ROLE OF INTRALESIONAL CORTICOSTEROID THERAPY IN PEMPHIGUS

By

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The usual procedure is to satisfactorly control the acute manifestations of the disease first with adequate dose of the systemic corticosteroid. Once this is achieved and marked improvement of the patient becomes evident, reduction in dosage is effected gradually taking care that the equilibrium established between the activity of the disease and the effective dose is not disturbed. This reduction is continued till one comes to the lowest effective maintenance dose. The aim of this maintenance dose is to permit the patient to carry on a fairly normal life free of symptoms as far as possible. However, this aim may not be achieved at times. We, at least, have run in difficulties in a few instances, two of which encountered recently are described below.

Mrs. S. Kaur, aged 26 years, was admitted in our ward in July, 1966 with history of blisters on various parts of the body of one month's duration. Diagnosis of pemphigus vulgaris of her illnes was established and treatment with prednisolone instituted. Her disease was controlled with 60 mg. prednisolone a day, which could subsequently be tailed off to 15 mg. a day. She was discharged on that dose on 10.10.66, and asked to continue taking it at home. She remained well for 1½ years, but then her condition relapsed. When she returned to us on 23.9 63, she presented characteristic clinical picture of pemphigus erythematosus. Her dose of steroid was again increased to 60 mg. a day. But it induced only a partial remission in her this time, and crusted lesions persisted on her face in butterfly distribution (Fig. 1). The patient complained of feeling severe stiffness in that region and was very much distressed. This impelled us to increae her prednisolone dose further to 80 mg. a day to control this refractory disabling lesion. This increase of the dose, however, had no beneficial effect. It, on the other hand, precipitated hypertension, severe diabetes, and marked osteoprosis of the spine. To counter these complications, we had no other alternative except to taper the oral dose, but we decided to supplement it with local corticosteroid intralesional infiltration. Three weekly infiltrations were done. These not only cleared the lesions on her face completely (Fig. 2), but also enabled us once again to bring her oral dose down to 20 mg. a day, rather quickly and to control her diabetes, hypertension, osteoprosis and depression more effectively.

Our second case, Mrs. M. Joshi, aged 41 years, developed lesions first on her buccal mucous membrane in July, 1968, and later scattered lesions on her trunk. Treatment in her was started with 4.0 mg. of dexamethasone on 9.9.68, but increased to 8 mg. on 25.9.68. As she started complaining of pain in abdomen

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after swallowing tablets, she was switched on to injectable instead of oral dexametoasone. Though there were no fresh lesions with this dose, it did not control her disease adequately. The pre-existing lesions were still spreading by slow peripheral extension, and most of them showed either irregular collar of loose soddened epidermis raised by subjacent fluid, or raw areas (Fig. 3). The dose was, therefore, further pushed up to 12 mg. and then to 16 mg., but even this big dose did not prove very effective. Instead of helping her, we found that we had induced in her too, severe diabetes and psychic symptoms. Encouraged by our experience with the previous case, we decided to infiltrate her lesions also in addition to oral corticosteroid therapy. The infiltration was done on 5.12.68. After infiltration, the lesions looked better the same evening, and were completely dry by 3rd day and appeared mildly erythematous and just imperceptibly scaly (Figs. 4 and 5). We were also able to decrease her dose and on 18.2.69 she was on 8 mg. injectable dexamethasone a day only without any recrudescence of the disease so far.

Ledercort suspension in one case and Wycort in the other were used. It was diluted 1:10 with a mixture of the equal quantity of isotonic saline and 2% procaine hydrochloride. Injections were made intradermally and about half to one inch apart. No more than 0.2 to 0.3 cc was injected at any one site. Maintenance of asepsis during injections was observed strictly and there was no secondary infection present in the lesions.

Having used this intra-lesional infiltration with corticosteroids in these two cases, I am feeling very gratified and heartened especially when I find that it is quickly effective. These encouraging results clearly suggest that though systemic corticosteroid may alone be adequate in most cases of pemphigus, intra-lesional infiltration is an effective supplement in management of some difficult cases at least as ours.

I am also feeling tempted to try it in lesions of mucous membrane which are well known to be refractory to moderate dose of systemic corticosteroid.

LEGENDS

Fig. 1 Showing crusted lesion on face in Case No. 1.

Fig. 2 Face of Case No. 1 after Intralesional corticosteroid.

Fig. 3 Showing active lesions on breast of Case No. 2.

Fig. 4 Breast lesions in case No. 2 after intra-lesional corticosteroid.

Fig. 5 Healed lesions on shoulder of Case No. 2 after intralesional corticosteroid.