Original Research Article

Sudden cardiac death: An autopsy study

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Abstract

Background: The most common cause of sudden cardiac death is ischaemic. Such patients may have an occlusive recent thrombosis in one or more major coronary arteries. An area of stenosis of 85 per cent is the best discriminating level for increased mortality. **Objective:** To study the cardiac causes of sudden death. **Materials and Methods:** 200 medicolegal cases of sudden death requested for forensic and pathological examination of heart were studied. A careful study of the information given by the police and forensic medicine experts was done. The heart was examined macroscopically for clots, thrombi, any anomalies, tumors or any other pathology. The organ was dissected, bits taken and sections studied under the microscope. **Results:** A total of 200 cases were studied. 120 were male and 80 were female. Majority of them(70%) weighed less than 300 grams. Only 2 weighed more than 350 grams. Cardiomyopathies were recognized in 30 specimen. Dilated cardiomyopathy was most common among them and accounted for 20 cases. **Conclusion:** Personal experience of studying such patients, suggests that the majority of deaths are indeedcardiac. Personal experience of studying such patients, suggests that the majority of deaths are indeedcardiac. The various causes of sudden cardiac death must be correlated with pathological findings to arrive at a definitive diagnosis.
Key Words: Autopsy, Heart, Ischemia, Thrombosis, Ventricular fibrillation.

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INTRODUCTION

The most common cause of sudden cardiac death is ischaemic[1,2]. Such patients may have an occlusive recent thrombosis in one or more major coronary arteries. An area of stenosis of 85 per cent is the best discriminating level for increased mortality. Most subjects who die of ischaemic heart disease suddenly, have this degree of stenosis in two or three major arteries[3,4]. Severe aortic stenosis, hypertrophic and obstructive cardiomyopathy are non ischemic causes for sudden death[5]. When the heart is macroscopically normal, review of previous electrocardiograms is the most helpful guide and may disclose conditions such as a long QT interval or pre-excitation. Use of the term "cardiomyopathy" by pathologists to cover all non-ischaemic sudden cardiac death is clinically misleading. The pathologist, when faced with investigating sudden cardiac death in an adult or a child over1 year of age, can usually place the heart into one of three categories[6,7,8]. In order of frequency these are ischaemic heart disease, conditions recognized macroscopically known to be associated with sudden death and, finally, hearts which are, at least to thenaked eye, normal.[9,10] Annually many thousands of patients dying suddenly from ischaemic heart disease come to autopsy for examination by forensic experts and pathologists[11,12], there is surprisingly little detailed knowledge of the pathology of ischaemic heart disease related to sudden death.[13] A proportion of these patients do have a recent occlusive thrombus in a major coronary artery. Atherosclerosis with fibrofatty atheroma is a common finding in coronary arteries. Death can be assumed to be from subsequent ventricular fibrillation.[14,15] The proportion of cases with such a thrombus is reported to be from 4 to64 per cent[15]. Such widely divergent figures reflect, in part, the degree of care taken or the beliefs of the individual pathologist.
Even within a single pathological department the proportion of thrombi found by different individual

consultant pathologists varies widely.[16] Other factors which militate against comparability of series are different temporal definitions of the term "sudden", varying proportions of smokers to non-smokers [17,18].

MATERIAS AND METHODS

200 medicolegal cases of sudden death requested for forensic and pathological examination of heart were studied at asram medical college, elluru from Jan 2015 to June 2017. A careful study of the information given by the police and forensic medicine experts was done. The heart was examined macroscopically for clots, thrombi, any anomalies, tumors or any other pathology. The organ was dissected in the direction of flow of blood. Multiple bits were taken from left anterior descending coronary artery, right coronary and left circumflex coronary artery. Bits were also taken from ventricles, valves, root of aorta and pericardium. Sections were taken and stained with H and E, and special stains like congo red and others. Sections were studied under the microscope.

RESULTS

A total of 200 cases were studied. 120 were male and 80 were female. (Table 1) Majority of them (70%) weighed less than 300 grams. Only 2 weighed more than 350 grams. (table2). Cardiomyopathies were recognized in 30 specimen. Dilated cardiomyopathy was most common among them and accounted for 20 cases. (Table3) Coronary vessels showed macroscopic thrombi in 80 of them. (Table 4). Microscopic examination of coronary vessels revealed atherosclerosis in 150 specimen. 100 were complicated by thrombi. Non ischemic causes including hypertrophic cardiomyopathy (HOCM) accounted for lesser numbers of cases (Table 5).

