8. Parslew R, Lewis-Jones MS. Localized unilateral hyperhidrosis secondary to an eccrine naevus. Clin Exp Dermatol 1997; 22: 246–247.
9. Martius I. Lokalisierte ekkrine schweibdrüsennävus mit hyperfunktion. Dermatol Wochenschr 1954; 130: 1016.
10. Chan P, Kao GF, Pierson DL, Rodman OG. Episodic hyperhidrosis on the dorsum of hands. J Am Acad Dermatol 1985; 12: 937–942.
11. Bingel G, Ertle T. Naevus sudoriferus. Z Hautch 1985; 60: 372.
12. Van de Kerkhof PJCM, Den Arend JAJC, Bousema MT, Strolz E. Localized unilateral hyperhidrosis. Br J Dermatol 1987; 177: 779–782.
13. Mayou SC, Black MM, Russell Jones R. Sudoriferous hamartoma. Clin Exp Dermatol 1988; 13: 107–108.
14. Pippione M, Depaoli MA, Sartoris S. Naevus eccrine. Dermatologica 1976; 152: 40–46.
15. Hong CE, Lee SH. Multiple eccrine nevus with depressed patches. Yosei Med J 1997; 38: 60–62.
16. Kopera D, Soyer HP, Kerl H. Human eccrine hamartoma of forearm-antebrachial organ of the ringtailed Lemur (lemur catta). A posssible phylogenetic relationship? Am J Dermatopathol 1994; 16: 275–279.
17. Nightingale KJ, Newman P, Davies MG. A functioning hamartoma associated with Down's syndrome (46,xx,–21,+t(21q21q). Clin Exp Dermatol 1998; 23: 264–266.

Cutaneous *Mycobacterium chelonae* Infection in a Presumably Immunocompetent Host

R. Satta[1], F. Cottoni[1], P. Molicotti[2], A. Lissia[3] and D. Cerimele[1]
Departments of [1]Dermatology, [2]Microbiology and [3]Pathology, University of Sassari, Viale San Pietro, 43, I-07100 Sassari, Italia.
E-mail: rsatta@ssmain.uniss.it
Accepted January 29, 2002.

Sir,

Mycobacterium chelonae, *M. fortuitum* and *M. abscessus*, species belonging to the so-called *M. fortuitum* complex, are rapid growers and have been known to be pathogenic for humans for many years (1, 2). *M. chelonae* is a saprophyte which is ubiquitous in the environment, found in both soil and water. Laboratory identification is based on growth at temperatures ranging between 28°C and 32°C in less than 7 days; typical Gram stain and colony morphology, acid fastness, the absence of pigmentation and positive arylsulphatase results at 3 days (3, 4).

We describe here a 60-year-old HIV-negative, presumably immunocompetent, woman with primary local skin lesions caused by *M. chelonae*.

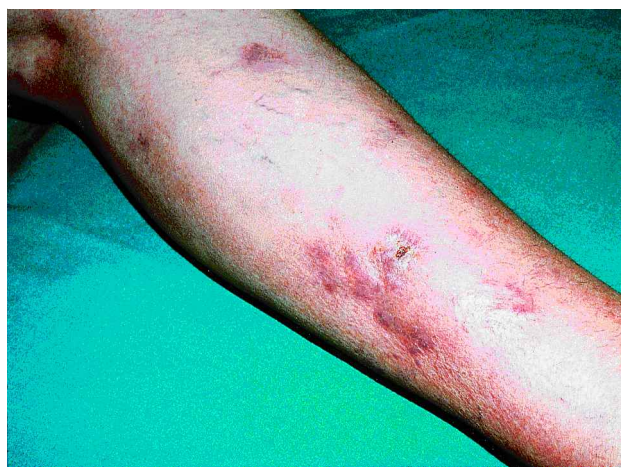

Fig. 1. Numerous cutaneous nodules on the left leg.

CASE REPORT

A 65-year-old housewife born and living in Sardinia was admitted to our Institute in March 1999 with a 10-year history of recurrent multiple subcutaneous nodular lesions on the lower left extremity. These had failed to respond to antibiotics. Examination revealed erythema and oedema of the lower left leg, with numerous spontaneous multiple violaceous nodules (Fig. 1). The left thigh presented an ill-defined, mildly erythematous and slightly hyperpigmented, indurated localized subcutaneous plaque. There was no palpable lymphoadenopathy. The lesions periodically improved without apparent cause. There was no history of trauma or iatrogenic procedures. A deep biopsy revealed a mixed granulomatous and acute inflammatory infiltrate composed of neutrophils, histiocytes and multinucleated giant cells in the dermis. No vasculitis or caseosis necrosis was present. PAS stain was negative and no acid-fast rods were present. The routine laboratory tests and HIV test were negative. Chest X-rays and abdomen and pelvic ecography were normal. *M. chelonae* grew on culture from tissue biopsy. After sensitivity testing, oral clarithromycin was initiated (500 mg twice daily). This treatment was continued for 6 months with total remission of the lesions. No relapse was observed for 2 months, but approximately 6 months later 2 new nodular lesions appeared. A new skin biopsy for histological examination revealed numerous acid-fast bacilli within a lobular panniculitis, with a mixed granulomatous and acute inflammatory infiltrate composed of neutrophils, histiocytes and multinucleated giant cells. At culture examination, *M. chelonae* grew from a tissue biopsy specimen within 7 days. This was confirmed by PCR. It was not possible to perform further sensitivity testing and the

therapy was modified to ciprofloxacin 500 mg twice daily. After a brief period of improvement, numerous new nodular lesions have appeared on the patient's left leg along with a discrete oedema. A new culture showed *M. chelonae* on tissue biopsy specimen. After sensitivity testing, therapy with ciprofloxacin 500 mg twice daily and doxycyclin 100 mg twice daily was initiated and is still underway.

