Cat-Scratch Disease

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Cat-scratch disease is a self-limited disease associated with contact with cats that causes regional lymphadenopathy in children and young adults. Recently identification of the probable causative organism, a gram-negative, non–acid-fast coccobacillus, has been achieved through special staining techniques from skin inoculation sites and involved lymph tissue. Differential diagnosis includes a variety of other infectious diseases and neoplasms. Diagnosis of cat-scratch disease can be made by presence of typical clinical findings, history of exposure to cats, results of skin testing to cat-scratch antigen, and in some circumstances, biopsy or fine-needle aspiration from skin inoculation site or involved lymph nodes. Future developments awaiting culture of the suspected bacterium include a vaccine for prevention, rapid diagnostic methods, and antimicrobial testing.

Cat-scratch disease was first noted by Debré and associates in the 1930s and described in 1950 in their publication, "La maladie des griffes de chat."[1] Carithers[2] has reported on his own experience with over 1,200 patients, providing much information on the natural history, epidemiology, and complications of this illness. Recent findings, first reported by Wear et al,[3] have led to the description of the probable causative organism. Until their report, a variety of virologic, bacteriologic, and pathologic inquiries had failed to yield the causative organism despite the strong known association between contact with cats and the typical course and findings of the illness.

NATURAL HISTORY

Inoculation by the causative organism, probably a small, gram-negative, non–acid-fast bacterium, occurs through a puncture wound, scratch, or perhaps a previous break in the skin, after exposure to a cat. Three to five days later a papule appears on the skin, progressing to a vesicular or crusty stage over the next two to three days. Within a week or two, regional lymphadenopathy occurs.[4] Because of the size and location of the lymph node, it may not be detected until some time after the initial papule has healed, making concurrent recognition difficult and the incubation time of the illness difficult to pinpoint. Figure 1 depicts the time course of major symptoms and signs in a patient with cat-scratch disease.

In more than three fourths of individuals affected, the illness is mild with constitutional symptoms of malaise, anorexia, fatigue, and rare additional complications, which will be described later. Less than 10 percent of patients have a temperature greater than 38.9 °C (102 °F) and one third have no elevation in temperature.

Regional lymph node enlargement, the most prominent feature, may increase and persist over a two- to ten-week period but rarely lasts longer. Suppuration of the nodes occurs in 10 to 30 percent of the patients, usually within five weeks after initial symptoms. In one study of nearly 500 patients,[5] lymphadenopathy consisted of a single node in 39 percent, single nodes in several regions in 24 percent, multiple nodes in one region in 24 percent, and multiple nodes at multiple sites in 37 percent of subjects. The majority of enlarged nodes involute spontaneously within six months. In the study previously cited by Carithers involving 1,200 patients, 85 percent had single-node involvement and fewer than 2 percent had bilateral lymphadenopathy. Seventy-two percent of enlarged nodes were located at the upper extremity, neck, or jaw.

EPIDEMIOLOGY

About 80 to 90 percent of the reported cases of cat-scratch disease occur in persons aged under 21 years. The disease affects all races and has a slight predilection for males. In temperate zones most cases occur during the fall and winter. In warmer climates seasonal variation is minimal.[6]

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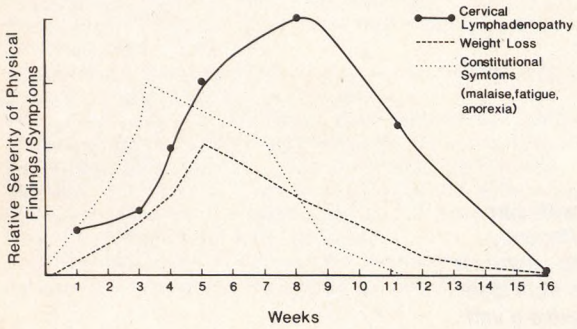

Figure 1. Time course and severity of signs and symptoms in a 33-year-old man with cat-scratch disease

