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CONTENTS

Pathological findings in congestive heart failure in dog: A case report

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ABSTRACT: This report describes a case of six-month-old male Labrador retriever presented for post-mortem examination at the Department of Veterinary Pathology, College of Veterinary and Animal Sciences, Pantnagar with a general appearance of overweight and oozing of blood-tinged fluid from nostrils. The owner reported that the dog was playing with his fellow dogs in the previous evening and died suddenly half an hour after the play after showing increased lethargy and occasional fainting episodes. Post-mortem examination revealed pale and irregular heart with dilated right ventricle. Tracheal mucosa was clear but there was sero-sanguinous frothy exudate in the tracheal lumen. All lobes of lungs were congested and dark red to purple colored. Sero-sanguinous frothy fluid was oozing from the cut surface of the lungs. Liver was enlarged and with dark reddish discoloration. Areas of necrosis were present on the surface of liver. Histopathological examination of liver revealed severe sinusoidal congestion throughout the parenchyma, a typical manifestation of the gross lesion, "Nutmeg liver". There was centrilobular necrosis of hepatocytes with replacement by fibrous connective tissue typically known as Cardiac Cirrhosis. Cardiac muscle fibres showed thinning and fragmentation. Lungs showed severe congestion of inter-alveolar wall capillaries, atelactasis, giant alveoli formation, interstitial fibrosis, and bronchiolar epithelial loss. This case highlights the progressive nature of congestive heart failure in dogs, emphasizing the need for early detection, comprehensive diagnostic evaluation, and long-term medical management.

Key words: Congestive heart failure, Dog

Dilated cardiomyopathy (DCM) and myxomatous mitral valve disease (MMVD) are the leading causes of congestive heart failure (CHF) in dogs, a progressive and life-threatening condition affecting cardiac function (Bagardi *et al*., 2022). While MMVD is prevalent in small breeds and leads to mitral regurgitation with left-sided heart remodeling, DCM primarily affects large breeds, resulting in ventricular dilation and biventricular failure leading to development of chronic general congestion and subsequently, congestive heart failure (CHF) and finally death. In CHF, the heart is unable to pump adequate supply of blood for the metabolic needs of the body. Clinical signs of CHF are due to backward failure (congestion and edema) and forward failure (reduced CO). Clinically, CHF manifests with dyspnea, tachycardia, arrhythmias, syncope, pleural effusions, ascites, and exercise intolerance (Silva *et al*., 2009-10). This communication describes a case of CHF in a Labrador dog.

Case

Six months old, white coloured, male, Labrador dog was presented to the Department of Veterinary Pathology, College of Veterinary and Animal Sciences, Pantnagar on 8-3-2025 for post mortem examination with a general appearance of heavily overweight according to the reported age by the owner (Fig.1) and oozing of sero-sanguinous fluid from nostrils (Fig. 2). The owner reported that the dog was playing with its fellow dogs in the previous evening and died suddenly approximately 30 minutes after the play, following lethargy and occasional fainting episodes. Owner also reported that the dog had access to *ad libitum* food and feed supplements like multivitamin throughout the day with no structured diet schedule maintained.

Post-mortem examination revealed lesions on heart, lung and liver. Heart appeared pale, irregular with dilated right ventricle (Fig. 3). Tracheal mucosa was clear but there was presence of sero-sanguinous frothy exudate in the tracheal lumen (Fig. 4). All lung lobes on both sides were congested and dark red to purple in colour. There was oozing of the sero-sanguinous frothy fluid from the cut surface of the lungs. Liver was enlarged and both the surfaces of all the lobes were dark reddish in

discoloration. There were focal necrotic areas on the liver surface (Fig. 5).

