Disease of Wild Canines I

- Rickets is a disease of the bony growth plate and thus only affects young, growing animals.
- The most common causes are dietary insufficiencies of phosphorus or vitamin D.
- Calcium deficiencies can also cause rickets, and while this rarely occurs naturally, poorly balanced diets deficient in calcium have been said to cause the disease.
- As in most diets causing *osteodystrophies*, the abnormal calcium:phosphorus ratio is most likely the cause.

• The characteristic lesions of rickets are failure of both vascular invasion and mineralization in the area of provisional calcification of the physis.

- This pathology is most obvious in the metaphyses of the long bones.
- Clinical signs:
- bone pain
- stiff gait
- swelling in the area of the metaphyses
- difficulty in rising
- bowed limbs
- pathologic fractures.

- On radiographic examination, the width of the physes is increased, the nonmineralized physeal area is distorted, and the bone may show decreased radiopacity.
- In advanced cases, angular limb deformity can be seen due to asynchronous bone growth.

- Typical microscopic lesions associated with rickets are impaired endochondral ossification, which are most prominent in fast-growing bones.
- Growth plates are widened and irregular, and joints appear enlarged.
- Trabecula of the spongiosa are thinner, predisposing to infarctions and hemorrhage.

- Canine adenovirus-1 (CAV-1), also known as infectious canine hepatitis virus (ICHV), has caused mortality in domestic canids and wild mammals in the families Canidae, Mustelidae, and Ursidae.
- CAV-1 also causes disease in wolves, coyotes, and bears, and encephalitis in foxes

- The <u>virus</u> is spread in the feces, urine, blood, saliva, and nasal discharge of *infected* dogs.
- The virus enters via the nasopharyngeal, oral, and conjunctival routes; initial infection occurs in tonsils, and then is spread to regional lymph nodes and to the blood.
- Viremia results in dissemination to saliva, urine, feces, and infection of endothelial and parenchymal cells in many tissues, leading to necrosis, especially in the liver, kidneys, spleen, and lungs.

- Symptoms:
- Fever, depression, loss of appetite, coughing, and a tender abdomen.
- <u>Corneal edema</u> and signs of liver disease, such as <u>jaundice</u>, vomiting, and <u>hepatic</u> <u>encephalopathy</u>, may also occur. Severe cases will develop <u>bleeding</u> disorders, which can cause <u>hematomas</u> to form in the mouth.
- Death can occur secondary to this or the liver disease. However, most dogs recover after a brief illness, although chronic corneal edema and kidney lesions may persist

- The lesions present in dogs with infectious canine hepatitis depend on the clinical course of infection.
- A rapid clinical course results in edema and hemorrhage of superficial lymph nodes, with multifocal to diffuse petechial and ecchymotic hemorrhages on serosal surfaces.
- The liver and spleen are enlarged, with mottling of the splenic parenchyma, and accumulation of fibrin on the serosal surfaces of the abdominal viscera.
- The wall of the gallbladder is characteristically thickened and edematous.
- Gross lesions in other organs may include cortical renal hemorrhages and multiple areas of pulmonary consolidation.
- Ocular lesions may include diffuse corneal edema and opacity.

- Histologic hepatic findings in acutely infected puppies include multifocal hepatocellular necrosis, and sometimes centrilobular hepatic necrosis as a consequence of disseminated intravascular coagulation.
- Intranuclear inclusions may be present within <u>Kupffer's cells and hepatocytes.</u>
- These characteristic inclusions also occur in <u>endothelial cells within the kidney</u> of affected dogs.
- There is typically widespread hemorrhage and necrosis associated with intravascular thrombosis in dogs that develop disseminated intravascular coagulation.

• In foxes, canine adenovirus 1 causes primarily central nervous system disease; infected animals may exhibit intermittent convulsions during the course of their illness and, terminally, may suffer paralysis of one or more limbs.

• Canine distemper (sometimes termed hardpad disease) is a <u>viral disease</u> that affects a wide variety of animal families, including domestic and wild species of dogs, coyotes, foxes, pandas, wolves, ferrets, skunks, raccoons, and large cats, as well as pinnipeds, some primates, and a variety of other species.

- Animals in the family <u>Felidae</u>, including many species of large cat as well as domestic cats, were long believed to be resistant to canine distemper, until some researchers reported the prevalence of CDV infection in large felids.
- Both large Felidae and domestic cats are now known to be capable of infection, usually through close housing with dogs or possibly blood transfusion from infected cats, but such infections appear to be self-limiting and largely without symptoms.

