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An unusual presentation of cat-scratch disease

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Abstract
Cat-scratch disease (CSD) is a relatively common entity, particularly in children. The most common sites of involvement are the axillary lymph nodes, followed by cervical, pre-auricular and submandibular lymph nodes. We present a case of cat-scratch disease in which the initial clinical features were indistinguishable from those of acute bacterial tonsillitis with jugulodigastric lymphadenopathy. This previously unreported apparent mode of presentation is discussed with reference to the current understanding of the disease.

Key words: Cat-Scratch Disease; Lymph Nodes; Tonsillitis

Introduction

Cervical lymphadenopathy is commonly seen in children, usually secondary to viral or bacterial oropharyngeal or cutaneous infection. Other causes include specific microbial infection (such as tuberculosis), inflammatory disorders and neoplasia. A careful history and physical examination along with laboratory tests may be required in order to reach a diagnosis.

Cat-scratch disease (CSD), historically known as benign non-bacterial regional lymph-adenitis, is considered to be a relatively common cause of lymphadenopathy in children and young adults.[1] The most common sites of involvement are the axillary, cervical, pre-auricular and submandibular lymph nodes,[2] although an array of clinical abnormalities may arise.

We present a case of CSD masquerading as acute tonsillitis with reactive cervical lymphadenitis. The clinical course and subsequent diagnosis in this case highlight the importance of considering CSD in the differential diagnosis of cervical lymphadenitis in children.

Case report

A 10-year-old female presented with a four-day history of fever, odynophagia and right-sided neck swelling. She had been vomiting for the preceding 24 hours. Relevant past medical history included recurrent acute tonsillitis. There was no stridor nor dysphonia.

Clinical examination revealed an unwell-looking child who was pyrexial (temperature 38° Celsius). The right tonsil was enlarged and there was bilateral Level II cervical lymphadenopathy, more marked on the right. The lymph nodes were firm, tender, non-fluctuant with erythema of the overlying skin. A provisional diagnosis of acute tonsillitis/infectious mononucleosis was made and the child was admitted for intravenous fluids and benzylpenicillin together with analgesia.

The white cell count was within the normal range but with lymphopenia present (lymphocyte count 0.9). A glandular fever screen and blood cultures were negative and throat swab cultured normal flora only. An ultrasound scan of the neck was performed on day two, demonstrating extensive right-sided Level II lymphadenopathy but no evidence of suppuration. By day three, the child's pyrexia was persisting, in spite of the addition of metronidazole. Surgical exploration was therefore performed, revealing necrotic jugulodigastric lymph nodes. Material was sent for histopathology and microbiology, including mycobacterial and actinomycosis studies. No suppuration was evident.

Microbiology and paediatric opinions were sought. Serum was sent for infectious mononucleosis, cytomegalovirus and toxoplasmosis serology all of which demonstrated no evidence of acute infection. In addition, culture from the lymph node was negative. Antibiotic therapy was changed to cefotaxime. The pyrexia persisted for a further five days during which time the patient's condition gradually improved.

The diagnosis was revised to CSD on the basis of the histological findings from the biopsied jugulodigastric lymph node. The normal lymph node architecture was effaced (Figure 1). There were irregular areas of microabscess formation surrounded by palisaded histiocytes (Figure 2). There were background reactive changes with lymphoid follicle formation.

The patient was discharged home on oral augmentin after nine days in hospital. Careful questioning revealed a close history of cat contact but with no evidence on examination of an inoculation site. At five months, the patient remains well with resolution of the cervical lymphadenopathy.

Discussion

Cat-scratch disease (CSD) is a bacterial infection caused by *Bartonella henselae*, a small Gram-negative rod which is extremely fastidious, requiring special culture conditions for isolation.[3] The entity was first described by Dubre in 1931[4] although Greer and Keefer, in 1951, published the first case report of the disease.[5] Other Bartonella-associated infections, usually affecting the immunocompromised, include bacillary angiomatosis and trench fever.

