DOI: 10.7860/NJLM/2024/70811.2869

Original Article

Pathology Section

Autopsy Pathology of Heart and Coronary Vessels in Sudden Death: A Cross-sectional Study from Southern India

MARY DIANA VINCENT[1], JAYASREE RAMAN[2], UNMESH AYYANCHIRA KUMARAN[3], SWAPNA SURESH[4]

ABSTRACT

Introduction: Cardiac diseases emerge as the primary culprits behind sudden and unexplained deaths. Morphological evaluation through autopsy is invaluable in establishing the prevalence of cardiac lesions and coronary atherosclerosis within the general population. It provides healthcare researchers with valuable insights to develop preventive measures and early detection strategies crucial for improving survival rates.

Aim: To estimate the prevalence of cardiac lesions in postmortem specimens of the heart from autopsies of sudden and unexplained deaths.

Materials and Methods: The present cross-sectional study was conducted in the Department of Pathology, Government Medical College (a public sector hospital), Cochin, Kerala, India, from January to December 2021 on 140 specimens of the heart. Medicolegal autopsies were performed in the Department of Forensic Medicine, with specimens forwarded to the Pathology Department for histopathological examination. Hearts dissected by the modified Virchow's method were examined for gross pathology in the cardiac wall, chambers, valves, coronaries and aorta. Microscopy findings were documented and the grading of coronary atherosclerosis was done using the Modified American Heart Association (AHA) criteria. Proportions were analysed from the compiled data using a Chi-square test. Statistical analysis was done using Statistical Package for the Social Sciences (SPSS) software version 21.0 software.

Results: In the study encompassing 140 adult autopsies, the mean age of the study population was 45.98 with a Standard Deviation (SD) of 15.82 years and the male to female ratio was 3.5:1. The prevalence of cardiac lesions was as follows: coronary artery atherosclerosis (80.70%), Ischaemic Heart Disease (IHD) (44.28%), cardiac hypertrophy and hypertension (10%), pericarditis (6.42%) and myocarditis (3.50%). The male-to-female ratio for coronary atherosclerosis was 4.4:1, with triple vessel disease observed in 42.90% of cases. Atherosclerosis severity grades included early non atheromatous lesions (19.3%), Pathological Intimal Thickening (PIT) (23.6%), and advanced atherosclerotic lesions (37.80%). Among cases associated with Coronavirus Disease 2019 (COVID-19)-related deaths (10.7%), changes noted included myocarditis, microhaemorrhages, oedema, capillary dilatation, fibrin deposition and inflammatory infiltrates.

Conclusion: The present study revealed a high prevalence of coronary atherosclerosis affecting all three vessels, particularly among the younger population. This underscores the significance of screening for cardiovascular risk factors and implementing preventive measures from an early age.

Keywords: Atherosclerosis, Coronary arteries, Ischaemic heart disease, Virchow's method

INTRODUCTION

The Global Burden of Disease study age-standardised estimates describe that a quarter of deaths in India are attributable to Cardiovascular Diseases (CVD) [1]. The rates of CVD vary markedly, with the highest rates found in the states of Kerala, Punjab and Tamil Nadu [2]. The most common cardiac lesions include coronary atherosclerotic disease and IHD, followed by hypertensive heart disease, hypertrophic cardiomyopathy, myocarditis, infective endocarditis, rheumatic heart disease and aortic dissection. Coronary insufficiency due to atherosclerotic narrowing and acute plaque changes in atheroma, thrombosis and vasospasm of coronary arteries can lead to the clinical syndromes of IHD. The clinical presentations of IHD are angina pectoris, myocardial infarction, chronic IHD with heart failure and sudden cardiac death [3]. Atherosclerosis is a progressive degenerative inflammatory disease characterised by the accumulation of lipids, both intracellular and extracellular, macrophages, T cells, proteoglycans, collagen and calcium in arterial vessels [4]. The AHA criteria guidelines used for grading atherosclerotic lesions follow a temporal sequence of histological morphologies in the development and progression of the disease, designated with numerals [5]. The modified AHA-recommended classification by Virmani R et al., uses a simplified classification based on morphological description as described in [Table/Fig-1] [6].

