ISSN: 0975-833X

Available online at http://www.journalcra.com

International Journal of Current Research
Vol. 9, Issue, 10, pp.59781-59783, October, 2017

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

RESEARCH ARTICLE

CAT-SCRATCH DISEASE AND MANAGEMENT

***Jembulingam Sabarathinam and Dr. Preetha**

Department of Physiology, Saveetha Dental College, Chennai 77, Tamilnadu, India

ARTICLE INFO

Article History:
Received 20th July, 2017
Received in revised form
10th August, 2017
Accepted 15th September, 2017
Published online 31st October, 2017

Key words:
Act, Childhood disease,
Cat scratch syndrome.

ABSTRACT

Cat-scratch disease (CSD) is a common and usually a contagious disease caused by the bacterium bartonella *henselae* It is most commonly found in children following a scratch or bite from a cat in the household within about one to two weeks. The current best diagonstic tool is the Polymerised chain reaction which gives accurate results within a short span of time. The preferred antibiotic for treatment is azithromycin since this agent is the only one studied in a randomized controlled study.

Citation: Jembulingam Sabarathinam and Dr. Preetha, 2017. "Cat-Scratch disease and management", *International Journal of Current Research*, 9, (10), 59781-59783.

INTRODUCTION

Cat-scratch disease (CSD) is one of the most prevalent infections among humans caused by a microorganism *Bartonella* species. Cat-scratch disease has an international distribution of affected individuals and has been more prevalent in all the areas of North America. This CSD occurs mostly in humid and warm weather conditions which is the usual climate in North America which supports its increased occurrences of this diseased condition. Approximately there are about 22,000 new cases of Cat-Scratch disease in the United States of America. (Jackson *et al*., 1993) *Bartonella henselae* is the causative bacteria which causes Cat-Scratch disease. It has been observed in feline erythrocytes and fleas, which contaminates saliva and induces the disease to humans by biting or clawing of cats. So cats are the transmitting agents of Cat-scratch disease which can be stray cats or domestic pet cats. The cat flea, *Ctenocephalides feli, is* the vector which is responsible for transmission of the disease between cats (Zangwill *et al*., 1993), and the infected cats bite can cause human infections also. In addition tick bites can transmit more bacteria to the humans. Over 50% of the cats host the bacteria and are completely asymptomatic in nature. (Massei *et al*., 2005)

Symptoms and Clinical findings

Cat-Scratch disease is commonly prevalent among children, but adults may also get affected by this disease. The important symptom and clinical finding of this disease is unilateral lymphadenopathy with a history of exposure to kittens and cats. Studies have proven that localized lymphadenopathy is caused by CSD4. When the patient is exposed to the surface of the infected cat (Massei *et al*., 2005), they are vulnerable to primary skin lesions which start as a vesicle at the inoculation site. Only a fraction people do not develop skin lesions when they are in contact with the infected cats. Lymphadenopathy is developed after couple of days following the exposure to infected cats which are usually characterized by ipsilateral. According to studies the lymphadenopathy is regional and about 46% of the population develop lymphadenopathy in upper extremities, while 26% develop lymphadenopathy in jaw and neck region and 18% develop in groin and 10% develop lymphadenopathy in other areas including chest (Carithers, 1985). On examination and palpation of lymph nodes, the lymph nodes appear tender, swollen and supprative. About two-third of the infected population develop complications such as aching, anorexia and malaise while one-third develop low-grade fever (Carithers, 1985). The other complications include bone related disorders like myalagia, arthralgia, and arthritis which are common in infected patients. (Maman *et al*., 2007) Even visceral organs are found to be affects in this disease where the manifestations include hepatosplenomegaly (Margileth *et al*., 1987; Lenoir *et al*., 1988). Prolonged fevers of unknown origin in children are described (Tsujino *et al*., 2004; Jacobs and Schutze, 1998). Rare cases of mengititis and eye related disorders are reported in immunocompetent patients. (Wong *et al*., 1995; Baorto *et al*., 1998) Only one neurological manifestation of CSD is encephalopathy, which has symptoms of severe headache and acute confusion on

Corresponding author*: **Jembulingam Sabarathinam,
Department of Physiology, Saveetha Dental College, Chennai 77, Tamilnadu, India.

