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Sudden cardiac death in South India: Incidence, risk factors and pathology

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ABSTRACT

Background: Sudden cardiac death (SCD) is a major cause of mortality secondary to coronary artery disease (CAD) in the industrialized societies. Although South Asians have a high prevalence of CAD, the frequency and underlying pathology of SCD among this population are unknown.
Methods: Medical records of consecutive patients presenting with unexplained sudden death (USD) in a tertiary care center were reviewed. Patients with trauma, violent deaths, positive toxicology and non-cardiac pathology were excluded to determine sudden cardiac death (SCD). Cardiac pathological findings were analyzed by autopsy. SCD rate was estimated based on census and government vital statistics for the years studied.
Results: During a two year period, 223 patients (mean age 55 + 10 yrs, 78.9% male, body mass index 26 + 4, 60% smokers, 39% known CAD, 46% hypertension, 43% diabetes) presented to hospital with USD. SCD was attributed to myocardial infarction (MI) in 87% of cases; 69% were acute (96% anterior MI); 76% were small/moderate infarct and 9.9% of the cohort had normal hearts. Based on official municipal vital statistics, the SCD rate in those >35 yrs of age was estimated as 39.7/100,000 with male/female ratio of 4.6.
Conclusions: SCD in this south Indian city occurred predominantly in men of relatively young age and was most frequently associated with small or moderate-sized acute MI. Improved health care access, preventive measures and enhanced emergency management may reduce SCD from acute MI in this locale.

1. Introduction

In developing countries such as India, cardiovascular disease is a major cause of mortality [1]. It is estimated that 60% of the world's coronary artery disease (CAD) patients are South Asians [2] who have a high prevalence of CAD risk factors at a relatively young age [3]. Acute myocardial infarction (AMI) is frequent and the associated mortality is high in this group. In addition, preventive measures, and myocardial revascularization are both low and heart failure is common in India [4,5].

In Western societies, CAD is the major cause of sudden cardiac death (SCD) [6]. By contrast, there is a paucity of data regarding the incidence and causes of SCD in South Asian societies [7]. This deficiency is related to multiple factors such as absence of systematic record acquisition, non-uniform billing codes, variations in payment strategies, lack of reporting and social aversion to autopsy. Knowledge of the rate and underlying pathology of sudden death could promote strategies for prevention and therapy of this tragedy.

We sought to assess the incidence and causes of out of hospital SCD as determined by postmortem findings among patients presenting with unexplained sudden death (USD) to a suburban tertiary care center in south India.

2. Methods

After obtaining approval from the institutional ethics committee, the records of patients presenting with unexplained sudden

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death (USD) from January 1, 2010 to December 31, 2011 undergoing autopsy were reviewed for demographics, medical history and pathological findings. Patients were considered to have USD if they died at home or were found dead out of hospital without a known cause, and were brought to the autopsy suite for evaluation of cause of death. Patients with trauma, violent deaths and positive toxicology were excluded from the study. Resuscitation history was not available for these patients, but none were alive at presentation to the emergency department (ED), however there are no standardized emergency response systems available in the geographic area of this study. All study patients were known to have died within 24 h of presentation to the ED.

2.1. Autopsy

Autopsy methodology followed previously described procedures [8]. Gross anatomy of the heart, the great arteries and the cardiac valves were assessed in detail. The coronary arteries were examined for size, shape, position, number, and ostial patency, as well as course and dominance. Multiple transverse cuts at three mm intervals were made along the course of the epicardial arteries and their major branches to assess patency. If calcified coronary arteries could not to be opened, they were removed intact, decalcified and opened.

Stenosis of the coronary arteries was graded I-IV based on cross sectional area of the lumen [9].

Grade I - Artery appeared grossly normal but had microscopic findings of atherosclerosis such as inflammatory infiltrates and lipid deposits.

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

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

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

The intact heart weight was assessed and compared with the nomogram for age and gender. Left ventricular (LV) wall thickness was measured at the base of the papillary muscles and compared with normal values for age and gender [8]. LV free wall slices (anterior, lateral and posterior), ventricular septum, free wall of the right ventricle and right ventricular outflow tract, and a block from each atrium as well as any area with macroscopic abnormalities, were examined with hematoxylin and eosin stain and a connective tissue stain (van Gieson, trichrome).