Histological terms	Morphology	Gross appearance. Thrombus +/-
Non atherosclerotic lesions		
Intimal thickening	Intimal Smooth Muscle Cells (SMCs)+, No lipid/foam cells	No thrombus.
Intimal xanthoma, or "fatty streak"	Foam cells without necrotic core	
Advanced atherosclerotic lesions		
Pathological Intimal Thickening (PIT)	SMCs, extracellular lipid without necrosis	Intermediate lesion. No thrombus
PIT+erosion	Luminal thrombosis; plaque same as above	Thrombus mural, infrequently occlusive
Fibrous Cap Atheroma (FCA)	Well-formed necrotic core with fibrous cap	No thrombus
+Erosion	Luminal thrombosis; no communication with necrotic core	Thrombus mural, infrequently occlusive
Thin Fibrous Cap Atheroma (TFCA)	Thin fibrous cap, macrophages, lymphocytes, rare SMCs, necrotic core	No thrombus
Plaque rupture	Fibroatheroma cap disruption; thrombus communicates with necrotic core	Thrombus occlusive
Calcified nodule	Eruptive nodular calcification, fibrocalcific plaque	Non occlusive thrombus
Fibrocalcific plaque	Collagenised calcified plaque, stenosis, inflammation, necrotic core may be+	No thrombus

[Table/Fig-1]: Modified AHA classification based on morphological description [6].

Myocarditis can present as asymptomatic cases to cardiogenic shock and sudden death. The Dallas criteria provide consensus-derived histologic criteria for the diagnosis of myocarditis: an inflammatory infiltrate of the myocardium with necrosis and/or degeneration of adjacent myocytes not typical of ischaemic damage [7]. The World Health Organisation (WHO) defines acute myocarditis (Marburg criteria) as a minimum of 14 infiltrating leucocytes per mm^2, preferably T cells, with necrosis [8]. The Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) virus, which emerged in late 2019, has spread across the world, causing more than ten million infections and over half a million deaths [9]. COVID-19 patients with pre-existing cardiovascular co-morbidities had a case fatality rate of 10.5% compared with an overall cohort fatality rate of 2.3% [10]. Fibrin deposition, capillary dilatation and microhaemorrhage were observed in the myocardium of COVID-19-associated coagulopathy and death [11].

Considering the magnitude of the problem of CVD among the population in a geographically diverse country like India, regional data are essential for surveillance, prevention and management. The present study aimed to describe the histomorphology patterns of cardiac lesions in sudden deaths in the region of Central Kerala, India and to grade atherosclerotic changes using the modified AHA classification of atherosclerosis.

MATERIALS AND METHODS

The present cross-sectional study was conducted in the Department of Pathology, Government Medical College (a public sector hospital), Cochin, Kerala, India, from January to December 2021 on 140 specimens of the heart. Out of the 381 medicolegal autopsies of adults, which included individuals of all age groups, genders and ethnicities, performed in the Department of Forensic Medicine, 146 specimens were sent for pathology examination. The study was approved by the Institutional Ethics Committee, with the IEC number being 53/2021.

Inclusion criteria: Specimens of whole hearts from medicolegal adult autopsy cases of sudden unexpected deaths above 18 years of age received in pathology were included in the study.

Exclusion criteria: Bits of the heart and autolysed samples were excluded; therefore, six samples were not included in the study, resulting in a total of 140 cases being included.

Sample size calculation: According to a study conducted by Nisha M et al., the proportion of cardiac pathology among autopsy specimens was 56%. The sample size calculation formula used was $N=4pq/d^2$ [12]. The minimum sample size required for the present study was 140 (139.68), where, 4-square of the Z value of the alpha error at 5%, which is 1.96 (approximated to 2), P= proportion from the previous study (56%), Q= 100-P (44%) and D= relative precision of 15% (8.4).

Study Procedure

The details of the autopsy and clinical history were collected from the requisition forms. Formalin-fixed specimens were dissected using an inflow-outflow method, emptied of post-mortem clots and weighed. Measurements of the thickness of the right ventricular wall, left ventricular wall and interventricular septum were recorded. The valves were examined for stenosis and calcification. Areas of myocardial ischaemia, recent or old, their locations and sizes were recorded. The ascending aorta was checked for dilatation, thickening, or atheromatous plaque. The coronary arteries were examined for stenosis and sectioned at regular intervals of 4-5 mm. Sections were taken from the right and left ventricular walls, Right Coronary Artery (RCA), Left Anterior Descending (LAD), Left Circumflex coronary Artery (LCX), and the stump of the aorta for microscopic examination. Tissues were processed using paraffin processing, and 4 µm sections were stained with Haematoxylin and Eosin (H&E). The changes in the myocardium in acute ischaemia included wavy fibres, neutrophil infiltrates, oedema, contraction band necrosis and granulation tissue. Areas of fibrosis with loss of myocytes and mononuclear cell infiltrate were the features identified in cases of chronic IHD. In COVID-19 autopsies, changes of myocarditis and coagulopathy-associated fibrin deposition, capillary dilatation and microhaemorrhages were recorded.

