Cat Scratch Disease Presenting as Acute Mastoiditis

Veronique Wan Fook Cheung, MDCM1; J.Paul Moxham, MD1,2

1University of British Columbia, Division of Otolaryngology-Head and Neck Surgery
2BC Children’s Hospital, Division of Pediatric Otolaryngology

ABSTRACT

Objective: To present the first published case of Cat Scratch Disease presenting as acute mastoiditis and review the relevant literature to discuss the Otolaryngologic manifestations of this disease and its treatment.

Design: A case report and literature review of the Otolaryngologic manifestations of Cat Scratch Disease.

Methods: A case report of a clinical scenario followed by a standard literature review. PubMed, EMBASE, and Cochrane database were used to find articles related to the Otolaryngologic manifestations of Cat Scratch Disease.

Results: A 6 year-old female presented to the Otolaryngologist with the typical appearance of acute mastoiditis. CT Scan confirmed breakdown of the osseous septae of the mastoid and mastoidectomy was undertaken. Granulation tissue and infected lymph nodes adjacent to the mastoid cortex were positive for Cat Scratch Disease. The patient was treated expectantly and recovered uneventfully.

Conclusion: This is the first literature report of Cat Scratch Disease presenting as an acute mastoiditis.

Keywords: Mastoiditis, Cat-Scratch Disease, Bartonella

CASE REPORT

A 6 year-old girl presented to her community Otolaryngologist with a past medical history of chronic otitis media with effusion and previous ventilation tube surgeries. She had an acute worsening of a presumed episode of acute otitis media with low-grade fever (38°C), bulging right tympanic membrane and a postauricular fullness with displacement of the auricle. Presuming acute mastoiditis, a CT Scan was undertaken which confirmed destruction of bony septae. She was taken to the operating room and a cortical (simple) mastoidectomy and myringotomy and tube placement was accomplished. There was purulent material in the middle ear but the mastoid appeared filled with thin fluid and granulation tissue. There was no evidence of cholesteatoma. Biopsy of the granulation tissue was reported as noncaseating granulomas with chronic inflammatory change.

Post-operatively, she developed a chronic serous drainage through the post-auricular incision with increasing non-tender lymphadenopathy post-auriculally and in the upper neck. After several courses of antibiotics (amoxicillin, clindamycin, and clarithromycin) without any change, she was transferred to BC Children’s Hospital for management.

A repeat CT Scan showed a mastoid cavity with soft tissue in it (Figure 1). A differential diagnosis including Langerhan’s Cell Histiocytosis, atypical mycobacterial infection, missed cholesteatoma, or malignancy mandated a revision mastoidectomy and lymph node biopsy, which was accomplished. Two liquefied lymph nodes post-auriculally and extensive mastoid cavity granulations were removed and sent to pathology and microbiology. The cavity was cleaned and smoothed thoroughly. Antibiotic soaked gelfoam was placed in the cavity and presuming atypical mycobacterial infection, the patient was placed on Rifampin and Clarithromycin. The wound was closed. The ear was inspected and found to be clear with a patent ventilating tube in place.

DISCUSSION

Cat Scratch Disease (CSD), although seen in all ages, is the most common cause of unilateral lymphadenopathy in children (other than non-specific viral and bacterial adenitis) [1]. It usually presents 3-10 days after being in contact with an infected cat: non-tender red-brown papule of 1-10 mm would be noted at the site of contact; regional lymphadenopathy about 3 weeks after inoculation is also seen in 80% of cases of CSD. Affected lymph nodes depend on the site of inoculation, with 37% of cases having multiple lymphadenopathy sites; however, no previous documentation of CSD presenting with mastoiditis was found. Due to its non-specific presentation, CSD should be considered in the differential diagnosis of mastoiditis, especially in presence of unilateral lymphadenopathy and a history of animal (especially cat) contact. Serological antibodies testing and PCR for detection of Bartonella Henselae should thus be included in the investigation to rule out this fastidious and often unrecognised pathogen.

RESULTS

Pathology (Figure 2) showed necrosis with granulation tissue with acute and chronic inflammatory infiltrate, with microabscesses surrounded by histiocytes and fibroblasts in a palisading arrangement (stellate abscesses); multinucleated macrophages were also present. These findings were suggestive of Cat Scratch disease. There were no suggestion of malignant processes, nor presence of fungus or acid-fast organisms on special stains.

Acid-fast bacilli (AFB) smear and culture of mastoid tissue and cat flea, Ctenocephalides felis, is the main vector of transmission for Bartonella henselae [2]. Bartonella henselae is the causative agent of Cat Scratch disease [2,3]. Bartonella henselea is a challenging organism to diagnose especially when there is no reported history of animal contact or if the primary infection site had healed. Culture of this fastidious gram-negative bacillus usually ends in false negative results. Papules and pustules along cat scratches can easily be overlooked, and enlarging lymph nodes that appear 3 weeks after inoculation are not usually associated with the infection [2]. Lymphadenopathy due to Bartonella Henselae is mostly self-limiting, lasting 2 to 4 months, but may lead to suppuration and abscess formation. Rare cases lasting up to 2 years have been reported. Systemic symptoms such as fever are rare (25%) [3].

In terms of management, prevention measures of infection include hand washing after close contact with animals or animal bites cleaning with mild soap and water. Control of vector (flea) infestation is important when immunocompromised patients are involved [3]. Surgical intervention is usually delayed in self-limiting cases of CSD, but may be useful for symptomatic relief of painful lymph node abscesses. Although there is no standardized antibiotic regimen in the treatment of CSD, azithromycin (oral for 5 days) has been shown to decrease lymphadenopathy if administered within the first 30 days of illness. No significant difference was seen with treatment 2 months and 4 months post infection, and treatment does not prevent suppuration [6]. Our patient was well past 4 months post infection, but was treated with Rifampin with Clarithromycin successfully. The wound closed without further complications and patient was seen to be fully healed at the 1 year follow-up.

CONCLUSIONS

CSD should be considered in the differential diagnosis of mastoiditis, especially in presence of unilateral lymphadenopathy and a history of animal (especially cat) contact. Serological antibodies testing and PCR for detection of Bartonella Henselae should thus be included in the investigation to rule out this fastidious and often unrecognised pathogen.

REFERENCES