

Scrape and Graze-Cat Scratch Disease

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Cat scratch disease typically occurs as a self limiting lymphadenitis induced by infection with *Bartonella henselae*, a bacterium which is transmitted by cats.

Cat scratch disease occurs due to infection by *Bartonella henselae*, a rod shaped bacillus acquired via arthropod vector as a cat flea.

Alternatively, bacterial inoculation due to cat scratch may engender the disorder. A reddish cutaneous papule arises within 3 days to 10 days following contact and exposure to the organism.

Cutaneous lesion may gradually become crusted or pustular and is associated with subsequent localized or regional lymphadenopathy within one to two weeks. Morphologically, a characteristic necrotizing, stellate micro-abscess encompassed by palisading histiocytes is observed.

The organism stains appropriately with Warthin Starry stain. Alternatively, specific immunohistochemistry may be employed. Disease confirmation may be obtained with serological techniques or polymerase chain reaction (PCR).

Generally, the condition undergoes spontaneous resolution. Exceptionally, antimicrobial therapy may be necessitated.

Cat scratch disease may occur within paediatric population or adults. However, subjects ≤ 14 years are commonly implicated and the bacterium frequently engenders benign lymphadenopathy, especially within young subjects [1,2].

Cutaneous lesions configure as a reddish papule or wheal which may subsequently appear as a crusted or pustular nodule.

Commonly, regional lymphadenopathy arises within upper extremities and cervical or inguinal lymph nodes wherein singular or multiple lymph node sites may be implicated. Hepatic parenchyma, spleen or bone may display bacteria-induced granulomas [1,2].

Exceptionally, central nervous system (CNS), ocular parenchyma or haematological involvement may occur with concurrence of cogent complications [1,2].

Parinaud oculoglandular syndrome is associated with follicular conjunctivitis and regional lymphadenopathy, possibly arising due to direct inoculation of conjunctiva.

Cat scratch disease predominantly arises due to infection by bacterium *Bartonella henselae* which is commonly harboured within kittens and young cats. The bacterial rods are transmitted between cats via diverse cat fleas [2,3].

Cat fleas comprehensively (~100%) configure as a habitat of *Bartonella* spp wherein the organism may be discerned within cat saliva, blood, cutaneous surfaces, claws and faeces [2,3].

Owning a kitten and being bitten, scratched or licked by a kitten with fleas emerges as a significant cause of disease occurrence. However, history of exposure to cats may be absent in ~25% subjects with cat scratch disease [2,3].

Clinically, a non pruritic, reddish cutaneous papule occurs within 3 days to 10 days following contact with an infected cat. Lesion may gradually become crusted or pustular. Regional lymphadenopathy within cervical or axillary nodes may occur within one week to two weeks. Pre-eminently, lymphadenopathy arises comprehensively (~100%) within diseased subjects although ~85% instances may depict involvement of a singular lymph node [3,4].

Pyrexia and constitutional symptoms may emerge. Necrotizing granulomas occur within hepatic parenchyma, spleen or bone. Exceptionally, complications as granulomatous conjunctivitis, thrombocytopenic purpura or disease occurrence within central nervous system may appear in up to 10% individuals [3,4].

Akin to morphological features, cytological smears depict epithelioid cell granulomas commingled within a smattering of inflammatory cells as epithelioid histiocytes, neutrophils or polymorphous lymphocytes. Focal necrosis may be discerned [3,4].

Upon microscopy, preliminary lesions depict follicular hyperplasia and aggregates of histiocytes with micro-abscesses confined adjacent to thick lymph node capsule.

Intermediate stage demonstrate capsulitis and sub-capsular amalgamation of granulomas [3,4].

Delayed stage delineates generally stellate micro-abscesses circumscribed by palisading histiocytes. Focal necrosis is observed.

The paracortex expounds proliferation of vascular articulations.

Lymph node sinuses are permeated with monocytoid B cells. However, epithelioid cells are absent [4,5].

Miniscule bacterial rods may encircle miniature vascular articulations and lymphatic structures and may be appropriately stained by silver stains, especially within preliminary disease stage. Cutaneous surfaces expound focal dermal necrosis encompassed with histiocytes. Aggregates of multinucleated giant cells, lymphocytes and eosinophils may concur [4,5].

Upon ultrastructural examination, extracellular bacteria configure miniature groups which are enmeshed within bundles of collagen fibrils. The gram negative, pleomorphic bacterial rods demonstrate thick, homogeneous cell walls [4,5].

Prognostic outcomes and risk stratification may be assessed with

- International extra-nodal lymphoma study group (IELSG) score comprised of:
- Eastern cooperative oncology group (ECOG) performance score.
- Age of incriminated subject.
- Protein concentration of cerebrospinal fluid.

- Serum lactate dehydrogenase (LDH) levels.
- Deep seated involvement of brain.

Two year proportionate survival rate is contingent to aforesaid risk factors assessed as:

- 0 to 1 adverse factor - 80% proportionate survival.
- 2 to 3 adverse factors - 48% proportionate survival.
- 4 to 5 adverse factors - 15% proportionate survival.

