CASE REPORT

Mycobacterium chelonae hand infection after steroid injection in a patient with rheumatoid arthritis receiving long-term linezolid therapyJens Steen Olesen,¹ Mikala Wang,² Christian Wejse^{1,3}¹Department of Infectious Diseases, Aarhus University Hospital, Aarhus N, Denmark²Department of Microbiology, Aarhus University Hospital, Aarhus N, Denmark³Department of Public Health, Center for Global Health, Aarhus University, Aarhus, Denmark**Correspondence to**
Professor Christian Wejse,
wejse@dadlnet.dk

Accepted 14 January 2017

SUMMARY

A 66-year-old multimorbid man with rheumatoid arthritis developed an infection after a steroid injection in the hand. *Mycobacterium chelonae* was cultured 1-month after presentation. In the mean time, his third finger had been amputated. Further treatment was based on preliminary susceptibility testing and the American Thoracic Society guidelines. No regression of the infection was observed before the addition of linezolid (600 mg×1/day) to a combination antimicrobial therapy also consisting of clarithromycin (500 mg×2/day) and moxifloxacin (400 mg×1/day), even though two methods of susceptibility testing, the E-test and broth microdilution, had shown susceptibility to other antimicrobial drugs. The healing was complete 12 months after presentation. There were no serious side effects observed with the use of linezolid in reduced dosage of 600 mg×1/day for a duration of 9 months.

BACKGROUND

Mycobacterium chelonae is a rapid growing, nontuberculous mycobacterium. *M. chelonae* and *M. abscessus* were formerly considered the same organism, but in 1992 they were reclassified as independent species.^{1,2} The incidence of infections caused by nontuberculous mycobacteria has increased significantly,^{3–5} while the incidence of *M. chelonae* remains unknown.

M. chelonae is ubiquitous in the environment and has been isolated from soil, water and human sources.^{6–9} *M. chelonae* can be acquired from environmental exposure by inhalation of aerosols or traumatic inoculation. *M. chelonae* has no pathognomonic findings. This makes diagnosis challenging and necessitates cooperation between clinicians and microbiologists. The clinical presentations are skin, bone and soft tissue diseases. Disseminated disease has been described in immunocompromised individuals.¹⁰ The treatment of infections with *M. chelonae* is difficult due to its resistance to most antimicrobial agents.

CASE PRESENTATION

A 66-year-old man with rheumatoid arthritis, leukocytoclastic vasculitis, diabetes mellitus, hypertension and ischaemic heart disease presented with a red and painful swelling between the metacarpophalangeal joints of the second and third finger on the left hand. He did not report any previous trauma and there was no sign of infection, for example, fluctuation. An ultrasonography revealed

tenosynovitis of the flexor tendons of the fingers as a complication to rheumatoid arthritis. He was treated with a steroid injection in the peritendineum by a rheumatologist.

The patient returned 1 week later with severe pain for which he needed opioids. The patient had elevated C reactive protein and leucocytosis, but no fever. Six months prior to presentation he had a similar presentation of an abscess on the right foot. Treatment with methotrexate had been paused and he was treated for rheumatoid arthritis with prednisolone (40 mg/day). The abscess on the foot was treated successfully with incision and dicloxacillin therapy on the suspicion of infection with *Staphylococcus aureus*. As no culture of pus was submitted, it cannot be ruled out to have been an infection with mycobacteria. However, as the wound healed with no complications, it seems unlikely to be correlated with the presented hand infection.

On suspicion of purulent tenosynovitis on the left hand, he was treated with dicloxacillin (1 g×3/day) and benzylpenicillin (1.2 g×3/day), and referred to surgical treatment with opening of the tendon capsule of the third finger. The necrotic tissue was removed from the proximal phalanx to the mid-palmar space. Pus was submitted for culture. The carpal tunnel was opened due to severe pain, but no pus was found. Two days after surgery, the third finger had lost vitality and was amputated. The wound was left open to heal with the intention of secondary closure.