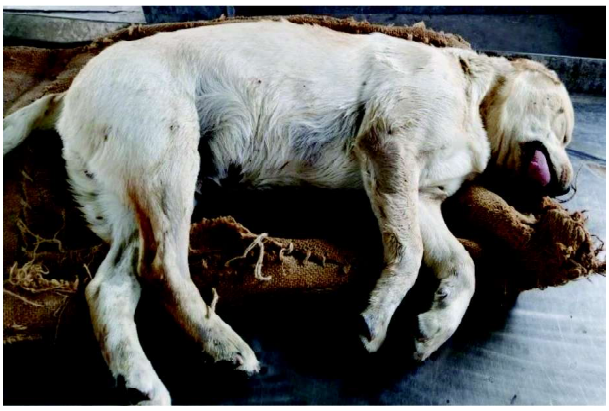

Fig. 1: Overweight dog

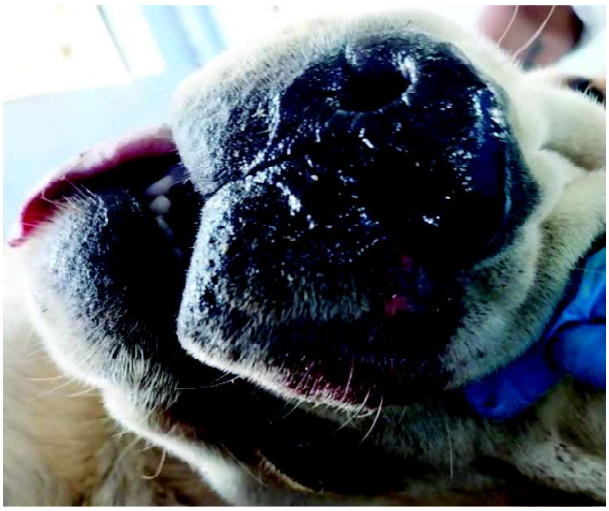

Fig. 2: Oozing of sero-sanguinous fluid from nostrils

Histopathological examination of the liver exhibited severe congestion of the large blood vessels throughout the parenchyma. There was severe congestion of the sinusoids in the hepatic lobules, throughout the parenchyma giving the liver a typical histopathological manifestation of "Nutmeg liver appearance". There was necrosis of hepatocytes around the blood vessels i.e. centrilobular necrosis with replacement of dead hepatocytes by the fibrous connective tissue, typically known as Cardiac Cirrhosis (Fig. 6). Lungs showed severe congestion of large blood vessels as well of interalveolar wall capillaries throughout the parenchyma leading to thickening of the interalveolar wall (Fig. 7). There was collapse of alveolar walls throughout the parenchyma (Fig. 7) except at few places where there was formation of giant alveoli indicating alveolar emphysema (Fig. 8). There was severe fibrous connective tissue proliferation around many of the blood vessels throughout the parenchyma (Fig. 9). Loss of epithelium throughout the circumference of the bronchioles was seen in many bronchioles. Heart revealed thinning of cardiac muscle fibre bundles along with fragmentation of muscle fibre bundles at places. The nuclei in the cardiac muscle bundles were pyknotic and pushed towards the periphery in many of cardiac myocytes. It was flattened in many of the cardiac myocytes indicating coagulative necrosis in cardiac myocytes (Fig. 10).

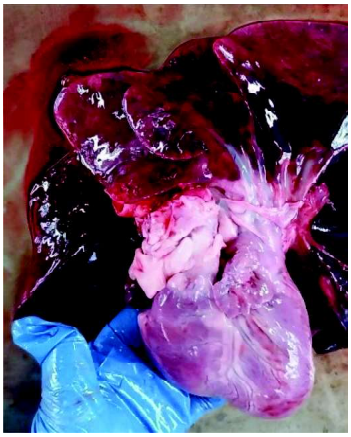

Fig. 3: Pale heart with dilated right ventricle

Fig. 4: Presence of sero-sanguinous frothy exudate in the tracheal lumen

Fig. 5: Enlarged liver with dark reddish discoloration

DISCUSSION

In the present case, overweight and exercise intolerance was there which is in confirmation with the findings of Silva *et al.,* 2009-10. Dilated cardiomyopathy in dogs is the most common form of cardiomyopathy representing an end stage of myocardial failure (Fuentes and Swift, 1998) which eventually affects the liver, lungs and other body systems. The DCM is a progressive disorder characterized by reduction of systolic myocardial contractibility leading to a drop in cardiac output and subsequent development of CHF and death (Silva *et al.,* 2009-10). The development of DCM in the present case might be due to disturbed body weight to heart weight ratio leading to more work load on heart, subsequently leading to development of DCM and CHF. Grossly, there was dilation of right ventricle of heart, severe dark discoloration of liver and dark purplish discoloration of lungs. These findings are in corroboration with that of Silva *et al.,* 2009-10 and Jadhav *et al.*, 2024 in Labrador dog and Sidhu *et al.*, 2022 in crossbred cattle. Histologically, liver showed congestion of large blood vessels along with congestion of sinusoids which is a typical manifestation of the gross lesion "Nutmeg liver". Due to cellular hypoxia, the hepatocytes surrounding the central veins are the first

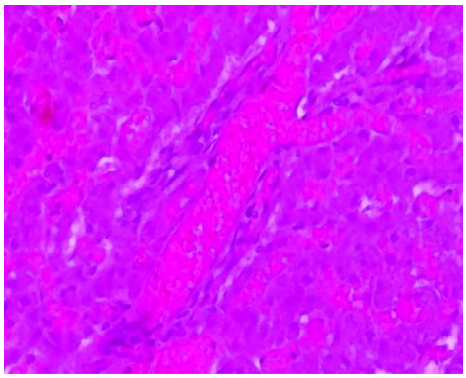

Fig. 6: Liver showing severe sinusoidal congestion, centrilobular necrosis and replacement by the fibrous connective tissue (H&E, ×400)

