Mycobacterium Avium Complex: Cutaneous Infection in an Immunocompetent Host

Sir,

Mycobacterium avium complex (MAC) is commonly present in the environment, but it is of low pathogenicity. It is widely believed that an underlying immune alteration is required for pathogenicity. Human infection caused by MAC organisms manifest as chronic pulmonary disease, but disseminated types are recognized in immunocompromised patients, especially AIDS sufferers (1). Cutaneous infection results from either haematogenous dissemination from pulmonary lesions or direct invasion from traumatic inoculation to the skin. It is exceedingly rare to encounter a patient with a purely cutaneous MAC infection; indeed, only few cases have been reported in the literature (2).

We describe here a 60-year-old, HIV negative, immunocompetent male who had a primary cutaneous infection with positive culture for MAC without involvement of other organs.

CASE REPORT

A 60-year-old man, a breeder by profession of sheep, presented with a 10-year history of recurrent cutaneous violaceous, reticulate, erythemato-squamous patches with pustules (Fig. 1). The lesions were localized on the thighs, buttocks, groin, legs and abdomen. The lesions started as small papules and gradually enlarged until they underwent spontaneous healing, which recurred periodically without apparent motivation. The biopsy specimen showed an epidermis with hyperkeratosis and a chronic inflammatory cell infiltration of the dermis with multinucleate giant cells and perivascular lymphocytes. Areas of dermal necrosis with numerous multinucleated granulocytes were seen. Periodic-acid-shiff (PAS) stain resulted negative. Fite staining of the biopsy specimen showed no acid-fast rods; however, after 1 month MAC bacteria developed in cultures from a small skin sample. Specimens from other organic fluids gave negative results. Routine laboratory tests, including full blood count and lymphocyte subset analysis, proved normal. A serum test for HIV antibody was negative. X-ray and scan tests were within normal limits. Treatment was started with a combination of oral tetracycline (100 mg/day) and cyprofloxacin (500 mg/day). All lesions regressed after 2 months of therapy, but new lesions appeared subsequently. The therapy was therefore modified to clarithromycin (12 mg/kg/day). The course of treatment is still ongoing.

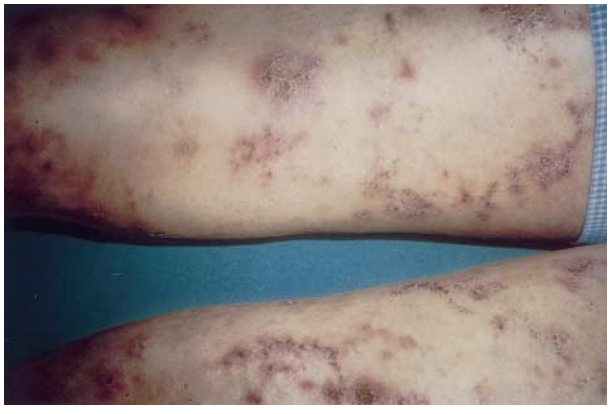

Fig. 1. Erythemato-squamous patches with pustules on the buttocks.

DISCUSSION

In primary cutaneous infection, MAC, which is present in the environment, enters into contact with the organism through inoculation to the skin. Cutaneous manifestations of MAC disease present a wide range of patterns: papules, nodules, ulcers, pustules and leprae, lupus and rosacea-like granuloma (3 – 4). In most cases the diagnosis can only be made via histological specimens but culture of small skin samples is necessary for final diagnosis.

The differential diagnosis in our case, characterized by erythemato-squamous lesions with pustules, over a wide skin surface, could have been eczema or mycosis, but the histological pattern excluded both. The finding of a granulomatous lesion with multinucleate giant cells, perivascular lymphocytes and areas of dermal necrosis, indicated another category of disease. Culture examination of a small skin sample permitted the diagnosis of mycobacterium infection. The absence of MAC localization in other organs, demonstrated by culture tests, X-ray and scan, excluded the likelihood of haematogenous dissemination. For this reason, we feel that infection came about via cutaneous inoculation. The spread of lesions in non-contiguous areas we feel was probably caused by autoinoculation through scratching of affected areas. Both these hypotheses, are supported by the literature (5). A characteristic of our patient was the periodic recurrence in lesions, which then resolved spontaneously. A possible explanation for this could be an attempt on the part of the immune system to eliminate MAC from the organism.

As regards therapy, there are no standard protocols and the necessity for prolonged treatments has been reported (6). Combination therapy is used with drugs such as streptomycin, isoniazid and ethambutol, but this treatment is unsatisfactory as the organisms are usually resistant. More promising results have been reported with oral administration of cyprofloxacin, tetracyclin and clarithromycin (7 – 8).

REFERENCES

1. Kakinuma H, Suzuki H. Mycobacterium avium complex infection limited to the skin in a patient with lupus erythematosus. Br J Dermatol 1994; 130: 785 – 790.
2. Hautmann G, Katsambas A, Lotti T. Non tubercolous mycobacterial skin infections. J Eur Acad Dermatol Venereol 1997; 9: 1 – 35.
3. Epps RE, El Azhary RA, Hellinger WC, Winkelmann RK, Von Scoy RE. Disseminated cutaneous mycobacterium avium-intracellulare resembling sarcoidosis. J Am Acad Dermatol 1995; 33: 528 – 531.
4. Kullavanijaya P, Sirimachan S, Surarak S. Primary cutaneous infection with mycobacterium avium intracellulare resembling lupus vulgaris. Br J Dermatol 1997; 136: 264 – 266.
5. Fujii K, Otha K, Kuze F. Multiple primary mycobacterium avium infection of the skin. Int J Dermatol 1997; 36: 54 – 56.
6. Bachelez H, Ducloy C, Pinquier L, Rouveau M. Disseminated varioliform pustular eruption due to mycobacterium avium intracellulare in an HIV-infected patient. Br J Dermatol 1996; 134: 801 – 803.
7. Philippot V, Yassir F, Balme B, Perrot H. Abces sous-cutanè a mycobacterium avium-intracellulare apres injections d'interferon alpha chez une maladie traitèe pour lymphome. Ann Dermatol Venereol 1996; 123: 103 – 105.