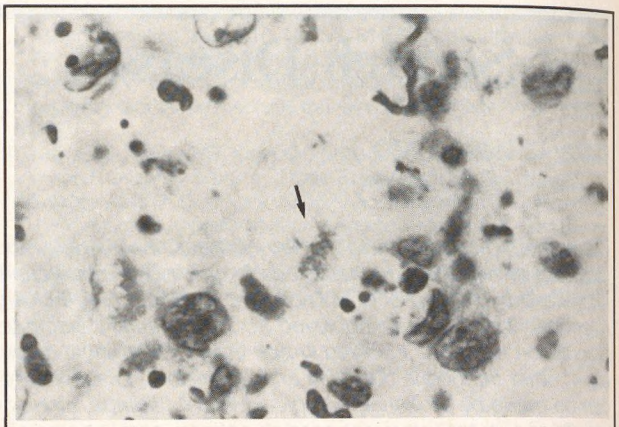

Figure 2. Coccobacillary forms of cat-scratch disease organism (arrow), cervical lymph node section, from patient represented in Figure 1 (Brown-Hopps tissue Gram stain, 450×)

There have been no true epidemics of cat-scratch disease reported. The attack rate of cat-scratch disease is unclear, but the disease does not appear to be easily acquired. For household contacts of patients affected with cat-scratch disease to acquire this disease is unusual, but occasional familial clustering has been noted. Cat-scratch disease has been reported from all countries and throughout the United States.

ETIOLOGY

Wear and co-workers[3] found a small gram-negative bacillus in histologic sections from lymph nodes removed from patients who had cat-scratch disease. These organisms were not stained by usual methods, but were best seen when silver stains (Warthin-Starry), previously used to identify spirochetal organisms, or the Brown-Hopps tissue Gram stain were employed, which may explain why previous investigators were unsuccessful in identifying an organism responsible for this disease. Later, similar organisms were reported by Margileth et al[7] from stained biopsy specimens taken at the inoculation site on the skin of several patients with cat-scratch disease. Recently Wear et al[8] have isolated cat-scratch disease bacilli from the conjunctiva of patients with Parinaud's oculoglandular syndrome, a variant of cat-scratch disease. More than one dozen laboratories[9-11] have now reported finding the cat-scratch disease organism using the Brown-Hopps tissue Gram stain or Warthin-Starry stains of lymph nodes sections. An example of these findings is shown in Figure 2.

Like many infectious diseases, the search for a cause for cat-scratch disease has had many blind alleys, false leads, and various inaccurate candidates for an etiologic agent that could never be conclusively implicated. Initial efforts to show a bacterial cause were met by failure. A viral cause was therefore favored, and viral culture methods and serologic studies were attempted but failed to yield definitive results. One of the difficulties in identifying the causative organism for cat-scratch disease may have been the selection of involved, tender lymph nodes as the place to look for an organism. It appears that lymphadenopathy is a late stage of the disease, a time when few viable organisms may be present. The skin inoculation site appears to provide a higher yield of organisms in specimens studied.

The gram-negative, pleomorphic organism suspected to cause cat-scratch disease is seen just at the limits of resolution of the light microscope. It has not been reported to have been cultured in the laboratory or in a variety of living animals. Electronmicroscopy of the bacillus thought to be the causative agent of cat-scratch disease has been described.[12] The organism seems to clump in vessel walls, which may explain the hematogenous spread of infection to distant sites that occasionally occurs or the presence of constitutional symptoms, which are more commonly noted.

It appears likely that only the domestic cat transmits directly, or indirectly, the organism responsible for cat-scratch disease. In Carithers' series of 1,200 patients,[2] 99.1 percent had a history of cat contact. Although there are reports connecting cat-scratch disease to contact with other animals (monkeys, squirrels, rabbits, and dogs) or inanimate objects (rose thorns, porcupine quills), there are not enough convincing data to substantiate this association. An additional piece of supporting evidence for the association of cat contact and cat-scratch disease is

the higher dermal reaction rate to cat-scratch antigen among contacts of patients who express liking for cats as opposed to the small number of reactions among contacts who state they dislike cats and avoid them.

