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COMMON CAUSES OF DEATH IN DOGS

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Canine herpesviral infection

Canine herpesvirus 1 causes abortion, stillbirths, and infertility in adults and a fatal systemic viremia in neonates. Affected puppies are often less than a week-old. Neonates become infected either in utero or through exposure to infected maternal secretions.

Infected neonates exhibit ocular and nasal discharge, dyspnea, and sudden death. Commonly, the whole litter of puppies is affected. The clinical signs and postmortem changes are very characteristic. Sporadically, immunologically suppressed puppies of up to 3-4 weeks of age die of CHV1 infection.

At necropsy, there is multifocal hemorrhage in all visceral organs including the lung, liver, and kidney. The petechiae and ecchymoses in the kidneys are commonly referred as “turkey egg kidney.” This lesion is considered pathognomonic for canine herpesviral infection.

Laboratory tests

Histopathology

PCR – lung, liver, kidney

“Canine parvoviral infection” should be in bold and needs to move to up-front like Canine herpesviral infection above.

Canine parovirus-2 is responsible for the commonly observed canine parvoviral enteritis. There is canine parvovirus-1 (aka. Minute virus of canines); CPV-1 is relatively nonpathogenic and only very occasionally causes myocarditis, gastroenteritis, and interstitial pneumonia in very young puppies.

The classical clinical presentations include depression, anorexia, vomiting, diarrhea, and intestinal bleeding. Some dogs can have mild or even subclinical disease. Generally, GI symptoms are first recognized within 24h to 48h of infection and blood is frequently absent in the early stages of diarrhea. Progressive intestinal crypt necrosis is responsible for intestinal hemorrhage. Secondary bacterial complication leads to high fever and septic shock.

Puppies that are infected in utero or before 8 weeks of age can develop necrotizing myocarditis.

At necropsy, segmentally dilated, flaccid small intestine containing red to red-brown fluid is characteristic. Mesenteric lymph nodes are commonly enlarged. The infected young puppies might have red and white stricks in the myocardium. The main differential diagnosis for hemorrhagic enteritis in a puppy is hookworm (*Ancylostoma caninum*) infection.

Laboratory tests

Histopathology and immunohistochemistry

PCR – Small intestine, mesenteric lymph node, spleen (pooled)

Fecal floatation to rule out hookworm diseases

Canine Distemper

Canine distemper virus is a morbillivirus that is antigenically related to the human measles virus, rinderpest virus in horses, and “peste de petit ruminants” virus in sheep. Distemper virus invades the upper respiratory mucosa, proliferates in lymphoid tissues, and becomes viremic. Distemper virus infects various types of cells including epithelial cells, lymphocytes, and neurons.

Extensive vaccination has significantly diminished the incidence of distemper virus but outbreaks among unvaccinated dogs and sporadic cases among vaccinated dogs still occur. Initial clinical signs are ocular and nasal discharge, coughing, dyspnea, vomiting, and diarrhea. Neurological signs develop 1 to 3 weeks after dogs start to recover from the systemic illness.

At necropsy, the lungs fail to collapse and are edematous, multifocally mottled red, and slightly firm. Occasionally in chronic cases, hyperkeratosis of foot pads (hard pad disease) can be seen. The differential diagnoses for the bronchointerstitial pneumonia include canine adenovirus-2 virus, canine parainfluenza virus, and *Bordetella bronchiseptica*. In dogs with CNS signs caused by distemper, rabies should be ruled out as a differential diagnosis.

Laboratory tests

Histopathology and immunohistochemistry

PCR test – lung, spleen, LN, and brain (pooled)

Hepatic lipidosis of neonatal canines

Hepatic lipidosis in neonatal canines is not a distinct disease entity like feline hepatic lipidosis. The hepatic lipidosis in young puppies (approximately 1-2 months of age) is a gross morphological diagnosis in emaciated puppies that often have severe diarrhea caused by *E. coli*, coccidiosis, coronavirus, or parvoviral enteritis. The negative energy balance due to diarrhea results in massive mobilization of internal fat stores which must be processed by the liver.