8. Alfandari S, Faiza A, Senneville E, Delaporte E, Chidiac C, Mouton Y. Isolated cutaneous mycobacterium avium complex infection in AIDS. Int J Dermatol 1997; 36: 276 – 301.

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Herpetic Pharyngitis with Mammary and Genital Herpes due to Sexual Contact

Sir,
Extragenital herpetic lesions can sometimes occur in patients with primary genital herpes simplex virus (HSV) infections. The route of infection is thought to be auto-inoculation or acquisition of the co-infection from a sexual partner (1). We report on a woman who developed primary HSV-1 infection of the pharynx, nipple and genitalia due to sexual contact.

CASE REPORT

A 48-year-old Japanese woman with no history of herpes simplex visited our hospital in April 1997. Nine days before presentation, she had had sexual contact with a man other than her husband. She did not notice whether he had any herpetic lesions. Four days later, she felt chills, and on the fifth day, pain in the right inguinal area. She noticed the development of mucocutaneous lesions on the right nipple and external genitalia on the seventh day. Physical examination showed that she had crusts and erosions on the right nipple and erosions on the right labium minus pudendae. In addition, the right inguinal lymph nodes were swollen. No pharyngeal signs, symptoms, nor any swelling of the cervical lymph nodes was noticed. The patient was administered oral acyclovir 1000 mg/day for 7 days and cefpodoxime proxetil 300 mg/day for 3 days. On the sixth day of treatment, the lesions on the nipple and genitalia had almost disappeared. We attempted to isolate the virus by using the Vero cell tissue culture system. HSV was isolated in the samples taken from the nipple and genitalia, but not in a pharyngeal swab specimen. Both isolates were identified as HSV-1 by a direct immunofluorescence technique using anti-HSV-1 and 2 monoclonal antibodies (Syva MicroTrak®, Genetic Systems Co., Seattle, WA, USA) (2). A polymerase chain reaction (PCR) was then performed in order to detect HSV DNA in the same pharyngeal specimen. The specimen was treated with Gene Release® (BioVenture, USA), which was used as a template. The primer sequences are located in the regions of the HSV-1 and HSV-2 DNA polymerase genes (3). After 30 PCR cycles consisting of 1 min at 94°C, 2 min at 55°C and 3 min at 72°C, HSV DNA was detected in the pharyngeal swab specimen. Both serum HSV-specific IgG and IgM antibody titres on the fourth day after onset were less than 1 : 10, respectively, by the indirect immunofluorescence technique.

Serum HSV antibody was not tested for in the extramarital sexual partner or the patient's husband because they did not visit our hospital.

DISCUSSION

Although this patient showed mild clinical features consistent with primary HSV infection, she had no previous history of HSV infection at any site and gave sero-negative results, suggesting that it was a primary infection.

In 1992 the prevalence of serum antibody against HSV-1 in Japanese women in their forties was about 80%. Herpes simplex of the nipple is a not infrequent clinical feature of the infection. There have been two reports on this condition (4, 5). There are two published case reports on inoculation of the nipple due to infant-mother transmission. In the case we describe here the infection appears to have been caused by sexual activity. The pathogenesis in these cases seems to be that of a bite of the nipple.

Although there are various causes of pharyngitis, HSV is an important pathogen among adults. It has been reported that oropharyngeal HSV infections complicated with primary genital herpes were seen in 10 – 15% of all patients (6). The major clinical findings of HSV pharyngitis are erythema, exudate in the pharynx, enlarged cervical lymph nodes and fever, but asymptomatic cases have been reported (7).

REFERENCES

1. White C, Wardropper AG. Genital herpes simplex infection in women. Clin Dermatol 1997; 15: 81 – 91.
2. Yoshida M, Hondo R, Tezuka T, Hiruma M. Male genital herpes complicated with urethral infection. J Dermatol 1994; 21: 595 – 597.
3. Cao M, Xiao X, Egbert B, Darragh TM, Yen TSB. Rapid detection of cutaneous herpes simplex virus infection with the polymerase chain reaction. J Invest Dermatol 1989; 92: 391 – 392.
4. Dekio S, Kawasaki Y, Jidoi J. Herpes simplex on nipples inoculated from herpetic gingivostomatitis of a baby. Clin Exp Dermatol 1986; 11: 664 – 666.
5. Sealander JY, Kerr CP. Herpes simplex of the nipple: infant-to-mother transmission. An Fam Physician 1989; 39: 111 – 113.
6. Arbesfeld DM, Thomas I. Cutaneous herpes simplex virus infections. Am Fam Physician 1991; 43: 1655 – 1664.
7. Glenzen WP, Clyde WA, Senior RJ, Sheaffer EI. Group A streptococci, mycoplasmas, and viruses associated with acute pharyngitis JAMA 1976; 202: 119 – 124.

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