Original Article

An Autopsy Study of Sudden Cardiac Death

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Abstract

Objective: To determine the cause of sudden cardiac death and observe the morphological changes in the heart after death.

Methods: A cross sectional survey of 66 cases was carried out at Armed Forces Institute of Pathology (AFIP) Rawalpindi from 1992 to 1994. A detailed autopsy including external and internal postmortem examination was done. Representative sections were taken for histopathological examination and some viscera were sent to Chemical Examiner at Lahore for chemical analysis.

Results: All the cases were male with age range from 21 to 58 years. These cases died within 60 minutes with or without onset of symptoms. Fifty two cases revealed mild to severe atherosclerosis. Coronary occlusion with thrombosis was seen in 37 cases whereas 9 cases showed critical narrowing without evidence of thrombosis. Five showed both symmetric and asymmetric hypertrophy. Changes in conduction system of the heart were seen in 4 individuals. Myocarditis was noted in 2 cases only. The cause of death could not be determined in 3 cases.

Conclusion: Majority of the cases died of coronary atherosclerosis whereas 17% cases revealed hypertrophic cardiomyopathy, changes in conduction system and myocarditis and 4% cases were undetermined. A detailed and thorough postmortem examination is mandatory to ascertain the cause of sudden cardiac death (JPMA 55:149;2005).

Introduction

Sudden cardiac death is commonly defined as an unexpected natural death due to cardiac cause within a short time period (usually within one hour) with or without onset of symptoms and without any prior conditions that would appear fatal.[1] Some prodromal symptoms like palpitation, chest pain and dyspnoea may suggest a cardiovascular etiology.[2,3]

The cardiac etiology of sudden death in an autopsy - based study has been reported in 60-70% of such victims.[2,4] Sudden cardiac death has a much higher incidence in men than women that increases with age because of high prevalence of ischemic heart disease in older ages.[5,6]

The most common underlying pathologic conditions in children and adolescents are myocarditis, hypertrophic cardiomyopathy, congenital coronary artery anomalies, atherosclerotic coronary artery disease, conduction system abnormalities, mitral valve prolapse and aortic dissection.[7,8] In adults, coronary atherosclerosis and acquired forms of cardiomyopathy are the most common findings of autopsies.[9,10]

Ventricular tachycardia and fibrillation and less often bradycardia and asystole are responsible for sudden cardiac death. In most cases, the fatal arrhythmia is triggered by electric irritability of myocardium distant from the conduction system, induced by ischaemia, other cellular abnormalities or infiltration.[11] With detailed postmortem examination most of the time the cause of sudden cardiac death can be determined but in a few cases, the pathologist faces a great difficulty especially in the hearts which appear normal to the naked eye.

The purpose of this study was to determine the cause of sudden cardiac death and observe the morphological changes in the heart after death.

Material and Methods

This study was conducted at Armed Forces Institute of Pathology (AFIP) Rawalpindi from 1992 to 1994. To accomplish the goal, a detailed proforma was designed and circulated to all Armed Forces Hospitals throughout the country. This proforma consisted of two parts. Part +1 was to be completed by the pathologists of various hospitals, who conducted initial postmortem examination. The information was collected from attendants and medical case history sheets. This part included all particulars of the deceased, physical activity prior to episode, any previous sick report, smoking habit, any prodromal signs and symptoms, time lapse between onset of symptoms and death, results of any investigations, any pathology in organs other than cardiovascular system or any other relevant finding. About time lapse between onset of symptoms and death, an eyewitness was consulted if needed.

Part II of the performa was completed at AFIP Rawalpindi where unopened hearts alongwith other vis-

viscera were received from military medical units all over the country. These specimens belonged to personnel of Defence Forces of Pakistan.

Each heart was examined by a senior pathologist and following parameters were assessed:

Weight of heart, ventricular surface fat and thickness of fat at base of right coronary artery, apparently dominant artery, coronary atherosclerosis, thrombosis, occlusion and its site from origin of vessel, state of coronary ostia, recent/old infarcts along with their size and location.

The coronary arteries were examined by transverse cuts at 2-3 mm intervals all along their length. The lesions of atherosclerosis were visually graded as:

Grade I - Artery appeared grossly normal but had microscopic findings of atherosclerosis.

