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CASE REPORT

Cat Scratch Disease - a case report

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Abstract

Cat Scratch Disease (CSD) presents most commonly with fever and lymphadenopathy, but up to 25% of cases develop systemic manifestations. **Objective:** to report a pediatric case of hepatosplenic form of CSD. **Method:** this is a case report of a nine-year-old child hospitalized from October to November 2019. The report was written based on medical records and literature review, through Pubmed platform. Informed consent was given by the legal sponsor and the assent by the child. **Result:** a nine-year-old girl presents with swollen, tender and erythematous nodes in the right popliteal fossa and fever. Initial suspicion was of cellulitis, complicated with a popliteal abscess. Treatment was initiated with oxacillin for 3 days and later on with clindamycin and ceftriaxone for 11 days. On the eleventh day of hospital stay, the patient manifested hepatosplenic abscesses and antibiotic treatment was upscaled to vancomycin and piperacillin-tazobactam until she was diagnosed with a hepatosplenic form of CSD, which happened seventeen days after admission. Therefore, treatment was provided with Rifampicin and Clarithromycin for 14 days, with significant clinical improvement. **Conclusion:** this report aims to raise awareness regarding the wide clinical spectrum of this infection and how to better manage it.

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INTRODUCTION

Cat Scratch Disease (CSD) is caused by bacteria of the genus *Bartonella*.^{1,2} The disease has a variable clinical presentation, from the most common form, characterized by fever and lymphadenopathy, to systemic forms, in up to 25% of cases.^{1,2}

This study reports a case of CSD in the hepatosplenic form and aims to contribute to the dissemination of one of the systemic forms of the disease and raise awareness of its early recognition and treatment.

CASE DESCRIPTION

LSR, female, 9 years and 10 months old, born and coming from São Paulo-SP, previously healthy, started experiencing pain in the right popliteal region 3 weeks ago; and 15 days ago, with a fever of up to 38 degrees Celsius, for three days, associated with local phlogistic signs. She was diagnosed with cellulitis and was prescribed Cephalexin for 10 days and an anti-inflammatory drug. Since only the fever improved, she sought out our service. Upon admission, she was in good general condition, eutrophic, with no changes in vital signs, cardiopulmonary auscultation was unremarkable, abdomen without visceromegaly and the right lower limb presented bulging in the popliteal fossa, with phlogistic signs and impaired leg extension, and was admitted with the hypothesis diagnosis of popliteal abscess. We then ordered a blood count (Hemoglobin 12.8 g/dL, Hematocrit 36.9% Leukocytes 9520/ μ L (Neutrophils 56%, Eosinophils 5%, Lymphocytes 31%, Monocytes 6%) platelets 285,000/ μ L), C-Reactive Protein 13.8 mg/L and blood culture was collected. Ultrasonography of the right lower limb showed a multilobulated collection, measuring 3.3 x 1.4 x 2.6 cm (6.3 mL) and reactive lymph node enlargement, in the popliteal region, without flow changes on Doppler.

Regarding the relevant background, her vaccinations were up to date, she had a home with basic sanitation and had 1 dog and 18 cats.

She received Oxacillin 200 mg/kg/day, switched to Ceftriaxone 1g every 12 hours and Clindamycin 30 mg/kg/day after three days due to no clinical improvement. The admission blood culture was negative. Serology for *Bartonella* was ordered due to positive epidemiology, but the result was only available after 13 days.

On the ninth day of antibiotic therapy, she developed a fever of 38.7 °C, associated with pain in the epigastric region, which progressively worsened over the following three days. Abdominal ultrasound showed scattered heterogeneous nodular areas in the liver and a nodular area with the same characteristics in the spleen.

Abdominal tomography showed hypodense images scattered throughout the liver parenchyma (the largest approximately 2.0 x 1.4 cm) and splenomegaly, with a

lobulated hypodense nodular image (approximately 3.0 x 2.4 cm), compatible with abscesses, without indication of surgical drainage.

Expanded treatment with Vancomycin 40 mg/kg/day and Piperacillin + Tazobactam 300 mg/kg/day. She had the following test results: C-Reactive Protein 109.17 mg/L, Erythrocyte Sedimentation Rate 49 mm, Total Bilirubin 0.18 mg/dL, Direct Bilirubin 0.13 mg/dL, Alkaline Phosphatase 229 U/L, Gamma Glutamyl Transferase 142 U/L, Aspartate Amino Transferase 78 U/L, Alanine Amino Transferase 59 U/L. After two days of this new therapeutic regimen, the serology result for *Bartonella* came back positive, IgG with a titer of 1/1024, and the diagnosis of systemic CSD was made. Clarithromycin 15 mg/kg/day and Rifampicin 600 mg/kg/day were then started.

The patient evolved with clinical and laboratory parameters improvement. Abdominal tomography on the eighth day of Clarithromycin + Rifampicin showed no evidence of new lesions. She was discharged after 26 days of hospitalization, with medications until she completed 15 days of treatment. Upon outpatient return, 25 days after discharge, she was asymptomatic. Control tomography performed three months after hospitalization showed a significant reduction in the dimensions of the hepatic and splenic nodular images.

