The European guideline on the management of NGU recommends performing NAAT for MG and for screening for macrolide resistance,[4,5] despite the fact that these techniques are not available at many sites. At our unit, we test patients with NGU who do not respond to treatment and who have negative NG-CT NAAT results for MG, but we do not screen for macrolide resistance. The high rate of resistance of MG to this antibiotic group has resulted in single-dose azithromycin no longer being recommended for the empirical treatment of NGU in Europe.[4] In patients who show no clinical or microbiological response to a 1 g single dose of oral azithromycin, the guideline recommends assuming that the patient has a macrolide-resistant strain and the recommended regimen would be to treat directly with moxifloxacin,[4] as shown in this case report.

The role of rectal MG as a reservoir and the need to treat asymptomatic rectal MG carriers have been controversial.[6] A recent study in MSM with MG urethritis showed that up to 40% of sexual contacts undergoing a rectal exam were positive for MG.[7] The presence of rectal MG is often asymptomatic and rectal MG load is higher among patients with symptomatic proctitis than among asymptomatic rectal MG carriers.[8] However, the prevalence of oropharyngeal MG is very low and does not appear to constitute a major reservoir.[9]

The European guideline on the management of MG infections recommends examining the sexual contacts of those infected with MG and treating them in the case of positive test results.[4,5] There are no cost-effectiveness studies on the treatment of the sexual contacts of patients with MG infection, although this does seem to be an effective tool for reducing incidence rate. There is also no evidence of aspects such as the natural history of the infection or preventable morbidity with treatment, and therefore screening asymptomatic patients for MG is not currently recommended.[10] In high-risk populations, such as HIV-positive MSM with higher rates of asymptomatic rectal MG carriers,[6] targeted screening could be more cost-effective.

To conclude, cases such as the one observed in our case report support the need for and the importance of screening the sexual partners of patients with MG infection and for treating rectal MG carriers in order to reduce the incidence of MG infections. Furthermore, macrolide resistance testing using molecular test methods at the same as screening would allow targeted treatment.

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Infección parotídea por *Mycobacterium malmoense*

Mycobacterium *malmoense parotid gland infection*

Case report

A 2-year old girl visited the clinic with a right preauricular mass. Prior to this, she was healthy, with no medical history of interest and had received all her immunisations. Her parents are from Ecuador and her father had tuberculosis which was treated correctly 10 years ago. Examination revealed a soft, tender mass in the parotid gland measuring 0.5 cm in diameter and high fever at the onset of symptoms. Viral parotitis was suspected and therefore blood tests were ordered, which were negative. Anti-inflammatory drugs were prescribed for one week. Twenty days after onset, the lesion had increased in size with signs of inflammation. Ultrasound images showed a multilocular lesion measuring 25 mm in diameter in the right parotid gland and lateral cervical and mastoid adenopathies suggestive of an inflammatory process with abscess. Antibiotic therapy with amoxicillin/clavulanic acid was commenced, with no response after 7 days. A broader study was therefore recommended. The Mantoux test was positive (12 mm) at 48 h. A QuantiFERON test was performed, which was negative. An ultrasound-guided fine-needle aspiration (FNA) biopsy was performed and the specimen culture was positive for *Mycobacterium malmoense*. Treatment with isoniazid, rifampicin, ethambutol and clarithromycin was started.

The lesion was confirmed by MR imaging (Figs. 1 and 2). The lesion was drained and its size decreased to 0.3 cm in diameter after three months of treatment. After 9 months of treatment, the lesion was stable and was surgically removed (Figs. 3 and 4).

