Original Article

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Pathology Section

Histopathological Spectrum of Cardiac Lesions in Sudden Cardiac Death- An Autopsy Study

RASHMI REKHA MAHAPATRA[1], KALYANI PRAVA GOUDA[2], RUPA DAS[3], PUNYANSHU MOHANTY[4], GOURANGA CHARAN PRUSTY[5]

ABSTRACT

Introduction: Sudden Cardiac Death (SCD) is a serious health concern, and the incidence of SCD is rising globally. A number of causes can result in SCD in apparently healthy individuals and in people with undiagnosed cardiac disease. The study was done to evaluate the probable cause of death by observing various histomorphological changes in cardiac autopsies.

Aim: To establish the cause of SCD and study the histopathology, age and sex distribution, frequency, and location of different types of cardiac lesions.

Materials and Methods: The study was a descriptive, cross-sectional, observational study carried out in the Department of Pathology and Forensic Medicine and Toxicology (FMT) at PRM Medical College and Hospital, Baripada, Odisha, over a period of three years. Gross and microscopic findings on Haematoxylin & Eosin (H&E)-stained cardiac sections were studied. The final diagnosis was made on the basis of clinical, autopsy, and histopathological findings. The study was compared with other relevant studies. The data was analysed using Microsoft excel 2019 software.

Results: A total of 164 cases of SCD were included in this study. The maximum number of deaths occurred in the age group of 51-60 years (42 cases). The male to female ratio was 2.6:1, indicating an overall male preponderance. Out of 164 autopsied hearts, 85 cases of Ischaemic Heart Disease (IHD), including new, old, and mixed lesions were found, followed by Hypertrophic Cardiomyopathy (HCM) in 25 cases, multiple lesions in 12 cases, dilated cardiomyopathy in four cases, tubercular pericarditis with myocarditis in one case, infective endocarditis in three cases, atherosclerosis in one case, coronary insufficiency in one case, cardiac myxoma in one case.

Conclusion: Many factors can lead to SCD in apparently healthy individuals or in people with cardiac disease. In the present study, the most common cause contributing to SCD was IHD. The cause of SCD can be identified by a thorough postmortem examination and histological analysis.

Keywords: Cardiomyopathy, Heart, Histopathology, Myocardial infarction, Postmortem

INTRODUCTION

According to the World Health Organisation (WHO), SCD is defined as a sudden, unexpected death that occurs either within one hour of the onset of symptoms, if it is witnessed or within 24 hours, if it is unwitnessed and was previously seen to be alive and symptom-free [1,2]. American College of Cardiology/American Heart Association defines SCD as "A natural death due to cardiac causes, heralded by abrupt loss of consciousness" [3]. Sudden and unexpected death occurring within an hour of the onset of symptoms, or occurring in patients found dead within 24 hour of being asymptomatic and presumably due to a cardiac arrhythmia or haemodynamic catastrophe [4]. Although, many definitions of SCD have been put forth in the past, no single definition can be employed in all circumstances because there are numerous pathways that can result in such deaths [2]. Refining WHO definition it was suggested that "conventional SCD definition can be improved to better specify sudden arrhythmic death by restricting witnessed SCDs to ventricular tachycardia/fibrillation or non pulseless electrical activity of rhythms and unwitnessed cases to <1 hour since last normal" [5]. According to autopsy, the majority of sudden and unexpected fatalities are caused as a sequel to cardiovascular disease [6,7]. Myocardial Infarction (MI), which results from coronary artery insufficiency due to atheroma and thrombosis, is likely the most frequent cause of death noted in autopsies [8]. Myocarditis, HCM, congenital coronary artery anomalies, atherosclerotic coronary artery disease, conduction system abnormalities, mitral valve prolapse, and aortic dissection are the most prevalent underlying pathologic diseases in children and adolescents [9,10]. The most frequent autopsy findings in adults are coronary atherosclerosis and acquired types of cardiomyopathies [11,12]. The primary diagnostic technique for examining diverse histomorphological alterations in healthy and diseased hearts is cardiac autopsy [13]. Establishing the definitive diagnosis and, whenever possible, determining the cause of death is the main goal of the autopsy [14]. The concordance between clinical and pathological causes of death is said to be moderate, although autopsies are still a crucial process for determining causes of death [15]. It has frequently been seen that histopathology can clearly determine the implicated cardiac pathology when gross pathology is unable to determine the cause of death [16,17].

