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Unveiling Heart Failure in Animals: Insights from Necropsy Diagnosis and Clinical Manifestations

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Introduction

Heart failure (HF) manifests as a clinical syndrome marked by symptoms like shortness of breath, fatigue, swelling, and reduced exercise capacity. These symptoms arise from the heart's inability to adequately compensate for its pumping function, a condition brought about by structural and/or functional abnormalities within the heart. Necropsy diagnosis plays a pivotal role in understanding the impact of heart failure on animals. It provides a comprehensive postmortem examination, shedding light on histopathological changes, cardiac defects, and associated complications, facilitating a deeper comprehension of cardiac diseases in diverse species.

Acute Heart Failure

Acute heart failure is the sudden loss of effective cardiac contraction, potentially leading to death within minutes. Triggers include anoxia, drug exposure, myocardial necrosis, shock, cardiac tamponade, and sudden blockage of the pulmonary artery and aorta. It causes lesions involving pulmonary and systemic congestion, with acute dilation primarily affecting the right ventricle. The left ventricle, resistant to dilation, exhibits a thick wall and an outward curvature from the atrioventricular level to the apex. Understanding these aspects highlights the critical nature of acute heart failure and its life-threatening structural changes.

Systolic Heart Failure

Systolic heart failure, also known as forward failure. This will result in reduced cardiac output, compromising blood pumping into the aorta or pulmonary artery, impacting arterial pressure maintenance. This leads to symptoms like lethargy, syncope, and hypotension. Various mechanisms contribute to systolic heart failure, including dilated cardiomyopathy, infectious myocarditis, doxorubicin toxicity, and myocardial infarcts. Volume and pressure overload, stemming from conditions like valvular diseases and hypertension, further contribute to this

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complex condition. Understanding these mechanisms is vital for identifying and managing the underlying causes of systolic heart failure.

Diastolic Heart Failure

Diastolic heart failure, also known as backward failure, involves the damming back of blood in the venous system, leading to congestive heart failure. This condition poses challenges, manifesting in complications like ascites, pleural effusion, and pulmonary edema. Specific mechanisms, including impaired ventricular relaxation and abnormal chamber properties, contribute to diastolic heart failure. Factors such as ventricular hypertrophy, stenosis, heartworm disease, and pericardial abnormalities play a role. Obstructions at veins, atria, and atrioventricular valves add complexity. Recognizing these multifaceted mechanisms is crucial for a comprehensive approach to diagnosis and management.

Congestive Heart Failure

Congestive heart failure appears in various forms – right-sided, left-sided, or bilateral – with cardiac dilation or hypertrophy. Right-sided failure results in systemic congestion signs like ascites and peripheral edema. Left-sided failure exhibits pulmonary congestion signs such as pulmonary edema and dyspnea. In small animals, pleural effusion is linked to bilateral failure. Recognizing these patterns is crucial for accurate diagnosis and management.

Cardiac Defects in Dogs

Common cardiac defects in dogs include Pulmonic Stenosis (PS), Subaortic Stenosis (SAS), Patent Ductus Arteriosus (PDA), and Ventricular Septal Defect (VSD). PS is the most prevalent, followed by SAS and PDA. VSD ranks fourth with a lower occurrence.

Concentric hypertrophy

Concentric hypertrophy, a response to pressure overload, involves compensatory cardiac chamber dilation to overcome resistance, sustaining stroke volume but increasing myocardial oxygen consumption. Over time, this reliance can lead to myocardial failure, with ischemia causing fibrosis and reduced diastolic filling. Eccentric hypertrophy, a response to volume overload, entails mild wall thickness increase and substantial left ventricular enlargement. This adaptation allows efficient handling of increased blood volume while maintaining functional equilibrium.

Left sided heart failure

Left-sided heart failure can occur from diverse causes affecting cardiac functionality. Different causes include hypertension, aortic valvular disease, mitral valvular disease, congenital heart disease, myocarditis, myocardial degeneration, adhesive pericarditis. Clinically, dyspnea and shortness of breath are prominent signs. The development of lung lesions in left-sided heart failure is linked to the left ventricle's inability to manage venous return efficiently from lungs.

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This results in elevated pulmonary venous pressure, leading to pulmonary congestion and edema. Within the pulmonary structures, heart failure cells and fibrotic changes occur, culminating in brown induration of the lungs, indicative of chronic congestion. In summary, left-sided heart failure initiates a cascade of events in the lungs, causing increased pressures, congestion, edema, and fibrotic changes, ultimately affecting pulmonary function.