HISTOPATHOLOGY

Cytologic examination of lymph node specimens or fine-needle aspirations of lymph nodes involved in cat-scratch disease reveal findings that vary with the stage of the disease.[13] Early lesions have reactive follicular hyperplasia with proliferation of lymphoid elements caused by microabscesses that usually form near or within germinal centers. Later lesions have characteristic, stellate granulomas with suppurative centers, usually without evidence of caseation. These granulomas can be identified on fine-needle aspiration of intermediate and advanced lesions.

While the aspiration cytology and lymph node sections of cat-scratch disease are characteristic, they are not diagnostic. Differential diagnosis of suppurative granulomas include lymphogranuloma venereum, Yersinia lymphadenitis, tularemia, brucellosis, listeriosis, and melioidosis. Definitive diagnosis of these latter considerations can only be made through culture confirmation, serologic evidence, and compatible clinical history.

DIAGNOSIS

At this time, there is no single test that can be used to diagnose conclusively cat-scratch disease. Diagnosis is suggested by regional lymphadenopathy developing within several weeks of cat contact together with a primary inoculation papule or pustule at the site of inoculation. Confirmation of the diagnosis has been agreed upon if the criteria in Table 1 have been met.[14]

While demonstration of cat-scratch bacilli in the primary skin lesion or lymph node appears to be definitive, lymph node biopsy or fine-needle aspiration is not indicated when the first three criteria in Table 1 confirm a typical case, particularly in those aged under 21 years.

The cat-scratch skin test has been available since 1957 and is a safe, reliable, and specific means of diagnosis.[15–17] The antigen, however, is not commercially available or standardized, but it can be prepared by the interested physician or obtained from those consultants who have a particular interest in cat-scratch disease.

The skin test antigen is prepared from aspirated pus from an individual with a typical case of cat-scratch disease, diluted with saline 1:4, and heated for six to eight hours at 60 °C. The antigen is then tested for sterility and hepatitis antigen. After intradermal inoculation of 0.1 mL of antigen, a positive reaction usually consists of a wheal or papule with 5 mm or more of induration within 48 to 72 hours. There is a false-positive and false-negative rate of 5 percent. Two negative reactions four weeks apart in an immunocompetent patient can reasonably exclude the disease, especially if two different antigen batches have been used. Administration of antigen prepared in this manner to several thousand patients over the last 20 years has not resulted in transmissibility of either cat-scratch disease or other illnesses.

TABLE 1. CRITERIA FOR DIAGNOSIS OF CAT-SCRATCH DISEASE

Diagnosis confirmed if three of the first four, or one of the first four plus No. 5 are present:

1. History of cat contact and presence of a scratch or primary dermal or eye lesion
2. Positive cat-scratch disease skin test
3. Negative studies for other causes of lymphadenopathy
4. Characteristic histopathology of a biopsied lymph node
5. Presence of typical silver-staining bacteria in histopathologic sections of lymph nodes or primary skin or eye lesions

DIFFERENTIAL DIAGNOSIS

A number of other illnesses causing regional lymphadenopathy must be distinguished from cat-scratch disease. The diagnosis of cat-scratch disease is not difficult in most patients. Most who meet the criteria outlined earlier are children who have tender lymphadenopathy usually in a single region, less often bilaterally. Other causes of lymphadenopathy to be considered can be divided into those that are infectious and noninfectious.

The major infections to be distinguished from cat-scratch disease are listed in Table 2 along with some distinguishing features and diagnostic aids. Many, but not all, of the infectious causes of lymphadenopathy are associated with tender lymphadenopathy, as is the adenopathy associated with cat-scratch disease; mycobacteria, toxoplasmosis, and sporotrichosis may be exceptions. Most of the noninfectious causes of regional lymphadenopathy produce nontender lymph node enlargement.

