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FELINE health topics

for veterinarians

inside

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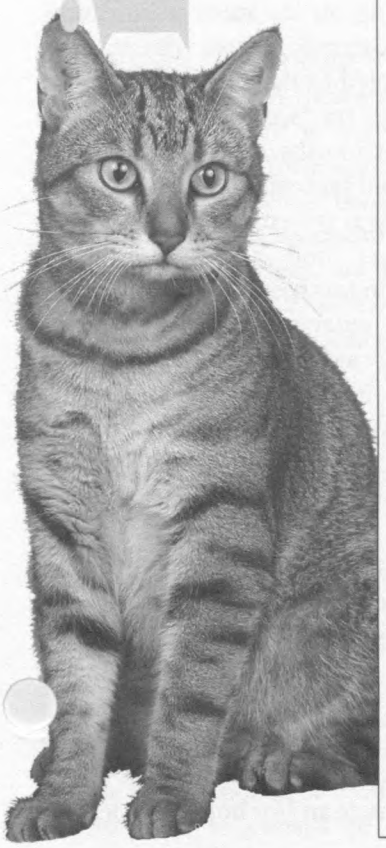

Feline Tuberculosis

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Several species of mycobacteria can cause disease in veterinary species, being either primary pathogens, or becoming pathogenic under certain circumstances.

Tuberculosis can be caused by a number of different, but closely related, bacteria. Members of the tuberculosis complex include *Mycobacterium* (*M.*) *tuberculosis*, *M. bovis* and *M. microti*. *M. tuberculosis* causes over 90 percent of tuberculosis in man, but rarely infects other mammals except for dogs. *M. bovis* is the main cause of tuberculosis in cattle. It can also infect various other mammals, including humans, dogs, cats and pigs. *M. microti* causes tuberculosis in voles and cats (in the latter it was previously termed *M. microti*-like and has culture characteristics between *M. tuberculosis* and *M. bovis*). *M. avium* causes tuberculosis in birds, and can also infect man, dogs and cats. *M. avium*-intracellular complex (MAC) organisms are mainly saprophytic, but they are often considered with the tuberculosis complex, since they can cause clinical disease indistinguishable from that caused by members of this group.

Other potentially pathogenic mycobacteria include *M. lepraemurium*, which causes leprosy in rats, and a similar, or possibly the same, organism which causes feline leprosy. Opportunistic mycobacteria are usually saprophytic, but a number of species have been reported to cause disease in cats. These include *M. chelonae-abscessus*, *M. fortuitum/peregrinum* group, *M. smegmatis, M. phlei, M. genavense, M. similae, M. thermoresistible, M. xenopi* and *M. terrae* complex.

Mycobacterial syndromes seen in cats therefore include tuberculosis, feline leprosy and opportunistic mycobacteriosis. All three syndromes have been reported in the United Kingdom (UK) and North America, where the majority of cases appear to be cutaneous in nature. All three syndromes can present with nodules, draining tracts and/or ulceration. In some cases, the disease may become generalized secondary to skin inoculation, but only occasional cases present with primary systemic disease. Where systemic disease is seen, infection with a member of the tuberculosis group is most likely. In many cases of feline mycobacteriosis, infection can be related to percutaneous injury, contamination via soil or the presence of devitalized tissue. These factors tend to be reflected in the distribution of the lesions.

Epidemiology and Etiopathogenesis

In cats, tuberculosis has classically been described as being caused by *M. bovis*. Historically, infection resulted from the ingestion of milk from tuberculous cattle. With the virtual eradication of tuberculosis from the national herd there has been a marked decline in the prevalence of the disease seen in cats.

Currently, tuberculosis in cats is seen infrequently. When diagnosed, it is usually caused by infection with either the cattle form of the infection (*M. bovis* [41 cases]) or the vole form (*M. microti* [22 cases - recent figures from Keith Jahans, Veterinary

Laboratories Agency, 2004, data collected since 1980). Infection of cats with *M. tuberculosis* is incredibly rare, probably because cats are naturally very resistant to it. (Interestingly, this is quite different from the picture seen in humans, where over 90 percent of cases result from infection with *M. tuberculosis*; approximately 1 percent is caused by *M. bovis* and disease due to *M. microti* is incredibly rare). Cats can occasionally develop disease due to infection with MAC.

