Editorial

Sudden Cardiac Death (SCD) -One of the most difficult challenges in modern cardiology.

Introduction:

Sudden cardiac death is a challenging problem to the cardiologists all over the world. As it involves a widely varied array of clinical and pathological conditions, understanding the mechanism leading to sudden cardiac death poses a problem. Even some different opinion among different authors regarding its definition.

Definition:

Sudden cardiac death may be defined as -unexpected natural death from a cardiac cause -i. within a short time period,generally = 1 hour from the onset of the symptoms.ii. In a person without any prior condition that would appear fatal.3,4,Prodromal symptoms may be present but are often non specific.40% sudden deaths are unwitnessed.

Epidemiology: Incidence:

No such study on SCD is available in Bangladesh. SCD accounts for more than 400,000 deaths yearly in the United States, with exact totals depending on the definition used.6,8. When the definition of SCD is restricted to death less than 2 hours from onset of symptoms, 12% of all natural deaths are sudden and 88% of these are a result of cardiac disease. In autopsy-based studies, a cardiac etiology of sudden death has been reported in 60% to 70% of sudden death victims.7

Risk Factors for SCD:

Hypertension, Diabetes mellitus, Family history of SCD, Dyslipidaemia, Smoking, Alcohol, History of IHD, Heart Failure, On ECG- LVH, Arrhythmia, PVCs On CXR- Enlarged heart, Physical inactivity, Obesity.

CLINICAL FEATURES OF THE PATIENT WITH CARDIAC ARREST &SCD[1,2]- Clinical cardiac arrest and SCD can be described in the framework of four phases of the event used to establish

temporal definitions-Prodromes, Onset of the terminal event,The cardiac arrest, and Progression to biological death or survival.

Prodromal Symptoms: Patients at risk for SCD can have prodromes such as chest pain, dyspnoea, weakness or fatigue, palpitations, syncope, and a number of nonspecific complaints.

Onset of the Terminal Event: The period of 1 hour or less between acute changes in cardiovascular status and the cardiac arrest itself is defined as the "onset of the terminal event." Ambulatory recordings fortuitously obtained during the onset of an unexpected cardiac arrest have indicated dynamic changes in cardiac electrical activity during the minutes or hours before the event.5

Cardiac Arrest: Cardiac arrest is characterized by abrupt loss of consciousness caused by lack of adequate cerebral blood flow. It is an event that uniformly leads to death in the absence of an active intervention, although spontaneous reversions occur rarely. The most common cardiac mechanism is VF, followed by asystole-pulseless electrical activity and sustained VT. Other mechanisms include rupture of the ventricle, cardiac tamponade, acute mechanical obstruction to flow, and acute disruption of a major blood vessel.

Progression to Biological Death: The time course for progression from cardiac arrest to biological death is related to the mechanism of the cardiac arrest, the nature of the underlying disease process, and the delay between onset and resuscitative efforts. The onset of irreversible brain damage usually begins within 4 to 6 minutes after loss of cerebral circulation related to unattended cardiac arrest, and biological death follows quickly.

Mechanism of SCD[1, 2]:

Relationship between Structure and Function in SCD: The vast majority of patients who have experienced SCD have cardiac structural abnormalities. In the adult population, these consist predominantly of CHD, cardiomyopathies, valvular heart disease, and abnormalities of the conduction system.

Tachyarrhythmias versus Bradya-rrhythmias in SCD: Ventricular fibrillation is the first recorded rhythm in approximately 75% of patients who have cardiac arrest.9 Sustained ventricular tachycardia is only rarely (less than 2%) documented as the initial rhythm, but it is unknown how often it precedes and precipitates ventricular fibrillation.Electromechanical dissociation and asystole are found in approximately 30% of patients experiencing cardiac arrest, and this finding is usually related to the time interval from collapse to first monitoring of the rhythm, suggesting that it is often a later manifestation of cardiac arrest.[9]

Mechanoelectrical Feedback: Left ventricular dysfunction has been identified as the strongest independent predictor of SCD.10 Despite the clinical recognition that acute heart failure can precipitate ventricular tachyarrhythmia's, the mechanism by which this occurs is incompletely understood. Besides mechanisms related to acute and chronic ischemia, it has been shown that acute changes in the mechanical state of the heart related to altered preload and contractility can have direct electrophysiologic effects that may precipitate arrhythmias; this relationship is referred to as mechanoelectrical feedback.

Cardiac abnormalities associated with SCD:

i. IHD: Coronary atherosclerosis (CAD-AMI, Ischemic Cardiomyopathy),Anomalous origin of coronary arteries,Hypoplastic coronary arteries,Coronary artery dissection,Coronary arteritis.