A number of noninfectious diseases causing adenopathy should also be distinguished from cat-scratch disease. Kawasaki disease, or mucocutaneous lymph node syndrome, in children usually presents with nontender cervical and scattered adenopathy following fever, conjunctivitis, reddening of palms and soles, fissuring of the lips, and desquamation from the fingertips during convalescence.

TABLE 2. INFECTIOUS DISEASES TO BE DISTINGUISHED FROM CAT-SCRATCH DISEASE

Infection	Causative Organism	Distinguishing Features	Laboratory Aids
Brucellosis	Brucella abortus and others	Occupational exposure to animal tissues, milk Chills, sweats, and fevers in 90% May have anorexia, weight loss, headaches May have splenomegaly, hepatomegaly	Culture of blood Antibody titers Brucella agglutination test (STA)
Coccidioidomycosis	Coccidioides immitis	Geographic exposure—Southwest USA, Central and South America Papular, verrucous, or ulcerative skin lesions	Culture of sputum, urine, pus Serologic tests Chest x-ray examination Skin test
Cytomegalovirus	Cytomegalovirus	Mononucleosis-like syndrome Hepatomegaly	Virus isolation Immunofluorescent antibody Atypical lymphocytosis
Herpes simplex	Herpes simplex virus	History of or presence of genital or oral ulceration prior to regional adenopathy Pharyngitis	Culture of organism from ulcer CF titers
Histoplasmosis	Histoplasma capsulatum	Fever, weight loss, malaise Upper respiratory tract ulceration Splenic or hepatic calcification	Culture Serologic tests Chest x-ray examination
Infectious mononucleosis	Epstein-Barr virus	Fever, sore throat, malaise Occipital plus cervical adenopathy May have splenomegaly	Heterophile antibody test Lymphocytosis with atypical lymphocytes Epstein-Barr virus antibody titers
Lymphogranuloma venereum	Chlamydia trachomatis	Primary genital lesion—painless vesicle, ulcer, or papule Tender inguinal, perirectal, or pelvic adenopathy	Culture of aspirated material Immunofluorescent staining or antibody tests CF testing
Mycobacterial lymphadenitis (scrofula)	Mycobacterium avium-intracellulare Mycobacterium scrofulaceum Mycobacterium tuberculosis	Unilateral cervical adenitis, firm, mildly tender to nontender Prolonged, subacute course	Culture and acid-fast stain of aspirate or excised node PPD 0–14 mm if due to atypical mycobacterium, >15 mm if due to tuberculosis PPD-B (battey) > 15 mm if caused by atypical mycobacterium (nontuberculous) Histopathology of lymph material Chest x-ray examination
Plague	Yersinia pestis	Fever and chills, abdominal pain Inguinal, axillary tender nodes Exposure to infected fleas from rats, squirrels, prairie dogs, rabbits	Culture of blood, pus, sputum Serologic tests
Rat bite fever	Streptobacillus moniliformis Spirillum minus	History of rat bite Chills and fever followed by morbilliform, petechial rash of feet and hands	Culture of blood, joint fluid, or pus Serologic tests
Sporotrichosis	Sporothrix schenkii	Occupational or recreational exposure to vegetation, history of splinter Nonhealing, painless red papule of extremity with spreading, nontender subcutaneous lesions along lymphatic lines No systemic signs or symptoms	Culture Serologic tests