The objective of this cross-sectional study was to emphasise the histomorphological profile and demographic distribution of SCD in the Government Medical College and Hospital, Baripada, Odisha, India, which is a newly established tertiary care hospital that caters to patients from the tribal areas of Northern Odisha.

MATERIALS AND METHODS

The present study was a descriptive, cross-sectional, observational study carried out over a period of three years, from April 2019 to March 2022, in the Department of Pathology and FMT at PRM Medical College and Hospital, Baripada, Odisha, India. A total of 164 heart specimens were received during this period in the centre and were included in the study after taking due approval from Institutional Ethical Committee (Ref No 3/5th IEC Meeting-21, Date- 10/03/2021). Available clinical history, epidemiological data, medical diagnosis, and postmortem findings of all cases were noted from postmortem papers and police requisition forms, and histopathological analysis was performed.

Inclusion criteria: All the medicolegal autopsy cases of sudden, unexpected cardiac death without having a history of past illness were included in the study.

Exclusion criteria: Autopsy cases with deaths due to accidents, sudden deaths of non cardiac origin, intoxication, and autolysed samples were excluded from the study.

Study Procedure

Weight and measurements of the heart were noted. The external surface was examined for pericardial diseases and for signs of recent or old infarcts. After fixation with 10% formalin, the heart was dissected using the inflow outflow method as per the standard autopsy protocol. The thickness of the Left Ventricular Wall (LVW), Right Ventricular Wall (RVW), and Interventricular Septum (IVS) were measured. The valves were evaluated for the presence of calcification, stenosis, and vegetation. Bread-loaf sectioning of the ventricles was performed, beginning from the apex and moving transversely at a 10 mm interval, and the position and extent of recent and old myocardial infarcts, if present, were recorded. The right coronary artery, left anterior descending artery, and left circumflex coronary artery were all examined for thrombosis, calcification, and stenosis using serial sections every 4-5 mm. Then, evaluation of the aorta was done for atherosclerosis. Sections from the right ventricular free wall, left ventricular anterior, lateral, and posterior walls, IVS, the apex, the valves, the stump of the aorta, and sections from the coronary arteries were obtained. Whenever necessary, sections from suspicious pathological lesions were taken.

All sections were processed as per standard procedure, stained with H&E stain, and viewed under a light microscope. Histopathological changes in heart in various cardiac diseases were evaluated. Special staining was used whenever required.

STATISTICAL ANALYSIS

The data were collected and entered in a Microsoft excel spreadsheet in tabulated form. Statistical parameters like the relative frequency of various lesions, the site of distribution, and socio-demographic data like the distribution of diseases with respect to age and sex were evaluated using Microsoft excel 2019 software.

RESULTS

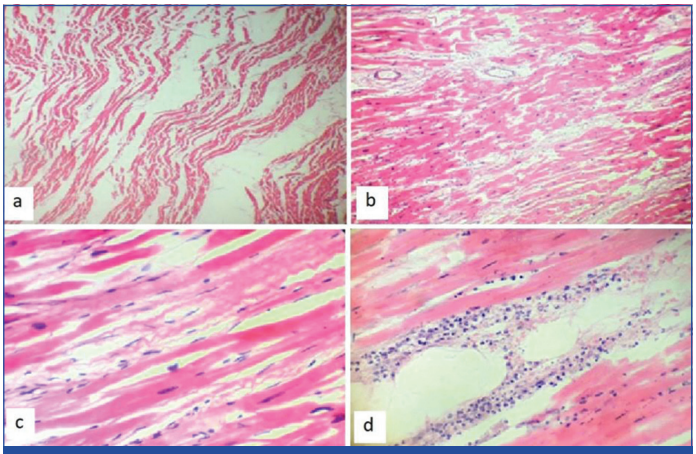
A total of 164 specimens of hearts were included in the study. The distribution of cases was assessed with respect to age and gender. The highest number of cases occurred in the age group of 51-60 years. The age group involved, from 9-80 years. Minimum age of involvement was a nine-year-old male child with a cardiac autopsy showing features of myocarditis. Out of 164 cases, 119 (72.56%) were male and 45 (27.43%) were female, in a ratio of 2.6:1. Male predominance was observed in this study [Table/Fig-1].

Age group (years)	Male (n)	Female (n)	Total	%
0-10	1	0	1	0.60
11-20	5	6	11	6.70
21-30	12	7	19	11.58
31-40	23	5	28	17.07
41-50	27	9	36	21.95
51-60	33	9	42	25.60
61-70	15	8	23	14.02
71-80	3	1	4	2.43
Total	119	45	164	100

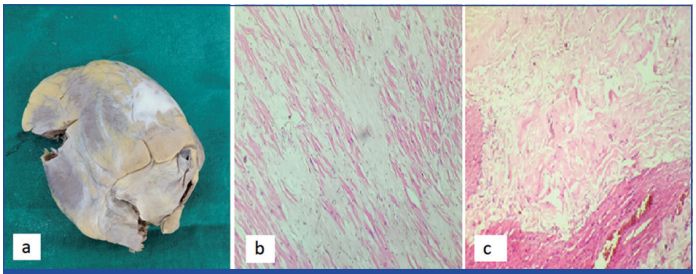
[Table/Fig-1]: Demographic distribution of cardiac cases of sudden death.

Histopathological evaluation showed a wide variety of changes, the most frequent being Acute Myocardial Infarction (AMI) in 70 cases (42.68%) [Table/Fig-2]. Other lesions were HCM in 25 cases (15.24%), dilated cardiomyopathy in four cases (2.43%), multiple lesions in 11 cases (6.7%), AMI with an old infarct in four cases (2.43%), and an old healed infarct in one case (0.6%) [Table/Fig-3], four cases (2.43%) had myocarditis [Table/Fig-4], tuberculous pericarditis with myocarditis in one case (0.6%) [Table/Fig-5], infective endocarditis with vegetations over the mitral and aortic valves in three cases (1.82%) [Table/Fig-6], atherosclerosis, cardiac myxoma [Table/Fig-7], and coronary insufficiency in one case each. Tubercular pericarditis and myocarditis of the heart revealed caseous material over pericardium and myocardium grossly. Histopathological examination showed multiple caseating granulomas. Ziehl-Neelsen staining was done, and the result of the staining was negative. However, due to the presence of specific findings like caseating necrosis and langhans giant cells, the histopathological diagnosis was tubercular pericarditis and myocarditis. Normal histomorphology of the heart was observed in 23 cases (14.02%) [Table/Fig-8].

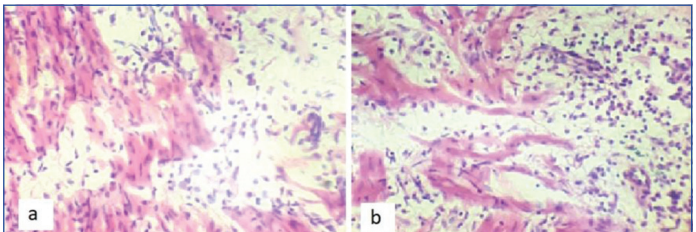
Multiple cardiac lesions were found in 11 cases (6.7%), with combinations of two or more cardiac lesions such as AMI, old healed infarction, HCM, Left Ventricular Hypertrophy (LVH), and thrombus in left anterior descending coronary artery [Table/Fig-9]. Non specific findings were identified in 16 cases (9.75%) that included haemorrhage between muscle fibres, break-up of myocardial fibres, oedema, sparse inflammatory cell infiltration, etc. Due to the lack of specific histological findings, no definitive diagnosis was made.