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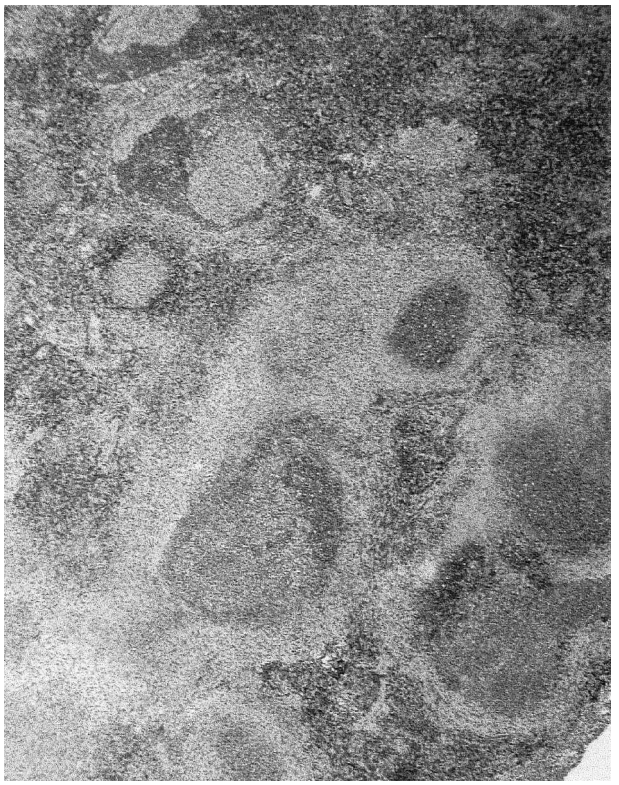

FIG. 1
Lymph node showing effacement of normal architecture with microabscesses and background lymphoid follicles. (H & E; ×50)

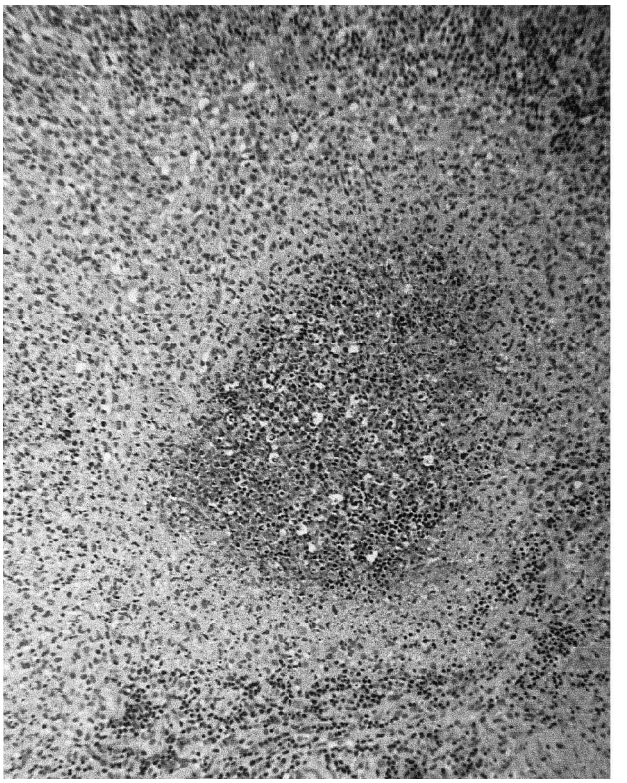

FIG. 2
A focus of microabscess formation with a rim of palisaded histiocytes (H & E; ×200)

The hallmark finding of CSD is regional lymphadenopathy.[6] The primary inoculation lesion, occurring in over 90 per cent of patients, typically arises three to 10 days after a cat scratch, bite or lick. The lesion, which progresses through a vesicular, erythematous and papular stage, typically lasts for one to three weeks.[6] Regional lymphadenopathy develops approximately two weeks (range three to 50 days) after inoculation.[6]

Whilst the axillary lymph nodes are most commonly affected, head and neck adenopathy has been reported in 26 to 73 per cent of patients.[2,7,8] The lymph nodes are typically firm and tender early in the disease. Various series report wide variation in the rate of lymph node suppuration, ranging from 11.8 to 48 per cent.[2,9] Lymphadenopathy may persist for more than six months in up to 20 per cent of patients.[2] One-third of patients have a history of pyrexia of greater than 38.3° Celsius lasting for one to two weeks which is usually associated with lethargy.