At necropsy, the most striking feature is severe fatty change within the liver. Thin sections of liver may float in formalin fixative. The stomach, small intestine, and large intestine are often empty, gas-filled, may contain a small quantity of watery ingesta.

Laboratory tests

Histopathology and immunohistochemistry (coronavirus and parvovirus)

Bacteriology – Small intestine

PCR test – gastrointestinal system

Fecal floatation

Cardiomyopathies

Dilated (congestive) cardiomyopathy (DCM): DCM is characterized by poor myocardial contractility. Most cases are idiopathic. As is the case with felines, genetic factors are thought to play a role in dogs. Affected dogs are commonly middle-aged, large-breed, males, including Doberman pinchers, Scottish deerhounds, Cocker spaniels, Boxers, Great Danes, etc. A juvenile form has reported in Doberman pinchers and Portuguese water dogs. Clinical signs of weakness, lethargy, tachypnea, exercise intolerance, cough, anorexia, ascites, and syncope, may develop rapidly, and there is an increased risk of sudden death.

At necropsy, the heart is enlarged with severe biventricular dilatation (particularly left-sided) and endocardial fibrosis. Pulmonary edema and ascites can be seen.

Arrhythmogenic right ventricular cardiomyopathy: ARVC is a variant of DCM. It is a familial, primary myocardial disease in humans and in Boxers. Sudden heart failure and death are common.

At necropsy, the heart exhibits right ventricular dilatation. Microscopic findings are very distinct and are characterized by myocardial loss with replacement and infiltration by adipose or fibrofatty tissue.

Hypertrophic cardiomyopathy: HCM is common in cats and infrequent in dogs. The cause is unknown and a genetic basis is suspected. Myocardial hypertrophy causes stiffness of the ventricular wall and diastolic dysfunction. Affected dogs are young to middle-aged, large-breed, male dogs. Sudden death or death during anesthesia is common.

At necropsy, the ventricular walls (particularly the left) are markedly thick with a narrow ventricular lumen.

Cardiac measurement reference ranges (%)

$$TC/BW = 0.58 - 0.94$$

$$RV/BW = 0.10 - 0.18$$

$$(LV+S)/TC = 52.21 - 66.25$$

$$(LV+S)/RV = 2.76 - 3.88$$

$$RV/TC = 15.22 - 20.94$$

* Compendium on continuing education for the practicing veterinarian, Vol 3 No 11, p 421-426, 1983

Gastric dilatation and volvulus (GDV)

The exact cause of GDV is uncertain but it may involve abnormal gastric motility. Dogs with a deeper thorax relative to width, familial history of the disease, once daily meals, eating from an elevated platform, and

repeated episodes of gastric dilation (causing gastrohepatic ligament relaxation) are at increased risk. GDV principally occurs in large breed dogs but small dogs and cats can be rarely affected.

At necropsy, the stomach is markedly distended with gas and generally rotated 180, 270, or 360 degrees clockwise. The gastric mucosa and wall are red to violet from congestion and cyanosis. Concurrent splenic displacement and congestion occurs.

Hemangiosarcoma

The most common neoplasm that causes sudden death in dogs is hemangiosarcoma. Hemangiosarcomas occur frequently in the spleen and right atrium of the heart. Visceral hemangiosarcomas are locally invasive and may metastasize to other organs. The prognosis is grave.

At necropsy, dogs with splenic hemangiosarcoma often have hemoabdomen and a large, tumor-associated hematoma in the spleen. Exsanguination and DIC are the cause of death in these cases. In the case of cardiac hemangiosarcoma, rupture of tumor-associated hematomas can produce hemopericardium, which results in cardiac tamponade.

