

HERPES SIMPLEX: CICATRICAL TYPE

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Abstract. An example of a cicatricial form of recurrent herpes simplex is presented. Although the initial appearance of the lesions was identical with non-scarring forms of herpes simplex, this patient's lesions progressed insidiously to eschar formation. Healing was delayed for four to seven weeks, with subsequent atrophic scar formation. Scarring and local recurrence could be prevented by curettage, and acridine orange and ultraviolet light therapy within 48 hours of an attack. Other modalities employed over a fourteen year period were largely ineffectual. It was concluded that the ulceration and scar formation were an intrinsic part of the herpetic process in this patient.

Key words: Herpes simplex; Scars; Photo therapy; Acridine orange

There is scant recognition that recurrent herpes simplex can be a scarring disease. Indeed most modern texts, monographs and reviews do not describe a cicatricial form of herpes simplex (5, 6, 8, 9, 11). One text suggests that any scarring which develops is a sequela of the disease due to trauma or secondary infection (4). Yet, a generation ago the Suttons (13) wrote of atrophic scars following severe cases, and in 1905 Radcliffe-Crocker (10) wrote, "I have once seen a gangrenous spot a quarter of an inch in diameter in an *H. labialis*".

The present report is presented to document the fact that a cicatricial type of herpes simplex does exist.

CASE REPORT

The patient was a 32-year-old woman referred to us by Xavier J. Chiampi, M.D., for further management of severe recurrent bouts of vesicular lesions of her lips. The attacks had begun 10 years ago following measles and have kept on occurring at irregular intervals, never greater than 6 months. Generally the attacks have been severe, leading to ulceration, eschar formation and requiring from 4 to 8 weeks for healing. They have resulted in marked local atrophic scarring. There have been no known trigger factors, and the patient has had no other cutaneous or genital lesions.

The therapeutic history disclosed an indifference to a series of smallpox vaccinations, oral and topical steroids,

as well as alcohol, camphor, silver nitrate, glycoxide and iododeoxyuridine locally.

The patient has been under our care for a subsequent 4 years. A clinical diagnosis of recurrent herpes simplex was made and a Tzanck smear was consistent, showing balloon cells and multinucleated epithelial giant cells. Viral typing was attempted once but without success. Four attacks observed by us showed clustered vesicles in varying sites on the upper and lower lips, both on the vermillion border as well as on the cutaneous portion. The vesicles became confluent and by one week were followed by the formation of a firm red eschar which required 4 to 7 weeks for separation and healing (Fig. 1). The patient denied any manipulation or trauma to the lesions. There was no evidence of artefactual interference, nor was the appearance that of impetigo or ecthyma.

The course was unaffected by treatment with systemic clindamycin, minocycline or avlosulfone. Topically, vioform was without effect, nor could the lesions be aborted by early liquid nitrogen treatment. A course of 16 intracutaneous injections of herpesvirus hominis antigen was without effect on either the frequency or severity of attacks. Nor was there any unusual inflammatory or destructive local effect from the intradermal antigen. This antigen is a formaldehyde-inactivated type 1 herpes virus grown on rabbit kidney tissue culture, prepared by Eli Lilly Co., Indianapolis, on a trial basis. It is analogous to that being studied elsewhere (7). Skin tests to commercially available bacterial antigens including streptococci pyogenes and staphylococcus albus were without any severe local or scarring reaction.

In 1971 treatment was instituted with acridine orange as a photo-inactivator. Using essentially the technique outlined by Rubin & Heaton (12), the patient was seen within 48 hours of the onset of an attack. The area was anesthetized with intradermal xylocaine, the vesicular lesions completely curetted away, and hemostasis achieved by pressure. The area was then painted with acridine orange solution (1% in pH 7.4 buffered saline) and the area exposed to Westinghouse black light fluorescent bulb lighting at 6 inches for 15 minutes.

This treatment reduced the healing time to less than 2 weeks and scarring did not ensue. Two subsequent attacks at new sites occurring within the following 6 months were treated with equal success by the acridine orange, ultraviolet light therapy. There were no more attacks for over 2 years. In the last 6 months two further recurrences developed in sites not previously treated with curettage and acridine

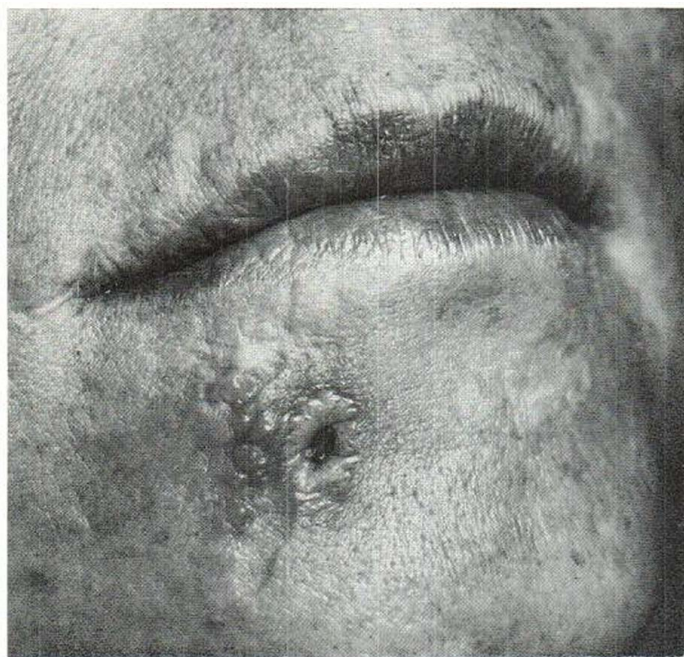


Fig. 1. Initial appearance of cicatrizing herpes simplex. Note the grouped vesicles and incipient destructive eschar formation.

orange. Treatment of these limited sites with the same technique was followed by complete healing within 7 days and without ulceration or scarring.