DISCUSSION

M. chelonae have been implicated as pathogens in skin, soft tissue and bone infections. A retrospective review of 100 isolates of *M. chelonae* reported disseminated disease in 53%, localized disease in 35% and catheter infections in 12% of cases (2). The most common type of cutaneous presentation is disseminated disease with multiple lesions in the form of erythematous nodules, usually on the extremities. Over 94% of patients who developed disseminated cutaneous infections were receiving corticosteroid treatment for organ transplantation or autoimmune diseases and a small number had HIV infection (4). Rarely, multiple cutaneous lesions, usually on the extremities, have been reported in immunocompetent hosts (5, 6). In contrast to disseminated disease, the localized form typically occurs in immunocompetent hosts (7, 8), usually induced by trauma or in patients undergoing medical procedures such as injection or surgery. This type of infection usually manifests as a single site of cellulitis, subcutaneous abscess or osteomyelitis (9). In patients who develop a localized infection without history of trauma the immune status should be evaluated (10, 11). Catheter-associated *M. chelonae* disease infection may be localized or associated with bacteremia, often in immunosuppressed patients (5).

M. chelonae skin histopathology may show neutrophilic abscesses along with granulomatous inflammation. *M. chelonae* can be acid-fast, although the staining may be weak, but it can also be non-acid-fast. Because a negative acid-fast smear does not eliminate Mycobacterium as the cause of lesion, multiple biopsies and cultures may be needed to establish the diagnosis. If skin infection with Mycobacterium spp. is suspected, specimens should be incubated at a temperature between 28°C and 32°C and, if present, *M. chelonae* colonies will develop in 7 days. In addition, PCR has recently become available to confirm the identification of the organism (12).

As regards therapy, the use of clarithromycin in our patient, on the basis of sensitivity testing, determined a temporary remission. The subsequent reappearance of the disease is in accordance with the cases reported in the literature. Indeed, although excellent response rates have been reported with claryithromycin in monotherapy (13, 14), cases of acquired (mutational) resistance to this antibiotic have been reported (15, 16). Additional antimicrobic agents should be selected on the basis of sensitivity testing in the case of disease reappearance, and at least two active drugs should be given.

REFERENCES

1. Palenque E. Skin disease and nontubercolous atypical mycobacteria. Int J Dermatol 2000; 39: 659–666.
2. Nathan DL, Singh S, Kestenbaum TM, Casparian JM. Cutaneous Mycobacterium chelonae in a liver transplant patient. J Am Acad Dermatol 2000; 43: 333–366.
3. Hautmann G, Katsambas A, Lotti T. Non-tuberculous mycobacterial skin infections. J Eur Dermatol Venereol 1997; 9: 1–35.
4. Schlossberg M. Tubercolosis and non tubercolous. IV Ed 1999.
5. Wallace RJ, Brown BA, Onyi GO. Skin, soft tissue, and bone infections due to Mycobacterium chelonei. J Infect Dis 1992; 166: 405–412.
6. Herrero JB, Borbujo C, Roman MT, Serrano O, deLucas R, Borbujo J, et al. Cutaneous infection caused by Mycobacterium chelonae. Presentation of three cases of multiple lesions. Rev Clin Exp 1996; 196: 606–609.
7. Fenske NA, Millns JL. Resistant cutaneous infection caused by Mycobacterium chelonei. Arch Dermatol 1981; 117: 152–153.
8. Kelley LC, Deering KC, Kaye ET Cutaneous Mycobacterium chelonei presenting in an immunocompetent host: case report and review of the literature. Cutis 1995; 56: 293–294.
9. Saluja A, Peters NT, Lowe L, Johnson TM. A surgical wound infection due to Mycobacterium chelonae successfully treated with clarithromycin. Dermatol Surg 1997; 23: 539–543.
10. Valencia IC, Weiss E, Sukenik E, Kerdel FA. Disseminated cutaneous Mycobacterium chelonae infection after injection of bovine embryonic cells. Int J Dermatol 1999; 38: 769–783.
11. Weitzul S, Eichhorn PJ, Pandya AG. Non-tuberculous mycobacterial infections of the skin. Current Therapy 2000; 18: 359–377.
12. Talenti A, Marchesi F, Bolz M, Bally F, Bottger EC, Bodmer T. Rapid identification of Mycobacteria to the species level by polymerase chain reaction and restriction enzyme analysis. J Clin Microbiol 1993; 31: 175–178.
13. Bordet AL, Machet L, De Muret A, Francois-Ramanantsoa C. Mycobacterium chelonae cutaneous infection: efficacy of prolonged treatment by clarithromycin. Ann Dermatol Venereol 1997; 124: 251–253.
14. Franck N, Cabiè A, Villette B, Amor B, Lessana-Leibowitch M, Escande JP. Treatment of Mycobacterium chelonae-induced skin infection with clarithromycin. J Am Acad Dermatol 1993; 28: 1019–1021.
15. Driscoll MS, Pharm D, Tyring SK. Development of resistance to clarithromycin after treatment of cutaneous Mycobacterium chelonae infection. J Am Acad Dermatol 1997; 36: 495–496.
16. Banuls J, Ramon R, Pascual E, Navas J. Mycobacterium chelonae infection resistant to clarithromycin in a patient with dermatomyositis. Br J Dermatol 2000; 143: 1345.