occurrence of lymphadenopathy (Wong *et al*., 1995). Parinaud oculoglandular syndrome is the most prevalent ocular manifestation (Cunningham and Koehler, 2000) and consists of granulomatous conjunctivitis and ipsilateral periauricularly mphadenopathy. In immune suppressed patients, *B.henselae* can cause bacillary angiomatosis and peliosis (Koehler and Tappero, 1993). Liver and sometimes spleen is affected by bacillary pelicosis. *B.henselae* and *bartonella Quintana*, which involves skin and bones. It can also affect internal organs. lesions consist of red and purple papules along with internal bleeding like purpura13. Patients suffering from AIDS and having a low CD4 cell count (Regnery *et al*., 1995) where the first to get affected by bacillus angiomatosis. Immunodeficient people are much more affecting by these diseases and high prevalence is seen in sanfransico and brazil (Lamas *et al*., 2010; Koehler *et al*., 2003).

to be conducted to identify the infection. (Sander *et al*., 1998; Spach and Kaplan, 2010) A positive immunoglobulin M test suggest acute disease, but production of this immunoglobulin is brief and does not have significant relation to *Bartonella* as that of immunoglobulin G which has a cross reactivity between *B.quintana* and *B.henselae*. PCR (polymerase chain reaction) can also detect different *Bartonella* species. Lymphnode biopsy of the CSD patient shows lymphoid hyperplasia and stellate granulomas. *B.henselae* microscopically is small curved and aerobic gram negative bacilli that stain by special silver stains with silver as key component. In bacillary angiomatosis, histologically, lobular proliferation occurs of small blood vessels in the presence of bacilli in adjacent connective tissue. In a study of about 786 lymph node specimens in whom CSD was suspected, only 245 patients (31%) had evident CSD while 13 of 245 patients had concurrent neoplasms which proves that