Coronary vascular atherosclerotic changes were classified as per the modified AHA classification based on morphologic descriptions as depicted in [Table/Fig-1]. Lesions were categorised into three main groups: 1) Non atherosclerotic/ early intimal lesions, including intimal thickening and intimal xanthoma with foamy cells, fatty streaks and extracellular lipids; 2) PIT are intermediate lesions similar to raised fatty streaks mentioned in the AHA classification; 3) The progressive/Advanced atherosclerotic lesion included PIT±erosion, Fibrous Cap Atheroma (FCA)±erosion, Thin Fibrous Cap Atheroma (TFCA), calcified nodule and fibrocalcific plaque.

STATISTICAL ANALYSIS

Statistical analysis was done using SPSS software version 21.0. Qualitative variables were summarised using frequency and percentage. Quantitative data were summarised using the mean and SD.

RESULTS

A total of 140 cases in the present study were in the age range from 18-92 years, with a mean±SD age of 45.98±15.82 years. Within the study group, 37 (26.4%) cases fell within the age bracket of 41-50 years. The male-to-female ratio was 3.5:1, including 109 (77.9%) males and 31 (22.1%) females. The age distribution of the study population is shown in [Table/Fig-2].

Age group (years)	Number of cases (n)		n (%)
	Females	Males	
18-20	1	3	4 (2.9)
21-30	3	18	21 (15)
31-40	10	25	35 (25)
41-50	8	29	37 (26.40)
51-60	2	18	20 (14.3)
61-70	0	11	11 (7.9)
71-80	3	3	6 (4.3)
81-90	2	2	4 (2.9)
91-100	2	0	2 (1.4)
Total (N)	31	109	140 (100)

[Table/Fig-2]: Age distribution of study population in age groups (N=140).

The cause of death was unknown in the majority of cases at autopsy. Collapse and death were the histories obtained in 117 (83.60%) cases. Histories of known cardiac disease were available in 8 (5.80%) cases. Co-morbidities recorded were diabetes mellitus in 5 (3.50%) and hypertension in 6 (4.30%) cases, with SARS-CoV-2 infection established by positive RT-PCR testing in 15 (10.70%) cases. On gross examination, the weight of the heart ranged from 125 g to a maximum of 625 g, with a mean weight of 327 g. An increase in heart weight to more than 400 g was seen in 20 (18.35%) males and to more than 350 g in 4 (12.90%) females.

The spectrum of cardiac pathology observed in the present study is given in [Table/Fig-3]. Coronary atherosclerosis was present in 113 (80.70%) cases, IHD in 62 (44.28%), and cardiac hypertrophy and hypertension were seen in 14 (10%) cases as the most common lesions. The male-to-female ratio for coronary atherosclerosis was 4.4:1, with triple vessel disease observed in 42.90% of cases. There were 5 (3.50%) cases of myocarditis, two of which were seen in association with COVID-19 positive cases. Of the 7 (5%) cases of pericarditis, two cases were granulomatous pericarditis associated with disseminated tuberculosis. One (0.71%) case of sickle cell disease showed vaso-occlusive crisis involving coronary vessels and myocardial ischaemia. One (0.71%) case of cardiac amyloidosis had hypertrophic cardiomyopathy and ischaemia. One case (0.71%) of infective endocarditis had mitral stenosis. Of total,

Histopathological findings	No. of cases, n	Percentage (%)
Coronary atherosclerosis	113	80.70
Ischaemic Heart Disease and Hypertension	62	44.28
Hypertesion/left ventricular hypertrophy	14	10
Myocarditis	5	3.50
Pericarditis (Tuberculous-2, Non tuberculous-5)	7	5.00
Infective endocarditis /mitral stenosis	1	0.71
Amyloidosis	1	0.71
Sickle cell disease	1	0.71
No significant pathology	27	19.3

[Table/Fig-3]: The histological spectrum of cardiac pathologies in the study (N=140).