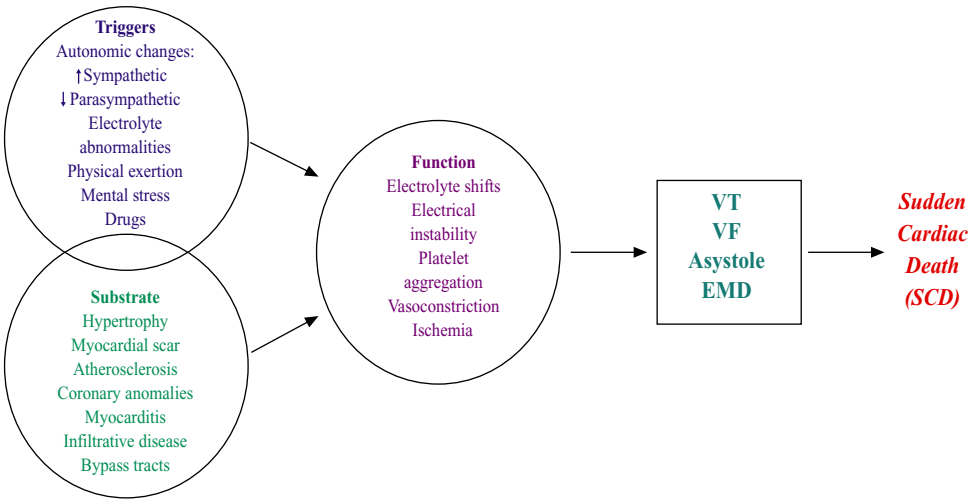

Fig 1: *Interaction between structural cardiac abnormalities, functional changes, and triggering factors in the pathophysiology of SCD. The role of triggering factors, such as changes in autonomic tone or reflexes, is increasingly being recognized. EMD(electromechanical dissociation); VT(ventricular tachycardia.); VF(ventricular fibrillation);*

Electrophysiologic Effects of Ischemia: Within the first 3 days of myocardial infarction, SCD may occur as a result of ventricular fibrillation initiated by early, frequent premature ventricular complexes (PVCs). Such PVCs have been shown in experimental models to be predominantly caused by impulse formation consistent with abnormal automaticity.

ii. Non Ischemic heart disease:

Cardiomyopathies,Infiltrative & inflammatory heart disease,Valvular heart disease (AS, AR, MVP), Congenital heart disease (TOF, TGA, Ebstein's, PVOD, AS), Primary electrical abnormalities (long Q-T syndrome, WPW, CHB, Brugada's syndrome, Jervell-Lange- Nielsen syndrome)

iii. Drug induced and other toxic agents: Anti arrhythmic drugs - Class 1A, Class III, Class IV;Non anti arrhythmic drugs-Erythromycin, Terfenadine, Pentamidine,Psychotropic drugs, Cocaine Alcohol,Phosphodiesterese inhibitors, Diuretics.

iv. Electrolyte abnormalities: Hypokalaemia,Hypomagnesemia,Hypercalcaemia.

v. Others: Abnormalities of autonomic nervous system during myocardial infarction may cause ventricular arrhythmia

Reduction of SCD:

Incidence of SCD is reduced by taking following measures-

Evaluation of a Patient and Risk stratification by-

History: A prior history of cardiac arrest is the most significant risk factor for recurrent cardiac arrest. Structural heart diseases which are considered as substrates for SCD.

Family history- sibling having congenital long QT, syncope, hypertrophy in familial idiopathic cardiomyopathy ,Drug history - that may prolong QT,In children- syncope, pre-syncope, dyspnoea, chest pain on exertion, palpitation- premonitoring features in children who die suddenly.Family with a history of syncope, arrhythmia, Marfan syndrome, HCM, sudden or unexplained death.

Investigations: Coronary perfusion e.g. CAG, ETT, Holter, ST-T changes in ECG, Pump function e.g. LVEF, NYHA classification, Arrhythmias e.g. Holter, Signal averaged ECG, QT interval, dispersion, T wave abnormality, Neurohumoral e.g. heart rate variability, baroreflex sensitivity, Psychosocial e.g. depression

1. Primary prevention:

- **i. Pharmacological management-** B blocker and amiadarone-can reduce frequency of sudden cardiac death after MI. Statin, ACEi ,aspirin,clopidogrel-Prevention of Plaque rupture,ACEi,B blocker- Stabilizing Autonomic function.ACEi and B blocker- Improving pump function, B blocker&Anti-Arrhythmic drugs-Preventing arrhythmia.
- **ii. Intervention:** PCI-PTCA with or without stenting-to reduce coronary heart disease, Automated Implantable Cardiac Defibrillator (CRT/AICD): Treatment of choice in patient of: Documented VT(non MI), VT (hemodynamically poor tolerated), History of unexplained syncope in impaired LV function. EP study & radiofrequency catheter ablation: to prevent sustained ventricular arrhythmia.
- **iii. Cardiac surgery-** CABG and anti-arrhythmic surgery-cardiac reconstructive surgery.

2. Secondary prevention:

If patients successfully resuscitated from an episode of cardiac arrest the secondary prevention is needed. As most causes of SCD occurs in the population of CAD, secondary prevention therapy is directed to the patient with CAD specially to survivors of AMI. Following measures should be taken- Anti-ischaemic drugs, Anti arrhythmic drugs, CRT/AICD, Revascularization-PCI or CABG, cardiac reconstructive surgery.

3. Resuscitation- Out of Hospital-

Promptness and restoring circulation is essential, it is done by Basic Life support (BLS) followed by Advanced Cardiac Life Support (ACLS) and cardiac Defibrillator (At hospital).

Conclusion:

It is evident that as SCD is a challenge even in this era of modern cardiology with hi-tech support system. High degree of suspicion, proper & detail history taking ,meticulous physical examination & necessary investigations are advised to triage the patient for the prevention of Sudden Cardiac Death (SCD) by cardiologists & at the same time need to grow attention & awareness for in-time referral of risky patients by other physicians & also health education for all.

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