Viral diseases of dogs

Rabies

It is an acute fatal infectious disease of worm-blooded animals and man characterized by abnormal behaviors, encephalomyelitis, and progressive ascending paralysis ending with death.

Annually, rabies causes the deaths of more than 50,000 humans and millions of animals worldwide. Once symptoms appear, the disease results in fatality.

Etiology

The disease is caused by neurotropic RNA *lyssa virus* of family *Rhabdoviridae.* It is classified as genotype 1, serotype 1 in this genus.

- It is a bullet shaped virus 75X180 *nm* in diameter.
- It is relatively fragile; dies in dried saliva in few hours and inactivated by exposure to ultra violate radiation, 1 % formalin or 3% cresol, boiling, 70% ethanol, soap solutions, ether, chloroform, acetone, 1% sodium hypochlorite, 2% glutaraldehyde, iodine preparations, quaternary ammonium compounds, and formaldehyde.
- It is rapidly inactivated in sunlight, and it does not survive for long periods in the environment except in a cool dark area.
- Rabies virus remains infectious in a carcass for less than 24 hours at 20c and survives days when the carcass is refrigerated.
- Slight antigenic variation among viruses isolated from various sources has been recorded using monoclonal antibodies where four strains had been detected.
- Strains of rabies virus isolated from natural cases are called **street viruses** and strains adapted to secondary hosts are called **fixed viruses.**
- Strains isolated from the brain of human patients and have been passed repeatedly in a variety of host systems are referred to as **Flury strains**.
- Accumulations of viral proteins in the cytoplasm constitute the inclusions seen histopathologically in infected nerve cells described as **Negri bodies**.

Epizootiology

Geographical distribution

It is a disease of a worldwide distribution, and enzootic in Africa

including Egypt. Few countries are free from rabies as Australia, New Zealand, Britain, Hawaii and Japan (Island countries).

Susceptibility

All worm blooded animals are susceptible. Carnivores and bats are the primary vector species. Dogs, cats, Jackals, Mangoes, Wolves, Raccoons, Coyotes, Skunks and Foxes are highly susceptible.

- Asymptomatic carriers are possible in species adapted to rabies.
- Bats are symptomless carriers in which replication of the virus occurs in fatty tissues without invasion of nervous system.

Sources of infection

1. The virus is present in saliva of infected animal two to five days before appearance of clinical signs (incubatory carrier).
2. Domestic and wild carnivores as well as bats are the main sources of infection especially stray dogs and cats.
3. In countries where dogs and cats are well controlled, Foxes, Vampire, insectivorous and fruit eating bats are the major sources of infection.
4. Vampire bats secrete the virus for several months.

Transmission

A- Common routes of transmission are:

- Bite of an infected animal where saliva is deposited in the bite wound.
- Contamination of skin wounds or lacerations by fresh saliva of an infected animal.
  - Not all bites from rabid animals result in infection because the virus is not always present in saliva and may not gain entrance to the wound if clothing or the coat of the animal wipes saliva.

B- Less common routes of transmission are:

1. Aerosol infection: Two cases of rabies occurred in a cave in Texas without bite wounds. A number of Foxes and Coyotes were subsequently placed in that cave. All animals died of rabies and the virus was isolated from the air of the cave.
2. Ingestion: Infection may occur because of ingestion of an infected carrier as dead rabid bat or infected tissue.

- Infection in humans may have been acquired in a laboratory (by aerosol). In addition, transmission has been occurred in man following transplants of corneas taken from infected patient.

The main animals involved differ from continent to continent.

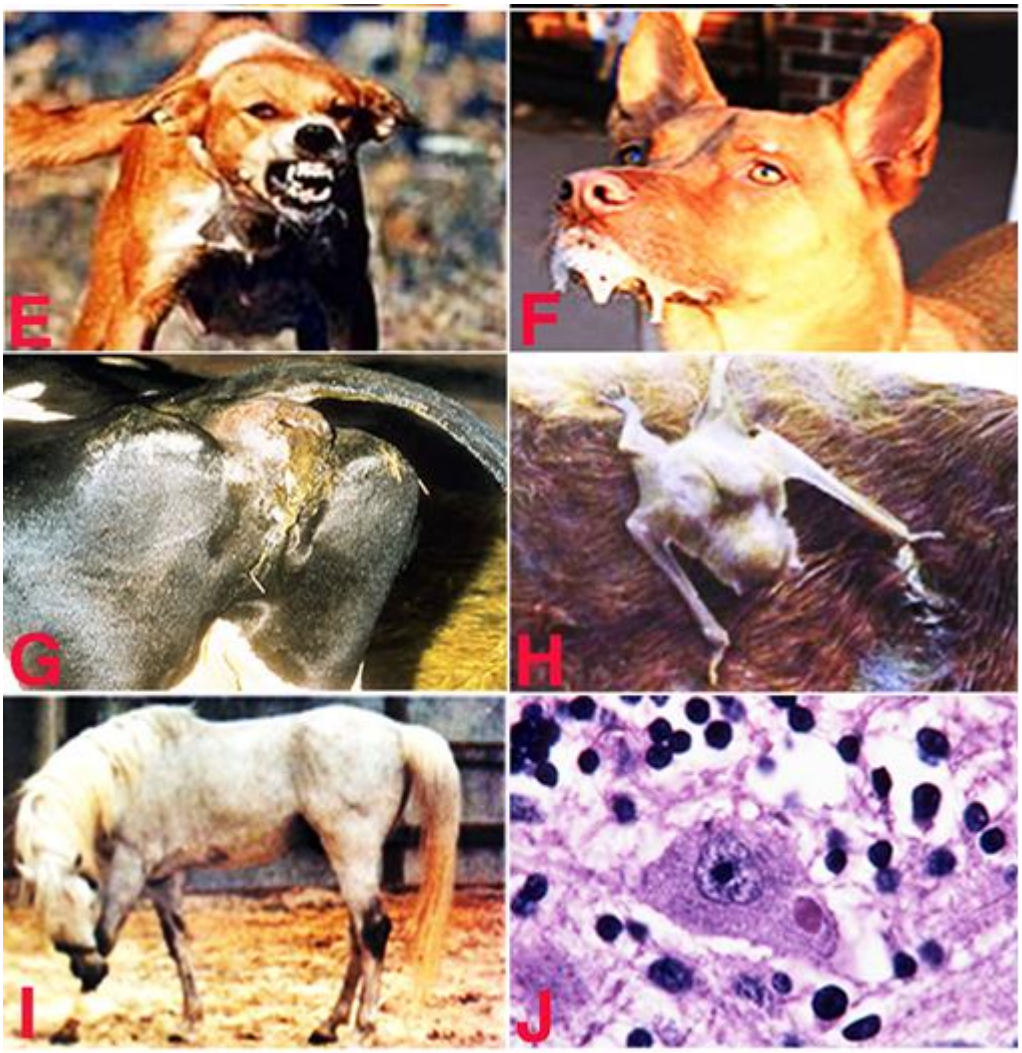
Europe: fox, bats
Middle East: wolf, dog

Asia:	dog
Africa:	dog, mongoose, antelope
N America:	foxes, skunks, raccoons, insectivorous bats
S America;	dog, vampire bats

Epidemiological cycles

The disease is characterized by different "epidemiological cycles":

- **Urban rabies:** The transmission cycle in urban rabies is principally sustained in dogs it is also called the “terrestrial cycle”. Dogs account for most human rabies cases. This cycle occurs in areas where there is high proportion of unvaccinated dogs especially in Africa, Asia and South America.
- **Sylvatic or “wildlife rabies”:** It is the dominant transmission cycle in Europe and North America e.g. Fox rabies and Raccoon rabies.
- **Bat rabies or “aerial cycle”:** in which the bat is the major sylvatic rabies vector transmitting rabies especially to cattle and humans.