Hypertrophy was diagnosed if the thickness of either ventricular wall exceeded the average for age and gender. Hypertrophic cardiomyopathy (HCM) was considered present if the ratio of interventricular septum to posterior wall was greater than 1.3 [10]. Myocardial infarction (MI) was considered acute or chronic based on the types of inflammatory cells and stage of necrosis of the infarcted LV segment. The presence of polymorphonuclear cells and soft necrosis was evidence of acute infarction and the presence of fibrosis, histiocytes and collagenization was indicative of chronic infarction. Infarct size was calculated using a derivation of the area by geometry and volume and multiplying by greatest depth after histological confirmation of the infarction. This factor was divided by total weight of the respective ventricular slices, yielding percent of ventricular involvement: small (<8%), moderate (8–14%), large (>14–22%) and very large (≥23%) [11]. Coronary thrombus was defined as the presence of clot with collections of platelets, fibrin, and trapped erythrocytes and leukocytes; atherosclerotic plaque was defined by presence of lipid core and disruption of the luminal fibrous cap with fissure or rupture into the lipid core [12]. Presence of inflammatory infiltrates in the coronary arterial wall was defined as coronary arteritis [13].

Patients with non-cardiac pathology were considered to have non-cardiac causes of USD. Cardiac abnormalities when present were considered to be the cause of SCD; conversely, absence of cardiac or non-cardiac pathological findings indicated non-structural cause of SCD.

2.2. Statistics

Results are expressed as mean (±standard deviation) or percentages. Chi square test and Student's t-test were used to compare proportions and means, respectively, using STATA 10. Differences were considered significant if $p < 0.05$.

2.3. Estimation of SCD incidence

Tirunelveli Medical College hospital is a tertiary care center which exclusively serves as an autopsy site for the City of Tirunelveli and its surrounding areas. All deaths in the district referred by physicians or by law enforcement requiring identification of cause of death to undergo autopsy at this institution were included in the study per criteria elaborated in methods section. Based on the number of SCDs, as determined by our pathological findings, an estimate of the SCD incidence was made from the death rate reported in the vital statistics for the district population during the period of this study.

3. Results

During the two year interval of this study, 223 patients underwent autopsy for USD, 80% of whom were male (Table 1). Mean age of the entire study cohort was 55 + 10 yrs, and BMI was 26 + 4. Patients' backgrounds included smoking in more than half; one third had a history of alcohol consumption; and more than a third had a history of CAD. Hypertension and diabetes were present in less than half of the total study group.

3.1. Autopsy findings

In forty two patients (19%), USD was attributable to non-cardiac causes such as infection, cerebrovascular accident and pulmonary embolism. The remainder of those who presented with USD were deemed to have died of cardiac causes, of which structural heart disease was present in a large majority (Table 2).

Coronary artery atherosclerosis was present in 163 (73%) patients, coronary thrombosis in 73 (33%), coronary arteritis in nine (4%), and ruptured coronary artery plaque in eight (2.7%). Coronary stenosis was present in 72% of the SCD cohort. In 17% of individuals, the stenoses were concentric and 55% had eccentric stenosis (grade 1: 26% of total cohort, grade 2: 11%, grade 3: 17%, and grade 4: 18%). Coronary plaque ulceration in the absence of thrombus was present in eight patients (3.5%). Mean mass of LV and right ventricle was 163 + 33 and 59 + 13 g, respectively. Twenty one percent of the individuals had LVH.

Of the cardiac causes, the most common LV pathology was MI in

Table 1
Baseline characteristics (n = 223).

Age, mean (SD),yrs	55 (10)
Gender, No.(%) (male)	176 (78.9)
BMI, mean (SD)	27 (4)
Known CAD, No. (%)	87 (39)
Hypertension, No. (%)	103 (46)
Diabetes mellitus, No.(%)	95 (43)
Smoker, No. (%)	133 (60)