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REVIEW ARTICLE

SUDDEN CARDIAC DEATH

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Sudden cardiac death (SCD) may be defined as unexpected death, usually instantaneous but uniformly fatal within one hour of onset of the terminal symptoms. If death is unwitnessed, the individual should have to have been seen alive in the past 24 hours for the death to be considered as SCD.[1] The highest incidence of SCD is in patients with coronary artery disease (CAD). Approximately 40% of patients may die due to acute coronary thrombosis and never reach hospital. In patients who survive the acute phase of myocardial infarction, the mortality rate in the first year is 1-20% with 50 of these deaths being sudden.

Subsequent mortality is 5% per year for at least five years, and many deaths are sudden.[2-3] Patients discharged from hospital following resuscitation showed, in one study, a 26% mortality rate at the end of one year and 36% at two years with three-quarters of the deaths due to recurrence of the sudden cardiac death syndrome.[4].

Other causes of SCD are: hypertrophic cardiomyopathy, dilated cardiomyopathy, acute myocarditis, valvular disorders such as aortic stenosis and mitral valve prolapse, electrophysiological abnormalities such as Wolff-Parkinson-White (WPW) syndrome, long QT interval syndrome, conduction tissues, congenital heart disease and arrhythmogenic right ventricular dysplasia.

Pathophysiologically three mechanisms have been involved, viz, left ventricular (L.V.) dysfunction, myocardial ischemia and ventricular arrhythmia.[3] In the overwhelming majority of individuals, SCD results from paroxysmal ventricular tachyarrhythmia, which usually strikes outside the hospital and without warning;[3] less frequently, SCD arises from brady-arrhythmias. 84% of SCD have been reported from ventricular tachyarrhythmia's and 16% from bradyarrhythmias.[6] Ventricular tachycardia (VT) usually precedes ventricular fibrillation (VF) The pathogenesis of VF includes acute myocardial infarction, new ischaemia, acute ischaemia and prior myocardial infarction, coronary reperfusion and remote myocardial infarction. Three mechanisms of SCD have been described: (a) VF of sudden appearance (primary) 8.2% which means that VF is usually preceded by one ventricular premature beat (VPB), i.e., R on T or by a very short run of VT; (b) VT (62%) or more rarely ventricular flutter that precipitates SCD usually through VF or, much less often, directly or through idioventricular rhythm; (c) Torsades de pointes (12.74%) occurs more frequently in patients receiving anti arrhythmic drugs and electrolytes (potassium, magnesium deficiency)[6]. An acceleration of heart rate is characteristically observed in the hours prior to SCD providing evidence that cardiac parasympathetic function is depressed in patients prone to SCD. The altered autonomic function contributes to development of electrical instability in many individuals, predisposing to lethal ventricular tachyarrhythmias.[5].

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PREDICTION OF SUDDEN DEATH

There is an urgent need to identify the various predisposing and precipitating factors that, in the presence or absence of SCD and ischemia, may lead to fatal arrhythmias. In men free of overt CAD, the risk factors for SCD cannot be distinguished from the classic risk factors for other manifestations of CAD[7]. The patients with ambulatory cardiac arrest in new study* had cardiac pain in 33% and dyspnoea in 26%. The overall incidence of ischemic events was higher than 70%. In patients who have been resuscitated from out of hospital cardiac arrest, there is a high incidence of ST segment depression in the exercise test performed after the resuscitation. Silent ischemia can trigger SCD while some reports suggest that unstable angina with silent ischemia has a poor prognosis and that stable angina patients with silent ischemia have more coronary involvement[6].

The pathologic process in sudden ischemic death involves a rapidly evolving coronary artery lesion in which plaque fissuring and resultant thrombus formation are present. Among 100 subjects who died of ischemic heart disease in less than 6 hours, coronary thrombi were found in 74. Among 26 eases without an intraluminal thrombus, plaque fissuring was found in 21 cases. Only 5 cases demonstrated no acute arterial lesion. There was no difference in incidence between those who died in less than 15 minutes, in 16-60 minutes or after one hour[8]. Cobb and colleagues have reported'[1] that if VF is associated with transmural infarction, there is only 2% rate of recurrence of VF or SCD in the year following resuscitation, compared to tenfold increase in patients without acute transmural infarction, who have chronic propensity for recurrence. Mukherjee documented 9 and 18% incidence of SCD in post myocardial infarction patients who had both frequent VPBs (more than 10 per hour) and LV dysfunction with radionuclide injection fraction less than 0.40. Congestive heart failure is responsible for 27.5% of deaths due to cardiac causes, and recurrent myocardial infarction for 34.5%[2].