Atypical manifestations of CSD occur in approximately five per cent of patients.

Parinaud's oculoglandular syndrome, in which granulomatous conjunctivitis is accompanied by pre-auricular lymphadenopathy, accounts for the vast majority of atypical cases.[2] Rare cases of encephalitis,[10,11] osteolytic lesions[12,13] and hepatosplenic involvement[14] have been reported.

The diagnosis of CSD has historically rested on the fulfilment of a number of criteria. The most widely-accepted current criteria, advocated by Carithers[15,16] is the presence of certain clinical or laboratory findings which are assigned a score. Subacute or chronic regional lymphadenopathy is assigned one point. Cat contact, presence of an inoculation site on examination and positive serology or Hanger-Rosen skin test are each assigned two points. A total of five points is considered strongly suggestive of CSD whilst seven points is definitive. The skin test is now considered obsolete. The diagnosis is reached once other causes of lymphadenopathy have been excluded.

Serological and histopathological findings play an important contributory role in reaching the diagnosis of CSD. Serological tests for *B. henselae* are now performed by a number of commercial and reference laboratories. The indirect immunofluorescence and enzyme immunoassay tests are reported to have a sensitivity of between 88–95 per cent.[17,18] However, the role of these tests is not clearly defined.[6] In the case we present, no serological study for CSD was undertaken as no test was locally available. Culture of organisms from involved lymph nodes is difficult,[6] the aspirate typically being sterile with conventional culture methods. However, detection of *B. henselae* nucleic acid by polymerase chain reaction amplification of nodal material is highly sensitive and specific.[19] At present, this technique is confined to research laboratories.

The histopathologic findings of lymph nodes involved in CSD may be strongly supportive of the diagnosis. They may vary according to the stage of infection. Lymphoid hyperplasia and arteriolar proliferation are features found in the early stages. This is followed by the appearance of granulomas with central necrosis.[6] Multiple stellate microabscesses develop later. Warthin-Starry silver impregnation staining may reveal clumps of pleomorphic bacilli in the walls of blood vessels and in the micro-abscesses. The features of all three stages may appear concurrently.[20]

The treatment of CSD is primarily supportive. There is a lack of data demonstrating a clear benefit of antimicrobial therapy for patients with disease of mild to moderate severity.[6] One recommendation, based on limited data and experience in the immunocompromised, is a 10–14 day

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course of oral doxycycline or erythromycin.[6] Severe symptoms can be managed by rifampicin, ciprofloxacin or trimethoprim-sulfamethoxazole which in clinical trials have been found to be efficacious in 58–88 per cent of cases.[21]

The role of surgery in the management of CSD is limited. Needle aspiration of a suppurative node is the treatment of choice and may relieve local pain.[1,2,22] Excision of the involved lymph nodes may be indicated when there is diagnostic uncertainty, upper airway obstruction or for the management of a persistently draining sinus.[2,22] Incision and drainage should be avoided because of the risk of sinus tract formation.[1,2]

The case of CSD we present is unique in that the initial presentation of acute tonsillitis overshadowed the progressive cervical lymphadenopathy. The patient's fever persisted in spite of the operative finding of an absence of suppuration. The diagnosis was reached following the histological findings.

We report a case of CSD in a 10-year-old girl in whom the initial presentation was that of acute tonsillitis. CSD should be considered in the differential diagnosis of infective lymphadenopathy, particularly when the patient fails to respond rapidly to conventional therapy.

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