INVESTIGATIONS

Culture of the amputated necrotic finger tissue revealed growth of acid-fast bacilli after 4 days and the isolate was identified as *M. chelonae* by partial sequencing of the *rpoB* gene by the local microbiology laboratory. The isolate was referred to the International Reference Laboratory for Mycobacteriology (IRLM), Statens Serum Institut for antimicrobial susceptibility testing.

After a week of treatment, preliminary susceptibility testing of the isolate by E-test showed susceptibility to moxifloxacin, clarithromycin, tobramycin, amikacin, imipenem and linezolid and resistance to ciprofloxacin and tetracycline.

At the IRLM, susceptibility testing by broth micro dilution was performed on the isolate, showing clarithromycin 0.25 mg/L (S), doxycycline 16 (R), ciprofloxacin 1 (S), sulfamethoxazole-trimethoprim 4/76 (R), moxifloxacin 2 (I), cefoxitin

CrossMark

To cite: Olesen JS, Wang M, Wejse C. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2016-217257

128 (R), amikacin 8 (S), tobramycin 2 (S), minocycline 8 (R), tigecycline 0,06 (S), linezolid 4 (S), imipenem 8 (I).

DIFFERENTIAL DIAGNOSIS

S. aureus is naturally occurring on the skin and can cause infection after entering small wounds on the hand. However, infection with *S. aureus* has an even more aggressive course than was seen in the presented case. *S. aureus* is known to cause abscesses, which can spread to almost every organ in the body, including soft tissue and bone. Most *S. aureus* isolates in Denmark are susceptible to dicloxacillin, the primary antibiotic drug used in the presented case. No culture of *S. aureus* was reported, making it an unlikely cause of the infection.

Nocardia is found in soil, fresh and salt water like *M. chelonae*.¹¹ The most common presentation is pulmonary nocardiosis after inhalation of the bacteria. However, primary cutaneous nocardiosis may occur due to traumatic inoculation, especially in immunocompromised individuals. Symptoms of *Nocardia* infection include skin infection, ulcerations, cellulitis and subcutaneous abscesses making it difficult to distinguish from other pyogenic bacteria. *Nocardia* are weakly acid-fast bacteria and can be visualised by the modified Kinyoun acid-fast stain comparable to the Ziehl-Neelsen stain used to visualise mycobacteria. *Nocardia* was not cultured from the wound.

Erysipelotrix rhusiopathiae is a bacterium found in several animals.¹² Infection in humans are most often related to occupational exposure, but have also been reported after dog bites. Infection with *E. rhusiopathiae* presents with swelling and violaceous discolouring, which is followed by central clearing as the infection spreads. As it usually resolves without treatment and is highly susceptible to penicillin, it seems to be an unlikely cause of the infection in the presented case.

Francisella tularensis is a bacterium that is transmitted to human through tick bites, handling of infected animals or contact with contaminated water.¹³ The bacterium is very infectious and may cause skin ulcers and lymphadenopathy. Infection with *F. tularensis* is accompanied by abrupt fever and other influenza-like symptoms, which was not reported in the presented case.

Fungi, including *Aspergillus*, *Cryptococcus*, *Scedosporium apiospermum*, *Pseudallescheria boydii* and *Sporothrix schenckii*, are other differential diagnoses in the presented case.^{14–18} Fungi are found in fresh and salt water, sewage, soil and in animals such as poultry and cattle. Though differences in clinical

presentation exist, they also cause pulmonary disease after inhalation or primary cutaneous disease after traumatic inoculation. Infection most often occurs in immunocompromised individuals, for example, after long-term therapy with steroid.

The comorbidity may have caused the poor healing response due to a reduced tissue perfusion. Based on the culture of *M. chelonae* and the healing of the wound after introduction of a combination therapy of antibiotics, it seems the comorbidity was only a contributing factor to the disease and not a cause.

TREATMENT

After amputation of the third finger, the patient was referred to the Department of Infectious Diseases for further antibiotic treatment (table 1).