The current epidemiology of tuberculosis in cats is still unclear. While there are occasional clinical cases, few are believed to relate to direct infection from cattle. This is because when tuberculosis is gained by drinking contaminated cows' milk the infection settles within the cat's intestines, and disease results in diarrhea and weight loss. It is probably because almost all cows' milk is now pasteurized that this type of tuberculosis is currently very rare. The tuberculosis now seen most frequently in cats affects their skin, where it causes non-healing sores and lumps that fail to heal. This is often associated with swollen lymph nodes, especially those under the chin, and some cases show only the swollen lymph nodes. In chronic cases where the infection has spread to the lungs, cats may develop a soft cough or have difficulty breathing.

It is important to try to determine how cats become infected. Most infected cats are keen hunters, regularly catching small rodents. Interestingly, studies have shown that in the UK, wild mice and voles quite often carry *M. microti*, while moles and rats carry *M. bovis*. It is, therefore, most likely that cats become infected by hunting small wild rodents. This also accounts for the distribution of the skin lesions seen on these cats, which occur most frequently on the face and legs, i.e., the areas most likely to be bitten when playing with prey. In some areas of Britain, *M. bovis* has become endemic in badgers. While cats and badgers rarely interact directly, there may be a potential risk for cats to become infected via local environmental contamination. *M. bovis* can also be endemically present in many other species of free-ranging wildlife (e.g., deer), so the risk of feline infection will vary in each country dependent on the likely interaction between these species and domestic cats.

All members of the tuberculosis complex pose potential zoonotic risks. However, to date, there have been no reported cases of cats passing tuberculosis onto humans. By far the greatest tuberculosis risk to humans is spending time with infected humans or, less frequently, handling infected cattle.

Interestingly, *M. tuberculosis* and *M. bovis* can both cause reverse zoonoses and there have been a small number of cases where humans have infected their cats (usually with *M. bovis*). This may be significant because with the current increase in human tuberculosis associated with HIV infection and poor housing, we may see a concurrent increase in feline tuberculosis caused by these organisms.

Predisposition

Most cases of feline tuberculosis are probably subclinical in nature. Infection usually occurs after protracted exposure, e.g., repeated exposure to infected small mammals, living on a farm housing tuberculous cattle, or living for prolonged periods with infected humans or poultry. Tuberculosis is, therefore, seen mainly in adult cats and, interestingly, most commonly in males. No evidence of immunosuppression has been found and those cats tested for FIV and FeLV have usually been negative.

Clinical Signs

Depending on the route of infection, affected cats may present with systemic signs related to the alimentary, and/or respiratory tracts, or with localized disease affecting the skin. Currently, the most usual presentation for tuberculosis in cats is the cutaneous form, with respiratory and alimentary forms being seen less frequently.

In the cutaneous form, the lesions probably arise from infected bite wounds, local spread or hematogenous dissemination to the skin. The lesions often involve the face, extremities, tail base or perineum, i.e., "fight and bite sites." Less frequently they involve the ventral thorax and tail base. They generally take the form of firm, raised, dermal nodules, ulceration, or non-healing wounds with draining sinus tracts. Extension of granulomatous tissue may, in some cases, involve the subcutaneous structures, muscle and/or bone. Skin lesions

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The ultimate purpose of the Cornell Feline Health Center is to improve the health of cats everywhere by developing methods to prevent or cure feline diseases, and by providing continuing education to veterinarians and cat owners. All contributions are tax-deductible.

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are commonly associated with either local or generalized lymphadenopathy. On occasion, submandibular or prescapular lymphadenopathy may be the only clinical finding.

When the infection spreads to the lungs, or where it is acquired through inhalation, tubercles arise in the lungs and/or hilar lymph nodes and affected cats present with weight loss, anorexia, dyspnea and coughing.

In the alimentary form, tubercles arise in the intestines and/or mesenteric lymph nodes. Affected cats commonly develop intestinal malabsorption and present with weight loss, anemia, vomiting and diarrhea. Occasionally tubercles arise in the tonsils, resulting in signs of oropharyngeal disease.

