Case Report

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Cat-scratch disease in young male: a case report

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ABSTRACT

Cat scratch disease (CSD) is an infectious disease caused by infected cat when it scratches a person hard enough to break the surface of skin. Although it's a rare disease, the frequency of disease is 1 in 10,000 people. Here we are presenting a case of CSD which was diagnosed with the help of HPE and confirmed by history of scratch from patient's pet cat. The patient was successfully treated with antibiotic therapy without any complications. However, prognosis is generally good, negligence of disease may lead to serious complications. A correlation of detailed history with clinical presentation as well as high index of suspicion will aid in accurate diagnosis of CSD.

Keywords: Cat-scratch disease, Histopathology, Lymphadenopathy, Antibiotics

INTRODUCTION

Cat scratch disease (CSD)/cat scratch fever is an infectious disease caused by gram-negative, acid-fast bacilli Bartonella henselae spread by either scratch or bite of an infected cat. Cats could be infected with B. henselae through intradermal inoculation by flea feces containing B. henselae.1 CSD was initially described in the 1930s, and the association of the illness with cats was identified in the 1950s.CSD causes a febrile illness with subacute regional lymphadenopathy. In most cases, there is a spontaneous resolution within 2 to 4 weeks. More severe and disseminated disease can occur in both immunocompetent and immunocompromised hosts. CSD should be considered in the differential diagnosis of any acute, subacute chronic lymphadenopathy.²⁻⁴ Here we are presenting a case of CSD which was diagnosed with the help of HPE and confirmed by retaking history from patient, successfully treated without any complications.

CASE REPORT

A 26-year-old male patient came to OPD with complaints of fever, malaise and swelling in his right axilla from last one week. He complaints of dry cough from last 1 month.

No significant family, psychosocial, medical, drug or surgical history was noted.

General physical examination revealed moderately built, moderately nourished, febrile with temperature 100°c, blood pressure 110/70 mmHg and pulse rate 76/min. No abnormality detected clinically in systemic examination. On local examination he was found to have matted axillary lymphadenopathy (Figure 1). USG of right axilla showed matted lymphadenopathy with abscess.

The lymphnode abscess present in right axilla was drained via I and D with excisional biopsy of lymph nodes done under local anaesthesia. The wound was left open to heal with secondary intention for three days, later suturing was done using ethylon 3-0. The lymph node (Figure 2) specimen was sent for histopathological examination. Patient was advised amoxicillin clavalunate potassium 625 mg twice daily prior to HPE results.

Histopathologic examination

On gross examination a single lymph node measuring $2.0\times1.5\times1$ cm and the cut surface of lymph node shows necrotic gray yellow areas, representative sections were

taken and studied with H and E stain, on microscopic examination lymph node revealed partially affected architecture with subcapsular micro-abscess, stellate necrotizing granulomas composed of central foci of geographical necrosis with neutrophils, cell debris surrounded by palisades of epithelioid histiocytes, the paracortical zone showed vascular proliferation and sinus are filled with monocytoid B cells which is typical of CSD was seen.

Surprisingly on repeated enquiry, the patient gave a history of scratch from his pet cat two months ago. Then antibiotic was shifted from amoxicillin clavalunate to azithromycin 500 mg bd for 7 days after confirming the diagnosis as CSD. Then the lymphadenopathy was resolved within 3 weeks. The patient was completely cured with no fresh complaints in four months of follow-up.



Figure 1: Right axillary lymphadenopathy.



Figure 2: Gross specimen of lymph node.

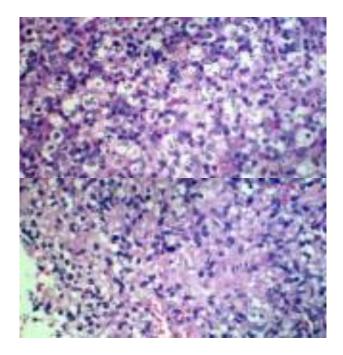


Figure 3: Microscopy of lymph node showing microabscess.

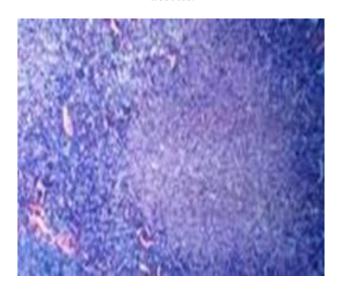


Figure 4: Microscopy of lymph node showing palisades of histiocytes.

