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INFECTIOUS CAUSES OF ABORTION IN THE CAT

Donald H. Lein, DVM, Ph.D.
Director
Diagnostic Laboratory
Associate Professor
Pathology and Theriogenology
New York State College of Veterinary Medicine
P.O. Box 786
Ithaca, NY 14851

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Several infectious diseases are the cause or associated with the female and occasionally the male reproductive system of the cat.[1,2,3,4,5,6,7,8] Most notable are the viral diseases that cause infertility, embryonal-fetal death and resorption, malformations, abortion, mummification, stillborns and kitten neonatal death. Two classical viral infections, panleukopenia and feline viral rhinotracheitis (Feline herpesvirus-1) cause the above clinical manifestations by transplacental fetal viral infections. Feline leukemia virus and feline infectious peritonitis virus have been associated with the above clinical manifestations but have not been directly incriminated. Since both of these viruses cause immunosuppression in cats, this mechanism may be involved in the above clinical reproductive disorders.

Other viral infections, such as the calicivirus infection, may cause stress during active infections of pregnant queens resulting in abortions. Their direct role as a primary cause of infection has not been documented.

Bacterial infections have been incriminated with infertility in the queen, resulting in abortion and infertility caused by endometritis, pyometritis, cervicitis and vaginitis. Fetal death, emphysematous fetuses, and septic metritis can be caused by bacterial infections. Postpartal endometritis, metritis, mastitis and neonatal septicemias are caused by bacteria. Several common bacteria are involved including Escherichia coli, Streptococci, Staphylococci, Salmonella spp. and Pseudomonas spp. Mycoplasma and Ureaplasma agents are frequently isolated from the urogenital tract of the cat, with special media,but their role in causing disease and inflammation of this organ system has not been studied.[9] Certainly the role of Mycoplasma and Ureaplasma in causing reproductive disorders in man and cattle have been well documented and more recently their role and association in dog and horse infertility is being investigated more fully.[10,11,12,13,14]

Viral Infection

Feline Panleukopenia (FPL)

Inutero infections with FPL virus, a parvovirus may result in early neonatal death and resorption, abortion, mummification, stillbirths, weak neonates, early neonatal death and malformations such as cerebellar hypoplasia.[15,16,17] These clinical manifestations depend on the age of the embryo or fetus when the viral infection takes place. Any cell in mitotic division is a target for this virus to multiply, resulting in various clinical effects. Microscopic lesions can be noted in various tissue deficiencies; granular cells of the cerebellum, decreased Purkinje cells, decrease neuronal cells in cortical substance with internal hydrocephalus, are the most notable cells affected.

Feline viral rhinotracheitis (FVR), Feline herpesvirus I

Inutero infection with FVR virus can result in abortion, fetal death and congenital fetal infections. Experimental infection between 42 and 50 days of gestation by intravenous injection of the virus resulted in placental lesions which included multiple infarcts in the placental labyrinth, thrombosis of maternal vessels in the endometrium and placenta, and multifocal necrosis of the giant-cell trophoblast and endometrial epithelium in the junctional zone of the placenta.[18] The affected cells contained eosinophilic intranuclear inclusion bodies. Abortions after intranasal inoculation revealed no virus, viral antigen or significant lesions in placental or fetal tissue. It was thought that these abortions were secondary to the severe maternal disease and induced probably by stress alone.

Feline herpesvirus 1(FVR) can cause viral vaginal infections, similar to that seen with herpes infection in the canine, bovine and human herpes viruses with vaginal or vulvar infections. Kittens born during the active experimental induced herpes vaginal infection had generalized herpes infections, fibrinous purulent rhinitis and tracheitis, fibrinous suppurative bronchopneumonia, and focal areas of hepatic necrosis with intranuclear inclusion bodies in affected bronchial epithelium and hepatic cells.[19]