Table 1: Gender distribution

Gender	No.	%
Male	120	60
Female	80	40
Total	**200**	**100**

Table 2: Weight of heart

Weight	No.	%
<300 g	140	70
300-350g	58	29
>350 g	02	01
Total	**200**	**100**

Table 3: Cardiomyopathies

Type	No.	%
Dilated	20	66.6
Restrictive	05	16.6
Hypertrophic	05	16.6
Total	**30**	**100**

Table 4: Macroscopic Thrombi in coronary vessels

Lesion	No.	%
Seen	80	40
Not seen	120	60
Total	**200**	**100**

Table 5: Microscopic atherosclerosis in coronary vessels

Lesion	No.	%
Present	150	75
Absent	50	25
Total	**200**	**100**

DISCUSSION

To demonstrate a morphological cause for sudden death is the wish of many pathologists[19,20]. The reported proportion of patients dying with ischaemic heart disease is very wide, from 12 to 85 per cent[3,21,22]. The cases with an occlusivethrombus can be predicted to develop regional myocardial infarction [23,24] but when death occurs within12 hours it is difficult for the pathologist to demonstratenecrosis[25]. Figures from Seattle [26] indicate that only 19 per cent of patients resuscitated from "sudden death" do develop myocardial infarction. The occlusion when presentdoes, however, act as a trigger to provoke ventricularfibrillation[27,28]. Platelet emboli are postulated to cause sudden death. [29,30,31] It is certainly possible that a shower of disintegrating platelets may not only block small arteries but their "pharmacological" contents could provoke intense spasm more distally[32,33]. There is some evidence suggesting that right coronary artery occlusions are more often associated with sudden death.[34,35] The limited published data available suggest that the ratio of right to left anterior descending artery occlusions is lower in patients dying in hospital of infarction than sudden death patients not reaching hospital[36,37](Table 6).

Table 6: Frequency of recent occlusive coronary thrombosis in sudden death

Author	No. of cases	Thrombosis %
Friedman *et al*[1]	27	4
Kuller *et al*[2]	486	18
Lie and Titus[3]	120	17
Spain andBradess[4]	189	18
Titus *et al*[5]	286	19
Perper *et al*[6]	171	22
Davies and popple[10]	120	33
Baba *et al*[11]	121	38
Crowford *et al*[12]	75	64
Present study	200	40

James [29,30] has reviewed the possible causes of this association which leads the right coronary to be regarded as the artery of sudden death [38]. The major factor is the role of the right coronary artery in supplying both sinuatrial andatrioventricular nodes.[39,40] The role of pure coronary artery spasm is clearly notamenable to

investigation in the dead heart andmust remain speculative.[41,42]

Table 7: Site of occlusive coronary thrombosis in sudden death

Author	No.	Main left	LAD	RCA	LCX
Friedman *et al*[1]	21	01	11	07	02
Davies and Popple[10]	39	00	18	16	09
Crowford *et al*[12]	48	00	27	15	06
Basha *et al* [11]	46	03	16	19	08
Present study	80	02	38	26	14