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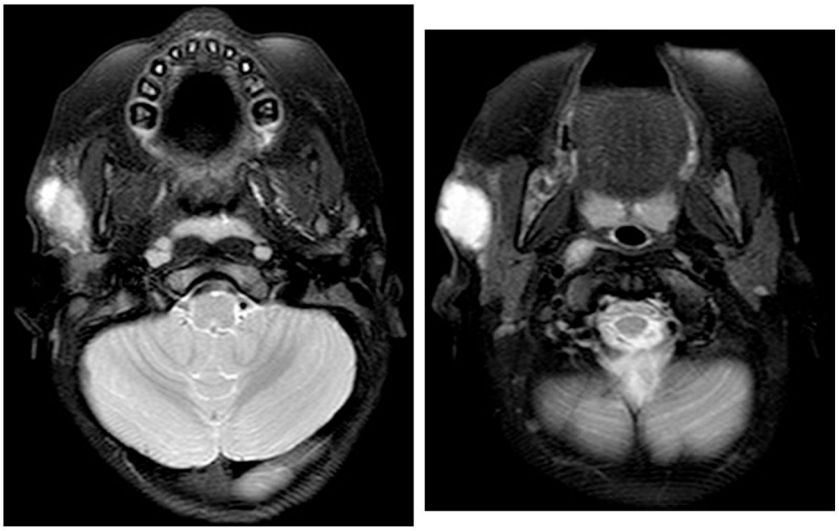
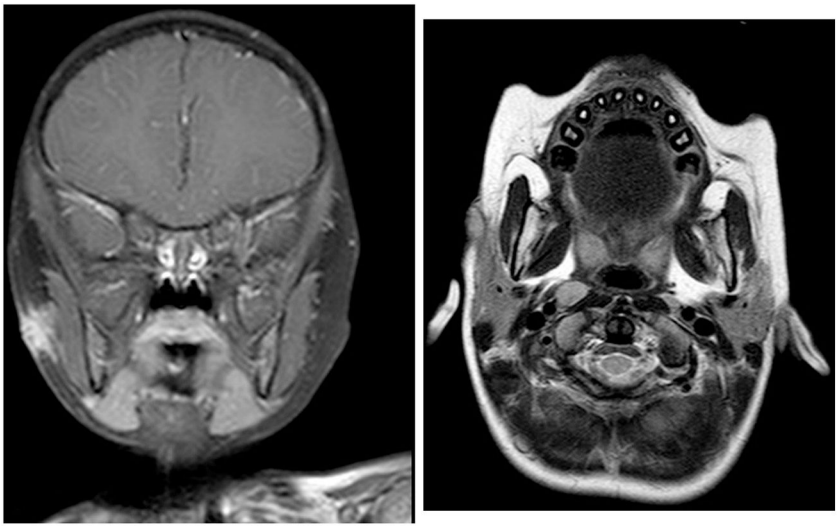


Figs. 1 and 2. T1 and T2-weighted sequences with gadolinium: septated cystic mass in the right parotid gland compatible with an abscess.



Figs. 3 and 4. T1 and T2-sequences with gadolinium: change in signal intensity at the anterior pole of the parotid gland compared to subcutaneous lesion with no clear path of the fistula. Compared to the initial study, there is significant improvement with a reduction in lesion size and signal intensity.

An intraparotid lymph node was resected, showing chronic granulomatous inflammation and parotid parenchyma with interstitial fibrosis. The surgery was completed without complications.

Discussion

Cervical lymphadenitis is the most common manifestation of infection due to non-tuberculous mycobacteria (NTM) in children, affecting children under the age of 5 years with no associated comorbidites.[1–3] Parotid gland involvement is uncommon.

Mycobacterium avium-intracellulare complex is the most prevalent pathogen.[2,4,5] In northern Europe, *M. malmoense* is one of the most prevalent NTM.[6] The mechanism of transmission of NTM remains unclear.[3,4,7]

Lesions tend to be unilateral and non-tender, starting subacute but becoming more chronic, and gradually increase in size with fistula formation, although they may heal spontaneously.[1–3,8] The differential diagnosis must include bacterial and tuberculous lymphadenitis, and, less commonly, parotid tumours.[7]

The diagnosis is confirmed by isolating pathogens from specimens obtained by FNA biopsy or excision.[3,5,7]

Ultrasound imaging is the method of choice; computed tomography (CT) and MRI scans are indicated prior to surgery for large, complicated lesions or to delimit their size.[1,5]

Mantoux skin test reactions are positive (induration $\geq$ 10 mm) in 30–60% of children with NTM, but this is not very useful for establishing a differential diagnosis with *Mycobacterium tuberculosis* (MT).[2,4,5] Nevertheless, interferon gamma release assays (IGRA) are typically negative since most of the NTM involved do not express antigens that induce the tested cellular immune response.[2,4,5]

Identification of NTM using molecular diagnostic techniques allows rapid isolation of the pathogen, with even $\geq$90% sensitivity.[2,3]

Treatment is controversial; there is not enough evidence to prove the superiority of expectant management, drug therapy or surgical intervention.[2,8]

Combination therapy, including a macrolide antibiotic plus rifabutin, fluoroquinolones or ethambutol,[5–8] for a minimum of 6 months is recommended to prevent resistance.

