REVIEW PAPER

SUDDEN CARDIAC DEATH IN NIGERIA: PATHOPHYSIOLOGY AND EVIDENCE BASED INTERVENTION STRATEGY

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ABSTRACT

Sudden death is an emerging phenomenon in Nigeria, a country with a burden of preventable communicable and non communicable diseases. The rapid adoption of western lifestyle, the almost complete dependence on vehicular transport as well as stress that accompany infrastructural development and processes of human development act synergistically to predispose individuals to preventable non-communicable atherosclerotic diseases, which culminate in sudden cardiac death. Health education, which teaches individuals and families in the community to value health as a personal asset and which modifies noxious behavioral lifestyles into health promoting behavioral lifestyles, is capable of preventing non-communicable diseases that are direct causation of sudden death. There is therefore a need to research into the direct causes of sudden cardiac death in Nigeria with the aim of providing evidence-based information to the policy makers for sustainable social environmental and behavioral modification that will nick this ugly trend in the bud.

Key words: Sudden cardiac death, Nigeria, Pathophysiology, Predisposing factors.
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INTRODUCTION

A sudden cardiac death is a natural death due to cardiac causes, heralded by abrupt loss of consciousness within one hour of the onset of acute symptoms; pre-existing heart disease may have been known to be present, but the time and mode of death are unexpected (Myerburg and Castellanos, 2007). On the other hand, it is important to point out that the operational definition of Sudden Cardiac Death differs among clinicians, epidemiologists, pathologists and scientists attempting to clarify the patho-physiological mechanism (Myerburg and Castellanos, 1997). It is estimated that sudden cardiac death accounts for up to 50% of all heart disease related deaths (Engelstein and Zipes, 1998). The dramatic nature of sudden cardiac death in Nigeria makes many people believe it is a voodoo initiated phenomenon.

Sudden cardiac death is a major health problem in developed countries. It is however an emerging problem in developing countries, especially the least developed countries (Zipes and Wellens, 1998). It has been said that the incidence of sudden cardiac deaths in Nigeria is largely unknown (Ogunlade, 2011). However, in a survey of 2,529 medico-legal autopsies over a 10 year period (1987-1997) in Ile-Ife, Nigeria, only 79 (3%) cases of sudden cardiac death were recorded (Rotimi et al, 2004). The reason for the lower incidence in Nigeria may be under-reporting. The lower incidence may also be explained by the use of unadulterated edible seeds, nuts and legumes (Eilat-Adar et al., 2013) as food by most Nigerians especially those in rural areas. These nutrients may have a beneficial effect on blood lipids as well as other coronary heart disease risk factors such as oxidation and inflammation. It is also possible that the substitution of high saturated fatty acids, sodium, and sugary food by nuts, whole grains, legumes and dietary fibers can also explain this beneficial effect (Eilat-Adar et al., 2013).

However, human behavior is not static. Therefore as individuals and families in communities are challenged by higher income, new environmental conditions and opportunities, behavioral lifestyles that predispose to sudden cardiac death are acquired. These include urbanization and acquisition of new dietary habits, vehicular transport and consequent physical inactivity. Dietary changes associated with urbanization are related to the fact that rural dwellers tend to be more self-reliant in obtaining food and also tend to eat traditional diets that are high in grains, fruit and vegetables, but low in fat. Once they arrive in urban areas, these same people tend to rely more on processed food for sustenance due to external forces such as socioeconomic conditions, resulting in a shift from production of their own food to the purchase of processed foods which predispose to obesity. This may be accompanied by over indulgence in sugary and alcoholic beverages. According to Hoffman, (2001), these processed foods such as high fat diets, high energy dense carbohydrate diets and alcoholic beverages, are often referred to as western diet.

CLASSIFICATION

Sudden cardiac deaths are generally of two main types:

- Atherosclerotic sudden cardiac death
- Genetic sudden cardiac death.

Atherosclerotic Sudden Cardiac Death

This is mainly due to coronary artery disease and certain acquired cardiomyopathies, which tend to occur at the age of 35 years and above. The risk of sudden cardiac death from atherosclerosis is 1-2 per 1000 per year (Myerburg and Spooner, 2001). Ingestion of excess quantities of animal fat and saturated fat of vegetable origin predisposes to atherosclerosis (Siri-Tarino et al., 2010), which accounts for 80% of all sudden cardiac deaths due to coronary artery disease (Mazeika, 2001). However, hypertensive heart disease was responsible for 83.5% of sudden cardiac death in a Nigerian study (Rotimi et al, 2004). Hypertension, a major risk factor for coronary heart disease, is strongly associated with obesity, physical inactivity, sodium intake, and alcohol intake. In fact, a study in Benin City, Nigeria, reported the most common cause of sudden cardiac unexpected natural death were cardiovascular diseases resulting from complication of Hypertension (Aligbe et al., 2002). Obesity, physical inactivity, and smoking are the major modifiable risk factors for diabetes mellitus, which is another important cause of coronary heart disease. Serum cholesterol, an established risk factor for coronary heart disease, is also influenced by diet and physical activity (Maruthur et al., 2009).

