Research Article

AN AUTOPSY STUDY OF THE HISTOPATHOLOGICAL SPECTRUM OF CARDIAC DISEASES IN CASES OF SUDDEN DEATH

Shubhangi V. Agale*., Pragya Virendrakumar Jain., Grace F. D'Costa., Megha Kinake Pratik Chide., Mayur Sonawane and Brizellda Rose D'Cunha

Department of Pathology, Grant Government Medical College & Sir J. J. Group of Hospitals, Mumbai

ABSTRACT

Objectives: Sudden death may be due to cardiac or extra-cardiac causes. Sudden cardiac death incidence has been increasing all over the world. This study was planned to observe various histomorphological cardiac changes in patients presenting with sudden death of cardiac etiology. The main objective of this study was to evaluate cardiac findings in forensic autopsy and clinical autopsy pathology cases.

Materials & Methods: This was a descriptive study carried out over a period of 6 months. Total 537 medico-legal and clinical autopsies were performed at our centre out of which sudden death was seen in 289 cases.

Results: This study included 289 cases of sudden cardiac death. Maximum number of cases was in the age-group of 31-40 years and average age was 35.05 years. An overall male preponderance was observed with male to female ratio being 1.83. Grossly, the average weight of heart was 255 gms. Ventricular wall hypertrophy was found in 107(37.02%) cases, left ventricular hypertrophy being more common than right.

Out of 289 cases, 104 cases showed atherosclerosis, 28 cases showed myocarditis with pericarditis. Myocarditis was seen in 20 cases, pericarditis in 16, ischemic heart disease (acute and chronic) in 13 cases, pericardial or myocardial hemorrhage in 6 cases, tuberculosis in 2 cases and vaso-occlusive sickle cell crisis in 1 case.

Conclusion: Ischemic heart disease was the leading cause of death with coronary atherosclerosis as the commonest pathological lesion in adults whereas in children and young adults myocarditis was the common finding.

INTRODUCTION

Sudden death is an enigma which may occur due to cardiac or extra-cardiac causes.[1] World Health Organization has defined sudden death as death occurring within 24 hours from the onset of symptoms.[2] According to other authors death within 1 hour or less from the onset of symptoms is sudden death.[1] Sudden cardiac death (SCD) is currently defined as a natural death from cardiac causes occurring within 1 hour of symptom onset and heralded by abrupt loss of consciousness.[3-4] Additional criteria for SCD include the absence of trauma as a mechanism and the unexpected timing and mode of death.[4]

SCD incidence in the United States ranges from 0.36 to 1.28 per 1000 persons per year.[4] A study carried out in Netherlands found that 21% of deaths in men and 14.5% of deaths in women were due to SCD.[4] Rao *et al.* in 2012, in the first ever study for evaluation of SCD found that SCD contributed to 10.3% of overall mortality.[5]

Incidence of sudden cardiac death has been steadily increasing all over the world[6,7] The annual incidence of SCD is three to four times higher in men than in women.[3] As per World Health Federation 35% of all deaths are caused by cardiovascular disease in India. According to Rao *et al.* in India incidence of ischemic heart disease has increased to ten percent.[6]

MATERIALS AND METHODS

The present study was a descriptive study carried out in the Department of Pathology, Grant Govt. Medical College and Sir J. J. Group of Hospitals, Mumbai over a period of 6 months. Total of 537 cases were received (medico-legal and clinical autopsies) during this period at our centre.

Of these cases, heart specimens were received in 297 cases out of which 8 heart specimens were autolysed. So, total 289 heart specimens were included in the study. Available clinical history, medical diagnosis and postmortem findings of all cases were noted. The heart was fixed in 10% formalin, weighed, externally examined and dissected within 12-24

Corresponding author: **Shubhangi V. Agale**
Department of Pathology, Grant Government Medical College & Sir J. J. Group of Hospitals, Mumbai

hours of fixation using standard autopsy protocol for heart grossing.