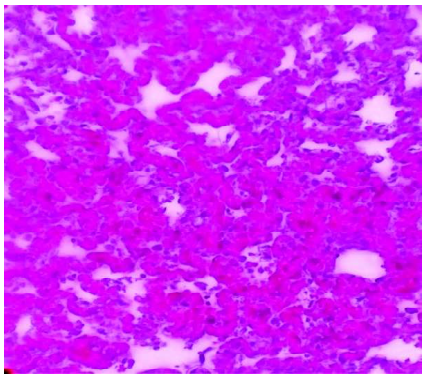

Fig. 7: Lungs showing severe congestion of interalveolar wall capillaries and collapse of alveolar walls. (H&E, ×100)

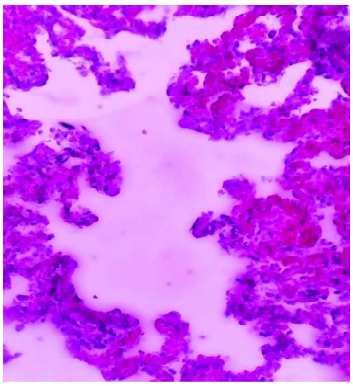

Fig.8: Lung depicting emphysema with formation of giant alveoli. (H&E, ×400)

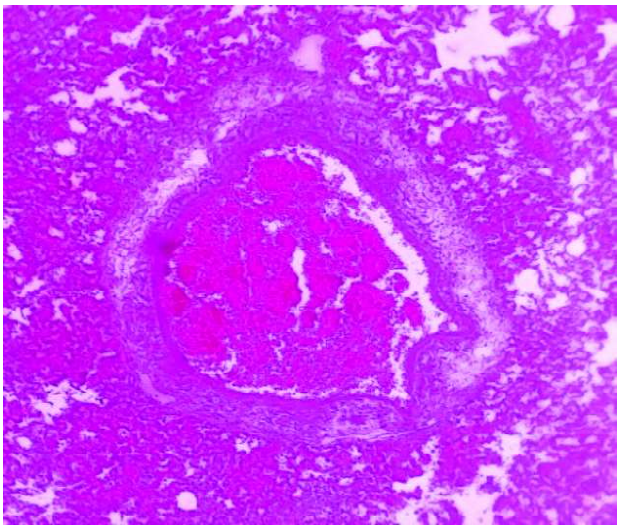

Fig. 9: Lung showing severe fibrous connective tissue proliferation around the blood vessel. (H&E, ×100)

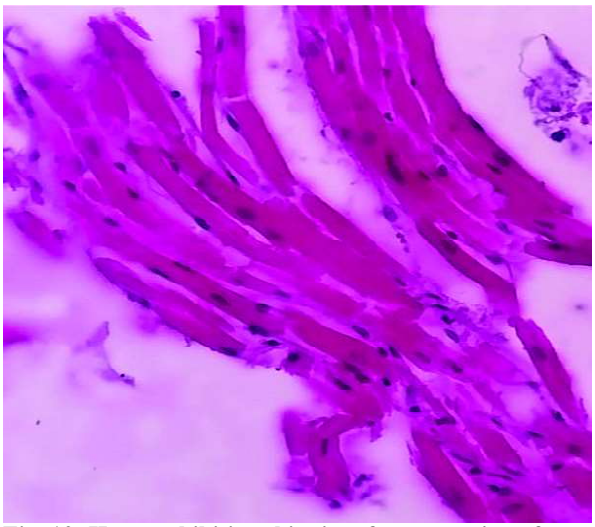

Fig. 10: Heart exhibiting thinning, fragmentation of cardiac muscle, pyknotic nuclei and peripheral location of nuclei. (H&E, ×400)

to die and thereby get replaced by the fibrous connective tissue, typically known as cardiac cirrhosis. These findings in liver are in corroboration with Jadhav *et al.*, 2024 in Labrador dog and Jones *et al.*, 2006. Lungs showed severe congestion of large blood vessels, interalveolar wall capillaries, thickening of the interalveolar wall, collapse of alveolar walls and severe fibrous connective tissue proliferation around blood vessels i.e. induration of lungs. Similar lesions have been reported by Jadhav *et al.*, 2024 and Jones *et al.*, 2006. Absence of heart failure cells in the present study might be due to delayed involvement of left side of the heart and consequently, the lungs, leading insufficient time for the development of heart failure cells, as also evident by the right DCM in the present case. Heart revealed thinning of cardiac muscle fibre bundles, fragmentation of muscle fibre bundles, and coagulative necrosis in cardiac myocytes. Similar lesions in heart have reported by Jadhav *et al.*, 2024 and Jones *et al.*, 2006.

The present case demonstrates severe congestive heart failure characterized by right ventricular dilation, pulmonary congestion and chronic hepatic congestion. The associated myocardial degeneration and necrosis indicate significant cardiac dysfunction leading to systemic venous congestion and sudden death. Early diagnosis and management of cardiac disorders in predisposed breeds are essential to prevent fatal outcomes.

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