Original Article

Sudden Death Causes: An Autopsy Study in Adults

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Abstract

Background and Objectives: Incidence of sudden death (SD) is quiet frequent to determine the cause of SD and observe the morphological changes in the heart after death.

Materials and Methods: A study for 50 cases was carried out in KAPV Government Medical College, Tiruchirappalli, in Pathology Department, in coordination with forensic medicine department from October 2015 to May 2016. A detailed autopsy finding with histopathological examination was done to analyze the cause of SD. We studied only heart specimen to explore the cause of SD.

Results: All cases revealed to between the age group 31 and 75 years with male preponderance common; out of 50 cases, 23 cases showed features of lesions; about 8 cases showed severe atheromatous change in aorta followed by 4 cases with early myocardial infarction (MI) and 4 cases with MI and complications such as atheromatous aorta with calcification, thrombus both old and fresh and a case myocarditis and medial hypertrophy of left ventricle. Rest of the cases the cause of death was undetermined.

Conclusion: The major causes of death were due to atherosclerosis, whereas MI and acute coronary events (thrombus) observed in 4% of cases of SD.

Key words: Atheroma, Autopsy, Heart, Morphology, Sudden death

INTRODUCTION

Sudden death (SD) is defined as an unexpected natural death due to cardiac cause within a short period (usually within 1 h) with or without onset of symptoms and without any prior condition that would appear fatal.[1] As the definition of SD varies, it is difficult task to compare one set of published data with another death from natural causes, in which interval between onset of signs and symptoms and death was not more than 24 h were regarded as SD.[2-4] It is by definition natural and it excludes all deaths due to poison, trauma. Various workers in this field have given different definitions. Although SD is a relative concept, this concept is currently described as unexpected death occurring within 1 h of new symptoms.[5] If the patient died instantaneously in

the presence of witness or died while asleep, their death was classified as SD.

The incidence of sudden cardiac death has been steadily increasing all over the world. When SD occurs in adults and elderly persons, coronary atherosclerosis is the usual cause. These diseases are frequently concealed and discovered with surprise only at post mortem using through macroscopic and microscopic examination.

This present study was done for 50 cases with the cause of SD with autopsy examination, with coordinance with Pathology Department and Forensic Medicine at KAPV Government Medical College, Tiruchirappalli, Tamil Nadu, India.

MATERIALS AND METHODS

This present study is based on autopsy observation carried out in 50 cases and analyzed for the cause of SD. The cases were chosen as per the definition of SD and autopsied. The heart was examined grossly and microscopically to observe various histomorphological changes and findings were correlated.

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The randomized study on 50 cases was submitted for postmortem analysis; formalin fixed heart was inspected; sections were taken from right and left ventricular wall, stump of aorta and coronaries; multiple sections were taken at 4-5 mm interval; additional sections were made if needed. The tissue was processed, and paraffin sections were made at 4 mm thickness, stained with routine H and E staining.[6]

RESULTS

Out of 50 cases of SD, only 23 cases showed heart changes while rest 27 cases no known cause was made out as in Figure 1.

Our study was done for 50 cases with age range 25-80 years. The most common age group in our study with heart changes was in 51-60 years, and most predominance was in males as in Table 1.

The number of cases involved showed 21 male and 2 female cases; male: female ratio was 10.5:1.

The histomorphological changes were observed most common being solely atheromatous change in aorta (atherosclerosis) in 8 cases and next common being early changes in coronaries and aorta 4 cases; other complications such as calcification of plaques were also seen; thrombus formation was noted in 4 cases, of which one had an old thrombus formation and another with infected ball wall valve thrombus in aortic orifice; and rest one was reported to have focal myocardial infarction (MI) with atheromatous aorta and old thrombus formation. One case was noted with left ventricular hypertrophy with focal calcification of coronaries and one more case with medial wall hypertrophy of coronaries and one with myocarditis.

