

ACUTE MYOCARDIAL INFARCTION IN A LABRADOR DOG: A NECROPSY-BASED STUDY

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SUMMARY

A carcass of a Labrador male dog was presented for necropsy examination at the postmortem hall of the Department of Veterinary Pathology, LUVAS, Hisar. Dog was obese and had a history of sudden respiratory distress and seizures. Detailed necropsy of dog was conducted and tissue samples were collected for histopathological examination. Necropsy examination revealed voluminous, heavy and dark red lungs with petechial hemorrhages on surface. The ventricles of heart were enlarged and dilated indicating cardiomegaly. Coronary blood vessels were congested and myocardium showed grayish elongated foci at the base of coronary blood vessels. Foci of ecchymotic hemorrhages were observed at right atrioventricular junction of endocardium. Histopathology revealed severe congestion, haemorrhages with marked hemosiderosis and presence of heart failure cells in lung. Microsection of heart showed wavy appearance of myocardial muscle fibers, necrosis of myocytes with loss of striations and congestion of blood vessels indicating acute myocardial infarction.

Keywords: Acute myocardial infarction, Congestive heart failure, histopathology, Labrador, Necropsy

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Myocardial infarction (MI) is a pathologic diagnosis characterized by the development of acute myocardial ischemia leading to myocardial injury or necrosis (Thygesen *et al.*, 2007). Myocardial infarction is the end result of either acute or chronic myocardial ischemia. Myocardial ischemia differs slightly from myocardial hypoxia in that ischemia results in a stasis of waste products of cellular metabolism in addition to a lack of oxygen delivery, leading to cellular damage above and beyond that from hypoxemia (Meier and Oyama 2007). Heart failure is the state of any heart disease in which, despite adequate ventricular filling, the heart's output is decreased. Heart is unable to pump blood at a rate adequate for satisfying the requirements of the tissues with function parameters remaining within normal limits (Denolin *et al.*, 1983). Acute myocardial infarction (AMI) is the most important form of the ischemic heart disease in humans, it is rare in dogs and cats. It is associated with number of common diseases and may contribute to morbidity and mortality in those dog and cats that are critically ill (Driehuys *et al.*, 1998).

Few case reports on necropsy-based investigations on acute myocardial infarction were found in mixed breeds of dogs and cats (Driehuys *et al.*, 1998). However, there are no reports on necropsy based pathomorphological investigations on acute myocardial infarction leading to congestive heart failure along with other vital organs in Labrador dog. Therefore, the present report describes the necropsy based pathological alterations in case of acute myocardial infarction which leads to congestive heart failure in Labrador breed of dog.

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A carcass of a Labrador male dog, died with history of sudden respiratory distress and seizures was presented for necropsy examination at postmortem hall of Department of Veterinary Pathology, LUVAS, Hisar. Post mortem was performed within 24 hrs and samples from affected organs were collected. Tissues were fixed in 10% neutral buffered formalin, tissues were processed using standard method (Luna 1986) and tissue sections of 3-4 micron thickness were made. Sections were stained with, with haematoxylin and eosin (Luna, 1986). At necropsy body condition of carcass was obese and healthy (Fig. 1A). Lungs were voluminous, heavy and dark red with petechial hemorrhages on surface. (Fig. 1B). Cut surface showed oozing of dark red coloured blood. Coronary blood vessels of heart were congested and myocardium showed foci of grey discoloration at the base of coronary blood vessels. (Fig. 1C). Endocardium revealed multifocal or ecchymotic hemorrhages at right atrioventricular junction of endocardium. (Fig. 1D). Spleen was blue, double in size than the normal with 2-3 raised flabby areas around 3-4 cm in diameter on the surface. (Fig. 1E). Cut surface of spleen showed oozing of dark red coloured blood. Liver was dark red and firm in consistency (Fig. 1F). Cut surface showed extensive oozing of dark red coloured blood. Brain showed diffuse dark red discolouration of meningeal blood vessels indicating marked congestion and swollen appearance of gyri and sulci. (Fig. 1G). Cut surface of kidney showed whitish necrotic streaks on the cortical area and congestion in corticomedullary junction (Fig. 1H).