A range of clinical signs may be seen with disseminated disease. These include splenomegaly, hepatomegaly, pleural or pericardial effusions, generalized lymphadenopathy, weight loss and fever. Lameness may result from bone involvement. Granulomatous uveitis and signs referable to central nervous system involvement have been seen in some cases.

Diagnostic Techniques

Non-specific tests: A thorough evaluation of the patient is necessary to assess the extent of local infection and the degree of systemic involvement. Changes in serum biochemistry and hematology, if present, are non-specific and vary with the severity of the disease. However, hypercalcemia has been seen in a number of cases and appears to correlate with a poorer prognosis. Radiography can be useful in the appraisal of lung involvement. However, changes are extremely variable and include tracheobronchial lymphadenopathy, interstitial or miliary lung infiltration, localized lung consolidation or pleural effusion. Abdominal radiography may reveal hepatic or splenic enlargement, abdominal masses, mineralized mesenteric lymph nodes or ascites. Bone lesions tend to consist of areas of bony lysis and sclerosis, osteoarthritis, discospondylitis or periostitis.

Specific tests: Specific tests for the diagnosis of tuberculosis have been investigated in cats, but have generally proved unhelpful. Unlike other species, cats do not react strongly to intradermally administered tuberculin and the results from intradermal skin testing are unreliable. Tests for specific serum antibody responses have also proved unhelpful.

To confirm mycobacterial involvement, aspirates and/or biopsy samples of affected tissue should be stained with Ziehl Neelsen (ZN) or other specific special stains. The number of acid fast bacilli seen within affected macrophages may be variable, depending on the species of mycobacteria involved, the location of the granuloma and, probably most importantly, the nature of the cat's immune response. While finding acid fast bacilli confirms the presence of mycobacteria, it is essential to culture the organism to determine the exact species involved. Once the species has been identified it is possible to evaluate zoonotic risk, potential sources of infection, and feasible treatment options. Unfortunately, many samples that are seen to have ZN positive organisms fail to culture positive, and even those that do typically take approximately two months.

Correct handling of biopsy material: In practice, this usually involves taking a biopsy from a case where mycobacterial disease is only one of a large number of possible differential diagnoses. If in-house facilities are available for ZN staining, this can be performed on aspirates or biopsy impression smears. However, in most cases biopsy material must be sent to a veterinary diagnostic laboratory. It is practical to collect the biopsy, cut it into three pieces, fix one in formalin for histopathological examination and ZN staining and, pending results, place one in a sterile container and freeze it. Where other bacterial infections are suspected, the third sample should be sent unfixed for routine bacterial culture at which time ZN staining can also be requested. That way, if the sample is found to have ZN positive organisms, the frozen portion can be defrosted and sent to:

USDA/APHIS National Mycobacterial/Brucellosis Laboratory
800 Delaware Avenue
Ames, IA 50014
Telephone: (575) 663-7676

Until the organism has been properly characterized, it should be considered a potential human pathogen.

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Newly Emerging Infectious Diseases - Part II

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Feline Spongiform Encephalopathy

Etiology

Feline spongiform encephalopathy (FSE) is one of a group of naturally occurring transmissible spongiform encephalopathies (TSEs). The TSE agents are unlike any other micro-organisms. All TSE diseases are characterized by the accumulation of an abnormal form of a host-coded protein, the prion-protein (PrP). PrP is a cell surface glycoprotein of unknown significance found in all animals. However, while the PrP isolated from normal individuals (PrPc) and the PrP isolated from TSE infected individuals (PrP-res) have the same amino acid sequence and secondary structure, PrPc is totally degraded by proteinase K, while PrP-res resists digestion. Once present, PrP-res is believed to induce additional copies of itself by interacting with normal PrPc. In doing this, PrP-res acts as an infectious agent. Once the host-coded PrPc has been transformed into PrP-res it accumulates in fibrils (SAF) and leads to disease.