Herpesvirus - Induced Feline Urolithiasis

A distinct feline herpesvirus different from type 1 has been incriminated as a cause of infectious urolithiasis in cats. This cell associated herpes virus (CAHV) is frequently isolated from the ovaries of infected queens.[20] Pathology studies of affected ovaries have shown a necrotic oophoritis similar to that reported for cattle infected with IBR.[21]

Feline Leukemia Virus (FeLV)

Feline leukemia virus, a retrovirus, has been associated with infertility, embryonal-fetal death and resorption, abortion, stillborns,

endometritis and birth of fading kittens.[22] Its direct involvement has not been proven. It appears that queens with FeLV can have the above clinical manifestations. Since immunosuppression is a clinical manifestation of FeLV, one should also look for other factors involved with that syndrome: bacterial endometritis, toxemia, upper respiratory viral infections and toxoplasmosis. One report of 30 kittens, born from latent FeLV carrier queens, revealed that only 1 was born with active FeLV infection.[23] This kitten appeared healthy, fast growing, with no clinical lymphadenopathy or anemia and was the fastest growing kitten in the litter. Both negative litter mates became viremic and developed generalized lymphadenopathy, one at 10, the other at 13 weeks of age. It is reported in another study that nearly 75% of FeLV infected queens experienced abortions and/or fetal resorptions.[24]

Feline Infectious Peritonitis (FIP)

Like FeLV, FIP, a coronavirus has also been associated with infertility and embryonal and fetal death. It has not been directly implicated as the causative agent.[6,25] Again, it can be immunosuppressive and may be causing a secondary effect.

In both FeLV and FIP infected catteries, the author has noted continuous repeating infertility, embryonal-fetal death, abortions, weak and dying neonatal kittens in several cycles of active queens leading to sterility in some or eventual improvement of fertility in others. This is much different than the previous described viral infections that result in a postinfection solid immune state and resumed fertile cycles.

The Cornell Feline Health Center has compiled data from 1975 to 1980, on the phenomenon known as kitten mortality complex (KMC), revealing a kitten mortality rate of 31% on 9,517 kittens from 2,309 litters representing 28 breeds. 9.3% of these were stillborn.[26] Of 8,630 kittens born alive, 5.8% died within 24 hours of birth, 2.8% died the second day, 13.2% had died by the first week and 24.2 % failed to reach one year of age. KMC was characterized by 3 main problems: reproductive failure, kitten mortality and various diseases in the adult. Reproductive failures included repeat breeders, fetal resorption, abortions, stillbirths and congenital malformations.

Toxoplasmosis

Toxoplasmosis has been incriminated as a causative agent of abortion in man and sheep. Pregnant queens, with an active toxoplasmosis infection, will have a toxoplasemia with transplacental infection, placentitis and fetal infection resulting in fetal deaths, abortions, and infected neonates. Abortions during clinical toxoplasmosis in pregnant queens have been reported.[27,28] Demonstration of the organism by histopathology or isolation by mouse inoculation and developing toxoplasma serum titers in the queen are needed for a positive diagnosis.

Bacterial, and possibly mycoplasma, and ureaplasma septicemias in the pregnant queen certainly can result in endometritis, placentitis and fetal infection resulting in abortion and weak septic neonates. E. coli, Beta-hemolytic streptococci and staphylococci, are most frequently found.

Mycoplasma and ureaplasma agents are not cultured for routinely and reports of spontaneous infertility disease for these agents in cats are lacking.

Streptococcus canis was identified as a cause of kitten mortality at 7 to 10 days after birth in catteries.[29] Each kitten had acquired an umbilical infection at birth from a carrier state found in the queens vagina while delivering the kittens. Internal abscesses at the end of the umbilical cord containing S. canis were found at necropsy. Following queening, the vagina remained clear until about 2 weeks postpartum when recolonization by S canis took place. Prevention by dipping the kittens umbilical cord in iodine immediately following birth was successful.

