Sudden Cardiac Death; Definition, Prevalence, Causes, Risk Factors and Management: Review Article

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ABSTRACT

Background: Sudden cardiac death (SCD) is described as an unexpected death that occurs within 1h of the symptom onset or during sleep in a previously fine person. SCD remains a significant public health concern globally because the majority of SCDs arise in persons with no previously diagnosed heart disease who do not follow the high-risk guidelines identified in observational studies and clinical trials. This review aimed to provide a summary of the occurrence, causes, and management of SCD. Conclusion: SCD is complex and has scientific and therapeutic concerns for a number of decades. As our understanding of this disease continues to progress through epidemiological research, laboratory experiments, and clinical trials, methods to reduce the prevalence and lethality of SCD through the community remain primary targets. Approaches and improved identification of high-risk individuals who will benefit from ICDs are critical to prevent SCD events and improve patient consequences.

Key Words: *occurrence, causes, management, sudden cardiac death*

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INTRODUCTION

Sudden cardiac death (SCD) is described as an unexpected death that occurs during sleep or within one hour of the symptom onset in a previously fine person [1]. SCD is generally used in cases where a patient dies unexpectedly without any signs that suggest an immediate danger of quick death in the next few minutes. It is still non-traumatic and can be spontaneous and immediate [2].

SCD remains a significant public health concern globally because the majority of SCDs arise in persons with no previously diagnosed heart disease who do not follow the high-risk guidelines identified in clinical trials and observational studies and is estimated to account for 15-20 percent of all deaths. Coronary artery disease is the most common cause of SCD, accounting for up to 80% of all cases [3, 4]. Annually, sports-related sudden death from any cause is 0.5 to 2.1 per 100,000 in general people. Sports-related, sudden deaths are higher in elite athletes with an incidence of 1:8,253 per year per the National Collegiate Athletic Association (NCAA) [5]. NCAA Division I male basketball players have a 1:5200 incidence of sudden death [6, 7].

Cardiomyopathies and genetic channelopathies account for the remaining causes. The most common causes of non-ischemic SCD are cardiomyopathy related to alcoholism, obesity, and fibrosis [8]. Multiple genetic, autonomic, structural, and clinical risk factors have been identified [9]. However, autopsy findings of patients with sudden death found nearly three-quarters of deaths due to heart disease and just nearly one quarter due to non-cardiac causes, mainly due to pulmonary embolism 18%, aortic collapse 4%, and intracranial bleeding 3% [10]. In patients aged 14 to 24 years, the cause of SCD is attributed to Marfan syndrome, Wolff-Parkinson-White syndrome, myocarditis, genetic channelopathies, congenital coronary anomalies,

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arrhythmogenic right ventricular cardiomyopathy (ARVC), and hypertrophic cardiomyopathy (HCM) [6].

ECG testing helps to detect ischemic heart disease, myocardial infarction, and genetic channelopathies. Echocardiograms assess signs of heart failure, cardiomyopathy, valve heart disorder, and congenital heart disease. Coronary angiography also assesses cardiac heart dysfunction, congenital heart problems, and coronary spasms [11].

Common antiarrhythmic drug therapy has not reduced and, in some cases, increased incidence of SCD when the implantable cardioverter-defibrillator (ICD) improves regeneration in high-risk patients [12]. The biggest improvement of SCD of patients with clinically evident heart disease stemmed from the use of beta-blockers [13].

Healthcare professionals should inform the family members of SCDs that they might still be at an elevated risk of ischemic heart disease and ventricular arrhythmias.

This review article aims to provide a summary of the occurrence, causes, and management of sudden cardiac death.

METHODS:

Study Design:

Review article.

Study duration:

Data was collected during the period from 1– 31 May 2020.

Data collection:

Medline and PubMed database searches were performed for articles about the sudden cardiac death, especially in Saudi Arabia and to provide a summary of the occurrence, causes, and management of SCD, published in English around the world. The keyword search headings include “occurrence, causes, management, SCD", and a combination of these was used. References list of each included study was searched for further supportive data.

