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# Case Report

## CAT SCRATCH DISEASE

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**ABSTRACT**

Approximately 24,000 people are infected with cat scratch disease (CSD) every year. CSD is caused by the bacteria Bartonella henselae, a gram-negative bacteria most often transmitted to humans through a bite or scratch from an infected cat or kitten. Although CSD is often a benign and self-limiting condition, it can affect any major organ system in the body, manifesting in different ways and sometimes leading to lifelong sequelae. It is a disease that is often overlooked in primary care because of the wide range of symptom presentation and relative rarity of serious complications. It is important for health care providers to recognize patients at risk for CSD, know what laboratory testing and treatments are available, and be aware of complications that may arise from this disease in the future.

***Keywords:*** Bartonella henselae, cat scratch disease, cat scratch fever.

## INTRODUCTION

Cat scratch disease (CSD) is a bacterial infection caused by the bacteria Bartonella henselae, a small, slow-growing, fastidious, intracellular, gram-negative bacillus that exists worldwide, transmitted to humans through a bite or scratch from an infected cat or kitten, dogs, foxes and even coyotes in USA.( Loutit JS, 2001) Most patients have a history of a cat scratch or contact. Extension beyond the local site occurs in about 14% of patients, resulting in encephalitis, osteomyelitis, peritonitis, pneumonia, exanthemas, and hepatosplenomegaly.

## Case Report

A 34 – year female patient presented at the clinic with a three months' history of weakness, sweating, fever to 38,5$^{o}$ C, heaviness, and pain in the left upper quadrant of the abdomen, weight loss about 30 kg. for a year and a half. There was a history of contact with cats but no bites or scratches. During the last year serological tests for leishmaniasis, tuberculosis, and hydatid disease are made. The results are negative. Laboratory tests included a white blood cell count 17,25 x10$^{9}$ / l; Hb – 85gr/l.; Hct – 0,25; Platelet count- 452 x10$^{9}$ / l. serological tests - Mycoplasma pneumoniae IgG – 0,71; Chlamidia trachomatis IgG – 0,27; Chlamidia trachomatis IgA – 0,50; Borrelia burgdorferi IgG – 0,50; Borrelia burgdorferi IgM – 0,49 reference values N < 1.

CT scan was performed – hepatosplenomegaly liver 233/181mm., spleen 163mm. fig1. multiple focal hypoechoic lesions in the spleen fig.2 lymphadenopathy in the regions of the aorta, lwer caval vein, mesentery vessels inguinal lymphadenopathy biggest sized 20mm.fig.3

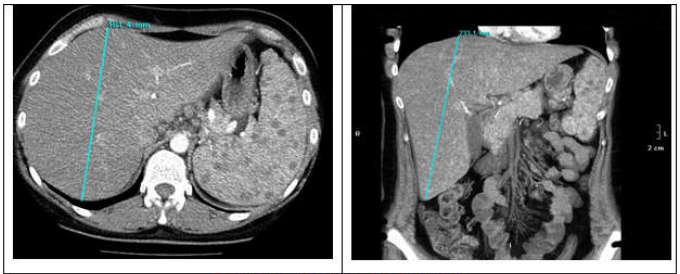

fig.1 CT of abdomen – hepatomegaly

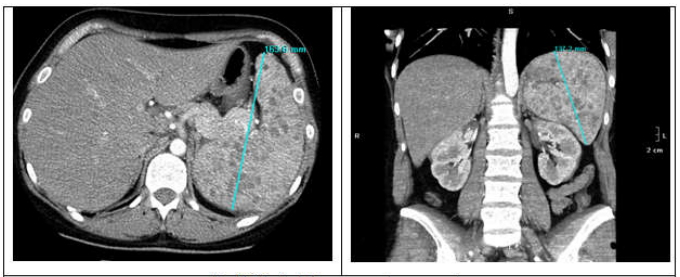

fig.2 CT of abdomen – splenomegaly

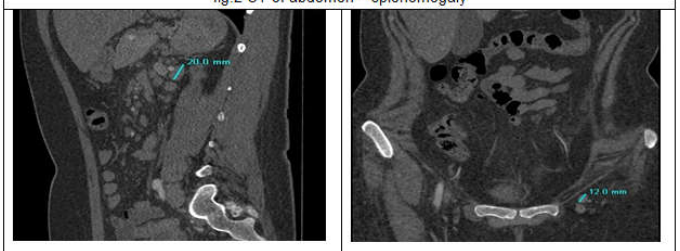

fig.3 CT of abdomen– aortal and inguinal lymphadenopathy.