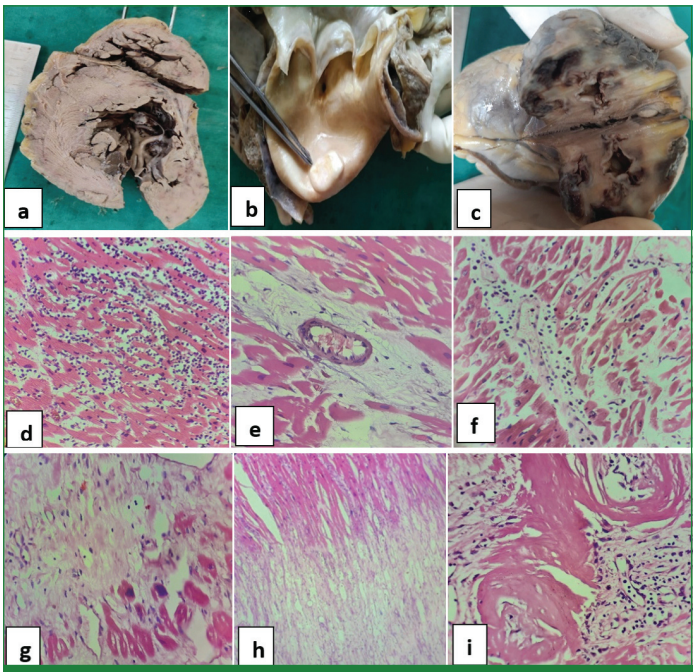
27 (19.3%) cases showed no significant pathology in the heart. The spectrum and prevalence of lesions in 10-year age groups are shown in [Table/Fig-4].

The youngest case of AMI occurred in a 27-year-old male, while the oldest case involved a 66-year-old male, with a mean age of 44.33 years (SD: 10.48 years). There were 20 (33.33%) cases below 40 years of age. The maximum number of IHD cases were seen in the age group of 31-40 years, with 16 cases including seven cases of AMI. Other than coronary vascular disease, sickle cell disease, amyloidosis and endocarditis were identified as causes of myocardial ischaemia in the present study. Atherosclerotic changes in coronary vessels were found in 113 (80.70%) cases in the study. Gender-wise prevalence was 92/109 (84.4%) males and 21/31 (67.74%) females. Above 40 years of age, aortic atherosclerosis was seen in 77 out of 80 (96.25%) cases, and coronary atherosclerosis was seen in 72 out of 80 (90%) cases. The prevalence of atherosclerosis increased with advancing age in both genders. The prevalence of coronary atherosclerosis in both genders before and after 40 years is shown in [Table/Fig-6].

Age range (years)	Normal	Coronary atherosclerosis, n (%)	Ischaemic heart disease, n (%)	HT/LVH (n)	Myocarditis (n)	Pericarditis (n)	Other; IE/SCD/Amyloidosis (n)
18-20 (n=4)	3	1 (25)	-	-			SCD-1
21-30 (n=21)	8	13 (61.9)	5 (23.80)	-	1	1	
31-40 (n=35)	7	28 (80)	16 (45.71)	5	1		IE-1
41-50 (n=37)	6	31 (83.78)	14 (37.83)	4	1	1 (TB)	Amyloid-1
51-60 (n=20)	2	18 (90)	10 (50)	2	2	1 (TB)	
61-70 (n=11)	-	11 (100)	10 (90.91)	1		2	
71-80 (n=6)	1	5 (83.33)	2 (33.33)	1		1	
81-90 (n=4)	-	4 (100)	3 (75)	1		1	
91-92 (n=2)	-	2 (100)	2 (100)				-
Total (N=140)	27 (19.30)	113 (80.7)	62 (44.27)	14 (10)	5 (3.50)	7 (5)	3 (2.14)

[Table/Fig-4]: Prevalence of spectrum cardiac lesions in age-wise distribution (N=140).
HT: Hypertension; LVH: Left ventricular hypertrophy; TB: Tuberculous; SCD: Sickle cell disease; IE: Infective endocarditis

The overall prevalence of IHD was 62 (44.28%) in the present study. There were 23 (16.42%) cases of Acute Myocardial Infarction (AMI). Old infarcts were seen in association with acute ischaemia in 8 out of 23 cases. In 39 (27.86%) cases, changes of chronic IHD were identified. Gross and microscopy images are shown in [Table/Fig-5a-i].