The combination antimicrobial therapy consisting of clarithromycin, moxifloxacin and linezolid was continued for a total of 9 months. The erythrocyte sedimentation rate in mm and figures illustrates the clinical evolution during the period.

The wounds were not healing and were filled with pus and necrotic tissue (figure 1A, B). When *M. chelonae* was isolated, dicloxacillin and benzylpenicillin treatment was discontinued and he was treated with a three-drug combination therapy of doxycycline (100 mg×2/day), clarithromycin (500 mg×2/day) and ciprofloxacin (500 mg×2/day) based on preliminary susceptibility testing and the American Thoracic Society (ATS) guidelines.¹⁰ Treatment for 6 months was planned due to bone involvement.¹⁰

After a susceptibility testing was performed at IRLM, further treatment was planned with 2 months of tobramycin (240 mg/day) and 6 months of moxifloxacin (400 mg×1/day) and clarithromycin (500 mg×2/day). However, after 1 month of treatment, the patient still had elevated C reactive protein and poor healing of the wound. Negative pressure wound therapy was initiated as a supplement.

Owing to the poor healing response, a change in the antibiotic regimen was made. In order to avoid the potential neurotoxic effects and myelosuppression of linezolid, the patient was treated with amikacin (750 mg×1/day), sulfamethoxazole-trimethoprim (2×400 mg/80 mg per day) and clarithromycin (500 mg×2/day). However, after a few days, there was a severe renal impairment which led to discontinuation of amikacin and sulfamethoxazole-trimethoprim.

Three months after presentation it was decided to resume moxifloxacin treatment despite intermediary susceptibility to

Table 1 Fifty-two weeks of antimicrobial therapy for the treatment of *Mycobacterium chelonae*

Antibiotics/week	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	51	52
Sedimentation rate	69					36									14	7	4
Figures					1a		1b									2	
Benzylpenicillin																	
Dicloxacillin																	
Doxycycline																	
Ciprofloxacin																	
Clarithromycin																	
Tobramycin																	
Moxifloxacin																	
Amikacin																	
Sulfamethoxazole-Trimethoprim																	
Linezolid																	

The combination antimicrobial therapy consisting of clarithromycin, moxifloxacin and linezolid was continued for a total of 9 months. The erythrocyte sedimentation rate in mm and figures illustrates the clinical evolution during the period.

Figure 1 (A) Prior to antimicrobial treatment at the department of infectious diseases. (B) After 2 weeks of antimicrobial treatment at the department of infectious diseases.

Figure 2 After 5 months of antimicrobial treatment at the department of infectious diseases.

this antimicrobial agent, as in vitro susceptibility testing of nontuberculous mycobacteria does not correlate well with clinical response.¹⁹ Linezolid was also given in reduced dose (600 mg×1/day). The wound began to heal and there was no sign of infection (figure 2) 2 months after the introduction of linezolid to the combination therapy.

Treatment was given for a total of 12 months, due to multiple risk factors for recurrence. In 9 of 12 months, the patient was treated with a long-term combination therapy of clarithromycin (500 mg×2/day), moxifloxacin (400 mg×1/day) and linezolid (600 mg×1/day). Oral candidiasis and distorted taste emerged after initiating linezolid treatment, but no other form of neurotoxicity or myelosuppression was reported.

OUTCOME AND FOLLOW-UP

After 12 months of treatment the healing was complete and the patient had reconstructive surgery of the hand as well as arthroplastic surgery of the knee. In both cases, there was an excellent

Figure 3 Six months after completion of combination antimicrobial therapy.

wound healing. Six months after cessation of antibiotic treatment, there was no sign of recurrence (figure 3).

The patient, however, died 1 year after completing treatment. Progressive congestive heart failure was observed in the months prior to his death, and he was admitted to the emergency ward and most likely died from pulmonary oedema. No autopsy was performed according to the wishes of the family.