DISCUSSION

The term SD has no agreed universal definition; Goldstein proposed that SD should be defined as witnessed death within 1 h of the onset of symptoms, emphasizing the need for a uniform definition but saying that the definition of

SD may be expressed in different terms depending on the nature and scope of the investigation.[7]

In our study, out of the total 50 cases of SD, 47 cases (94%) were male cases and 3 (6%) cases were females as shown Figure 2; male:female ratio was 15.6:1 for SD in general. This finding was consistent with study of Sarkojia *et al.* (82%),[8] Thomas *et al.* (73.9%),[8] Nordrum *et al.* (79.67%),[8] and Singh *et al.* (94.5%).[8]

The main aim of this study was to read out the cause of SD and the contribution of cardio vascular cause being the most common one. Data from post mortem of SD parallel the clinical observation on the prevalence of coronary disease, as the major structural etiological factor. More than 80% of cases have pathological findings of coronary heart disease; pathologic descriptions include a combination of long-standing atherosclerosis of the coronary arteries, and acute active coronary lesions, which include a combination of fissured or ruptured plaques, platelet aggregates, hemorrhage, and thrombosis.[9]

Although there are numerous causes of SD, cardiovascular causes are the principle cause among SD in the present
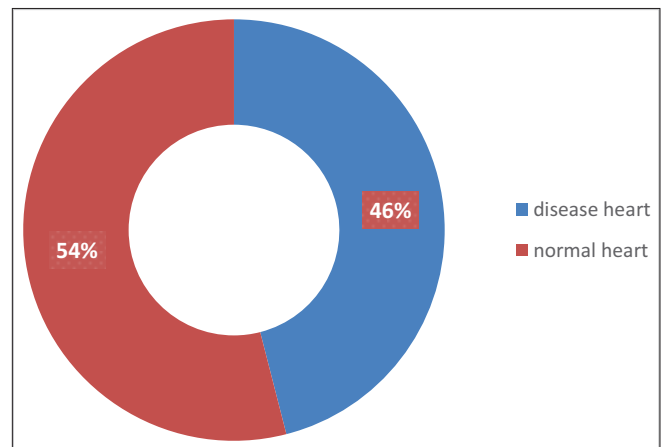

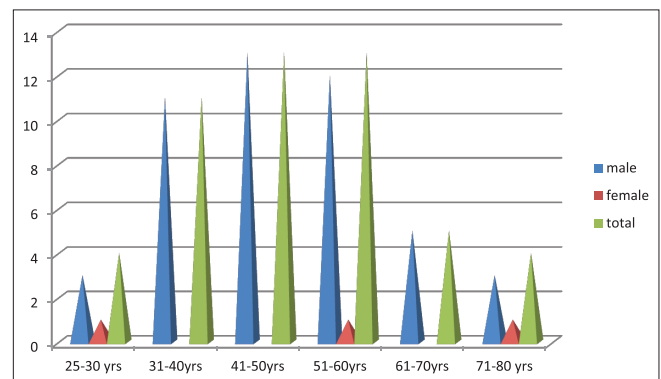**Figure 1: Autopsy changes in heart 50 cases**

Figure 2: Age- and sex-wise distribution among sudden death cases (50 cases)

Table 1: Age and sex of occurrence in 23/50 cases

Age group (years)	Male	Female
25-30	-	-
31-40	4	-
41-50	5	-
51-60	8	1
61-70	3	-
71-80	1	1
Total	21	2

study. In our study, autopsy of 50 cases SD revealed heart changes in 23 cases (46%) with atherosclerosis (35%) of aorta followed by MI with early changes (18%) and MI with complication (18%), thrombus in coronaries (18%), coronary medial wall hypertrophy, left ventricular wall hypertrophy, and myocarditis each (4%) as shown in Figure 3, and the Figures 6-8 depicting the histopathological and microscopic changes in H and E.