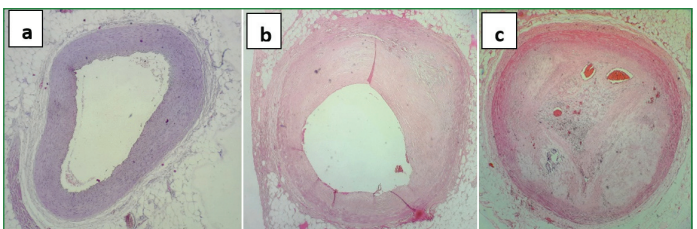


[Table/Fig-5]: a) Gross image of Left Ventricular Hypertrophy; b) Atheroma Aorta; c) Tuberculosis of Heart; d) Lymphocytic Myocarditis (H&E, 40x); e) Vaso-occlusive Crisis- Arteries Clogged By Sickle Cells (H&E, 40x); f) Eosinophilic Myocarditis (H&E, 40x); g,h) Myocardial Infarction- Necrosed myocardial fibres with acute inflammatory infiltrate (H&E, 10x and 40x); i) Amyloid deposition in the wall of coronary arterioles with atrophic myocytes (H&E, 40x).

Gender	Coronary atherosclerosis		Total
	Age <40 years	Age >40 years	
Females (n=31)	7 (22.58)	14 (45.16)	21/31 (67.74)
Males (n=109)	32 (29.36)	60 (55.04)	92/109 (84.40)
Total (N=140)	39/60 (65)	74/80 (92.50)	113/140 (80.70)

[Table/Fig-6]: Prevalence of coronary atherosclerosis below and above 40 years of age.
Results are presented as n (%)

Out of the 113 cases of coronary atherosclerosis, triple vessel disease was seen in 60 (42.90%) cases, two vessels were involved in 39 (27.9%) cases and single vessel disease was seen in 14 (10%) cases. No significant atherosclerosis was found in 27 (19.3%) of cases. As per the modified AHA criteria in [Table/Fig-1], the atherosclerotic intimal lesions of major coronary arteries were grouped into three severity grading groups: 1) Non atheromatous; 2) PIT (intermediate lesion); and 3) advanced lesions. LAD was the most common coronary artery involved in 113 (80.71%) cases with advanced atherosclerotic lesions in 71 (62.83%), followed by RCA with 57 (50.44%). Images and grading of coronary atherosclerosis based on modified AHA criteria are shown in [Table/Fig-7,8], respectively.



[Table/Fig-7]: Modified AHA grade of coronary atherosclerosis (H&E, 40X): a) Non atheromatous lesions; b) Pathological Intimal Thickening (PIT) (intermediate lesion/ preatheroma); c) Progressive/Advanced atherosclerotic lesions.

Morphology	LAD%	LCX%	RCA%
Non atheromatous intimal lesions	27 (23.89)	51 (47.66)	39 (34.51)
Pathological intimal thickening (intermediate lesion/preatheroma)	15 (13.27)	8 (7.47)	17 (15.04)
Progressive/advanced atherosclerotic lesions	71 (62.83)	48 (44.86)	57 (50.44)
Total	113/140 (80.71)	107/140 (76.43)	113/140 (80.71)

[Table/Fig-8]: Severity grading of atherosclerotic lesions in each coronary vessel based on modified AHA criteria.
Results are presented as n (%)

There were 15 (10.7%) confirmed SARS-CoV-2 deaths. Out of the 15 COVID-19 associated deaths, 11 (73.33%) cases had coronary atherosclerosis, 3 (20%) had chronic IHD and 3 (20%) cases showed AMI. Other histological findings noted in these cases were myocarditis, capillary dilatation, interstitial oedema, microhaemorrhage, inflammatory infiltrates and capillary fibrin.

DISCUSSION

In sudden and unexplained deaths where there is no history of previous CVDs, coronary occlusion with a plaque change in an atheromatous lesion can be the cause of death, irrespective of age. The incidence of coronary atherosclerosis in the Indian population is on the increase, leading to sudden deaths in the past few decades. This increase can be attributed to dietary habits, physical inactivity, the increased incidence of lifestyle diseases, diabetes and hypertension.

In the present study of adult medicolegal autopsies, the maximum number of cases, n=37 (26.4%) were in the age group 41-50 years. There were 109 (77.9%) males and 31 (22.1%) females, with a male to female ratio of 3.5:1. The most common age group observed in a study by Rao D et al., was 50-60 years [13]. Joseph A et al., had a male to female ratio of 6.9:1 in their study [14]. Rani E et al., reported a male to female ratio of 11:1, observing that more cases were in males, indicating that sudden natural death from all causes (cardiac and non cardiac) was common in men [15].

In the present study, out of 140 cases, 113 (80.7%) had coronary artery atherosclerosis in one or more major vessels. Atherosclerotic heart disease (80.7%) was the most common pathology observed, followed by IHD (44.28%), myocardial hypertrophy (10%), myocarditis (3.50%), pericarditis (3.57%), tuberculosis pericarditis (1.42%), infective endocarditis (0.71%) and cardiac amyloidosis (0.71%). A comparison of the prevalence of histopathological findings in the heart and coronaries in different studies is shown in [Table/Fig-9] [12,16-18].

