

Cat Scratch Disease Revealed with Elbow Swelling in 10 Year-Old Child: Case Report

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Abstract

Case Report

Introduction: Cat Scratch Disease (CSD), caused by *Bartonella henselae*, is the most common *Bartonella* infection in humans, typically presenting as self-limiting regional lymphadenopathy following a cat scratch or bite. **Case Presentation:** A 10-year-old child developed an atypical form of CSD, characterized by significant elbow swelling and abscess formation. Diagnosis was confirmed through imaging and PCR testing. The case required surgical drainage and antibiotic therapy, highlighting the variability in CSD presentation. **Discussion:** CSD is primarily transmitted through household cats, with higher incidence in children and seasonal peaks in fall and winter. While most cases resolve without intervention, severe forms may involve systemic or visceral complications. Diagnosis relies on clinical evaluation, serology, and PCR. Imaging techniques such as ultrasound and MRI are essential for identifying atypical presentations. **Conclusion:** CSD can manifest beyond typical lymphadenopathy, necessitating early recognition and appropriate management. In pediatric patients with suspected hepatosplenic involvement, abdominal ultrasound is recommended. Though often self-limiting, severe cases may require prolonged antibiotic therapy and surgical intervention. **Keywords:** *Bartonella henselae*, elbow swelling, cat-scratch disease, child, lymphadenopathy.

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INTRODUCTION

Bartonella henselae is the primary causative agent of Cat Scratch Disease (CSD), the most common form of human infection with *Bartonella*. In the United States, around 12,000 outpatient cases and 500 hospitalizations for CSD occur annually [1, 2]. Cats, the main reservoir, often become asymptotically infected after being bitten by cat fleas (*Ctenocephalides felis*), younger cats are more frequently infected [2, 3].

The U.S. Public Health Service and the Infectious Diseases Society of America have issued guidelines to reduce the risk of *Bartonella* transmission from cats to humans [4]. Studies suggest that approximately 51% of domesticated cats in the U.S. are chronically infected with *B. henselae*, and 24% have bacteremia [5]. Clinically, CSD typically presents as self-limiting regional lymphadenopathy following a cat scratch or bite [6].

Tick bites and animal exposure have also been associated with a 9-46% seroprevalence of *B. henselae* antibodies in humans [7-10]. CSD has been recognized for over 100 years, often presenting as prolonged regional lymphadenopathy in healthy individuals,

though atypical cases, affecting 5-14% of patients, may include systemic symptoms such as prolonged fever, myalgia, arthralgia, fatigue, weight loss, and Parinaud's oculoglandular syndrome [11, 12]. Serious complications like endocarditis, bacillary angiomatosis, and bacillary peliosis hepatis can also occur, particularly in children and immunocompromised individuals [13,14].

The identification of *B. henselae* as the causative agent of CSD was a major breakthrough in microbiology, as the disease's etiology remained a mystery for many years [15, 16]. Interestingly, *B. henselae* was first isolated not from CSD patients, but from bacteremic, immune-compromised individuals [17]. Experimental evidence confirms that cat fleas can transmit *B. henselae* between cats [18].

OBSERVATION

A 10-year-old child with a history of asthma, managed with intermittent Ventolin use, presented with left axillary adenopathy and a sore throat but no fever. Initial treatment with amoxicillin-clavulanate and ceftriaxone showed no improvement after four days.

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The patient remained in good general condition, afebrile, with stable hemodynamics and respiratory status. Clinical examination revealed a warm, painful swelling on the anteromedial aspect of the left forearm, while the left elbow joint was unaffected. The left axillary adenopathy showed no signs of inflammation. Abdominal examination was unremarkable, with no hepatomegaly, splenomegaly, or hernial abnormalities detected.