Sudden Cardiac Death of Genetic Origin

Certain cases of sudden cardiac death display a strong familial pattern and these include the following (Myerburg and Spooner, 2001):

- Long QT interval syndromes
- Familial hypertrophic cardiomyopathies
- Right ventricular dysplasia
- Brugada's syndrome and
- Idiopathic ventricular Fibrillation

These tend to manifest at ages less than 35 years. The risk of sudden cardiac death from this variant is about 1 per 1000,000 per year (Myerburg and Spooner, 2001). The most common inherited cardiac diseases have an autosomal-dominant pattern of inheritance. This implies that half of the first-degree relatives are at risk of having or developing the same disease (Myerburg and Spooner, 2001).

EPIDEMIOLOGY

Sudden cardiac death is found all over the world though it is by far more common in the developed countries. It is more common in males above the age of 35 years. It is said to be especially common in affluent persons who abuse alcohol and eat excess of fatty meat and saturated fatty acids. It is also common among sedentary obese persons, especially those who are smokers, hypertensive and diabetic (Maruthur et al., 2009). Stress may be an exacerbating

factor. Around 80% of sudden cardiac deaths occur in the home (de Vreede-Swagemakers et al., 1997), 40% are unwitnessed, and bystander cardiopulmonary resuscitation is initiated in only 10%–20% of cases (Mazeika, 2001).

PATHOGENESIS

This depends on the type. Atherosclerotic sudden cardiac deaths are believed to occur as a result of myocardial infarction following coronary artery thrombosis or rupture of atherosclerotic plaques. The immediate patho-physiological events that culminate in sudden cardiac death in individuals with coronary atherosclerosis include atheromatous plaque vulnerability, consequent thrombosis, and electrical instability (Mazeika, 2001). The resulting ischemia provokes ventricular arrhythmias, which may be aggravated by vigorous unaccustomed exercise and profound emotional stress. Myocardial ischemia has therefore been suggested as the initiator of the terminal event (Mazeika, 2001). Arrhythmias may also be precipitated by diuretics therapy, which cause hypokalemia and drugs, such as antihistamines, anti-depressants and major tranquillizers, which can prolong QT interval (Mazeika, 2001).

Myocardial infarction may occur as a result of the combination of a complex interaction of multiple predisposing factors. A change in lifestyle due to sudden affluence may encourage an individual to eat excessively and depend on vehicular transport rather than take a walk. Both changes predispose the individual to obesity, hyperlipidemia and subsequently coronary atherosclerosis. Environmental stress predisposes to emotional disturbances, which may induce the individual to engage in smoking and alcohol abuse as well as predispose him to hypertension especially if he is above 35 years of age (Park, 2007). While hypertension causes thickening of arterial walls, smoking induces the production of increased quantities of catecholamines. Coronary atherosclerosis in the presence of thickened arterial walls predispose to coronary occlusion, myocardial ischemia and finally myocardial infarction (Park, 2007).

Genetically determined arrhythmogenic entities including ion channelopathies and catecholaminergic ventricular tachycardia have been regarded as important etiologies of Sudden Cardiac Death, particularly in young victims (Yalta et al., 2010). Genetic mutations are believed to be the basis of Sudden Cardiac Death susceptibility in individuals with rare inherited arrhythmias. These mutations may cause alterations in structure and function of membrane elements involved in electrogenesis and trans-cellular activation at the level of ion channel exchange through gap junction proteins. This may occur during disease induced cardiac remodeling, or in response to hypertension, structural failure or aging (Myerburg and Spooner, 2001).

CLINICAL FEATURES

Sudden cardiac death (SCD) may be the first and final manifestation of several heart diseases. There may or may not be evidence of existing cardiac disease. Most affected persons complain of severe constricting chest pain (angina pectoris) before the loss of consciousness. For those who are still alive when they are brought to the hospital, there may be features of acute respiratory distress syndrome and aspiration pneumonitis. Seizures are common occurrences due to anoxic encephalopathy (Mazeika, 2001).

INVESTIGATIONS

These are not commonly available in many hospitals in most developing countries including Nigeria. According to Mazeika (2001), they include

- Coronary angiography, stress testing
- Coronary artery spasm
- Ergonovine study
- Endomyocardial biopsy
- Echocardiography
- Magnetic resonance imaging,
- Signal averaged electrocardiogram.

TREATMENT

Management of a victim of cardiac arrest follows cardiopulmonary resuscitation, early cardiac defibrillation and cardiac life support. In most cases, the affected person dies before getting to the hospital. This is especially true in developing countries, where ambulance services are poor. Even where ambulance services are available, the delays

due to decision making, contacting the ambulance service, and bad roads make it almost impossible to get the affected person to the health facility alive. Besides, the primary and secondary health facilities, which are relatively close to most people, have no facilities for the management of sudden cardiac arrest. In developed countries, defibrillators are stationed in strategic places in the hospital and can be used in resuscitation of affected persons.

PREVENTION OF SUDDEN CARDIAC DEATH

The events leading to sudden cardiac death require technologically advanced diagnostic facilities, which are only available in a few tertiary health facilities in Nigeria. Furthermore, the management of sudden cardiac death requires implantable Cardioverter Defibrillator, which is hardly available in the best centers in developing countries.