Atherosclerotic coronary artery disease was reported as the leading cause of death in most of the studies [12,17-19]. Garg S et al., reported the lowest percentage (55.3%) of deaths due to atherosclerotic CAD [16]. The percentage of coronary atherosclerosis in different studies is shown in [Table/Fig-10] [12,16-19].

In the authors observation of coronary atherosclerosis, the non atheromatous intimal lesions were present in 23.89% of cases, while the intermediate and advanced lesions together constituted 76.10% in the left anterior descending coronary artery. Triple vessel involvement, two-vessel and single-vessel involvement were found in 42.9%, 27.9%, and 10% of cases, respectively. Nisha M et al., reported three-vessel disease in 56.94% of cases [12]. In the present study, the degree of involvement of atherosclerosis in LAD, LCX, and RCA were 72.14%, 57.14% and 65.71%, respectively. Bhanvadia VM et al., observed similar findings that the degree of atherosclerosis in LAD, LCA and RCA was 42%, 40% and 39%, respectively [20]. Sudha ML et al., observed that LAD was the most common site for atheromatous plaques (47%) [21]. Findings by Thej MJ et al., showed that LAD (60%), followed by RCA (50%) and LCA (42.5%), was the pattern of involvement [22]. In the present study, the prevalence of non atherosclerotic early intimal lesions, such as intimal thickening and fatty streaks according to the modified AHA classification, is examined alongside atherosclerotic lesions. However, the criteria utilised in other studies are not explicitly delineated.

The percentage of different forms of IHD were as follows: AMI in 10.71%, AMI with changes of chronic IHD in 5.71% and chronic IHD in 27.86%. Similar results were reported in studies by Farb A et al., Friedman M et al., Davies MJ and Thomas A, who classified MI into two categories as, recent and old MI [23-25]. Farb A et al., reported old MI in 41% of cases and AMI in 10% of cases [23]. Myocarditis and hypertrophic cardiomyopathy as causes of death showed a high degree of variation in different studies. The highest percentage of myocardial hypertrophy was reported by Joshi C (52.7%), whereas the present study showed 10% [17]. The prevalence of myocarditis was less than 3.5% in the present study, which was similar to the data in other studies [12,16,18]. Joshi C reported a higher percentage of myocarditis at 9.56% [17].

Histopathological findings	Nisha M et al., [12], Haryana	Garg S et al., [16], Haryana	Joshi C, [17] Raipur	Verma R et al., [18], Haryana	Present study, Kerala
Year of the study	2011	2018	2016	2021	2024
Coronary atherosclerosis	71%	55.3%	64.34%	61%	80.7%
Ischaemic heart disease	35.9	14.1%	28.69%	34.6%	44.28%
i) Chronic IHD	25.5%	4.25%	-	21.6%	27.86%
ii) Acute MI	7%	9.92%	-	8.5	10.71%
iii) Acute on chronic IHD	3.4%	-	-	4.5	5.71%
Myocardial hypertrophy	2.5%	7.09%	52.17%	2.3%	10%
Myocarditis	1.5%	3.5%	9.56%	1.6%	3.50%
Pericarditis (non TB)	0.5%	2.8%	0.86%	2.7%	3.57%
Pericarditis tuberculous	0.5%	-	-	0.7%	1.42%
Infective endocarditis/ valve lesions	1.61%	0.07%	-	0.2%	0.71%
Vaso-occlusive crisis-sickle cell disease	-	-	5.21%	-	0.71%

[Table/Fig-9]: Comparison of prevalence of histopathological findings in heart and coronaries in different studies [12,16-18].
IHD: Ischaemic heart disease; MI: Myocardial infarction; TB: Tuberculosis

No.	Study	Place, year	% of coronary atherosclerosis
1	Nisha M et al., [12]	Haryana, India 2011	71.00%
2	Kasthuri AS et al., [19]	Bengaluru, India 2002	76.92%
3	Garg S et al., [16]	Haryana, India 2018	55.3%
4	Joshi C [17]	Raipur, India 2016	64.34%
5	Verma R et al., [18]	Haryana, India 2021	61.00%
6	Present study	Kerala, India 2024	80.70%

[Table/Fig-10]: Comparison of prevalence of coronary atherosclerosis in different studies [12,16-19].

