

pustules were present concurrently. The patient complained of occasional pruritus and burning. Chronic plaque psoriasis on elbows was confirmed seven years before, for which she received topical corticosteroids. Focal infections were not found on clinical examination. It is important to note that the patient's smoking habit aggravated the condition. Palmoplantar pustular psoriasis was diagnosed based on clinical picture and histopathologic appearance. Histopathologic analysis of skin lesion of the sole showed epidermal acanthosis with parakeratosis and large accumulation of neutrophils within the stratum spinosum, known as spongiform pustule of Kogoj. In the dermis, the capillaries were elongated and tortuous. PUVA cream phototherapy was administered five times weekly for four weeks of her hospital stay. Topical corticosteroids were applied under hydrocolloid occlusion, which significantly enhanced regression of the skin lesions. Most patients with palmoplantar pustular psoriasis have an underlying disease that can be identified, but in our case the onset, fluctuations and duration of the disease were not associated with focal infections. It is important to note that smoking aggravates the disease and has unfavorable impact on treatment success. PUVA cream phototherapy and topical corticosteroids result in dramatic improvement of the disease with significant psychosocial benefit.

#### **DETERMINATION OF EGFR, BCL-2 AND KI 67 IN PATIENTS WITH ORAL LICHEN PLANUS**

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Oral lichen planus (OLP) affects 1% to 2% of the population. Typically, it affects middle-aged and elderly women, although it can affect men, and rarely children. The cause of OLP is not known, but it is known to be mediated through T-lymphocytes to as yet unknown antigen. There is about a 1% risk of cancerous change over a 10-year period. The main problem is to identify lesions that will transform into cancer. Nor-

mally, tumor markers are used to identify cancer, but in some instances they can suggest potentially malignant lesions. Therefore, we evaluated OLP lesions using immunohistochemistry markers (epidermal growth factor receptor (EGFR), bcl-2, Ki67) in comparison to the density of subepithelial band inflammatory infiltrate. OLP patients were divided into smokers and non-smokers. There were 15 OLP patients in smoker group (age range 28-70 years) and 49 OLP patients in non-smoker group (age range 21-72 years). The mean age at OLP diagnosis was lower in OLP smokers ( $48.7 \pm 10.6$ ) than in OLP non-smokers ( $55.8 \pm 11.5$ ). Conventional hematoxylin and eosin staining showed no difference in the diagnosis of OLP between smoking and non-smoking group. Spearman's correlation test for EGFR expression showed no between-group difference ( $P=0.4$ ). Comparing EGFR, Ki67 and bcl-2 expression in squamous epithelium according to density of subepithelial band inflammatory infiltrate (using semi-quantitative method; low-1, medium-2, high-3), we found significant difference ( $P<0.01$ ) between smokers and non-smokers with OLP. Immunohistochemical expression of EGFR, bcl-2 and Ki67 in squamous epithelium in relation to the density of subepithelial inflammatory infiltrate showed significant difference between OLP smokers and OLP non-smokers ( $P=0.0005$ ). Study results suggested that smokers were younger than non-smokers at the time of OLP diagnosis, which may imply the possibility of cancer development at younger age than statistically reported for oral carcinoma. Additional immunohistochemical analysis revealed smokers with OLP to show a statistically significant expression of EGFR, Ki67 and bcl-2 markers in squamous epithelium in relation to the density of subepithelial band inflammatory infiltrate as compared to OLP non-smokers. These findings could contribute to understanding the carcinogenesis and pathogenesis of OLP. Additional researches in a larger sample are needed to confirm our presumption.