Kuller *et al.*[10] studied showed that SDs are due to atherosclerotic disease, which correlated with our study.
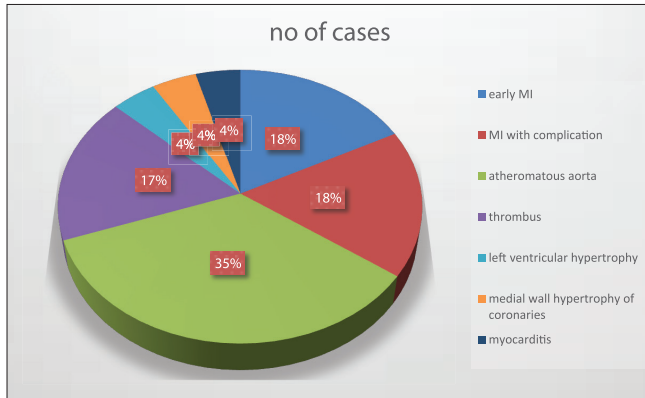

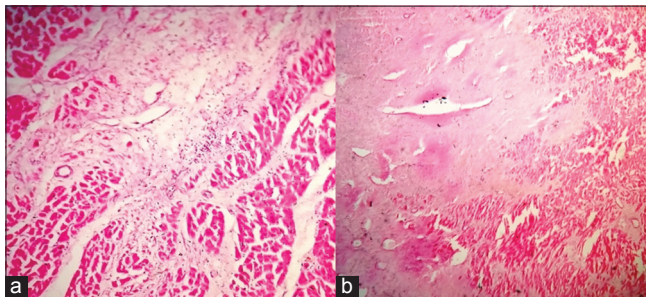**Figure 3: Histomorphological changes in heart (23 cases)**

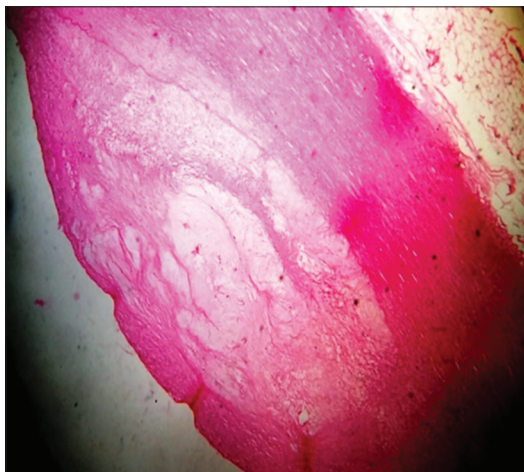**Figure 4: (a and b) Myocardial infarct changes in cardiac myocytes (H and E, ×100)**

Figure 5: Atheromatous plaque aorta (H and E, ×100)

A study by Zanjad and Nanadkar showed SD due to cardiac causes (49.55%); Reddy and Nandy[8] stated that the most of the death were due to cardiovascular causes about 45-50%. Similar findings were seen in study of Kuller *et al.* (49.50%),[8] Siboni *et al.* (46.20%),[8] and Luke *et al.* (38%).[8]

Our study shows that incidence of male was high (21 cases male and 2 cases female) and average ratio of male: female was 10.5:1 which proved to be coherent with studies conducted by Farb *et al.*[10] that SD was more predominant in male, and Framingham[10] studies regarding male: female ratio.

The average age group of incidence was between 50 and 59 years in one study, and study series of Pentilla[10] also showed age group 55-66 years which was compatible with our study and they were the average age group was 50-60 years as shown in Figure 7.

The risk of SD increases dramatically beyond age 35 years and greatest rate of increase is between 40 and 65 years. Among older than 30 years of age, with structural heart
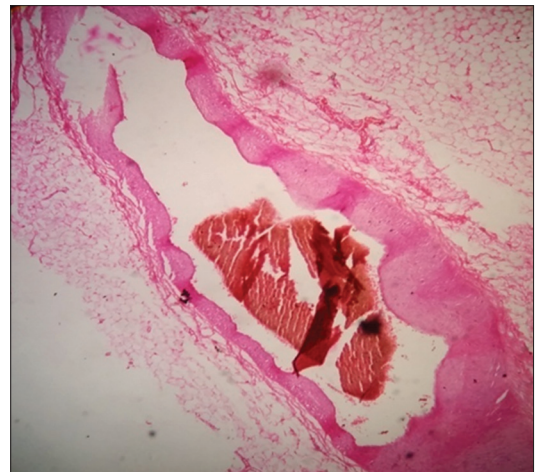