On post-mortem examination, the dimensions of heart, thickness of left and right ventricular walls and interventricular septum were measured. The valves were checked for any abnormalities. While sectioning the ventricles transversely at 10 mm intervals starting from the apex, regions of either old or recent myocardial ischemia were checked and their location and size were noted. The three main coronary arteries were dissected out and regularly sectioned at 4-5 mm and examined for thickening, plaques, stenosis or gritty feel on cutting.

For the histopathological examination of the heart, representative sections were taken from the anterior, lateral and posterior left ventricular wall as well as multiple sections from the coronary vessels. In addition, sections were taken from any regions with suspected pathological lesions.

All sections were routinely processed, stained with Haematoxylin and Eosin staining and examined under light microscope. Histological grading of cases of atherosclerosis was done according to American Heart Association.

RESULTS

In the present study 289 heart specimens were included. Age-wise and sex-wise distribution of cases was studied. Overall, maximum number of cases were in the age-group of 31-40 years and minimum number of cases were in the age-group of 0-10 years. Age range was 5 months to 75 years. Average age in present study was 35.05 years [Table 1].

Table 1 Age-wise distribution of case

S. No	Age groups (years)	Number of cases	% of cases
1.	0-10	05	1.73
2.	11-20	32	11.07
3.	21-30	63	21.80
4.	31-40	72	24.91
5.	41-50	48	16.61
6.	51-60	36	12.46
7.	61-70	26	8.99
8.	71-80	07	2.42
	Total	289	100

Our study included 187 (64.71%) males and 102 (35.29%) females. An overall male preponderance was observed with male to female ratio being 1.83 [Table 2].

Table 2 Gender-wise distribution of cases

S. No	Gender	Number of cases	% of cases
1.	Male	187	64.71
2.	Female	102	35.29
	Total	289	100

On gross examination, the average weight of heart as measured in post-fixation state was 255 gms. Ventricular wall hypertrophy was found in 107 (37.02%) cases. Left ventricular hypertrophy (wall thickness > 1.5 cm) (Fig 1a) cases predominated over cases with right ventricular hypertrophy (wall thickness >0.5 cm) [Table 3].

Table 3 Split-up of cases showing hypertrophy of the heart wall (n= 107)

S. No	Heart wall involved	Number of cases	% of cases
1	Left ventricular hypertrophy	41	38.32
2	Right ventricular hypertrophy	30	28.04
3	Left and right ventricular hypertrophy	36	33.64
	Total	107	100

Figure 1a Photograph showing left ventricular hypertrophy.

On gross examination of coronaries more than 75% block was observed in 22 (21.15%) (Fig1b).

Figure 1b: Photograph showing marked thickening of coronary artery due to atherosclerosis**.**

Chronic ischemic heart disease was seen as grayish white, firm areas on bread loafing (Fig 1c).

Figure 1c Photograph showing gray white firm area due to chronic ischemic heart disease

Histopathological examination revealed a wide spectrum of changes, the most common being atherosclerosis found in 104 (35.98%) cases (Fig 2a & 2b), myocarditis with pericarditis in 28 (9.69%) cases (Fig 2c & 2d), myocarditis in 20 (6.92%) cases (Fig 3), pericarditis in 16 (5.54%), chronic ischemic heart disease in 12 (4.15%) (Fig 4), acute ischemic heart disease 1 (0.35%), pericardial or myocardial hemorrhage in 6 (2.08%), tuberculosis in 2 (0.69%) (Fig 5) and vaso-occlusive sickle cell crisis in 1 (0.35%) case [Table 4].
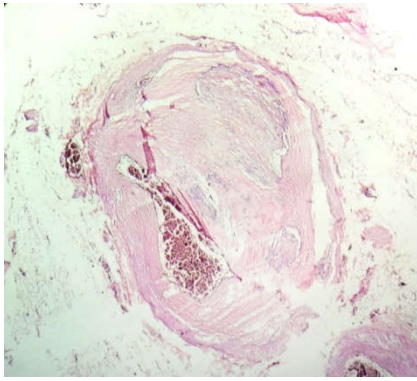
Figure 2a Microphotograph showing near complete obliteration of coronary due to atherosclerosis.
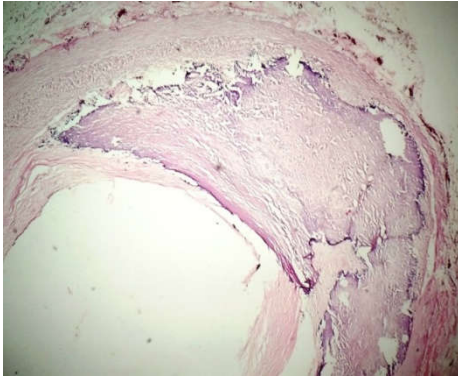