DISCUSSION

M. chelonae infection is a serious and debilitating disease which can be resistant to treatment, but long-term linezolid, clarithromycin and moxifloxacin therapy was effective in the case presented here.

The genus *Mycobacterium* currently consists of 175 species,²⁰ excluding subspecies, which can be classified into four groups based on biochemical, growth and pigmentation characteristics:

1. *Mycobacterium tuberculosis* complex
2. *Mycobacterium leprae*
3. Slowly growing nontuberculous mycobacteria
4. Rapidly growing nontuberculous mycobacteria

The group of rapidly growing nontuberculous mycobacteria include *M. fortuitum*, *M. abscessus* and *M. chelonae* among others. *M. abscessus* was described in 1953 as synonymous to *M. chelonae* or as a subspecies within the *M. chelonae-abscessus* group.² *M. abscessus* was first acknowledged as an independent species in 1992. *M. chelonae* and *M. abscessus* have similar clinical presentations, are phenotypically and biochemically alike and are indistinguishable by 16S rDNA sequencing.²¹ *M. abscessus* has for these reasons previously been mistaken for *M. chelonae*.² The differentiation between the two species is of critical importance, as *M. abscessus* is intrinsically resistant to most antibiotics, while *M. chelonae* is susceptible to a wider range of antimicrobial agents. Rapidly growing mycobacteria, for example, *M. abscessus*, *M. fortuitum* and *M. chelonae*, often show susceptibility to macrolides according to Clinical and Laboratory Standards Institute procedures.²² However, induction of resistance to macrolides may occur as a result of rRNA methylation caused by the *erm* gene, located in *M. abscessus* and *M. fortuitum*, but not in *M. chelonae*.²³ Thus, monotherapy with macrolides against rapidly growing mycobacteria is not recommended without differentiation. The two species, *M. abscessus* and *M. chelonae*, are now differentiated by partial sequencing of the RNA Polymerase B (*rpoB*) gene making identification definitive.²¹

Among rapidly growing mycobacteria, *M. fortuitum* is the most common. While *M. fortuitum* causes infection primarily

by direct inoculation, *M. chelonae* often causes infection in immunosuppressed patients. *M. chelonae* infection should be considered in patients with chronic soft tissue infections non-responsive to commonly used antimicrobial therapies.²⁴ The source of *M. chelonae* infection is usually not identified due to the ubiquitous presence in the environment. A possible route of transmission could be everyday life activities, as the patient was both gardening and walking his dogs. *M. chelonae* has also been found in tap water and on medical instruments.^{7 25–27} It has been known to cause infection after healthcare procedures, such as filler injection, acupuncture and liposuction.^{28–30} Only sporadic cases of infection occur because of the low pathogenic potential of *M. chelonae*. Usually a preceding trauma is necessary to develop disease. Sterile technique during healthcare procedures is therefore important to prevent infections with nontuberculous mycobacteria. We suspect the most likely route of transmission in this case is traumatic inoculation following the steroid injection in the peritendineum for the treatment of tenosynovitis, even though the procedure was performed under sterile conditions. Contamination of the steroid ampoule may have been a possible route of transmission, but was not suspected at the time. As the lot number of the ampoule was not registered, the authorities were not notified. Furthermore, the most likely source of infection was due to the skin flora.

Patients with rheumatoid arthritis are known to have an increased risk of infection.³¹ The increased risk seems to be associated with both the disease itself and the immunosuppressive medications used to treat it. Even before the use of steroid, increased susceptibility to infection due to alterations of the immune system was reported in rheumatoid arthritis.³² Prednisolone most likely also increases the risk of infection.^{32 33} Some studies however do not find an association between prednisolone and increased risk of infection.³² Clinicians need to be vigilant for infections in patients treated with prednisolone, even though studies may be inconclusive. In a review of 100 skin, bone or soft tissue infections with *M. chelonae*, more than half of the patients were receiving prednisolone. Almost everybody, 49 of 53, with disseminated disease were receiving prednisolone, although in low doses of <20 mg/day.³³ The multiple and resistant infections of the patient in this case while receiving prednisolone therapy, support the possibility of rheumatoid arthritis and/or prednisolone treatment as risk factors for infections, including *M. chelonae* infection.