DISCUSSION

CSD has two clinical forms: 1. Typical CSD: This is more common form characterized by sub-acute regional lymphadenopathy (most common being axillary lymph nodes) and painless erythematous papule or pustule, that develops at the site of cat scratch 2. Atypical CSD: It is characterized by extra nodal manifestations such as hepatitis, splenitis, and retinitis. The wide spectrum of diseases connected with these bacteria ranges from asymptomatic cases, to skin inflammation, fever of unknown origin, lymphadenopathy, eye disorders, encephalitis and endocarditis. In immunocompromised patients, *Bartonella* sp. can cause opportunistic infections

like bacillary angiomatosis and peliosis hepatitis. In immunocompetent individuals, B. henselae infection presents as cat-scratch disease (CSD). The reservoirs of B. henselae are domestic animals like cats, guinea pigs, rabbits and occasionally dogs. In Poland, positive IgG antibodies for B. henselae are found in 50-90% of cats. The cat flea Ctenocephalides felis (Siphonaptera: Pulicidae) is the best-recognized vector henselae and transmission between cats and humans which mainly occurs through infected flea faeces. People become infected by being bitten or scratched by an infected animal.⁶ Bartonella infection in humans produces prolonged bacteraemia in blood.⁷

In typical CSD, regional lymphadenopathy occurs 1 to 3 weeks after inoculation and lasts for up to several months. Eighty-five per cent of patients have only a single node involvement. Asymmetric lymphadenopathy occurs most frequently in the axillary and epitrochlear nodes (46%), head and neck (26%), and the groin (17.5%). The lymph nodes are painful and movable with solid consistency. In 20-30% of patients, inflamed lymph nodes produce suppuration with purulent fistulas to the skin; approximately 10% of nodes require drainage. About 50% of patients present CSD with mild systemic symptoms like generalized aches, malaise, anorexia, nausea, and abdominal pain.8 The disease is benign in character in a majority of patients. Surgical removal of involved lymph nodes or biopsy of lymph nodes or inoculation sites is not necessary for diagnosis or management. To establish a diagnosis of CSD in patients presenting with superficial lymphadenopathy in one isolated area, we propose the use of an etiological approach which consists of looking first for the presence of B. henselae DNA by PCR analysis. In the case of PCR positivity, CSD may be retained on account of the excellent specificity. In the case of a negative PCR result, the diagnosis could rely on the presence of at least two of the following criteria: (i) positive serology, (ii) histology compatible with CSD, The definitive diagnosis of CSD is one by special stain Warthin starry, stain which is positive for small rod like bacterial organism *B. henselae* which are seen around the blood vessels and Immunohistochemistry (iii) contact with cats during the days or weeks preceding lymphadenopathy, together with elimination of any other cause of lymph node enlargement.9 Treatment of CSD depends on the disease presentation. Most patients, especially children, have self-limited lymphadenopathy lasting two to eight weeks and do not require antibiotics. Up to 14% of persons develop dissemination to the liver, spleen, eye, or central nervous system and antibiotics may help. 10 Suppurative nodes should be drained by large-bore needle aspiration and not by incision and drainage in order to avoid chronic draining tracts. Immunocompromised patients must always be treated with systemic antimicrobials. Typical cat-scratch disease-antibiotics not routinely indicated but for patients with extensive.¹¹

Some unusual presentation of CSD published in database are as follows-3 of cases Parinaud's oculo-glandular syndrome, cough (AIDS), encephalitis, solitary splenic abscess with no lymphadenopathy. 12-15 TB with CSD, mesenteric lymphadenitis transformed into arthritis of hand joints, erythema nodosum with CSD. 16-18 Here the presented case has atypical presentation of cough. The patient was successfully responded to antibiotic therapy and lymphadenopathy was resolved within one month. He was completely cured with no fresh complaints in four months of follow-up. The clinicopathological correlation revealed history of cat as pet and cat scratch in recent period.

CONCLUSION

However, the number of reported cases of CSD has been low, there has been a significant increase in incidence of cases in urban and suburban areas which may be due to increased rearing of kittens at home and negligence to vaccinate their cats. However, prognosis is generally good, recover within 4 months, negligence of disease may lead to serious complication. A correlation of detailed history with clinical presentation as well as high index of suspicion will aid in accurate diagnosis of CSD.

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