Pathogenesis

After introduction of the virus into a bite wound, limited multiplication may occur in epithelial cells and monocytes. The virus passes through sensory nerves to skin. A virus journey within axons of nerves is at a rate of 0.5 to 15 inches per day. It may take weeks before the virus reaches the spinal cord motor neurons of the CNS. This is why post-exposure vaccination is possible. The virus proliferates centripetally along peripheral nerves to the CNS. The virus proliferates rapidly in spinal cord and brain and travels centrifugally to the salivary glands through the axon of trigeminal, facial, olfactory, and glosso-pharyngeal nerves. The virus passes into oral and nasal secretions. There are some evidences that the virus may be secreted in milk and reach to the fetus.

Incubation period ranges from 3-8 weeks and it depends upon:

1- Site of the wound bite.
2- Virulence of the virus.
3- Quantities of the virus.

Clinical signs

Dogs

The clinical course of the disease in dogs is divided into three phases: **prodromal, exitative (furious) and paralytic (dumb) phases**. The term furious rabies refers to animals in which the exitative phase predominates. Dumb rabies refers to dogs in which the exitative phase is extremely short or absent.

A- Prodormal phase

This phase is manifested by a change in normal behavior of the animal. In dogs it is two to three days, in cats, one day and in cattle about few hours.

1. Infected animals stop eating and drinking
2. Friendly dogs become irritable.
3. Stimulation to the urogenital tract with frequent urination and erection.
    - After this period, infected animal either shows signs of paralysis or becomes vicious.

B- Furious phase (classical mad-dog syndrome)

1. In this phase the animal is hypersensitive to external stimuli and becomes aggressive and noise invites attack.
2. Animal loses all caution and fear of natural enemies.
3. Infected dogs frequently roam streets and high ways.
4. Infected dogs bite other animals, people, and any moving objects.
5. in this stage Infected dogs commonly swallow foreign objects as stones and chew wires and frame of the cages and break their

teeth.

٦. Rabid dog will follow a hand moved in front of the cage attempting to bite.

٧. Infected dogs rarely live beyond ten days from onset of the signs.

- Death may occur in this stage or may continue to the paralytic phase

C- Paralytic form

This form is manifested by:

1. Paralysis of the throat and masseter muscles associated with profuse salivation and inability to swallow.
2. Dropping of the lower jaw is a common sign.
3. Paralysis progresses rapidly to all parts of the body with coma and death (flaccid paralysis and prostration).

- Hydrophobia is not a common feature.
- Death is the result of progressive paralysis.

Cats:

Cats having dumb rabies are depressed or attempting to hide or isolate themselves from their owners. Cats may also appear to be paralyzed in the head and neck region. In the furious form cats are overly excited or aggressive towards anything around them, attacks suddenly, bites and scratches viciously.

Fox:

Infected Foxes attack people, dogs, and other animals

Cattle

1. The animals stop eating and drinking with drop in milk production.
2. Pharyngeal paralysis and salivation occur.
3. Characteristic bellowing sound (Low pitched voice).
4. Animal may become aggressive and attack other animals and man.
5. Sexual excitement.
6. Paralysis of the anus and bladder also occurs.

Horses

1. Colic, rolling and chewing of foreign materials are characteristics.
2. Horse may bite viciously.
3. Intense sexual excitement occurs.
4. The animal usually suffers from self-inflected wounds especially in the genitalia.
5. Ataxia and paresis, hyperesthesia, pharyngeal paresis or paralysis, tail, perineal and anal sphincter hypotonia, circling and hypersalivation.

Postmortem lesions

Presence of foreign bodies in stomach of the dog, broken teeth and

wearing of the foot pads are the common findings.

Diagnosis

1. Epizootiological situation of the disease.
2. Clinical signs.
3. Laboratory diagnosis.

Samples: The head should be removed and send to the laboratory on ice. Cerebellum and brain stem are the best tissues. Saliva and salivary glands should be submitted to the laboratory on ice or refrigerated.

a. Virus isolation from brain or saliva on monolayer cell culture of neuroblastoma cells. The virus can be identified by FAT.
b. PCR. Real-time PCR not only allow rapid diagnosis but also differentiation between lyssa virus species.
c. Detection of the viral antigen in impression smears from Ammon's horn or corneal impression before death using FAT. It is a rapid and highly specific test.
d. The use of immunoperoxidase technique for tissue sections to demonstrate the viral antigen.
e. Serodiagnosis: The presence of neutralizing antibodies is not diagnostic. Sero - tests include VNT, CFT and ELISA.
f. Demonstration of the intracytoplasmic inclusion bodies (eosinophilic cantaining basophilic granules) called Negri bodies, in impression smears or tissue sections stained with sellers or Giemsa stain. Negri bodies are not present in the early stages of the disease and present only in 70-80% of cases of bovines. Failure to find Negri bodies not excludes rabies.
g. Intracerebral inoculation of suckling laboratory mice is indicated in negative finding for Negri bodies. Mice are inoculated intracerebrally with 10% brain suspension and observed for 21 days with examination of the brain for presence of Negri bodies. The test is considered negative after 28 days.

Differential diagnosis

1. Dogs: The disease should be differentiated from Pseudorabies and Canine distemper.
2. Cattle and sheep: The disease is confused with Listeriosis, BSE, MHC, Pseudorabies, Scrabie, Louping ill, acute lead poisoning, vitamin A deficiency and ketosis

Control

Control of rabies depends upon:

1. Elimination of stray dogs.
2. Reduction of excess number of wild life vectors.

3. Mass vaccination of dogs.

These steps can be achieved through:

1. Destruction of all rabid animals.
2. Destruction of stray dogs.
3. Muzzling, licensing and vaccination of all other dogs.
4. Washing of bite wounds and scratches using soap 20%, water and 70% alcohol.
5. Control of wildlife:
   a. Trapping of raccoons and skunks to vaccinate them and then return them to the wild.
   b. Oral rabies baits containing vaccine also may be distributed to create immunity and prevent rabies from spreading.
   c. Control of vampire bats. In central and south America, this is done through vaccination of bovine hosts as well as injection of anticoagulant into cattle as coumarin, which kills bats during the four following days.
6. Vaccination: Both live attenuated and inactivated vaccines are used.
   a. Inactivated tissue culture vaccine is used at three months of age then at six months and then annually. It is used for dogs by intra-muscular route in the hind limb.
   b. Attenuated living vaccine. HEP flurry vaccine is given by intra-muscular rout at six months of age and then annually.
   c. Modified live virus vaccine placed in baits is used for control of rabies in wild life.

By 28-30 days post-primary immunization, a peak rabies antibody level is achieved. At this time, the animal can be considered immunized.

- Dogs less than four months of age must be confined at home.
- Regardless of the age of the animal at primary immunization, a second rabies vaccination should be given one year later, and the three-year booster schedule followed thereafter.
  - A country is considered free from rabies when no indigenous rabies has occurred in animals or humans for two years.
  - The quarantine period in free countries is four to six months.

<u>Management of animals and humans that have been bitten by a rabid or suspected rabid animal:</u>

1. Unvaccinated dogs or cats that have been bitten by a known rabid animal should be destroyed immediately but if the owner is unwilling to do that, the animal should be vaccinated and placed in strict isolation in a kennel for six months.
2. Previously vaccinated dog that has been bitten by a known rabid animal should be revaccinated and restrained for 90 days.

3. A bovine that has been bitten by a rabid animal should be destroyed immediately. If such an animal is slaughtered within seven days of exposure, the tissues may be eaten discarding the tissues of the exposed area. Tissue or milk from confirmed rabid animal must not be used for human consumption.
4. In case of human exposure, the suspected animal should be confined for observation for at least ten days, fed, and watered in a special iron cage. The animal should not be killed but be allowed to die. If the animal shows signs of rabies during the ten days observation, it should be killed and submitted for rabies diagnosis.

- In humans, vaccines, antiviral drugs such as ribavirin, interferon-alpha, passively administered anti-rabies virus antibodies (human immunoglobulin. Ig), ketamine and/or the induction of a coma have been tried.
- The bitten person should be provided with prompt scrubbing of the bite site combined with Ig and post exposure vaccine. Vaccinated person should receive two-booster doses 3-days apart, whereas unvaccinated person should receive five-doses of vaccine over 1-month and one dose of Ig at 0 day. (Antibodies prevent the spread of the virus to the CNS, stopping the disease).

Aujesky's Disease

Pseudorabies-Mad Itch

It is an acute fatal neurologic disease of worm-blooded animals with the exception of swine in which the disease may be mild or subclinical. The disease is characterized by marked localized pruritus, paralysis, and fatal termination.

Etiology

The disease is caused by *type 1 porcine herpes* DNA virus.

- The virus is shed abundantly in nasal and oral secretions.
- The virus remains infectious for months in cold temperature or for weeks in worm temperature.
- The virus is readily inactivated by most disinfectants and detergents as 1% NaOH, 2% formalin and 5% phenol.

Epizootiology

Geographical distribution

The disease is enzootic in Midwestern USA, Europe, Middle East, and

Africa.

Susceptibility

Wild life as fox, raccoon, skunks, and domestic animals as swine, cattle, sheep, goats, dogs and cats are susceptible.

The virus infects both dogs and cats primarily those living on farms as well as other domestic animals such as swine, cattle, sheep, and goats.

Sources of infection

Infected animals are the main source of infection. Swine are considered the principal reservoir. The virus is shed in nasal and oral secretions of infected animals.

Transmission

Transmission occurs through:

1. Contact through abraded skin (direct and indirect).
2. Inhalation and ingestion.
3. In pigs, transmission through coitus has been reported.
4. In dogs, ingestion of infected carcass, uncooked meat, offals from swine, or ingestion of infected rats can transmit the infection.

Pathogenesis

The virus replicates in the cells of nasopharyngeal mucosa and the virus reaches the CNS via the peripheral nerves. The virus causes nerve cells damage resulting in encephalomyelitis and pruritus.

Clinical signs

The disease is manifested by:

1. Pruritus which stimulates rubbing, licking, scratching and chewing at the pruritic site.
2. Anorexia, dullness, coughing and vomiting.
3. Pharyngeal paralysis with drooling of saliva may occur.
4. Convulsions, paresis, incoordination, and paralysis. The animal dies in a coma within 24-72 hours.

Postmortem lesions

These include:

1. Severe local trauma because of self-mutilation at the pruritic site.
2. Pulmonary edema.
3. Congestion of the meninges.

Diagnosis

1. Epizootiological situation of the disease.
2. Clinical signs and lesions.

3. Laboratory diagnosis.

Samples: Brain, nerve trunk, tonsils, lungs, and blood serum should be collected.

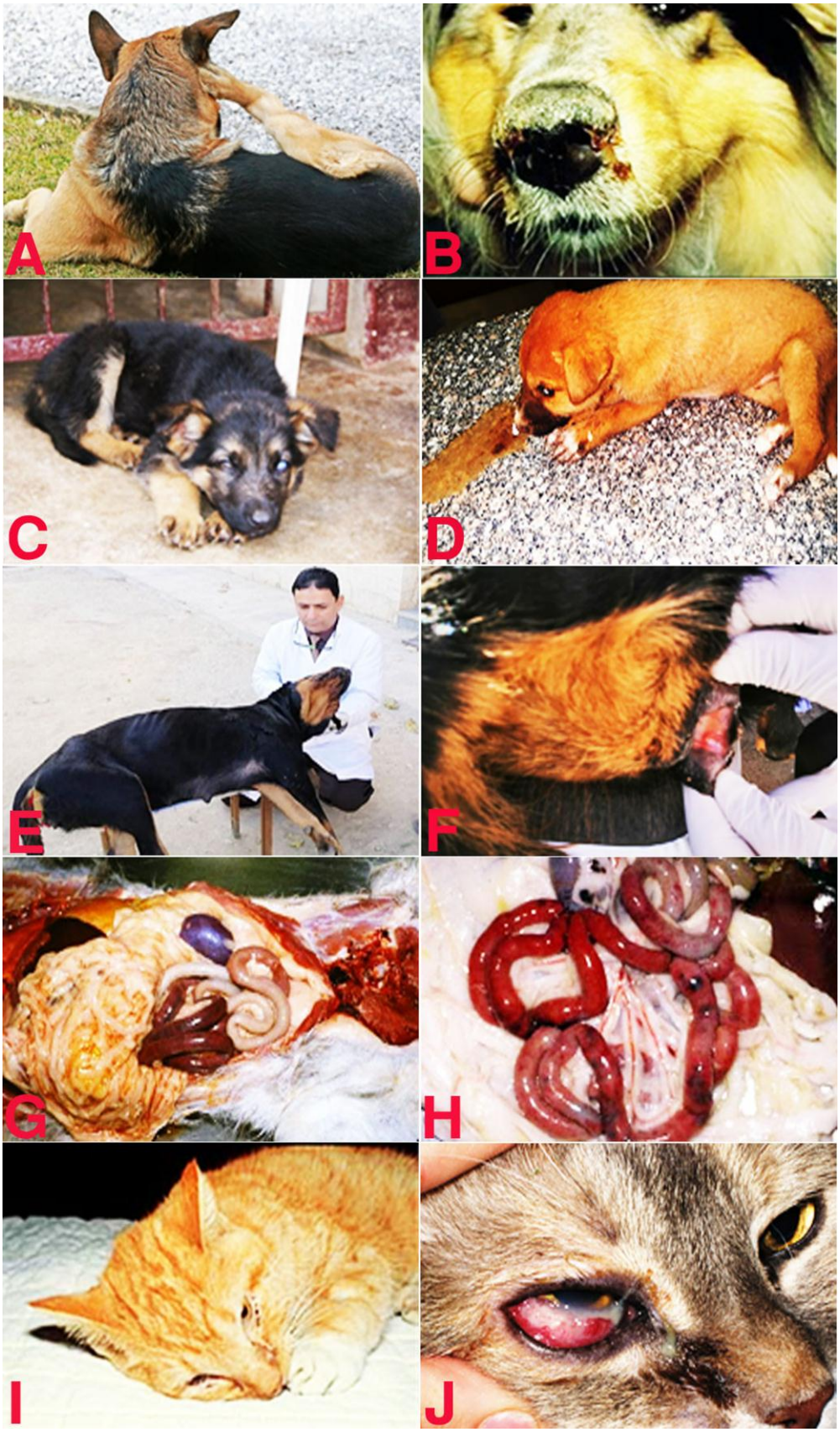
a. Isolation of the virus on tissue culture and identification of the virus using FAT.
b. Laboratory animal inoculation: Inoculation of rabbits by the suspected material produces itching and local inflammation with death.
c. VNT and ELISA for detection of antibodies
d. FAT for detection of the viral antigen in clinical specimens.