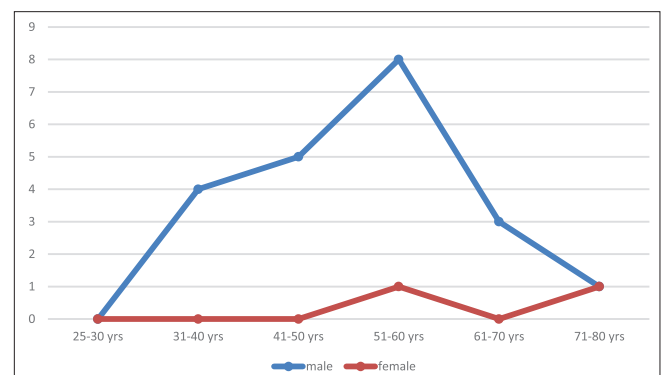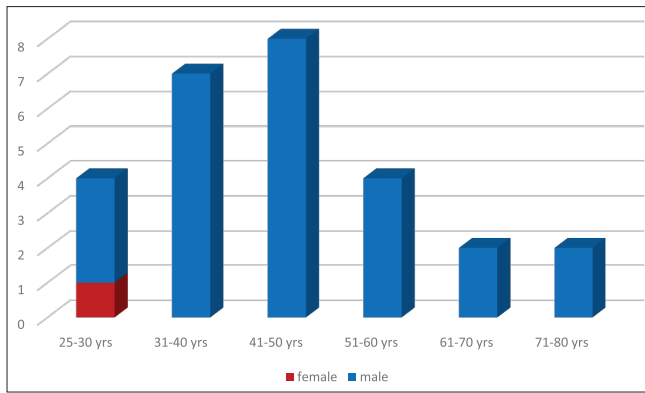**Figure 6: Coronary vessel thrombus (H and E, ×100)**

Figure 7: Age- and sex-wise incidence showing diseased heart changes in 23 cases (Total cases: 23, male - 21, female - 2)

Figure 8: Age-wise and sex with normal heart findings (27 cases) in autopsy

disease and markers of high risk for cardiac arrest, the event may exceed 25% per year and age-related risk

The non-cardiac causes for SD have been reported in the central nervous system as intracerebral hemorrhage due to ruptured berry aneurysm, primary intracerebral hemorrhage, and epilepsy. The respiratory causes could be pneumonia, pulmonary embolism, chronic bronchitis, pulmonary hypertension, and acute asthma and rest of the causes elicited to be acute alcoholic poisoning, alcoholic fatty liver.[11] A variety of sedatives, tranquilizers, and mild analgesic drugs do contribute to causes of SD without cardiac change.[11] Cigarette smoking also increases the risk for SD.

The cause of SD in rest of 27 cases (54%) are of non-cardiac origin (Figure 8), so SD is not a just heart attack, and there remains a group, in which no cause can ever be found to explain death or undetermined attenuates.

CONCLUSION

Ischemic heart disease may be the leading cause of SD, with coronary atherosclerosis being the most significant pathogenic mechanism. This factor is being emphasized by many authors, and in our study, the cases have proven to that atherosclerosis was the major cause of sudden cardiac death, and we received only heart specimen pertained to this study.

SD is source of concern and detail post mortem examination is mandatory to ascertain the cause. The study helped us to find out cardiac causes but the non-cardiac causes were not definite in our study. So, more emphasis has to be made by obtaining complete history data, autopsy details, and histopathological examination of other organs would help out to find the cause. No one putative explanation for SD could be made.

Sudden cardiac death accounts for approximately over half of the total death due to the cardiovascular cause, to prevent this SD in the population.

Certain strategies have to be followed to achieve major population impact; effective prevention of underlining disease and development of new epidemiologic and clinical probes, for better individual risk prediction and specific high-risk groups in the population in needed.

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