Laboratory test

Histopathology

Heartworm disease

Various species of mosquitos in the world act as vectors for heartworm (*Dirofilaria immitis*) disease. Heartworm disease is an important cause of pulmonary hypertension in dogs. The adult worms lodge in the pulmonary arteries, inciting verminous arteritis that, along with the occlusive affects of the worms themselves, results in pulmonary hypertension. Most affected dogs are 4-8 years old; however, there is no specific age or breed predilection. Many dogs are asymptomatic but symptomatic dogs often have histories of exertional dyspnea, fatigue, syncope, cough, and weight loss. Sudden death can occur.

At necropsy, the main gross finding is an enlarged heart with a rounded apex, marked right ventricular dilation, and large numbers of adult heartworms in the pulmonary arteries. The liver is enlarged, heavy, and has accentuated lobular pattern (due to chronic passive congestion). In some cases with severe arteritis (sometimes 5 to 30 days after adulticide therapy), the lungs are multifocally mottled in dark-red, wet, and firm, resulting from arterial thromboembolism. Cross-sections of lungs contain large to medium-sized pulmonary arteries that are occluded by dead adult heartworms. In very severe cases of heartworm diseases (caval syndrome), large numbers of adult worms lodge in pulmonary artery, right heart, and vena cava, obstructing venous flow and resulting in severe passive congestion in the liver. Ascites secondary to hepatic congestion are common. The liver often exhibits an accentuated lobular pattern (nutmeg liver). Large numbers of adult heartworms in the vessels cause mechanical hemolysis, which results in dark-red kidneys and red urine in the urinary bladder.

Laboratory test

Histopathology

Renal failure

Acute renal failure: The kidneys are very susceptible to hypoxic conditions and toxic materials. Acute renal failure is often related to acute severe hemolysis or toxicosis (aminoglycosides, NSAIDs, ethylene glycol, cisplatin, lead, etc.). Dogs with acute renal failure usually present with a sudden onset medical emergency such as collapse, vomiting, and possibly even coma. These animals are often oliguric or anuric.

At necropsy, kidneys are swollen, wet and heavy and the renal capsule is opaque and thick due to edema. Perirenal edema is sometimes severe. The urinary bladder is frequently empty.

Laboratory tests

- Histopathology
- Toxicology

Chronic renal failure: Chronic inflammation of any component (glomerulus, tubule, interstitium) of the kidney eventually leads to chronic renal failure. Therefore, the exact etiology of chronic renal failure can be difficult to determine. The typical clinical presentation is characterized by polydipsia-polyuria, weight loss, poor body condition, nonregenerative anemia, and an ammonia-like odor on the animal's breath.

At necropsy, the kidneys are small and irregular. Typical uremia-associated lesions can include ulcerative glossitis and stomatitis, gastric ulcers, mineralization of the stomach wall, lung, kidney, or intercostal muscles, and parathyroid hyperplasia.

Laboratory tests

- Histopathology
- PCR tests - leptospirosis

Toxicosis

Anticoagulant rodenticides: Affected dogs exhibit multiple hemorrhages in various organs, intestinal hemorrhage, subcutaneous hemorrhage, epistaxis, hemothorax, and hemoabdomen.

Ethylene glycol (antifreeze): Ethylene glycol toxicosis has three clinical phases. The first phase (0.5 to 12 hr) is basically alcohol intoxication, which results in depression and incoordination. The second phase (12 to 24 hr) is cardiopulmonary due to metabolic acidosis, which leads to pulmonary edema, tachypnea, and tachycardia. The final phase (24 to 72 hr) is acute renal failure. The kidneys are swollen and edematous, the urinary bladder is empty, and sometimes perirenal edema is evident. Often, observation of calcium oxalate crystals on histopathology is enough to make a definitive diagnosis.

Chocolate: Chocolate toxicity is caused by theobromine (an alkaloid in cocoa) and caffeine. In general, the darker the chocolate is, the greater the theobromine content in the chocolate. The potential lethal dose of milk chocolate is less than 2oz (about 60g)/kg. Baking chocolate contains about 10 times more theobromine than milk chocolate. Therefore, ingestion of less than 0.2oz (about 6g)/kg baking chocolate is potentially lethal in dogs.