogenic concept and almost all currently available effective remedies ranging from topical tar, corticosteroids, cytotoxic agents

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to PUVA are capable of inhibiting epidermal mitosis. Perhaps, what remains to be found is whether the increased epidermal mitosis is a primary event or it is secondary to some yet unknown pathogenic mechanism. Many schools of thought prevail and it is not yet established whether psoriasis is primarily a disease of the epidermis or the dermis. A working model is however required to build the arguements and on the existing information drawn from histological and immunological studies it appears pertinent to analyse the data.

The two possibilities are (1) that the basic fault is in the startum corneum which is antigenically altered in patients with psoriasis. This is supported by results of direct immunofluorescent studies on tissue sections from psoriatic lesions which reveal the presense of an IF band within the stratum corneum[8,9]. Consequently, the presence of stratum corneum antibodies 'SCAB' has been hypothesized[9]. These antibodies are demonstrable in the blood and permeate from time to time into the stratum corneum to initiate an antigen-antibody reaction and subsequent cascade of events resulting in the formation of 'munro-abscess'. This, in turn, initiates the epidermal proliferation at the basal zone resulting in the clinical lesion. The serum, however, fails to demonstrate abnormalities in the immunoglobulin levels even in the active stage of the disease[8]. (2) The fault lies in the dermis within or outside the vascular compartment. Either some epidermal factor is reabsorbed in the blood and alter the epidermopoiesis or a blood borne factor acts directly on the epidermal cells to initiate the formation of lesion involving immunological phenomena or otherwise.

The mechanism of induction of remission by dialysis in psoriasis is not known. Most psoriatic patients have normal renal functions and none of our patients studied had any evidence of renal failure. This rules out the possibility of clearing of lesions being attributable to the removal of a dialyzable toxin. We believe that dialysis eliminates from the blood a 'psoriatic factor' either derived from the epidermis or the dermis. This factor is either not excreted by the normal kidney or if it is excreted it is reabsorbed within the tubules. Hemodialysis is known to cause loss of factors from the plasma that are essential for DNA synthesis[10] and removal of such factors results in clinical regression of the psoriatic lesion.

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