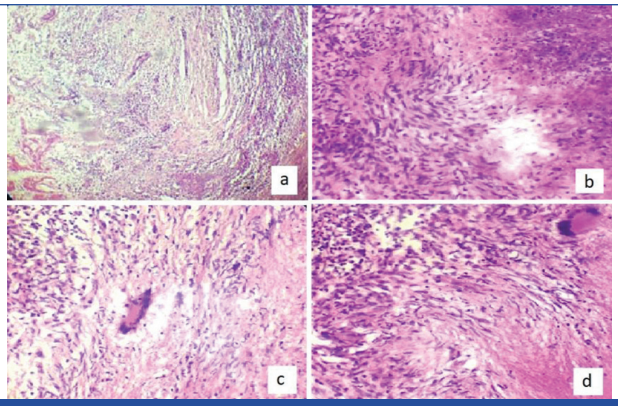
[Table/Fig-2]: Photomicrographs showing features of myocardial infarction at different stages: a) Waviness of myocardial fibres (H&E stain, 10X); b) Fragmentation of myocardial fibres (H&E stain, 10X); c) Coagulative necrosis of myocardial fibres showing hypereosinophilia (H&E stain, 40X); d) Heavy neutrophilic infiltration in-between myocardial fibres (H&E stain, 40X).



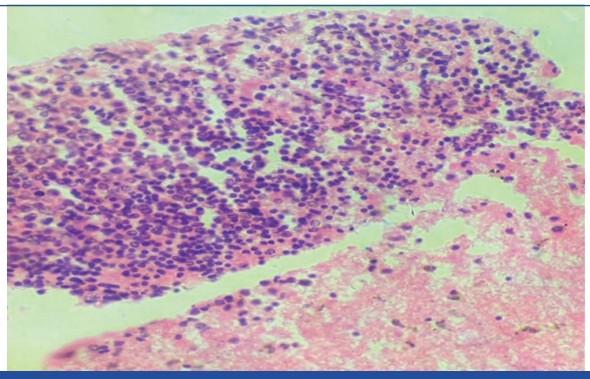
[Table/Fig-3]: Photographs of old healed myocardial infarct: a) Gross appearance showing whitish scar over heart; b) Photomicrograph showing dense fibrous scar replacing cardiomyocytes (H&E stain, 10X); c) Showing extensive myocardial fibrosis (H&E stain, 10X).



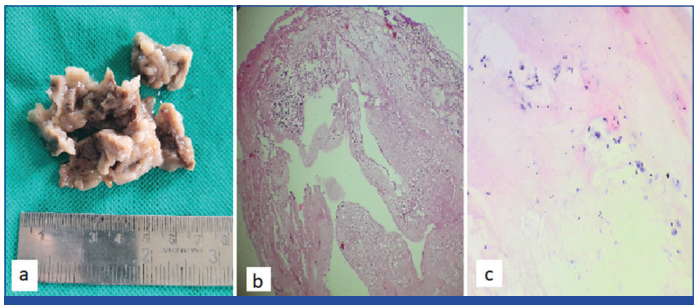
[Table/Fig-4]: Photomicrographs of myocarditis: a) showing interstitial oedema and lymphocytic infiltration in between myocardial fibres (H&E stain, 40X); b) showing oedema in between cardiomyocytes and heavy infiltration of lymphocytes (H&E stain, 40X).



[Table/Fig-5]: Photomicrographs of tuberculous pericarditis and myocarditis: a) Tuberculous granuloma involving myocardium and pericardium (H&E stain, scanner view); b) Granuloma showing collection of epithelioid cells, caseous necrosis and lymphocytes (H&E stain, 40X); c) and d) showing Langhans giant cell along with epithelioid cells and lymphocytes (H&E stain, 40X).



[Table/Fig-6]: Photomicrograph of infective endocarditis reveals infiltration of lymphocytes, macrophages, and a few neutrophils over a fibrinous background (H&E stain, 40X).



[Table/Fig-7]: Photographs of cardiac myxoma: a) Gross picture showing papillary lesion with gelatinous appearance; b) Photomicrograph showing stellate myxoma cells embedded within an abundant ground substance (H&E stain, scanner view); c) Polygonal to stellate cells surrounded by stroma rich in acid mucopolysaccharides (H&E stain, 40X).

