Post-Facelift Infection Due to *Mycobacterium Fortuitum*: A Case Report

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Mycobacterium fortuitum complex belongs to a group of rapidly growing mycobacteria (RGM). Very few cases of infections with *M. fortuitum* have been reported after a facelift.

PATIENT DESCRIPTION

A 70 year old retired nurse was referred to the infectious diseases outpatient clinic 2 weeks after a cosmetic facelift procedure, presenting with purulent secretion from the drainage areas that grew *Pseudomonas aeruginosa*, susceptible to ciprofloxacin. Her medical history was significant for diabetes mellitus that was well-controlled with oral medications. Physical examination revealed no systemic signs of infection: no fever, peripheral white blood cell (WBC) count 9380 cells/mm^3, neutrophils 57.7%, erythrocyte sedimentation rate (ESR) 45 mm in the first hour, and C-reactive protein (CRP) 1.2 mg/L.

The patient was treated with ciprofloxacin. Two weeks later, the secretion had decreased, yet there were new areas of swelling with induration and some fluctuation, without signs of cellulitis around the lesions, which had spread bilaterally along the neck and cheeks [Figure 1]. On examination, there were no systemic signs of infection, such as fever. A complete blood count (CBC) was within normal ranges, but there was an increase in CRP (11.6 mg/L).

A thick fluid sample was obtained by needle aspiration from several lesions and sent for bacterial, fungal, and *Mycobacterium* culture, as well as polymerase chain reaction (PCR). All four samples grew *M. fortuitum* and one sample was also PCR positive for the same isolate. By antibiogram, the pathogen was sensitive to amikacin, clarithromycin, ciprofloxacin, doxycycline, and imipenem, and resistant to trimethoprim/sulfamethoxazole, cefoxitin, and tobramycin.

Figure 1. Before treatment

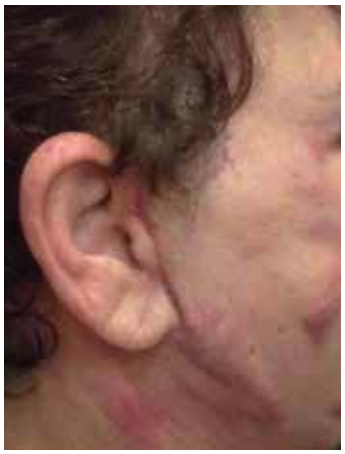


Figure 2. During treatment

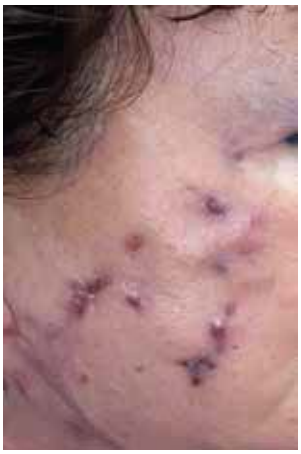
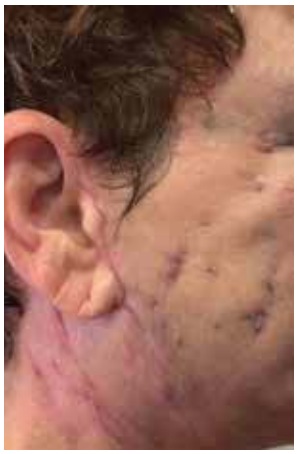


Figure 3. After treatment



COMMENT

Our patient presented with a sporadic case of nontuberculous mycobacteria (NTM) post-surgical infection with no identified source of infection. She was treated with a combination of clarithromycin (minimum inhibitory concentration [MIC] was 1 µg/ml), ciprofloxacin, and doxycycline for 2 months followed by clarithromycin and ciprofloxacin for 6 months of treatment with a clinical, laboratory, and microbiological cure. Samples aspirated from residual lesions after 4 months of treatment were negative for NTM by culture and PCR [Figure 2]. Two months after treatment was completed, there was a clear cosmetic improvement but hyper-pigmented scars were still present [Figure 3].