Laboratory results showed an elevated C-reactive protein level of 60 mg/L and a white blood cell count of 9,050/ μ L. Imaging studies included a left elbow X-ray, which revealed significant subcutaneous soft tissue thickening, suggestive of probable posteromedial epitrochlear cellulitis (Figure 1). Left elbow ultrasound demonstrated widespread synovial thickening without bone lesions, cortical irregularities, or periosteal apposition, a mixed hypoechoic lesion (14 x 20 mm) with irregular contours and peripheral hypervascularization, along with central venous flow, suggested a possible infectious focus or mass, with no intra-articular effusion

(Figure 2). Abdominal ultrasound confirmed the absence of hepatomegaly or splenomegaly. Left elbow MRI identified an intramuscular mass at the anteromedial aspect of the distal biceps brachii, highly suggestive of an abscess, though a tumor could not be entirely ruled out (Figure 3). Surgical intervention revealed thick, compartmentalized pus mixed with subcutaneous fatty tissue, confined to the muscle without bone involvement.

One week after incision and drainage of the elbow mass, the left axillary adenopathy became suppurative (Figure 4), confirmed by ultrasound (Figure 5), necessitating a second surgical intervention for drainage. Bartonella serology was positive, with PCR confirming *Bartonella henselae* infection. Following axillary drainage, the patient was treated with a five-day course of azithromycin. Due to persistent purulent discharge from the axillary site, clindamycin was added for an additional 15 days. Upon follow-up, the scars on the left elbow and axilla were clean, with no signs of residual infection.



Figure 1: X-ray of left elbow: Significant subcutaneous soft-tissue thickening

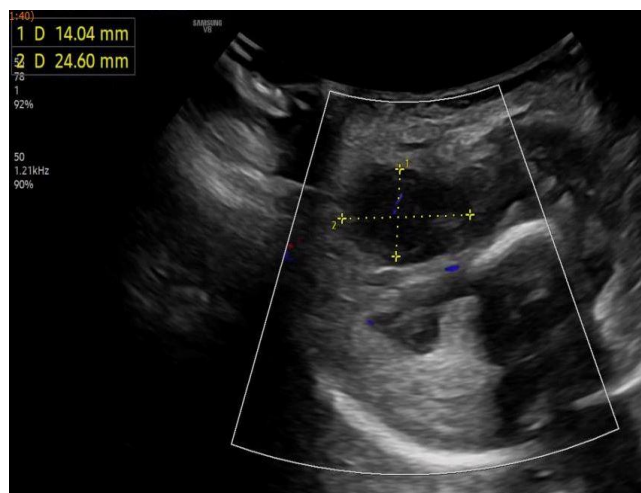


Figure 2: Ultrasound of left elbow: posterior soft tissue thickening (9 mm), mixed hypoechoic lesion (14 x 20 mm)



Figure 3: Left elbow MRI performed in sagittal, frontal, and transverse views: intramuscular mass at the anteromedial aspect of the distal biceps brachii



Figure 4: Left axillary adenopathy

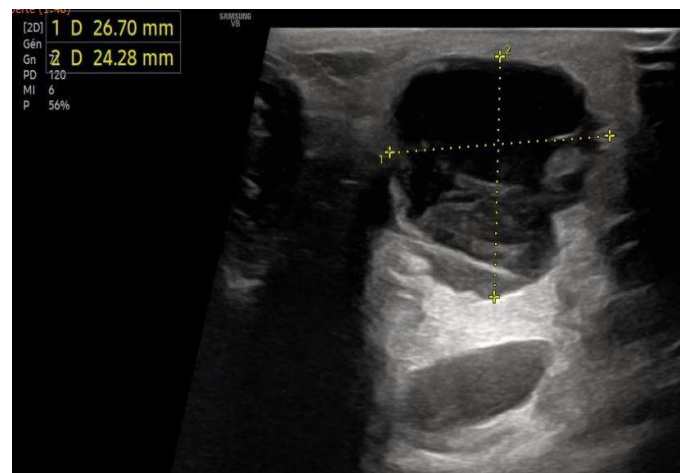


Figure 5: Left axillary ultrasound: Adenopathy measures 26 x 24 mm with a collected and partitioned part, suggesting infectious adenitis in part collected

DISCUSSION

Household cats serve as the primary reservoir for *B. henselae*, with seroprevalence varying geographically. Studies have reported that up to 50% of cats have antibodies against this bacterium, with higher infection rates in stray or multi-cat households [19-21]. In France, regional studies identified seroprevalence rates of 36% among pet cats and higher infection rates in stray cats [24, 25].