Cardiac death not caused by coronaryatheroma: The second major group of hearts are those with non-ischaemic cardiacdisease. [43,44] Severe left ventricular hypertrophy particularly due to aortic valve stenosis maybe associated with sudden death.[45] In practice, hearts of a total weight over 550 g will have sufficient ventricular hypertrophy to be reasonably associated with sudden death.[46] In cases of aortic stenosis subendocardial recent necrosis is usually demonstrablein the left ventricle. Gross right ventricularhypertrophy also carries a risk of sudden death usually associated with previously unrecognized pulmonary valve stenosis, obstructive cardiomyopathy, or primary pulmonary hypertension.[47,48,49] Sudden death is seen particularly in men (Table 4)and has been reported to occur particularly incertain families.[37,38]There are no morphological features to distinguish those cases dying suddenly from those not. The pathologist should suspect hypertrophiccardiomyopathy in any heart showing ventricular hypertrophy with a small left ventricularcavity. Inclusion as a standard autopsy practice of measurement of the septum and posterior wall with ratios over 1.6confirms many of these cases to be hypertrophiccardiomyopathy.(Table 5). Inhypertrophic cardiomyopathy gross hypertrophy of the free left ventricular wall, on occasions, tends to mask the septal asymmetric hypertrophy leading toan erroneous diagnosis of "hypertensive" cardiomegaly.[50] On rare occasions the mass of abnormal muscle is not septal. A reversal of the septal/posterior wall ratio may also therefore indicate hypertophiccardiomyopathy provided that no old septalinfarction is present.[40] Subaorticendocardial thickening is always a valuable confirmatory feature in cases of HOCM with outflow obstruction. Cardiomyopathy of the congestive form is not associated to any obvious degree with sudden death without a prior long period of left ventricularfailure.[51] Deposition of amyloid can cause sudden death particularly when extensive and involving the conduction system[52,53]. A high proportion of elderlypatients at autopsy have nodules of a substancestaining as amyloid in the left atrium with a tendency to be associated with atrial fibrillation.[4,54] Acute myocarditis of all forms can be associated withsudden death [55,56,57]; with history of some days' malaise, fever, and tachycardia or palpitation. It is possible to suspect the diagnosismacroscopically; the myocardium is mottled, theleft ventricle dilated but with no cardiomegaly. Pericarditis is also present. Idiopathic giant cell myocarditis has serpiginous areas of myocardial necrosis[58] Myocardial sarcoidosis is easily confirmed histologically. Once again caution must be used to avoid overdiagnosis of myocarditis at autopsy [59,60]. Isolated fociof lymphocytes in the atrial myocardium, are common in all elderly hearts and maybe erroneously related to death by pathologists. In cases of death actually caused by myocarditis virtually every histological block from the ventricular muscle, and often from the conduction system itself, is involved. Sudden death may occur in patients with floppymitral valves, yet without severe mitral regurgitation.[61,62]The frequency of the valve abnormality in the population is of the order of 5 per cent [42,63] so the risk of death to any individual patient with a floppymitral valve must be very small.[43,44]Patients with a mild floppy valve and a normal electrocardiogram probably have no such risk. The mechanism underlying these electrocardiographic abnormalities is debatable.[46,64] It has been ascribed to be associated with a primary muscleabnormality, mechanical traction on papillarymuscles, endocardial impact with the valve, anomalous coronary arteries, and interference with left circumflex flow[65,66,67]. If this is a subtleab normality of myocardial repolarisation, it is uncertain if the association with a floppy mitralvalve is fortuitous. Anomalous coronary artery anatomy[47,68] may be perfectly benign or produce serious functional effects. Where the abnormality is a simple one of both coronary orifices arising from the same sinusor a single orifice there is no risk of sudden death. The commonest form is to find a single or two orifices in the right coronary sinus[69]. The left anterior descending coronary artery crosses in front of the right ventricular outflow.[70,71] Sudden death is a risk either when a segment of the coronary artery tree is aplastic or when there is a coronary shunt. Most frequently the former is a single right coronary orifice with the left anteriordescending artery passing behind the pulmonaryartery and being represented as a fibrous strand without a lumen[72,73]. Coronary shunts occur with afistula from an artery into the ventricles, atria, orcoronary sinus. Aneurysmal dilatation of thecoronary artery involved ensues and myocardialperfusion becomes abnormal. Anomalous origin of one coronary artery from the pulmonary artery also leads to an aortic-pulmonary shunt[74,75].Coronary embolism is a cause of sudden death. Emboli occur most frequently from aortic valve thrombus as in bacterial endocarditis. All myxomatous polyps on the aortic valvemay prolapse into a coronary orifice but

areextremely rare.[9] Isolated dissection of coronaryarteries occurs occasionally to produce sudden death in Marfan's syndrome and also in pregnancy.[50,51,76] Coronary arteritis occurs in polyarteritisnodosa [52,77] and sudden death is well described.[19]InJapan[78] a striking syndrome of lymphadenopathy, skin rash, conjunctivitis, and fever in young children with a high risk of sudden death from coronaryarteritis is relatively common [63,79]When all other known causes of sudden death have been excluded macroscopical examination ofthe area of the conduction system is, on occasion, helpful. The small benign mesothelialtumour of the atrioventricular node[54,56] is usually visible as a1 to 2 cm cystic mass in the atrial septum anteriorto the coronary sinus. It is when the pathologist is faced with a heartapparently totally normal to external examinationthat practical problems arise.[80] A number of conditions deserving better recognitionis also squeezed into a "cardiomyopathy" group by pathologists. Isolated increase in the heart weight is better termed idiopathic cardiomegaly. Most examples are probably an excessive hypertrophy response to unrecorded hypertension [81]. Widespread interstitial or focal myocardialfibrosis without other morphological abnormalityis best termed idiopathic myocardial scarring andmay well be due to post-viral myocarditis. This group ofpatients, clinically, may be associated with arrhythmicproblems entirely without evidence of abnormalmyocardial contractile function.[82] Macroscopically, normal hearts are also en-countered in which a selective and progressive loss of conduction fibres occurs followed by replacemen tfibrosis. These hearts are again not associated with evidence of loss of contractile function but developarrhythmias and conduction defects. [83]

CONCLUSION

The initial step is to exclude unnatural death, in particular a concealed suicide. Blood should be screened for drugs by gas chromatography. After these steps have been carried out death can be presumed to be cardiac in origin. Adetailed medical history from the family hasto be sought and any electrocardiogram ever taken must be reviewed. The conduction system has to be examined histologically. Personal experience of studying such patients, suggests that the majority of deaths are indeed cardiac. The various causes of sudden cardiac death must be correlated with pathological findings to arrive at a definitive diagnosis.

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