DISCUSSION

CSD is caused by bacteria of the genus *Bartonella*, gram-negative and aerobic bacilli, with *Bartonella henselae* being the main one.^{1,2} Cats are its main reservoir, transmitted from one feline to another through the flea *Ctenocephalides felis*.^{1,3,4} Transmission to humans occurs through scratching, biting or contact of non-intact skin with the animal's saliva.^{5,6,7}

It is estimated that in the United States the annual incidence is 4.5 cases/100,000 inhabitants in the adult population and 9.4 cases/100,000 in children aged 5 to 9 years.^{8,9} In Brazil, the incidence is unknown and the number of cases is underestimated.^{1,10}

The localized form of CSD is characterized by fever and lymphadenopathy, most common in the axillary, epitrochlear, cervical and inguinal chains,^{1,6,11,12} in addition to malaise, headache and odynophagia.^{1,6,11,12} In children, constitutes around 85 to 90% of cases.²

CSD can also present as Fever of Undetermined Origin (FUO) in 10 to 30% of cases and should therefore be included in the differential diagnosis during its investigation.⁶

The hepatosplenic manifestation involves the formation of microabscesses in the liver and/or spleen, in addition to fever, abdominal pain, hepatosplenomegaly and lymph node enlargement.^{1,5,11} These abscesses are characterized by hypoechogenic lesions on ultrasound and hypoattenuating on tomography.^{1,6}

In immunosuppressed patients, CSD causes a pro-angiogenic immune response, leading to a systemic form called bacillary angiomatosis.^{1,4} In 90% of cases, it presents

with dermatological lesions, consisting of red-brown papules, which make a differential diagnosis with Kaposi's sarcoma, epithelioid hemangioma and pyogenic granuloma.^{1,4} There may also be liver involvement, called bacillary hepatic peliosis.^{5,6}

The ocular manifestations of CSD are Parinaud Oculoglandular Syndrome (which occurs in approximately 5% of patients³ and consists of fever, pre-auricular, submandibular or cervical lymphadenopathy), conjunctival necrotic granuloma, follicular conjunctivitis^{1,5,6} and neuroretinitis, which can result in sudden loss of vision.^{1,3,5}

CSD rarely affects the Nervous System. In children, it manifests as encephalopathy, presenting changes in mental status, headache, seizures, neck stiffness, weakness, changes in tone and reflexes.^{1,5,6} The CSF may be normal or show increased cellularity or protein. Imaging exams are rarely changed. The electroencephalogram may show slowing of waves. The prognosis is favorable, with spontaneous recovery in more than 90% of cases.^{1,3,6} Other less frequent neurological manifestations are neuropathies, Guillain-Barré Syndrome, ataxia, transverse myelitis, among others.^{1,3,6}

The diagnosis is currently based on a combination of epidemiological, clinical, serological, histological data and imaging findings, as there is no gold standard method.¹² In cases of typical presentation of the disease, the diagnosis is clinical.^{1,2} In less common presentations, the main diagnostic method is serology, with immunofluorescence being the most used. Sensitivity ranges from 14 to 100% for IgG and 2-50% for IgM, and specificity ranges from 34-100% for IgG and 86-100% for IgM.^{1,12} The disadvantages are: negative IgM does not exclude the disease, as its levels are detectable for only 3 months, and positive IgG does not necessarily mean current infection, as it can remain positive for up to a year.^{2,5,6} IgG titers above 1:256 strongly suggest current infection.^{1,5}

Treatment for localized lymphadenopathy consists of supportive measures.^{5,6} If there is lymph node suppuration, its contents must be aspirated. Incision and drainage are not recommended due to the risk of recurrence.^{4,7,11} In systemic forms, localized conditions that do not resolve spontaneously and in comorbidities or immunosuppression, antimicrobial treatment is recommended.³ Azithromycin can be used for 5 days, Rifampicin or Metronidazole for 2 to 3 weeks or Sulfamethoxazole + Trimethoprim for 7 to 10 days.^{1,2,4}

Treatment recommendations for systemic forms of CSD are based on literature with a low level of evidence, with observational studies and a wide variety of antimicrobial regimens among them.^{1,4,6} For the hepatosplenic form and prolonged fever, some studies recommend Rifampicin for 14 days, associated or not with Azithromycin, Gentamicin or Sulfamethoxazole + Trimethoprim.^{4,6,12,13}

In immunosuppressed patients, bacillary angiomatosis is treated with Erythromycin or Doxycycline or in combination with Rifampicin in more severe cases, for 3 to 4 months, due to the risk of relapse.^{1,3,6}

For neuroretinitis, Doxycycline (due to its penetration into the central nervous system) or Erythromycin (in children under 8 years of age) is recommended in association with Rifampicin for 2 to 4 weeks in immunocompetent patients and for 4 months in immunosuppressed patients.^{1,5} In encephalopathy, antimicrobial therapy is controversial and, if performed, the same regimen as neuroretinitis is recommended for 4 to 6 weeks.^{1,5}

Prevention measures include controlling fleas on cats, health education, hand hygiene after contact with cats and supervising children when playing with pets.^{2,8}

In summary, the case described illustrates the wide clinical spectrum of this infection, which, combined with the delay in obtaining the serology results, can make its diagnosis a challenge. Therefore, it is worth remembering this disease when investigating cases of prolonged fever, lymphadenopathy, abscesses and/or abdominal pain.

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