Management of Nonstaphylococcal Toxic Epidermal Necrolysis

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We were interested to read the paper of Tegelberg-Stassen et al. [1], and we adhere without restriction to their conclusions. As a matter of fact, at the Department of Dermatology, we have treated all our cases of toxic epidermal necrolysis (excluded of course the cases of staphylococcal scalded skin syndrome) in the same way, i.e. reserved barrier nursing, control of fluid balance, timely antibiotics and overall high doses of corticosteroids.

From our experience, we would like to add some comments:

High doses of corticosteroid must be resorted to as soon as possible in order to control the intensity of the eruption.

The treatment does not prevent the shedding of the epidermis already severely aggressed, but it minimizes the oozing.

Improvement must be achieved in a few days only; if delayed for more than 7 days, prognosis gets much worse, and infections multiply unavoidably.

The comfort of patients and the rapidity of recovery are definitively enhanced.

Reference In Reply

1 Tegelberg-Stassen MJAM, Vloten WA van, Baart de la Faille H Management of nonstaphylococcal toxic epidermal necrolysis Follow-up study of 16 case histories. Dermatologica 1990; 180 124-129.

Chronic Vulvar Purpura

Sir,

In a recent issue of Dermatologica, Kato et al. [1] described a chronic vulvar purpura in a 56-year-old Japanese woman. The purpuric lesion was persistent during a follow-up time of 8 years and has been related by the authors to an abdominal ptosis.

A cause of persistent purpuric lesions with hemosiderin deposits in the vestibulum vaginae is the plasma cell vulvitis of Zoon (vulvitis circumspecta plasmacellularis). This circumscribed chronic vulvitis is usually located around the posterior circumference of the introitus vaginae or on the inner side of the labia minora; most often it presents as a dark red colored smooth lesion or a slightly erosive inflammatory erythroplasia [2].

The main histologic hallmarks are: atrophy of the epithelium, dense lymphoplasmocytic infiltration of the upper corium with invasion of the lower epithelial layers and extravasation of erythrocytes leading to a less or more dense deposition of hemosiderin [3]. Persistent lesions heavily laden with hemosiderin look clinically dark pigmented. In histologic specimens of older
lesions, with a degree of fibrosis, plasma cells progressively shift to the inferior border of the inflammatory infiltrate. They may even disappear and are not considered by all pathologists as an essential feature for diagnosis [4]. For this reason the synonym ‘lichenoid telangiectatic purpuric vulvitis’ is sometimes proposed. The same clinical and histological evolution occurs in Zoon’s plasma cell balanitis in noncircumcised male patients.

Concerning the reported case, I suggest that the authors discuss also Zoon’s vulvitis, either as a differential diagnosis or as a possible identical entity. Immunohistologic staining for B cells or immunoglobulins would eventually be helpful to clarify the nosology of this chronic genital pigmentary purpura.

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References

Dear Sir,
We thank the pertinent comments of Dr. Grosshans for kindly indicating to include Zoon’s vulvitis in the differentiation of purpuric vulvar lesions.

From the specific location of the lesion, we have also thought of the possibility of Zoon’s vulvitis, but histologic inability to demonstrate a dense plasmacytic infiltration in our biopsy specimens obtained three times during the follow-up period of 3 years led us to rule it out. Histologic features as well as clinical features were almost the same, and consistent. T cells and histiocytes were predominant, and only a few intermingling plasma cells that can be understood from the site of involvement were observed. In addition to this inflammatory infiltration, the most striking features in our case [1] are changes of blood vessels. A thickening of the vascular wall was consistently associated with an extravasation of erythrocytes as well as a dense deposition of hemosiderin. These features, suggesting that the lesions in our case are still in an active stage persistently stimulated by venous stasis, do not conform to those described in the chronic phase of Zoon’s vulvitis cited by Grosshans.

A moderate lymphohistiocytic infiltration and swelling of the vascular wall are common features of the microvascular system wherever there is stasis. Therefore, as the most plausible mechanism, we speculated that the abdominal ptosis plays an important role in the pathogenesis in our case. After the publication of our paper, we experienced a quite similar case in a 47-year-old woman associated with abdominal ptosis.

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Reference
Flagellate Dermatitis after Bleomycin

Sir,

We have read with interest the paper by Cortina et al. [1]. We observed a similar case [2] in which we performed a histological and immunohistochemical study [3]. Our histological findings were quite similar to those described by Cortina et al. [1], but we were not able to find any anaplastic-like cells or any atypical mitotic figures in the inflammatory infiltrates. Our immunohistochemical study showed that the infiltrates were composed mainly by T helper lymphocytes (ratio CD4/CD8 3:1). Thus, a strong HLA-DR expression against a mild positivity for interleukin-2 and a high degree of CD1-positive cells was noted in the inflammatory infiltrates. The low degree of interleukin-2 we found can be explained...