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The Foot 15 (2005) 104–106
www.elsevierhealth.com/journals/foot

Case report

Mycobacterium chelonae as a cause of forefoot infections

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Abstract

A 33-year-old lady presented to our unit after multiple failed debridements of a foot ulcer developing after wading in stagnant water. Despite culturing polymicrobial flora and receiving long-term antibiotics the ulcer failed to improve. After presenting to our unit, all antibiotics were stopped for 7 days and several deep samples were taken which grew avid fast bacilli in several samples. *Mycobacterium chelonae* was cultured. Appropriate antibiotics were given resulting in complete healing of the ulcer.

Discussion: Chronic foot ulceration is usually associated with the presence of underlying vascular insufficiency or neuropathic disturbance. The presence of a persistent ulcer and the absence of these risks should alert one to initiate a search for alternative culprits. Atypical mycobacteria are ubiquitous. They have been recovered from water, soil, milk and food products. Risk factors for atypical bacterial infections should be ascertained and should involve a comprehensive review of their occupational and recreational activities in particular any history of penetrating injury or regular exposure to fresh water or seawater. Successful treatment involves suspecting and identifying the organism and treating with appropriate antibiotics.

Keywords: Infection; Mycobacterium

1. Case history

A 33-year-old woman was referred to our specialist unit for investigation of a persistent foot ulcer. The ulcer had appeared 18 months prior to this and involved the sole of the left foot. Shortly before the appearance of the ulcer, she had developed several verrucas on that foot and had received topical treatment. She was unemployed and lived on a boathouse on a local canal. Several months earlier, the canal towpath had flooded and for several weeks, she recounted a history of wading in stagnant canal water. She also recounted a history of a gunshot wound some 6 years earlier and had undergone minor surgery to remove the pellet from the dorsum of the foot not related to the site of the ulcer. She kept three ferrets and a dog as pets. Her medical history was unremarkable and she did not have diabetes and was not immunosuppressed. No risks for HIV were elicited.

Clinical examination revealed a shallow ulcer on the forefoot measuring 3 cm × 4 cm approximately. There were several verrucas present adjacent to the ulcer. She had protective sensation (assessed using a Semmes-Weinstein 10 g monofilament) and a normal vascular examination. Her inflammatory markers were normal including CRP, ESR and WCC. Repeated radiographs and MR imaging of the foot revealed no evidence of residual foreign body in the foot but revealed changes consistent with the diagnosis of osteomyelitis. Histology showed changes consistent with acute and chronic infection with the presence of macrophages and some giant cells.

She had undergone repeated surgical debridements in an attempt to eradicate the infection and repeated courses of antibiotics had failed to control the disease. The culture results from the initial debridements yielded polymicrobial flora including *Streptococcus constellatus*, *Staphylococcus aureus*, *Streptococcus viridans*, *coliforms*, *anaerobes*, *Streptococcus milleri* and group B *Streptococcus*. Cultures from the later debridements yielded multi-resistant organisms including *Enterobacter aerogenes*, *Morganella morganii*, *Proteus mirabilis* and *multi-resistant aeromonas*.

She received repeated courses of targeted antimicrobial treatment, which at one point was complicated by a moderately severe reaction to teicoplanin, when she experienced hy-

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0958-2592/$ – see front matter © 2005 Published by Elsevier Ltd.
doi:10.1016/j.foot.2005.01.001

perpyrexia, tachycardia hypotension and a generalised maculopapular rash. Despite treatment however, the ulcer persisted.

According to our standard protocol, all antibiotics were stopped 7 days prior to a further debridement to achieve reliable sampling. She also received orthotic advice on appropriate off-loading of the ulcer. Multiple deep samples of bone and soft tissue were sent for histological and microbiological analysis, which included fungal and mycobacterial culture. Several samples revealed the presence of acid-fast bacilli and subsequent cultures confirmed the presence of *M. chelonae*.

She was initially commenced on rifampicin, clarithromycin and ethambutol and later continued on clarithromycin and ciprofloxacin as guided by susceptibilities. She has currently completed 9 months of treatment and the wound has healed well with no evidence of recurrence. We plan to continue treatment for a further 3 months. She has tolerated her treatment without adverse effects.