Differential diagnosis

This disease should be differentiated from diseases causing nervous signs in dogs such as rabies and canine distemper.

Control

1. Isolation of infected animals
2. Proper disinfection
3. Vaccination


A
B
C
D
E
F
G
H
I
J

Canine Distemper (CD)

Hard Pad Disease (HPD)

It is a highly contagious disease of dogs characterized by fever, gastrointestinal and respiratory tract catarrhal inflammation, leucopenia, and neurological complications.

Etiology

The disease is caused by Canine distemper virus, genus *morbillivirus* of family *Paramyxovirdae.*

- The virus is closely related to Rinderpest, PPR, Measles, and Phocin distemper viruses.
- The virus is extremely sensitive to heat, dryness and ultra violet rays. It does not persist in kennels after removal of infected dogs. In exudates it survives only for 20 minutes.
- The virus is destroyed by ether, formalin, phenol, and chloroform.
- The virus causes massive destruction of lymphocytes and immune suppression.

Epizootiology

Geographical distribution

It is a disease of worldwide distribution.

Susceptibility

Dogs, foxes, raccoon, ferret, wolves, mink and chunks are susceptible. Dogs of 3-6 months are highly susceptible. Exotic cats and pigs may be infected.

Sources of infection

The virus is present in respiratory and conjunctival exudates, saliva, feces, and urine for up to 60-90 days post infection.

Transmission

1. Aerosol infection is the common method.
2. In utero infection has been reported.

Pathogenesis

The virus is attached to the epithelium of the upper respiratory tract and replicates in macrophages then spreads to the tonsils. Cell associated viremia arises and the virus is spread to retro pharyngeal, bronchial and mesenteric lymph nodes, respiratory and intestinal mucosa, spleen, liver, eye, skin and CNS.

Incubation period is 2-9 days.

Clinical signs

The severity of clinical signs depends on virulence of the virus, age of the host, immune status as well as complications and super infection with other pathogens.

A. Systemic signs

1. Diphasic fever, the temperature rises for 1-3 days and then subsides for several days followed by second elevation for a week or more.
2. Anorexia and depression.
3. Coughing which is dry then becomes moist, sneezing and dyspnea
4. Nasal and ocular discharges.
5. Audible crackling sound and increase of bronchovesicular sound.
6. Keratoconjunctivitis and optic neuritis with blindness may occur.
7. Vomiting and diarrhea which contains mucous and blood.
8. Dehydration and emaciation.

B. Neurological signs

1. It occurs 1-3 weeks following systemic illness.
2. Hyperesthesia, cervical rigidity, circling, disturbances of gait and ataxia occur.
3. Chorea myoclonus or neuromuscular twitching or rhythmic jerking "localized twitching of group of muscles" occurs in the form of forceful simultaneous contractions in the legs and facial muscles. It occurs due to irritation of lower motor neurons of spinal cord. It is characteristic and specific for CDV infection.

C. Hyperkeratosis of nose and foot pads.

D. Pustular dermatitis of the lower abdomen is common in dogs with neurological symptoms.

- Young puppies which are infected transplacentally develop neurologic signs during the first month of life with permanent immune deficiency in survived puppies.
- Infection of puppies before eruption of permanent dentition may cause severe damage of their enamel.
- Super infection by infectious canine hepatitis or toxoplasmosis is common.
- The course of the disease is 10 days and sometimes is prolonged for several weeks.

Postmortem lesions

These include:

1. Catarrhal enteritis.
2. Pneumonia.

3. Hyperkeratosis of nose and foot pads.
4. Pustular dermatitis of lower abdomen.

Diagnosis

1. Epizootiological situation of the disease.
2. Clinical signs and lesions.
3. Laboratory diagnosis.

Samples

- Transtracheal or pharyngeal washing.
- Smears from conjunctiva, tonsils, spleen, lungs, respiratory and genital epithelium, urine, blood for buffy coat separation and blood serum.
- Foot pad biopsy.

a. Virus isolation on canine or ferret reticuloendothelial tissue cell culture.
b. Detection of the viral antigen on smears from buffy coat or tissues using FAT.
c. The use of PCR for detection of the virus.
d. Serological examination using VNT, ELISA, and immunoperoxidase.
e. Animal inoculation: Intracerebral inoculation in mice, ferrets and hamsters produces CNS signs.
f. Histopathology: Examination of sections from epithelial cells, neurons and conjunctival cells to detect intracytoplasmic and intranuclear inclusion bodies.
g. Hematology: There is marked leucopoenia.

Differential diagnosis

The disease should be differentiated from Infectious canine hepatitis, Toxoplasmosis, Leptospirosis and Rabies.

Treatment

1. Antibiotics to avoid secondary infection.
2. Antipyretics, antiemetics and antidiarrheal drugs.
3. Ringer's solution I.V.
4. Vit. B complex and ascorbic acid.
5. Dogs should be kept in clean warm place and oculonasal discharges should be removed.
6. Anticonvulsant may be used for neurological disturbances.

Control

1. Isolation of infected dogs and symptomatic treatment.
2. Cleaning and disinfection of kennels
3. Removal of all sources of infection.
4. Vaccination using modified live virus vaccines.
5. Puppies born to non-vaccinated bitches are vaccinated at 1-4 weeks of age and those born to vaccinated bitches are vaccinated at 6-16

weeks of age. Another dose is given after 2-4 weeks interval and vaccination is repeated every year (one-year protection).

Infectious Canine Hepatitis (ICH)

Canine Adenovirus Disease

It is an infectious disease of dogs characterized by fever, vomiting, diarrhea, leucopenia and prolonged bleeding time.

Etiology

The disease is caused by *Canine adenovirus-1* (CAV-1). It is antigenically related to CAV-2 which causes a respiratory disease in dogs.

- The virus survives for days in contaminated fomites. The virus is resistant to environmental inactivation and disinfectants as ether, acids, chloroform, and formalin but inactivated by iodine, phenol, and sodium hydroxide.
- The virus persists in renal tubules with shedding of the virus for 6-12 months after recovery.

Epizootiology

Geographical distribution:

It is a disease of worldwide distribution.

Susceptibility

Dogs and foxes are susceptible. Dogs under one year of age are more susceptible.

Source of infection

Saliva, feces, and urine of dogs during the acute stage of the disease contain the virus.

Urine of dogs contains the virus up to 6-12 months post recovery.

Transmission

1. Infection occurs through:
2. Ingestion of contaminated food.
3. Inhalation of urine aerosol.
4. Some ectoparasites may play a role in transmission of the disease.

Pathogenesis

Infection of dogs results in localization of the virus in tonsils and regional lymph nodes where primary replication occurs and the virus is released from infected cells causing viremia which lasts for a week and then the virus localizes in hepatic parenchymal cells, reticuloendothelial cells and vascular endothelial cells. This will result in acute hepatic necrosis, glomerular injuries, and corneal opacity. Hepatic regeneration

occurs in dogs surviving the acute phase of the disease. Chronic latent infection of the liver may also occur with chronic active hepatitis.
Incubation period is 4-10 days.

