Cat Scratch Disease: Follow up of a Clinical Case

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Abstract: Cat scratch disease is a non-epidemic infection caused by Gram negative bacillus Bartonella henselae. The most common course of the disease includes regional lymphadenitis and skin papule at the entrance site of the infection. We report a case of a female, 22 years of age, with cat scratch disease. Clinically we observed the typical skin papule at the entrance site of the infection with severe suppurative lymphadenitis of the parotid region and the neck. We have observed the course of the disease.

Keywords: Cat scratch disease; lymphadenopathy, head and neck

1. Introduction

Cat scratch disease /CSD/ is caused by Bartonella henselae. Perinaud over hundred years ago described inflammation of the conjunctiva and periauricular lymphadenopathy as a result from an animal contact. In 1931 Debré observes regional lymphadenitis after cat scratch and 20 years later describes the disease (8). In 1983 Wear et al. (40) are the first to discover small Gram-negative microorganism in the lymph nodes of a CSD patient.

2. Epidemiology of CSD

Every year CSD is diagnosed and verified in 22 000 patient in the USA. Over 2000 among them are treated in hospitals. The peak of the disease is in autumn and winter (16). The frequency is from 1,8 to 9,3 cases over 100 000 (16, 43). Most common is among youngsters, 87% being under 18 years old (3). A newer research found that 43% of the patients are over 21 years old (13). The patients report cat contact in more than 90%. Licking of the face, scratching or bite by a kitten (less than 12 months) can be attributed to the disease (43). Eighty four percent of the CSD patients are seropositive for B henselae (43). The examination of the cats of the CSD patients found positive blood result for B. henselae in 68%, while all cats had flees (10).

Clinical characteristics

It is typical for CSD to present as 3-5 mm papule at the site of inoculation. The papule develops from the second up to 3-4 weeks after the inoculation (3, 23, 27). The lesion evolves into macula, pustule and vesicle. Primary skin lesion is found in 60-93% of the cases, which refers to the CSD diagnosis (3, 34). Lymphadenopathy can be observed between 7-th to 60-th day of the papule eruption and in half of the cases, it is isolated to the head and neck region and upper limbs. The lymph nodes are tender or painful in 80% of the cases. Suppurative lymphadenitis is found in 12-15% of the cases, cellulitis is rarely observed. Lymphadenitis resolves spontaneously in 2 to 4 months (3, 23). In 30% of the patient fever and indisposition can be observed, and in 15% - anorexia (23).

3. Diagnosis

Bartonella henselae is demanding microorganism and as such, it is difficult to be isolated. Routine culturing is difficult (11), which can be contributed to the long incubation period of 2-4 weeks (18). Histopathological findings are not characteristic for the disease, but contribute for the clinical and diagnostic correlations (10). Serological tests for Bartonella henselae are sensitive and specific (31, 43). Serological testing for IgA and IgG through indirect immunofluorescence method shows high sensitivity of 88%
and specificity of 98% (2, 28). For positive is considered titre of 1:512 or higher (6). Polymerase chain reaction (PCR) is considered most sensitive method for confirming Bartonella infection (24).

4. Case Report

The patient, female, 22 years old, came for consultation in the Maxillofacial surgery department of St. Anna University hospital with complaints from painful lymphadenitis of the right preauricular and cervical lymph nodes. The complaints started 3 weeks ago, when the lymph nodes enlarged quickly over few days. The patient was suffering from severe pain and tension in the affected region. She complained also from several periods of fever over the past few weeks. Treatment for 14 days with broad-spectrum antibiotic didn’t lead to relief. The clinical examination revealed lymphadenopathy affecting preauricular, parotid and neck lymph nodes from II and III level. The affected nodes were round, non-inosculated, with firm-elastic structure, tender and with no suppuration. A distinctive, non-suppurative periadenitis was observed in the affected region. (Fig. 1) The size of the lymph nodes was ranging from 3x2 cm to 1x1 cm. Skin examination found erythematous papule on the lower eyelid on the same side of the face. (Fig. 2) The patient reported about a scratch on the eyelid from a kitten. An elevated, non-suppurating, and non-healing slightly painful papule gradually appeared at the scratch site. The clinical symptoms and the patient history strongly suggested a possible cat-scratch infection. At admission, the patient was in moderately disturbed general health, with complaints of weakness and pain in the preauricular area. The blood count showed the following results: Leu - 8,3 x10⁹ / L, Lymph - 2,8 x 10⁹ / L; Mono – 0,4 x 10⁹ / L; Gran - 5,1 x10⁹ / L; Lymph % - 32,2%; Mono % - 5,2%; Gran % - 61,6%; Hemoglobin – 125 g/L; Ery - 4,31 x10¹² / L; Thr – 385,0 x10⁹ / L; ESR – 16. Microbiological culturing was without result. Histopathological examination of a curettage material found granulomatous necrotizing lymphadenitis.