2. Discussion

Chronic foot ulceration is usually associated with the presence of underlying vascular insufficiency or neuropathic disturbance. The presence of a persistent ulcer in the absence of these risks should alert one to initiate a search for alternative culprits.. There have been several descriptions of musculoskeletal and cutaneous infections caused by mycobacterium chelonae in immunocompromised hosts [3–6], and infections with other atypical mycobacteria are well described [7–11]. However, osteomyelitis caused by *M. chelonae* has only been rarely described in immunocompetent hosts [1,2].

Atypical mycobacteria are ubiquitous. They have been recovered from water, soil, milk and food products [12,13]. It is recognized that they colonise body surfaces and secretions without causing disease. Water is the likeliest source and the classical “fish tank” or “swimming pool” granuloma caused by *Mycobacterium marinum* is frequently described in fish tank handlers or those with regular exposure to freshwater or seawater [14]. Nosocomial outbreaks of skin/soft tissue infections have also been described. These include associations with intravenous or peritoneal catheters, post injection abscesses and surgical site infections [15,16,17]. The presence of penetrating trauma, i.e. standing on a nail, is an important risk factor [2].

The extrapulmonary manifestations of these infections are protean. They may present as solitary or multiple papules usually affecting the extremities which may progress to shallow ulceration. Nodular lesions may be present and follow a lymphatic distribution, in a similar fashion to sporotrichosis. However, unlike sporotrichosis, regional lymphadenopathy is infrequently described. They have been described as causing disease in tendon sheaths, bursae, joints and bone usually as a result of direct inoculation. Abscess formation with or without fistulae is also well described.

Over 100 species of mycobacterium have now been described [18]. All mycobacterial species share one characteristic – the presence of “acid-fastness” – the ability to withstand decolorisation after appropriate staining, i.e. with carbol fuchsin or auramine-rhodamine. Although insensitive, the presence of AFB (acid fast bacilli) is virtually synonymous with mycobacteria, the main exception being nocardia—which is partially acid-fast.

Culture remains the cornerstone of identification and is critical for confirming susceptibility patterns. Most mycobacteria grow at 35–37 °C however; those responsible for cutaneous infections tend to grow at lower temperatures 28–34 °C, including *M. chelonae*, *Mycobacterium haemophilum*, *M. marinum*. It is essential that the laboratory be informed of the clinical suspicion so that these specialized requirements can be initiated. Histological analysis may confirm the presence of a granulomatous process.

There is lack of consensus on treatment of these infections, which is therefore guided by anecdotal and observational evidence. Expert committees including the American Thoracic and British Thoracic Society have proposed guidelines however; these concentrate primarily on *M. kansasi* infection and pulmonary disease rather than esoteric muskuloskeletal infections such as this case. To date, there have been no randomized trials on optimal treatment and duration. *M. chelonae* is not susceptible to anti-tuberculosis agents and a quinolone is usually recommended sometimes with an aminoglycoside or imipenem. A combination of other agents has been used including cotrimoxazole, macrolides, tetracyclines, rifampicin and ethambutol [19]. Later on, the treatment should be guided by culture and clinical response.

We think this case highlights several important points in dealing with seemingly intractable muskuloskeletal infection. Risk factors for atypical mycobacterial infections should be ascertained and should involve a comprehensive review of their occupational and recreational activities in particular any history of penetrating injury or regular exposure to freshwater or seawater. In our case, we speculate that the patient’s barefoot exposure to canal water was the source of the pathogen. An occupational history is important as fish handlers are more likely to develop infections.

Risks for immunosuppression should be elicited. Surgical debridement should be carried out only after cessation of antibiotics for at least 7 days in order to obtain high quality specimens. These samples should be sent to histopathology and microbiology for microscopy, culture and for auramine or other appropriate staining to identify acid-fast bacilli. The laboratory should be alerted to the case, as special incubation requirements may need to be arranged. Treatment of these cases should be guided by culture results with appropriate surgical debridement; however the optimal duration of therapy remains controversial ranging from 6 months to 2 years depending on the extent of debridement undertaken and clinical response. Further randomized, placebo controlled and blinded trials need to be performed to determine the optimal treatment for these rare infections.

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