TABLE 2. INFECTIOUS DISEASES TO BE DISTINGUISHED FROM CAT-SCRATCH DISEASE, CONTINUED

Infection	Causative Organism	Distinguishing Features	Laboratory Aids
Staphylococcal and streptococcal adenitis	Staphylococcus aureus Streptococcus group A, B or H	Abrupt onset and rapid suppuration, erythema, warmth of nodes	Culture of pus, blood, throat secretion Gram stain
Syphilis	Treponema pallidum	Initial papule becomes painless, indurated ulcer (chancre) Regional adenopathy follows	Dark-field examination of chancre scraping Positive VDRL or RPR Positive FTA-ABS
Toxoplasmosis	Toxoplasma gondii	Consumption of undercooked meat Exposure to cat feces Fever, malaise—may be asymptomatic Usually nontender, rubbery nodes—cervical or general	Serologic tests Isolation of organism from body fluids, blood Histologic diagnosis from biopsy, body fluids
Tularemia	Francisella tularensis	Exposure to rabbits, rabbit skins, ticks, or deer flies Ulceration developing from a papule of extremity	Serum agglutinating antibodies Culture of blood, pus, sputum Skin test antigen (available from CDC)

CDC—Centers for Disease Control; CF—complement fixation; FTA-ABS—fluorescent treponemal antibody-absorption (syphilis); PPD—purified protein derivative (tuberculosis); RPR—rapid plasma reagin (syphilis); STA—Standard tube agglutination test (Brucella); VDRL—Venereal Disease Research Laboratory test for syphilis

Sarcoidosis also presents with scattered adenopathy, possible splenomegaly, hilar adenopathy, and an elevated angiotensin-converting enzyme level.

A number of congenital cysts may present with cervical swelling, which can be mistaken for adenopathy.[18] These cysts usually occur in children who are otherwise well. A diffuse, soft, spongy mass may be a vascular malformation such as a hemangioma (bluish hue) or a cystic hygroma (may transilluminate). A thyroglossal duct cyst presents as a midline mass that retracts with swallowing. Branchial cleft cysts, another source of neck masses in children, may be confused with cat-scratch disease.

A number of malignant diseases can also present with regional adenopathy. Lymphoproliferative disorders, including Hodgkin's disease, non-Hodgkin's lymphoma, leukemia, and histiocytosis can present with regional adenopathy, often accompanied by fever and leukocytosis. It is important to note that the nodes are firm and nontender. Over time they progress without remission or drainage and become matted and rubbery, and are usually associated with hepatomegaly and splenomegaly. Fine-needle aspiration or excisional biopsy provides material for pathologic assessment. A number of additional rare tumors and metastatic disease can also present with regional adenopathy.

Typical presentation in the young patient aged under 21 years involving single-node enlargement or regional lymphadenopathy meeting at least three of the first four criteria outlined in Table 1, along with observation, is usually sufficient to avoid confusing cat-scratch disease with any of the large number of illnesses that can present with lymphadenopathy.

COMPLICATIONS

The most common unusual manifestation of cat-scratch disease is Parinaud's oculoglandular syndrome. First described in 1899,[19] this symptom complex involves a unilateral, granulomatous lesion of the conjunctiva or eyelid associated with preauricular lymph node enlargement. Although Pesme[20] suggested in 1950 a connection between this syndrome and cat-scratch disease, it was not until 1985 that Wear et al[8] reported finding bacteria in conjunctival lesions of patients with Parinaud's oculoglandular syndrome identical to bacteria found in lymph nodes and skin from cat-scratch disease patients. In the series of 1,200 patients reported by Carithers, 48 presented with the symptom complex of Parinaud's oculoglandular syndrome.

Other complications of cat-scratch disease include erythema nodosum and a variety of central nervous system effects,[20–24] including encephalopathy, transverse myelitis, and radiculitis. Less commonly associated symptoms and findings include osteolytic lesions of bone,[25] hemolytic anemia with hepatosplenomegaly,[26] pleural effusion,[27] anicteric hepatitis, thrombocytopenic purpura, and atypical pneumonia. Most of these changes are reversible, and the course is usually benign. Death has been associated rarely with encephalitis.

Cat-scratch disease in the immunocompromised host can produce life-threatening illness. One report of sepsis with hypotension in a renal allograft recipient demonstrates the potentially systemic nature of this usually benign, self-limited illness.[28] Cat-scratch disease may have the potential for serious complications in the host without normal immune defenses.