Reason for Interest

TSE diseases occur in many mammalian species including man. They include scrapie in sheep and goats, bovine spongiform encephalopathy (BSE) in cattle, chronic wasting disease (CWD) of deer and elk, transmissible mink encephalopathy (TME) in mink, and Creutzfeldt-Jakob disease (CJD), Gerstmann-Sträussler-Scheinker disease (GSS), and kuru in humans. Experimentally, TSEs can be transmitted to an even wider range of species, including rodents and non-human primates. While the widespread interest in TSEs developed only recently, mainly associated with the BSE epidemic in the UK and the recognition of variant CJD (variant CJD), this type of disease is far from new. Historical records show that scrapie was first recognized almost three hundred years ago. FSE was first recognized in 1990.

Geographical Distribution

TSEs have been seen in many countries throughout the world. However, while scrapie and human TSEs have a widespread distribution, BSE has been seen mainly in Europe, particularly in the United Kingdom (UK). The situation with FSE is similar to BSE, with the majority of cases being seen in the UK.

Transmission

The agent responsible for FSE is believed to be the same as that for BSE. The BSE agent is believed to have entered the UK cat population in contaminated pet food. BSE was first reported in 1987. It is believed to have resulted from the inclusion of scrapie-infected sheep carcasses, in the form of meat and bonemeal, into feedstuffs for cattle. A change in the rendering process had enabled the TSE agent to survive the processing procedures. Transmitting the scrapie agent through cattle is believed to have resulted in a change to its pathogenicity, making it more infectious to cattle (and cats). Cattle succumbing to BSE were then included into the meat and bonemeal, thereby amplifying the transmission and spreading the infection. The feeding of meat and bonemeal to ruminants was then banned in 1988, and the Pet Food Manufacturers Association (PFMA), recommended to its members that 'specified bovine offal' (SBO) be banned from inclusion in pet food. This later became law in the UK. Subsequent changes to the handling of offal material should have removed any further risk of cats being exposed to the BSE agent in commercial pet food.

Consistent with the hypothesis that FSE is caused by the BSE agent is the temporal distribution of FSE cases. Since its recognition in 1990, ~90 cases of FSE have been confirmed. The majority of these were seen between 1990 and 1994. Since that time there has been a general decline in the number of cases. It shows that after a time interval representing the incubation period of the disease, the removal of SBO from the diet of cats in the UK resulted in a marked reduction in the incidence of FSE. Also, a retrospective study of brain sections from cats with neurological disease revealed no cases of FSE prior to 1990. However, since very few domestic cats are subject to routine post mortem examination it is likely that the total number of FSE cases has been underestimated.

The natural route of FSE transmission is unclear, although it is believed to result from ingestion. While maternal transmission of spongiform encephalopathies may occur in some species, there is no evidence that it occurs in cats. TSE diseases are exceptional as they are not only infectious, but in some cases, they can be transmitted genetically. However, while this is true of certain human TSEs (e.g., GSS) and, to some extent, scrapie in sheep, it does not appear to be the case for BSE or FSE.

Signalment

Consistent with a food-related infection, FSE shows no breed predisposition, and cats from all types of households have been affected. While FSE has been seen in both sexes, it has been seen more frequently in males. The mean age at onset is approximately 5-7 years, with a range of 2-12 years of age.

Clinical Signs

FSE is characterized by a long asymptomatic incubation period followed by an insidious onset of progressive

behavioral and motor disturbances. Cases may present with progressive hind limb ataxia, increased aggression or affection, hyperesthesia to touch, sound and/or light, altered grooming patterns, increased salivation, dilated pupils with an unusual staring expression, polyphagia and/or polydipsia, abnormal head posture, jaw chattering, aberrant defecation/urination, muscle fasiculations, or an inability to retract their claws. Behavioral changes are usually noted first, followed by progressive locomotor dysfunction. The cats tend to show ataxia, with dysmetria or hypermetria, which often leads to an erratic crouching gait. They also show an inability to judge distances.

Duration of Disease

The disease is generally progressive, warranting euthanasia within 8-12 weeks of the onset of clinical signs.

Diagnosis

Premortem diagnosis is rarely possible. While clinical signs may be suggestive of FSE, and non-specific tests, like electroencephalography (EEG) or magnetic resonance imaging (MRI) may indicate the presence of diffuse CNS disease, specific tests are generally lacking. Diagnosis of FSE is usually made by histopathological examination of the brain (formalin fixed tissue), and the ultrastructural detection of SAF in brain extracts (fresh frozen brain or spinal cord). After euthanasia, any case suspected of having FSE should have a full post-mortem examination performed by a trained veterinary pathologist. If the diagnosis is confirmed, contact the *National Veterinary Services Laboratories at* ***www.aphis.usda.gov/vs/nvsl/***.