Diagnosis	Number of cases	Percentage
Acute Myocardial Infarction (AMI)	70	42.68
Old healed infarct	1	0.60
AMI+Old healed infarct	4	2.43
Tuberculous pericarditis and myocarditis	1	0.60
Myocarditis	4	2.43
Infective endocarditis	3	1.82
Hypertrophic Cardiomyopathy (HCM)	25	15.24
Dilated cardiomyopathy	4	2.43
Cardiac myxoma	1	0.60
Coronary insufficiency	1	0.60
Multiple lesions	11	6.70
Non specific findings	16	9.75
Normal histomorphology	23	14.02
Total	164	100

[Table/Fig-8]: Histopathological spectrum of changes in autopsied hearts and their relative frequency.

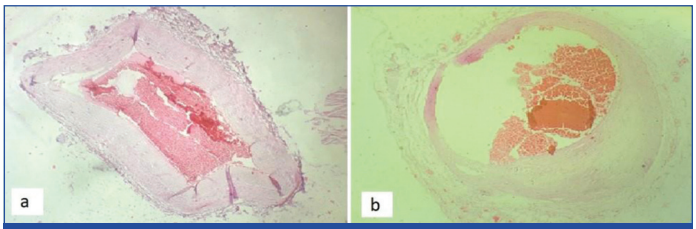
Diagnosis	Number of cases	Percentage
AMI+HCM	7	63.63
AMI+HCM+Old healed infarct	2	18.18
Old healed infarct+HCM	1	9.09
LVH+thrombus in left anterior descending artery	1	9.09
Total	11	100

[Table/Fig-9]: Multiple lesions in heart.
AMI: Acute myocardial infarction; HCM: Hypertrophic cardiomyopathy; LVH: Left ventricular hypertrophy

Out of all IHD cases, 70 cases (82.35%) of recent infarcts, one (1.17%) old infarct, four (4.7%) acute on chronic MIs, and ten (11.76%) mixed lesions were found [Table/Fig-10]. Three vessels involvement were more prevalent, and the left anterior descending coronary artery was the most frequently affected vessel [Table/Fig-11].

Myocardial Infarction (MI)	Number of cases	Percentage
AMI	70	82.35
Old infarct	1	1.17
AMI+Old infarct	4	4.70
AMI+HCM	7	8.23
Old infarct+HCM	1	1.17
AMI+Old infarct+HCM	2	2.35
Total	85	100

[Table/Fig-10]: Types of Myocardial Infarction (MI) (Isolated and mixed).
AMI: Acute myocardial infarction; HCM: Hypertrophic cardiomyopathy



[Table/Fig-11]: Photomicrographs showing left coronary artery thrombosis: a) Near complete obliteration by congestion and thrombus formation (H&E stain, 10X); b) Partial obliteration by thrombus and haemorrhage (H&E stain, 10X).

A total of 39 cases (45.88%) of infarction among IHD patients were found on LVW; 28 cases (32.94%) were found on LVW and Interventricular Septum (IVS); and five cases (5.88%) on Apex+LVW+IVS. On 12 cases (14.11%) where the diagnosis was done microscopically, no noticeable gross changes were visible over the heart [Table/Fig-12].

Areas involved	AMI	AMI with HCM	Chronic IHD	Chronic IHD with HCM	Acute on chronic IHD	Acute on chronic IHD with HCM	Total (%)
Only LVW	30	4	0	1	3	1	39 (45.88)
Only RVW	0	0	0	0	0	0	0
Only IVS	0	0	0	0	0	0	0
LVW and IVS	22	3	1	0	1	1	28 (32.94)
Apex	0	0	0	0	0	0	0
Apex and IVS	1	0	0	0	0	0	1 (1.17)
Apex+LVW +IVS	5	0	0	0	0	0	5 (5.88)
No gross change	12	0	0	0	0	0	12 (14.11)
Total	70	7	1	1	4	2	85 (100)