M. chelonae is considered to be among the most drug-resistant nontuberculous mycobacteria as it is resistant to most antibiotics, for example, all antituberculous drugs.³⁴ It is therefore necessary to test for antimicrobial susceptibility on all isolates of *M. chelonae*.³⁵ According to ATS guidelines, *M. chelonae* is susceptible to clarithromycin (100%) and tobramycin (100%), linezolid (90%), amikacin (50%), doxycycline (25%) and ciprofloxacin (20%).⁶ Clarithromycin is the drug of choice.^{24 36–38} A combination antimicrobial therapy is however recommended to avoid development of mutational resistance. Moxifloxacin has proven to be a successful supplement to clarithromycin in this case as well as in other case reports.²⁴ A minimum of 4 months is necessary to treat skin or soft tissue infection. It is recommended to treat for a minimum duration of 6 months in case of bone involvement.¹⁰ Surgery is often necessary as antimicrobial therapy is difficult.

The toxicity profile of linezolid given for more than 28 days is more or less unknown. Monitoring of blood count is required due to risk of myelosuppression. Similar cases have reported successful treatment with combination therapy of clarithromycin and linezolid, even though most cases only reported a few months of treatment with linezolid.^{37 38} No myelosuppression was observed in several case reports during treatment with

linezolid in reduced doses of 600 mg×1/day.^{37 39} The only observed side effect in this case was distorted taste, despite treatment with linezolid of 600 mg×1/day for 9 months. This case report demonstrates that long-term treatment with linezolid may be tolerated by some patients.

A case of severe *M. chelonae* infection in an immunosuppressed patient is presented. Amputation was imminent, but avoided through addition of linezolid (600 mg×1/day) to the treatment regime, also consisting of clarithromycin and moxifloxacin, and was well tolerated despite severe comorbidity.

Learning points

- ▶ Rheumatoid arthritis and prednisolone may be risk factors for nontuberculous mycobacteria (NTM) infections.
- ▶ Prolonged infections where the most commonly used antimicrobial drugs are ineffective may be caused by NTM, particularly in immunocompromised individuals.
- ▶ Clarithromycin is the first drug of choice in combination with two other antimicrobial drugs to prevent developing of resistance. Both moxifloxacin and linezolid were shown to be successful in treatment of *Mycobacterium chelonae* osteomyelitis.
- ▶ The use of linezolid may not be restricted to short-term treatment in the future as reduced dosage of 600 mg×1/day had no serious side effects in this and several other case reports.

