University of Mosul
Lecture No.:
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Date:
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Lecture title: Canine Diseases

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Summary:

1. Rabies

Synonym: Hydrophobia

Definition:

Rabies is a deadly, zoonotic and neurological disease of mammals that is almost fatal once the clinical signs develop. Humans are usually infected when they are bitten by an infected animal, or exposed to its saliva or central nervous system (CNS) tissues.

Etiology

Rabies results from infection by the rabies virus, a neurotropic virus in the genus *Lyssavirus*, family Rhabdoviridae.

Pathogenesis of rabies

After inoculation into the subcutaneous tissues and muscle, rabies virus replicates locally within muscle cells and then attaches to peripheral nerve endings. Local replication around the bite site can continue for months before the virus enters peripheral sensory and motor nerve endings.
Once the virus is within the CNS, there is massive viral replication, with cell-to-cell transmission of the virus across synaptic junctions. The spinal cord, medulla oblongata, gray matter, and cerebellum are particularly affected. The virus also moves outwards from the CNS in somatic and autonomic nerves and is deposited in a variety of tissues, including cardiac and skeletal muscle, the eye, the kidney, pancreas, nerves around hair follicles, and the salivary glands. Production of new virions through budding from the plasma membranes occurs primarily within the salivary glands, which results in the shedding of virus that can be transmitted to other hosts. Virus is shed by some dogs for up to 13 days before the onset of clinical signs.
The clinical presentations of rabies virus infection have been divided into excitatory (“furious”) and paralytic (“dumb”) forms.
Excitatory (Furious) form characterized by hyperactivity, aggression, highly irritable and restless. Hydrophobia (fear of water) and aerophobia (fear of air drafts) are common. Muscle spasms and difficulty swallowing due to throat paralysis. Excessive salivation due to impaired swallowing. In later stages, seizures, coma, and death occur.
Paralytic (Dumb) form is less common than the furious form. It characterized by progressive muscle weakness leading to paralysis. No signs of aggression, making it harder to diagnose. Eventually leads to respiratory failure and death.

GROSS PATHOLOGY

- There are no pathognomic gross findings.
- Externally, there may be fresh or healed bite wounds, and sometimes gross trauma due to self-mutilation.
- In the CNS there may be congestion of meningeal vessels, the brain tissue may appear pinker than usual an there may be mild cerebral edema.

HISTOPATHOLOGY

- Histopathological changes do not reflect the severity of the clinical disease.
- The general CNS findings are those of viral encephalitis, including Perivascular cuffing, vascular congestion, neuronophagia, neuronal degeneration and focal to diffuse gliosis. Lesions may be most severe in the brain.
- The presence of Negri bodies is considered pathognomic for rabies, but these are only seen in about 50 - 75% of cases. These are found most commonly in ganglionic cells of the hippocampus and in Purkinje cells of the cerebellum.

Diagnosis:

1- The direct fluorescent antibody test (dFA) is the test most frequently used to diagnose rabies.
2- Electron microscopy (EM).
3- Histologic examination and immunohistochemistry (IHC).
4- PCR, and isolation in cell culture

2. <u>Infectious Canine Hepatitis</u>

Infectious canine hepatitis (ICH) is a worldwide, contagious disease of dogs with signs that vary from a slight fever and congestion of the mucous membranes to severe depression, marked leukopenia, and coagulation disorders. It also is seen in foxes, wolves and bears.

Etiology and Pathogenesis:

ICH is caused by a DNA virus, canine adenovirus 1 (CAV-1).

Ingestion of urine, feces, or saliva of infected dogs is the main route of infection. Recovered dogs shed virus in their urine for ≥6 months. Initial infection occurs in the Peyer patches, followed by viremia and disseminated infection. Vascular endothelial cells are the primary target, with hepatic and renal parenchyma, spleen, and lungs becoming infected as well. Chronic kidney lesions and corneal clouding (“blue eye”) result from immune-complex reactions after recovery from acute or subclinical disease.

Clinical Findings:

Signs vary from a slight fever to death. The mortality rate ranges from 10%–30% and is typically highest in very young dogs. The incubation period is 4–9 days. The first sign is a fever of >40°C, which lasts 1–6 days. If the fever is of short duration, leukopenia may be the only other sign, but if it persists for >1 day, acute illness develops.

Signs are depression, anorexia, thirst, conjunctivitis, serous discharge from the eyes and nose, and occasionally abdominal pain and vomiting. Intense hyperemia or petechiae of the oral mucosa, as well as enlarged tonsils, may be seen. There may be subcutaneous edema of the head, neck, and trunk. It may be difficult to control hemorrhage, which is manifest by bleeding.

GROSS PATHOLOGY

Endothelial damage results in "paint-brush" hemorrhages on the gastric serosa, lymph nodes, thymus, pancreas, and subcutaneous tissues. Hepatic cell necrosis produces a variegated color change in the liver, which may be normal in size or swollen. Grayish white foci may be seen in the kidney cortex.

HISTOPATHOLOGY

There is necrosis, with neutrophils and macrophages infiltration, and hepatocellular intranuclear inclusions. The gallbladder wall is typically edematous and thickened; edema of the thymus may be found.

Diagnosis:

1- Usually, the onset of illness and bleeding suggest ICH, although clinical evidence is not always sufficient to differentiate ICH from distemper.
2- ELISA, serologic, and PCR testing.
3- Postmortem gross changes in the liver and gallbladder are more conclusive and characteristic intranuclear inclusion bodies in the liver.
4- Diagnosis is confirmed by virus isolation and immunofluorescence,