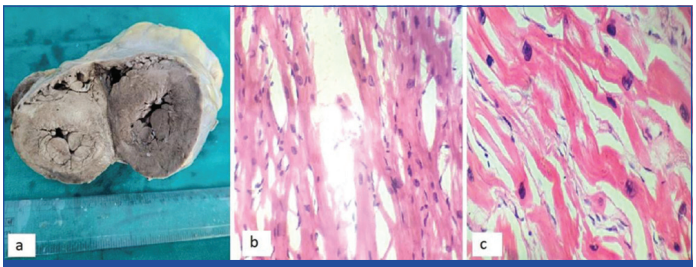
[Table/Fig-12]: Frequency distribution of areas involved in Ischaemic Heart Disease (IHD) (Isolated and mixed lesions).
LVW: Left ventricular wall; RVW: Right ventricular wall; IVS: Interventricular septum; IHD: Ischaemic heart disease; AMI: Acute myocardial infarction; HCM: Hypertrophic cardiomyopathy

Microscopic features of 70 cases of AMI and one case of a healed infarct were evaluated, out of which in 41 cases (57.74%) the age of the infarct was 12-24 hours [Table/Fig-13]. Another 4 cases showed both AMIs and old healed infarcts, in which the age of one recent infarct was 4-12 hours, and the age of the rest of recent infarcts was 12-24 hours. The age of all the old infarcts was more than two months as determined by their histomorphology. Among mixed lesions, out of 7 cases of AMI with HCM, the age of the recent infarct was 12-24 hours in 5 cases and 1-3 days in 2 cases. In one case of AMI with a healed infarct and HCM, the age of the recent infarct was 12-24 hours. The age of old infarcts in a mixed lesion was over two months.

Light microscope	Gross appearance	No of cases	Percentage	Time
Waviness of fibres	None	2	2.81	30 min to 4 hours
Early coagulative necrosis; oedema; haemorrhage	None	10	14.08	4-12 hours
Coagulative necrosis; pyknosis of nuclei; myocyte hypereosinophilia; marginal contraction band necrosis; early neutrophilic infiltrate	Dark mottling	41	57.74	12-24 hours
Coagulation necrosis, loss of nuclei and striations; brisk interstitial infiltrate of neutrophils	Mottling with yellow tan infarct centre	16	22.53	1-3 days
Disintegration of myocytes with dying neutrophils; early macrophages infiltration at infarct border	Hyperemic border; central yellow tan softening	1	1.40	3-7 days
Dense collagenous scar	Scarring complete	1	1.40	>2 months

[Table/Fig-13]: Morphological features of Acute Myocardial Infarction (AMI) and old healed infarct (n=71).

Thirty five cases (21.34%) of HCM, including isolated and mixed lesions, were evaluated in this study. Grossly, the heart revealed a thick wall with myocardial hypertrophy. Microscopically, there was remarkable myocyte hypertrophy along with focal myocyte disarray and interstitial fibrosis [Table/Fig-14].



[Table/Fig-14]: Photographs of Hypertrophic Cardiomyopathy (HCM): a) Gross picture (cross-sectional view) showing thickening of ventricular wall; b) Photomicrograph showing disarray and disorganised branching cardiac myocytes (H&E stain, 40X); and c) Photomicrograph showing enlarged nuclei of cardiac myocytes (H&E stain, 40X).

DISCUSSION

In our study, the age range of 51-60 years had the highest number of cases, which was consistent with the findings of Shah SN et al., study [18]. In the study by Ndoye EHO et al., the age range of 50 to 59 was the most affected [19]. The studies conducted by Nisha M et al., Joshi C and Ding Z et al., reported that the age group of 41-50 years had the highest number of cases [14,15,20]. Khandekar S and Mahadani J, found that the majority of cardiovascular deaths occurred between the ages of 31 and 60 in their study, demonstrating the importance of age as a risk factor for heart disease [16]. The age range with the highest number of cases in the study by Agale SV et al., was 31-40 years [21].

In the present study, there was a remarkable male predominance with 119 (72.56%) of the 164 cases being male and 45 (27.43%) female. Research by Khandekar S and Mahadani J, (128 males, 72 females), Agale SV et al., (male to female ratio of 1.83), Chugh SS (male to female ratio of 2.33), Risgaard B et al., (male to female ratio- 659/234- 2.81), Braggion-Santos MF (male to female ratio- 599/300- 1.99), Ifteni P et al., (male to female ratio- 749/336- 2.23), and other studies likewise revealed a male predominance, showed that SCD was more prevalent in men [16,21-27]. The male to female ratio in this study was 2.6:1, but it was significantly higher in the studies by Nisha M et al., (184 males,16 females), Shah SN et al., Ndoye EHO et al., Ding Z et al., and Shanthi B et al., [14,18,19,20,28].