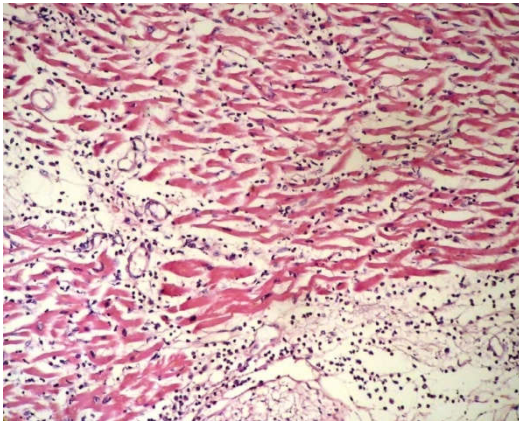**Figure 2b** Microphotograph showing complicated atherosclerosis

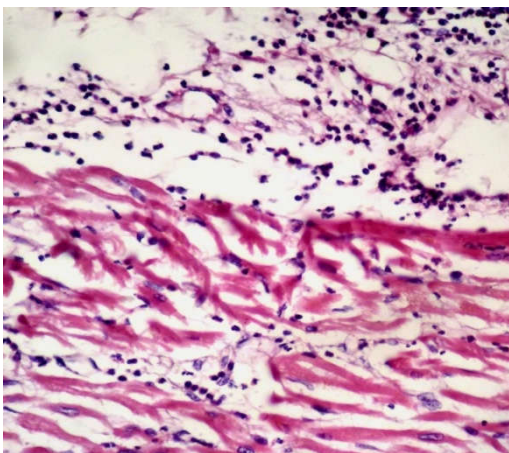

Figure 2c Microphotograph showing myocarditis

Figure 2d Microphotograph showing pericarditis

Multiple cardiac lesions were seen in 10(3.46%) cases which consisted of combination of two or more cardiac lesions like chronic IHD, pericarditis, myocarditis, atherosclerosis and pericardial or myocardial hemorrhage [Table 5].
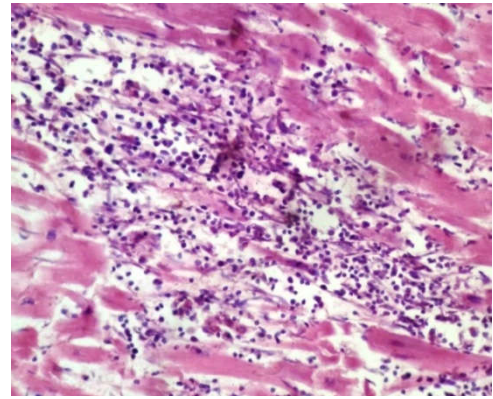

Figure 3 Microphotograph showing chronic inflammatory infiltrate in the myocardium
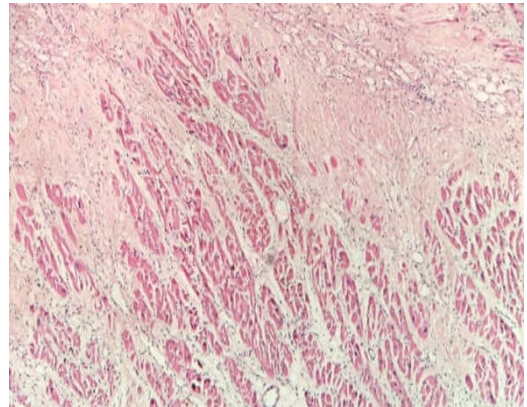

