

Pemphigus Vulgaris Possibly Triggered by Cilazapril

Sir,

Cilazapril is a new angiotensin converting enzyme inhibitor (ACEI) which, like other ACEIs, is a non-thiol molecule, containing an active amide group. Since the early 1990s an increasing number of case reports have been published pointing to the role of this family of drugs in inducing or triggering pemphigus (1). In 1992, the ACEI Enalapril was found to be the most powerful acantholytic drug *in vitro* (2). Buzon et al. (3) reported the first case of pemphigus foliaceus associated with Cilazapril. We describe here the possible association of the drug with pemphigus vulgaris.

CASE REPORT

A 67-year-old Ashkenazi Jewish male, born in Germany, was admitted to our department with painful oral and pharyngeal erosions of several months duration. He had a 2-year history of hypertension for which he was being treated with Cilazapril. There was no other cutaneous involvement. The patient reported losing his job prior to the outbreak of the oral lesions.

Histology of a biopsied specimen and direct and indirect immunofluorescence studies were consistent with pemphigus vulgaris. HLA studies revealed HLA A2, A26, B38, B35, DR5 and DR 4.

Cilazapril was discontinued and the erosions gradually improved. Despite the improvement the patient insisted on systemic therapy and was given a medium dose of oral steroids, prednisone 60 mg/day. He is currently responding to this treatment, with old lesions healing gradually and no new lesions. In the absence of new lesions, the steroids are being tapered. Interestingly, all blood pressure measurements were within normal limits without antihypertensive treatment.

COMMENT

The literature contains several reports of ACEIs associated with pemphigus, but only one concerning the new drug cilazapril (3). While Buzon's patient (3) developed pemphigus foliaceus with severe cutaneous involvement 3 months after starting Cilazapril, our patient developed oral pemphigus vulgaris more than 12 months after initial exposure to the drug. This variability of clinical manifestations (type of pemphigus, distribution of lesions) is intriguing and may be related to the genetic background of our patient, an

Ashkenazi Jew; pemphigus vulgaris is relatively common in this population and there is a predominance of HLA DR4 (4) as seen in our case. We also consider the emotional stress of losing his job to be an important factor in triggering the disease (5).

The subsidence of the lesions following withdrawal of Cilazapril indicates that the pemphigus vulgaris was triggered by the drug. There is a growing body of evidence that induced or triggered pemphigus can be of the vulgaris variant and not solely of the foliaceus or erythematosus one (6). Our and Buzon et al.'s (3) patients demonstrate this variability.

Our case also points up the importance of obtaining a detailed drug history in every case of pemphigus. Such a meticulous approach might disclose more information about the relationship between Cilazapril and pemphigus.

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Accepted January 12, 2000.

Edith Orion¹, Ephraim Gazit² and Sarah Brenner¹

¹Department of Dermatology, Tel Aviv-Elias Sourasky Medical Center, 6 Weizman Street Tel Aviv 64239 and ²Tissue Typing Laboratory, Chaim Sheba Medical Center, Tel Hashomer, Sackler School of Medicine, Tel Aviv University, Israel.