Statistical analysis:

No software was utilized to analyze the data. The data was extracted based on the study objective. These data were reviewed by the group members to determine the initial findings. A double revision of each member’s outcomes was applied to ensure the validity and minimize the mistakes.

Prevalence:

Increased occurrence of SCD will be important consequence of this increasing population of patients with coronary disease and SCD [14]. A substantial delay in the implementation of successful risk stratification interventions and the avoidance of SCD can be specifically linked to a poor understanding of the pathways involved in fatal arrhythmogenesis [15]. In many investigations, the annual incidence of treated primary cardiac arrest ranges from 41 to 89/100,000 [16-18].

The recorded results occurrence of SCD in the US (a total population of about 300,000,000) is between 180,000 and 250,000 cases a year. For the globe (a global population of about 6,540,000,000), the projected average risk of SCD will be between 4 and 5 million cases each year [19]. SCD rates is 50-100 per 100,000 in the Netherlands, Ireland, and China's general population as well. A national survey of out of hospital cardiac arrests in Canada reported an incidence of (56/100,000) [20]. The annual incidence of 50-year-old men is approximately 100 per 100,000 populations compared to 800 per 100,000 for 75-year-old men [20]. The annual incidence of SCD-related atherosclerotic CAD is 0.7 per 100,000 in 18–35-year-old people and increases to 13.7 per 100,000 in those >35 years of age [21].

Causes:

Arrhythmia:

The most frequent causes of SCD are congenital defects in those 0–13 years of age, primary arrhythmia in the 14–24 year age range, and CAD in those with > 25 years old [22]. Monomorphic VT typically does not result in a loss of consciousness or SCD, it only tends to SCD if other medical circumstances relate to a lack of circulation or if it degenerates into ventricular fibrillation or polymorphic VT [23]. Polymorphic VT is the cause of SCD in nearly 25% of cases and is especially prevalent in acute myocardial ischemia [24]. SCD may even be the outcome of primary VF combined with acute myocardial ischemia or inherited channelopathies such as Brugada or long QT syndrome. Ventricular flutter and VF are responsible for 23% of out-of-hospital cardiac arrests treated by emergency care, which may result from initial VT degenerating into less coordinated rhythms [25]. Bradyarrhythmia leads to sudden death only in rare cases because, in most patients, endogenous secretion of catecholamines causes and maintains an escape rhythm that is enough to keep the patient alive [26].

Other Heart Disease:

Coronary heart disease or congestive heart failure considerably increases the risk of SCD. The occurrence of SCD after MI decreased in comparison with CHD mortality through time and was reported to be just as low as 1% a year in patients undergoing proper medical therapy and revascularization [27]. Even so, the incidence in some subsets of post-MI patients with SCD is still high. While open structural heart disease greatly raises SCD risk, most patients with cardiac arrest would not have LVEF reported less than 35% prior to SCD. Scar development after myocardial infarction is an important structural pre-requisite for re-entry into the unidirectional block [28]. This

is demonstrated in cardiac magnetic resonance imaging studies in which variation of the scar is a reliable indicator of ventricular arrhythmias. The occurrence of SCD due to coronary artery disease was already decreased by improved revascularization and the use of ACE inhibitors, beta-blockers, and statins [29]. SCD in DCM is most commonly triggered by ventricular tachyarrhythmia (50%), specifically VF, and may even be due to electromechanical dissociation and bradycardia. If SCD happens in the absence of heart failure or coronary artery disease, ARVC and HCM are the primary triggers. Many evidence on the occurrence of SCD in younger, healthier patients come from SCD research of athletes. Congenital cardiac disorders, myocarditis, Wolff – Parkinson – White Syndrome and Marfan Syndrome, with blunt trauma, commotio cordis and thermal pulse being less common causes [28-30].

Implanted defibrillator:

In ICD patients, up to 80% of all device-treated ventricular tachyarrhythmias is monomorphic ventricular tachycardia (VT). Patients with an ICD have differing annual mortality, the lowest (< 1 percent) among young patients without systemic heart disease, and the highest (> 7 percent) among patients with heart failure (especially ischemic heart disease) and the defibrillator for cardiac resynchronization therapy [31, 32].