Clinical signs

Peracute form

In this form, sudden death may occur due to damage of vital organs as brain and lungs or due to shock.

Acute form

This form is characterized by:

1. Fever (1-6 days) which may be transient or biphasic.
2. Tonsillar enlargement and coughing.
3. Serous nasal and ocular discharges.
4. Abnormal chest sounds due to pharyngitis, laryngitis and pneumonia.
5. Vomiting, abdominal pain and diarrhea.
6. Cervical lymphadenopathy.
7. Subcutaneous edema of head, neck, and trunck.
8. Ascites also occurs.
9. Icterus may occur in dogs survive the acute phase.
10. Hemorrhage on mucous membranes of mouth and around teeth and sometimes into the aqueous chamber of the eye.
11. Epistaxis and bleeding form vein puncture sites.
12. CNS signs as disorientation and terminal coma.
13. Corneal edema and ulceration, blepharospasm, photophobia and uni or bilateral corneal opacity (blue eye).

- Death occurs due to hepatic insufficiency and hepatoencephalopathy.
- Course of the disease is 5-7 days and may be prolonged in complicated cases.

Postmortem lesions

1. Edema and hemorrhage of superficial and cervical lymph nodes.
2. Petechial and echymotic hemorrhages on all serosal surfaces.
3. Abdominal cavity contains clear to bright red fluid.
4. The liver is enlarged, dark and mottled in appearance.
5. Spleen is enlarged.
6. Focal hemorrhage in renal cortex.
7. Consolidation of the lungs.
8. Hemorrhage in midbrain and caudal brain stem.
9. Clouding of the aqueous humor.

Diagnosis

1. Epizootiological situation of the disease.
2. Clinical signs and lesions.
3. Laboratory diagnosis.

Samples: Swabs from oropharynx, tonsillar crypts, kidney, brain, blood and blood serum are collected.

a. Virus isolation on cell culture. The virus can be isolated only in the early stage post infection.
b. The use of PCR.
c. Detection of the viral antigen in blood and tissues.
d. Serological examination using CFT and ELISA.
e. Hematology: There is marked leucopenia
f. Histopathology: Detection of intranuclear inclusion bodies in hepatic and endothelial cells.

Differential diagnosis

The disease should be differentiated from Canine distemper, Leptospirosis and Toxoplasmosis.

Treatment

1. Antibiotic therapy for secondary infection as ampicillin or gentamicin.
2. Fluid therapy as Ringer's solution, 45 ml/kg body weight, I.V.
3. The use of fresh plasma or whole blood.
4. Application of atropine ophthalmic ointment to decrease ciliary spasm.
5. The use of non-absorbable oral antibiotics as neomycin to reduce ammonia-producing bacteria in intestine.
6. Oral potassium therapy and ascorbic acid.
7. Decrease of protein intake.

Control

1. Isolation of infected dogs, symptomatic treatment, and good nursing.
2. Cleaning and disinfection of kennels.
3. Vaccination using modified live vaccine or inactivated vaccine.
4. Dogs are vaccinated at 12-14 weeks of age with two doses with 3-4 weeks interval.

- Modified live vaccine may produce subclinical interstitial nephritis and shedding of the virus in urine.

Canine parvo-virus infection (CPV)

It Is a highly contagious and common cause of acute, infectious gastroenteritis in young dogs. It is characterized by enteritis and myocarditis.

Etiology

The disease is caused by a nonenveloped, single-stranded DNA *Canine parvovirus* of family *Parvoviridae.*

- The virus is resistant to environmental conditions as well as many common detergents and disinfectants. There is evidence that the virus can live in ground soil for up to a year
- The virus targets stem cells in bone marrow, lymphatics and intestinal crypts, myocardial cells and external granular layer of cerebellum.

Epizootiology

Geographical distribution

It is a worldwide disease reported in USA, Australia, and Japan

Susceptibility

- Dogs, foxes, wolves and coyotes are susceptible.
- Puppies of few days age are affected by acute or chronic mycocarditis. Dogs of more than six weeks age are affected with enteritis.
- Certain dog breeds, such as Rottweilers, Pit Bulls, Labrador Retrievers, Doberman Pinschers, German Shepherds, English Springer Spaniels and Alaskan sled dogs, are particularly susceptible to the disease.
- All isolates of canine parvovirus affect dogs, wolves, and foxes, but only some of them will infect cats.

Sources of infection

- Infected dogs act as source of infection.
- Infectious CPV can persist indoors at room temperature for at least 2 months outdoors, if protected from sunlight and desiccation; it can persist for many months and possibly years.
- Virus is shed in the feces of infected dogs within 4–5 days of exposure (often before clinical signs develop), throughout the period of illness, and for about 10 days after clinical recovery.

Transmission

The disease occurs through:

1. Ingestion and inhalation (oral fecal link)
2. Indirectly through contact with virus-contaminated fomites.
3. In- utero-infection

Pathogenesis

After exposure, the virus enters through M cells to get to Peyer's patches and replicates in variety of tissues including spleen, thymus and lungs.

The virus preferentially infects and destroys rapidly dividing cells of the small-intestinal crypt epithelium, lymphopoietic tissue, and bone marrow. Destruction of the intestinal crypt epithelium results in epithelial necrosis, villous atrophy, impaired absorptive capacity, and disrupted gut barrier function. Infection in utero or in pups of less than 8 weeks old or born to unvaccinated dams without naturally occurring antibodies can result in myocardial infection, necrosis, and myocarditis. Viremia occurs after 2-7 days post exposure. The virus localizes in small intestine, lung, spleen, thymus, and mesenteric lymph nodes. Inflammatory process and vasculitis occur. Panleukopenia also occurs because of virus-induced cytolysis of rapidly dividing stem cells in the bone marrow.

- Parvoviruses require a target cell–derived duplex transcription template, which is available only when cells divide during the S phase of the cell cycle. They must wait for target cells to enter the S phase of the cell cycle.
- Virus probably infects macrophages or dendritic cells migrating in the mucus layer and on the surface of mucosae.

Clinical signs

1. Cardiac form: This form occurs in cases of in-utero infection or infection in the early neonatal period. There are signs of cardiac arrhythmia, dyspnea, cough, pulmonary edema, and death.
2. Intestinal form: This form occurs in dogs of six weeks to nine months. It is manifested by:

a. Fever, depression, lethargy and anorexia
b. Diarrhea which may be bloody tinged.
c- Vomiting and dehydration.

- Some infected dogs show no clinical signs.

Postmortem lesions

1. Liquid bloody feces in the intestine.
2. Pulmonary edema.
3. Enlargement of mesenteric lymph nodes.

Diagnosis

1. Epizootiological situation of the disease.
2. Clinical signs and lesions.
3. Laboratory diagnosis.

Samples: blood, serum, lung, spleen and fecal swabs are collected.

a. Isolation of the virus on canine fetal lung cell culture.
b. Serological examination using VN or HI.
c. The use of latex agglutination test on feces.
d. The use of electron-microscope.
e. Histopathology of heart and intestine.

f. Radiography (enlargement of the heart and intestinal obstruction) and ECG.
g. Biochemical and urine analysis may reveal elevated liver enzymes, lymphopenia, and electrolyte imbalances.
h. Abdominal ultrasound may reveal enlarged lymph nodes in the groin, or throughout the body, and fluid-filled intestinal segments.