Seasonality plays a role in the incidence of CSD, peaking in autumn and winter. Studies in the U.S. and Japan have shown seasonal variations, with most cases occurring from September to December, coinciding with increased flea activity [22, 23, 27]. Our patient had a history of close contact with kittens, aligning with known risk factors for *Bartonella* infection.

Classically, CSD presents with regional lymphadenopathy, often following a cat scratch or bite. Lymph nodes become tender and may suppurate in about 10% of cases [28]. However, our patient exhibited suppurative lymphadenopathy requiring drainage, a higher rate than the 10% reported in the literature, aligning more closely with studies indicating an incidence of 21.1% for abscess formation in lymph nodes [36].

Musculoskeletal involvement is relatively uncommon, affecting approximately 10% of patients and typically manifesting as myalgia, arthralgia, or arthritis [29]. Our case presented with a rare intramuscular abscess without joint involvement, a less frequently reported complication. Similar cases have been documented in the literature, but they remain exceptional, emphasizing the need for a high index of suspicion in atypical presentations.

Visceral involvement, including hepatosplenomegaly, has been documented in 30% of pediatric patients with Parinaud's oculoglandular

syndrome (PFS) caused by *B. henselae* [32]. While our patient did not show hepatosplenic involvement, Nawrocki *et al.*, found that children under 14 years had a 1.76 times higher risk for such complications than those older than 15 years [21]. This reinforces the importance of abdominal ultrasound in suspected disseminated cases [37].

Serological testing remains the cornerstone of *B. henselae* diagnosis. PCR offers high specificity but lower sensitivity than serology [19-20]. The timing of serological testing is crucial; IgM has a short-lived presence (~3 months) and may be undetectable early in the disease course [38, 39]. Our case was confirmed via serology and PCR, highlighting the importance of multimodal diagnostic approaches when clinical suspicion is high.

Cat Scratch Disease is generally a self-limiting condition, with most cases resolving within 2 to 6 months without antibiotic treatment [40]. However, up to 14% of cases may involve systemic dissemination to organs such as the liver, spleen, eye, or central nervous system (CNS), where antibiotics can be beneficial [40]. A randomized trial supports the use of a 5-day azithromycin regimen for mild to moderate cases, adjusted based on patient weight [41, 42]. Antibiotic therapy is generally reserved for complicated cases. Persistent lymphadenopathy (>30 days) and febrile syndrome are the most common indications for treatment [43]. Severe or disseminated infections may require additional agents such as rifampicin, ciprofloxacin, gentamicin, or trimethoprim-sulfamethoxazole, though their efficacy varies [20, 45]. In our case, initial empirical treatment with amoxicillin-clavulanate and ceftriaxone was ineffective, consistent with the known resistance of *Bartonella henselae* to β -lactam antibiotics. Therefore, our case required azithromycin followed by clindamycin due to persistent purulent discharge, consistent with recommendations for complicated cases [43-45].

CONCLUSION

Cat Scratch Disease presents with a broad clinical spectrum, ranging from self-limited lymphadenopathy to severe systemic complications. Recognizing atypical manifestations, such as musculoskeletal or visceral involvement, is crucial for timely diagnosis and appropriate management. While most cases resolve without intervention, severe or persistent infections may require prolonged antibiotic therapy and surgical drainage. In pediatric patients, abdominal ultrasound should be considered when hepatosplenic involvement is suspected. A multidisciplinary approach, combining clinical evaluation, imaging, microbiological confirmation, and targeted treatment, is essential to optimize patient outcomes. Increased awareness of CSD's varied presentations helps ensure early diagnosis, avoid unnecessary treatments, and prevent complications.

Competing interests: The authors declare no competing interest.

Author Contributions:

All authors contributed to the creation of this article. The authors also declare that they have read and confirmed the final version of this article.

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