Group B streptococcus was isolated with hemolytic Escherichia coli from the uterus of a queen 2 days postpartum.[30] The queen delivered 5 kittens. Two were delivered without assistance, the others following an oxytocin injection. Two days postpartum, the cat was presented in a febrile, depressed state with a bloody vaginal discharge and enlarged uterus. The uterus contained retained placentas. Recovery was uneventful following post surgical treatment with trimethoprin-sulfadiazine for 10 days. Group B streptococcus are a common cause of neonatal sepsis in human infants, a common pathogen causing mastitis in cattle and referred to as S. agalactiae, and a cause of canine neonatal death.

Infertility in the tom is infrequently seen or reported[8,31,32] although purebred or inbred toms should be examined closely. Since the role of the tom in possible venereal spread of infectious diseases is unknown, negative status for toxoplasmosis, feline leukemia virus and feline infectious peritonitis FIP virus is important when obtaining a breeding tom. Scrotal distension, which is usually cool, painless and fluctuating, may be associated with the effusive peritonitis of FIP. Congenital testicular hypoplasia may be a consequence of fetal or neonatal panleukopenia infection. Prior to breeding, adequate vaccination and preventive health care are essential.

It is important to culture the prepuce and/or penis of the tom for bacteria when queens in a colony are affected with vulvar discharges and infertility. Frequently similar organisms, usually E. coli, staphylococci or streptococci, will be found in the queens and tom cats of these colonies. In this situation, the tom should be separated from the queens and treated with the antibiotic of choice similar to the queen before resuming breeding practices. A case of suppurative prostatic disease has been reported in a tom cat, but this is rare[33].

Bite wounds of the scrotum and testes are frequently found following territorial fights. A fever and bite wound cellulitis or abscessation of the scrotum are indications for treatment with antibiotics and drainage, but if resolution is not rapid, the affected testis may have to be removed to save the nonaffected testis from heat degeneration and extension of the infection. chronic epididymitis, orchitis, or periorchitis will lead to degeneration of the contralateral testis from heat-induced and immune-mediated injury. This can be prevented by removal of the affected testis.

Infectious feline infertility and kitten mortality in catteries can be

reduced by proper scheduling vaccinations prior to breeding and vaccination of kittens early, possibly at 4 or 6 weeks of age instead of 8, if respiratory infections are a problem.[26] Serological test for FeLV and toxoplasmosis should be conducted and positives segregated or eliminated. Cats returning from shows or breeders should be isolated for 2 or more weeks, and retested for and new diseases. New cats should be properly vaccinated and tested free of FeLV, FIP and Toxoplasmosis. Outdoor animals should not be allowed free access to mingle with cattery cats. Pregnant queens, nursing queens and kittens should be separated from other cattery cats. Non-productive cats, show cats, etc., should be separated from the breeding colony. Separate clothing and cleaning hands prior to entering the nursery are important. General cleanliness of the cattery should be optimal with frequent replacement of litter and pans. Floors, litter pans, and food dishes should be regularly cleaned and disinfected with a detergent and chlorine diluted at 1:32. Stress should be eliminated: rest queens between litters, with not more than 2 litters per year, avoid stress of shows for breeding cats, avoid toxic substances- phenol, smoke-exhaust fumes, formaldehyde-based insulation, drugs during pregnancy (griseofulvin is teratogenic in the pregnant queen). Avoid inbreeding or line breeding which may result in lethal genes and genetic malformations and other disorders. Taurine deficiency has been recognized to cause infertility, mainly fetal resorption, abortions, stillborns and weak neonates.[34]

The diagnosis of feline abortion and neonatal death requires the full cooperation of owners and veterinarians to conduct an intensive investigation with a well documented history, clinical examination and collection and submission of all aborted fetuses, placentas and vaginal discharges (swabs) along with serum from the queen to a diagnostic laboratory. Fetal tissues should be placed in 10% buffered formalin or better, Bouin's solution for best demonstration of viral inclusion bodies and also fresh chilled or refrigerated tissue (not frozen if possible), along with an acute blood for serology and sent to a competent diagnostic laboratory for viral, bacterial, toxoplasma, mycoplasma and ureaplasma isolations and serological titers. A convalescent serum, 10 to 21 days later should be resubmitted. Vaginal discharges following suspected fetal resorptions should be collected with a sterile swab or aspirated into a sterile syringe and placed in transport media for viral, bacterial and mycoplasma agents. Amies transport media is excellent for both bacteria and mycoplasma agents.

