Cutaneous Amebiasis in an Iranian Immunodeficient Alcoholic: Immunochemical and Histological Study

F. Loschiavo a
B. Guarneri b
T. Ventura-Spagnolo a
F.P. Purello C’Ambrosio c
L. Ricciardi c
S.P. Cannavò b

aDepartment of Medical Pathology and Tropical Medicine, and bDepartment of Dermatology, cSchool of Allergy and Clinical Immunology, University of Messina, Italy

Key Words
Cutaneous amebiasis
Alcoholism
Immunological deficiency

Prof F. Loschiavo, Policlinico Universitario, Pad. H, Viale Gazzi, I-98125 Messina (Italy), Tel. +90 2212075, Fax +90 694773

Although amebiasis can appear at unusual extraintestinal localizations, especially in the presence of a variable immunological balance between host and parasite, cutaneous localization with lesional parasites is rarely found [1-6]. We report on a case of cutaneous amebiasis with lesional protozoa. The observation is remarkable because of particular immunologic, histologic and clinical aspects.

A 30-year-old Iranian male, who had been living in Messina (Italy) for several months, was found to be positive for Entamoeba histolytica cysts during a routine screening among immigrants. In Iran he had been working as an agricultural laborer who regularly used fertilizers. His personal history revealed that he was an alcoholic.

Clinical examinations showed the presence of hepatomegaly. In the mesogastric site it was possible to observe a circular-like ulceration (diameter 3 cm approximately, fig. 1) with a reddish coating over a period of 2 months; the lesion margins were sclerotic and painful on touch. The histological examination of the skin biopitic fragment showed the infiltration of monocytoid elements, with regressive phenomena of the cutaneous cells and the presence of E. histolytica trophozoites in the exudate (fig. 2). The patient’s hematologic and immunologic parameters revealed that he was an alcoholic with cellular immunodeficiency but without HIV positivity.

Once it had been demonstrated that the amebic infection was the cause of this lesion, an appropriate therapy was prescribed, first with metronidazole (400 mg twice daily) and second with a high dosage of parenteral pararnomicine (250 mg 4 times daily) for 6 weeks. At the same time treatment for correcting the cellular immunological deficit and disorders caused by alcohol was started. After 3 weeks of this treatment Ravault’s paste, containing dihydroemetine at a high concentration, was applied locally. Four weeks later the lesion had completely healed and did not recur.
In the literature there are few studies concerning the correlation between alcoholism, immunological deficiency and skin lesions due to E. histolytica [6-9]. Nevertheless in chronic alcoholics some authors [10, 11] have observed a considerable modification in the regulation process of the T and B lymphocyte function. In addition, in alcoholics the skin presents atrophy of the epidermis and of the sebaceous glands. Such modifications can cause a notable weakening of the cutaneous barrier layer which, together with the depression of the cell-mediated immunity, facilitates the implant of parasites on the epidermic surface and subsequent colonization. In conclusion, we think this case of cutaneous amebiasis in an immunodeficient alcoholic should be considered as an opportunistic infection.

References
Serum Antibodies to Parvovirus B19 in Patients with Pityriasis rosea

B.S. Marcus-Farber’, R. Bergman”, E. Ben Porathb, J.V. Zaltzmanb, R. Friedrnann-Bimbaunib

Departments of “Dermatology and bMicrobiology.

Rambam Medical Center and the Brace Rappaport Faculty of Medicine, Technion Israel Institute of Technology, Haifa, Israel

Parvovirus B19 is a DNA virus of the Paroviridae family, of which it is the only one considered to be a human pathogen [1]. It has been shown in recent years to be the causative agent of erythema infectiosum [2]. More recently, it has also been implicated as a cause of papular-purpuric (petechial) gloves-and-socks syndrome [3,4]. The cause of pityriasis rosea is still unknown although a viral etiology is strongly suspected [5]. To the best of our knowledge, the role of parvovirus B19 as a possible causative agent of pityriasis rosea has not been examined yet. We report herein the results of serological tests for this virus in 13 patients with pityriasis rosea.

Thirteen patients with classical pityriasis rosea, 7 females and 8 males, aged 17-25 years (mean 23.3), were included in the study. Blood samples were collected 7-30 days (mean 13 days) after the appearance of the rash (including the herald patch). The presence of IgG and IgM antibodies directed against parvovirus B19 was studied using a standard IBL ELISA kit (Kurzarbeitsanleitung, Hamburg, Germany). IgG antibodies were found in 5 (38%) patients. IgM antibodies were not detected in any of the 13 patients.

The percentage of pityriasis rosea patients with positive IgG antibodies against parvovirus B19 is close to the reported prevalence of 40-60% of this antibody in the general population [1]. IgM antibodies to parvovirus B19 appear a few days after infection with this virus [1]. Therefore, the absence of IgM antibodies, which reflect recent infection, in all of our patients does not lend support to a possible role of parvovirus B19 in the pathogenesis of pityriasis rosea.

References


Bat Sheva Marcus-Farber, MD,
Department of Dermatology, Rambam Medical Center,
Haifa 31096 (Israel)

Dermatology 1997;194:371-373

The Petrified Ear – A Manifestation of Dystrophic Calcification

R. Strumia, A.R. Lombardi, E. Altieri Clinica Dermatologica, Università di Ferrara, Italy
Key Words
Petrified ear · Dystrophic calcification · Diabetes mellitus

The petrified ear (PE) is a rare condition in which the ears become stone-hard [1-4]. The real aetiology is still unknown, but many patho-genetic factors are reported in the literature (table 1). We report a case in which the only demonstrable cause was diabetes. Dystrophic damage of the cartilage following diabetic micro-angiopathy might be one of the pathogenetic stimuli of this rare entity.

A 75-year-old male who worked as a guard until he was 60, presented with an ulcerated plaque localized on his left ear. The lesion

Letters to Dermatology
371