

Short Communication**Out of Hospital Sudden Cardiac Arrest**Vanita Arora*, Pawan Suri¹, Vivek Kumar²

1. Dr. Pawan Suri MD; DM; FSCAI; FESC Chief Cardiologist, SGL Super specialty Hospital, Jalandhar, Punjab.

2. Dr. Vivek Kumar MD; DM Consultant Interventional Cardiologist Max Healthcare Super specialty Hospital, Saket, New Delhi.

***Corresponding Author: Dr. Vanita Arora** MD; DNB (Cardiology); FRCP (Edin)

Director & Head Cardiac Electrophysiology Laboratory & Arrhythmia Services
Max Healthcare Super Specialty Hospital, Saket, New Delhi, India.

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Out of hospital cardiac arrest (OHCA) and the resulting sudden cardiac death, is a major unmet need in the healthcare system worldwide. The WHO defines SCD as a sudden unexpected death within 1 hour of symptom onset or within 24 hours of having been last seen well. The exact incidence of SCD in the general population worldwide is difficult to ascertain, but in the western population, it is estimated to account for 20% of deaths. (1) By one estimate for an unselected adult population 35 years and older worldwide, the overall incidence is calculated to be in the range of 0.1% to 0.2% per year (1 to 2 per 1000 population). The temporal trend of SCD by age indicates two peaks: within the first year of life and between 45 and 75 years of age. (2)

The cause of SCD in infants are mostly complex congenital heart diseases (3). In children, adolescents, and young adults the incidence of SCD is less and mostly unexplained. But genetic causes were confirmed in 27% of such cases in a post-mortem study.

Coronary artery disease (CAD) becomes a dominant cause of SCD beyond the age of 30 years. Then the incidence of SCD over time parallels the increase in the prevalence of coronary heart disease with advancing age (4). But rare disorders, such as hypertrophic cardiomyopathy,

Brugada syndrome, long-QT syndrome, and right ventricular dysplasia, remain significant contributors for SCD in the young adult population.

Overall there is a male preponderance in SCD syndrome relative to women during the young adult and early middle-age years because of the lesser CAD events in women before menopause (5). But even afterward as a risk for coronary events increases in postmenopausal women and risk for SCD increases proportionately, still men remain at higher risk than women across the entire age spectrum.

Apart from age and sex, family history of SCD is a very important risk factor. Familial incidence SCD is because of various rare but important hereditary disease of primary arrhythmic syndromes (e.g., long-QT syndromes, Brugada syndrome, catecholaminergic polymorphic ventricular tachycardia/fibrillation) or structural diseases associated with risk for SCD (e.g., hypertrophic cardiomyopathy, right ventricular dysplasia) (6).

In the acquired causes, CAD is the most common cause for SCD, and it is plausible that the risk factors for CAD would be the risk factors for SCD e.g. diabetes, dyslipidaemia, hypertension, smoking, obesity, and sedentary lifestyle. Approximately 50% of all SCDs occur in 10% of the population in the highest-risk group based on multiple risk factors.

A reduction in the left ventricular ejection fraction (EF) is the most powerful of the known predictors of total mortality and SCD in patients with chronic ischemic heart disease, as well as other acquired structural disease (e.g. dilated cardiomyopathy, valvular heart disease) (7).

Additionally, left ventricular hypertrophy is an independent risk factor for SCD which is often associated with scarring and subendocardial ischemia. The resulting electrical inhomogeneity is a physiologic contributor to mechanisms of potentially lethal arrhythmias.

Though SCD is considered a sudden but insignificant number of cases there could be prodromal symptoms that are often neglected which occur weeks or months before an event, are generally predictors of an impending cardiac event, but not specific for SCA itself.

Such symptoms include chest pain, dyspnoea, fatigue, palpitations, syncope. The patient may even have visited a physician for such symptoms. Ultimately symptoms that occur within the last hours or minutes before cardiac arrest may include symptoms of arrhythmias, ischemia, or heart failure. Loss of responsiveness marks the onset of the terminal event and signifies SCA.

The loss of consciousness is caused by a lack of adequate cerebral blood flow because of the failure of the cardiac pump function. The most common electrical mechanism of out of hospital cardiac arrest (OHCA) as per the available data is asystole (50%), followed by VF/pulseless VT and pulseless electrical activity (PEA) (25% each) (8).