Differential diagnosis

This disease should be differentiated from other diseases causing diarrhea.

Treatment:

1. Antibiotics as enrofloxicin and gentamycin.
2. Anti-acids as Zentac.
3. Antiemetics
4. Plasma transfusion.
5. Intravenous fluid and nutrition therapy are essential in maintaining a dog's normal body fluid after severe diarrhea and dehydration.

Control

1. Isolation of infected dogs.
2. Disinfection using hypochlorite 1:10.
3. If you need to clean up a parvovirus-contaminated area, first pick up and safely dispose of all organic material (vomit, feces, etc.), and then thoroughly wash the area with a concentrated household bleach solution, one of the few disinfectants known to kill the virus.
4. If a dog has had parvovirus in a home, it is best not to have a puppy in that home for several years.
5. Vaccination of bitches prior to breeding using inactivated or attenuated vaccine. To prevent CPV infection, vaccination with a modified-live vaccine is recommended at 6–8, 10–12, and 14–16 weeks of age, followed by a booster administered 1 year later and then every 3 years.

Canine Infectious Tracheobronchitis

Kennel Cough Complex

It is a highly contagious disease of dogs characterized by paroxysmal coughing.

Etiology

1. *Canine Parainfluenza* virus (labile virus)
2. Other viruses may be included as canine distemper, CAV_1 and CAV_2).
3. *Bordetella bronchiseptica*
4- Mycoplasma Spp.

Epizootiology

Geographical distribution

The disease is widely distributed in the world.

Susceptibility

Dogs of all ages especially young dogs are more susceptible.

Sources of infection

Respiratory discharge of infected dogs acts as the main source of infection.

Transmission

Infection occurs by inhalation.

Pathogenesis

The virus multiplies in the epithelium of nasal mucosa, trachea, bronchi, bronchioles, and prebronchial lymph nodes. Damage of the tracheobroncheal mucosa facilitates the colonization of bacteria.

Incubation period is about ten days.

Clinical signs

A- Un-complicated form

It is characterized by:

1. Dry cough, which is frequently paroxysmal in type.
2. Rhinitis with serous to mucopurulent nasal discharge.
3. Conjunctivitis.
4. Spontaneous recovery within one to two weeks.

B- Complicated form

Chronic bacterial bronchopneumonia develops, manifested by fever, nasal and ocular discharge and dyspnea

Postmortem lesions

Inflammation of respiratory tract, consolidation of the lungs and enlargement of bronchial lymph nodes.

Diagnosis

1. Epizootiological situation of the disease.
2. Clinical signs and lesions.
3. Laboratory diagnosis.

Samples: Nasopharyngeal swaps, tracheal washing fluids, blood, and serum are collected.

a. Isolation of the virus.
b. Isolation of the causative bacteria.
c. Serological examination against para-influenza virus and *Bordetella bronchoseptica*.
d. Examination of tracheal wash fluid for detection of high number of neutrophils.

Differential diagnosis

The disease should be differentiated from other diseases causing respiratory manifestation

Treatment

a. Antimicrobial therapy

-Tetracycline 20mg/kg body weight, orally every eight hours for seven days.
-Cephalexin 30 mg/kg body weight, orally every 12 hours for seven days.
-Trimethoprim-sulphonamide 50 mg/kg body weight, every 12 hours for seven days.

b. Glucocorticoides: Prednisolone 0.25-0.50 mg/kg body weight, every 12 hours for seven days.

c. Bronchodilators: Aminophylline dihydrate, 11 mg/kg body weight, orally every 12 hours for seven days

d. Expectorants.

Control

1. Isolation of infected dogs.
2. Disinfection using sodium hypochlorite.
3. Vaccination.

Canine Herpes Virus Infection

It is an infectious fatal hemorrhagic neonatal disease of dogs characterized by rhinitis, vesicular vaginitis, and death.

Etiology

Canine herpes virus is the causative agent. It is primarily an opportunistic pathogen in the immunocompromised dogs.

Epizootiology

Geographical distribution

The disease is widely distributed all over the world.

Susceptibility

The disease affects young puppies of 1-3 weeks and pregnant bitch. It is rare in dogs older than four months.

Sources of infection

Infected dogs are the source of infection. The virus is secreted in oral, respiratory and vaginal secretions.

Transmission

The disease is transmitted by inhalation.

Pathogenesis

The virus multiplies in nasal epithelial cells and tonsils. The virus disseminates through macrophages and viremia occurs. The virus multiplies in different organs. Prenatal death, stillbirth or abortion may occur.

Clinical signs

The disease is manifested by:

1. Death of young puppies within 24 hours of illness.
2. Mild rhinitis, pharyngitis, and conjunctivitis in older dogs.
3. Vesicular vaginitis, stillbirth, infertility, or abortion may occur in bitches.

Postmortem lesions

These include Focal necrosis and hemorrhages in lungs, kidneys, liver and intestinal tract.

Diagnosis

1. Epizootiological situation of the disease.
2. Clinical signs and lesions.
3. Laboratory diagnosis.

   Samples: Respiratory and vaginal secretions, kidneys, liver, lymph nodes, blood and serum are collected.

   a. Isolation of the virus on cell culture.
   b. Serological examination as VNT or ELISA.
   c. Histopathology for detection of the intranuclear inclusion bodies.

Differential diagnosis

The disease should be differentiated from Canine Infectious Hepatitis, Canine distemper and Toxoplasmosis.

Prognosis of this disease is very poor.

Control

1. Isolation of infected pregnant bitch three weeks prior to parturition and isolation of infected puppies.

2. Disinfection of infected kennels.
3. Examination of bitches for vesicular vaginitis should be carried out before breeding.

Viral Diseases of Cats

Feline Leukemia

It is a chronic oncogenic virus infection of cats characterized by different syndromes including both proliferative and degenerative conditions

Etiology

The disease is caused by a single stranded RNA, *Oncorna virus,* family *Retroviridae*.

- The virus contains reverse transcriptase, an enzyme necessary for synthesis DNA copy of the viral RNA. This DNA incorporates into the DNA of the host's chromosomes of the infected cell.
- Outside the host, the virus survives only for several hours.
- The virus is immunosuppressive and causes destruction of lymphocytes.

Epizootiology

Geographical distribution

The disease is common in Australia and NewZeland.

Susceptibility

Cats of all ages especially of two to four years are susceptible

Sources of infection

Infected cats during the long incubation period secrete the virus in saliva and nasal secretions.

Milk and feces also contain the virus.

Carriers also secrete the virus.

Transmission

The disease occurs though:

1. Direct transmission from cat to cat by cat bite.
2. Congenital transmission.
3. Venereal transmission.

Pathogenesis

After oronasal infection the virus infects the lymphocytes in the local lymph nodes of the head and neck. The virus infects the circulatory lymphocytes and monocytes and viremia arises. The virus spreads to the cells of bone marrow where it replicates to high titers and enters the blood. The virus is profoundly immunosuppressive causing destruction of lymphocytes and

suppression of their functions. Production of immune complexes also occurs.

Incubation period ranges from months to years.

Clinical forms:

This disease is characterized by presences of several forms.