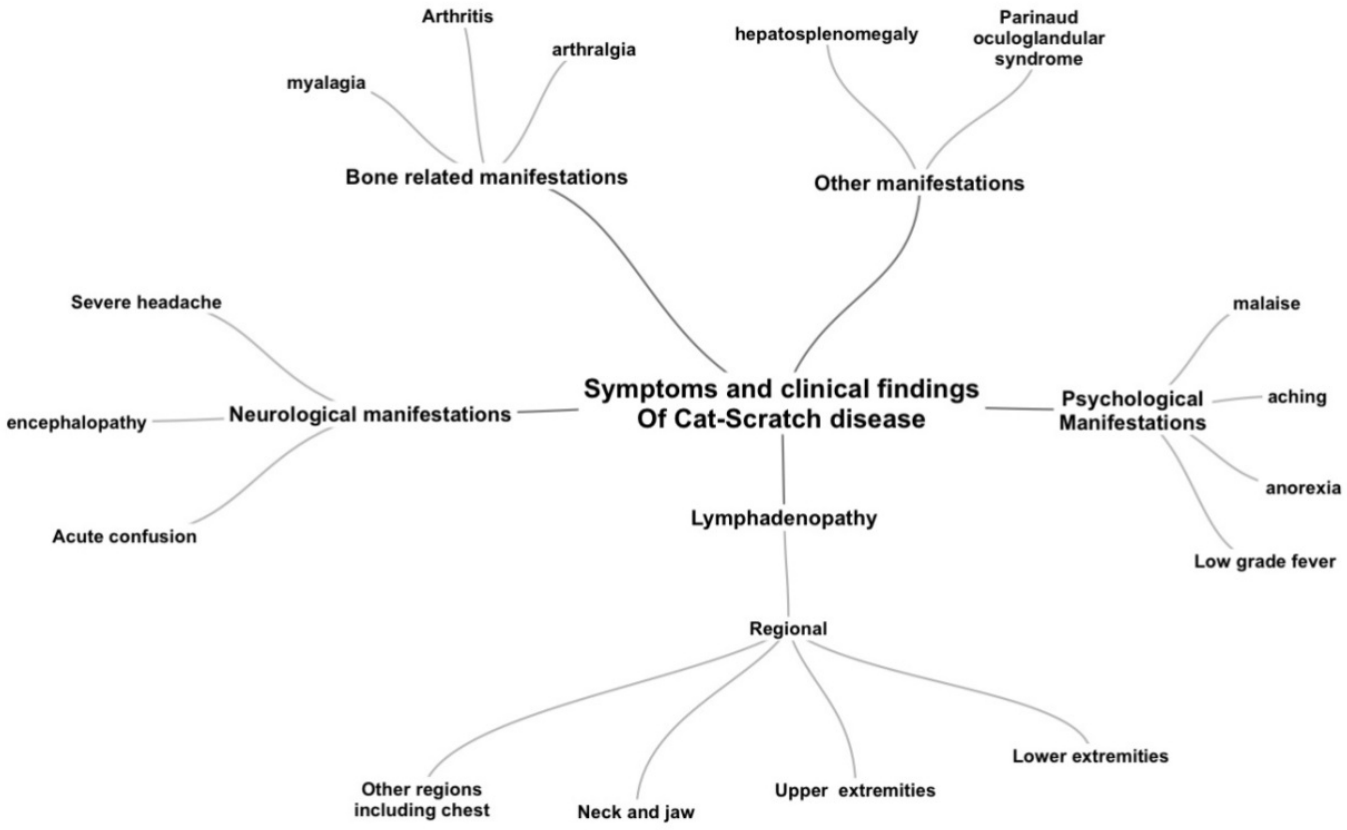

Figure 1. Signs and symptoms of CSD's

Diagonsis

The *Bartonella species* are very difficult to isolate by culture because of their high specificity androtinue culture methods are not advisable, where in serological test are the best preliminary test which is performed by ELISA (enzyme linked immunosorbent essay) and indirect immunoflurocscentassay. Although serological test are more sensitive than culture, serological test lack specificity because the population infected with asymptomatic condition also turns out to be positive (Bergmans *et al*., 1997) because of previous exposure to cats. The cat owners had high serological positive test when compared to our general population (Bergmans *et al*., 1997). There is specific range for immunoglobulin G titre where if the titre is less than 1:64 then the patient is not infected by *bartonella* infection, while if the titre is between 1:64 and 1:256 represents possibility of being infected while more than 1:256 are strong indicators of this disease. Repeated test have unilateral lymphadenopathy is seen in patients with Cat-scratch disease (Rolain *et al*., 2006).

Treatment

Treatment of the Cat-Scratch syndrome depends on the signs and symptoms, where children with self-limited lymphadenopathy lasting a couple of weeks do not require whereas adults with dissemination of liver, spleen and central nervous system 4 need antibiotics to help relieve from their problems (Rolain *et al*., 2004) In a study from 1985, a single investigator who evaluated 1200 patients with lymphadenopathy 4 who ever believed to have CSD found that antibiotics were rarely used and their effect was minimal. In a randomized control trial of oral administration of azithromycin 500mg/day22, for 29 adult patients, the use of azithromycin led to rapid resolution of lymphadenopathy than placebo, were 8 people out of 14 were taking this antibiotic had 80% more

resolution at 30 days when compared to placebo. (Bass *et al*., 1998) There are two contradicting recommendation in united states where the infectious disease society of America guidelines suggests (Stevens *et al*., 2005) and favors the use of antibiotics for CSD while there is a panel of authorities which restrict its use against patients with mild or uncomplicated disease. Other antibiotics used are Rifarmpin, ciprofloxacin, trumethprin and gentamycin (Margileth, 1992).

REFERENCES

Baorto E, Payne RM, Slater LN, *et al*. 1998. Culture-negative endocarditis caused by Bartonella henselae. *J Pediatr*., 132(6):1051-1054.

Bass JW, Freitas BC, Freitas AD, *et al*. 1998. Prospective randomized double blind placebo-controlled evaluation of azithromycin for treatment of cat-scratch disease. *Pediatr Infect Dis J*., 17(6):447-452.

Bergmans AM, Peeters MF, Schellekens JF, *et al*. 1997. Pitfalls and fallacies of cat scratch disease serology: evaluation of Bartonella henselae-based indirect fluorescence assay and enzyme-linked immunoassay. *J ClinMicrobiol*., 35(8):1931-1937.

Carithers HA. 1985. Cat-scratch disease. An overview based on a study of 1,200 patients. *Am J Dis Child*, 139(11):1124-1133.