Pathological changes are confined to the CNS and consist of variable degrees of vaculation of the neuronal parenchyma and an astrocytic response.

Treatment

There is no effective treatment.

Zoonotic Risk

Although it is generally difficult to transmit a TSE agent from one species to another by mouth, BSE appears to have been transmitted naturally, not only to cats, captive exotic felids, and captive exotic ungulates, but also to humans (as variant CJD). Thankfully, with the introduction of strict laws regulating the slaughter and rendering of ruminants, and the overall decline in the incidence of BSE, the possibility of the BSE agent continuing to be included in the food chain is extremely small. However, because the incubation period is long and variable we are likely to continue to see new cases of variant CJD for a few years yet to come.

It is very unlikely that cats present a zoonotic risk. Not only is the disease now extremely rare, it was never very common, and the likelihood of FSE-infected brain or spinal cord entering the human food chain is almost non-existent. While there has been one case of human CJD and FSE occurring within the same household, the strain of TSE with which both individuals were affected appears to have been a variant more typically associated with spontaneous CJD, not BSE. The method of transmission in this case is not known.

Borna Disease

Etiology

Borna disease virus (BDV) is a neurotropic RNA virus.

Reason for Interest

Classical Borna disease (BD) is seen predominantly in horses and sheep in endemic areas of Germany and Switzerland. However, while the disease was initially thought to be limited to these species and this location, it is now known that natural infections can also be seen in cats, ostriches, and occasionally, rabbits, cattle, goats, deer, and dogs. Experimentally, BDV can be transmitted to a wide variety of species, including birds, rodents and monkeys. Far from being a regional infection, serological evidence has documented BDV in central and northern Europe, North America, the UK, Japan, Iran, Israel and Australia. The presence of a higher prevalence of BDV infection in humans with neurologic or psychiatric disorders has suggested that the infection may have zoonotic potential.

Geographical Distribution

BD in cats is also known as "staggering disease." Serological surveys or surveys looking for BDV RNA within peripheral blood samples have shown that BDV infection is often asymptomatic; ~6 percent of healthy cats in the UK are seropositive. The prevalence of seropositive cats appears to increase with age. However, while BDV may be detected in many normal cats, BDV RNA or antibodies against BDV are seen most frequently in cats with neurological disease. While fatal BD is seen most commonly as a rare, isolated event, it can occasionally be seen in large outbreaks, where as many as 30-40 cases may be seen in a week. While most documented cases of feline BD have originated from northern and central Europe, probable cases of BD have also been seen in other countries. Given the difficulty in making a diagnosis, and the low index of suspicion, it is likely that BD is underdiagnosed.

Signalment

Natural BD has been reported in over 100 cats. It is seen most frequently in male cats, with no particular breed predisposition. While a wide age range of cats may be affected (from five months to 11 years), young adults appear to be most at risk. Affected cats have usually been allowed to roam outdoors, particularly in rural or woodland areas.

Transmission

The natural reservoir host of BDV is

not known. However, since the virus can infect so many different species, a single reservoir may not be required. In feline BD, hunting mice appears to be a risk factor. Most natural infections are believed to occur via saliva or nasal secretions from clinically affected animals or apparently normal virus carriers.

It is currently not clear whether there is a single biotype of BDV that can infect and cause disease in a wide range of species, or whether there are species-specific biotypes. Either way, BDV appears to be a ubiquitous virus that is very well adapted to its various host species. In most cases, infection causes little or no sign of disease. It is only when a host is particularly susceptible, or mounts an abnormal response to the virus, that clinical signs develop.