Animals suspected of repeated fetal resorption problems should be monitored for early pregnancy diagnosis with ultrasound. Ultrasonic techniques for pregnancy and fetal well being throughout pregnancy can start at day 15 post breeding to term. Viral infections can be adequately protected against by administrating good commercial vaccines prior to breeding. Adequate programs must be maintained in any cattery. No modified live vaccines should be given to queens during pregnancy. Catteries negative for FeLV and FIP, and toxoplasmosis are ideal. Serological monitoring of all cats and new cats prior to addition is beneficial to control. Catteries with Toxoplasma problems should be examined for fecal shedding individuals. Infected fecal shedders should be isolated from the colony, treated and monitored. Sources other than shedding cats, such as raw meat and rodents should be prevented. Fetal tissues from suspected Toxoplasma abortions should include brain, heart,

liver, spleen, lung, bone marrow and kidney for histopathology and mouse inoculation. Histological lesions may show only local area of necrosis and mineralization. These areas should be checked carefully for evidence of Toxoplasma cyst with silver stains.

Cystic hyperplasia-endometritis-pyometra complex is a condition seen in queens ranging from 3 to 14 years of age but especially in nulliparous queens over 5 years of age.[35] The incidence is decreased in free-roaming queens that have consecutive or frequent pregnancies. Dow showed that this syndrome is similar to that in the bitch, starting with cystic endometrial hyperplasia induced by estrogens and progesterone of consecutive periods of pseudocyesis or infertile breedings followed by subacute, acute and chronic endometritis and open or closed pyometra.[36] Escherichia coli is most frequently isolated, but staphylococci, streptococci and Pseudomonas spp. have also been incriminated.

Endometritis and pyometra can be a postcoital infection. These affected queens are usually seen in high density colonies where a high number of bacteria will be isolated from vaginal discharges following breeding or from aborted fetuses, stillborns or neonatal septicemia. Most frequently, hemolytic E. coli or beta hemolytic streptococci are found in pure or mixed cultures. Staphylococcal and Pseudomonas infection have also been incriminated. Tom cats breeding these queens may have high carrier states of these organisms on their prepuce and penis. Catteries with FeLV and/or FIP infected population appear to be highly prone to these urogenital tract infections due to the immunosuppressive role of these viruses. This syndrome is characterized by poor conception, early embryonic death and resorption, abortion, macerated fetuses, stillborn and neonatal septicemias and mucopurulent vaginal discharges.

Affected queens may appear normal, with or without a mucopurulent vulvar discharge or may show various degrees of clinical signs resulting in a possibly fulminating septic acute endometritis with a bloody vulvar discharge, depression, toxic bone marrow depression, septicemia and toxemia leading to rapid death.[35] Pyometra may be closed or open with drainage and bloody to mucopurulent discharge. The uterus can be paper thin in the closed condition and rupture with extensive peritonitis and rapid death is possible. In older queens with cystic endometrial hyperplasia and pyometritis, large single or multiple cystic endometrial polyps may be present. They frequently are pedunculated and may prolapse through the cervix resulting in constant persistent drainage. Use of ultrasonography or radiographs to diagnose the polyp is helpful prior to therapy since medical treatment of this condition is unsuccessful and panovariohysterectomy is the treatment of choice when endometrial polyps are present. Possibly in valuable breeding queens the polyps could be surgical removed and the infection treated.

Queens with endometritis or pyometra have total white blood cell counts that may be in the high normal range, 20,000 mm^3, to a leucocytosis, 100,000 $+/mm^3$, or may appear leukopenic because of toxic bone marrow depression. About 50 per cent of cats with pyometra have a normocytic, normochromic anemia.[35] Culture of vulvar discharges (vaginal culture), or uterine contents, with antibiotic sensitivities for antibiotic choice, supportive therapy and ovariohysterectomy are indicated.