Grade II - Thickening of vessel wall with 25 to 50% narrowing of lumen.

Grade III- Thickening of vessel wall with 50 to 75% narrowing of lumen.

Grade IV - Thickening and calcification with more than 75% narrowing of lumen.

Coronary artery dominance was assessed by noting the origin of posterior descending branch from either right or left artery. Representative sections were taken from all coronary arteries.

The hearts were then opened by modified Virchow's[12] method following the direction of blood flow. All the chambers were washed off any blood clots and examined for any pathology of valves or endocardium. Thickness of ventricular walls and interventricular septum was also measured. Blocks for histopathological examination were taken from right atrium, SA node, AV node, base and middle of septum; anterior and posterior walls of both ventricles and from left anterior and posterior papillary muscles. Serial sections were taken from areas of infarcts if any. These tissues were processed in automatic tissue processor "SAKURA - JAPAN" for 16-18 hours. Three to five micron thick sections were made with the help of manual microtome "LEITZ - GERMANY". These were stained with haematoxylin and eosin, elastic von Gieson and Masson trichrome stain. The slides were examined under light microscope. Some of the viscera along with their contents (liver, kidney, stomach and small gut) in saturated saline were sent to the Chemical Examiner to the Government of Punjab, Lahore for detection of poisoning.

Results

A total of 656 autopsy specimens of serving soldiers were received at this institute during two years. Of these 66 cases fulfilled the criteria of sudden death as defined and were studied according to above mentioned protocol. All were male with age range between 21 to 58 years. Maximum number of cases were seen during 4th and 5th decades.

The commonest symptoms in order of their frequency were: chest pain 58%, sudden collapse 50.0%, dyspnoea 42%, cold sweating 16% and vomiting in 14% cases. Majority of the cases died at rest whereas 10 died during moderate to severe exertion. Electrocardiography could be done in 5 individuals only, which revealed ventricular fibrillation and asystole.

In 76% of the cases posterior descending artery originated from right coronary artery. Most of the cases (27) revealed triple vessel disease followed by double vessel disease (15 cases) and single vessel disease (10 cases). The predominant involvement was seen in left anterior descending artery (LAD) followed by right coronary artery, left circumflex coronary artery and left mainstem coronary artery. Most common cause of sudden cardiac death was coronary atherosclerosis found in 52 cases (79%). Lipid rich atheromata were found in 65% and fibrofatty atheromata in 35% of cases. Atherosclerotic coronary occlusion alongwith thrombosis was found in 37 cases (56%), out of which 30 (45%) revealed recent thrombosis with plaque rupture and erosion as a substrate. Recent and old infarcts were seen in 20.8% and 35.1% cases respectively. Conduction system of the heart showed necrosis of SA and AV nodes in one and two cases respectively due to narrowing of nutrient arteries. Only one case revealed nonspecific fatty infiltration in Bundle of His. Both symmetric and asymmetric hypertrophic (increased thickness of left ventricular wall and interventricular septum) was seen in 5 cases. In two hearts interstitial lymphocytic infiltrate (myocarditis) was observed. The cause was undetermined in 3 cases. Other findings are depicted in Table and Figure. Sixteen cases of traumatic sudden deaths were included as controls. The age range of these cases was 21 to 50 years. Three of such cases showed mild degree of atherosclerosis in LAD. None of them revealed signs of thrombosis or recent and old myocardial infarct. In one case only, fatty infiltration

Table. Different grades of coronary atherosclerosis (n = 52).

Grade	No. of cases	Percentage
I	2	4%
II	4	8%
III	10	19%
IV	36	69%

Figure. Age-wise distribution in cases of sudden cardiac death.

thrombosis or recent and old myocardial infarct. In one case only, fatty infiltration was seen in the A-V node. The chemical analysis of the viscera and their contents sent to the Chemical Examination at Lahore did not reveal evidence of poisoning in any case.

Discussion

Following trauma and poisoning, heart disease is the most common cause of sudden death and in these cases coronary atherosclerosis is the predominant lesion. However 10-20% cases of sudden cardiac death are of non-atheromatous origin.[7] So sudden death is not just heart attack. There remains a group in which no cause can be ever found to explain death. Myocardial ischemia in the absence of coronary artery atherosclerosis can be caused by coronary spasm, probably secondary to inappropriate adrenergic receptor activity, or by compression of a major epicardial artery by contraction of an over bridging left ventricular muscle.This intramyocardial location of LAD may represent a potentially lethal variant. Systolic contraction causing occlusion of such LAD artery may precipitate death in selected subjects especially during exercise.[13]

In studied sample, (out of 656 autopsies) sudden cardiac death was 10%. The percentage of cardiac cases below 40 years was 45.5% which is closer to previous series (42.5%)[14] but lesser to a local study10 (71%). This difference is probably due to less number of cases (14 cases) in the second study

In adults the most common cause of sudden cardiac death is coronary atherosclerosis. In a clinicopathological study of sudden death by Kasthuri et al[15], in India, 76.9% individuals died of coronary artery diseases and triple vessel disease was seen in 8 of 10 cases. Similar observations were made in this study where 79% cases showed various grades of atherosclerosis and triple vessel disease was the predominant finding. Twenty one percent of our cases did not show coronary atherosclerosis and this finding agrees to other studies where 10-20% cases of sudden cardiac death of non-atheromatous origin have been reported.[7]

The right coronary artery was dominant in 76% of our cases which correlates with another study[16] where 80% of the hearts had right dominant artery. The right coronary artery has been suggested as the artery of sudden death because of its possible role in supplying both sinu-atrial and atrioventricular nodes.

The evidence of occlusive coronary thrombi as a cause of sudden cardiac death in this autopsy study was 56% which is closer to other previous studies by Crawford et al[17] (64%) and James et al[18] (62%). However in a previous study by Luqman et al[10] complete blockage with thrombosis was not seen in any case. Majority of our cases with recent thrombosis showed plaque rupture and erosion. Similar findings were seen in 81% young adults in a study carried out at Netherlands.[19]

In the present study recent infarcts were found in 20.8% of cases and old infarcts in 35.1% individuals. This figure correlates with other studies in which healed infarct has been reported in 40% of cases and recent infarct in upto 25% of cases.[7]

Sometimes examination of conduction system of the heart is helpful in establishing the cause of sudden death. These changes could be in the form of necrosis or nonspecific findings like fatty infiltration and fibrosis. We observed changes in cardiac conduction system in 6% individuals whereas Cohle et al[20] noted such lesions considered to be lethal in 3% cases. In this study one and two cases revealed necrosis of S.A. node and A-V node respectively. The necrosis was due to narrowing caused by atheromatous intimal thickening of nutrient arteries. This observation is in conformity to a previous study[11] where 2 of 49 cases showed acute changes in the conducting system.

Morphological changes in conduction system may or may not be seen. The transient conduction block may be explained on the basis of efflux of potassium from the surrounding necrotic tissue and the release of lysosomes from the infiltrated leukocytes, creating transient functional defect.[11]

In this series five (8%) young individuals (age range 21-30 years) revealed gross and microscopic features of hypertrophic cardiomypathy which is in conformity to two pervious studies by Mckenna et al[21] and Nocod et al.[22] The mechanism of sudden death due to hypertrophic cardiomyopathy are not completely understood but the available data suggests the role of arrhythmia

arrhythmia and/or haemodynamic disturbances as precipitating factors.[23,24]

In a Spanish study[25], hypertrophic cardiomyopathy and myocarditis was seen in 6.5% and 3.2% cases respectively whereas in this series similar percentage (3%) of myocarditis cases was noted. However, 8% of hypertrophic cardiomyopathy cases is comparable to an Indian study (7.6%).[15]

Undetermined cases of sudden cardiac death in our study were 4% which is in contrary to two previous studies where it was 11.8%[20] to 16.3%.[24] Such cases may be because of myocardial ischaemia caused by coronary spasm secondary to overdriven adrenergic activity.

In this study coronary atherosclerosis was the major cause of sudden cardiac death. Sudden death is a source of concern and a detailed postmortem examination is mandatory to ascertain the cause.

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