Cunningham ET, Koehler JE. 2000. Ocular bartonellosis. *Am J Ophthalmol*., 130(3):340-349.

Jackson LA, Perkins BA, Wenger JD. 1993. Cat scratch disease in the United States: an analysis of three national databases. *Am J Public Health*, 83(12):1707-1711.

Jacobs RF, Schutze GE. 1998. Bartonella henselae as a cause of prolonged fever and fever of unknown origin in children. *Clin Infect Dis*., 26(1):80-84.

Koehler JE, Sanchez MA, Tye S, *et al*. 2003. Prevalence of Bartonella infection among human immunodeficiency virus-infected patients with fever. *Clin Infect Dis*., 37(4):559-566.

Koehler JE, Tappero JW. 1993. Bacillary angiomatosis and bacillary peliosis in patients infected with human immunodeficiency virus. *Clin Infect Dis*., 17(4):612-624.

Lamas CC, Mares-Guia MA, Rozental T, *et al*. 2010. Bartonella spp. infection in HIV positive individuals, their pets and ectoparasites in Rio de Janeiro, Brazil: serological and molecular study. *Acta Trop*., 115(1-2):137-141.

Lenoir AA, Storch GA, DeSchryver-Kecskemeti K, *et al*. 1988. Granulomatous hepatitis associated with cat scratch disease. *Lancet*, 1(8595):1132-1136.

Maman E, Bickels J, Ephros M, *et al*. 2007. Musculoskeletal manifestations of cat scratch disease. *Clin Infect Dis*., 45(12):1535-1540.

Margileth AM, Wear DJ, English CK. 1987. Systemic cat scratch disease: report of 23 patients with prolonged or recurrent severe bacterial infection. *J Infect Dis*., 155(3): 390-402.

Margileth AM. 1992. Antibiotic therapy for cat-scratch disease: clinical study of therapeutic outcome in 268 patients and a review of the literature. *Pediatr Infect Dis J*., 11(6):474-478.

Massei F, Gori L, Macchia P, Maggiore G. 2005. The expanded spectrum of bartonellosis in children. *Infect Dis Clin North Am*., 19(3):691-711.

Regnery RL, Childs JE, Koehler JE. 1995. Infections associated with Bartonella species in persons infected with human immunodeficiency virus. *Clin Infect Dis*., 21(suppl 1):S94-S98.

Rolain JM, Brouqui P, Koehler JE, Maguina C, Dolan MJ, Raoult D. 2004. Recommendations for treatment of human infections caused by Bartonella species. *Antimicrob Agents Chemother*., 48(6):1921-1933.

Rolain JM, Lepidi H, Zanaret M, *et al*. 2006. Lymph node biopsy specimens and diagnosis of cat-scratch disease. *Emerg Infect Dis*., 12(9):1338-1344.

Sander A, Posselt M, Oberle K, Bredt W. 1998. Seroprevalence of antibodies to Bartonella henselae in patients with cat scratch disease and in healthy controls: evaluation and comparison of two commercial serological tests. *Clin Diagn Lab Immunol*., 5(4):486-490.

Spach DH, Kaplan SL. 2010. Microbiology, epidemiology, clinical manifestations, and diagnosis of cat scratch disease. UpToDate. http://www.uptodate.com. Accessed September 20, 2010.

Stevens DL, Bisno AL, Chambers HF, *et al*. 2005. Infectious Diseases Society of America. Practice guidelines for the diagnosis and management of skin and soft-tissue infections. *Clin Infect Dis*., 41(10):1373-1406.

Tsujino K, Tsukahara M, Tsuneoka H, *et al*. 2004. Clinical implication of prolonged fever in children with cat scratch disease. *J Infect Chemother*., 10(4):227-233.

Wong MT, Dolan MJ, Lattuada CP Jr, *et al*. 1995. Neuroretinitis, aseptic meningitis, and lymphadenitis associated with Bartonella (Rochalimaea) henselae infection in immunocompetent patients and patients infected with human immunodeficiency virus type 1. *Clin Infect Dis*., 21(2):352-360.

Zangwill KM, Hamilton DH, Perkins BA, *et al*. 1993. Cat scratch disease in Connecticut. Epidemiology, risk factors, and evaluation of a new diagnostic test. *N Engl J Med*., 329(1):8-13.

****