Transepithelial Elimination in Cutaneous Leishmaniasis

Sir.
Azadeh & Abdulla made the challenging statement that transepithelial elimination in cutaneous leishmaniasis has not been previously reported. Kurban et al. (1966) said exactly the same thing. A later paper by Malik & Kurban (1971) proposed the term "Catharsis" for such elimination.

REFERENCES


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Reactivation of Healed Primary Syphilitic Ulcer: A Manifestation of Febrile Herxheimer Reaction in Secondary Syphilis

Sir.
Febrile Herxheimer reaction frequently occurs when patients with early syphilis are treated with treponemical drugs (1). About 95% of patients with early secondary syphilis experience the reaction after treatment with penicillin (2). Patients usually develop fever, chills, malaise, arthralgia, nausea and vomiting. There may be an exacerbation of cutaneous lesions. Several mechanisms have been postulated for the pathogenesis of febrile Herxheimer reaction. It has been attributed to the release of treponemal breakdown substances like endotoxins. There is also some experimental evidence to support the speculation that cell-mediated immunity may play an important role. We here report a patient with secondary syphilis, who developed reactivation of a healed primary syphilitic lesion after treatment with parenteral benzathine penicillin. This phenomenon has not been reported earlier.

CASE REPORT
A 16-year-old unmarried man presented with a 4-week history of several asymptomatic, erythematous lesions on the trunk and upper thighs. He gave a history of a single unprotected heterosexual exposure with a professional 4 months earlier. Two weeks after the sexual contact, the patient developed an asymptomatic papule on the coronal sulcus; this ulcerated in a few days. The ulcer healed spontaneously in 2–3 weeks, to leave behind a hypopigmented scar. The patient had been asymptomatic for 8–9 weeks when he noticed the present rash. The patient did not give a history of any constitutional symptoms. He had not received any treatment for this.

Physical examination revealed a small, non-tender, discrete lymphadenopathy in the cervical, axillary, epitrochlear and inguinal regions. He had several asymptomatic erythematous papulosquamous, annular lesions on the trunk and upper thighs. In addition he had eroded papules in the perianal region. There were no oral or palmpoplantar lesions. There was a single hypopigmented atrophic scar on the coronal sulcus.

The patient's routine investigations were within normal limits. His serology for syphilis (VDRL) was reactive 1: 64. Dark-field examination performed from the perianal lesion was positive for Treponema pallidum. A biopsy from the papulosquamous lesion was consistent with a diagnosis of secondary syphilis. The patient was given 2.4 million units of parenteral benzathine penicillin after ruling out sensitivity to the drug. Within 3 h he developed fever, which reached a peak of 39.4°C 9 h after the dose of penicillin and lasted for 48 h. He also complained of giddiness and nausea. However, he did not develop any new cutaneous lesions.

About 6 h after treatment with penicillin, the patient noticed swelling at the site of the healed primary lesion. This ulcerated after 2 days. At that time there was a single indurated ulcer on the coronal sulcus (Fig. 1). Dark-field examination performed from the ulcerated genital lesion was negative. The patient refused biopsy from the ulcer. The ulcer healed spontaneously within a fortnight, without any further treatment.

DISCUSSION
In febrile Herxheimer reaction, there may be an exacerbation of the skin lesions in the form of either appearance of new

Fig. 1. Single indurated ulcer at the site of a healed primary syphilitic ulcer.

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lesions or and erythema and edema of the pre-existing lesions, but this manifestation is decidedly infrequent (4, 5). Our patient developed a moderately severe febrile Herxheimer reaction but did not have exacerbation of cutaneous lesions. It is unique that he developed an indurated ulcer at the site of the healed primary lesion.

REFERENCES


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Cytokine Profile of Bilateral Pseudocyst of the Auricle

Sir,

Pseudocyst of the auricle (PA) occurs on the upper part of the ear with smooth surface as the result of trauma, mechanical stimuli or infection. Although the pathomechanism(s) of the blistering process in PA is still unknown, an overproduction of glycosaminoglycans by repeated minor trauma or mechanical stimuli to the ear cartilage is suggested to be the primary stage of this disorder (1). Recent investigations have revealed that various cytokines are released from cartilages, fibroblasts, activated lymphocytes or monocytes. We examined the cytokine profile of the blister fluids and sera of a patient with bilateral PA and 2 patients with solitary PA.

Sera and blister fluids were collected from a 45-year-old male with bilateral PA, a 64-year-old male with solitary PA, and a 49-year-old female with solitary PA, who were diagnosed by clinical and histopathological examinations, before treatment. Specimens were stored at -80°C until use. Interleukin-1β (IL-1β), IL-2, IL-6 and tumor necrosis factor-α (TNF-α) were measured by ELISA technique. In normal sera, cytokine levels were as follows: IL-1β <10.0 pg/ml; IL-2 <0.8 U/ml; IL-6 <4.0 pg/ml; TNF-α <5.0 pg/ml.

We demonstrated a marked increase of IL-6 and a slight increase of IL-1β in the blister fluid in the case of bilateral onset, while serum levels were within normal range (Table I). IL-6 was also elevated in the two solitary PA fluids. Cartilage degeneration is thought to be further accelerated by various cytokines released by the resident mesenchymal cells, the infiltrating neutrophils, monocytes or macrophages (2). IL-1 is the most important mediator of inflammation and cartilage destruction, which increases the synthesis of proteases and PGE2 by chondrocytes, and inhibits the formation of extracellular matrix components (3). IL-6 is induced by IL-1 and is a stimulator of chondrocyte proliferation (4). Furthermore, analysis of the LDH iszyme pattern of the blister fluids revealed an increase in LDH4 (31.6% in lt BF, 23.2% in rt BF) and LDH5 (51.7% in lt BF, 42.5% in rt BF) isozymes, while the serum isozyme pattern exhibited a dominance of LDH1 of 25.3% (LDH4: 9.8%; LDH5: 18.8%) in the bilateral case. The LDH isozyme pattern of articular cartilages demonstrates mostly LDH4 and LDH5 isozymes (5). It is suggested that the release of cytokines is derived from infiltrating inflammatory cells or damaged cartilages, which play a role in the formation of PA.

REFERENCES


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Table I. Cytokine profiles of sera and blister fluids of patients with pseudocyst of the auricle

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