1. Abdominal form: Diarrhea, inappetance, loss of weight, vomiting and palpable abdominal masses.
2. Thymic form: Dyspnea, cough and hydrothorax.
3. Multicentric form: Enlargement of superficial lymph nodes, spleen, liver, kidneys, development of uremia in addition to the signs of immunosuppression.
4. Lymphoid leukemia: Increase of WBCs due to circulating malignant lymphoblasts and decrease of reticulocytes

Clinical signs

1. The disease is characterized by chronic wasting nature, anemia and lethargy
2. Secondary infections in the form of oral ulcers, gingivitis, diarrhea, uremia and ocular lesions (bilateral hyphaema).
3. Mortality rate reaches up to 100%.
4. Infertility in queens and abortion.

Postmortem lesions

These include Hydrothorax, enlargement of lymph nodes, spleen, liver and kidneys.

Diagnosis

1. Epizootiological situation.
2. Clinical signs and lesions.
3. Laboratory diagnosis.

Samples: Saliva, tears, pleural fluid, blood, and serum are collected.

a. Viral isolation
b. IFAT and ELISA for detection of the virus in leukocytes, saliva, and tears
c. Hematological findings: Increase number of WBCS, decrease in reticulocyte numbers and presence of immature lymphocytes in blood films.

Differential diagnosis

The disease should be differentiated from diseases causing immunosuppression of cats, such as feline immunodeficiency virus infection and feline panleukopenia.

Control

1. Elimination of positive or carrier cats after blood testing.
2. Proper disinfection of cat's premises.

3. Vaccination using inactivated or subunit vaccines. Cats at risk of exposure are vaccinated.

Feline Immunodeficiency Virus Infection

(Feline AIDS)

It is an infectious disease of cats characterized by the development of opportunistic infections as a consequence of an acquired immunodeficiency.

Etiology

The disease is caused by a single stranded RNA *Lentivirus* of the subfamily R*eteroviruses*.

- The virus contains reverse transcriptase enzyme which is necessary for synthesis of DNA copy of the viral RNA which incorporates into the DNA of the host's chromosomes of infected cells.
- The virus is T cell lymphotropic causes a loss of T cells and immunodeficiency.
- The virus infects many white blood cell types including T. lymphocytes, B. lymphocytes and macrophages. FIV eventually leads to debilitation of the immune system and exhaustion of T helper cells.

Epizootiology

Geographical distribution

The disease is reported in many countries, Japan, Germany, Switzerland and Holland.

Susceptibility

Cats and cheetahs are affected.

Sources of infection

Saliva of infected cats is believed to be the main source of infection.

Transmission

The virus is transmitted through deep bite wounds, congenital transmission is reported. The virus could be isolated from seminal plasma and seminal cells of experimentally infected cats.

Pathogenesis

The virus multiplies within macrophages causing lymphopenia and neutropenia. A syndrome of generalized lymphadenopathy persists for 2-9 months. Signs of opportunistic infections occur.

Clinical signs

The disease has a prolonged non-symptomatic period of latent infection followed by a period of clinical disease.

1. Latent phase: Non-symptomatic phase may range from months to years.
2. Acute phase: fever, enlargement of lymph nodes associated with bacterial infections of skin and GIT.
3. Chronic phase: Different clinical signs due to secondary infections as stomatitis, gingivitis, dermatitis, eye affections, diarrhea, and neurological disorders.

Diagnosis

1. Epizootiological situation of the disease.
2. Clinical signs and lesions.
3. Laboratory diagnosis.

Samples: blood, serum and specimens of infected organs are collected.

a. Virus isolation on cell cultures.
b. Serological examination using IFA, ELISA and Western blot.
c. Hematological findings: lymphopenia and neutropenia.

Differential diagnosis

The disease should be differentiated from other diseases causing immunosuppression in cats as feline leukemia and feline panleukopenia

Control

1. Isolation of infected cats and symptomatic treatment.
2. A vaccine to prevent the disease is available and recommended for use in high risk cats.

Feline Panleukopenia

Feline infectious enteritis, Feline parvoviral enteritis, feline distemper, or cat plague

It is a highly contagious often fatal, viral disease of cats characterized by fever, vomiting, diarrhea, dehydration, marked leucopenia and death.

The name panleukopenia comes from the low white blood cell count (leucocytes) exhibited by affected animals.

Etiology

The disease is caused by a single stranded non-enveloped DNA, *Parvo virus* of the family *Parvoviridae.*

- The virus is extremely resistant to many chemicals including alcohol, ether and chloroform. It survives for longer than year in room temperature in organic material.
- Virus particles are abundant in all secretions and excretions during the

acute phase of illness and can be shed in the feces of survivors for as long as 6 weeks after recovery.

- The virus is partially deficient and requires actively dividing cells to complete its replication cycle.

Epizootiology

Geographical distribution

It is a disease of worldwide distribution.

Susceptibility

All members of the family Felidae; cats, raccoons, mink and ferrets are susceptible.

It is predominantly a disease of young kittens of 2-4 months old.

Sources of infection

Diseased cats shed the virus in all body secretions and excretions and up to six weeks after recovery.

Transmission

Infection occurs by direct contact between susceptible and infected cats. Flies may play role in transmission during warm periods.

Pathogenesis

The virus enters through mouth and replication occurs in the lymphoid tissues of oropharynx. Within 24 hours, the cat becomes viremic. The virus invades the rapidly multiplying cells as lymphoid cells, bone marrow and epithelial cells of ileum and jejunum causing destruction of these cells. The virus primarily attacks the lining of the gastrointestinal tract, causing internal ulceration and, ultimately, total sloughing of the intestinal epithelium. This results in profuse and usually bloody diarrhea, severe dehydration, anemia, and often death. The virus also invades the uterus of pregnant cat and infects the fetus. In addition, it crosses the blood brain barrier and infects the cerebellum.

Depletion of lymphoid tissues and progressive drop in circulating white cells because of bone marrow destruction and endotoxin absorption from the bowel are characteristic.

Incubation period ranges between four to ten days.

Clinical signs

1. Fever, depression and anorexia.
2. Vomiting usually develops 1–2 days after the onset of fever; it is typically bilious (yellow) and unrelated to eating.
3. Severe fetid diarrhea, that sometimes is bloody, after two to four days of the onset of fever.
4. Dehydration.
5. Oral ulceration, purulent otitis and eye lesions as retinal dehydration.

6. Affected cats may sit for hours at their water bowl, although they may not drink much.
7. Terminally the temperature is subnormal followed by coma and death.

Postmortem findings

The intestinal tract is dilated, hyperemic with petechial hemorrhage and contains bloody fetid feces.

Diagnosis

1. Epizootiological situation of the disease.
2. Clinical signs and lesions.
3. Laboratory diagnosis.

Samples: Urine, feces, lymphoid tissues, blood and serum are collected.

a. Virus isolation.
b. Serological examination using VNT, CFT and FAT.
c. Histopathology: Eosinophilic intranuclear inclusions in lymph nodes and intestinal epithelium.
d. Hematology: There is marked leukopenia

Differential diagnosis includes salmonellosis and infections with feline leukemia virus (FeLV) and feline immunodeficiency virus.

Treatment

1. Fluid therapy as ringer's solution 44 ml/kg body weight/day intravenous. B. vitamins should be added to the infusion, together with 5% glucose if hypoglycemia is suspected.
2. Plasma or blood transfusion 10-20 ml blood/kg body weight /day.
3. I.V. broad spectrum antibiotics to prevent septicemia. Nephrotoxic drugs (eg, gentamicin, amikacin) should be avoided until dehydration has been corrected.
4. Anti-emetics eg, metoclopramide
5. GIT protectants.
6. Injections of vitamins: A, B, and C.
7. Recombinant feline interferon omega.