Contributors CW treated the patient, MW handled the laboratory analyses and discovered that it was a *Mycobacterium chelonae* infection. JSO wrote the first draft of the manuscript and CW and MW contributed to the final version.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Levy-Frevault V, Grimont F, Grimont P, *et al.* Deoxyribonucleic acid relatedness study of the mycobacterium fortuitum-mycobacterium chelonae complex. *Int J Syst Bacteriol* 1986;36:458–60.
- 2 Kusunoki S, Ezaki T. Proposal of *Mycobacterium peregrinum* sp. nov., nom. rev., and elevation of *Mycobacterium chelonae* subsp. abscessus (Kubica *et al.*) to species status- *Mycobacterium abscessus* comb. nov. *Int J Syst Bacteriol* 1992;42:240–5.
- 3 Menzies D, Nahid P. Update in tuberculosis and nontuberculous mycobacterial disease 2012. *Am J Respir Crit Care Med* 2013;188:923–7.
- 4 Marras TK, Chedore P, Ying AM, *et al.* Isolation prevalence of pulmonary nontuberculous mycobacteria in Ontario in 1997–2003. *Thorax* 2007;62:661–6.
- 5 Lai CC, Tan CK, Chou CH, *et al.* Increasing incidence of nontuberculous mycobacteria, Taiwan, 2000–2008. *Emerg Infect Dis* 2010;16:294–6.
- 6 Khan IUH, Selvaraju SB, Yadav JS. Occurrence and characterization of multiple novel genotypes of *Mycobacterium immunogenium* and *Mycobacterium chelonae* in metalworking fluids. *FEMS Microbiol Ecol* 2005;54:329–38.
- 7 Santos R, Oliveira F, Fernandes J. Detection and identification of mycobacteria in the Lisbon water distribution system. *Water Sci Technol* 2005;52:177–80.
- 8 Vaerewijck MJM, Huys G, Palomino JC, *et al.* Mycobacteria in drinking water distribution systems: ecology and significance for human health. *FEMS Microbiol Rev* 2005;29:911–34.
- 9 Lin SS, Lee CC, Jang TN. Soft tissue infection caused by rapid growing mycobacterium following medical procedures: two case reports and literature review. *Ann Dermatol* 2014;26:236–40.
- 10 Griffith DE, Aksamit T, Brown-Elliott BA, *et al.* An official ATS/IDSA statement: diagnosis, treatment, and prevention of nontuberculous mycobacterial diseases. *Am J Respir Crit Care Med* 2007;175:367–416.
- 11 Wilson JW. Nocardiosis: updates and clinical overview. *Mayo Clin Proc* 2012;87:403–7.