A comparison of the prevalence of histopathological findings in the heart and coronaries in different studies is shown in [Table/Fig-10].

In the current study, cardiac pathology in SARS-CoV-2 virus infection included coronary atherosclerosis in 11/15 (73.33%) cases and AMI in 3/15 (20%) cases. Myocardium in COVID-19 infection showed lymphocytic myocarditis in two (13.33%) cases, capillary dilatation and interstitial oedema in 11 (73.33%) cases and interstitial fibrosis in 9 (60%) cases. The cardiac findings associated with COVID-19 infections in the present study were non specific and mirrored the observations made by Sang CJ et al., [26]. In their study focusing on the cardiac pathology of 50 COVID-19-related deaths, they reported acute myocardial ischaemia in eight (16%) patients, lymphocytic inflammatory infiltrates in eight (16%) patients and focal myocarditis in two (4%) patients [26]. Ferrer-Gómez A et al., in a series of 30 COVID-19 deaths, observed coronary atherosclerosis as the most frequent histopathological finding in eight (26.7%) patients and myocarditis in two (6.66%) cases. Cardiac pathology was only modest in most patients with severe COVID-19 [27].

Limitation(s)

Information regarding the clinical details and risk factors contributing to CVDs could not be obtained, which was a limitation.

CONCLUSION(S)

In the current study, a notable rise in the prevalence of coronary atherosclerosis was observed with advancing age. Three-vessel disease emerged as the predominant finding, with LAD artery involvement being the most frequently encountered among the major coronaries. Particularly striking was the elevated prevalence of atherosclerosis observed in the study group, affecting a relatively younger demographic without any previous history of CVD. While the current study encompassed a limited number of cases, it underscores the early emergence and escalating incidence and intensity of atherosclerotic lesions within the south Indian population. Additionally, it underscores the necessity for Institutional screening initiatives and early-age preventive and management strategies against atherosclerosis. In SARS-CoV-2 deaths, COVID-19-related cardiac changes seen were inflammatory changes, microvasculopathy-associated changes and myocarditis.

