Remission of Localized Cutaneous Leishmaniasis in a HIV-Positive Patient Using Systemic Terbinafine

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Key Words

Terbinafine
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Patients with advanced HIV infection may develop localized as well as disseminated leishmaniasis. Treatment includes intralesional or systemic antimonial pentavalents. In some resistant cases amphotericin B, pentamidine isethionate and interferon-gamma [1] have also been used. We present a case of localized leishmaniasis with remission following oral terbinafine.

The patient was a 26-year-old male with a previous history of parenteral drug addiction, HIV infection and hepatitis C since May 1993. Laboratory values showed a normal hemogram, CD4 of 165 lymphocytes/mm³ and a mild elevation of liver enzymes. Zidovudine treatment was recommended as well as prophylactic co-trimoxazole.

The patient was not cooperative and did not follow the controls. In May 1995 after a 14 months' stay in Colombia he presented cutaneous desquamative erythematous lesions disseminated on the trunk and limbs, especially on the legs, which suggested tinea corporis; he also presented onychomycosis of the fingers and a periauricular and auricular plaque with superficial excoriation for 1 month (fig. 1). Physical examination was otherwise normal. CD4 was 42 lymphocytes/mm³. Therapy with terbinafine 250 mg/day was recommended to treat the tinea, until the histopathology results of the auricular lesions were available. After 14 days of treatment the lesions disappeared and so did those located on trunk and limbs. Culture of skin and nail lesions showed *Trichophyton mentagrophytes* and *Trichophyton rubrum*. Biopsy of the outer ear showed abundant *Leishmania* (fig. 2).

The history, clinic and histology confirm the diagnosis of American cutaneous leishmaniasis [2]. It is caused by *L. braziliensis*, *L. mexicana* or *L. amazoniensis*, and it is endemic in Central and South America. Although metronidazole, clofazimine and ketoconazole have also provided good results, the experience with these drugs is limited and they are not in the routine prescription. Terbinafine, an epoxidase squalene inhibitor antifungal agent, also presents in vitro activity against some species of *Leishmania*, specially *L. mexicana* [3] and *L. amazoniensis* [4]. In our patient's condition terbinafine's clinical efficacy was accidentally demonstrated since it was prescribed to treat tinea corporis and onychomycosis.

Oral terbinafine should be further investigated as an alternative systemic therapy for localized leishmaniasis.

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Fig. 1. Lesion of cutaneous leishmaniasis with periauricular and auricular excoriation.

Fig. 2. Dense dermal infiltration of lymphocytes and macro-phages with intracellular Leishmania.

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References

Kolde G, Luger T, Sorg C, Sunderkötter C: Successful treatment of cutaneous leishmaniasis using systemic interferon-gamma. *Dermatology* 1996;192:56-60.

Pearson RD, de Queiroz Sousa A: Leishmania species: Visceral (kala-azar), cutaneous and mucosal leishmaniasis; in Mandell GL, Douglas RG, Bennet JE (eds): *Principles and Practice of Infectious Diseases*. New York, Churchill Livingstone, 1990, pp 2066-2077.

Balfour JA, Faulds D: Terbinafine. *Drugs* 1992;43:259-284.

Vannier-Santos MA, Urbina JA, Martiny A, Neves A, de Souza W: Alterations induced by the antifungal compounds ketoconazole and terbinafine in Leishmania. *J Eukaryot Microbiol* 1995;42:337-346.

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Schönlein-Henoch Purpura Associated with Gastric Helicobacter pylori Infection

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Helicobacter pylori

Schönlein-Henoch purpura (SHP) is a common vasculitis affecting the skin, gastro-intestinal tract, joints and, occasionally, other organs. IgA glomerular mesangial deposits are characteristic of the disease. Infectious agents affecting the upper airways or the gastrointestinal tract have been suggested as the cause.

Case Report

A 65-year-old man was admitted in January 1995 for numerous extensive papular and purpuric lesions located mainly on the lower limbs. However the trunk and upper limbs were also affected. Cutaneous biopsy revealed moderate leukocytoclasia vasculitis with fibrinoid necrosis of the dermal capillaries. The purpuric rash was accompanied by abdominal pain, diarrhoea and melaena. Oesophago-gastroduodenoscopy performed 2 days after onset of the rash revealed petechial haemorrhage of the stomach. Histologic examination showed atrophic chronic gastritis with haemorrhagic suffusions in the superficial chorion. This was associated with marked presence of *Helicobacter pylori* within the lumina of dilated antral foveolae. Bacteriological culture was not performed. Colonoscopy was performed because of persistent abdominal pain and rectorrhagia and showed diffuse purpuric involvement of the ileum and right bowel. Biopsies demonstrated acutely ulcerated and haemorrhagic ileitis. Histological examination showed focal oedematous and haemorrhagic lesions in the chorion.

Except for arthralgia and cutaneous and digestive involvement, clinical examination was normal. Blood cell count and biological tests (including creatinaemia 84 µg/l) were normal except for proteinuria (maximum 3 g/24 h), elevated C-reactive protein (53 mg/l) and increased IgA level (4.1 G/l, normal range 1.01-3.26). HCV, HBV and

HIV serology tests were negative. Antinuclear antibodies were negative. Renal biopsy showed focal glomerular lesions with abundant mesangial IgA and fibrin deposition. The patient was treated with 2g/day amoxycillin and 1 g/day clarythromycin for 8 days, associated with 20 mg/day omeprazole for 1 month and 100 mg/day dapsone. Cutaneous lesions disappeared within 2 months. Creatinaemia remained within the normal range and proteinuria returned to normal within 6 months. One year later, clinical examination and biological tests showed no relapse.

Comments

SHP is a syndrome with multiple aetiologies. It has been reported after infectious diseases, but the search for non-infectious aetiologies of vasculitis is necessary. In our case only *H. pylori* was evidenced by histological examination in antral ulcerations. *H. pylori* is considered to play an important pathogenic role in duodenal ulcers and gastritis [1]. Treatment usually consists of omeprazole associated with antibiotics (amoxycillin alone or with macrolides or imidazoles). Another case of SHP associated with antral ulcerations, probably secondary to *H. pylori* infection, was recently reported [2]. In this case *H. pylori* infection was treated with omeprazole and amoxycillin and signs of SHP disappeared. A relapse occurred 10 months later, once again associated with *H. pylori* gastric infection. This case was strongly suggestive of a causative role of *H. pylori* in the occurrence of SHP. In addition, some cases of SHP have been reported after infection with related bacteria, *Campylobacter jejuni* [3,4].

Because abdominal pain is frequent in SHP, fibroscopic examination is not systematically performed: thus association of SHP with *H. pylori* infection may be underestimated. This may be of importance for the treatment and prevention of relapses of SHP, since eradication of *H. pylori* is possible [1].

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References

de Boer WA, Tytgat GNJ: 90% cure: Which *mu*-Helicobacter treatment can achieve this treatment goal? *Am J Gastroenterol* 1995;90:1381-1382.

Reinauer S, Megahed M, Goerz G, Ruzicka T, Borchard F, Suasanto F, Reinauer H: Schönlein-Henoch purpura associated with gastric *Helicobacter pylori* infection. *J Am Acad Dermatol* 1995;33:876-879.

Lind KM, Gaub J, Pedersen RS: Henoch-Schönlein purpura associated with *Campylobacter jejuni* enteritis. *Scand J Urol Nephrol* 1994;28: 179-181.

Schonheyder HC, Sogaard P, Frederisen W: A survey of *Campylobacter* bacteremia in three Danish counties, 1989 to 1994. *Scand J Infect Dis* 1995; 27:145-148.

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