Memorial Sloan Kettering Cancer Centre (MSKCC) prognostic score designates distinctive prognostic subgroups contingent to:

- Age ≤ 50 associated with median overall survival of 8.5 years.
- Age > 50 and Karnofsky performance status (KPS) ≥ 70 delineating median overall survival of 3.2 years.
- Age > 50 and Karnofsky performance status (KPS) < 70 enunciating median overall survival of 1.1 years [4,5].

Silver stains as Warthin Starry stain appear appropriate for staining *Bartonella* spp. Besides, immunohistochemistry cogent for *Bartonella henselae* may be employed. In contrast to adoption of singular technique, combination of Warthin Starry stain with immunohistochemistry contributes to enhanced proportionate sensitivity and bacterial detection.

Gram stain appears unsatisfactory for staining *Bartonella* spp as staining is inadequate [5,6].

Granulomatous inflammation induced by cat scratch disease require segregation from conditions as lymphoma, especially classic Hodgkin's lymphoma, sarcoidosis, Kikuchi syndrome, Kimura's disease and various infectious agents inducing granulomatous lymphadenitis as *Mycobacteriaceae*, *Chlamydia trachomatis*, *Francisella tularensis*, *Treponema pallidum* or diverse fungi [5,6].

Cat scratch disease may be appropriately diagnosed by techniques as polymerase chain reaction (PCR), serological methodologies or immunofluorescence. Causative organism of cat scratch disease may be suitably discerned by polymerase chain reaction (PCR).

Bartonella spp emerge as fastidious organisms and diagnostic culture may be challenging.

Upon radiography, regional lymphadenopathy is encountered. Spleen and hepatic parenchyma are be-riddled by granulomas [5,6].

Cogent therapeutic intervention appears superfluous. Therapy is not recommended for uncomplicated disease or immunocompetent subjects. Disseminated lesions or complicated disease may be managed with antibiotics as doxycycline or rifampin.

Majority of individuals represent with self limiting disease wherein lymphadenopathy regresses in up to 6 months [5,6].

Immunocompromised subjects may manifest with disseminated disease or diverse conditions or syndromes engendered due to *Bartonella* as bacillary angiomatosis [5,6].

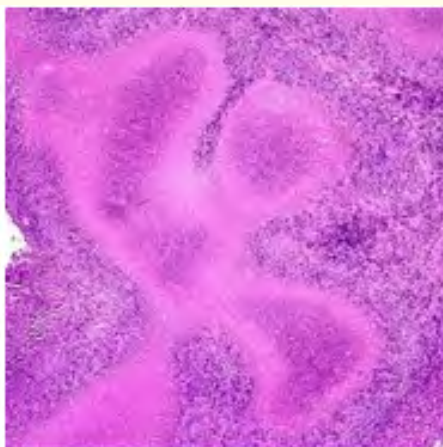


Figure 1: Cat scratch disease delineating stellate micro-abscesses and epithelioid cell granulomas entangled with a polymorphous inflammatory infiltrate of neutrophils, lymphocytes and histiocytes [7].

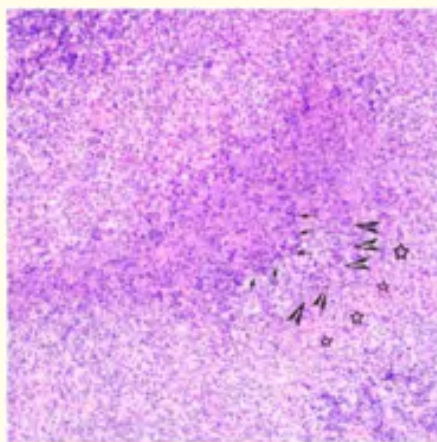


Figure 2: Cat scratch disease delineating stellate micro-abscesses infiltrated with bacterial rods and epithelioid cell granulomas along with a polymorphous inflammatory exudate of neutrophils, lymphocytes and histiocytes [8].

Bibliography

1. Martínez Lindado MA., et al. "Cat scratch disease in pediatrics: who has systemic involvement?" *Pediatric Infectious Disease Journal* 44.1 (2025): 18-23.
2. Baranowski K and Huang B. "Cat scratch disease". Stat Pearls International. Treasure Island, Florida (2025).
3. Hempel A., et al. "Hemophagocytic lymphohistiocytosis secondary to unrecognized *Bartonella henselae* infection: a case report". *Tropical Diseases, Travel Medicine and Vaccines* 9.1 (2023): 14.

4. Cheslock MA and Embers ME. "Human bartonellosis: an underappreciated public health problem?" *Tropical Medicine and Infectious Disease* 4.2 (2019): 69.
5. Yang T, *et al.* "Hemophagocytic lymphohistiocytosis is associated with *Bartonella henselae* infection in a patient with multiple susceptibility genes". *Annals of Clinical Microbiology and Antimicrobials* 19.1 (2020): 28.
6. Ud Din MA, *et al.* "Anaplastic large cell lymphoma presenting as haemophagocytic lymphohistiocytosis with underlying *Coxiella burnetii* and *Bartonella henselae* seropositivity". *European Journal of Case Reports in Internal Medicine* 7.11 (2020): 001850.
7. Image 1 Courtesy: Basic medical key.
8. Image 2 Courtesy: Spandidos publications.

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