REFERENCES

[1] Prabhakaran D, Jeemon P. Global burden of cardiovascular disease cardiovascular diseases in India. J Am Heart Assoc. 2016;133(16):1605-20.
[2] Kumar AS, Sinha N. Cardiovascular disease in India: A 360-degree overview. Med J Armed Forces India. 2020;76(1):01-03.
[3] Kumar V, Abbas A, Aster J, Turner J. Robbins & Cotran Pathologic Basis of Disease Tenth Edition. Elsevier. 2021; P. 112-112.
[4] Stary HC, Chandler AB, Dinsmore RE, Fuster V, Glagov S, Insull W, et al. A Definition of advanced types of atherosclerotic lesions and a histological classification of atherosclerosis. Arteriosclerosis, Thrombosis, and Vascular Biology. 1995;15(9):1512-31. Doi:10.1161/01.atv.15.9.1512.
[5] Stary HC. Arteriosclerosis, thrombosis, and vascular biology. natural history and histological classification of atherosclerotic lesions. Arterioscler Thromb Vasc Biol. 2000;20(5):1177-78. Doi: 10.1161/01.ATV.20.5.1177.
[6] Virmani R, Kolodgie FD, Burke AP, Farb A, Schwartz SM. Lessons from sudden coronary death. Arterioscler Thromb Vasc Biol. 2000;20(5):1262-75. Doi:10.1161/01.ATV.20.5.1262
[7] Mason JW, Trehan S, Renlund DG. Myocarditis. In: Cardiovascular Pathology. 5th edition.
[8] Pfeifer JD, Dehner LP, Humphrey PA. The Washington Manual of Surgical Pathology. 3rd edition. Philadelphia: Wolters Kluwer Health; 2020: p. 543-606.
[9] Chen G, Wu D, Guo W, Cao Y, Huang D, Wang H, et al. Clinical and immunological features of severe and moderate coronavirus disease 2019. J Clin Invest. 2020;130(5):2620-29.
[10] Team NCPERE. Vital Surveillances; The epidemiological characteristics of an outbreak of 2019 Novel Coronavirus Disease (COVID-19). China CDC Wkly. 2020;2(8):113-22.
[11] Haslbauer J, Tzankov A, Mertz K. Characterisation of cardiac pathology in 23 autopsies of lethal COVID-19. J Pathol CR. 2021;7(4):326-37.
[12] Nisha M, Bhawna S, Sumiti G, Amrita D, Sunita S, Rajeev S. Histomorphological spectrum of various cardiac changes in sudden death: An autopsy study. Iran J Pathol. 2011;6(4):179-86.
[13] Rao D, Sood D, Pathak P, Dongre2, Sudhir.D. A cause of sudden cardiac deaths on autopsy findings; A four-year report. J Emerg Med. 2014;2(1):12-17.
[14] Joseph A, Ackerman D, Talley JD, Johnstone J, Kupersmith J. Manifestations of coronary atherosclerosis in young trauma victims-an autopsy study. J Am Coll Cardiol. 1993;22(2):459-67.
[15] Rani E, Kumar S, Mehrolia V. Morphological patterns in heart diseases- An autopsy study. Int J Curr Adv Res. 2018;6(8):5391-93.
[16] Garg S, Hasija S, Sharma P, Kalhan S, Saini N, Khan A. A histopathological analysis of prevalence of various heart diseases: an autopsy study. Int J Res Med Sci. 2018;6(4):1414-18.
[17] Joshi C. Postmortem study of histopathological lesions of heart in cases of sudden death- An incidental findings. J Evid Based Med Heal. 2016;3(06):184-88.
[18] Verma R, Singh S, Marwah N, Pawar R, Rana D. Histopathological array of cardiac lesions: An autopsy-based study in a tertiary care centre. IP Arch Cytol Histopathology Res. 2021;6(3):173-80.
[19] Kasthuri AS, Handa A, Niyogi M, Choudhury JC. Sudden death: A clinicopathological study. J Assoc Physicians India. 2002;50:551-53.
[20] Bhanvadia VM, Desai NJ, Agarwal NM. Study of coronary atherosclerosis by Modified American Heart Association Classification of Atherosclerosis-an autopsy study. J Clin Diagnostic Res. 2013;7(11):2494-97.
[21] Sudha ML, Sundaram S, Purushothaman KR, Kumar PS, Prathiba D. Coronary atherosclerosis in sudden cardiac death : An autopsy study. Indian J Pathol Microbiol. 2010;52(4):486-89.
[22] Thej MJ, Kalyani R, Kiran J. Atherosclerosis in coronary artery and aorta in a semi-urban population by applying modified American Heart Association classification of atherosclerosis: An autopsy study. J Cardiovasc Dis Res. 2012;3(4):265-71.
[23] Farb A, Tang AL, Burke AP, Sessums L, Liang Y, Virmani R. Sudden coronary death. Frequency of active coronary lesions, inactive coronary lesions, and myocardial infarction. Circulation. 1995;92(7):1701.
[24] Friedman M, Manwaring JH, Rosenman RH, Donlon G, Ortega P, Grube SM. Instantaneous and sudden deaths. Clinical and pathological differentiation in coronary artery disease. JAMA. 1973;225(11):1319-28.
[25] Davies MJ, Thomas A. Thrombosis and acute coronary-artery lesions in sudden cardiac ischemic death. N Engl J Med. 1984;310(18):1137-40.
[26] Sang CJ, Burkett A, Heindl B, Litovsky SH, Prabhu SD, Benson PV, et al. Cardiac pathology in COVID-19: A single center autopsy experience. Cardiovasc Pathol. 2021;54(December 2019):107370.
[27] Ferrer-Gómez A, Pian-Arias H, Carretero-Barrio I, Navarro-Cantero A, Pestaña D, de Pablo R, et al. Late cardiac pathology in severe COVID-19. A postmortem series of 30 patients. Front Cardiovasc Med. 2021;8:748396. Doi: 10.3389/fcvm.2021.748396.

PARTICULARS OF CONTRIBUTORS:
1. Senior Resident, Department of Pathology, Government Medical College, Cochin, Ernakulam, Kerala, India.
2. Professor, Department of Pathology, Government Medical College, Cochin, Ernakulam, Kerala, India.
3. Professor, Department of Forensic Medicine, Government Medical College, Cochin, Ernakulam, Kerala, India.
4. Senior Resident, Department of Pathology, Government Medical College, Cochin, Ernakulam, Kerala, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Jayasree Raman,
Professor, Department of Pathology, GMC, Ernakulam-683503, Cochin, Kerala, India.
E-mail: jayasreeraman2007@gmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Mar 20, 2024
- Manual Googling: May 09, 2024
- iThenticate Software: May 20, 2024 (11%)

ETYMOLOGY: Author Origin

EMENDATIONS: 6

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: **Mar 19, 2024**
Date of Peer Review: **Apr 26, 2024**
Date of Acceptance: **May 21, 2024**
Date of Publishing: **Jul 01, 2024**