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RETROSPECTIVE EVALUATION OF CARDIOVASCULAR LESIONS IN CATS: PREVALENCE, GROSS, AND HISTOPATHOLOGICAL FINDINGS ENCOUNTERED FROM NECROPSY CASES AT PATHOLOGY LABORATORY, UNIVERSITY VETERINARY DIAGNOSTIC CENTRE (UVDC), UNIVERSITI MALAYSIA KELANTAN (UMK) FROM 2020 TO 2025

By

HO HUI SIN

A RESEARCH PAPER SUBMITTED IN PARTIAL FULFILMENT
OF THE REQUIREMENT FOR THE
DEGREE OF DOCTOR OF VETERINARY MEDICINE

2025

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ABSTRACT

Cardiovascular (CVS) disease is a major cause of morbidity and mortality in felines with hypertrophic cardiomyopathy (HCM), showing the highest incidence rate of CVS-related deaths. Despite advanced diagnostic tools like echocardiography, electrocardiogram and cardiac biomarkers, many cases remain underdiagnosed due to sudden death and asymptomatic. This study retrospectively evaluated the prevalence, gross and histopathological characteristics of cardiovascular lesions in feline necropsy cases encountered at the Pathology Laboratory, University Veterinary Diagnostic Centre (UVDC), Universiti Malaysia Kelantan (UMK), from 2020 to June 2025. A total of 52 feline necropsy cases were reviewed, representing 22.13% of the 235 total necropsy submissions during the study period. The findings revealed an increasing trend in feline necropsy prevalence, with the highest recorded rate of 36% in 2025. Histopathological cardiovascular lesions were identified in 69.23% of 52 feline necropsy cases examined between 2020 and June 2025, showing an overall increasing trend with a transient decline from 75% to 50% between 2021 and 2023 and a peak prevalence of 88.89% as of June 2025. Infectious diseases and trauma, including wound-related, motor vehicle, and high-rise trauma, were the most common antemortem diagnoses. Kittens (birth to one year old), male cats, and domestic shorthair (DSH) breeds represented the highest proportions of necropsy submissions at 46.15%, 65.28%, and 76.92%, respectively. The most frequently observed feline cardiovascular gross lesions were left ventricular hypertrophy (53.57%), congestion (46.43%), pericardial effusion (39.29%), and valvular endocardiosis (35.37%). Histopathologically, the predominant lesions were myocardial fibrosis (59.46%), myocarditis (56.76%), ventricular hypertrophy (51.36%), and ventricular dilatation (40.54%). Fisher's exact test demonstrated no significant association between antemortem diagnosis (infectious, non-infectious, trauma-related, and congenital anomalies) and the cardiovascular histopathological lesions (myocardial fibrosis, myocarditis, ventricular hypertrophy and ventricular dilatation) ($p > 0.05$), although infectious and trauma-related causes appeared to contribute to the lesions' development. This study provides insights into the prevalence and characteristics of feline cardiovascular lesions in Malaysia, enhancing the understanding of their potential causes and aiding clinicians in improving diagnosis, treatment and prognosis of feline cardiovascular diseases.

Keywords: Cardiovascular lesion, feline necropsy cases, prevalence, retrospective, gross lesions, histopathological lesions

ABSTRAK

Kajian ini dijalankan bagi menilai secara retrospektif prevalens serta ciri-ciri makroskopik dan histopatologi lesi kardiovaskular dalam kes nekropsi kucing yang dijalankan di Makmal Patologi, Pusat Diagnostik Veterinar Universiti (UVDC), Universiti Malaysia Kelantan (UMK) dari tahun 2020 hingga Jun 2025. Sebanyak 52 kes nekropsi kucing telah dikaji, mewakili 22.13% daripada jumlah keseluruhan 235 kes nekropsi sepanjang tempoh kajian. Kajian ini menunjukkan peningkatan dalam bilangan dan prevalens kes nekropsi kucing, dengan kadar tertinggi direkodkan pada tahun 2025 iaitu sebanyak 36%. Lesi histopathogi kardiovaskular dikenal pasti dalam 69.23% daripada 52 kes nekropsi kucing yang diperiksa bagi tempoh tahun 2020 hingga Jun 2025. Dapatan kajian menunjukkan trend peningkatan prevalens secara keseluruhan, walau bagaimanapun terdapat penurunan sementara daripada 75% kepada 50% antara tahun 2021 hingga 2023, dengan prevalens tertinggi direkodkan pada tahun 2025 (sehingga Jun) iaitu sebanyak 88.89%. Penyakit berjangkit dan trauma, termasuk trauma akibat luka, kemalangan kenderaan serta sindrom jatuh dari tempat tinggi, dikenal pasti sebagai diagnosis antemortem yang paling lazim. Anak kucing (lahir hingga satu tahun), kucing jantan dan baka *Domestic short-haired* (DSH) merupakan kumpulan utama yang dihantar untuk nekropsi, masing-masing mewakili 46.15%, 65.28% dan 76.92% daripada jumlah keseluruhan kes. Lesi makroskopik kardiovaskular yang paling kerap diperhatikan ialah hipertrofi ventrikular kiri (53.57%), kongesi (46.43%), lelehan perikardium (39.29%) dan endokardiosis valvular (35.37%). Sementara itu, penilaian histopatologi menunjukkan fibrosis miokardium (59.46%), miokarditis (56.76%), hipertrofi ventrikular (51.36%) dan pendilatan ventrikular (40.54%) sebagai lesi utama yang diperhatikan. Analisis statistik menggunakan *Fisher's exact test* menunjukkan tiada hubungan yang signifikan antara diagnosis antemortem (berjangkit, tidak berjangkit, trauma dan anomali kongenital) dengan lesi histopatologi (fibrosis miokardium, miokarditis, hipertrofi ventrikular, pendilatan ventrikular) ($p > 0.05$). Walau bagaimanapun, penyebab berjangkit dan trauma didapati berpotensi menyumbang kepada perkembangan lesi-lesi tersebut. Keterbatasan kajian ini termasuk saiz sampel yang kecil akibat rekod yang tidak lengkap serta kehilangan blok tisu bagi kes terdahulu, yang mungkin menjejaskan kekuatan analisis statistik. Oleh itu, disarankan agar kajian masa hadapan diperluaskan meliputi kawasan geografi yang lebih luas serta melibatkan populasi kucing pelbagai baka bagi meningkatkan kebolehpercayaan data. Di samping itu, sistem pendokumentasian dan pengarkiban data nekropsi yang lebih tersusun dan piawai perlu

diwujudkan bagi memastikan kesempurnaan dan kebolehcapaian data. Kajian ini memberikan sumbangan penting terhadap pemahaman tentang prevalens dan ciri-ciri lesi kardiovaskular dalam kucing di Malaysia. Dapatan kajian ini dapat membantu klinikal veterinar dalam mempertingkat ketepatan diagnosis, strategi rawatan serta penilaian prognosis bagi penyakit kardiovaskular kucing, seterusnya meningkatkan tahap kesihatan dan kesejahteraan kucing secara keseluruhan.

Kata kunci: Lesi kardiovaskular, kes nekropsi kucing, prevalens, retrospektif, lesi makroskopik, lesi histopatologi

CERTIFICATION

This is to certify that we have read this research paper entitled **'Retrospective Evaluation of Cardiovascular Lesions in Cats: Prevalence, Gross, and Histopathological Findings Encountered from Necropsy Cases at Pathology Laboratory, University Veterinary Diagnostic Centre (UVDC), Universiti Malaysia Kelantan (UMK) from 2020 to 2025'** by Ho Hui Sin, an in our opinion, it is satisfactory in terms of scope, quality, and presentation as partial fulfilment of the requirements for the course DVT 55204 – Research Project.

DR. FATHIN FAAHIMAAH BINTI ABDUL HAMID
Lecturer
Faculty of Veterinary Medicine
Universiti Malaysia Kelantan

Dr. Fathin Faahimaah Binti Abdul Hamid
DVM (UMK), MSC in Pathology (UMK)
Lecturer
Faculty of Veterinary Medicine
Universiti Malaysia Kelantan
(Supervisor)

Dr. Norhidayah Binti Noordin
DVM (UMK), MVSc. (UPM)
Lecturer
Faculty of Veterinary Medicine
Universiti Malaysia Kelantan
(Co-Supervisor)

ACKNOWLEDGEMENTS

First and foremost, I would like to express my deepest gratitude to my supervisor, Dr. Fathin Faahimaah Binti Abdul Hamid, and my co-supervisor, Dr. Norhidayah Binti Noordin, for their unwavering guidance, patience and encouragement throughout my research journey. Their thoughtful advice, constructive feedback, and constant support have shaped not only this thesis but my learning experience in research. Their help has been invaluable throughout the entire process.

Besides, I am truly grateful to the Faculty of Veterinary Medicine (FPV), Universiti Malaysia Kelantan (UMK), for providing the facilities and opportunities that made this study possible. I also wish to thank the Histopathology and Rnomic laboratory assistants, Encik Mohd Faiz Bin Juha and Encik Suhaimi Bin Omar, for their technical help and consistent support throughout this project. I would also like to express my sincere gratitude to my partner in this study, Ng Hooi Ean, for your encouragement, help and companionship. Without your support, this thesis would not have been possible.

To my family, I owe my deepest appreciation. Their unconditional love, endless patience and constant belief in me have been the foundation of my strength. Thank you for walking beside me through every challenge and celebrating every small victory along the way.

Lastly, I am thankful to all who have contributed directly or indirectly, offered their support and encouragement during this journey. This thesis is as much yours as it is mine.

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LIST OF ABBREVIATIONS

UMK	Universiti Malaysia Kelantan
UVDC	University Veterinary Diagnostic Centre
FPV	Faculty of Veterinary Medicine
RVFW	Right ventricular free wall
IVS	Interventricular septum
HCM	Hypertrophic cardiomyopathy
DCM	Dilated cardiomyopathy
RCM	Restrictive cardiomyopathy
ASD	Atrial septal defects
VSD	Ventricular septal defects
FATE	Feline aortic thromboembolism
NT-proBNP	N-terminal pro-B-type natriuretic peptide
cTnl	Cardiac troponin I
ARVC	Arrhythmogenic right ventricular cardiomyopathy
UCM	Unclassified cardiomyopathy
LVH	Left ventricular hypertrophy
CHD	Congenital heart defect
FCoV	Feline Coronavirus
ATE	Aortic thromboembolism
FPV	Feline parvovirus
FIV	Feline Immunodeficiency Virus
SPSS	Statistical Packages for the Social Sciences
DSH	Domestic short-haired

DLH	Domestic long-haired
BSH	British short-haired
FIP	Feline infectious peritonitis

LIST OF SYMBOLS

%	Percentage
>	Greater than
<	Lesser than
x	Times
X	Magnification
µm	Micro-meter
°C	Degree Celsius

CHAPTER 1

1.0 INTRODUCTION

1.1 INTRODUCTION

The heart is the central organ of the cardiovascular system, together with the blood vessels. It contracts and relaxes rhythmically to pump blood through the blood vessels, supplying the entire body. Anatomically, the heart is located at the mediastinum and enclosed by the pericardial sac, which contains several millilitres of serous fluid to act as a lubricant when the heart is contracted and relaxed (Maxie, 2016). Structurally, the heart is a four-chambered, hollow, muscular sac divided into left and right sides, with each side composed of an atrium and a ventricle. The two atria function to collect blood and ensure sufficient blood fills both the ventricles. Besides, the blood flow in the heart is unidirectional with the aid of the heart valves, such as the mitral valve, tricuspid valve and semilunar valves, to prevent reflux (König & Liebich, 2020). A heart's weight varies with different factors such as species, age, sex, nutritional status and fitness level of the animal. The reference range of relative heart weight is about 1% of body weight of a newborn, while 0.3-0.8% in juveniles and adults (Maxie, 2016). Furthermore, the ventricular thickness varies greatly. In a healthy animal, the left ventricular free wall is thicker than the right ventricular free wall. The ratio of right ventricular free wall (RVFW) to interventricular septum (IVS) to left ventricular free wall (LVFW) should be 1:3:3 (Wilkie *et al.,* 2015). Any deviation from the macroscopic and microscopic evaluation of a heart during necropsy indicates pathological changes.

According to Zakaria Ahmad (2016), feline heart diseases can be categorised into congenital and acquired, with the latter more frequently diagnosed. Hypertrophic cardiomyopathy (HCM) (47%) is the most common acquired heart disease, followed by dilated cardiomyopathy (DCM) (18%) and restrictive cardiomyopathy (RCM) (15%). The other heart disease types (11%) include pericardial effusion, aortic insufficiency, heart base tumours, and feline heartworm disease, myocarditis (7%), and congenital heart disease (2%), which includes atrial septal defects (ASD) and ventricular septal defects (VSD). However, most of the cats with heart disease were asymptomatic and only presented with various clinical signs due to the deterioration of heart diseases such as abnormal heart sounds (heart murmur, gallop sounds, muffled heart sound), dyspnoea, tachycardia, cardiac arrhythmias and hypotension, congestive heart failure, feline aortic thromboembolism (FATE) and eventually leading to cardiac death (Ferasin, 2009; Fox et al., 2015). In clinical practice, there are various diagnostic tools in diagnosing feline heart diseases, such as radiography, echocardiography, electrocardiography and cardiac biomarkers such as NT-proBNP and cardiac troponin I (cTnl) (Côté *et al.,* 2011; Fox *et al.,* 2015). The common treatments used in managing feline myocardial disease are diuretics, calcium channel blockers, beta blockers, and inotropic drugs. Calcium channel blockers like diltiazem and verapamil are commonly used in managing symptomatic feline HCM. The prognosis of feline cardiomyopathy cases depends on the presence of clinical signs associated with congestive heart failure and/ or evidence of arterial thromboembolism, which represent negative prognostic indicators (Ferasin, 2009).

Wilkie et al (2015) and Stalker (2009) found that cardiac disease was the most common condition discovered in cats that died unexpectedly. The presumed cause of death by pathologists for feline that presented for necropsy with unexpected death with cardiac

disease are HCM (79%), arrhythmogenic right ventricular cardiomyopathy (ARVC) (4%), RCM (2%), unclassified cardiomyopathy (UCM) (1%) and non-cardiomyopathic cardiac conditions (13%) such as congenital defects such as peritoneal pericardial diaphragmatic hernia, ventricular septal defect and mitral valve dysplasia (4%), inflammatory lesions such as epicarditis and myocarditis (2.3%), endomyocarditis with left ventricular endomyocardial fibrosis (4.4%), endocardial fibroelastosis (1.2%) and myocardial fibrosis (1.2%). On the other hand, in the group of felines with unexpected death without cardiac disease, 15% were diagnosed with incidental cardiac diseases by pathologists such as HCM, left ventricle hypertrophy (LVH), pericarditis, myocarditis and myocardial fibrosis.

However, based on the current knowledge, there is still a knowledge gap in understanding the prevalence, pathological evaluation, including gross and histopathological findings of cardiovascular lesions in felines, particularly when the feline patients are asymptomatic from cardiovascular disease.

1.2 PROBLEM STATEMENT

Cardiovascular diseases represent a leading cause of morbidity and mortality in feline patients with hypertrophic cardiomyopathy (HCM), accounting for the highest incidence rate of cardiovascular-related deaths. However, many cardiovascular diseases have still often remained underdiagnosed despite many advanced diagnostic workups being available, such as echocardiography, electrocardiogram and cardiac markers, due to asymptomatic or non-specific clinical signs and sudden cardiac death. Hence, pathological evaluations, encompassing both gross and histopathological assessments, are crucial for diagnosing and elucidating the underlying causes of cardiovascular lesions. These assessments play a vital role in setting up the fundamentals in facilitating the early diagnosis of cardiovascular diseases in feline patients, even in the absence of clinical signs.

Nevertheless, significant knowledge gaps persist as there is limited regional data on the prevalence and the predominant type of cardiovascular lesions in felines, particularly in Malaysia and Southeast Asia. Besides, the other knowledge gap included the scarcity of comprehensive retrospective pathological studies on cardiovascular lesions in felines has further constrained our understanding of trends and cardiovascular lesion patterns over time. Hence, this study is essential to deepen the understanding of cardiovascular lesions in felines, which ultimately equips veterinarians with data-driven guidance to refine their diagnostic strategies and promote early diagnosis of feline cardiovascular diseases in clinical practice.

1.3 RESEARCH QUESTIONS

I. What is the prevalence of feline necropsy cases encountered in the Pathology Laboratory, University Veterinary Diagnostic Centre (UVDC), Universiti Malaysia Kelantan (UMK) from 2020 to June 2025?

II. What is the prevalence of histopathological cardiovascular lesions in feline necropsy cases encountered in the Pathology Laboratory, University Veterinary Diagnostic Centre (UVDC), Universiti Malaysia Kelantan (UMK) from 2020 to June 2025?

III. What are the common cardiovascular lesions observed in feline necropsy cases encountered in the Pathology Laboratory, University Veterinary Diagnostic Centre (UVDC), Universiti Malaysia Kelantan (UMK) from 2020 to June 2025?

1.4 RESEARCH OBJECTIVES

I. To determine the prevalence of feline necropsy cases encountered in the Pathology Laboratory, University Veterinary Diagnostic Centre (UVDC), Universiti Malaysia Kelantan (UMK) from 2020 to June 2025.

II. To determine the prevalence of histopathological cardiovascular lesions in feline necropsy cases encountered in the Pathology Laboratory, University Veterinary Diagnostic Centre (UVDC), Universiti Malaysia Kelantan (UMK) from 2020 to June 2025.

III. To determine the common cardiovascular lesions observed in feline necropsy cases encountered in the Pathology Laboratory, University Veterinary Diagnostic Centre (UVDC), Universiti Malaysia Kelantan (UMK) from 2020 to June 2025.

1.5 RESEARCH HYPOTHESIS

I. Prevalence of feline necropsy cases encountered in the Pathology Laboratory, University Veterinary Diagnostic Centre (UVDC), Universiti Malaysia Kelantan (UMK) is in an increasing trend.

II. Prevalence of histopathological cardiovascular lesions in feline necropsy cases encountered in the Pathology Laboratory, University Veterinary Diagnostic Centre (UVDC), Universiti Malaysia Kelantan (UMK) is in an increasing trend.

III. The common cardiovascular lesions observed in feline necropsy cases are valvular endocarditis, myocarditis and ventricular hypertrophy.

CHAPTER 2

2.0 LITERATURE REVIEW

2.1 RISK AND PREDISPOSING FACTORS TO CARDIOVASCULAR LESIONS

Hypertrophic cardiomyopathy represents the most prevalent cardiovascular disease in cats, whereas it is considered rare in dogs. (Atkins *et al.,* 1992; Ferasin, 2009; Payne *et al.,* 2015; Wilkie *et al.,* 2015). However, few studies were conducted to determine the risk factors of feline HCM. According to Atkins et al (1992) and Payne et al (2015), middle-aged castrated male cats possessed a significantly greater risk than the neutered female cats. Besides, Ferasin (2009) and Meurs (2010) stated that Maine Coon, Ragdoll and British Short Hair are prone to having HCM due to the inherited autosomal dominant trait in Maine Coon that causing single base-pair genetic mutation in myosin binding protein C (MYBPC3) gene, but the mode of inheritance of gene mutation in MYBPC3 gene in Ragdoll remain unknown. Moreover, feline dilated cardiomyopathy (DCM) is commonly seen in cats fed taurine-deficient diets. However, not all cats fed taurine-deficient diets will develop DCM, suggesting that other factors such as taurine depletion associated with a potassium-deficient diet or a genetic predisposition may also play a role (Ferasin, 2009).

Schrope (2015) stated that ventricular septal defects represent the most common congenital heart defect (CHD) in mixed-breed cats, followed by aortic stenosis and hypertrophic obstructive cardiomyopathy. Among felines, certain congenital heart diseases show sex-predisposition in which valvular pulmonic stenosis, subvalvular and valvular aortic stenosis, and ventricular septal defects are more frequently reported in

males, while pulmonary artery stenosis shows a higher prevalence in females. Prevalence of CHD is lower in mixed-breed cats (0.14%), suspected due to the lack of purebreds in the study population or the actual changes in the disease prevalence.

2.2 ETIOLOGICAL AGENT ASSOCIATED WITH CARDIOVASCULAR LESIONS

Kent et al (2022) reported that cancer was the leading cause of death in cats (35.81%), followed by renal failure (10.81%), feline infectious peritonitis (6.72%), and cardiac disease (5.18%). Among feline cardiac pathologies, hypertrophic cardiomyopathy is the most frequently associated with mortality in cats, followed by endocardiosis, myocarditis and myocardial fibrosis. The etiological agent associated with cardiovascular lesions seen in felines can be categorised into 2 types, which are infectious causes and non-infectious causes. Infectious causes include all the cardiovascular lesions caused by the pathogen, including bacteria such as *Bartonella* spp, *Staphylococcus* spp, *Streptococcus* spp, *Escherichia coli* and *Enterococcus hirae*, which can lead to infectious endocarditis in felines (Chomel *et al.,* 2003; Palerme *et al.,* 2016; Loon *et al.,* 2020); viruses such as Feline Coronavirus (FCoV) that can cause fluid distension of the pericardial sac leading to cardiac tamponade and heart failure (Pedersen, 2009); and parasites, etc, while non-infectious causes include trauma and congenital anomalies.

On the other hand, congenital anomalies are a non-infectious cause of cardiovascular lesions in cats, often linked to inheritance or breed predisposition. For example, hypertrophic cardiomyopathy (HCM) is common in Maine Coon, Ragdoll, and British Shorthair cats due to an autosomal dominant mutation in the MYBPC3 gene (Ferasin, 2009; Meurs, 2010). Other breeds with a predisposition to HCM include the British

Shorthair, Norwegian Forest, Turkish Van, Scottish Fold, Bengal, Siberian, and Rex cats (Zachary, 2016). Moreover, the Burmese cat is prone to have primary endocardial fibroelastosis, which is a congenital anomaly of the heart (Zook & Paasch, 1982; Zachary, 2016)

2.3 TYPE OF CARDIOMYOPATHY

According to Luis Fuentes et al (2020), cardiomyopathy is defined as a heart muscle disease in which the myocardium is structurally and functionally abnormal in the absence of any other cardiovascular disease sufficient to cause the observed myocardial abnormality. Cardiomyopathy can be further classified based on phenotypic features into a few categories, which are hypertrophic cardiomyopathy (HCM), dilated cardiomyopathy (DCM), restrictive cardiomyopathy (RCM), arrhythmogenic right ventricular cardiomyopathy (ARVC) and non-specific phenotype. Among them, HCM is the most common type of cardiomyopathic phenotype seen in cats. HCM is defined as diffuse or regional increased left ventricular wall thickness with a nondilated left ventricular chamber. (Atkins *et al.,* 1992; Fox, 2003; Ferasin, 2009; Wilkie *et al.,* 2015). Dilated cardiomyopathy (DCM) is characterised by the dilation and impaired contraction of the left ventricle or both ventricles (Richardson et al., 1996). However, Ferasin (2009) and Karp et al (2022) pointed out that modern cat diets containing adequate taurine have led to a drop in feline DCM prevalence, limiting it to about 10% of all feline cardiomyopathic conditions. Besides, RCM is characterised by the restrictive filling and reduced diastolic volume of one or both ventricles with normal or near-normal systolic function and wall thickness while ARVC is characterized by progressive fibrofatty replacement of right ventricular myocardium, initially with typical regional and later global right or some left ventricular involvement with relative sparing of the septum. For

cases that do not clearly align with any group previously, they will fall under unclassified cardiomyopathies (UCM). However, Kittleson et al (2021) highlighted that the cardiomyopathies are usually indistinguishable clinically. This is because cats with subclinical cardiomyopathies may or may not exhibit the characteristic physical examination findings, such as a heart murmur and gallop, or radiographic findings. However, cats with severe cardiomyopathies will manifest the clinical signs of congestive heart failure, such as dyspnea and tachypnea or feline atrial thromboembolism, which is characterised by pain and paralysis. Sudden cardiac death is possible in cats with severe cardiomyopathies. The prognosis of felines with cardiomyopathies is usually guarded and poor once the clinical signs are manifested. Hence, a classification system was established for staging heart disease or heart failure in cats with cardiomyopathy. The classification system is as below:

Stage	Description
A	• Cats that are predisposed to a particular type of cardiomyopathy • Have no identifiable evidence of cardiomyopathy on physical examination
B1	• Subclinical cardiomyopathy (showing no overt clinical manifestation of heart failure, thromboembolism or syncope) • Cats with no to mild left or right atrial enlargement upon echocardiography • No at high risk of developing heart failure or aortic thromboembolism (ATE)
B2	• Subclinical cardiomyopathy (showing no overt clinical manifestation of heart failure, thromboembolism or syncope)

	• Cats with moderate to severe atrial enlargement upon echocardiography • At higher risk of developing heart failure or aortic thromboembolism (ATE) and may require medical intervention such as clopidogrel administration to prevent thrombosis in severely enlarged left auricle
C	Cats in left heart failure or those that have experienced ATE
D	Cats that have become refractory to a loop diuretic (> 6mg/kg/day furosemide)

Table 2.1: Classification system of heart disease or heart failure in cats with cardiomyopathy (Luis Fuentes *et al.,* 2020; Kittleson *et al.,* 2021)

2.4 INFECTIOUS ENDOCARDITIS

Infectious endocarditis is defined as a polypoid ulcerative inflammation of the endocardium (Sereda *et al.,* 2022). Infectious endocarditis is usually bacterial in origin and rarely seen in mycotic infection. The etiological agents that can cause infectious endocarditis in cats are many, such as *Bartonella* spp, *Staphylococcus* spp, *Streptococcus* spp, *Escherichia coli* and *Enterococcus hirae*, similar to the pathogens that cause infectious endocarditis in canines (Chomel *et al.,* 2003; Palerme *et al.,* 2016; Loon *et al.,* 2020). The lesions are often very large by the time of death, and it is often difficult to diagnose in clinical practice. The diagnosis remains presumptive at the clinical level until it is confirmed through bacterial isolation and identification and histopathological examination. Infectious endocarditis can be divided into 2 types based on the affected location of the heart. For instance, valvular endocarditis is defined when the lesion is present on the heart valves, but mural endocarditis is used when the lesion involves the

endocardium, spreads from the adjacent affected valve, or originates from the underlying myocardium. Valvular endocarditis mainly affects the mitral and aortic valves rather than the tricuspid valves. The pathogenesis of infectious endocarditis is believed to involve Virchow's triad in thrombogenesis, which includes endothelial injury, turbulence and hypercoagulability. Affected animals frequently have a pre-existing extracardiac infection that leads to bacteremia. The endothelial injury on the heart valves facilitates bacterial adhesion, proliferation, and initiation of an inflammatory reaction that leads to the fibrin deposition and subsequent vegetation formation on the heart valves. In some cases, the loosely attached vegetation may detach and form septic emboli and travel to other organs via the bloodstream, such as the heart, liver, kidney and meninges, leading to infarction, localised inflammation, or abscess formation. Mortality may result from cardiac failure secondary to valvular dysfunction or the systemic effects of bacteremia (Zachary, 2016).

2.5 MYOCARDITIS

Myocarditis is defined as the inflammation of the myocardium, which is a response of the myocardium to injury. There are various infectious factors that can lead to myocarditis, such as *Toxoplasma gondii*, *Trypanosoma cruzi*, feline immunodeficiency virus (FIV), *Staphylococcus* spp and feline parvovirus (FPV), *Bartonella henselae* and opportunistic fungi. Maxie (2016) reported that *Bartonella henselae* shows an increasing prevalence of causing both pyogranulomatous myocarditis and endocarditis in cats. Myocarditis is commonly associated with systemic infection that spreads hematogenously to the myocardium. There are different types of myocarditis, such as suppurative, necrotising, haemorrhagic, lymphocytic and eosinophilic. The suppurative myocarditis manifests due to the localisation of septic emboli that are composed of

pyogenic bacteria such as *Listeria monocytogenes* and *Actinobacillus equuli* in the myocardium, which most commonly originates from the vegetation of the valvular endocarditis.

2.6 ENDOCARDIAL FIBROELASTOSIS

Primary endocardial fibroelastosis is a congenital disorder characterised by diffuse thickening of the endocardium due to fibrous and elastic tissue proliferation, predominantly affecting the left heart and resulting in chamber dilation and hypertrophy (Zook & Paasch, 1982; Yoshida *et al.,* 2022; Zachary, 2016). This condition is recognized as an inherited congenital defect in Burmese and Siamese cats, although it is frequently underdiagnosed despite being a relatively common cardiac abnormality in felines. Clinical signs typically emerge between 3 and 4 weeks of age and include tachycardia, a gallop rhythm, systolic murmurs, cardiomegaly, and symptoms of congestive heart failure, such as dyspnea and terminal cyanosis. In milder cases, affected cats may exhibit minimal or no clinical signs, allowing them to survive to adulthood and potentially transmit the defect to offspring, although the precise mode of inheritance has not been established. Zachary (2016) further notes that it remains uncertain whether endocardial fibroelastosis represents a true congenital defect or a secondary response to left atrial dilation, as it may reflect an endocardial reaction to hypoxia commonly associated with cardiac diseases that lead to chamber dilatation.

2.7 GROSS AND HISTOPATHOLOGICAL LESIONS OF CARDIOVASCULAR LESIONS

For a feline with HCM, the heart will have increased cardiac mass, in which the fresh heart weight is over 20g. The left ventricular free wall and interventricular septum are thickening (non-dilated concentric hypertrophy) with or without concurrent thickening of the right ventricular free wall, grossly causing the heart lumen to be narrowed. (Gerdin *et al.,* 2011; Zachary, 2016). Not only that, but left ventricle volume is also reduced, and the papillary muscles are hypertrophied. Microscopically, cardiomyocyte hypertrophy, myofiber disarray, and myofiber interstitial fibrosis can be appreciated. Cardiomyocyte hypertrophy is distinguished by thick myofibers with the presence of oval and vesicular nuclei with prominent nucleoli. (Biasato *et al.,* 2015). Myofiber disarray is considered a histopathologic gold standard for the diagnosis of HCM in felines, in which the myofibers appear in the form of a peculiar and disorganized cellular structure, characterised by an interweaving pattern instead of the parallel arrangement. Besides, we can also observe intramural arteriosclerosis, which is characterised by severely narrowed small coronary vessels due to smooth muscle and connective tissue proliferation. (Fox, 2003; Biasato *et al.,* 2015; Wilkie *et al.,* 2015; Zachary, 2016) Myofiber interstitial fibrosis can be due to the increased interstitial matrix or myocardial replacement fibrosis. The myocardial fibrosis may be due to ischemia that is predisposed by the myofiber disarray and intramural arteriosclerosis (Cesta *et al.,* 2005; Biasato *et al.,* 2015).

In cases of infectious valvular endocarditis, Zachary (2016) stated that the affected valves exhibit as reddened and roughened heart valves with large, friable, yellow to grey fibrinous masses adhered to their surface, which can enlarge and potentially obstruct the valvular orifice. These fibrinous accumulations are referred to as vegetations. In chronic

stages, the fibrin deposits may become organised into fibrous connective tissue, forming irregular, nodular structures known as verrucae (wart-like lesions). Microscopically, the lesions are made up of accumulated layers of fibrin and numerous bacterial colonies, supported by a zone of dense leukocytic infiltration, especially neutrophils and developing granulation tissue.

For myocarditis caused by Feline Immunodeficiency Virus (FIV) concurrent with hypertrophic cardiomyopathy, the heart will appear to have cardiomegaly, left ventricular hypertrophy, myocardial pallor and multifocal to coalescing white-tan areas in myocardium and epicardium grossly. Microscopically, multifocal to coalescing areas of infiltration of inflammatory cells between the myocardium, such as lymphocytes, macrophages, neutrophils and plasma cells, can be appreciated microscopically (Rolim *et al.,* 2016).

For endocardial fibroelastosis, the affected cat will have a prominent, white and thickened endocardium, particularly the left ventricle, due to the proliferation of fibroelastic tissues. Not only that, the left atrium and left ventricle are often dilated, causing the hypertrophied left ventricle wall to appear thin. Additionally, Yoshida et al (2022) reported that a false tendon connecting the left ventricular free wall and the interventricular septum can be observed in affected kittens. Microscopically, significant deposition of collagen fibres and elastic fibres, mainly in the endocardium, can be observed (Zook & Paasch, 1982; Yoshida *et al.,* 2022). The older fibres will become thick and more organised as the new fibres form near the heart surface and are less organised.

Despite endocardial fibroelastosis, the endothelium remains intact without infiltration of fibrin and inflammatory cells.

Myocardial fibrosis is characterized by increased extracellular matrix (ECM), particularly collagen fibers within the myocardium (Weber *et al.,* 1994; Aupperle *et al.,* 2011). According to Hort (2000), there are three patterns of myocardial fibrosis, which are interstitial fibrosis, perivascular fibrosis and replacement fibrosis. Grossly, hearts with myocardial fibrosis show increased weight, a rounded shape, and a brown myocardium with occasional pale fibrotic areas, mainly affecting the papillary muscles and left ventricular wall. Microscopically, interstitial fibrosis is characterised by multifocal areas of spindle-shaped cells with elongated nuclei (fibroblasts) within the interstitium surrounding individual cardiomyocytes and perivascular fibrosis, which is characterised by affected arteries with angiosclerosis. Most of the fibrocytes will remain inactive with slender hyperchromatic nuclei and a small amount of cytoplasm, and predominance of collagen type I was seen in both types of fibrosis. (Aupperle *et al.,* 2011).

Vasques-Nóvoa et al. (2022) defined myocardial edema as the accumulation of cardiac water in the interstitial or intracellular compartments of the heart. Cardiac fluid balance relies on microcirculation, vascular integrity, interstitial structure, and lymphatic drainage. An imbalance in any of these factors can disturb fluid regulation and result in myocardial edema. Additionally, ischemia and inflammation stimulate leukocyte infiltration and the activation of matrix-degrading enzymes, which impair lymphatic function and reduce lymph flow. When capillary filtration exceeds the lymphatic system's capacity for fluid clearance, fluid accumulates in the myocardial interstitium.

Cardiomegaly is defined as cardiac enlargement with an overall increase in external dimensions of the heart (Maxie, 2016). Cardiac enlargement may affect all chambers or be limited to either the left or right chambers (Ware *et al.,* 2021). Cardiomegaly can be caused by ventricular hypertrophy or ventricular dilatation. For necropsy, cardiomegaly can be diagnosed based on the gross evaluation. Heart shape and heart weight can be an indicator of cardiomegaly (Winklhofer *et al.,* 2014). Generally, the enlarged heart will have a rounded apex with an overall increase in external dimension of the heart (Maxie, 2016). Kittleson et al (2021) reported that a healthy cat's heart typically weighs less than 20 grams, while in cases of hypertrophic cardiomyopathy (HCM), the heart weight exceeds 20 grams and can reach up to 38 grams. Normally, the heart-to-body weight ratio in cats falls between 3 and 4 grams per kilogram. In contrast, cats affected by HCM show an average ratio of 6.3 grams per kilogram. Since cardiomegaly can be caused by ventricular dilatation and ventricular hypertrophy, the transverse ventricular thickness ratio is important to determine the severity of the ventricular wall hypertrophy. It can be counted by using the formula below:

$$(LV + IVS) \div RV = 3.45 \text{ (Range } 2.94 – 4.17)$$

CHAPTER 3

3.0 METHODOLOGY

3.1 STUDY AREA

This research was conducted at the Pathology Laboratory, University Veterinary Diagnostic Centre (UVDC), Universiti Malaysia Kelantan (UMK), which includes the Postmortem Laboratory and Histopathology Laboratory. A collection of case records from laboratory usage logbook was conducted at the Postmortem Laboratory, while the analysis of the cases and the histopathology slides observation were conducted at the Histopathology Laboratory.

3.2 STUDY DESIGN

This research study used a retrospective cross-sectional design in which reliable case records were collected from the laboratory usage logbook and specimen submission and request forms from the Postmortem Laboratory and the Histopathology Laboratory, UVDC, UMK. The record was retrieved from 2020 to June 2025. The data of the feline cases that presented to the Postmortem Laboratory, UVDC, UMK, for necropsy were recorded in a data sheet.

3.3 STUDY POPULATION

The study population for this research consists of feline cases that were presented to the Postmortem Laboratory, UVDC, UMK for necropsy from 2020 to June 2025.

3.4 SELECTION CRITERIA

3.4.1 INCLUSION CRITERIA

The necropsy cases that involved felines that were conducted in the Postmortem Laboratory, UVDC, UMK from the year 2020 to June 2025, and the availability of historical data of the carcass.

3.4.2 EXCLUSION CRITERIA

The non-feline necropsy cases that presented to the Postmortem Laboratory, UVDC, UMK, from 2020 to June 2025 and the feline necropsy cases that are outside the range of the year needed.

3.5 SAMPLING METHOD AND PROCEDURE

3.5.1 DATA COLLECTION

The convenience sampling method was used in this study as the study population consists of all available feline necropsy cases from 2020 to June 2025 at the Postmortem Laboratory, UVDC, UMK. The data collection was started with the collection of the total number of necropsy cases, regardless of species, and the total number of feline necropsy cases that presented to the Postmortem Laboratory, UVDC, UMK. This was collected from the laboratory usage logbook of the Postmortem Laboratory, UVDC, UMK. Secondly, the data of the feline necropsy cases, such as patient ID, age, breed, sex, management, clinical history and antemortem diagnosis, were collected from the Histopathology Laboratory, UVDC, UMK from the specimen submission and request form. The study was focused on feline necropsy cases encountered from 2020 to June 2025. The data of feline necropsy cases will be recorded and compiled into a data sheet for analysis.

3.5.2 RESECTION OF TISSUE BLOCK FOR SLIDE INTERPRETATION

For the previous feline necropsy cases in which the histopathology slides cannot be retrieved, the tissue blocks were resected by using a rotary microtome (Leica RM2245, Germany) to obtain a thin tissue section at 2.5 µm in thickness for microscopic examination. The tissue block was fixed on the stage of the rotary microtome and gradually brought toward the blade with the help of an adjustable wheel until the block surface was in contact with the blade. Each rotation of the wheel produced a thin section, ideally under 2.5 µm in thickness, which forms a ribbon and was floated on a clean warm water bath at 40°C to achieve flattening and to ensure uniform thickness and optimal adherence to the glass slide. The ribbon was then carefully transferred onto a glass slide by placing the slide into the water bath to allow attachment of the tissue section and lift the slide out vertically from the water to avoid bubbles and excess water trapped underneath the section and the slides were placed on the slide holder and left it dry in the fume hood.

The histopathology slide preparation was started with the deparaffinization step to remove the paraffin from the glass slides by running the glass slides through xylene 1 and xylene 2 for 2 minutes each. Secondly, the hydration step was carried out by placing the slides into 100% alcohol, 75% alcohol for 2 minutes, respectively, to remove the xylene. Then, the slides were rinsed under running tap water and placed into distilled water for 3 seconds. Subsequently, the slides were stained with Haematoxylin and Eosin stain. The slides were first stained with haematoxylin for 3 minutes, followed by an 8-minute wash with running tap water and a brief immersion in an acid-alkaline solution. Then, the slides were rinsed again under running tap water and placed in distilled water for 3 seconds. Following that, the

slides were stained with 0.5% eosin stain, washed again under running tap water, and finally dipped in distilled water for 3 seconds. Afterwards, the slides were undergoing a dehydration and clearing process by placing the glass slides through 75% alcohol, 100% alcohol, and xylene 1 for 2 minutes. Then, the glass slides were run through xylene 2 and dipped into xylene 1 once. Following that, the mounting process will be done by adding one drop of DPX mounting medium to the tissue sample and placing the coverslip on top of the glass slide. The histopathological slides were undergoing microscopic evaluation by using a ZEISS Primostar 3 light microscope, and the slides were scanned using a Panoramic MIDI II scanner to obtain high quality images.

3.6 DATA ANALYSIS

The data analysis chosen in this research study was the combination of descriptive and statistical analysis, which is Fisher's exact test. Prevalence of the feline necropsy cases was calculated using the number of feline necropsy cases over a period divided by the total number of necropsy cases over the same period. The formula used is:

$$Prevalence = \frac{Number\ of\ feline\ necropsy\ cases\ over\ a\ period}{Total\ number\ of\ necropsy\ cases\ over\ the\ same\ period}\ x\ 100$$

(Tenny & Hoffman, 2023)

The data analysis was included a double bar chart to depict the yearly distribution of the total necropsy cases encountered, regardless of species, and the total feline necropsy cases encountered at the Pathology Laboratory, UVDC, UMK. Additionally, a bar chart was created to show the prevalence of feline necropsy cases throughout the study period.

Besides, the feline necropsy cases were categorised based on the antemortem diagnosis of the feline, such as infectious, non-infectious, trauma, euthanasia, congenital anomaly, and others. All suspected cases of bacterial, viral, parasitic, or mycotic infections were grouped into the 'infectious' category, regardless of the body system that is being affected. On the other hand, the 'non-infectious' category includes cardiovascular diseases, kidney diseases, endocrine diseases, nutritional diseases and other non-infectious conditions. The 'Trauma' category generally referred to those cases that involve physical injuries that result from distressing events such as skin barrier breakage and bone discontinuity. 'Trauma' category was subdivided into 'trauma (wound)', which involves bite wound, myiasis and other traumatic wounds and 'trauma (motor-vehicle and high rise) which was defined as the involvement of a motor-vehicle traumatic incident or high-rise syndrome. Those clinical histories that were not listed, neoplasia, sudden death and unclear antemortem diagnosis were categorised under the 'others' category. A pie chart was generated to illustrate the distribution of the antemortem diagnosis of felines that were submitted for necropsy at the Pathology Laboratory, UVDC, UMK throughout the study period.

Moreover, the age variable of the feline carcass was converted into categorical data by categorising the age range as follows:

Category	Age range
Kitten	Birth to 1 year
Young adult	1 – 6 years
Mature adult	7 – 10 years
Senior	>10 years

Table 3.1 Classification of age range in felines (Quimby, 2021)

The data analysis for the age range will include a pie chart to depict the age of the felines that submitted for necropsy at the Pathology Laboratory, UVDC, UMK. Furthermore, data on breed and sex variation among feline carcasses submitted for necropsy at the Pathology Laboratory, UVDC, UMK, were collected and analysed, with the results presented in a pie chart to illustrate the distribution of breeds and sex.

Furthermore, the prevalence of cardiovascular gross and histopathological lesions in feline necropsy cases was determined by recording the frequency of each lesion and calculating its prevalence using the prevalence formula. The findings were presented through a combined bar and line chart, illustrating the frequency and prevalence of the respective cardiovascular gross and histopathological lesions. All the charts were generated using a data sheet to effectively summarise and present the findings clearly.

On the other hand, the data was analysed by using Statistical Packages for the Social Sciences (SPSS) version 27. The data were categorical and non-continuous, thus a non-parametric test was used in data analysis. Subsequently, Fisher's exact test was carried out due to the small sample size to find out the association between the antemortem diagnosis and the top four cardiovascular histopathological lesions in felines.

CHAPTER 4

4.0 RESULTS

4.1 ANNUAL DISTRIBUTION OF FELINE NECROPSY CASES IN PATHOLOGY LABORATORY, UVDC, UMK FROM 2020 TO JUNE 2025

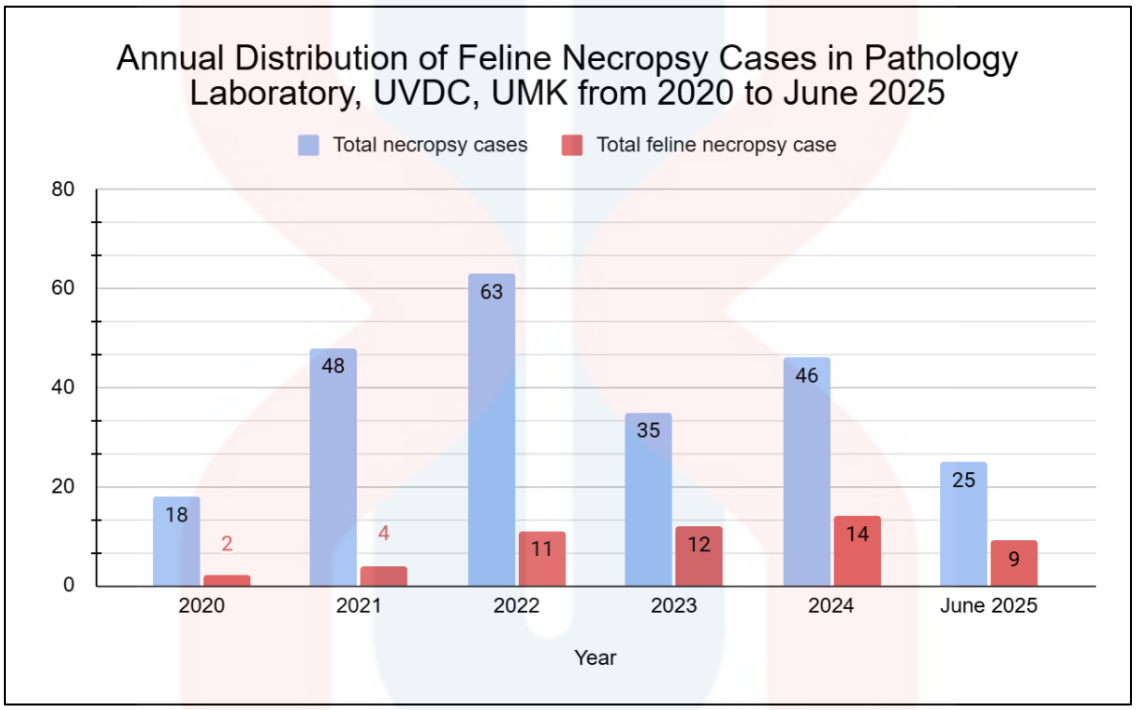

Figure 4.1: Annual distribution of feline necropsy cases in Pathology Laboratory, UVDC, UMK from 2020 to June 2025

Based on data obtained from the Pathology Laboratory, University Veterinary Diagnostic Centre (UVDC), Universiti Malaysia Kelantan (UMK), there were a total of 52 feline necropsy cases received from 2020 to June 2025. From 2020 to 2025, the number of feline necropsy cases received showed annual variation. In 2020, two cases were documented, increasing to four cases in 2021. A total of 11 and 12 cases were recorded in 2022 and 2023, respectively. The number of cases further increased to 14 cases in 2024. As of June 2025, nine cases had been received.

4.2 PREVALENCE OF FELINE NECROPSY CASES IN PATHOLOGY LABORATORY, UVDC, UMK FROM 2020 TO JUNE 2025

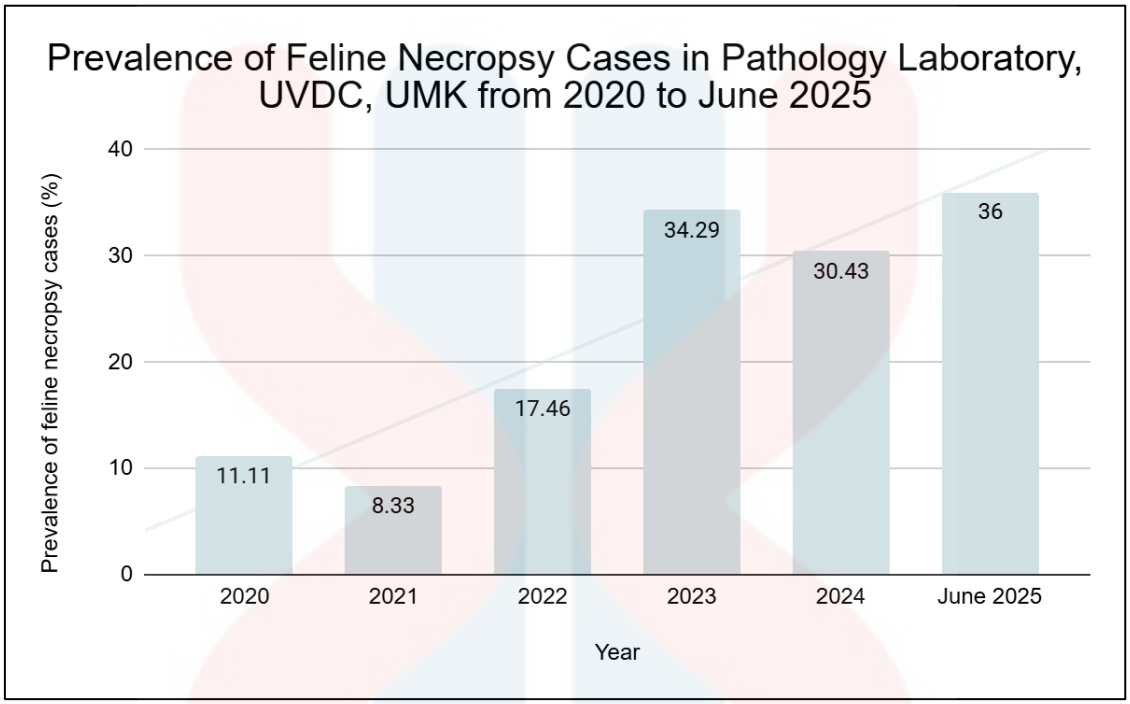

Figure 4.2: Prevalence of feline necropsy cases in Pathology Laboratory, UVDC, UMK from 2020 to June 2025

Year	Total necropsy case	Total feline necropsy case	Prevalence of feline necropsy cases (%)
2020	18	2	11.11
2021	48	4	8.33
2022	63	11	17.46
2023	35	12	34.29
2024	46	14	30.43
June 2025	25	9	36.00
Total	235	52	22.13

Table 4.1: Total necropsy, total and prevalence of feline necropsy cases in Pathology Laboratory, UVDC, UMK from 2020 to June 2025

The prevalence of feline necropsy cases in Pathology Laboratory, UVDC, UMK, from 2020 to June 2025 is 22.13%. The highest prevalence of feline necropsy cases was observed in 2025, based on the data collected until June 2025 at 36% followed by 34.29% in 2023 and 30.43% in 2024. Although minor fluctuations were observed in the annual

prevalence of feline necropsy cases throughout the study period, a marked increase was evident between 2020 and 2024, followed by a slight decline in 2024. In 2025, the number of feline necropsy cases did not exhibit a noticeable increase, which may be attributed to the fact that data collection was only conducted up to June 2025. Nevertheless, as of June 2025, the data indicate a potential continuation of the increasing trend in feline necropsy cases for the remaining months in 2025.

4.3 ANNUAL DISTRIBUTION AND PREVALENCE OF HISTOPATHOLOGICAL CARDIOVASCULAR LESIONS IN FELINE NECROPSY CASES IN PATHOLOGY LABORATORY, UVDC, UMK FROM 2020 TO JUNE 2025

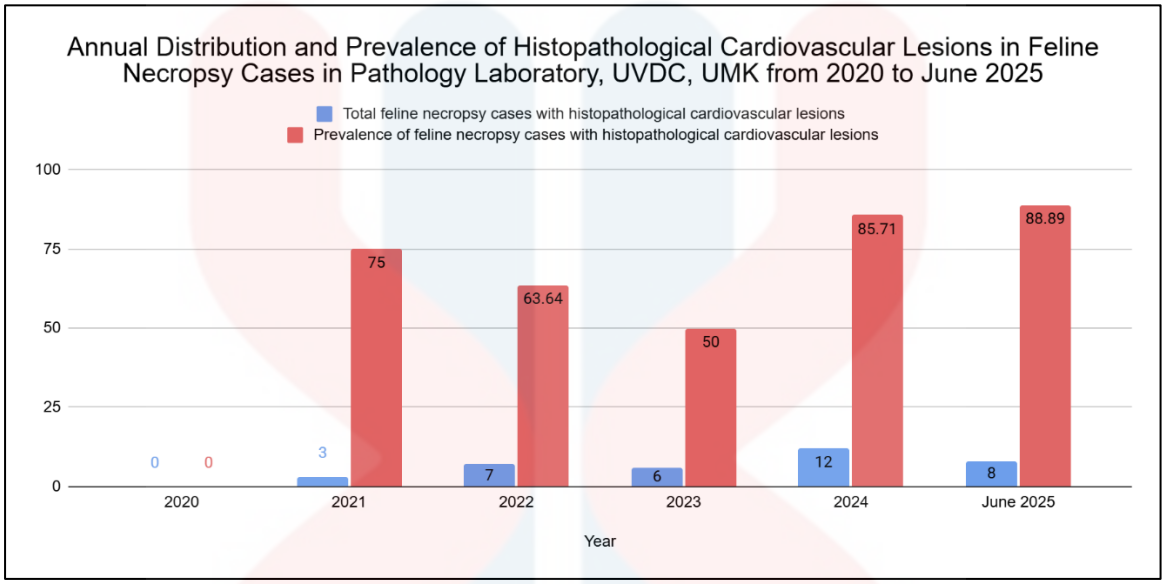

Figure 4.3: Prevalence of antemortem diagnosis of feline cases that submitted for necropsy in the Pathology Laboratory, UVDC, UMK, from 2020 to June 2025

Year	Total feline necropsy case	Total feline necropsy cases with histopathological cardiovascular lesions	Prevalence of feline necropsy cases with histopathological cardiovascular lesion (%)
2020	2	0	0
2021	4	3	75.00
2022	11	7	63.64
2023	12	6	50.00
2024	14	12	85.71
June 2025	9	8	88.89
Total	52	36	69.23

Table 4.2: Total feline necropsy, total and prevalence of feline necropsy cases with histopathological cardiovascular lesions in Pathology Laboratory, UVDC, UMK from 2020 to June 2025

Of the 52 feline necropsy cases examined between 2020 and June 2025, histopathological cardiovascular lesions were identified in 36 cases, resulting in an overall prevalence of 69.23% throughout the study period. An overall increasing trend in the prevalence of cardiovascular lesions was observed, with the highest prevalence recorded in 2025 (up to

June) at 88.89%. Nevertheless, a transient declining trend was noted between 2021 and 2023, during which the prevalence decreased from 75% to 50%.

4.4 PREVALENCE OF ANTEMORTEM DIAGNOSIS OF FELINES THAT SUBMITTED FOR NECROPSY IN PATHOLOGY LABORATORY, UVDC, UMK FROM 2020 TO JUNE 2025

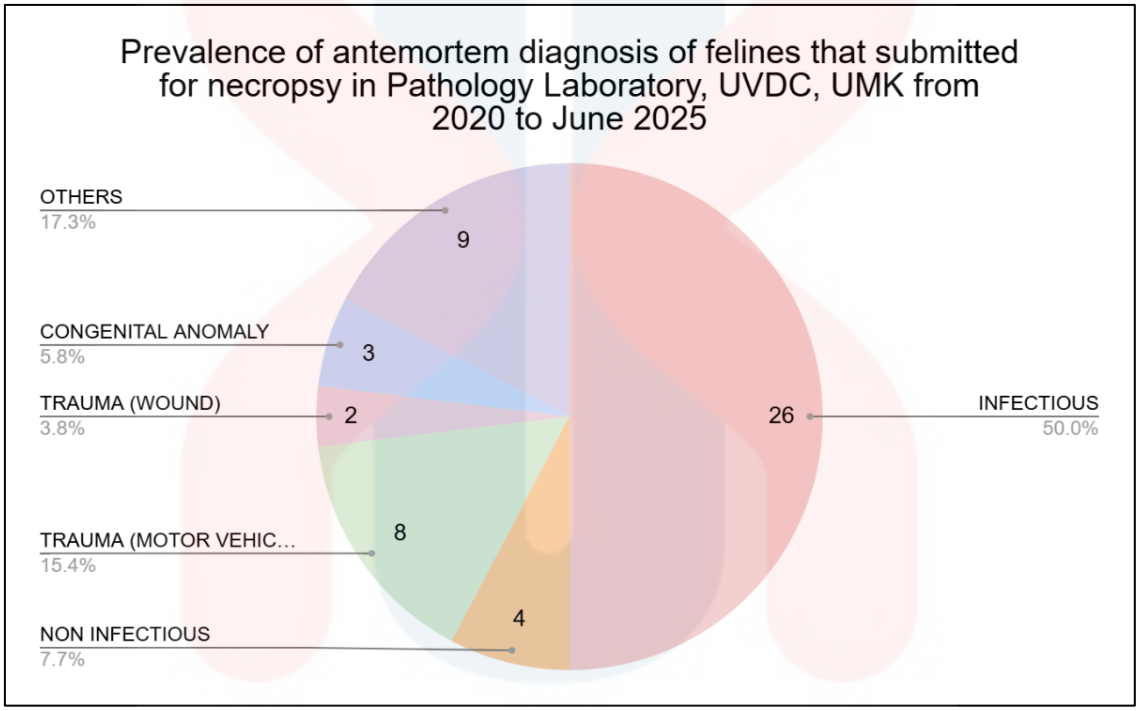

Figure 4.4: Prevalence of antemortem diagnosis of feline cases that submitted for necropsy in the Pathology Laboratory, UVDC, UMK, from 2020 to June 2025

Antemortem diagnosis of feline cases subjected to necropsy was classified into infectious, non-infectious, trauma (motor vehicle & high rise), trauma (wound), congenital anomaly and other causes. The data shows that half of the felines (50%) were diagnosed with an infectious disease, making it the most frequent cause. Trauma collectively represented 19.23% comprising 3.85% wound-related trauma and 15.38% motor vehicle-related or high-rise-related trauma. Notably, infectious causes were more than twice as prevalent as trauma-related causes.

4.5 AGE GROUP DISTRIBUTION OF FELINE NECROPSY CASES IN PATHOLOGY LABORATORY, UVDC, UMK FROM 2020 TO JUNE 2025

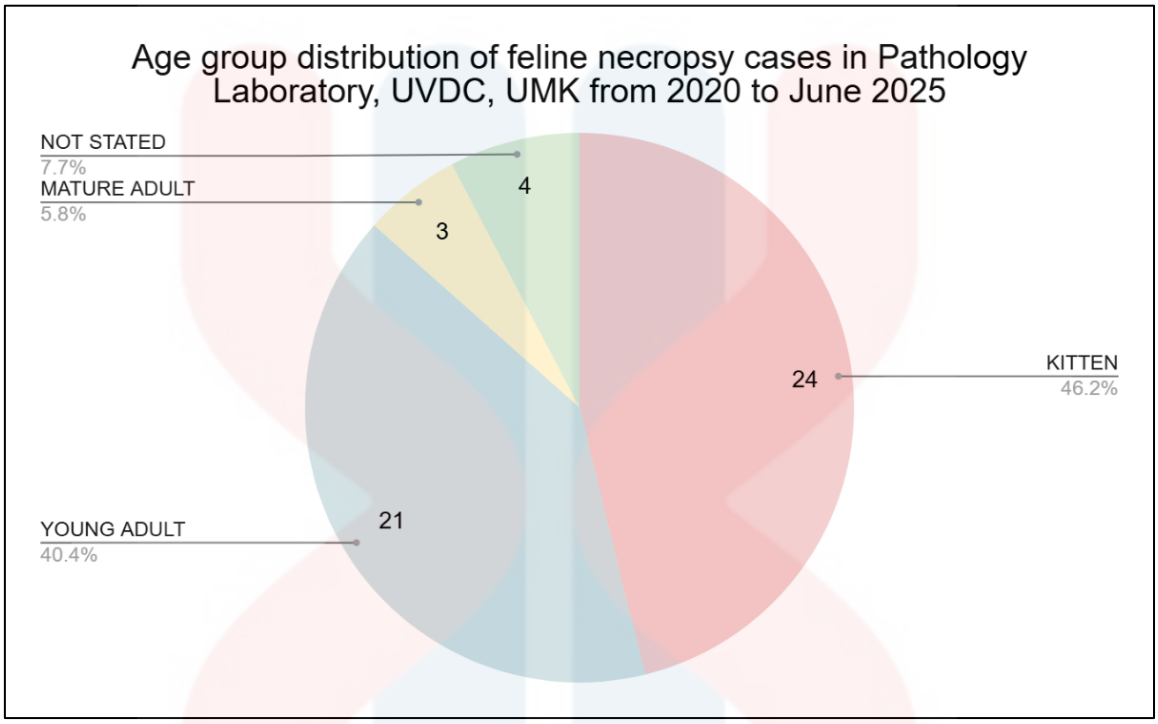

Figure 4.5: Age group distribution of feline necropsy cases in Pathology Laboratory, UVDC, UMK from 2020 to June 2025

In the distribution of feline necropsy cases by age, kittens (birth to 1 year) represented the largest proportion at 46.15%, followed by young adults (1 to 6 years) at 40.38%. Meanwhile, 7.7% of the feline necropsy cases did not have a clearly stated age. No cases were reported in the senior age group (0%).

4.6 BREED DISTRIBUTION OF FELINE NECROPSY CASES IN PATHOLOGY LABORATORY, UVDC, UMK FROM 2020 TO JUNE 2025

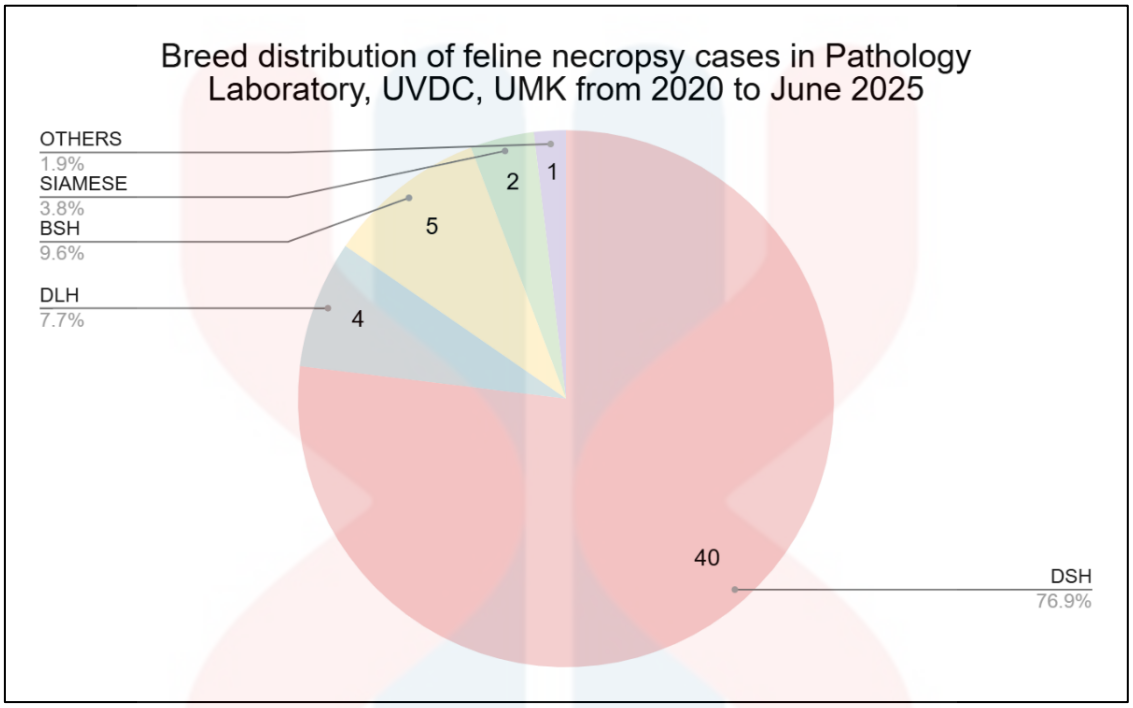

Figure 4.6: Breed distribution of feline necropsy cases in Pathology Laboratory, UVDC, UMK from 2020 to June 2025

In terms of breed distribution, domestic short-haired cats (DSH) constituted the majority of the feline necropsy submissions at 76.92%, followed by British short-haired (BSH) cats at 9.6% and domestic long-haired cats at 7.7%. Siamese cats accounted for 3.8% of the cases, while the 'others' category represented the lowest proportion at 1.9% due to not in listed.

4.7 SEX DISTRIBUTION OF FELINE NECROPSY CASES IN PATHOLOGY LABORATORY, UVDC, UMK FROM 2020 TO JUNE 2025

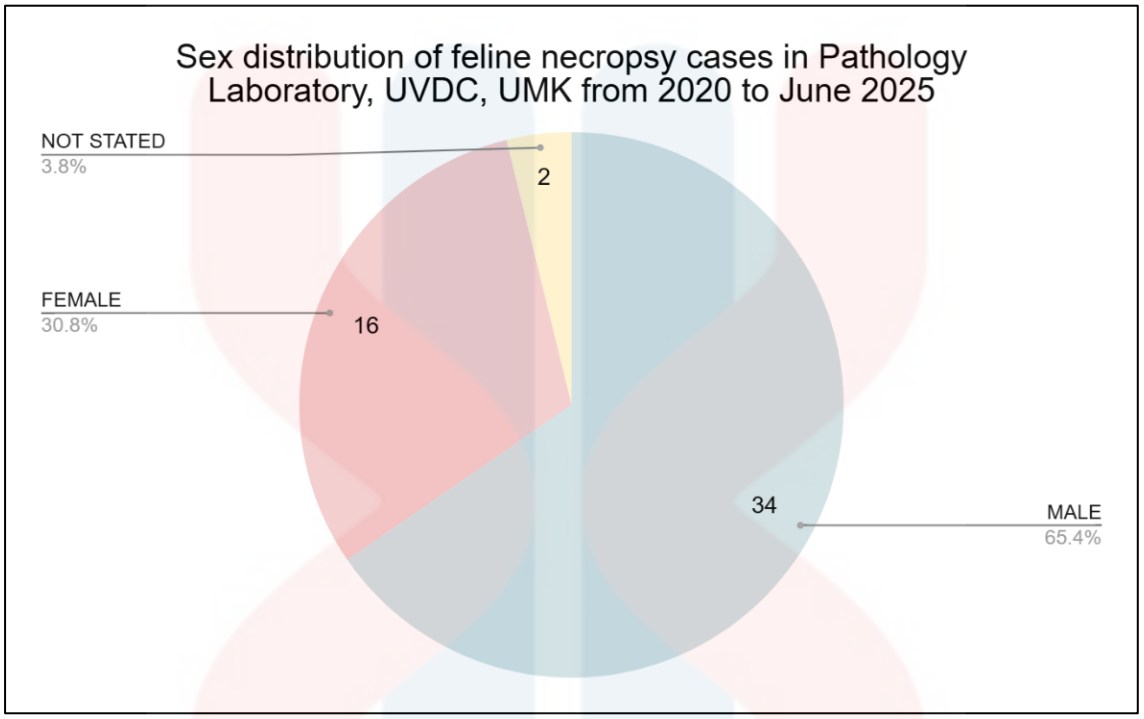

Figure 4.7: Sex distribution of feline necropsy cases in Pathology Laboratory, UVDC, UMK from 2020 to June 2025

Male cats predominated in the necropsy cases, accounting for 65.38%, which was more than twice the proportion of females at 30.77%, while 3.85% of cases had an unspecified sex.

4.8 PREVALENCE OF CARDIOVASCULAR GROSS LESIONS IN FELINE NECROPSY CASES IN PATHOLOGY LABORATORY, UVDC, UMK FROM 2020 TO JUNE 2025

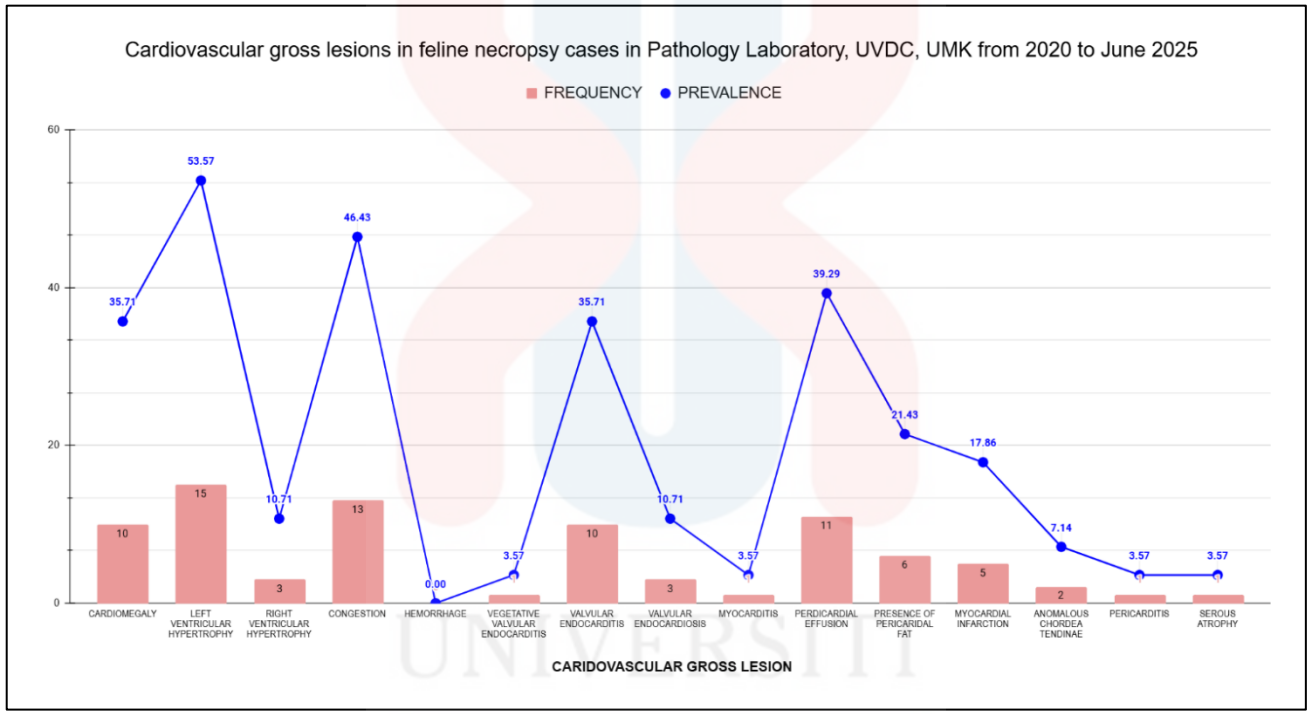

Figure 4.8: Distribution and prevalence of cardiovascular gross lesions in feline necropsy cases in Pathology Laboratory, UVDC, UMK from 2020 to June 2025

In the distribution of cardiovascular gross lesions among feline necropsy cases, left ventricular hypertrophy was the most common (53.57%), followed by congestion (46.43%) and pericardial effusion (39.29%). In contrast, vegetative valvular endocarditis, pericarditis and serous atrophy were the least frequent, each accounting for only 3.57% of cases.

4.9 PREVALENCE OF CARDIOVASCULAR HISTOPATHOLOGICAL LESIONS IN FELINE NECROPSY CASES IN PATHOLOGY LABORATORY, UVDC, UMK FROM 2020 TO JUNE 2025

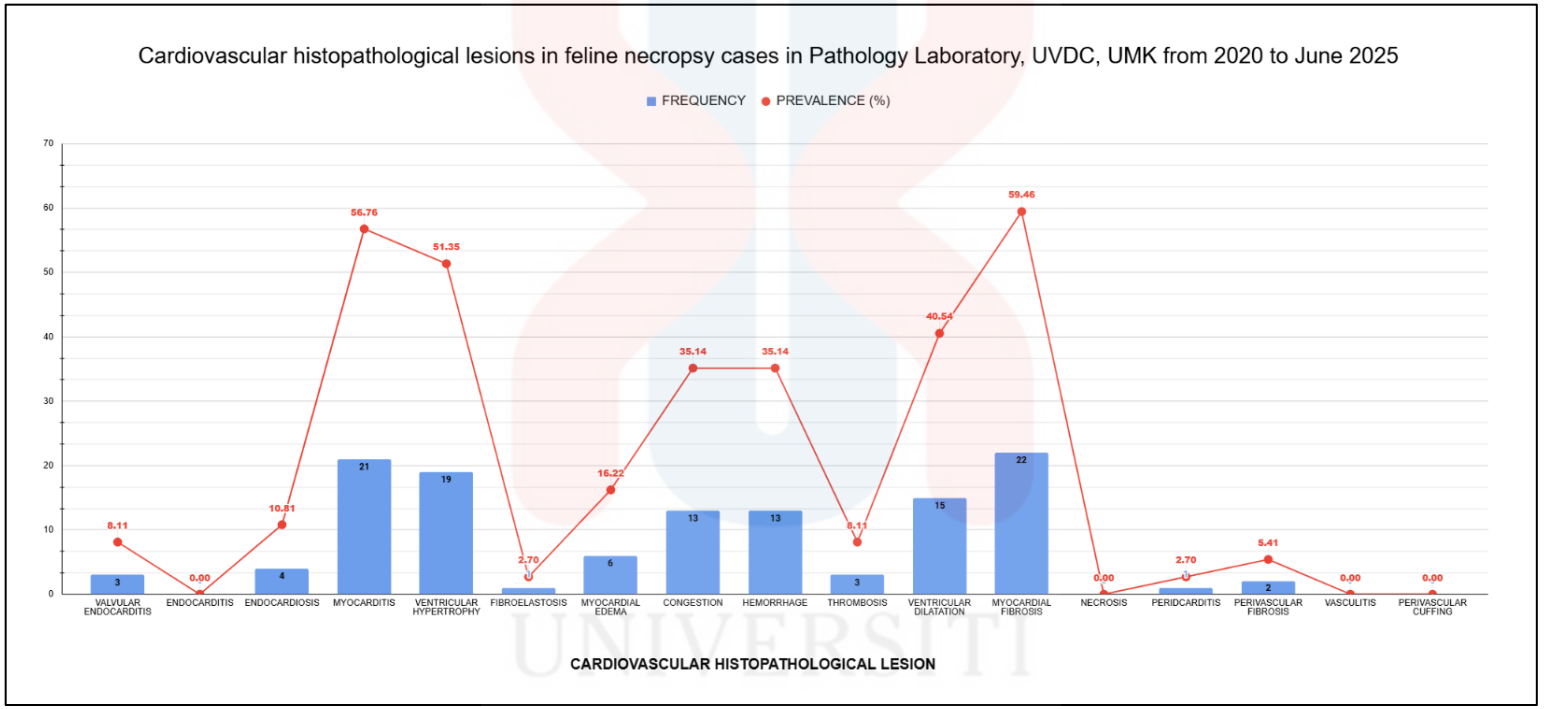

Figure 4.9: Distribution and prevalence of cardiovascular histopathological lesions in feline necropsy cases in Pathology Laboratory, UVDC, UMK from 2020 to June 2025

In the histopathological evaluation of the feline cardiovascular lesions, myocardial fibrosis has the highest prevalence (59.46%), followed by myocarditis (56.76%) and ventricular hypertrophy (51.35%). Conversely, fibroelastosis and pericarditis were the least common, each occurring in only 2.70% of the feline necropsy cases.

4.10 ASSOCIATION BETWEEN COMMON CARDIOVASCULAR HISTOPATHOLOGICAL LESIONS AND ANTEMORTEM DIAGNOSIS OF FELINE NECROPSY CASES

Cardiovascular histopathological lesions	Antemortem diagnosis	Positive	Negative	p-value
Myocardial fibrosis	Infectious	12 (46.2%)	14 (53.8%)	0.620
	Non-infectious	1 (25.0%)	3 (75.0%)	
	Trauma (motor vehicle & high-rise)	5 (62.5%)	3 (37.5%)	
	Congenital anomaly	1 (33.3%)	2 (66.7%)	
Myocarditis	Infectious	10 (38.5%)	16 (61.5%)	0.652
	Non-infectious	2 (50.0%)	2 (50.0%)	
	Trauma (motor vehicle & high-rise)	5 (62.5%)	3 (37.55)	
	Congenital anomaly	1 (33.3%)	2 (66.7%)	
Ventricular hypertrophy	Infectious	7 (26.9%)	19 (73.1%)	0.076
	Non-infectious	2 (50.0%)	2 (50.0%)	
	Trauma (motor vehicle & high-rise)	6 (75.0%)	2 (25.0%)	
	Congenital anomaly	2 (66.7%)	1 (33.3%)	
Ventricular dilatation	Infectious	10 (38.5%)	16 (61.5%)	0.573
	Non-infectious	1 (25.0%)	3 (75.0%)	
	Trauma (motor vehicle & high-rise)	1 (12.5%)	7 (87.5%)	
	Congenital anomaly	1 (33.3%)	2 (66.7%)	

Table 4.3: Statistical analysis test of the common cardiovascular histopathological lesions and antemortem diagnosis of feline necropsy cases (p-value)

The p-values obtained for the association between antemortem diagnosis and cardiovascular histopathological lesions, specifically myocardial fibrosis, myocarditis, ventricular hypertrophy and ventricular dilatation, were all greater than 0.05 ($p>0.05$). This indicates that none of the antemortem diagnoses, including infectious, non-infectious,

trauma (motor vehicle & high-rise) and congenital anomaly, showed a statistically significant correlation with these lesions. Nevertheless, infectious causes accounted for the highest proportion among the cases that exhibited myocardial fibrosis, myocarditis, ventricular hypertrophy and ventricular dilatation.

4.11 FELINE CARDIOVASCULAR HISTOPATHOLOGICAL FINDINGS ENCOUNTERED FROM NECROPSY CASES AT PATHOLOGY LABORATORY, UNIVERSITY VETERINARY DIAGNOSTIC CENTRE (UVDC), UNIVERSITI MALAYSIA KELANTAN (UMK) FROM 2020 TO 2025

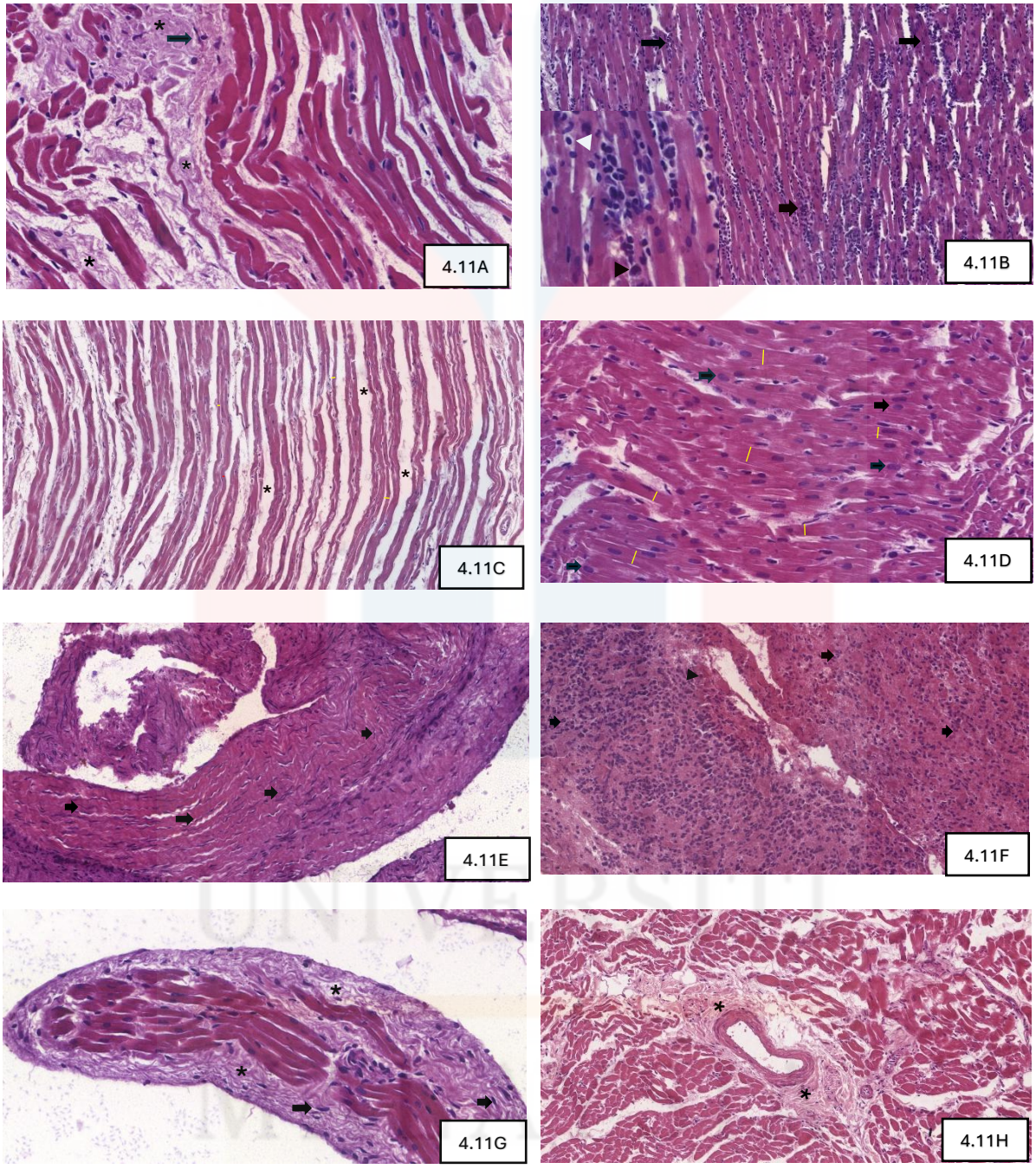

Figure 4.11 (A): Presence of collagen fibres (asterisk) and proliferations of fibroblasts, characterized by spindle-shaped cells with elongated nuclei (arrow) within the interstitium surrounding the myocardium, which indicates myocardial fibrosis (H&E, 32.9X). **Figure 4.11**

B: Hypercellularity of myocardium due to infiltration of mononuclear inflammatory cells (arrow), such as neutrophils, in between the myocardium, which indicates myocarditis (H&E, 20.1X). Infiltration of monocyte (black arrowhead) and lymphocyte (white arrowhead) were observed (H&E, 83.9X). **Figure 4.11 C:** Presence of uniform gap (asterisk) between the stretched cardiomyocytes (yellow line), which indicates ventricular dilatation (H&E, 17.1X). **Figure 4.11 D:** Presence of boxcar nuclei (arrow) with thickened cardiomyocytes (yellow line), which indicates ventricular hypertrophy (H&E, 40.9X). **Figure 4.11 E:** Thickening of the spongiosa layer of the mitral valve due to the presence of an increased number of fibroblasts, characterized by the spindle-shaped cells with elongated nuclei (arrow) and wavy arrangement, which indicates valvular endocardiosis (H&E, 20.6X). **Figure 4.11 F:** Presence of hypercellularity in the heart valve due to infiltration of mononuclear inflammatory cells, such as neutrophils (arrow) and macrophages (arrowhead) (see inset), which indicates valvular endocarditis (H&E, 31.8X). **Figure 4.11 G:** Thickening of the surrounding layer of papillary muscle due to the presence of an increased number of fibroblasts (arrow) and collagen fibres (asterisk) surrounding the papillary muscle, which indicates fibroelastosis (H&E, 25.4X). **Figure 4.11 H:** Presence of proliferations of collagen fibres (asterisk) surrounding the blood vessel, which indicates perivascular fibrosis (H&E, 22.9X).

CHAPTER 5

5.0 DISCUSSION

Feline cardiovascular diseases have been represented as a leading cause of morbidity and mortality in feline patients with hypertrophic cardiomyopathy (HCM), identified as the predominant cause of cardiovascular-associated mortality. Despite the availability of advanced diagnostic approaches, including echocardiography, electrocardiography, and cardiac biomarker analysis, underdiagnosis remains a common issue. This is primarily attributable to the asymptomatic nature of many cases, the presence of non-specific clinical manifestations, and the occurrence of sudden cardiac death, which have collectively obscured early detection and clinical management.

A total of 52 feline necropsy cases were documented over the five years, corresponding to a prevalence of 22.13% among the total necropsy submissions at UVDC, UMK. An upward trend in both the total number and prevalence of feline necropsy cases was observed from 2020 to 2024. Besides, histopathological cardiovascular lesions were observed in 69.23% of the 52 feline necropsy cases examined in this study. Temporal analysis revealed an overall increasing trend in prevalence over the study period, culminating at 88.89% in 2025 (as of June). This increase is presumed to be associated with the post-COVID phase, in which citizens resumed outdoor activities and promoted greater public mobility, potentially contributing to the increased trend. Increased public movement may have contributed to a higher incidence of road traffic accidents involving cats, resulting in increased mortality. Findings from this study indicate that trauma-related incidents, including motor vehicle accidents and high-rise syndrome, contributed to the rise in the annual distribution and prevalence of feline necropsy cases from 2024 up to

June 2025. In addition, the increasing number of feline necropsy submissions may reflect a higher incidence of sudden and unexpected deaths of unknown etiology, deaths associated with cardiovascular disorders and neoplastic conditions (Kelly-Bosma *et al.,* 2023). Furthermore, some cases may have been underdiagnosed or presented without significant clinical signs (Silva *et al.,* 2013), or the animals may have exhibited subtle signs that went unnoticed prior to death. Conversely, the rise in feline necropsy submissions could also indicate that affected cats were brought for veterinary care at an advanced stage of illness, when the clinical signs became apparent, causing late intervention. Moreover, this trend may reflect increased owner awareness and acceptance of post-mortem examinations as a means of investigating the cause of death in companion animals. From 2024 to June 2025, a slight decline was observed in the total number of feline necropsy cases, likely due to the fact that data collection only extended until June 2025. Nevertheless, the highest prevalence of feline necropsy cases was recorded in 2025 (36% as of June), suggesting that the increasing trend may have persisted. However, this figure may represent an overestimation, as the data only encompass the first half of the year and may not accurately reflect the overall annual distribution for 2025. On the other hand, a slight but consistent decline was observed in the prevalence of histopathological cardiovascular lesions from 2021 to 2023. However, the absolute number of feline necropsy cases presenting with these lesions continued to show an increasing trend throughout the study period. This discrepancy may be attributable to data related factors such as variations in case submissions, incomplete records or differences in sample size rather than a true reduction in cardiovascular pathology.

In this study, infectious causes constituted the majority of antemortem diagnoses (50%) among feline necropsy cases, followed by trauma-related causes (19.23%), which

comprised motor-vehicle accidents or high-rise syndrome (15.4%) and wound-associated trauma (3.85%). These findings are consistent with those reported by Withoeft et al. (2019). Additionally, male cats represented the predominant population among the necropsy cases (65.38%), exceeding more than twice the proportion of female cats (30.77%). This observation is in line with previous studies indicating that adult male cats are more frequently affected by infectious diseases, particularly feline infectious peritonitis (FIP) (Withoeft *et al.,* 2019). The greater susceptibility among males may be attributed to their wider roaming range and increased environmental exposure during the search for females in oestrus, leading to higher contact rates with other cats of unknown health status and, consequently, greater pathogen transmission. Similarly, earlier research has shown that young, sexually intact male cats aged between three months and three years are more susceptible to coronavirus infection (De Oliveira *et al.,* 2003; Rohrbach *et al.,* 2001). The present findings also align with Conroy et al. (2018), who reported that younger cats (6 months to 2 years), particularly males and crossbred individuals, are more prone to trauma-related deaths, such as motor-vehicle accidents and high-rise syndrome, due to behavioral tendencies like increased roaming activity compared to females.

Kittens (birth to 1 year) constituted a significant proportion of the feline necropsy cases in this study. This may be attributed to their immature immune systems, which render them more susceptible to pathogen invasion and infectious diseases compared to young (1–6 years) and mature adult cats (7–10 years) (Pereira *et al.,* 2023). In addition, kittens are more prone to trauma-related mortality, particularly those involving motor-vehicle accidents, as a result of high-risk behaviours such as road crossing (Loyd *et al.,* 2013), hunting along roadsides (Wilson *et al.,* 2017), lack of experience with artificial dangers such as motor vehicles (Nakanishi *et al.,* 2010), and exploratory tendencies due to curiosity

(Uccheddu *et al.,* 2022). In contrast, older and female cats tend to exhibit fewer risky outdoor behaviours, including road crossing, and consequently face lower mortality risks from motor-vehicle incidents (McDonald *et al.,* 2017; O'Neill *et al.,* 2015; Loyd *et al.,* 2013; Rochlitz, 2003). Logically, we assume that high human population density and urban environments pose greater hazards to roaming domestic cats due to increased traffic. However, McDonald et al. (2017) reported no significant association between human population density or the degree of urbanisation and the proportion of road traffic–related feline mortality.

Domestic short-haired (DSH) cats represented the majority of feline necropsy submissions in this study, followed by British short-haired (BSH) cats. This finding may be attributed to the predominance of DSH cats as the most common breed in Malaysia, along with the limited diversity of cat breeds presented to the Pathology Laboratory, UVDC, UMK. The relatively higher number of BSH cats may reflect their increasing popularity among pet owners in Malaysia. Besides, their underlying predisposing factor to cardiovascular diseases, such as hypertrophic cardiomyopathy (HCM) and lead to sudden death (Meurs *et al.,* 2009), could account for their significant representation in feline necropsy cases. In contrast, cat breeds such as Maine Coon and Ragdoll, which are commonly associated with cardiovascular conditions like HCM, were not represented in this study. This absence could be due to their status as imported breeds and their relatively higher cost, making them less common among cat owners in the local population.

Histopathologically, myocardial fibrosis represented the most prevalent cardiovascular histopathological lesion in cats, followed by myocarditis, ventricular hypertrophy, and

ventricular dilatation. The predominance of myocardial fibrosis may be explained by its role as a pathological cardiac remodelling process that initially serves as a reparative mechanism to preserve myocardial structural integrity and prevent further tissue damage. This response is typically activated following myocardial infarction, which may arise from infectious diseases, traumatic injury, or underlying cardiovascular disorders such as hypertrophic cardiomyopathy (HCM) and dilated cardiomyopathy (DCM), both of which were among the notable antemortem diagnoses in this study. However, excessive accumulation of extracellular matrix components, including collagen and fibroblasts, within the myocardium can result in increased stiffness of the cardiac muscle and predispose to arrhythmias, cardiomyopathy, heart failure, and ultimately death (Eijgenraam *et al.,* 2019). In addition, myocardial congestion, identified as the second most common gross cardiovascular lesion in this study, may contribute to the development of myocardial infarction due to blood stagnation and inadequate oxygen supply. This, in turn, could further promote the progression and higher prevalence of myocardial fibrosis observed in the present findings. Myocardial congestion can arise secondary to valvular endocarditis and valvular endocardiosis. Both valvular lesions can lead to valvular insufficiency and valvular regurgitation, causing poor ventricular filling from the atrium. Subsequently, this can reduce the cardiac output and promote blood pooling in the heart, ultimately manifesting as myocardial congestion on both gross and histopathological examination. Myocardial congestion may be accompanied by pulmonary congestion and pulmonary edema due to the increased left ventricular end diastolic pressure, causing backflow of blood from left atrium to the lung via the pulmonary vein and this further causes increased in hydrostatic pressure and lead to pulmonary edema (Jasni & Al-Sultan, 2020; Sato, 2017).

Valvular endocardiosis is considered clinically uncommon in cats. the present study demonstrates that this lesion can be identified histopathologically in feline heart valves. Despite this finding, there is a lack of published studies specifically investigating feline valvular endocardiosis. Côté et al. (2011) reported that cats with degenerative valve disease rarely to develop severe valve thickening or prolapse that is common in canine degenerative valve disease. This observation suggests that although cats are capable of developing valvular endocardiosis, clinical diagnosis remains challenging. A subset of affected feline patients may not exhibit an auscultable cardiac murmur due to factors such as a small volume of regurgitant blood, eccentric regurgitant jets directed away from the body surface, a low pressure gradient driving regurgitation, and physical limitations during auscultation, including purring and fractious behaviour. These factors may contribute to the underdiagnosis and underreport of degenerative valvular disease in cats. In contrast, valvular endocardiosis is more readily identified during pathological examination in this study, as it represents one of the degenerative changes affecting feline heart valves, potentially arising from chronic irritation, trauma, or congenital anomalies.

The high prevalence of myocarditis observed among feline cardiovascular histopathological lesions in this study is likely associated with the high incidence of infectious diseases identified as antemortem diagnoses in feline necropsy cases. Myocarditis is commonly linked to systemic infections that spread hematogenously to the myocardium, involving pathogens such as *Toxoplasma gondii*, *Trypanosoma cruzi*, feline immunodeficiency virus (FIV), *Staphylococcus* spp., feline parvovirus (FPV), *Bartonella henselae* (Maxie, 2016), and opportunistic fungi (Zachary, 2016). The condition represents an immune-mediated response to infection, characterized by inflammatory cell infiltration into the affected myocardial regions (Prisco *et al.,* 2025). In addition to infectious

etiologies, myocarditis may also arise secondary to trauma, particularly motor-vehicle-related injuries, as reported by Luz et al. (2025) and Hofmann and Tater (2019). Traumatic injury can result in an open wound that serves as a portal of entry for bacteria into the systemic circulation. Once disseminated via the bloodstream, these bacteria will localise in the cardiac tissue, eliciting the inflammatory response, which is characterised by vasodilation, increased vascular permeability and recruitment of the inflammatory cells to the injury site via chemotaxis, diapedesis and migration, which consequently give rise to myocarditis (Zachary, 2016).

