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Keratitis due to *Nocardia* nova after cataract surgery

Queratitis por Nocardia *nova tras cirugía de cataratas*

In the last few years, the genus *Nocardia* has undergone rapid taxonomic expansion due to the utilization of 16S sequencing[1] resulting in more than 100 recognized described species, although around two thirds of them are known as pathogen.[2]

Nocardia infections can be acquired by either inhalation, causing lung disease in immunocompromised patients[3] or traumatic inoculation. Ocular affectation caused by these organisms is a rare location, being keratitis more frequent than scleritis or endophthalmitis infection.[4] Here we describe one case of sideport infection of a self-sealing corneal tunnel incision following an uneventful phacoemulsification as a complication of cataract surgery due to *Nocardia nova*.

The patient was a 59-year-old male who underwent an uneventful clear corneal temporal incision phacoemulsification with a foldable intraocular lens (day 0). He recovered uneventfully a month after surgery (day 30) being treated with a combination of tobramycin/dexamethasone and diclofenac eye drops.

In the 48 day, the patient came to the hospital with complaints of redness, tearing, photophobia, blurry vision and irritation in the right eye during two days. Visual acuity was recorded as 20/30. The cornea showed a small stromal infiltrate of 3 mm diameter in the superior temporal quadrant of the right cornea with a 1+ anterior chamber reaction but no hypopyon, being 1 mm from the limbus (in the self-sealing tunnel incision), with an epithelial defect of 2 mm diameter, an ill-defined border and mild surrounding corneal edema (Fig. 1). Corneal scraping was taken for microbiological processing and inoculated on tryptic soy 5% sheep blood agar and chocolate agar (bioMérieux®, Marcy-LÉtoile, France), being incubated at 37 °C in 5% CO_2 aerobic atmosphere. He was empirically treated with hourly fortified vancomycin and ceftazidime eye drops. On follow-up the infiltrate did not improve.

After 3 days of incubation (day 51), the culture revealed a significant growth of a microorganism. The Gram-stain of the colony showed gram positive branching filamentous rods. A Ziehl–Neelsen stain and a modified Ziehl–Neelsen stain (5% sulfuric acid) were performed, being negative and positive respectively. The organism was identified as *Nocardia* spp. according to the morphology of the colony and the stain characteristics. The patient had not any risk factors for this infection and he did not travel abroad. Then, treatment was changed to oral trimethoprim–sulfamethoxazole (400/80 mg twice daily) and hourly fortified amikacin and moxifloxacin 0.5% eye drops. In the day 53 clinical improvement was obvious.

Due to partial resolution of the infiltrate, oral cotrimoxazole treatment was finished in day 61, and topical treatment with amikacin and moxifloxacin was finally stopped in day 77 because the lesion was finally cured.

An antimicrobial susceptibility test was performed (day 51) through broth microdilution test (Sensititre®, Trek Diagnostics Systems, West Sussex, England). Minimum inhibitory concentration (MIC) (μg/ml) values were obtained after 72 h of incubation (day 54) and they were as follows: susceptible to amikacin (<1) and cotrimoxazole (2); and resistant to ciprofloxacin (>4) and moxifloxacin (4).

Despite "in vitro" resistant to quinolones, topical moxifloxacin treatment was maintained due to the improvement of the patient.

The strain was then submitted to Instituto de Salud Carlos III (Majadahonda, Madrid, Spain) (day 55) for 16S rRNA gene sequencing, being identified as *Nocardia nova* (day 85).

Keratitis is an ocular infection affecting the cornea, with unusual reported cases due to *Nocardia* or *Mycobacterium*.[5]

The most effective agents toward most infections produced by *Nocardia* are cotrimoxazole and amikacin, being the latter the

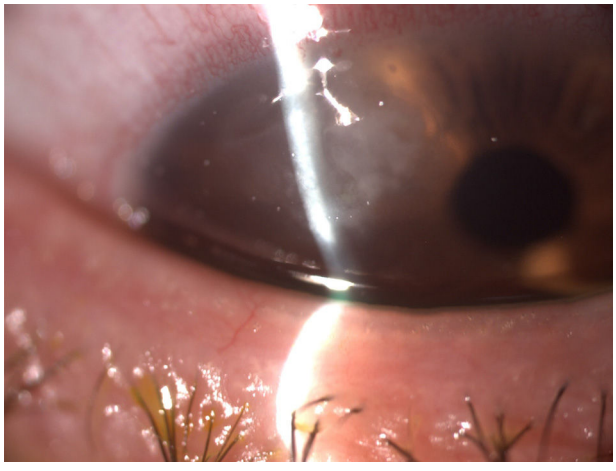

Fig. 1. Cornea infiltrate.

treatment of choice for *Nocardia* keratitis.[6] Other drugs that can be used are tetracycline, chloramphenicol and fluoroquinolones.[4] According to a paper developed in our country, all strains of *Nocardia nova* are susceptible to amikacin and cotrimoxazole, whereas almost all of them are resistant to fluoroquinolones.[5] However, moxifloxacin shows a minimum susceptibility[7] and its concentration in cornea are high,[8] which could be the reason why "in vitro" resistant is not always related to a failure treatment of keratitis.