Histopathological evaluation in the present study showed a wide spectrum of changes, the most prevalent of which was MI, of which 70 cases (42.68%) were AMI. MI caused by atherosclerosis was the most frequent cause of mortality in the study by Khandekar S and Mahadani J [16]. In contrast, Siddique MI et al., showed that the incidence of myocardial lesions was 2.8% in their study [29].

In this study, out of all cardiac lesions, recent infarcts were seen in 70 cases (42.68%), an old infarct in one case (0.6%), and acute on chronic infarcts in four cases (2.43%). Recent infarcts were discovered in 20.8% of cases and old infarcts in 35.1% of cases in the study by Ahmed M et al., [30]. Agale SV et al., in their study, found that recent infarcts were present in 0.35% of cases, while old infarcts were present in 4.15% of cases [21]. Infarcts were found to be recent in 7% of cases, old in 25.5% of cases, and acute on chronic in 3.5% of cases, according to research by Nisha M et al., [14]. Similar to the present study, Agale SV et al., discovered in their study that the left anterior descending coronary artery was the most frequently affected vessel (35.65%), followed by the left circumflex coronary artery (33.61%), and the right coronary artery (30.44%) [21]. The research conducted by Beelwal D et al., and Jha BM et al., revealed similar findings [31,32]. However, Porwal V et al., and Garg M et al., discovered that the left anterior descending artery was most frequently affected, followed by the right coronary artery and the left circumflex coronary artery [33,34].

According to the study by Nisha M et al., involvement of all three vessels occurred most frequently (52%), followed by involvement of one vessel (26.4%) and two vessels (21.6%) [14]. There was no distinct pattern of coronary involvement when compared to other research; however, involvement in three vessels was more prevalent in all the investigations, including the study [35,36].

Microscopic features of 70 cases of recent infarcts and one case of a healed infarct were studied, out of which in 41 cases (57.74%) the age of the infarcts was 12 to 24 hours in the present study. Siddiqui MI et al., found that the microscopic characteristics of IHD included waviness in the fibres, myocyte hypereosinophilia, neutrophil infiltration, reperfusion haemorrhage, formation of granulation tissue, fibrosis, and collagenisation [29].

Among IHD cases, 39 cases (45.88%) were located on the LVW, followed by 28 cases (32.94%) located on the LVW and IVS. In the study by Nisha M et al., 34 of the 72 cases (47.22%) of IHD involved all areas, followed by only the LVW (20.83%) [14]. Shah SN et al., tried to determine the frequency distribution of IHD in various areas, in which 37 of the 67 cases (55.22%) of IHD had involvement of all the areas, followed by involvement of the LVW and IVS (20.89%) [18]. Similar to this study, there was no involvement of the right ventricular wall in the study by Nisha M et al., and Shah SN et al., [14,18]. Findings of the present study were consistent with those that have been published in the literature [37-39].

In 23 cases (14.02%) of clinically diagnosed SCD, no specific finding with normal histomorphology of heart was observed in the present study. Unexplained SCD with no specific findings was identified in 115 cases in the study by Nisha M et al., 6.1% of cases in the study by Ding Z et al., and 22.49% of cases in the study by Agale SV et al., [14,20,21]. In the study by Wu Q et al., out of 1656 cases of SCD, the number of sudden unexplained deaths after the histopathological examination was 251 cases (15.2%) [26].

In this study, tuberculous pericarditis with myocarditis was seen in one case (0.6%). In the study by Agale SV et al., tuberculosis of the heart was observed in two cases (0.69%), one of which showed only pericarditis, and the other had both myocarditis and pericarditis due to tuberculosis [21]. Due to disseminated miliary tuberculosis, other organs were affected in both cases. According to autopsy studies, 2% of Human Immunodeficiency Virus (HIV) patients had heart involvement [40]. Tuberculous myocarditis is rare and occurs as a complication of tuberculosis elsewhere in the body, which can spread directly or through lymphatic and haematogenous dissemination [41].