- 12 Reboli AC, Farrar WE. Erysipelothrix rhusiopathiae: an occupational pathogen. *Clin Microbiol Rev* 1989;2:354–9.
- 13 Steiner DJ, Furuya Y, Metzger DW. Host-pathogen interactions and immune evasion strategies in Francisella tularensis pathogenicity. *Infect Drug Resist* 2014;7:239–51.
- 14 van Burik O-AH, Colven R, Spach DH. Cutaneous aspergillosis. *J Clin Microbiol* 1998;36:3115–21.
- 15 Neuville S, Dromer F, Morin O, et al. Primary cutaneous cryptococcosis: a distinct clinical entity. *Clin Infect Dis* 2003;36:337–47.
- 16 Cortez KJ, Roilides E, Quiroz-Telles F, et al. Infections caused by Scedosporium spp. *Clin Microbiol Rev* 2008;21:157–97.
- 17 Ishii S, Hiruma M, Hayakawa Y, et al. Cutaneous pseudallescheria boydii/scedosporium apiospermum complex (Molecular type: Scedosporium apiospermum [Clade 4]) infection: a case report and literature review of cases from Japan. *Japanese J Med Mycol* 2015;56:E25–30.
- 18 Mahajan VK. Sporotrichosis: an overview and therapeutic options. *Dermatol Res Pract* 2014;2014:272376.
- 19 Heginbotham ML. The relationship between the in vitro drug susceptibility of opportunist mycobacteria and their in vivo response to treatment. *Int J Tuberc Lung Dis* 2001;5:539–45.
- 20 List of Prokaryotic names with Standing in Nomenclature. Genus Mycobacterium. 2016. <http://www.bacterio.net/mycobacterium.html> (accessed 20 Nov 2016).
- 21 Arnold C, Barrett A, Cross L, et al. The use of rpoB sequence analysis in the differentiation of Mycobacterium abscessus and Mycobacterium chelonae: a critical judgement in cystic fibrosis?. *Clin Microbiol Infect* 2012;18:E131–3.
- 22 Clinical and Laboratory Standards Institute M24-A2. *Susceptibility testing of mycobacteria, Nocardiae, and other aerobic actinomycetes*. 2nd edn. PA: Wayne, 2011.
- 23 Nash KA, Brown-Elliott BA, Wallace RJ. A Novel gene, erm(41), confers inducible macrolide resistance to clinical isolates of mycobacterium abscessus but is absent from mycobacterium chelonae. *Antimicrob Agents Chemother* 2009;53:1367–76.
- 24 Cho SY, Peck KR, Kim J. Mycobacterium chelonae infections associated with bee venom acupuncture. *Clin Infect Dis* 2014;58:e110–113.
- 25 Dubrou S, Konjek J, Macheras E, et al. Diversity, community composition, and dynamics of nonpigmented and late-pigmenting rapidly growing mycobacteria in an urban tap water production and distribution system. *Appl Environ Microbiol* 2013;79:5498–508.
- 26 Chroniou A, Zimmerman SK, Cook S. Molecular typing of Mycobacterium chelonae isolates from a pseudo-outbreak involving an automated bronchoscope washer. *Infect Control Hosp Epidemiol* 2008;29:1088–90.
- 27 Negayama K, Terada S, Kiuchi H. Contamination of endoscopes and endoscope washers by atypical mycobacteria. *Kansenshogaku Zasshi* 1992;66:349–53.
- 28 Latorre-González G, García-García M, Martínez Martínez-Colubi M. [Disseminated cutaneous infection by Mycobacterium chelonae after botulinic toxin injection in an immunosuppressed patient]. *Med Clin* 2005;125:439.
- 29 Giannella M, Pistella E, Perciaccante A. Soft tissue infection caused by Mycobacterium chelonae following a liposculpture and lipofilling procedure. *Ann Ital Med Int* 2005;20:245–7.
- 30 Lee WJ, Kang SM, Sung H. Non-tuberculous mycobacterial infections of the skin: a retrospective study of 29 cases. *J Dermatol* 2010;37:965–72.
- 31 Listing J, Gerhold K, Zink A. The risk of infections associated with rheumatoid arthritis, with its comorbidity and treatment. *Rheumatology(Oxford)* 2013;52:53–61.
- 32 Baum J. Infection in rheumatoid arthritis. *Arthritis Rheum* 1971;14:135–7. <http://www.ncbi.nlm.nih.gov/pubmed/975760> <http://dx.doi.org/10.1002/art.1780140119>
- 33 Wallace RJ, Brown B, Onyi G. Skin, soft tissue, and bone infections due to mycobacterium chelonae chelonae: importance of prior corticosteroid therapy, frequency of disseminated infections, and resistance to oral antimicrobials other than clarithromycin. *J Infect Dis* 1992;166:405–12.
- 34 Strangfeld A, Eveslage M, Schneider M, et al. Treatment benefit or survival of the fittest: what drives the time-dependent decrease in serious infection rates under TNF inhibition and what does this imply for the individual patient?. *Ann Rheum Dis* 2011;70:1914–20.
- 35 García-Agudo L, García-Martos P, Jesús I, et al. Assessment of in vitro susceptibility to antimicrobials of rapidly growing mycobacteria by E-test. *Rev Med Chil* 2009;137:912–17.
- 36 Wallace RJ, Tanner D, Brennan P. Clinical trial of clarithromycin for cutaneous (disseminated) infection due to Mycobacterium chelonae. *Ann Int Med* 1993;119:482–482.
- 37 Brown-Elliott BA, Wallace RJ Jr, Blinkhorn R, et al. Successful treatment of disseminated Mycobacterium chelonae infection with linezolid. *Clin Infect Dis* 2001;33:1433–4.
- 38 Hoetzenecker W, Ulmer A, Klingel K. Dissemination of a localized cutaneous infection with Mycobacterium chelonae under immunosuppressive treatment. *Arch Dermatol* 2007;143:951–2.
- 39 Taguchi K, Miyakawa T, Ohmura T. A reduced linezolid dosage maintains favorable efficacy with minimal hematologic toxicity in a methicillin-resistant Staphylococcus aureus-infected patient with renal insufficiency. *Scand J Infect Dis* 2013;45:77–80.

Copyright 2017 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- Submit as many cases as you like
- Enjoy fast sympathetic peer review and rapid publication of accepted articles
- Access all the published articles
- Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow