

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 susceptibility⁷ 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 conjunctivitis⁹ and scleritis¹⁰ 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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References

- Schlager R, Fisher MA, Hanson KE. Susceptibility profiles of *Nocardia* isolates based on current taxonomy. *Antimicrob Agents Chemother.* 2014;58:795–800.
- Betran A. Clinical significance, antimicrobial susceptibility and molecular identification of *Nocardia* species isolated from children with cystic fibrosis. *Braz J Microbiol.* 2016.
- Bajracharya L, Gurung R. A case of nocardia keratitis treated successfully with topical amikacin. *Nepal J Ophthalmol.* 2012;4:170–3.

- Rao SK. *Nocardia asteroides* keratitis: report of seven patients and literature review. *Indian J Ophthalmol.* 2000;48:217–21.
- Garg P, et al. A cluster of *Nocardia* keratitis after LASIK. *J Refract Surg.* 2007;23:309–12.
- Lalitha P, et al. *Nocardia* keratitis: species, drug sensitivities, and clinical correlation. *Cornea.* 2007;26:255–9.
- Larruskain J, et al. Susceptibility of 186 *Nocardia* sp. isolates to 20 antimicrobial agents. *Antimicrob Agents Chemother.* 2011;55:2995–8.
- Chung JL, et al. Comparative intraocular penetration of 4 fluoroquinolones after topical instillation. *Cornea.* 2013;32:1046–51.
- Micheletti JM, et al. Chronic conjunctivitis due to *Nocardia nova* complex formation on a silicone stent: a case report and review of the literature. *Ophthalm Plast Reconstr Surg.* 2015;31:e131–2.
- Das S. Nodular non-necrotising anterior scleritis due to *Nocardia nova* infection. *Eye (Lond).* 2007;21:276–8.

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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/mm³, 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/mm³ (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.

Borrelia burgdorferi and *Coxiella burnetii* were negative and showed *Toxoplasma gondii* past infection. An immunofluorescence assay (IFA) against *Bartonella henselae* showed an IgM titer of 1:200 and IgG titer of 1:3200. Treatment was changed to oral doxycycline and rifampicin. Four days later the patient was discharged with a diagnosis of atypical CSD with parotid abscess and aseptic meningitis. Patient completed treatment for 2 weeks, and 1 month later he had fully recovered. A cervical CT-scan did not show abnormalities and CRP was within the normal range. Six months later, the IgG titer against *B. henselae* had decreased to 1:800.

To our knowledge, this is the first case reported of *B. henselae* infection that appeared as parotid abscess and aseptic meningitis. Some clinical aspects are worth mentioned.

Only 6% of CSD patients are elderly people. CSD is more frequent in women at this age, and atypical and severe forms are more frequent.³

Parotid involvement is present in 6% of cervical and head location in CSD^{4,5} and generally appears in the context of Parinaud's syndrome but this is very rare in elderly patients.^{1–3} The patient did not show the typical eye affectation and only had a parotid abscess that is the typical involvement of parotid in CSD.⁵ A rare sign was the presence of torticollis that has been reported in only 1% of cases in cervical CSD, generally associated with deep tissue involvement⁴ and that was excluded in our patient by MRI and CT-scans. Encephalitis and neuroretinitis are the most frequent CSD neurologic manifestations.^{1,2} Encephalitis is more frequent in elderly patients.³ Our patient did not develop convulsions, disorder of consciousness or any signs of focal neurological involvement that are typical of encephalitis. Meningitis is a very rare manifestation, and normally appears in the context of neuroretinitis.^{6–8} In our patient, the assessment of meningeal signs was difficult in context of neck stiffness and pain secondary to torticollis, but the CSF analysis was indicative of meningeal injury with findings of aseptic meningitis. This fact was essential for the choice of antimicrobials and duration of the therapy. Moreover, our patient did not show ocular involvement or loss of vision suggesting neuroretinitis.