Microscopic examination of heart showed presence of thrombus in blood vessel, severe congestion and

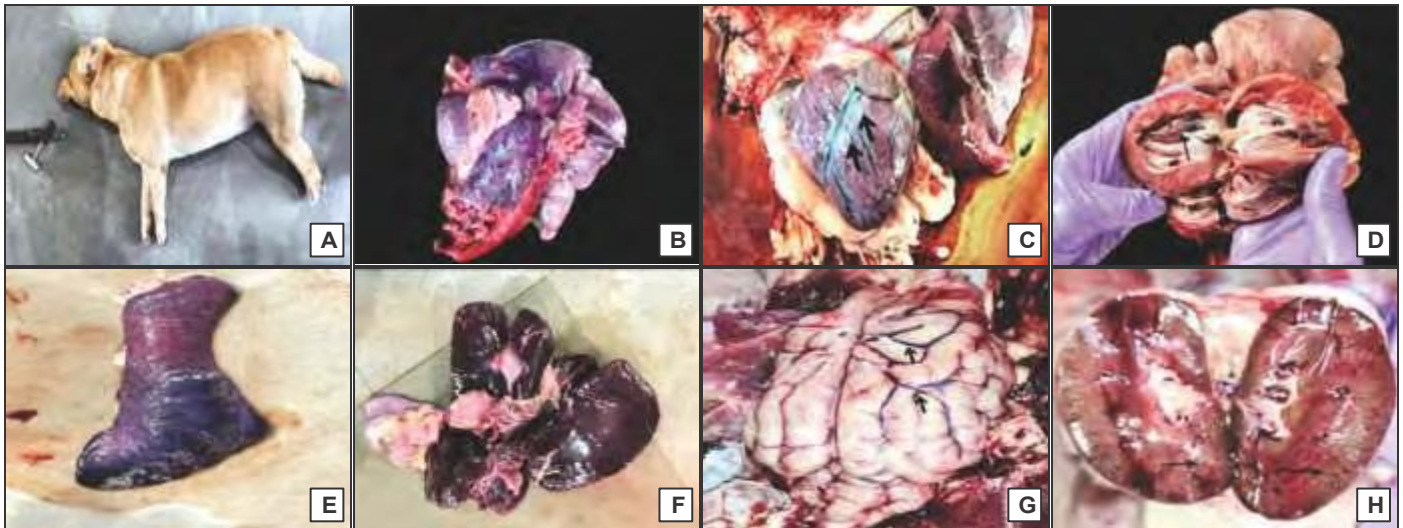


Fig. 1. Gross lesions in heart and other visceral organs in Labrador dog **A**: obese and healthy Labrador dog; **B**: Voluminous and heavy, dark red lungs with petechial hemorrhages on surface; **C**: Congestion of coronary blood vessels of heart with epicardial infarction at the base of coronary blood vessels; **D**: Endocardial ecchymotic hemorrhages at right atrioventricular junction **E**: Splenomegaly with blue discoloration and raised flabby areas on the surface; **F**: Liver showing dark red colour and firm consistency; **G**: Brain showing congestion of meningeal blood vessels and swollen appearance of gyri and sulci; **H**: Cut surface of kidney showing white streaks on the cortical area and congestion in corticomedullary junction.