Left ventricular hypertrophy is an independent risk for sudden death and acute myocardial infarction. The appearance of VPBs in asymptomatic hypertensive patients with LV hypertrophy is a grave sign and requires aggressive and specific therapeutic intervention. Low grade hypokalemia or other electrolyte disorders may have been a factor contributing to the increased ventricular ectopy[10]. Male individuals over 45 years of age are more likely to be victims of SCD. The incidence of SCD in women is delayed by 20 years compared to men. Diabetes mellitus is an independent risk factor which increases the cardiovascular mortality[1]. Daly et al showed[7] a lower total mortality in those who ceased smoking than the non-smokers and ex-smokers combined, and that stopping smoking reduced the rate of sudden death only in those with less severe coronary attacks. Both early and late onset of CAD appear to have a familial component which may or may not be genetic and act independently of the other risk factors.". Other variables related to incidence of SCD are obesity, vital capacity, use of digitalis and thiazide diuretics[9]. Attempts at defining risk stratification have been directed towards the identification of these factors.[3]

It is important to know the history, clinical examination and routine investigations of the patient for stratifications. Special investigations such as exercise testing, 24 hours Holler monitoring, echocardiography, radionuclide study and the new technique of recording a signal averaged ECG to look for delayed potentials may add to the accuracy of clinical assessment. Cardiac catheterization may be used to document the severity of CAD and LV function. Programmed

ventricular stimulation may identify high risk patients in terms of whether or not they develop sustained monomorphic VT and to guide therapy with various antiarrhythmic agents[5].

MANAGEMENT AND PREVENTION

Cardiac arrest often without warning symptoms may occur when medical attention is not immediately available. Survival is critically dependent on prompt cardiopulmonary resuscitation and delivery of definitive care, in particular dcfibrillation[12]. Primary VF is common in patients with ischemic heart disease. Prevention of death in these patients requires rapid application of prophylactic measures. [,3]. Ambulance staff trained in defibrillation techniques can successfully resuscitate patients from cardiac arrest outside hospital. However, the capability of paramedical staff is limited to stabilizing the hemodynamic state of the patients'[2]. Coronary care unit management of such patients may be necessary for cardiovascular support in the first 24 to 72 hours[4]. Koster and colleagues reported [13] a bolus of intramuscular lignocaine given by the paramedic reduced the incidence of primary VF in patients with acute myocardial infarction. There was 31% reduction in potential mortality in the treated group during the 60 minutes after the injection.

Beta blocking agents, antiarrhythmic drugs, antiplatelet agents, coronary artery bypass graft surgery, myocardial surgery and psychophysiological intervention have been proposed as possible means of preventing SCD[4]. Antiarrhythmic drug prophylaxis has generally been disappointing. None of antiarrhythmic agents has a favourable effect on the incidence of SCD or non-SCD between two groups of patients randomized to mexiletine or placebo[3]. Beta blockers are the only agents shown to exert a favourable effect on the incidence of SCD. They decrease the myocardial oxygen requirement rather than suppress arrhythmias. Procainamide appears to be an effective antiarrhythmic agent, preventing the initiation of sustained VT in 30% to 35% of cases. Class I antiarrhythmic agents such as flecainide and mexiletine do not appear to be better. Amiodarone is the most effective antiarrhythmic agent for the prevention of recurrent sustained VT. It has been effective in 62% of patients with documented sustained VT or VF, who were refractory to other antiarrhythmic agents. Overall, proarrhythmic effects occur in approximately 13% of patients in drug trials; in particular class Ic drugs aggravate ventricular arrhythmias[3]. The use of two class Ic antiarrhythmic agents, flecainide and encainide in CAST[14] to treat asymptomatic or minimally symptomatic or minimally symptomatic ventricular arrhythmias in patients after myocardial infarction is associated with a substantial increase in the sudden death rate and total mortality.

The automatic implantable cardioverter defibrillator and cardioverter is another option for patients who fail to respond to drug therapy.

Antiplatelet agents like aspirin in unstable angina have reduced total mortality by 51% at 3 months and 56% at 18 months[3].

Most deaths from CAD are sudden, unexpected events which occur outside the hospital. The prevailing concern for the patient resuscitated from out-of-hospital VF is the relatively high risk rate of recurrence, leading to considerable anxiety for the patient, family and doctor[1]. The integration of paramedical with medical prehospital coronary care has improved survival after out-of-hospital Cardiac arrest. Prehospital coronary care schemes dependent on paramedical staff acting alone

reported survival rates of 36% to 63% in such patients. A Community Training Programme in cardiopulmonary resuscitation improves the efficacy of prehospital coronary care[12] especially training for family members.

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