

Visceral involvement in an immunocompetent male: a rare presentation of cat scratch disease

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ABSTRACT

We report a case of an adult, immunocompetent male with lymphadenopathy of both groins, para-aortal lymph nodes and multiple lesions in the spleen. A neoplasm was excluded by histology of the largest lymph node from the left groin. The diagnosis of cat-scratch disease (CSD) became apparent when serological testing for *Bartonella henselae* showed to be positive. A review of literature shows that disseminated (visceral) infection is a rare presentation of CSD.

KEYWORDS

Bartonella henselae, cat-scratch disease, spleen, visceral

INTRODUCTION

Cat-scratch disease (CSD) is an infectious disease generally seen among children and young adults and is caused by *Bartonella henselae*. It presents with a primary dermal lesion and regional lymphadenopathy after a scratch from an infected cat. Disseminated infection with extended lymphadenopathy, visceral involvement, and neurological and ocular manifestations is rare in adults and immunocompetent persons.

CASE REPORT

Two weeks before admission to our hospital, a 49-year-old man noticed a painful swelling in the left groin. For a few days this was accompanied by fever and transpiration during the night. After his divorce, about a year ago, he suffered an unknown amount of weight loss. He had not had any unsafe sexual intercourse during the previous year.

At physical examination he was in a clinical good condition with a temperature of 38.6°C. In the left groin, a painful enlarged lymph node of 3.5 cm was palpable. There was also a slightly enlarged lymph node in the right groin. No enlarged lymph nodes were detectable in the cervical and axillary regions. Physical examination of the heart, lungs and abdomen was normal.

Laboratory testing showed a raised C-reactive protein (145 mg/l) and a peripheral white blood cell count of $12.0 \times 10^9/l$ with 15% monocytes. Serological studies for various infectious diseases were taken at that time. Needle biopsy of the largest lymph node in the left groin had no purulent aspect and was aseptic. The lymphocyte populations were normal (CD4/CD8 ratio 2.4 and kappa/lambda ratio 1.8). Abdominal computed tomography (CT) showed enlarged lymph nodes in both groins and a pathological enlargement of lymph nodes in the para-aortal region. Multiple hypodense round lesions were found in an enlarged spleen (figure 1). Because we could not exclude a neoplasm at this time, we decided to extirpate the largest lymph node from

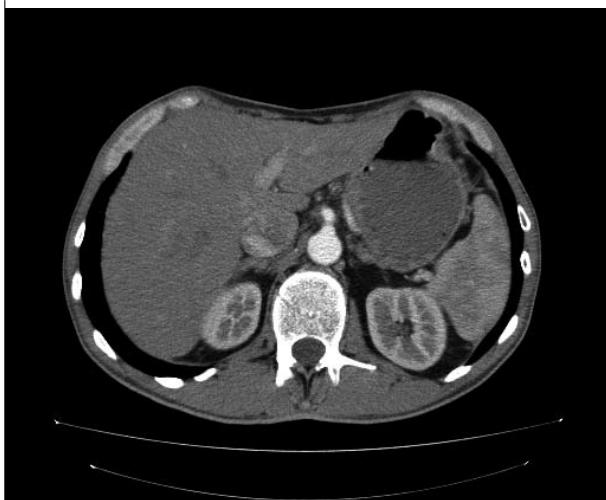
Figure 1. Multiple hypodense round lesions are visible in the enlarged spleen on admission



the left groin. Histology of this lymph node revealed a sclerosed aspect, without signs of malignancy or acute inflammation.

After two weeks the ELISA for *B. henselae* IgM proved to be positive and the IgG specific for *B. henselae* was 3,53 g/l. Afterwards he remembered that he had been scratched by a cat. Serological tests were negative for other pathogens, such as HIV and chlamydia trachomatis. We had no suspicion of immunodeficiency in this patient, because of the lack of infections in his medical history, the negative HIV serology and a normal serum electrophoresis. Afterwards we asked the pathologist to stain the lymph node for *Bartonella*. A Giemsa staining showed intracellular micro-organisms. We decided not to treat him with antibiotics because of his good clinical condition and the fact that CSD is a self-limiting disease. Three months later the CT scan of the abdomen showed that the lesions in the spleen had completely disappeared (figure 2). Seven months after presentation the ELISA for *B. henselae* IgM had become negative and serological tests showed a raise in specific IgG levels (5,5 g/l).

Figure 2. After three months, the spleen regained its normal size and the lesions resolved



DISCUSSION

CSD is a world-wide infectious disease caused by *B. henselae*, a Gram stain negative, intracellular rod. In the Netherlands the incidence of CSD is approximately two cases per 100,000 and about 300 to 1000 cases per year.¹ Studies show that about 14 to 53% of the cats and kittens have positive serology testing for *B. henselae*. There are indications that cats are not permanently infected, but are periodically virulent.²

CSD typically presents with a localised papule after a cat scratch. Lymphadenopathy affects the proximal lymph region and arises within one to two weeks of inoculation

and can persist over 12 months. One-third of the patients have systemic symptoms such as fever, headache and lethargy. The diagnosis of CSD can be made on the typical history of a recent scratch combined with typical clinical findings. Laboratory diagnosis is based on a positive IgM serology. The tiny *B. henselae* can not be seen in sections stained by tissue Gram methods, but can be found in Giemsa-stained sections.³ A positive polymerase chain reaction (PCR) for *B. henselae* on tissue is the ultimate evidence for CSD. This test is not available in many laboratories. The clinical findings with a positive IgM for *Bartonella* and seroconversion give enough evidence for the diagnosis of CSD. We did not perform PCR on the lymph node. CSD is a self-limiting disease and does not need antimicrobial treatment, although there are reported cases of disseminated CSD treated with antibiotics.⁴ Antibiotics with proved clinical efficacy are macrolides,^{5,6} gentamicin, rifampicin, tetracycline and ciprofloxacin. Bass *et al.* report a significant decrease in lymph node volume in the first month after treatment with five days of azithromycin.⁵ In our hospital only immunoincompetent patients with CSD are treated with antibiotics.

Lymphadenopathy and visceral involvement is rare in CSD. A small number of the patients present with lymphadenopathy and granulomas in the liver, bones, lungs and spleen. Less common manifestations of CSD include the oculoglandular syndrome, meningitis and pneumonia. In the last two decades, several reports about visceral involvement in CSD have been published. Two articles report a patient with granulomas in the liver and spleen without lymphadenopathy.^{7,8} Daybell *et al.* report a splenic rupture due to a disseminated CSD, which is a very rare complication. What is remarkable is the large numbers of immunoincompetent patients who suffer from disseminated CSD. Persons infected with HIV can present with bacillary angiomatosis and hepatic peilosis due to CSD.⁹

Our case shows the possibility of lymphadenopathy and lesions in the spleen due to CSD. The combination of lymphadenopathy and lesions in the liver and spleen can be seen in infectious diseases such as *Entamoeba histolytica*, mycobacterium tuberculosis or pyogenic bacteria such as *Salmonella* and *B. henselae*. On the other hand, neoplasms such as Hodgkin's and non-Hodgkin's lymphoma and acute leukaemia should always be included in the differential diagnosis.¹⁰ In this case, histology of the largest lymph node excluded malignancy. Our patient recovered spontaneously within a few months.

CONCLUSION

This case demonstrates the possibility of lymphadenopathy and involvement of the spleen due to CSD in an immunocompetent adult person. That is why we emphasise

the importance of considering CSD when lymphadenopathy and lesions in visceral organs are present, even though the patient is immunocompetent.

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