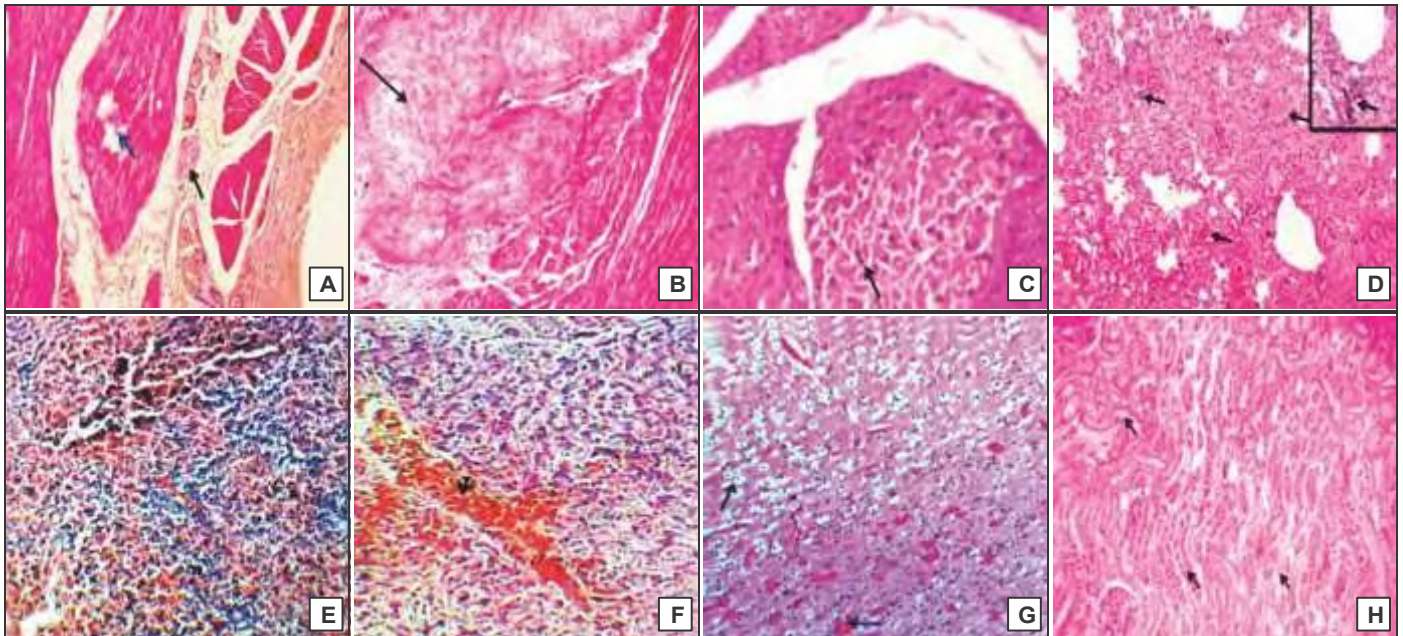


Fig. 2. Microscopic lesions in heart and other visceral organs in Labrador dog **A**: Heart showing presence of thrombus in blood vessel (arrow), severe congestion and necrosis of muscle fibers in myocardium (blue arrow) 100 x H & E Stain; **B**: Heart showing infarct i.e. area of coagulative necrosis in myocardium (arrow), 100 H & E Stain; **C**: Heart showing pyknotic nuclei and dark eosinophilic cytoplasm of muscle fibers (arrow) indicating necrosis 200 x H & E Stain; **D**: Lung showing severe congestion (arrow), hemorrhages and heart failure cells (thick arrow) along with presence of edematous fluid in alveoli; inset: heart failure cells in peribronchiolar region x 100 H & E Stain; **E**: Spleen showing diffuse hemorrhages with hemosiderosis H & E stain x 100; **F**: Liver showing marked congestion of portal vein and sinusoidal spaces x 100 H & E stain; **G**: Brain showing severe congestion of blood vessels, vacuolation of neurons and few glial cells x 100 H & E stain; **H**: Kidney Showing degeneration and necrosis of tubules in cortex x 100 H & E stain.

necrosis of muscle fibers in myocardium (Fig. 2A). Foci of myocardial coagulative necrosis i.e. infarct (Fig. 2B) was also noticed. Pyknotic nuclei and dark eosinophilic cytoplasm of muscle fibers indicated necrosis (Fig. 2C). All these findings led to diagnosis of acute myocardial infarction in heart. Lung showed severe congestion, haemorrhages with marked hemosiderosis and presence of

heart failure in lung parenchyma (Fig. 2D) which is important histopathological change confirming heart failure. Spleen showed extensive diffuse haemorrhages in white pulp and congestion along with hemosiderosis (Fig. 2E). Liver showed marked congestion of central vein, portal vein and hepatic sinusoids as well as atrophy and necrosis of hepatocytes with fatty change in periportal area

(Fig. 2F). Cerebrum showed severe congestion of blood vessels, vacuolation of neurons and few glial cells (Fig. 2G) Kidney showed degeneration and necrosis of tubules in cortex which is due to insufficient blood supply to tubules which are very much vulnerable to ischemia (Fig. 2H).

Predisposing factors for acute myocardial infarction in veterinary species have not been determined. It could be associated with aortic and pulmonic stenoses, patent ductus arteriosus, atherosclerosis, idiopathic hypertrophic cardiomyopathy (Sidhu *et al.*, 2022), primary coronary thrombosis associated with renal disease, as well as coronary vasculitis and thrombosis, arrhythmia or bacterial endocarditis in dogs (Driehuys *et al.*, 1998). In present case, the dog was obese which was one of the very important predisposing factor for cardiac failure. Obesity leads to blockage of blood vessels due to plaque formation leading to thrombosis and cardiac failure (Driehuys *et al.*, 1998). Gross and histopathological findings observed in present case are very characteristic such as severe congestion in liver, spleen and lungs along with typical heart failure cells in lung parenchyma. Acute myocardial infarction resulted into reduced blood supply leading to

necrosis of tubules in kidney, neuronal vacuolation due to oxygen insufficiency affecting their function and resulting into death of the dog. Regular health checkup, controlled diet and early detection can prevent occurrence of acute myocardial infarction.

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RETRACTION OF ARTICLE

This article earlier available at <https://www.luvas.edu.in/haryana-veterinarian/download/harvet2016-dec/1.pdf> entitled “*Occurrence of some organochlorine pesticide residues in poultry feed and meat*” has been retracted by the authors because of some error made during the data analysis process of the experimental observations due to counting the number of samples showing the concentration of pesticide below its corresponding Limit of Detection. All authors take full responsibility for this mistake and sincerely apologize for any inconvenience it may cause.

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