Queens intended for future breeding should be given sexual rest as soon as signs of vulvar discharge, endometritis or pyometritis are observed. A deep vaginal culture and cytologic smears are obtained by using a sterile saline-moistened swab or by flushing the vagina with sterile saline or water via a syringe with either a sterile bovine teat cannula or tom cat catheter or use of an eyedropper. The use of a sterile 4mm or less diameter otoscope head as a speculum will decrease contamination of the specimen from the perineal and vestibule tissue. Systemic treatment with antibiotics, sensitive to the isolated organism, for a prolonged period of time is highly beneficial. If this is a cattery problem, all affected cats should be treated, including breeding males with carrier states. Sexual rest is mandatory. Culling of non-breeding population is important to reduce the population density. A complete cleansing of the cattery with detergent and chlorine (1:32 dilution), replace all litter, litter pans, and if possible move treated animals to new clean quarters and leave the old cattery vacant for a period of time are all highly beneficial. Check ventilation for adequate air flow and if possible place treated cats in outside cages and let the cleaned indoor cattery remain empty for as long as possible before repopulating.

Closed or open pyometras may be treated successfully with systemic antibiotics and prostaglandin F_2 alpha at 25 ug/kg or up to 1000 ug/kg given b.i.d. or o.i.d. respectively, intramuscularly for 2 to 5 days or to effect.[37] The lower dose is preferred initially until adequate vulvar drainage and side effects are determined for the individual. This drug is not licensed for use in cats. Prostaglandins should not be used if endometrial polyps are present since cervical blockage may lead to retrograde passage of exudate into the peritoneal cavity via the oviducts. Daily flushing of the vagina with a tom cat catheter and syringe containing 10 to 20 ml of warm sterile saline may enhance uterine evacuation and vaginal flow during the prostaglandin therapy. Daily monitoring of vulvar discharge and palpation per abdomen of uterine size and tone will monitor the extent and need of further prostaglandin therapy. System antibiotic therapy should be carried on for several days (usually for 10 to 14 days) post prostaglandin therapy.

Two queens with E. coli pyometritis were reported to be successfully treated with 200 ug/kg/day for 2 days and 500 ug/kg/day for 5 days with prostaglandin F_2 alpha along with oral administration of trimethoprin-sulfadiazine, 30 mg/kg bid for 30 and 21 days respectively.[38] Both cats were successfully bred and produced healthy kittens post treatment.

Sexual rest, normal estrous without vulvar discharge, normal hemograms, small palpable uterus, negative vaginal cultures with normal vaginal cytology are necessary before attempting breeding. Tom cats in affected colonies should also be cultured post treatment to look for low numbers of bacterial flora. The prognosis for catteries or individual queens following the above is quite favorable if the treatment and hygienic cleaning of the cattery is successful. The author has seen catteries where successful litters and reproductive performance were severely disrupted by E. coli, Pseudomonas or Streptococcal infections. Successful therapy, drastic culling and strict hygienic cleaning of the cattery was successful in returning the catteries to reproductive sound states. Autogenous

bacterins have been tried, but their success cannot substitute for good management, strict hygiene, rational culling (over population) and proper nutrition. Taurine deficiency can lead to infertility, resorptions and small litter size which can be confused or misdiagnosed as infectious infertility.

Chronic cervicitis and vaginitis can be seen in the queen.[4] A discharge may be difficult to detect. These infections are caused by trauma and infection following a dystocia or delivery of large kittens. Stenosis may follow, with closure of the vagina at the vestibule or any area forward to the cervix with fibrosis. The cervix may be stenotic or closed and fibrotic. Diagnosis is possible using sedatives and examining the vaginal and cervical area with a sterile otoscope cone. Cervicitis and vaginitis should be treated with systemic antibiotics and local douching with warm sterile saline following cultures and antibiotic sensitivity of isolated organisms. E. coli, staphylococci and streptococci organisms are usually found.

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