Intraoperative findings – spleen sized up to 20 cm. adhesions to the omentum stomach and the tail of the pancreas. fig4. Splenectomy with distal pancreatectomy was performed.

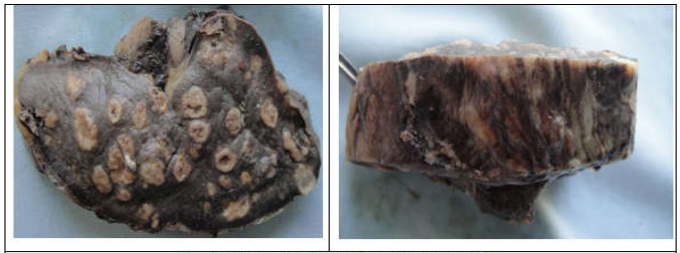

fig. 4 spleen – multiple abscesses up to 1см

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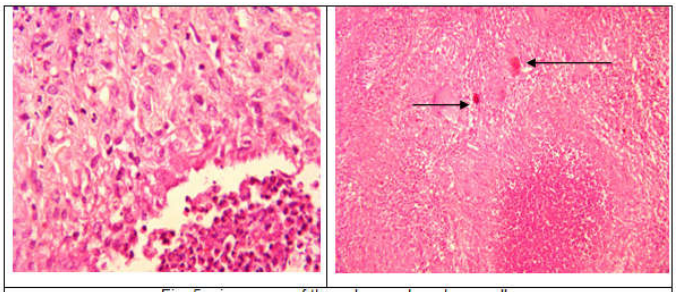

Fig. 5 microscopy of the spleen – Langhans cells

The path histological finding presented various sized granulomas: the small ones with central necrosis and gigantic cell Langhans type; the big ones are presented with central necrosis with abscesses fig.5.
Recovery and therapy with Metronidazol 3x1fl. and Cefazoloin 3x2 gr. for five days. Six months follow – up to the patient – without complaints. Serological tests repeated – antibodies against Bartonella henselae – IgG past medical history for feline disease.

## DISCUSSION

Splenomegaly occurs in less than 10% of patients (Loutit JS, 2001) and was first described by Inglis and Tonge in 1950. (Inglis and Tonge, 1950; Vanlemmens *et al.,* 1995) it has been associated with erythema nodosum" and hepatomegaly. (Bitterman and Cohen, 1985; Inglis and Tonge, 1950; Greenbaum *et al.,* 1988; Hadfield et al., 1985; Kinlaw, 1954; Korbitz, 1954; Rocco et al., 1985; Winship,1953) and occurs most commonly in children younger than 15 years of age.( Inglis and Tonge, 1950; Greenbaum *et al.,* 1988; Hadfield et al., 1985; Winship, 1953) Approximately 24,000 people are infected each year in the United States with CSD (Loutit JS, 2001). Only in two of the cases reported with hepatosplenomegaly there is lack of cat. There are common signs in the past medical history and in the clinical symptoms in the literature case – reports described:

- presence of a pet – cat;
- scars from scratching or biting;
- lymphadenopathy of non-infectious process or malignancy;
- clinical presentation during two – three weeks;

According to Greenbaum et al. the CT – scan images are infrequent and usually the description is “some small defects” *B. henselae* is difficult to isolate in human blood and tissue, although it is easy to isolate in the blood of cats. (Rocco *et al.,* 1985) Although further studies are warranted, *B. henselae* has also been identified in Ixodes ticks from North America, Europe, and Asia.(Giladi *et al.,* 2005) *B. henselae* is most often transmitted from cats to humans via a scratch or bite through a break in the skin, but can also be transmitted to humans via a bite from an infected flea. Multiple outbreaks within a family have been documented; however, there is no documentation that *B. henselae* is transmitted from person to person. Although CSD is typically a self-limiting condition of usually 6 to 12 weeks duration, rare situational manifestations, including optic nerve involvement, which presents as optic neuritis or neuroretinitis, endocarditic inflammatory breast disease, encephalitis, and hematological manifestations such as thrombocytopenic purpura and hemolytic anemia are seen. (Loutit JS, 2001) Ophthalmic manifestations of CSD are usually benign with an excellent prognosis for visual recovery. (Gray *et al.,* 2004)

## conclusions

1. CSD is more frequent disease. The main cause for this is increase the number of pet and from the other side the new serological tests.
2. The atypical clinical symptoms, are associated with hematological or the nervous system. Involvement of the organs in the abdomen have been reported increasingly.
3. The exact diagnosis and successful treatment depends on the interdisciplinary collaboration between microbiologists, immunologists, surgeons and pathologists.

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