Surgical intervention, which in some studies is considered the treatment of choice,[1,5,8,9] is not exempt from complications: superinfection of the surgical site and neurovascular lesions, such as facial nerve paralysis.[3,7]

Drug therapy before or after surgery may help decrease the size of the excision site, thereby reducing the risk of bilateral brain injury, advanced lesions, fistulisation or recurrence.[5,9]

Final comments

NTM-related infections of the parotid gland are rare and MT must be ruled out. Treatment must be personalised after assessing the risk-benefit ratio.

In our case, drug therapy prior to surgery considerably reduced the size of the lesion and minimised aesthetic sequelae.

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Streptobacillus moniliformis bacteraemia: A case report

Bacteriemia por Streptobacillus moniliformis*: a propósito de un caso*

Dear Editor:

Streptobacillus moniliformis is a fastidious, pleomorphic, Gram-negative rod that is part of rodents' upper respiratory tract microbiota. *S. moniliformis* as well as *Spirillium minus* cause "rat-bite fever" (RBF) disease.[1]

Here we report a case of bacteraemia and possible infectious endocarditis due to *S. moniliformis*. A 31-year-old man was admitted to the emergency department after a two-week history of fever and unspecific cutaneous lesions in some fingers and feet. He had little dotted lesions in both hands located only on the fingers but not on the palms. There were also other unspecific lesions on the sole of the feet. Blood samples were collected to perform further serologic studies. Considering that the patient general condition was good, he was discharged under paracetamol therapy and he was referred to the infectious diseases outpatient department. One week later, the patient returned to the hospital because of persistent fever as well as the appearance of arthralgia and additional skin lesions. After examining the patient, one and two millimetre-sized petechial and purpuric lesions were found on both hands (fingers and palms) and feet (the right toe and the left heel). Some of them were slightly bigger and similar to Osler's nodes. Neither cellulitis nor oedema was observed. Empiric therapy was started with intravenous (IV) ceftriaxone 2 g/24 h due to infectious endocarditis suspicion.

The initial peripheral blood analysis (obtained one week before) and the new analysis results demonstrated normocytic anaemia, thrombocytosis, C-reactive protein value of 15 mg/L, erythrocyte sedimentation rate of 40 mm/h and normal values of rheumatoid factor, and serologic studies were negative (HIV, hepatitis, toxoplasma and *Treponema pallidum* serologies). Histopathological studies of skin lesions found small thrombi causing occlusive vasculopathy. Neither the ophtalmoscopic exam nor the transthoracic echocardiogram revealed any abnormalities.

Gram stain of the blood cultures (positive after 20 h incubation) showed thin and long Gram-negative bacilli. Microorganism identification directly from blood culture using MALDI-TOF mass spectrometry was not achieved. After a 72-h incubation under capnophilic atmosphere, small and greyish colonies grew on sheep blood agar subculture. Those colonies were identified using MALDI-TOF analysis as *S. moniliformis* with a 1.95 score. Identification by 16S rRNA PCR and sequencing yield a 709 bp amplified fragment that shared 99% identity with *S. moniliformis* ATCC49940 (acc. num. KP657489.1). Susceptibility testing was performed by the gradient diffusion (Etest) and disk-diffusion methods on sheep blood agar plates. The isolate was susceptible to penicillin (MIC: 0.03 mg/L), cefotaxime (MIC: 0.012 mg/L), imipenem (MIC: 0.012 mg/L), tetracycline (MIC: 0.38 mg/L) and ciprofloxacin (MIC 0.19 mg/L) and was resistant to aminoglycosides (MICs: tobramycin 8 mg/L, amikacin 32 mg/L and gentamycin 4 mg/L), colistin (MIC: