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Original Research Article

An Array of Histomorphological Findings in Cardiac Autopsies: A Retrospective Study at a Tertiary Care Centre

T M Vincy*, S Hemalatha, S Brihadisvarar, S Nisha

Department of Pathology, Government Royapettah Hospital, Government Kilpauk Medical College, Chennai, Tamil Nadu
* Correspondence: Dr T M Vincy (tmvincy0641@gmail.com)

ABSTRACT

Background: Autopsy plays a vital role in identifying the cause and time of death. It also aids in understanding the emerging trends and pathogenesis in the course of the diseases and its consequences which leads to death. The aim of this study was to study the cardiac pathologies encountered during the histopathological examination in the autopsied heart specimens.

Methodology: We did a cross sectional retrospective study on heart specimens received in the Department of Pathology, Government Royapettah Hospital, Kilpauk Medical College, Chennai for a period of 3 years from 2020 to 2022.

Results: Out of 291 autopsy cases received during this period, 170 well preserved hearts obtained from medicolegal autopsies were studied. In this study 67% were males and 33% were females. In the current study we found that atherosclerosis and coronary artery disease are the most common findings. The most common coronary artery exhibiting atherosclerotic change was left anterior descending artery. An array of cases with rare cardiac pathologies such as giant cell myocarditis, hypoplastic left heart syndrome, infective endocarditis, hypertrophic cardiomyopathy, ventricular aneurysm and metastatic deposits in the heart were highlighted in the current study.

Conclusions: With the help of histopathological findings, the present study has contributed to a range of rare lesions in cardiac pathology determining the cause of death which were not diagnosed antemortem. Thus, this study emphasizes on the essentiality and multidisciplinary approach of postmortem histopathological examination in determining the causative factor of the disease significant enough to change the course of the disease if recognized before death.

Keywords
Heart, Coronary atherosclerosis, Aneurysm, Endocarditis, Cardiomyopathy, Giant cell myocarditis, Metastasis in the heart

INTRODUCTION

Sudden deaths due to cardiac events have increased in the last few decades. They are responsible for approximately 88% of sudden natural deaths.[1] In India, Coronary Heart Disease (CHD) prevalence shows an increasing trend particularly in the urban population due to lifestyle modifications and environmental changes.[2] Across the world, 25% of deaths are sudden cardiac deaths.[3] Cardiac autopsy serves as a tool to study different histo-morphological changes in normal and diseased heart. Thus, it is of great academic value and highlights the infrequent cardiac lesions which are unnoticed when the individual is alive. The aim of this study was to study the array of histo-morphological changes in the heart obtained from medicolegal autopsies.

MATERIALS AND METHODS

This was a 3-year cross sectional retrospective study conducted at the Department of Pathology, Government Royapettah Hospital, Kilpauk Medical College, Chennai, from January 2020 to December 2022. Hearts preserved in 10% neutral buffered formalin from 170 cases of

medicolegal postmortems were studied. Systematic examination of external surface of heart, ventricular wall, chambers, valves and coronaries was carried out. Irrespective of presence or absence of morphologically demonstrable lesions, a minimum of eight sections were studied from each heart including four heart chambers, great vessels and coronary arteries. Paraffin-embedded 5-micron thick tissue sections were assessed using haematoxylin and eosin stain. Special stains were used as and when required. The histopathology findings in each heart were recorded.

RESULTS

Out of 170 patients, 114 (67%) were males and 56 (33%) females, with male to female ratio of 2:1. Maximum number of patients 52 (30%) were observed in the age range of 31-40 years (Table-1). In the current study we found that Atherosclerosis (Figure-1A) and coronary artery disease were the most common findings. The most common coronary artery exhibiting atherosclerotic change was left anterior descending artery (38%). Figure-1B illustrates atheromatous plaque narrowing the lumen of left coronary artery. Complications of atherosclerosis, such as myocardial infarction (Figure-1C), was noted in 5%. Myocardial fibrosis (Figure-1D) due to old healed myocardial infarction was noted in 3% of study population.

Table-1: Demographic data of patients

Age in years	Males	Females	Total
0-10	09 (5 %)	06 (4 %)	15 (09 %)
11-20	19 (11 %)	05 (3 %)	24 (14 %)
21-30	20 (12 %)	09 (5 %)	29 (17 %)
31-40	31 (18 %)	21 (12 %)	52 (30 %)
41-50	16 (10 %)	12 (7 %)	28 (17 %)
51-60	13 (8 %)	02 (1 %)	15 (9 %)
61-70	04 (2 %)	01 (1 %)	5 (3 %)
Above 70	02 (1 %)	00 (1 %)	02(1 %)
Total	**114 (67 %)**	**56 (33 %)**	**170 (100 %)**

Congestive right heart changes such as right ventricular hypertrophy and right heart dilatation was evidenced by passive congestion of lungs, liver and spleen and were noticed in 8.2% of study population (Table-2). An array of cases with rare cardiac pathologies were highlighted in the current study, apart from the features of atherosclerosis, congestion, calcification and thrombus. The range of rare and incidental findings were: giant cell myocarditis, infective endocarditis, hypertrophic cardiomyopathy, left ventricular aneurysm with rupture, hypoplastic left heart syndrome and metastatic adenocarcinomatous deposits in the heart.

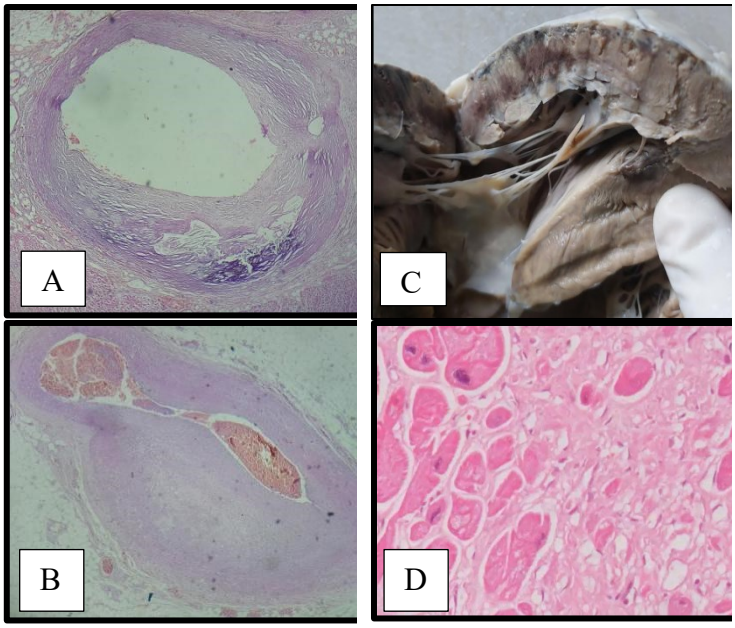

Figure-1 A) Atheroma showing lipid core with fibrous cap (H&E, X100). B) Left coronary artery showing atheromatous plaque causing occlusion of the lumen (H&E, X100). C) Cut open heart shows infarcted areas in the left ventricle. D) Myocardial fibrosis in an old healed infarct (H&E, X400)

Some of the interesting and rare cases are described here.

Case-1: A 45-year-old male who presented with shortness of breath, syncope and sudden death. Histo-morphological examination revealed the diagnosis of left ventricular aneurysm which appeared as thin circumscribed, fibrous outpouching from the left ventricular wall (Figure-2). Further examination revealed aneurysm occurring in the setting of old scarred myocardial infarction which was not detected antemortem.

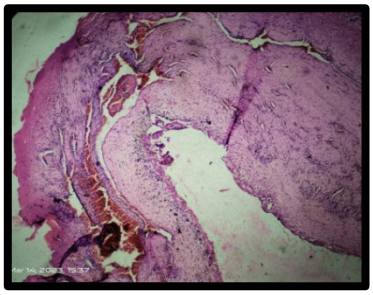

Figure-2: Histopathological examination showing ventricular aneurysm (H&E, X100)

Table 2: Histomorphological findings of heart

Findings			Number of cases
Fatty streaks in aorta			16 (9.41 %)
Myocardial infarction			10 (5.88 %)
Congestive right heart changes			14 (8.24 %)
Atherosclerosis	Right coronary artery	22 (33 %)	66 (38.82 %)
	Left circumflex artery	19 (29 %)	
	Left anterior descending artery	25 (38 %)	
Giant cell myocarditis			2 (1.18 %)
Cardiomyopathy			2 (1.18 %)
Myocarditis			2 (1.18 %)
Left Ventricular aneurysm			1 (0.59 %)
Left ventricular Hypertrophy			18 (10.59 %)
Infective endocarditis			1 (0.59 %)
Myocardial fibrosis			6 (3.53 %)
Hypoplastic left heart syndrome			1 (0.59 %)
Cardiac metastasis			1 (0.59 %)
Autolysis			8 (4.71 %)
Unremarkable			22 (13.53 %)

Case-2: Another interesting case was of a 27-year-old male who had past history of rheumatic heart disease and presented with fever and sudden collapse. Cut surface of the heart revealed large, irregular vegetations on the mitral valve cusps. Histo-pathological examination showed eosinophilic fibrinoid material with plenty of polymorphs and bacterial colonies along the mitral valve leading to a diagnosis of infective endocarditis.

Case-3: A 35-year-old male with history of hypertension had a sudden collapse. Macroscopic examination showed massive, concentric and symmetrical myocardial hypertrophy giving a banana shaped appearance of the heart which was typical of hypertrophic cardiomyopathy. Microscopic examination revealed massive myocyte hypertrophy with myofiber disarray along with interstitial fibrosis.

Case-4: An 8-months-old male infant presented with breathlessness and was brought dead to the hospital. Macroscopic examination revealed a hypoplastic left heart. Careful histopathological examination revealed marked myocyte disarray and vacuolated cardiomyocytes close to the epicardial surface.

Case-5: A 15-years-old female who was a known case of Systemic Lupus Erythematosus had recent viral infection. She presented with shortness of breath and collapsed on her way to the hospital. Histopathological examination revealed interstitial inflammatory cell infiltrate predominantly of lymphocytes and focal myocardial fibrosis prompting the possibility of viral myocarditis (Figure-3A).

Case-6: A 34-years-old male presented with history of shortness of breath and sudden death. Microscopic examination revealed giant cell myocarditis (Figure-3B) characterised by inflammatory cell infiltrate composed of multinucleated giant cells, lymphocytes, eosinophils and plasma cells.

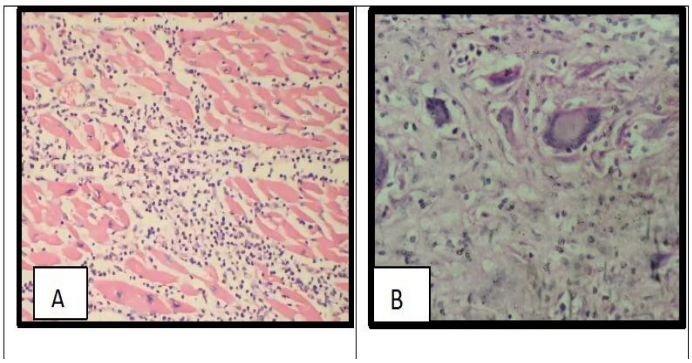

Figure-3 A) Viral myocarditis with myocardium showing lymphocytic infiltrates (H&E, X100). B) Giant-cell myocarditis with mononuclear inflammatory infiltrate with multinucleated giant cells and loss of muscle (H&E, X100)

Case-7: The current study also included a rare case of 30-years-old female with loss of weight and appetite, abruptly developed chest discomfort and soon succumbed to death on her way to the hospital. Gross examination revealed multiple grey white nodules in the pericardial fat, aorta and coronary ostia. Similar grey white nodules were noticed in her lungs. Histo-pathological examination revealed adenocarcinomatous tumour deposits in the pericardial fat of both ventricles, aorta and coronary ostia and primary adenocarcinoma in the lungs.

DISCUSSION

Sudden cardiac event arises as a complication and often is the first manifestation of heart disease. Sudden cardiac death is defined as a "natural, unexpected fatal event in an apparently healthy subject or in one whose disease was not so severe as to predict an abrupt outcome, occurring within one hour from the onset of symptoms".[4] In case of sudden cardiac deaths, gross and histopathological examination reveal the exact cause of death and also coexisting incidental pathologies. Fatty streaks in aorta were noted as minute multiple flat yellow streaks in 9.4% specimens of our study. They were noted in aortas of infants and virtually in all adolescents emphasizing the fact that fatty streak evolution starts at birth.[5] Though fatty streaks are not raised sufficiently to cause significant obstruction, they later lead to atheromatous plaque formation. Atherosclerosis (38%) remains the leading and the most common change among the cardiac findings observed in our study. This is similar to the study conducted by Sapna Patel et al[6] and Joshi et al.[7] In our study, we found that males are more affected than females and similar observation was noted by Sapna Patel et al.[6] In our study, atherosclerotic changes were seen more commonly in the age group of 40 to 50 years in males and 50 to 60 years in females. Atherosclerosis was frequently seen in patients with one or more risk factors such as diabetes, hypertension, hyperlipidaemia, obesity and history of smoking.[3] The most common coronary artery involved in our study was left anterior descending artery (38%) followed by right coronary artery (33%) and left circumflex artery (29%). Bansal et al found a similar observation in their study group where they noted male predominance in atherosclerotic changes and observed left anterior descending artery to be the most commonly involved vessel in their study.[7,8] Left ventricular aneurysm accounts for about 30 to 35% of acute transmural myocardial infarction.[9] Mural thrombus, arrythmias and heart failure remain the most important complications of ventricular aneurysm. Thus, our case reveals the importance of early screening and detection of cardiac events which can be life-saving to the individual.

Infective endocarditis is one of rare conditions which is associated with high morbidity and mortality. It should be suspected in cases where perforation or tear in valves with vegetations are noted.[10] High clinical suspicion and early diagnosis is important to prevent and treat the disease.[11] Thus, a prompt antibiotic prophylaxis could have prevented the death if diagnosed antemortem. Hypertrophic cardiomyopathy is a heterogeneous autosomal dominant genetic disorder[10] causing ventricular outflow obstruction. Its incidence is 1 in 500, affecting men and women equally. It is one of the most common causes of sudden unexplained death particularly in young athletes.[12] Hence, it is important to look for cardiac pathology in all young sudden deaths where significant death cause is unknown. Hypoplastic left heart syndrome is a rare congenital heart disease occurring in 3% infants with congenital heart disease. Its incidence is one in 5000 neonates.[13] It is due to underdevelopment of left sided heart structures such as aortic, mitral valves, left ventricle, arch of aorta and ascending aorta. It is due to decreased flow of blood through the left ventricle which inhibits the ventricular growth resulting in hypoplasia of the left heart during the development of foetus. Chaudhry et al suggested tests by using HLHS myocardial tissues to understand its aetiology and detect the biological pathways.[14]

The incidence of myocarditis is 10 to 22 per 100,000 persons.[15] Myocarditis can be asymptomatic and it can rapidly progress to heart failure. Hence, it is important to have strong clinical suspicion on myocarditis in individuals with autoimmune diseases. Giant cell myocarditis is a condition affecting young healthy individuals with a rapid progressive fatal course.[16] Though idiopathic, it is strongly associated with 20% of auto immune conditions.[17] Thus, it gains its importance by the fact that it represents fulminant course of the disease and carries a very poor prognosis.

At autopsy, cardiac metastases are detected in 0.7% to 3.5% in the general population and about 9.1% in patients with known cancers.[18] Metastases involving the heart are more frequently from carcinoma of lung[19] and breast, melanoma, leukaemia and lymphoma. Metastases reach the pericardial fat and heart by retrograde lymphatic extension, by hematogenous spread or direct contiguous extension or by direct extension. They can ultimately cause superior vena cava syndrome leading to death. With decreasing age of the patient, non-atherosclerotic causes of sudden cardiac events, such as congenital heart disease, cardiomyopathies, myocarditis and infective endocarditis, should be considered to detect the exact cause of death. Thus, these incidental findings emphasize that there is a need of a multidisciplinary approach in autopsy of sudden cardiac deaths and handling autopsy specimens which may be helpful in determining the cause of death.

CONCLUSIONS

From our study, we conclude that atherosclerosis is the main cause of cardiovascular ischemia and sudden cardiac death. In younger individuals, congenital and other incidental findings observed in our study should also be looked into as the other causes of sudden death. Hence, careful cardiovascular macroscopic examination and noting precise histomorphological changes in the heart play important role in finding the exact cause of death. Autopsy remains the first and last opportunity, to determine the cause of sudden cardiac death.

REFERENCES

1. Engelstein ED, Zipes DP. Sudden cardiac death. In: Alexander RW, Schlant RC, Fuster V, eds. The Heart, Arteries and Veins. New York, NY: McGraw-Hill; 1998:1081–1112.

2. Sreeniwas Kumar A, Sinha N. Cardiovascular disease in India: A 360 degree overview. Med J Armed Forces India. 2020 Jan;76(1):1-3. doi: 10.1016/j.mjafi.2019.12.005. Epub 2020 Jan 13. PMID: 32020960; PMCID: PMC6994761.

3. Mendis S, Puska P, Norrving B (2011) Global atlas on cardiovascular disease prevention and control, WHO, Editors: WHO; World Heart Federation; World Stroke Organization, NLM ID:101580439

4. Goldstein S. The necessity of a uniform definition of sudden coronary death: witnessed death within 1 h of the onset of acute symptoms. Am Heart J 1982;103:156–159.

5. McGill HC Jr, McMahan CA, Herderick EE, Malcom GT, Tracy RE, Strong JP. Origin of atherosclerosis in childhood and adolescence. Am J Clin Nutr. 2000 Nov;72(5 Suppl):1307S-1315S. doi: 10.1093/ajcn/72.5.1307s. PMID: 11063473.

6. Patel S, Rajalakshmi BR, Manjunath GV. Histopathologic Findings in Autopsies with Emphasis on Interesting and Incidental Findings-A Pathologist's Perspective. J Clin Diagn Res. 2016 Nov;10(11):EC08-EC12. doi: 10.7860/JCDR/2016/21106.8850. Epub 2016 Nov 1. PMID: 28050373; PMCID: PMC5198326.

7. Joshi C. Postmortem study of histopathological lesions of heart in cases of sudden death - An incidental finding. J Evid Based Med Healthc 2016; 3(6), 184-188.

8. Bansal YS, Mandal SP, Kumar S, Setia P. Prevalence of Atherosclerotic Coronary Stenosis in Asymptomatic North Indian Population: A Post-mortem Coronary Angiography Study. J Clin Diagn Res. 2015 Sep;9(9):HC01-4. doi: 10.7860/JCDR/2015/15106.6458. Epub 2015 Sep 1. PMID: 26500922; PMCID: PMC4606251.

9. Sattar Y, Alraies MC. Ventricular Aneurysm. [Updated 2023 Apr 3]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK555955/

10. Duncanson ER, Mackey-Bojack SM. Histologic Examination of the Heart in the Forensic Autopsy. Acad Forensic Pathol. 2018 Sep;8(3):565-615. doi: 10.1177/1925362118797736. Epub 2018 Aug 31. PMID: 31240060; PMCID: PMC6490581.

11. Rajani R, Klein JL. Infective endocarditis: A contemporary update. Clin Med (Lond). 2020 Jan;20(1):31-35. doi: 10.7861/clinmed.cme.20.1.1. PMID: 31941729; PMCID: PMC6964163.

12. Saikia UN, Bahl A, Mishra B, Sharma M, Kumari V, Talwar KK. Pathobiology of cardiomyopathies: Experience at a Tertiary Care Center. J Pract Cardiovasc Sci 2015;1:156 60.

13. Gobergs R, Salputra E, Lubaua I. Hypoplastic left heart syndrome: a review. Acta Med Litu. 2016;23(2):86-98.

14. Chaudhry B, Alqahtani A, Eley L, Coats L, Moldovan C, Annavarapu SR, Henderson DJ. The Left Ventricular Myocardium in Hypoplastic Left Heart Syndrome. J Cardiovasc Dev Dis. 2022 Aug 19;9(8):279. doi: 10.3390/jcdd9080279. PMID: 36005443; PMCID: PMC9409828.

15. Olejniczak M, Schwartz M, Webber E, Shaffer A, Perry TE. Viral Myocarditis-Incidence, Diagnosis and Management. J Cardiothorac Vasc Anesth. 2020 Jun;34(6):1591-1601. doi: 10.1053/j.jvca.2019.12.052. Epub 2020 Jan 7. PMID: 32127272.

16. Gadela NV, Krishnan AM, Mukarram O, Sthalekar N. Giant cell myocarditis. Proc (Bayl Univ Med Cent). 2021 Jan 22;34(3):401-402. doi: 10.1080/08998280.2021.1874775. PMID: 33953477; PMCID: PMC8059898.

17. Kandolin R, Lehtonen J, Salmenkivi K, Räisänen-Sokolowski A, Lommi J, Kupari M. Diagnosis, treatment, and outcome of giant-cell myocarditis in the era of combined immunosuppression. Circ Heart Fail. 2013 Jan;6(1):15-22. doi: 10.1161/CIRCHEARTFAILURE.112.969261. Epub 2012 Nov 13. PMID: 23149495.

18. Goldberg AD, Blankstein R, Padera RF. Tumors metastatic to the heart. Circulation. 2013 Oct 15;128(16):1790-4. doi: 10.1161/CIRCULATIONAHA.112.000790. PMID: 24126323.

19. Lichtenberger JP 3rd, Reynolds DA, Keung J, Keung E, Carter BW. Metastasis to the Heart: A Radiologic Approach to Diagnosis With Pathologic Correlation. AJR Am J Roentgenol. 2016 Oct;207(4):764-772. doi: 10.2214/AJR.16.16148. Epub 2016 Aug 4. PMID: 27490329.

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