Figure 4 Microphotograph showing myocardial fibrosis due to chronic ischemic heart disease
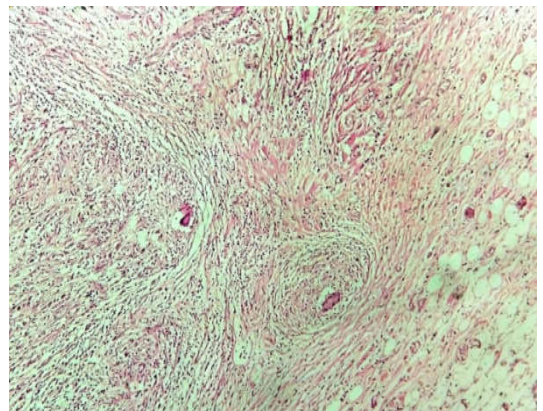

Figure 5 Microphotograph showing tuberculous granulomas comprising of epithelioid cells, lymphocytes and giant cells.

Table 4 Histopathological spectrum of cases found in our study (n=289)

S. No	Diagnosis	Number ofcases	% of cases
1.	Atherosclerosis	104	35.98
2.	Myocarditis and pericarditis	28	9.69
3.	Myocarditis	20	6.92
4.	Pericarditis	16	5.54
5.	Ischemic heart disease-acute	1	0.35
6.	Ischemic heart disease-chronic	12	4.15
7.	Haemorrhage(pericardial, myocardial)	6	2.08
8.	Sickle cell disease	1	0.35
9.	Tuberculosis	2	0.69
10.	Multiple lesions	10	3.46
11.	Non specific findings	24	8.30
12.	No specific findings	65	22.49
	Total	289	100

Table 5 Multiple lesions

S. No	Diagnosis	Number of cases	% cases
1.	Chronic IHD + Pericarditis	3	30
2.	Chronic IHD + Myocarditis	1	10
3.	Chronic IHD + Myocarditis + Pericarditis	1	10
4.	Myocarditis + Atherosclerosis	1	10
5.	Myocarditis + Pericarditis + Atherosclerosis	3	30
6.	Myocarditis + Myocardial & pericardial hemorrhage	1	10
	Total	10	100

Non-specific findings in the heart were seen in 24(8.30%) cases which comprised of congestion, myocardial fibre hypertrophy, myocardial fatty degeneration, brown atrophy, pericardial fibrosis, myocardial fatty infiltration, vessel interstitial fibrosis etc. No specific gross or microscopic findings were seen in 65(22.49%) cases [Table 6].

Table 6 Non specific findings

S. No	Diagnosis	Number	% cases
1	Congestion	10	
2	Myocardial fibre hypertrophy	8	3.57
3	Fatty degeneration	1	0.45
4	Brown atrophy	1	0.45
5	Pericardial fibrosis	1	0.45
6	Pericardial fibrosis and myocardial fatty degeneration	1	0.45
7	Myocardial fatty infiltration	1	0.45
8	Vessel interstitial fibrosis	1	0.45
	Total	24	100

Maximum number of cases with atherosclerosis belonged to the age group 31-40 years, 32 (30.77%) followed by 41-50 years, 24(23.08%) [Table 7A].

Table 7 A Atherosclerosis cases- Age wise distribution

S. No	Age groups (Years)	Number of cases	% of cases
1.	0-10	0	0
2.	11-20	2	1.92
3.	21-30	16	15.38
4.	31-40	32	30.77
5.	41-50	24	23.08
6.	51-60	11	10.58
7.	61-70	15	14.42
8.	71-80	4	3.8
	Total	104	100

All cases of atherosclerosis, based on the extent of occlusion of coronary vessel lumina by atherosclerotic plaques, were classified into categories, namely, 1-25% stenosis in 47(45.19%) cases, 26-50% stenosis in 23(22.12%), 51-75% stenosis in 12(11.54%) and 76-100% stenosis in 22(21.15%) [Table 7B].

Table 7 B Atherosclerosis cases- extent of vessel blockage

S. No	Grade of atherosclerosis	Number of cases	% of cases
1.	1-25 % block	47	45.19
2.	26-50 % block	23	22.12
3.	51-75% block	12	11.54
4.	76-100 % block	22	21.15
	Total	104	100

Histological grading of atherosclerosis as per American Heart Association revealed maximum number of cases in Grade III-36 (34.68), followed by Grade 4- 20 (19.32) [Table 7C].

Table 7 C Atherosclerosis cases - Histological grade according to American Heart Association

Grade	Histological features	Number of cases	% of cases
I	Isolated intimal foamy cells(minimal change)	8	7.60
II	Numerous intimal foamy cells in layers (fatty streaks)	12	11.50
III	Pools of extracellular lipids without a well defined core (preatheroma)	36	34.68
IV	Well defined lipid core with luminal surface covered by normal intima (atheroma)	20	19.23
V	Lipid core with fibrous cap with or without calcification (fibro-atheroma)	13	12.50
VI	Fibro-atheroma with cap defects- hemorrhage and thrombosis	1	0.96
VII	Calcification prominent	12	11.50
VIII	Fibrous change prominent	2	1.92
	Total	104	100

Most cases of atherosclerosis presented with double vessel involvement seen in 43(41.35%) cases followed by triple vessel involvement in 31(29.80%) cases and single vessel involvement in 30(28.85%) cases [Table 7D].

Table 7 D Atherosclerosis cases- blood vessel involvement

S. No	Number of vessels involved	Number of cases	% of cases
1.	Triple vessel involvement	31	29.80
2.	Double vessel involvement	43	41.35
3.	Single vessel involvement	30	28.85
	Total	**104**	**100**

Overall, the most common coronary involved is LAD with 82 (35.65%) cases, followed by LCX 78 (33.91%) cases while the least commonly involved coronary was RCA 70(30.44%) cases [Table 7E].

Table 7 E Atherosclerosis cases - overall blood vessel involvement

S. No	Blood vessel involved	Number of cases	% of cases
1.	LAD	82	35.65
2.	LCX	78	33.91
3.	RCA	70	30.44
	Total	230	100

Out of 104 cases, 88 (84.62%) cases showed only atherosclerosis, 12 (11.54%) cases showed calcification, 2 (1.92%) cases showed plaque rupture, 1 (0.96%) case presented with thrombosis of the coronary while 1 (0.96%) case showed recanalization of coronary [Table 7F].

Table 7 F Atherosclerosis cases- complications

S. No	Diagnosis	Number of cases	% of cases
1.	Atherosclerosis only	88	84.62
2.	Atherosclerosis + calcification	12	11.54
3.	Atherosclerosis + plaque rupture	2	1.92
4.	Atherosclerosis + thrombosis	1	0.96
5.	Atherosclerosis + recanalization	1	0.96
	Total	**104**	**100**

DISCUSSION

The cardiac autopsy is important to study the pathological lesions in the heart. The main aim of this study was to evaluate cardiac findings in forensic and clinical autopsy pathology cases. Our study is important as we have evaluated all gross and microscopic findings in these cases.

The age range was 5 months, 75 years and average age was 35.05 years. But the age range of maximum number of cases (31-40 years) is one decade earlier than the studies done by Marwah *et al*,[1] and Joshi C.[2] Ding *et al*.[8]

There was male preponderance [male- 187 (64.71%), female- 102 (35.29%) with male to female ratio of 1.83. All the other studies[8-15] have male preponderance but according to study done by Shanti *et al*.[7] the male to female ratio was 10.5:1, whereas Ding *et al*.[8] had male to female ratio of 5:1, which are very high as compared to our study and other studies. A study carried out by Winkel *et al*.[13] in children (1-18) also shows male preponderance.