Clinical Signs

"Staggering disease" is characterized by behavioral and motor disturbances resulting from meningoencephalomyelitis. In experimental infections, clinical signs include protrusion of the third eyelid, behavioral changes, circling, ataxia, and tremors. Natural infections may present with progressive hind limb ataxia, often with hypermetria and a crouching gait, behavioral changes, including increased timidity, aggression or affection towards the owner, fever, hypersensitivity to light and/or sound, unusual staring expression, loss of appetite (occasionally polyphagia), apparent pain over the sacrum, altered grooming patterns, seizures, increased salivation, an inability to retract the claws, muscle fasiculations, or constipation.

Duration of Disease

Disease is usually progressive, warranting euthanasia within one week to six months from the onset of clinical signs. In naturally infected cats, once clinical signs develop, mortality rates are usually high. Animals that survive the initial episode may remain chronically infected, and may experience recurrent episodes of disease.

Diagnosis

Premortem diagnosis can be difficult. In most cases, typical clinical signs in a cat from an endemic area will result in a presumptive diagnosis of BD. Detection of serum antibodies is not reliable. While raised serum antibodies may be present in some cats with BD (~40 percent), other affected cats may be antibody negative. Although clinical signs of BD tend to develop at the same time that BDV RNA can be detected within the peripheral white blood cells, the detection of BDV RNA within peripheral blood does not necessarily reflect the extent of the viral load in the CNS. Routine serum biochemistry and hematology are generally unremarkable, although some cats may show a leukopenia and mild elevations in glucose and liver enzyme levels. CSF analysis may show a leukocytosis with mononuclear cells predominating, and protein levels may be increased.

Pathologically, BD results from a non-suppurative meningoencephalomyelitis, which is usually particularly evident in the grey matter of the cerebral hemispheres, the limbic system and the brainstem.

Treatment

There is no specific treatment for BD. Supportive care and corticosteroids may be helpful. Prednisolone may be given at 1-2 mg/kg PO q24 hours until clinical signs regress, then reduced gradually over several weeks or months.

Zoonotic Risk

It is currently unclear what role BDV may play in the induction of human disease. Antibodies against BDV, viral proteins and RNA have been found in humans in Europe, North America and Asia. A higher prevalence of infection is seen in patients with neurologic or psychiatric disorders, particularly schizophrenia and uni- or bipolar disorders. However, since the virus has also been detected in clinically normal patients, a role for BDV in the development of these complex psychiatric disorders has still to be proven.

The presence of BDV infection in many domestic species, and evidence of cross-species transfer raise the possibility of zoonotic spread. However, while animal species may pose a potential risk to humans, finding BDV RNA in blood from normal human blood donors suggests that humans may perhaps be as much at risk from horizontal spread between humans. Considerably more investigation needs to be performed before the zoonotic potential of BDV can be determined.

Bartonellosis

Etiology

Bartonella (B.) hensalae is a small, curved, gram-negative, facultative intracellular bacterium. It is closely related to *B. clarridgeiae*. Both of these organisms can be found in the blood of asymptomatic cats.

Reason for Interest

While *B. hensalae* and *B. clarridgeiae* are currently believed to cause little or no disease in cats (see below), it is now known that in humans *B. hensalae* is the major cause of cat scratch disease (CSD), bacillary angiomatosis (BA), and visceral bacillary peliosis (BP). *B. clarridgeiae* has also been implicated in CSD.

Geographical Distribution

Serology has shown that *B. hensalae* and, to a lesser extent, *B. clarridgeiae* are common infections in cats throughout the USA. The presence of *B. hensalae* has also been confirmed in the UK, France, Portugal, the Netherlands, Australia, the Hawaiian islands, Japan, Israel, and Egypt. It is presumably found throughout most

emperate regions of the world. A higher prevalence of seropositive individuals is generally found in older cats, especially those coming from warm, humid, flea-infested areas, or feral populations.

Signalment

Most cats are infected with *B. hensalae* when they are kittens; this probably occurs when they are first bitten by fleas. Cats from households with a significant flea problem are therefore more likely to become infected. Most *B. hensalae* infected cats are asymptomatic. To date, only mild disease has been confirmed in cats experimentally.

Transmission

B. hensalae is an intra-erythrocytic bacterium. Once infected, cats can remain bacteremic for many months, and may experience relapsing bacteremia. While the infection can be transmitted from cat-to-cat in blood transfusions, the natural route of transmission appears to be via fleas. Whether the fleas act as mechanical or as biological vectors is currently unclear. While very few fleas are needed for transmission to occur between cats, transmission does not occur in the absence of fleas.

