Group G Streptococcal Epizootic in a Closed Cat Colony

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An epizootic of beta-hemolytic Lancefield group G streptococcal infections occurred in a specific-pathogen-free colony of laboratory cats. A total of 19 out of 68 animals in a single building were affected over a 10-day period. Clinical signs included fever, depression, lymphadenopathy, pharyngitis, and submandibular edema. The organism was recovered from the pharynx in two of five clinically normal cats from the affected building. Cultures from 12 animals in the same colony but housed in unaffected buildings were negative. Two doses of long-acting penicillin G 72 h apart stopped the outbreak and resulted in negative cultures for previously affected animals. Three months later, two new cases occurred in the same building. The disease was finally eradicated from the colony by depopulating the affected building.

Epizootics of a contagious streptococcal lymphadenitis caused by beta-hemolytic Lancefield group G streptococci have been reported twice previously for cats (5, 8). Each epizootic has been associated with laboratory cats and with group housing. In the first report, 32 cases were randomly scattered among 1,391 random-source cats in a large research facility over a 5-month period (5). In the second report, the infection was seen in four nursing kittens who were litter mates and one unrelated adult cat quartered in the same room (8). Although the disease has been reproduced experimentally by oral inoculation of healthy cats (9), the low attack rate seen in the first outbreak (2.3%) and the small number of animals involved in the second outbreak have made it difficult to appreciate its highly contagious nature. This report describes an explosive epizootic resulting from what appears to have been a single introduction of a pathogenic beta-hemolytic group G streptococcus to a closed, pathogen-free cat colony.

CASE REPORTS

On 26 January 1982, 2 out of the 20 6-month-old male cats located in room 4 of building W-4 developed submandibular abscesses. These were lanced, and the animals were given parenteral procaine penicillin G (20,000 U/kg) once daily for 5 days. The abscesses were lanced in the animal room. Exudates were carefully collected on cotton gauze, and two individual stainless-steel cages were placed in the room to house the animals while their wounds were draining. The animals made an uneventful recovery. The lesions were not cultured. By 29 January six additional animals in the same room displayed a syndrome characterized by submandibular edema, massive bilateral submandibular lymph node swelling, depression, salivation, and fever. One of these swollen nodes was aspirated percutaneously, and a beta-hemolytic streptococcus was obtained in pure culture from the aspirate. The isolate was not further characterized. These swollen nodes were not drained surgically. Each affected animal received penicillin as above and was clinically normal within 48 h after the initiation of antibiotic therapy. These animals remained at large within the animal room.

On 2 February 1982, seven more cats became acutely ill in the two rooms immediately adjacent to room 4. Five of the seven cats were housed together in room 3. Two of the seven were in individual cages in room 2. No animals had been moved from room 4 to these rooms, but caretakers had access to all three rooms. Four of these cats presented with submandibular lymphadenopathy as in the previous cases; three cases were sufficiently mature to require surgical drainage. The other three cats showed no submandibular swelling but were severely depressed. Two of these were febrile, with temperatures of 41.0 and 41.2°C and slightly elevated white blood cell counts (19,000 and 22,000 cells per mm³) with 85 and 87% neutrophils, respectively. The third cat was dyspneic and appeared to be moribund. Its temperature was normal (38.3°C) and its white blood cell count was 2,200 cells per mm³. The third cat died in a few hours despite antibiotic therapy and was necropsied. On necropsy it was found to have a copious fibrinopurulent pleural effusion which, when Gram stained, revealed numerous gram-positive cocci in long chains. A Lancefield group G streptococcus was obtained in pure culture from the pleural effusion and from the submandibular lymph node (which was not enlarged) and was found in mixed culture in the pharynx.

By 5 February a total of 19 out of 68 cats in building W-4 had been affected clinically. Eleven had presented with submandibular edema and lymphadenopathy but had not required surgical drainage, five had abscessed and been lanced, three had presented with acute prostration, probably representing septicemia, and
FIG. 1. Distribution of streptococcal infections in three rooms (numbers 2, 3, and 4) housing a cat colony. On 5 February and again on 26 April all cats were treated with penicillin; on 17 February, throat swab cultures for 17 cats were negative.

one of these had died with a pleuritis as described above. In all cases but the latter, the response to penicillin therapy was dramatic. Course and spread of the disease are shown in Fig. 1.

Beginning 2 February measures were instituted to attempt to control the epizootic. Caretakers working in W-4 were not allowed to enter any other buildings housing cats. All movement of cats between buildings occupied by the colony was stopped.

Between 2 and 5 February cultures were obtained from affected and unaffected cats to determine the prevalence of beta-hemolytic streptococcal infection in the colony and its association with the disease process. All beta-hemolytic streptococci recovered were characterized for their streptococcal grouping and carbohydrate fermentation pattern.

On 5 February and again on 8 February every cat in building W-4 received 150,000 U each of procaine penicillin G and benzathine penicillin G subcutaneously. During this period all surfaces in the building were cleaned thoroughly and disinfected with a quaternary ammonium disinfectant. The outbreak stopped immediately. No new cases appeared for nearly 3 months.

On 26 April two more cases presented in building W-3, room 2. One cat was found dead; it had been seen the previous day and had not seemed sick at that time. A necropsy was performed, and no gross pathology was noted except that one lung lobe was severely congested. Histological sections of the affected lobe revealed a severe, acute, diffuse pneumonia with vasculitis, suggestive of an acute septic thrombotic event.

The second cat presented with fever and lymphadenopathy exactly as were seen in the earlier cases. This cat was treated with penicillin as described above and recovered rapidly. Beta-hemolytic streptococci were obtained from the lung and pharynx of the first cat, and a lymph node aspirate and the pharynx of the second cat. All cats in the room were treated prophylactically with benzathine penicillin as before. No further cases were seen.

A decision was made to gradually depopulate building W-3. At present, the building has been restocked with specific-pathogen-free cats, and none of the animals who occupied the building at the time of the outbreak remain. The newly introduced cats and those in the other portions of the specific-pathogen-free cat colony in other buildings continue to remain free of any evidence of infectious disease.

MATERIALS AND METHODS

Animals. The cats were part of a large closed colony maintained for nutrition studies. The colony originated from cesarian-derived animals and was maintained under conditions of limited access. Workers were required to wash hands and change outer garments and footwear before entering rooms where animals were housed. Workers in this colony did not handle any other animals during working hours. The colony occupied several adjacent buildings in the area where the outbreak occurred and had breeding animals located in more-distant buildings. The colony had been in existence for 5 years and during that time had remained free of feline respiratory infections, panleukopenia, feline leukemia virus, feline infectious peritonitis, and all internal and external parasites. No attempt had been made to control or monitor bacterial flora. No infectious disease had ever been seen in any animal in this facility. The epizootic occurred entirely within three rooms of a single building, designated W-4. Room 4 of building W-4 housed 20 6-month-old male cats. Room 3 contained 23 3- and 4-month-old female cats. The cats in these two rooms were not under experimentation but were young animals being raised for resale. These cats were kept at liberty in large rooms, with resting boards and perches provided for exercise. Dry cat food and water were provided freely from common, open containers. The only exception to this type of husbandry in the affected building was one group of 25 cats housed in room 2; these cats were kept in individual cages and fed a zinc-deficient synthetic diet.

Collection of cultures. Pharyngeal swabs were taken by swabbing the tonsilar crypt area with a sterile swab and transporting this in 0.5 ml of modified Stuart transport medium (Culturette, Scientific Products, Inc., Detroit, Mich.). Swabs were plated on 5% bovine
blood agar within 3 h of collection. Exudates and necropsy materials were streaked directly on 5% bovine blood agar.

Identification. Gram-positive, catalase-negative cocci producing beta-hemolysis on 5% blood agar and hemolysis in blood broth were considered to be beta-hemolytic streptococci.

Media. Bovine 5% blood agar was prepared by combining 19 parts blood agar base (Difco Laboratories, Detroit, Mich.) with 1 part sterile defibrinated bovine blood to 5 ml of tryptose broth. Sugar fermentations were tested in 5-ml tubes containing 1% fermentable substrate in a bromocresol purple broth base to which 2 drops (0.1 ml) of sterile horse serum had been added. The broth base contained 10 g of peptone (Difco), 4 g of beef extract (Difco), 5 g of sodium chloride, and 0.016 g of bromocresol purple per liter.

Characterization. All beta-hemolytic streptococci obtained were tested for their ability to produce acid from lactose, trehalose, and sorbitol within 4 days of incubation at 37°C in the appropriate bromocresol purple broth.

Streptococcal grouping was performed by the Microbiology Service, Veterinary Medical Teaching Hospital. An enzyme extraction method (7, 10) was used with commercially obtained antisera for groups A, C, D, E, and G (Wellcome Research Laboratories, Beckenham, England) and with commercially obtained lysozyme and Streptomyces albus enzyme (Difco).

RESULTS

A Lancefield group G streptococcus was isolated from 11 samples from seven affected animals taken between 2 February and 26 April. Isolations were made from lymph nodes (two), pus (three), pleural effusion (one), throat swabs (four), and lung (one) of affected animals. The organism was isolated from every affected animal for which culture was attempted.

On 5 February, just before prophylactic antibiotics were administered, throat swabs were taken from five clinically normal animals in the affected rooms. Two of these samples produced beta-hemolytic group G streptococci. Throat swabs were also taken for 12 cats of the same colony from adjacent buildings in which no disease had been seen. No beta-hemolytic streptococci were obtained from these cats.

On 17 February, 9 days after the second antibiotic injection, throat swabs were taken for 17 cats in building W-4; 12 had been previously affected and 5 had been exposed but not ill. No hemolytic streptococci were recovered.

Since infections with group G hemolytic streptococci have been reported in human beings (6), all personnel working in building W-4 were asked to submit throat swabs during February. No hemolytic streptococci were found in these cultures.

All of the 11 beta-hemolytic group G streptococci that were recovered from animals with clinical disease fermented lactose but not trehalose or sorbitol. Two isolates were obtained from the pharynges of normal animals in affected rooms; both fermented lactose, neither fermented sorbitol, and one fermented trehalose whereas the other did not.

DISCUSSION

This report confirms the existence of a spontaneously occurring contagious streptococcal infection of cats caused by a beta-hemolytic Lancefield group G streptococcus. Many veterinary keys and textbooks refer to group G streptococci by the unofficial term Streptococcus canis (2-4). Group G streptococci that ferment lactose but not trehalose or sorbitol are commonly isolated from dogs, in which they show a predilection for the urogenital system (1). The natural reservoir of streptococcal strains pathogenic for cats is unknown, as is the prevalence of this organism in the cat population in general. The source of the outbreak described in this report could not be determined.

Of particular interest in this epizootic were the following observations. (i) The disease was highly contagious in a closed colony with no prior exposure. (ii) Group G beta-hemolytic streptococci were recovered from pharyngeal swabs of two normal animals in infected rooms. These animals may have had nonclinical infections or may have been incubating the disease. (iii) Four out of twenty-one cases occurred in animals housed individually; previously reported cases have only occurred in animals housed in groups. (iv) Either the infection was not eradicated from the colony with antibiotic therapy, or a second introduction into the same building occurred 3 months after the original epizootic.

LITERATURE CITED