Cardiac arrest survival depends upon how soon the patient receives resuscitation measures (BLS/ACLS) after the terminal event and how soon the return of spontaneous circulation (ROSC) occurs. The irreversible brain damage usually begins within 4 to 6 minutes after the loss of cerebral circulation, and biologic death may follow quickly in unattended cardiac arrest. Younger patients with less severe cardiac disease and the absence of coexistent multisystem disease may survive some resuscitation delays.

Management of OHCA survivor is complex and the prognosis remains bad. Patients who arrive in a coma majority may not awake and the most common causes of death are CNS injury, including anoxic encephalopathy and sepsis. Patients with short resuscitation delay and not so prolonged time lag before ROSC may have a better prognosis. Post ROSC ECG may help in such patients regarding the future course of management, as the need for a coronary angiogram and subsequent intervention. Apart from intensive care, therapeutic hypothermia in patients with post-cardiac arrest coma is considered beneficial.

Primordial prevention includes measures directed towards the prevention of CAD and its subsequent events. A person vulnerable to develop SCA because of familial or genetic diseases as mentioned above needs to be properly evaluated and followed up and if needed Automated Implantable Cardioverter Defibrillator (AICD) should be done. Similarly, GDMT should be given to patients with LV dysfunction, and indication for CRTD/AICD should be assessed and implanted. Patients with valvular or congenital heart disease should be treated timely before ventricular dysfunction sets in. The above measures are meant to be delivered by a cardiologist/electrophysiologist.

One important intervention that can save valuable lives is community intervention in form of BLS training to public servants and availability of Automated External Defibrillators (AED) at public places to facilitate early defibrillation by “first responders”. Patients with an initial rhythm of VT/VF have better survival than those with asystole/PEA (9).

Proper training of paramedical staff/nurses/healthcare workers/bystanders to be the first responders saves lives. Schools, clubs and organizations sponsoring athletic events should also have an established emergency response plan for SCA (10).

Essential elements of an emergency response plan include an effective communication system to alert “first responders” and retrieve the AED, training of anticipated responders in CPR and AED use, access to an AED for early defibrillation, integration of on-site AED program with the local emergency medical services system and practice and review of the response plan.

References

- (1) Wong CX, Brown A, Lau DH, et al. “Epidemiology of sudden cardiac death: global and regional perspectives”. *Heart Lung Circ* 2019; 28:6–14. doi:10.1016/j.hlc.2018.08.026
- (2) Atkins DL, Everson-Stewart S, Sears GK, et al. “Epidemiology and outcomes from out-of-hospital cardiac arrest in children: The Resuscitation Outcomes Consortium Epistry—Cardiac Arrest”. *Circulation*. 2009; 119:1484.
- (3) Bagnall RD1, Weintraub RG1, Ingles J1, et al. “A prospective study of sudden cardiac death among children and young adults”. *N Engl J Med*. 2016; 374:2441.
- (4) Myerburg RJ. “Sudden cardiac death: exploring the limits of our knowledge”. *J Cardiovasc Electrophysiol*. 2001; 12:369.
- (5) Bogle BM, Ning H, Mehrotra S, et al. “Lifetime risk for sudden cardiac death in the community”. *J Am Heart Assoc*. 2016;5(7).
- (6) Kannel WB, Shatzkin A. “Sudden death: lessons from subsets in population studies”. *J Am Coll Cardiol* 1985;5 [Suppl 6]:141B.
- (7) Stecker EC, Vickers C, Waltz J, et al. “Population-based analysis of sudden cardiac death with and without left ventricular systolic dysfunction”. *J Am Coll Cardiol*. 2006; 47:1161.
- (8) Field JM, Hazinski MF, Sayre MR, et al. “Part 1: Executive summary. 2010 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care”. *Circulation*. 2010;122(suppl 18): S 640.
- (9) Bernard SA, Gray TW, Buist MD, et al. “Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia”. *N Engl J Med*. 2002; 346:557.

(10)J A Drezner: “Preparing for sudden cardiac arrest—the essential role of automated external defibrillators in athletic medicine: a critical review”; British Journal of Sports Medicine September 2009 - Volume 43-9 doi.org/10.1136/bjism.2008.054890.

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