MANAGEMENT

Management is directed at alleviating symptoms, as cat-scratch disease is a benign, self-limited illness in the overwhelming majority of patients. Prognosis for complete and full recovery is excellent. Reassurance and limitation of activities, only as dictated by constitutional or local symptoms, seem prudent.

Suppurative lymphadenitis is probably the most frequent symptom requiring intervention in the patient with cat-scratch disease. Needle aspiration of purulent material can be helpful in symptomatic improvement. Incision and drainage are not favored by experts in the field, perhaps because of concerns of complication of the procedure including development of sinus drainage tracts in what is otherwise a usually self-limited problem. Excision of involved nodes is not routinely indicated.

The use of antibiotics has not been systematically studied and in most anecdotal reports appears to be of no benefit. Whether this finding represents failure to treat early in the course, resistance of the causative organism, or some combination is unclear. One anecdotal report, involving the immunocompromised patient mentioned earlier, noted prompt improvement after intravenous administration of sulfamethoxazole, trimethoprim, erythomycin, and tobramycin in combination. Steroids are ineffective.

Whether to perform a biopsy or a fine-needle aspiration of an involved node is a question frequently of concern to physicians. In the typical young patient with cat-scratch disease meeting the diagnostic criteria outlined earlier, observation without resort to lymph node biopsy or fine-needle aspiration appears warranted. The atypical case, such as is found in the adult aged over 21 years, in those with some of the unusual manifestations mentioned, in those with persistent pain, or conversely in those with nontender, regional lymphadenopathy or enlargement of nodes in multiple regions, is a candidate for consideration of examination of inoculation site or lymph node material. When the diagnosis of cat-scratch disease is in doubt, particularly in children, a dermal punch biopsy at the suspected inoculation site is safe, easy to perform, and highly specific for identification of the cat-scratch disease bacillus by special staining. Fine-needle aspiration of lymph node material, rather than open biopsy, can also be done as an office procedure.

If node biopsy or skin punch biopsy is performed, the specimen should be divided, placed in formaldehyde, and processed for histopathologic study including a Warthin-Starry silver stain and a portion cultured for fungi, mycobacteria, Brucella, and Francisella tularensis. Pathologists, even at remote sites, should be familiar with the histopathology consistent with cat-scratch disease. Familiarity with special staining techniques may be less available in small hospitals, but will become more available in the future as experience with this technique and diagnosis increases. At present, the nearest regional referral center can supply expertise in histopathologic preparation if not available locally.

PREVENTION

Hadfield et al[14] believe the cat-scratch disease organism may be part of the feline oral flora. Avoidance of bites, licking of open human wounds, and scratches (cat-scratch disease bacillus is transferred to claws during grooming) might reduce the risk of infection from cats.

The patient does not require isolation because of the low transmissibility of cat-scratch disease. The cat that is suspected of being the vector is not ill, and no one is sure of the length of time during which the cat may transmit the disease. Some authorities mention declawing if the cat is a pet, but no scientific effort has been reported demonstrating reduction in future cases of cat-scratch disease by this method.

FUTURE TRENDS

The next advance in the understanding of cat-scratch disease will come from the culturing of the suspected bacterium, which will allow for antimicrobial susceptibility testing and perhaps development of a rapid diagnostic test available commercially. Also, the resultant production of a vaccine may make cat-scratch disease preventable among cat owners, veterinarians, and others who handle cats frequently. Studies to develop a purified skin test and serologic test for cat-scratch disease are ongoing.

Recognition of the typical features of cat-scratch disease and demonstration of typical features or bacilli in lymph nodes or skin inoculation sites in the patient with unusual symptoms may reduce fear, inconvenience, and the cost of medical and surgical care for this illness. Future efforts at rapid diagnostic methods may aid this process. While some of the mystery surrounding cat-scratch disease has been solved with the identification of the causative or-

ganism in tissue stains, unanswered questions remain regarding this interesting illness.

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