Physical activity:

Physical exercise has both positive and negative impacts on the occurrence of SCD. Most studies have found inverse correlations between rising daily physical exercise and SCD or SCA. The results are more robust with modest levels of exercise where the majority of studies have reported beneficial correlations [33-36]. Rapid exercise decreases vagal activation due to an acute rise in the sensitivity of ventricular fibrillation, whereas daily exercise raises the basal vagal tone resulting in improved electrical cardiac consistency [37].

Psychological factors:

Anxiety, social isolation, lower socioeconomic status, depression, and psychological stress have been identified as a risk for cardiovascular mortality. Fearful anxiety has been associated with SCD but not non-fatal MI risk in 3 separate populations of women and men. Depression is a risk of cardiac arrest and SCD among women without CHD [38, 39].

Genetic factor:

Many studies have confirmed a genetic susceptibility to SCD. SCD events and lethal arrhythmias such as ventricular fibrillation (VF) are frequently the early signs of acute myocardial infarction and tend to be clustered in populations. Parental experience of fatal MI was only associated with an elevated risk of fatal MI and did not affect the likelihood of SCD [40]. Consistent correlations concerning the family history of arrhythmic mortality as an individual risk factor for SCD in the general population have led to many experiments based on the discovery of genetic variations that may affect the susceptibility of the population to ventricular arrhythmias and SCD [41].

Dietary factor:

Dietary intake and blood-based measures of identified nutrients have been directly correlated with SCD in observational studies. Several epidemiological findings indicate that elevated ingestion of n-3 polyunsaturated fatty acids (PUFAs) is inversely correlated with SCD to a greater degree than non-fatal MI [42]. Moreover, alcohol and magnesium consumption may have a selective impact on SCD risk. Strong alcohol use is associated with an increased risk of SCD. Mediterranean-style diet, consisting of higher intakes of vegetables, fruit, nuts, whole grains, fish, modest consumption of alcohol, and low intake of red/processed meat, has been linked with a lower risk of cardiovascular disease in clinical trials [43].

Others:

Modifiable risk factors for CHD that have been found to predict SCD in different studies include hypertension, hypercholesterolemia, diabetes, 67-69 renal impairment, obesity, and smoking [44, 45]. About 5% – 10 % of cases with SCD do not indicate underlying cardiac dysfunction at autopsy. Brugada syndrome caused by a genetic change of the sodium channel can cause loss of the action potential dome in the epicardium but not in the endocardium and accounts for about 20% of SCD in patients with structurally normal hearts [46].

Evaluation:

Emergency medical services (EMS) or laypeople can use an automatic external defibrillator (AED) before traveling to the hospital. Cardiac monitoring via ECG can occur until EMS arrives. ECG, echocardiogram, coronary angiography, fitness examination, electrophysiology, MRI, and cardiac biopsy may be used for complete cardiac assessment if the patient has survived. Screening of the first-degree relatives, particularly those with <35 years of age, is critical. When results indicate cardiomyopathy or hereditary channelopathy, it is often important to examine other family members [11, 47].

Management of Survivals and Individuals at Risk:

It could be necessary to recommend the placement of an ICD for primary prevention in patients that have been identified as at high risk for SCD. It is troubling that the epidemiology and clinical diagnosis of SCD-predisposing disorders are not normally treated in accordance with resuscitation research and procedure [48].

Targeted temperature management (TTM) was used to slow down pathophysiological activities and biochemical processes that trigger cell damage [49]. However, concerns persist about optimum optimal temperature, particular populations, period of hypothermia and methods of induction, restoration, and reversal. Extracorporeal membrane oxygenation (ECMO or eCPR) can be of possible benefit in patients with underlying congenital heart disease or where the reversible disease is present and where current procedures, staff, and facilities are developed and readily accessible [50].

CONCLUSION:

SCD is complex and had scientific and therapeutic concerns for a number of decades. As our understanding of this disease continues to progress through epidemiological research, laboratory experiments, and clinical trials, methods to reduce the prevalence and lethality of SCD through the community remain primary targets. Approaches and improved identification of high-risk individuals who will benefit from ICDs are critical to prevent SCD events and improve patient consequences.

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