On gross examination, the average weight of heart as measured in post-fixation state was 255 gms whereas it was found to be 303.88 gms in a study conducted by Porwal *et al*.[19] in a study of 103 cases. Of the 107 (37.02%) cases of myocardial hypertrophy, biventricular hypertrophy was seen in 36 cases. In a study done by Joshi *et al*.[2] biventricular hypertrophy was seen in 37 cases out of 60 (52%) cases. Myocardial hypertrophy was also reported by Shanthi *et al*.,[7] Cristina Basso *et al*.,[16] and Bora *et al*.[17] in their studies.

Histopathologically the study revealed a wide spectrum of disorders such as coronary atherosclerosis, myocarditis with pericarditis, myocarditis, pericarditis and chronic ischemic heart disease, but the most common finding was coronary artery atherosclerosis found in 104 (35.98%) cases. Similar findings were reported by Cristina *et al*. (21%),[16] Bora *et al*. (42%),[17] Beelwal *et al*. (52%),[18] and Porwal *et al*. (55.33%).[19]

Among the atherosclerosis cases studied maximum cases belong to the fourth decade (30.77%) which is earlier than the seventh decade as found by Garg *et al*.[20] Most common grade in our study was Grade 3(pre-atheroma), however Beelwal *et al*[18] found Grade 5 (fibro-atheroma) to be the most common.

As per our study the grade I coronary atherosclerosis was common and two vessel involvement was commonest (41.35%), followed by three vessel (29.80%), and one vessel (28.85) involvement. This is different from the studies done by Marwah *et al*.[1] and Porwal *et al*.[19] where three vessel involvement was seen in 52% and 40% respectively. The most common vessel involved was LAD (35.65%), followed by LCX (33.61%), and RCA (30.44%).

More or less similar results were found in the studies carried out by Beelwal *et al*.[18] and Jha *et al*.[21] However Porwal *et al*.[19] and Garg *et al*.[20] have found LAD to be the most commonly involved followed by RCA and LCX.

Out of 104 cases, 12 (11.54%) cases showed calcification, 2 (1.92%) cases showed plaque rupture, 1 (0.96%) case presented with thrombosis of the coronary artery while 1 (0.96%) case showed thrombosis with recanalization of coronary artery. Coronary artery wall calcification was reported by Joshi *et al*.[2] in 17% cases whereas thrombosis was reported by Joshi *et al*.[2] in 5% cases, Shanthi *et al*[7] in 18% cases and Bora Ozdemir *et al*.[17] in 4.8% cases.

Myocarditis with pericarditis was seen in 28 (9.69%) cases and only myocarditis in 20 (6.92%) cases. Myocarditis was reported by Kramer *et al*. (29%)[22] and Drory *et al*. (22%)[23] with more or less similar results as our study whereas other authors namely Cristina *et al*. (10%),[16] Joshi C. 11 (9%) cases,[2] and Shanthi *et al*. (4%)[7] reported lower incidence. The very low incidence of 0.6% of myocarditis was documented by Waller *et al*. in 1992.[24]

In the present study, pericarditis was found in 16 (5.54%) which is very high compared to study by Joshi C. (0.86%).[2]

We reported chronic ischemic heart disease in 12 (4.15%) which is in concordance with study done by Porwal *et al*.[19] and Garg *et al*.[20] but is lower compared to data given by Marwah *et al*.[1]

We found acute myocardial infarction in 1 (0.35%) case which is comparable with data given by Garg *et al* 3 (2.6%)[20] but very low as compared to studies conducted by Marwah *et al*. 14 (7%),[1] Porwal *et al*. 19 (18.44%),[19] and Gohel *et al*. 25 (25%) cases.[25] The low incidence of acute myocardial infarction in our study may be attributed to sudden death which occurred before the development of infarct and very small infarcts which were overlooked on gross examination This also may be related to pattern of sampling of tissue.[1] According to Gohel *et al*.[25] acridine orange detects youngest infarction (upto 2 hours of age).