Clinical Signs

In the vast majority of cats, infection with *B. hensalae* is subclinical. If it does cause disease, it is most likely to occur in chronically infected cats, associated with episodes of stress, or in conjunction with other diseases.

Experimentally, infection with *B. hensalae* can result in a self-limiting febrile illness of 2-3 days duration. Some cases have also shown anorexia, generalized peripheral lymphadenopathy, and inflammatory lesions in multiple organs. Other cases have demonstrated mild neurological dysfunction, with lethargy, staring expression, disorientation, unresponsiveness, and ataxia.

While not yet proven, there are a number of feline diseases for which *B. hensalae* infection is being investigated as a potential cause. These include persistent focal or generalized lymphadenopathy, peliosis hepatis, chronic relapsing uveitis, and cataract formation. However, because so many chronically bacteremic cats are healthy, it will be difficult to establish a causal relationship between bacteremia and these specific disease presentations.

Diagnosis

While serology may provide useful epidemiological information, it is of limited clinical use as its correlation with a positive blood culture is poor. While high titers do tend to correlate fairly well with a positive culture result, low titers may occur in culture-negative cats and, occasionally, culture-positive cats may be antibody negative. Blood culture needs to be performed by a specialist laboratory. *B. hensalae* may also be detected by specialist staining of peripheral blood smears.

Most cats show no pathological changes. Experimentally, cats have shown lymphoid hyperplasia, and inflammatory foci within the lymph nodes, spleen, and liver.

Treatment

Unfortunately, the efficacy of antibiotics to remove *B. hensalae* from cats has not been clearly established. Several antibiotics that are effective in humans are ineffective in cats, even after prolonged administration. Also, different studies have generated contradictory results. At present, it is suggested that enrofloxacin, doxycycline, and rifampin can be used either alone or perhaps in combination. Treatment should be given for 2-4 weeks. Repeat cultures should be taken 2-4 weeks after finishing the treatment.

Zoonotic Risk

CSD occurs world-wide. It is seen most commonly in children and adolescents, most frequently males. There is a seasonal incidence, with 60 percent of cases occurring between September and January. Occasionally, multiple cases may be seen within one household. Since the estimated incidence in the USA is nine cases per 100,000 people, it represents a very significant problem, both medically, and financially. CSD is a significant occupational hazard for the veterinary profession.

Initial suspicions that cats played a role in transmitting CSD came from serological studies. These showed that 90 percent of CSD cases had contact with a cat, and 80 percent of CSD cases had had a cat bite or scratch. At present it is not known whether the organism is passed by cats to humans by direct contact (e.g., bites or scratches), or indirectly (e.g., by fleas).

The vast majority of CSD cases are benign and self-limiting (> 85 percent cases). In these cases there is typically a subacute regional lymphadenopathy, a papular skin lesion at the site of inoculation, a low grade fever, malaise, and myalgia. Very rarely, atypical disease may occur. This may take the form of encephalopathy, an oculo-glandular syndrome (after inoculation into the eye), arthralgia/arthritis, hepatitis, or pneumonia. Even in these cases, the disease is rarely fatal.

In immunocompromised individuals, particularly those suffering from AIDS, *B. hensalae* infection may result in the development of either bacillary angiomatosis (BA), which consists of vasoproliferative blood-filled cysts in the skin, or bacillary peliosis (BP) which consists of vasoproliferative blood-filled cysts in internal organs, especially the liver.

Very rarely, *B. hensalae* infection can cause a relapsing bacteremia associated with fever, malaise, fatigue, and weight loss.

Since fleas appear to play a major role in the transmission of *B. hensalae* between cats and possibly from cats

to humans, it is advised that cats owned by immunocompromised individuals be checked for the presence of *B. hensalae* and then maintained under a strict flea-control regime. Also, since CSD is frequently associated with exposure to kittens, immunocompromised people should keep adult rather than young cats. It is advised that they do not play roughly with them, and that they wash their hands after handling them. In the longer term, attempts are being made to develop a vaccine to protect cats against *B. hensalae.*

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