DISCUSSION

Scarring in recurrent herpes simplex is so rare as to be disregarded or to be viewed as a complication, not a form of the disease. However, study and observation of the repeated scarring episodes in this patient led to the conclusion that there is a cicatricial type of herpes simplex. It manifests itself initially as an essentially typical vesicular eruption, involving only the epidermis. However, instead of healing within a week, it progresses to a necrotizing dermal process, with eschar formation. The ulcerative lesion requires 4 to 7 weeks to heal, leaving a permanent, depressed, atrophic scar (Figs. 2, 3).

The sequence bespeaks more than an intra-epidermal destructive change. One might think of the deeper involvement as being factitious in origin, but there was little to support this. The patient was emotionally stable, meticulous in following instructions, showed no lesions other than at the sites of herpes, and showed a course consistent with the severity of the initial herpetic lesions.

One might view the process as due to secondary infection such as ecthyma, but the failure of systemic

antibiotics to alter the inexorable long course is not in keeping with this view. Also noteworthy is the absence of lesions anywhere but at the site of the initial herpes. The patient had never had pyodermas, and showed no unusual bacterial sensitivities. This scarring seemed to be the direct result of the herpetic infection attacking the dermis as well as the classic epidermal locus.

Significantly, removal of the viral-infected epidermis by curettage and photo-therapy completely aborted the progress of lesion, preventing eschar as well as scar formation. It also prevented further attacks at that precise locus just as has been reported in the common non-scarring type of herpes simplex (3).

It is now known that the virus resides permanently in the trigeminal ganglion (2). Possibly the curettage and or photo-therapy locally limits future attacks by focal destruction of the normal axonal pathway used by the virus to re-enter the epidermis when it initiates a new lesion.

It is not evident how the herpes virus induced the destructive dermal change. The patient's immune status was normal and, interestingly, patients receiving immunosuppressants do not show a scarring form of herpes simplex (1). The patient showed no unusual sensitivity to stock herpes virus hominis antigen on skin testing. One could postulate that an

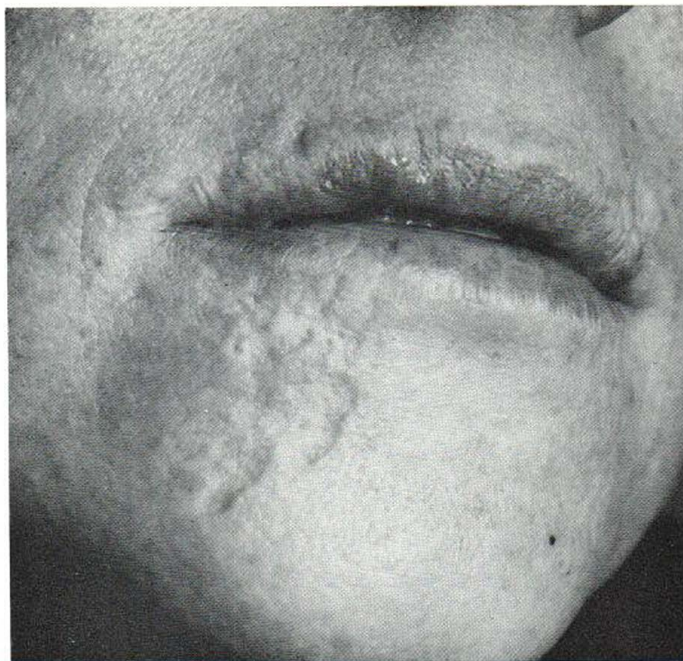


Fig. 2. Scarring of chin and lip at sites of recurrent herpes simplex.

unusually virulent strain or type of the herpes virus was present in this patient. This might be demonstrable on viral study such as inspection of pock size on chorioallantoic membrane culture, but such data were not available to us. Also, the herpes virus in this patient may have been dermatropic as well

as epidermotropic. For example, the possibility of invasion of the local cutaneous vasculature by the virus with consequent infarct cannot be disregarded. Further studies are needed to bring us understanding of the pathogenesis of this scarring type of herpes simplex.

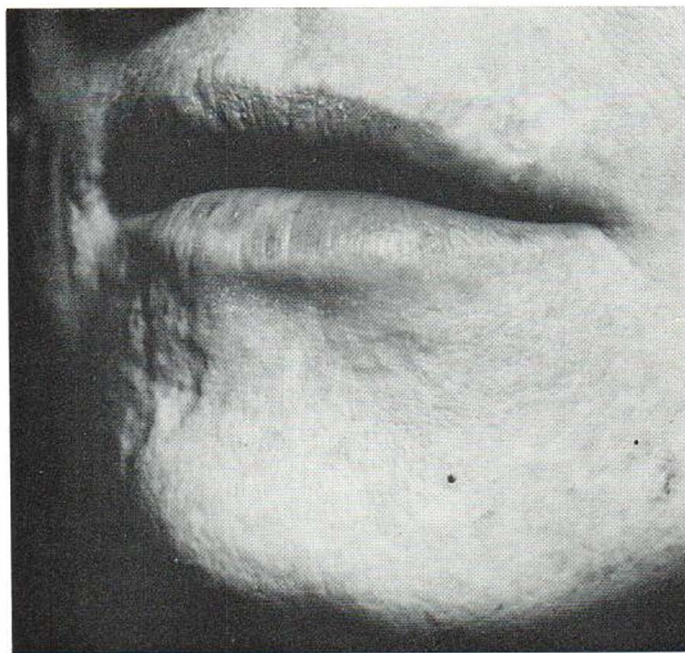


Fig. 3. Cross lighting of post-herpetic scars to show depth.

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