Tuberculosis of heart was seen in 2 cases of which one showed only pericarditis and the other case was of tubercular myocarditis and pericarditis. In both the cases there was involvement of other organs due to disseminated miliary tuberculosis. The autopsy studies suggest that heart was involved in 2% of cases of patients suffering from HIV/AIDS era.[26] Tuberculous myocarditis is uncommon and occurs as a complication of tuberculosis elsewhere in the body and occurs by direct extension or lymphatic/hematogenous route.[27] In the present study, we found only one case of vaso-occlusive crisis in sickle cell disease. However Joshi C. found 6 (5.21%) cases and is the only study reporting vaso-occlusive crisis in sickle cell disease.

No specific findings on gross or microscopy were seen in 65 (22.49%) cases. Similarly, Zijiao *et al*[8] found 6.1% and Marwah *et al*[1] found 115 cases of unexplained sudden cardiac death.

CONCLUSION

In sudden cardiac deaths, the cause of death can be determined by autopsy but we need to thoroughly examine and sample the heart for histopathological examination. The sampling of all the parts of heart (the coronaries, valves, endocardium, myocardium and pericardium) has to be adequate and represent the lesions present in the heart.

Ischemic heart disease was the most common cause of sudden cardiac death in elderly while myocarditis was most common cause in children and young adults. Atherosclerosis was the most important underlying cause for ischemic heart disease with double vessel involvement being the most common and LAD being the most commonly involved coronary. We found maximum number of cases of atherosclerosis in the age group of 31-50 years with male preponderance. Our study population mainly being urban cosmopolitan, the above statistics highlight the need for employing preventive lifestyle modification strategies like stress reduction, healthy diet and regular exercise especially in the young people. Better screening tests for early diagnosis and advanced management techniques like reperfusion should be employed.

So to conclude, as the causes of sudden cardiac death are varied, a detailed and meticulous post-mortem examination of whole heart is important to rule out the various aetiologies.

References

1. Nisha M, Bhawna S, Sumiti, Duhan Amrita, Singh Sunita, Sen Rajeev. Histomorphological Spectrum of Various Cardiac Changes in Sudden Death: An Autopsy Study. *Iranian Journal of Pathology* 2011;6 (4):179-186.

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

3. Honnekeri BS, Lokhandwala D, Panicker GK, Lokhandwala Y. Sudden cardiac death in India: A growing concern. *J Assoc Physicians India*. 2014; 62(12):36-40.

4. Sara JD, Eleid MF, Gulati R, Holmes DR. Sudden cardiac death from the perspective of coronary artery disease. In Mayo Clinic Proceedings 2014; 89(12):1685-1698. Elsevier.

5. Rao BH, Sastry BK, Chugh SS, Kalavakolanu S, Christopher J, Shangula D, Korabathina R, Raju PK. Contribution of sudden cardiac death to total mortality in India- a population based study. *Int J Cardiol* 2012; 154;163-167.

6. Rao D, Sood D, Pathak P, Dongre SD. A cause of Sudden Cardiac Deaths on Autopsy Findings; a Four-Year Report. *Emergency*. 2014;2:12-17.

7. Shanthi B, Saravanan S, Elangovan RS, Sudhan V. Sudden death causes: An autopsy study in Adults. *Int J Sci Stud*. 2016;4:176-179..

8. Ding, Z., Yang, M., Wang, Y. *et al*. Retrospective analysis of 769 cases of sudden cardiac death from 2006 to 2015: a forensic experience in China. *Forensic Sci Med Pathol* 2017; 13: 336. https://doi.org/10.1007/s12024-017-9888-z

9. Risgaard B, Winkel BG, Jabbari R, Behr ER, Ingemann-Hansen O, Thomsen JL, Ottesen GL, Gislason GH, Bundgaard H, Haunsø S, Holst AG, Tfelt-Hansen J. Burden of sudden cardiac death in persons aged 1 to 49 years: nationwide study in Denmark. *Circ Arrhythm Electrophysiol*. 2014;7(2):205-211. doi: 10.1161/CIRCEP.113.001421

10. Braggion-Santos MF, Volpe GJ, Pazin-Filho A, Maciel BC, Marin-Neto JA, Schmidt A. Sudden cardiac death in Brazil: a community-based autopsy series (2006-2010). *Arq Bras Cardiol*. 2015 Feb;104(2):120-7. doi: 10.5935/abc.20140178.