Right-sided heart failure

Right-sided heart failure primarily arises as a consequence of left-sided heart failure (LHF) and mitral stenosis. The causes of pure right-sided heart failure encompass a range of cardiac conditions. Myocarditis, inflammation of the heart muscle, and myocardial infarction with subsequent degeneration contribute to the onset of right-sided heart failure. Constrictive pericarditis and hydropericardium also cause the right sided heart failure. Additionally, endocarditis affecting the tricuspid valve, manifesting as incompetence and stenosis, plays a role in the development of right-sided heart failure. These all will lead to damming back of blood in both systemic and portal venous circulation leads to anoxia. These all will lead to edema. In specific species, such as horses and ox, edema is observed subcutaneously, while in dogs, it presents as ascites and in cats it is present as hydrothorax. In summary, right-sided heart failure stems from various causes, impacting both cardiac and renal functions, and culminating in the manifestation of edema with species-specific variations.

Cor-pulmonale, characterized by the enlargement or failure of the right ventricle that results from lung and pulmonary vessel diseases. In Right sided heart failure, liver is characterized by marked congestion, enlargement, and a nutmeg pattern. Histopathological changes in liver include degeneration, and centrilobular. Sinusoidal dilation contributes to hepatic alterations, giving rise to a yellow appearance in peripheral hepatocytes and contributing to the development of cardiac cirrhosis. In the kidneys, RHF causes congestion and hypoxia, leading to fluid retention and peripheral edema due to disrupted circulation and oxygen supply. The spleen also enlarges and histopathologically characterized by sinusoidal dilation. The distended sinusoidal walls undergo fibrous thickening, contributing to a state known as congestive splenomegaly.

Diagnostic Markers in Cardiac Myocardial Damage

Cardiac myocardial damage is detected through increased serum enzyme activity, such as creatine kinase (CK), lactate dehydrogenase (LDH), troponin T (TnT), troponin I (TnI), and aspartate amino-transferase (AST). Released selectively from injured cardiac muscle cells, these enzymes serve as reliable markers for detecting myocardial damage. Increased plasma concentrations of natriuretic peptides, including A-type (atrial) ANP and B-type (brain) BNP, further indicate cardiac disease presence. These hormones are synthesized and released in greater

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quantities during cardiac dysfunction episodes. The combined assessment of specific enzymes and natriuretic peptides offers valuable diagnostic insights into the occurrence and extent of myocardial damage in cardiac pathology.

Cardiac troponin I (cTnI) is a crucial biomarker for detecting myocardial fibrosis in dogs with chronic cardiac diseases, particularly in cases of Myxomatous Mitral Valve Disease (MMVD). Its concentration reflects pathological changes associated with MMVD, a condition marked by mitral valve degeneration leading to mitral regurgitation, chronic volume overload, left atrial dilation, and left ventricular eccentric hypertrophy, resulting in congestive heart failure. Most of the cTnI structurally bound within myocytes, is released into circulation after cell injury.

Creatine kinase, an enzyme found in the heart as well as other muscles, and plasma activities may increase with cardiac disease. The SGOT elevation is observed specifically in cases of actual myocardial infarction. Serum cardiac isoenzyme creatine kinase MB measurements prove to be a valuable additional diagnostic tool for autopsy diagnosis of ischemic heart diseases (IHDs). Given the limitations of histopathological findings, the establishment of diagnostic utility for various biochemical cardiac markers in biological fluids becomes crucial for postmortem diagnosis of myocardial infarction (MI).

In the context of congestive heart failure, the appearance of C-reactive protein is significant, but caution must be kept to rule out other conditions associated with its presence, such as active myocarditis, myocardial infarction, bacterial endocarditis, and pericarditis. Systemic infections, systemic embolism, and urinary tract infections are common accompaniments of congestive heart failure, also potentially contributing to the appearance of C-reactive protein.

Considering the compilation of various findings, including owner-provided history (clinical signs, medications), macroscopic and microscopic lesions, and different markers related to cardiac failure, the diagnosis of heart failure at the time of necropsy becomes a comprehensive process.

Conclusion

The exploration of heart failure (HF) and its various manifestations in animals revealed a complex clinical syndrome marked by distinctive symptoms. In essence, the culmination of these findings, along with owner-provided history and macroscopic and microscopic lesions, enriches the diagnostic process at necropsy. The comprehensive approach presented here not only enhances our understanding of cardiac pathology in animals but also provides a roadmap for improved diagnosis and tailored management strategies.

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