- In canines, distemper affects several body systems, including the gastrointestinal and respiratory tracts and the spinal cord and brain, with common symptoms that include
 - high fever,
 - eye inflammation and eye/nose discharge,
 - labored breathing and coughing,
 - vomiting and diarrhea,
 - loss of appetite and <u>lethargy</u>, and
 - hardening of nose and footpads.
- The viral infection can be accompanied by secondary bacterial infections and can present eventual serious neurological symptoms.

- Canine distemper virus tends to orient its infection towards the <u>lymphoid</u>, <u>epithelial</u>, and <u>nervous tissues</u>.
- The virus initially replicates in the lymphatic tissue of the respiratory tract. The virus then enters the blood stream and infects the <u>respiratory</u>, <u>gastrointestinal</u>, <u>urogenital</u>, epithelial, and <u>central nervous systems</u>, and <u>optic nerves</u>.
- The typical pathologic features of canine distemper include lymphoid depletion (causing <u>immunosuppression</u> and leading to secondary infections), interstitial <u>pneumonia</u>, <u>encephalitis</u> with <u>demyelination</u>, and <u>hyperkeratosis</u> of the nose and foot pads.

• The spread of CDV infection was from dogs to non-dog hosts and vice-versa, it should not be forgotten that wild carnivores could be a source of distemper infection for domestic carnivores.

- Rabies is an acute, viral infection of the central nervous system caused by a Rhabdovirus.
- Rabies virus is a bullet shaped enveloped virion belongs to Lyssavirus genus and Rhabdoviridae family.
- All mammals, including humans, can become infected with rabies. Once clinical signs appear, rabies is nearly 100% fatal. Rabies does not infect birds or reptiles
- Rabies is widespread in NY and raccoons, skunks, bats, foxes, and coyotes make up the vast majority of cases in the United States.

- Most common way of entry of rabies virus into the body is either through saliva or infected neural tissue via bite wounds or open cuts in the skin or mucous membrane and not through the intact skin.
- Non-bite exposure methods are inhalation of aerosolized rabies virus, cornea/organ transplants and contamination of abrasions, with infectious material such as brain tissue from a rabid animal

- Pathogenesis of rabies :
- Incubation period or eclipse phase is highly variable from 2 weeks to 6 years (average: 2 to 3 months) according to the *concentration of the virus*, *inoculation site* and density of innervation.
- Bites on the head, face, neck and hands with bleeding offer the greatest risk and are generally associated with shorter incubation period due to decreased length and greater number of neurons.
- Virus travels by rapid retrograde fast axonal transport at a rate of 12- 100 mm per day towards the CNS

• Clinical signs may vary and are not typical. Many diseases can have similar signs, including <u>distemper</u>. Nonspecific signs may include restlessness, anorexia, difficulty swallowing, vomiting, or diarrhea.

• Dumb form

- Animals with the dumb form of rabies may:
- lose their fear of humans
- become depressed and retreat to isolated places
- become partially paralyzed (watch for abnormal facial expression, drooling, drooping head, sagging jaw, strange sounds, or paralysis in the hind limbs that spreads to the rest of the body)
 - Furious form
- Animals with the furious form of rabies may:
- be extremely excited and aggressive
- gnaw at and bite their own limbs
- attack other objects or animals
- be alternately agitated and depressed
- Wild animals with rabies usually have rough, dirty fur and may be very thin and feeble.

- Specific **gross lesions** are not present at autopsy, but selfinflicted wounds and foreign bodies in the stomach of a carnivore should raise suspicion.
- The **histologic lesions** of rabies, when present, are typical of **nonsuppurative encephalomyelitis**, with **ganglioneuritis** and **sialadenitis**.
 - Perivascular cuffing and focal gliosis
 - Neuronal degeneration
 - Inclusion bodies of Negri.

- *Rabies diagnosis is done by direct fluorescent antibody (FAT) in a specialized laboratory. A specific section of brain tissue is required to make a definitive diagnosis.
- *There is no successful medical treatment for clinical rabies infection in humans, however prompt vaccination is highly effective at preventing the disease.
- ❖ If bitten by a suspect animal, flush the wound with soap and water. Contact the local health department!!!!!
- *Rabies can be controlled by <u>vaccination of domestic animals</u> and quarantine of domestic animals that may be exposed to rabies.