In this case, the diagnosis was made by serologic assays. PCR tests were negative. This fact may be due to the low sensitivity of PCR in some samples and the previous use of antimicrobials. Doxycycline and rifampicin were prescribed since this is the option that experts recommend for CNS involvement.^{9,10} Our patient favorably evolved, probably due to the early instauration of the treatment. It

is known that delayed treatment is a prognosis factor, especially for elderly people.^{6,9,10}

References

- Murakami K, Tsukahara M, Tsuneoka H, Iino H, Ishida C, Tsujino K, et al. Cat scratch disease: analysis of 130 seropositive cases. *J Infect Chemother.* 2002;**8**:349–52.
- Lamps LW, Scott MA. Cat-scratch disease: historic, clinical, and pathologic perspectives. *Am J Surg Pathol.* 2004;**121** Suppl. 1:S71–80.
- Ben-Ami R, Ephros M, Avidor B, Katchman E, Varon M, Leibowitz C, et al. Cat scratch disease in elderly patients. *Clin Infect Dis.* 2005;**41**:969–74.
- Ridder JG, Boedeker CC, Technau-Ihling K, Sander A. Cat scratch disease: otolaryngologic manifestations and management. *Otolaryngol Head Neck Surg.* 2005;**132**:353–8.
- Ridder GJ, Richter B, Laszing R, Sander A. Parotid involvement in Cat-scratch disease: a differential diagnosis with increased significance. *Laryngorhinotologie.* 2000;**79**:471–7.
- Pinto VL Jr, Curi AL, Pinto Ada S, Nunes EP, Teixeira Mde L, Rozental T, et al. Cat scratch disease complicated with aseptic meningitis and neuroretinitis. *Braz J Infect Dis.* 2008;**12**:150–60.
- Fukushima A, Yasuoka M, Tsukahara M, Ueno H. A case of cat scratch disease neuroretinitis confirmed by polymerase chain reaction. *Jpn J Ophthalmol.* 2003;**47**:405–8.
- Wong MT, Dolan MJ, Lattuada CP Jr, Regnery RL, Garcia ML, Mokulis EC, et al. Neuroretinitis, aseptic meningitis and lymphadenitis associated with *Bartonella (Rochalimaea) henselae* infection in immunocompetent patients and patients infected with immunodeficiency virus type 1. *Clin Infect Dis.* 1995;**21**:352–60.
- Rolain JM, Brouqui P, Koehler JE, Maguina C, Dolan MJ, Raoult D. Recommendations for treatment of human infections caused by *Bartonella* species. *Antimicrob Agents Chemother.* 2004;**48**:1921–33.
- Pérez-Martínez L, Blanco JR, Oteo JA. Tratamiento de las infecciones por *Bartonella* spp. *Rev Esp Quimioter.* 2010;**23**:109–14.

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Características de la infección por metapneumovirus humano, ¿es importante la edad?



Characteristics of human metapneumovirus infection, is it important the age?

Las infecciones respiratorias agudas son una de las principales causas de morbilidad en niños¹. En los últimos años, gracias al progreso de las tecnologías de diagnóstico molecular se han descrito nuevos virus implicados en estas infecciones, siendo uno de estos el metapneumovirus humano (MPVh)².

Aunque es el causante del 6–14% de los ingresos por infección de vías respiratorias en niños^{3,4}, menos frecuente que los principales causantes de hospitalización (VRS, parainfluenza), se le atribuyen hasta un 20% de estas infecciones virales en niños^{5,6}.

Existen pocos trabajos que evalúen la epidemiología de la infección por MPVh en nuestro medio, por lo que en esta carta científica queremos destacar diferentes aspectos epidemiológicos, clínicos,

diagnósticos y terapéuticos entre los niños hospitalizados en los que se ha demostrado infección por MPVh, y presentar las diferencias encontradas entre la infección en lactantes menores de 2 años y en niños de más edad, eligiendo este punto de corte, bajo la hipótesis de mayor sintomatología obstructiva y gravedad en los lactantes respecto a niños más mayores^{7,8}.

En un estudio retrospectivo llevado a cabo en nuestro hospital entre abril de 2009 y mayo de 2014 se incluyeron 192 aislamientos de MPVh en aspirados nasofaríngeos correspondientes a 190 pacientes de 0 a 14 años (en 2 pacientes se aisló MPVh en 2 ingresos en diferentes años). Todas las muestras fueron enviadas al departamento de virología del laboratorio del hospital en medio de transporte de virus (MTV), y fueron estudiadas mediante una técnica de inmunofluorescencia directa con anticuerpos monoclonales específicos (D3 Duet[®] DFA RSV/Respiratory Virus Screening Kit, DiagnostiC Hybrids).

El 77,6% de los pacientes fueron menores de 2 años. De los pacientes de nuestra serie, el 10,4% presentaron enfermedad de base (neurológica, cardíaca, oncológica o prematuridad), sin