About 90–95% of affected kittens that are two months or less of age, die regardless of treatment.

Control

1. Isolation of infected cats.
2. Proper disposal of infected material and disinfection.
3. FPV can be destroyed by exposure to a 1:32 dilution of household bleach (6% aqueous sodium hypochlorite), 4% formaldehyde, and 1% glutaraldehyde for 10 minutes at room temperature.
4. Inactivated and MLV vaccines that provide solid, long-lasting immunity are available. Kittens at 8-10weeks should be vaccinated and

repeated after 2-4 weeks. Live vaccines should not be given to cats that are pregnant, immunosuppressed, or sick.

Feline infectious peritonitis (FIP)

It is an infectious disease of cats characterized by vasculitis, fibrinous peritonitis or pleuritis and formation of disseminated pyogranulomas.

It is an immunological mediated disease. It is considered as the most feared disease in breeding catteries because there is neither effective treatment nor preventive vaccine.

Etiology

The disease is caused by a single stranded RNA virus; a member of *coronaviridae*.

- The virus is antigenically similar to the feline *enteric corona virus*.
- It is relatively unstable outside the host.
- The virus affects the wall of blood vessels causing vasculitis.

Epizootiology

Geographical distribution

It is a disease of worldwide distribution

Susceptibility

Cats and wild Felidae are susceptible.

Sources of infection

Diseased cats, infected cats in incubation period and healthy sero-positive cats are sources of infection.

Transmission

It occurs through ingestion or inhalation due to direct close contact with infected cats or their excreta.

Pathogenesis

The virus replicates in the epithelial cells of the pharynx and lungs. The virus invades the macrophages and infected macrophages in turn transport the virus throughout the body. Hematogenous dissemination of the virus results in viremia. The virus localizes within the cells in the wall of small blood vessels causing vasculitis with escape of fibrin rich serum, which accumulates in the body cavities.

Incubation period ranges between weeks, months, or years.

Clinical signs

The disease usually occurs in sporadic form.

1. Infected animals show fever that persists until the last few hours of life.
2. Weight losses and lethargy.

There are two main forms:

A. Effusive (wet) form: This form is characterized by peritonitis, pleuritis or both.

1. Accumulation of fibrin rich fluid within the peritoneal cavity and chest cavity.
2. Enlargement of the abdomen is a common sign.
3. Respiratory distress and dyspnea (pleuritic effusion).
4. At later stage, icterus, vomiting, diarrhea and constipation may occur.
    - The virus and viral proteins contribute along with antibody and complement to Arthus-like reaction. This form is believed to occur in cats that develop humoral but not cellular immunity.

B. Non effusive (dry) from: It is characterized by:

1. Fluctuating temperature and weight loss.
2. The lesions are of granulomatous appearance including lymph nodes, kidneys, lungs, and brain.
3. Persistent cough may occur due to pyogranulomatous pneumonia.
4. Renal or hepatic insufficiency may occur.
5. Unilateral or bilateral retinitis and hyphaema.
    - This form is believed to occur in cats that develop partial cellular immunity.

Postmortem lesions

1. The serosal surfaces of abdomen and thorax are covered with diffuse or multi-focal fibrinous exudate (omentum, mesentery, pleura and lungs).
2. Yellow fluid containing fibrin flakes may be present in the peritoneal and pleural cavities.
3. Pyogranulomatous lesions in the viscera.

Diagnosis

1. Epizootiological situation of the disease.
2. Clinical signs and lesions.
3. Laboratory diagnosis.

Samples: Abdominal and thoracic fluid, biopsy from internal organs, blood and serum are collected.

a. Virus isolation.
b. Serological examination using IFAT and ELISA but there is cross reaction with feline enteritis corona virus.
c. Histopathological examination (biopsy).
d. Detection of inclusion bodies within circulating neutrophils.
e. Radiography and ultra-sonography.

Control

1. Isolation of suspected cases.
2. Destruction of all high antibody reactive animals.
3. There is no effective treatment (fatal disease).
4. Vaccination using MLV vaccine gives only 40% protection.

Feline Respiratory Disease Complex

Feline Rhinotracheitis

It is an infectious disease of cats characterized by rhinitis, conjunctivitis, and oral ulceration

Etiology

Multiple pathogens including Feline calici-virus, *Feline herpes virus*, *Chlamydia psittaci*, Mycoplasama spp. and *Bordatella bronchisceptica* are incriminated.

Epizootiology

Geographical distribution

It is a worldwide disease.

Susceptibility

Young kitten and aged cats are severely affected. The disease is usually associated with stress.

Sources of infection

These include infected cats and contaminated fomites.

Transmission

Transmission of the infection occurs by aerosol infection through droplets transmitted through ocular and oropharyngeal discharges.

Pathogenesis

Generally viruses cause interference with pulmonary clearance mechanism, viremia or bacteremia occurs with localization in oral, respiratory and conjunctival mucous membranes. *Calicivirus* localizes in the epithelium of the oral cavity and tissues of the lungs.

Incubation period varies according to the etiology.

Clinical signs

Calicivirus infection

1. Fever, depression, anorexia, and salivation.
2. Ocular and nasal discharge, coughing and sneezing.
3. Ulceration is preceded by vesicles on the tongue, hard palate, and nostrils are regular features.
4. Some strains cause serous rhinitis, pulmonary edema, and interstitial pneumonia.
   Many recovered cats remain carriers of the virus, shedding infectious organisms from the pharynx.

Herpes virus infection

***Feline herpes virus* is a highly contagious virus that is one of the major causes of upper respiratory infections.**

Typical signs include conjunctivitis, ocular discharge, sneezing, nasal discharge, salivation, pharyngitis, lethargy, inappetence, fever, coughing, and sometimes keratitis

***Chlamydia psittaci* infection**

It is associated with fever, conjunctivitis, and lacrimation.

Mycoplasma infection is characterized by severe edema of the conjunctiva.

Bordatella bronchisceptica is a highly communicable bacteria more often found in dogs but also responsible for respiratory issues in cats. Symptoms include sneezing, nasal discharge, eye discharge, and possibly fever. Coughing is a more common symptom in dogs than cats. Treatment with antibiotics is usually affective.

Diagnosis

1. Epizootiological situation of the disease.
2. Clinical signs and lesions.
3. Laboratory diagnosis.

Samples: Nasal or tracheal wash, nasal and conjunctival swabs, lachrymal discharges, blood and serum are collected.

   a. Isolation of the causative viruses, chlamydia or mycoplasma spp.
   b. Examination of Giemsa stained films of exudates for detection of the elementary bodies of Chlamydia in cytoplasm of mononuclear cells of conjunctiva.

Treatment

1. Removal of nasal and ocular discharges.
2. The use of vasoconstrictor nasal drops.
3. Application of antiseptic solution for oral lesions.
4. The use of broad-spectrum antibiotics taking in consideration Chlamydia or Mycoplasma infection.
5. The use of antihistaminics.
6. Application of ophthalmic antibiotic ointment.

7. Fluid therapy may be helpful in some cases.

Control

1. Isolation of infected cats.
2. Proper disinfection.
3. Avoidance of stress factors.
4. Vaccination:

- MLV against calicivirus and herpes viruses is used. The first dose is at 9 weeks of age and the second dose 3 weeks thereafter then annually. They can be used parentrally or intranasally.