In recent years, NTM have emerged as important opportunistic pathogens. Among more than 140 species of NTM, the RGM, which include *M. fortuitum* and *M. chelonae*, are clinically significant pathogens that cause a number of human diseases [1,2]. RGM are widely spread and have been isolated from natural water, tap water, soil, dust, rocks, bioaerosols, and more [1,3]. NTM have been isolated from cutaneous and soft tissue infections, after skin injury or minor trauma, and after surgical procedures including plastic surgery [1]. Cases have been described after injections with contaminated solutions, tattooing, piercing, and cardiac surgery [3-5]. Cosmetic surgery has emerged as a significant source of RGM infections. Outbreaks investigated after facial procedures, abdominoplasty, liposuction, liposculpture, breast aug-

mentation and reconstruction, and nipple piercing have all been associated with post-procedure infection caused by RGM [3-5].

In most outbreaks, the source of infection was not identified; however, some cases have been attributed to the contamination of solutions used for skin marking, the presence of an ice machine in the operating room, and whirlpool foot baths in nail salons. In sporadic cases, the source of infection was probably contamination of the wound by tap water, directly or indirectly [3]. All the cases had a delayed onset (10 days to 3 weeks after surgery), with persistent non-healing wounds. Patients had painless lesions with discharging sinuses without fever or other systemic signs of infection, and a lack of response to antibiotics used for pyogenic infections [1].

M. fortuitum is the most common organism isolated (57%) [1], and co-infection with *S. aureus* and *P. aeruginosa* have been documented [3]. Most RGM are sensitive to ciprofloxacin, clarithromycin, amikacin, and tobramycin and are usually resistant to rifampicin and isoniazid. Clarithromycin and a combination of amikacin and doxycycline were found to be effective against NTM *fortuitum-chelonae*. RGM can develop macrolide resistance, thus monotherapy is not recommended, especially when macrolide MIC are in the 4–8 mg/ml range. A combination of two or more drugs provides effective therapy for RGM skin and soft tissue infections, and the mean duration of treatment is 3 to 9 months [1,3] or at least 3 months after cessation of discharge from the infected site [1].

CONCLUSIONS

Clinicians should suspect mycobacterial infection in patients with delayed wound healing, who are unresponsive to standard antibiotic treatment, especially in the absence of systemic signs of infection.

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Capsule

Evidence of alternative modes of B cell activation involving acquired fab regions of *N* glycosylation in antibody-secreting cells infiltrating the labial salivary glands of patients with Sjögren's syndrome

To better understand the role of B cells, the potential mechanisms responsible for their aberrant activation, and the production of autoantibodies in the pathogenesis of Sjögren's syndrome, **Koelsch** and co-authors explored patterns of selection pressure and sites of *N* glycosylation acquired by somatic mutation (ac*N* glyc) in the immunoglobulin G (V) regions of antibody-secreting cells (ASCs) isolated from the minor salivary glands of patients with Sjögren's syndrome and non-Sjögren's syndrome control patients with sicca symptoms. A novel method to produce and characterize recombinant monoclonal antibodies (mAb) from single cell-sorted ASC infiltrates was applied to concurrently probe expressed genes (all heavy- and light-chain isotypes as well as any other gene of interest not related to immunoglobulin) in the labial salivary glands of patients with Sjögren's syndrome and non-Sjögren's syndrome controls. V regions were amplified by reverse transcription-polymerase chain reaction, sequenced, and analyzed for the incidence of *N* glycosylation and selection pressure. For specificity testing, the amplified regions were expressed as either the native mAb or mutant mAb lacking the ac*N* glyc motif. Protein modeling was used to demonstrate how even an ac*N* glyc site outside of the complementarity-determining region could participate in, or inhibit, antigen binding. V-region sequence analyses revealed clonal expansions and evidence of secondary light-chain editing and allelic inclusion, of which neither of the latter two have previously been reported in patients with Sjögren's syndrome. Increased frequencies of ac*N* glyc were found in the sequences from patients with Sjögren's syndrome, and these ac*N* glyc regions were associated with an increased number of replacement mutations and lowered selection pressure. A clonal set of polyreactive mAb with differential framework region 1 ac*N* glyc motifs was also identified, and removal of the ac*N* glyc could nearly abolish binding to autoantigens. These findings support the notion of an alternative mechanism for the selection and proliferation of some autoreactive B cells, involving V-region *N* glycosylation, in patients with Sjögren's syndrome.

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Eitan Israeli

"As we express our gratitude, we must never forget that the highest appreciation is not to utter words, but to live by them"

John Fitzgerald Kennedy, (1917–1963), 35th American President, served from 1961 until his assassination in 1963