Among the eye infections, there are few cases of conjunctivitis[9] and scleritis[10] due to *Nocardia nova* complex. Here we describe the first case of keratitis due to *Nocardia nova* secondary to ocular surgery, with a good outcome after prolonged therapy, despite *in vitro* quinolone resistance.

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Cat-scratch disease presenting as parotid gland abscess and aseptic meningitis

Absceso de parótida y meningitis linfocitaria como presentación de enfermedad por arañazo de gato

Cat scratch disease (CSD) usually presents with a regional subacute lymphadenopathy after a cat scratch or bite. It is more frequent in children and teenagers, and usually it is a self-limited condition. Atypical and systemic clinical forms have been described in 5–20% of patients. Several organs including parotid gland and central nervous system (CNS) can be involved.[1,2] Herein, a case of a patient with CSD presenting a parotid abscess with aseptic meningitis is detailed.

A 74 year-old man, previously healthy, was admitted to the hospital because of fever, chills, night sweats and malaise for 2 weeks. Besides, he reported cervical pain 24 h before. He had been evaluated in the emergency room the previous day because of fever and diagnosed of respiratory infection, and was treated with azithromycin. The patient had a cat, although he did not remember any bite or scratch. Physical examination showed axillary temperature of 39 °C with normal heart rate and blood pressure. A cervical deviation to the right (torticollis) and small adenopathies were found in the neck. Meningeal signs were not assessable. No other alterations were observed. The white blood count (WBC) was 15,500/mm^3, and C reactive protein (CRP) was 48 mg/L. The remaining analysis was normal. Chest radiography and abdominal ultrasounds scan were normal. A lumbar puncture was performed and the cerebrospinal fluid (CSF) showed 27 cells/mm^3 (100% mononuclear) and 0.55 g/L of proteins with normal values of glucose and ADA. CSF Gram and auramine staining did not demonstrate microorganisms. Ceftriaxone, vancomycin and ampicillin treatment was started. Three days later the fever and the cervical pain disappeared. A painful tumor on the right parotid gland that was hot and erythematous and a pre-auricular adenopathy were detected. A cervical CT-scan revealed a hypodense lesion with uptake of contrast in the right parotid and bilateral maxillary sinusitis (Figure 1). Magnetic resonance imaging (MRI) of cervical region demonstrated the same findings. Blood cultures were negative. CSF culture and polymerase chain reaction (PCR) assays for *Borrelia burgdorferi*, *Mycobacterium tuberculosis* and *Bartonella* spp. were negative. Serological studies against HIV, *Brucella* spp.,

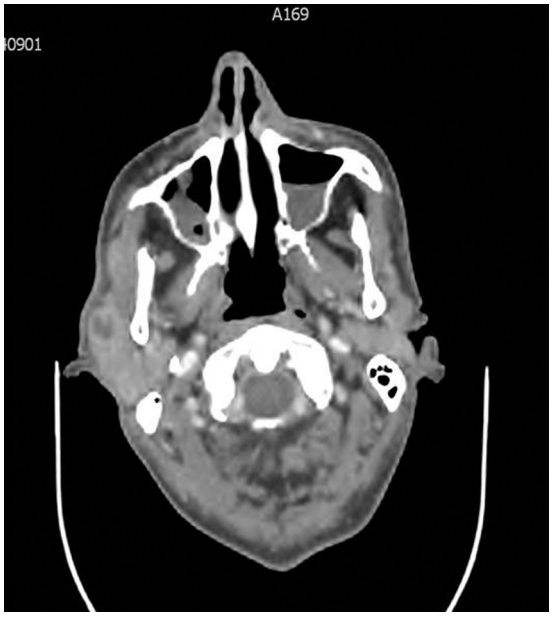

Fig. 1. CT-scan of the neck showing nodular hypodense lesion in right parotid gland, with fine peripheral enhancement compatible with abscess.