The term "Hypertrophic Cardiomyopathy" (HCM) refers to a genetic myocardial disease that is characterised by an asymmetric thickening of the LVW without dilation of the cavity and without the presence of any other cardiac or systemic disease that might explain the severity of the heart muscle hypertrophy (e.g., high blood pressure, aortic stenosis) [42]. It has a prevalence of 1:500, or 0.2% of overall population, and affects people of all genders and ethnicities [43]. Thirty five cases (21.34%) of HCM, including both isolated and mixed lesions, were studied in this study, and it was the second most common cardiac lesion. Myocardial hypertrophy and a thick heart wall were grossly visible. Myocyte hypertrophy was seen under the microscope, along with focal myocyte disarray and areas of interstitial fibrosis. Cardiomyopathies accounted for 54.9% (n=129) of the cases in Ndoye EHO et al., study, with 90% of dilated cardiomyopathies (n=116) and 10% of hypertrophic cardiomyopathies, with a male predominance [19]. In their study, Ahmed M et al., reported that five (8%) young individuals (age range 21-30 years) had gross and microscopic features of HCM, which was consistent with findings from two previous studies by Mckenna WJ et al., and Nocod P et al., [30,44,45]. According to the study by Matsumori A et al., Congestive Heart Failure (CHF) and arrhythmias were the major causes of death for Dilated Cardiomyopathy (DCM) and Hypertrophic Cardiomyopathy (HCM), respectively [46]. According to the studies conducted by Ahmed M et al., and Kasturi AS et al., 8% and 7.6% of cases with HCM, respectively, were reported in each study [30,36].

Four cases (2.43%) of myocarditis, excluding tubercular pericarditis with myocarditis, were reported in this study. Myocarditis was the most frequent lesion in the paediatric group in this study. In the study by Winkel BG et al., myocarditis accounted for 13% of all autopsied SCD and was the most common structural cause of cardiac death in children and adolescents aged 1-18 years [27]. In the study by Agale SV et al., it was revealed that only myocarditis was present in 20 (6.92%) cases, while myocarditis and pericarditis were present in 28 (9.69%) cases [21]. Myocarditis was also reported by Kramer MR et al., (29%) and Drory Y et al., (22%) [47,48]. But Joshi C and Shanthi B et al., and Basso C et al., reported a lower incidence that was 9%, 4% and 10% of cases, respectively, in their studies [15,28,49]. Myocarditis has a relatively low incidence of 0.6%, according to Waller BF et al., [50].

One case (0.6%) of cardiac myxoma in left atrium was found in the present study. The most common primary benign tumour of the heart is cardiac myxoma, and it is mostly seen in the left atrium. Histologically, myxomas are made up of globular or stellate myxoma cells that are embedded in a ground substance that is largely made up of acid mucopolysaccharides. Haemorrhage and mononuclear inflammation are mostly present [37].

Limitation(s)

Since, the current study was conducted in a tertiary care centre and convenient sampling was done, the results were not generalisable to the general population.

CONCLUSION(S)

This study revealed, a significant number of SCDs among cases in the age group of 41-60 years. MI was identified as the most common cause of SCD. Histopathological studies provide the best information for a better understanding of cardiovascular diseases. This study revealed the major heart-related health issue that exists in the society and the necessity to promote public awareness in order to prevent SCD and increase life expectancy.

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PARTICULARS OF CONTRIBUTORS:
1. Assistant Professor, Department of Pathology, PRM Medical College and Hospital, Baripada, Odisha, India.
2. Associate Professor, Department of Pathology, PRM Medical College and Hospital, Baripada, Odisha, India.
3. Associate Professor, Department of Pathology, PRM Medical College and Hospital, Baripada, Odisha, India.
4. Professor, Department of FMT, PRM Medical College and Hospital, Baripada, Odisha, India.
5. Tutor, Department of Pathology, PRM Medical College and Hospital, Baripada, Odisha, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Rashmi Rekha Mahapatra,
Assistant Professor, Department of Pathology, PRM Medical College and Hospital,
Rangamatia, Baripada, Mayurbhanj-757107, Odisha, India.
E-mail: rashmimahapatra123@gmail.com

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