5. Discussion

Cat scratch disease can express great variety of clinical signs and symptoms. The most common of them is the lymphadenopathy, which can be found in over 50% of the cases (37). Atypical course of the disease is reported in 5-14% of the patients (9, 23). The Parinaud syndrome (oculoglandular syndrome) is characterized by conjunctivitis and regional lymphadenopathy and can be found in 6% of the cases (23). There is a case report of orbital abscess and osteomyelitis (26). Systemic forms with hepatosplenic involvement have been observed (9; 19, 32). Periportal and periportal adenopathy can be observed. Involvement of the liver and spleen is not necessarily accompanied by enlargement or deviation in liver test results (37). Complaints from the central nervous system can occur in 2-6 weeks after the lymphadenopathy occurrence, and present as seizures, headache and encephalopathy (4, 20, 30). Neurological complaints can be found in 1-7% from the cases (3, 4). Temporary dysfunction of the cranial nerves and myelopathy (20), neuroretinitis, severe vision reduction or blindness (4, 25, 42) are amongst the symptoms, described by clinicians. Lung manifestations of the disease can be pneumonia or pleural effusion (1). Bone involvement in the form of osteomyelitis with radiological osteolytic lesions is related to Bartonella henselae infection (29, 35, 38). Bone lesions can be observed adjacent to the lymphadenopathy. Osteolytic lesions can occur with localized pain without erythema, tenderness or swelling (5, 39). The disease can present with skin eruption (5%) most often maculopapular rash, observed in the early stages. Vesicular rash, erythema nodosum, erythema multiforme, rashes, purpura and leukocytoclastic vasculitis are amongst possible symptoms. (7, 14, 21). There are reports of hematological test changes, such as thrombocytopenia and hemolytic anemia (12, 21). One has to consider CSD in the cases with non-clarified elevated body temperature (32). Prolonged fever without local infection can occur in immune-compromised and in immune-competent patients with persistent bacteremia of B henselae and B quintana (36, 41).

In the reported case, we observed the primary affect in the region of the scratch, which is also the entry point for the infection (Fig. 2). We found erythematous, slightly elevated papule, which stayed for about 3 weeks and gradually disappeared. During that time, we did not observed pus
formation in the lesion and the patient didn’t have any complaints related to it. By the time of hospitalization, the patient complained of pain and tension in the parotid and masseter region, lasting 3 days after the beginning of the antibiotic and anti-inflammatory treatment. The typical clinical characteristics suggested CSD. Serological tests found IgG antibodies against Bartonella, with positive titer of 1:512. We started the treatment with parenteral administration of Ciprofloxacin and consecutive per oral Azitromycin and Vibramycin. The analysis of the course of the disease led to the following conclusions: 1. The patient was afebrile with unbiased blood test results; 2. In the beginning the lymph nodes were firm and in the course of the disease all of them did form abscesses in different periods of time. Active antibiotic treatment didn’t affect this process and didn’t achieve regress of the lymphadenopathy. This advocates the statements, that disease is self-healing, and the antibiotic treatment is not always necessary (31). On Fig. 2, Fig. 3 and Fig. 4 we present the course of the disease led to the following conclusions: 1. The patient was afebrile with unbiased blood test results; 2. In the beginning the lymph nodes were firm and in the course of the disease all of them did form abscesses in different periods of time. Active antibiotic treatment didn’t affect this process and didn’t achieve regress of the lymphadenopathy. This advocates the statements, that disease is self-healing, and the antibiotic treatment is not always necessary (31). On Fig. 2, Fig. 3 and Fig. 4 we present the course of the lymphadenopathy. When there was obvious pus formation, we undertook incision and curettage of the affected lymph nodes, which resulted in quick resolution of the local symptoms. The disease took prolonged period to heal completely. From clinical point of view, we observed two distinctive phases of the disease. The characteristics of the first phase were the primary affect on the skin, regional lymphadenitis without suppuration, periadenitis and cellulitis of the affected region. This phase developed over period of 4 weeks. The second phase took longer time, up to 16 weeks, and during this period, we observed successive suppuration of the lymph nodes, with total resolution after treatment.

Treatment of choice is antibiotic therapy. Surgical treatment is recommended for abscess formation. Biopsy of lymph node is undertaken when in doubt for malignant disease.

Although in vitro tests suggest susceptibility of Bartonella henselae to macrolides, tetracycline, rifampicin, third generation of cephalosporins, in vivo there is insufficient treatment result (11). Positive result from erythromycin and doxycycline treatment is reported (42). Recent studies advocate azithromycin usage (15, 33). Moderate susceptibility is found to aminoglycosides, while the microorganism is resistant to first generation of cephalosporins (11). Retrospective study found treatment result in 87% with rifampin and 84% with ciprofloxacin (22). Doxycycline 100 mg every 12 hours is recommended for ocular bartonellosis, for high ocular penetration of the drug. The course of treatment should continue for 2 to 4 weeks in immune competent patients, and four or more months in immune compromised patients (17, 33).

6. Conclusion

CSD is caused by Bartonella henselae. The disease affects more often young patients and is characterized by the symptoms of lymphadenitis. Many different atypical forms of the disease are possible. The most significant epidemiological risk is the contact with kittens and cats with flees. The patients with systemic involvement or immune incompetence are treated with antibiotics. Surgical treatment is needed when there is obvious suppuration or malignant disease is suspected.

References