11. Ifteni P, Barabas B, Gavris C, Moga M, Burtea V, Dracea L. Sudden Cardiac Death: Autopsy Findings in 7200 Cases Between 2001 and 2015. *Am J Forensic Med Pathol*. 2017 Mar;38(1):49-53. doi: 10.1097/PAF.0000000000000274.

12. Wu Q, Zhang L, Zheng J, Zhao Q, Wu Y, Yin K, Huang L, Tang S, Cheng J. Forensic pathological study of 1656 cases of sudden cardiac death in southern China. *Medicine*. 2016;95(5):1-8.

13. Winkel BG, Risgaard B, Sadjadieh G, Bundgaard H, Haunsø S, Tfelt-Hansen J. Sudden cardiac death in children (1–18 years): symptoms and causes of death in a nationwide setting. *European heart journal.* 2013;35(13):868-75.

14. Ramazan Karanfil, Mete K Gulmen, Ahmet Hilal, *et al*. Evaluation of cardiac conduction system in sudden death cases. *J of Forensic Med* 2013;27(1):17-28. doi: 10.5505/additip 2013. 54154.

15. Stavroula A Papadodima, Emmanouil I, Panagiotis S, *et al*. Cardiovascular disease and drowning: Autopsy and laboratory findings. *Hellenic J Cardiol* 2007;48:198-205.

16. Cristina Basso, Fiorella Calabrese, Domenico Corrado, *et al*. Postmortem diagnosis in sudden death victims: macroscopic, microscopic and molecular findings. Elsevier, Cardiovascular Research 2001;50:290-300.

17. Bora Ozdemir, Osman Celbis, Rezzan Onal, *et al*. Multiple organ pathologies underlying in sudden natural deaths. *Medicine Science* 2012;1(1):13-26. 19.

18. Beelwal D, Pachori G, Sunaria RK, Goyal V. A post-mortem study of coronary atherosclerosis and relationship to myocardial infarction in Ajmer region. *Int J of Med Sci Public Health*. 2017;6:563-568.

19. Porwal V, Khandelwal S, Jain D, Gupta S. Histological classification of atherosclerosis and correlation with ischemic heart disease. A autopsy based study. *Ann of Pathol Lab Med* 2016;3:A99-104.

20. Garg M, Aggarwal A, Kataria S. Coronary Atherosclerosis and Myocardial Infarction. An autopsy study. *J Indian Acad Forensic Med* 2011;33:971-973

21. Jha BM Naik D, Agarwal A, Jana S, Patel M. Incidence of coronary atherosclerosis in different coronary arteries and its relation with myocardial infarction: a randomized study in 300 autopsy heart in tertiary care hospital. *Int J of Med Sci Public Health*. 2013;2:836-839.

22. Kramer MR, Drory Y, Lev B. Sudden death in young Israeli soldiers: analysis of 83 cases. *Isr J Med Sci* 1989;25:620-624.

23. Drory Y, Turetz Y, Hiss Y, *et al*. Sudden unexpected death in persons less than 40 years of age. *Am J Cardiol* 1991;68:1388-1392.

24. Waller BF, Waller B, Catellier MJ, Clark MA, Hawley DA, Pless JE. Cardiac pathology in 2007 consecutive forensic autopsies. *Clinical cardiology*. 1992; 15(10):760-765.

25. Gohel H, Desai N, Tripathi S, Gupta B, Santwani P. Post mortem, study of heart in cases of sudden cardiac death using acridine orange fluorescence and haematoxylin and eosin stain. *Indian Journal of Medicine and Healthcare*. 2012;1(5):115-23.

26. Mutyaba AK, Ntsekhe M. Tuberculosis and the Heart. *Cardiol Clin* 2017;35(1):135-144.

27. Jokhdar HA, Sayed SN, Omar SH. Case Report: tuberculosis presenting as myocarditis. *Med J Cairo Univ*.2009; 77(3):89-92.

****