Ventricular hypertrophy accounted for a notable proportion of feline cardiovascular lesions, both grossly (53.57%) and histopathologically (51.35%). The predisposition of British Shorthair (BSH) cats may have contributed to this finding, as 2 out of 5 BSH cats (66.7%) exhibited ventricular hypertrophy lesions at both gross and microscopic levels. This observation aligns with the findings of Granström et al. (2011). However, no hypertrophic cardiomyopathy (HCM)–associated genetic mutations have been identified in BSH cats to date (Granström *et al.,* 2011; Meurs *et al.,* 2009). In the present study, ventricular hypertrophy lesions were primarily associated with infectious causes (7/26; 26.9%) and trauma-related cases, including motor-vehicle accidents and high-rise syndrome (6/10; 75%). Rolim et al. (2016) suggested that further investigation is required to elucidate the potential link between feline immunodeficiency virus (FIV) infection and the development of HCM or myocarditis, indicating the possible role of infectious diseases in the pathogenesis of ventricular hypertrophy. However, there are no studies have specifically examined the association between trauma (motor-vehicle accidents or high-rise syndrome) and the occurrence of ventricular hypertrophy in cats. The findings of this study indicate that ventricular hypertrophy was predominantly associated with infectious diseases and

trauma cases (including motor vehicle accidents and high-rise falls). This pattern suggests that the hypertrophy observed may represent a compensatory response of the myocardium prior to death. When cardiac output declines, the heart may undergo hypertrophic changes in an attempt to maintain the cardiac output and adequate perfusion to tissues and organs. Nevertheless, to distinguish whether the ventricular hypertrophy is attributable to contractile dysfunction in sarcomeric hypertrophic cardiomyopathy (HCM) or to a compensatory mechanism secondary to cardiac insufficiency remains challenging, as both conditions present with similar gross and histopathological features. This finding is consistent with the conclusions of Janus-Ziółkowska et al. (2025), who noted that the pathological diagnosis of HCM remains difficult and often remaining ambiguous even after excluding other potential causes of left ventricular hypertrophy in cats such as chronic kidney disease, hyperthyroidism and systemic hypertension. Therefore, accurate interpretation requires consideration of the overall case context rather than reliance solely on morphological and histopathological lesions. Furthermore, most of the cardiovascular lesions formation are not entirely independent pathological process with notable interactions among different types of cardiovascular lesions and leading to its formation and progression. In the present study, ventricular hypertrophy was identified in 19 feline necropsy cases, of which 13 cases were concurrently associated with myocarditis. The exact mechanism by which myocarditis leads to ventricular hypertrophy are not fully understood but it is like related to the infiltration of inflammatory cells between cardiomyocytes, which can impair myocardial contractility and reduce cardiac pumping efficiency. As a compensatory response to this functional impairment, the myocardium may undergo adaptive hypertrophy and fibrosis as part of healing and remodeling process. These findings are consistent with those reported by Higashikuni et al. (2022), who demonstrated that interleukin-1β (IL-1β), a pro-inflammatory cytokine produced

following NLRP3 inflammasome activation via heart–brain interactions, plays a role in inducing adaptive cardiac hypertrophy and the progression to heart failure in murine models. Similarly, Khor et al. (2014) reported that multifocal myocarditis with predominantly lymphocytic and neutrophilic infiltration was frequently observed in the left myocardium of cats with pre-clinical hypertrophic cardiomyopathy (HCM). The authors further suggested that myocardial fibrosis observed in HCM may be modulated by inflammatory processes mediated by cytokines released from infiltrating inflammatory cells, including interleukin-1 (IL-1), interleukin-6 (IL-6), and tumour necrosis factor-alpha (TNF-α), as supported by Sivasubramanian et al. (2001). Comparable findings were observed in the present study, in which myocardial fibrosis was identified in 22 feline necropsy cases, with 14 cases concurrently exhibiting ventricular hypertrophy and 18 cases associated with myocarditis. Notably, 12 cases demonstrated the concurrent presence of myocarditis, ventricular hypertrophy, and myocardial fibrosis, further supporting the interrelationship between inflammatory processes, myocardial remodelling, and hypertrophic changes.

One of the limitations of this study is the relatively small sample size of feline necropsy cases, which was attributed to incomplete records at the Universiti Malaysia Kelantan (UMK) Veterinary Diagnostic Centre (UVDC). This limitation may have resulted from inadequate documentation regarding the utilization of the Postmortem Laboratory and the animal species submitted for necropsy, where inconsistencies in case recording, incomplete submission forms and insufficient data management practices led to the difficulties in tracking the necropsy submission and retrieving relevant case information The lack of a standardized recording system and proper data archiving further contributed to the loss of important case details, thereby limiting the comprehensiveness and reliability

of data set. For instance, several cases, particularly those from the years 2020 and 2021, were missing due to insufficient record-keeping of gross lesions and the unavailability of tissue blocks that could not be retrieved from the Histopathology Laboratory for further examination. The limited number of samples consequently reduced the statistical power of the study. This small sample size may have contributed to the absence of a statistically significant association between antemortem diagnoses (infectious, non-infectious, trauma-related, and congenital anomalies) and cardiovascular histopathological lesions (myocardial fibrosis, myocarditis, ventricular hypertrophy, and ventricular dilatation). Moreover, the predominance of infectious cases among the antemortem diagnoses could have influenced the outcome of Fisher's exact test, thereby contributing to the non-significant association observed between the selected antemortem diagnoses and cardiovascular lesions. Additionally, the limited diversity of feline breeds submitted to the Pathology Laboratory, UVDC, UMK, further constrained the representativeness of the findings on feline cardiovascular gross and histopathological lesions. Finally, the low number of necropsy submissions and documented cardiovascular lesions may also be attributed to the limited public demand for necropsy services, as many pet owners are either unaware of their importance or are discouraged by financial considerations.

CHAPTER 6

6.0 CONCLUSION AND RECOMMENDATION

In conclusion, the prevalence of feline necropsy cases encountered in the Pathology Laboratory, Veterinary Diagnostic Centre (UVDC), Universiti Malaysia Kelantan (UMK) from 2020 to June 2025 was 22.13%, accounting for 52 cases out of a total of 235 necropsy submissions. An increasing trend in both the number and prevalence of feline necropsy cases was observed throughout the study period, with the highest prevalence recorded up to June 2025 at 36%. Moreover, histopathological cardiovascular lesions were observed in the majority of feline necropsy cases examined during the study period, with an overall prevalence of 69.23% among 52 cases from 2020 to June 2025. Although a temporary decline in prevalence was noted between 2021 and 2023, decreasing from 75% to 50%, an overall increasing trend was evident, culminating in the highest recorded prevalence of 88.89% by June 2025. Among the antemortem diagnoses, infectious diseases and trauma, including wound-related, motor vehicle-related and high-rise trauma, were identified as the two most common causes leading to feline necropsy submissions. Kittens (birth to one year old), male cats, and domestic shorthair (DSH) breeds constituted the highest proportions of submissions, representing 46.15%, 65.28%, and 76.92% of the total cases, respectively.

The most frequently observed cardiovascular gross lesions among the feline necropsy cases were left ventricular hypertrophy (53.57%), congestion (46.43%), pericardial effusion (39.29%), and valvular endocardiosis (35.37%). Correspondingly, the most common cardiovascular histopathological lesions identified were myocardial fibrosis (59.46%), myocarditis (56.76%), ventricular hypertrophy (51.36%), and ventricular dilatation (40.54%). Although the study found no statistically significant relationship between antemortem

diagnoses (infectious, non-infectious, trauma-related, and congenital anomalies) and the major cardiovascular histopathological lesions, the findings suggest that infectious and traumatic causes, particularly motor vehicle and high-rise trauma, may contribute substantially to the development of these lesions.

To overcome the limitations encountered in this study, future research should consider expanding the geographical scope to include a wider range of feline cases and breeds, thereby enhancing the representativeness and statistical robustness of the findings. It is also recommended that the laboratory establish a standardized data recording and archiving system for the Postmortem Laboratory. This would facilitate more efficient tracking of necropsy submissions, retrieval of relevant case information, and prevention of data loss. Furthermore, comprehensive documentation of gross and histopathological lesions should be ensured for every necropsy case to support future reference and analysis. Tissue blocks should be properly organized and labelled according to the year of collection to prevent misplacement. It is advisable to retain necropsy records and tissue blocks for a minimum of ten years prior to disposal, as this practice would support long-term data accessibility, improve the reliability of prevalence studies, and contribute to both research and educational purposes.

Overall, this study contributes to the understanding of common cardiovascular lesions in felines and their possible etiological factors, which have been insufficiently described in previous literature. The findings provide valuable insights for small animal medicine by assisting clinicians in refining diagnostic and therapeutic approaches to feline cardiovascular diseases. Moreover, the knowledge generated from this study may enhance the overall well-being of cats

through earlier detection, improved treatment strategies, and more accurate prognostic assessments.

7.0 APPENDIX

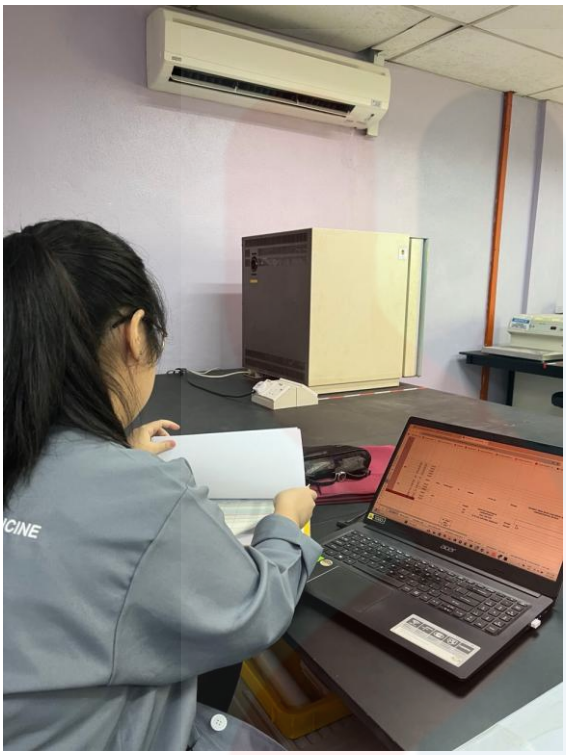

Figure A: Data retrieving from the sample submission forms in the Histopathology Laboratory, UVDC, UMK

Figure B: Retrieval of archived tissue blocks in the Histopathology Laboratory, UVDC, UMK

Figure C: Tissue block resection by using a rotary microtome (Leica RM2245, Germany) in the Histopathology Laboratory, UVDC, UMK

Figure D: Transfer of tissue 'ribbon' from water bath onto a glass slide in the Histopathology Laboratory, UVDC, UMK

Figure E: Automated Haematoxylin and Eosin staining of histopathological slides in the Histopathology Laboratory, UVDC, UMK

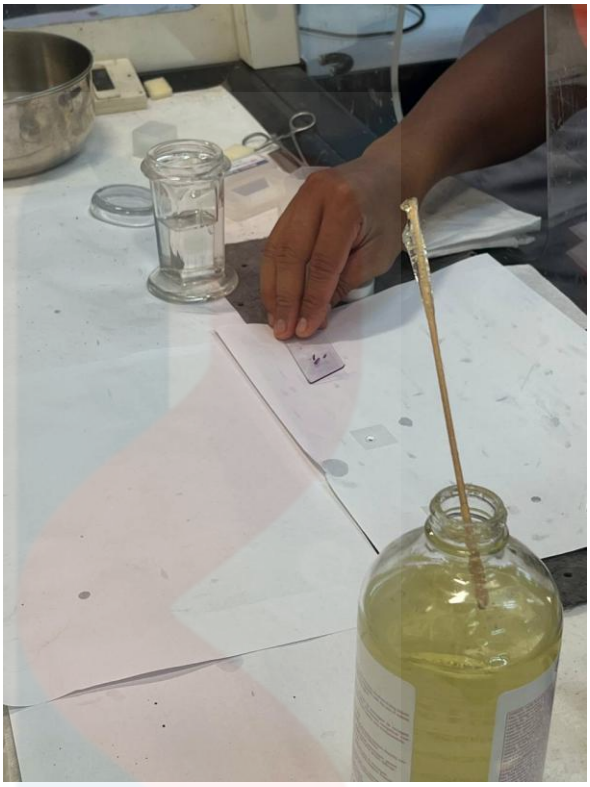

Figure F: Mounting process of histopathological slides by using DPX mounting medium in the Histopathology Laboratory, UVDC, UMK

Figure G: Sorting of archived histopathological slides in the Histopathology Laboratory, UVDC, UMK

Figure H: Microscopic examination of histopathological slides by using a microscope in the Histopathology Laboratory, UVDC, UMK

Antemortem diagnosis	2020		2021		2022		2023		2024		2025	
	Frequency	Prevalence (%)	Frequency	Prevalence (%)	Frequency	Prevalence (%)	Frequency	Prevalence (%)	Frequency	Prevalence (%)	Frequency	Prevalence (%)
Infectious	2	100	4	100	7	63.64	6	50	5	35.71	2	22.22
Non-infectious	0	0	0	0	3	27.27	0	0	0	0	1	11.11
Trauma (Motor vehicle & high rise)	0	0	0	0	0	0	0	0	3	21.43	5	55.56
Trauma (Wound)	0	0	0	0	0	0	1	8.33	1	7.14	0	0
Euthanasia	0	0	0	0	0	0	0	0	0	0	0	0
Congenital anomaly	0	0	0	0	0	0	0	0	3	21.43	0	0
Others	0	0	0	0	1	9.09	5	41.67	2	14.29	1	11.11
Total	2	100	4	100	11	100	12	50	14	100	9	100

Table 7.1: Annual distribution and prevalence of antemortem diagnosis of felines that submitted for necropsy in Pathology Laboratory, UVDC, UMK from 2020 to June 2025

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