Since advanced technological facilities are not available for early diagnosis and treatment, it is imperative that preventive strategies be developed through evidence based research into behavioral life styles, environmental stresses and factors that are conducive to sudden cardiac death. Health education methods, that can be used to limit the access of individuals and families in communities to adverse environmental and nutritional factors, should be developed through research.

It is also necessary to research into specific protection such as food fortification and supplementation. Certain fish oils found in sardines, which contain polyunsaturated fatty acids have been suggested as being capable of inhibiting coronary atherosclerosis (Seeley et al., 2011). Though a recent meta-analyses of randomized controlled trials found little evidence of a protective effect of omega-3 supplementation on the incidence of cardiovascular disease (Kwak et al., 2012), a review of 11 cohort studies involving 116,764 individuals suggested that fish consumption at 40–60 g daily is associated with markedly reduced coronary heart disease mortality in high-risk, but not in low-risk populations (Marckmann and Grønbaek, 1999). However, Omega-3 fatty acids have been shown to increase arrhythmic thresholds, reduce blood pressure, improve endothelial function, reduce inflammation and platelet aggregation, enhance plaque stabilization, and favorably affect autonomic tone (Kromhout et al., 2012). According to Mazeika (2001), sudden cardiac death is potentiated in individuals with coronary atherosclerosis by atheromatous plaque vulnerability, consequent thrombosis, electrical instability and ventricular arrhythmias. By implication, the enhancement of plaque stability and arrhythmic thresholds may be useful in the prevention of sudden cardiac death.

Also, Alpha-linolenic acids; are essential fatty acids which are convertible in-vivo to omega-3 fatty acids such as eicosa-pentanoic acid (EPA) and docosa-hexanoic acid (DHA), which can be used to synthesize prostaglandins, which prevent blood clotting (Seeley et al., 2011). It is known that eating sardine two or three times a week, lower the risk of heart attack, which is why Seeley et al. (2011), encourages persons who do not like eating fish to drink the oils from sardines. This principle can be used on a global scale by fortifying edible oils with EPA and DHA.

CONCLUSION

Based on the available facts on the etiology of Sudden Cardiac Death, it is suggested that locally available foods be analyzed with an aim of limiting the use of cholesterol rich foods, which predispose to coronary atherosclerosis. The closer to nature one is, the less the likelihood of developing predisposing factors to sudden cardiac death. Also, persons above the age of 35 years should undergo regular blood pressure check for early detection of hypertension which is the commonest predisposing factor to sudden cardiac death in Nigeria.

Finally, research should be done to determine the best methods of modifying behavioral life styles in the noveau-riche, who are likely to develop hyperlipidemic states of obesity, diabetes mellitus, hypertension, coronary atherosclerosis and finally sudden cardiac death.

REFERENCES

Aligbe, J.U., Akhiwu, W.O. and Nwosu, S.O. (2002). Prospective study of coronar's autopsies in Benin City, Nigeria. *Med. Sci. Law.;* 42: 318-324.

Eilat-Adar, S., Tali Sinai, T., Chaim Yosefy, C. and Yaakov Henkin, Y. (2013). Nutritional recommendations for cardiovascular disease prevention. *Nutrients*. 5(9): 3646–3683.

Engelstein, E. and Zipes, D. (1998). The heart, arteries and veins. McGraw-Hill, New York. pp. 1081-1112

Hoffman, D.J. (2001). Obesity in developing countries: Cause and implications. *Food Nutr. Agric.;* 28: 35-44.

Kromhout, D., Yasuda, S., Geleijnse, J. M. and Shimokawa, H. (2012). Fish oil and omega-3 fatty acids in cardiovascular disease: do they really work? *Eur. Heart J.;* 33(4):436-43.

Kwak, S.M., Myung, S.K., Lee, Y.J. and Seo, H.G. (2012). Efficacy of omega-3 fatty acid supplements (eicosapentaenoic acid and docosahexaenoic acid) in the secondary prevention of cardiovascular disease: a meta-analysis of randomized, double-blind, placebo-controlled trials. *Arch. Intern. Med.*; 172(9):686-94.

Marckmann, P. and Grønbaek, M. (1999). Fish consumption and coronary heart disease mortality. A systematic review of prospective cohort studies. *Eur. J. Clin. Nutr.;* 53(8):585-90.

Maruthur, N.M., Wang, N. and Appel, L.J. (2009). Lifestyle interventions reduce coronary heart disease risk. *Circulation;* 119(15): 2026–2031.

Mazeika, P. (2001). Aborted sudden cardiac death: a clinical perspective. *Postgrad. Med. J.*; 77: 363–370.

Myerburg, R.J. and Spooner, P.M. (2001). Opportunities for sudden death prevention: Directions for new clinical and basic research. *Cardio. Res.;* 50: 177–185.

Myerburg, R.J. and Castellanos, A. (1997). Cardiac arrest and sudden cardiac death. Heart disease: A textbook of cardiovascular medicine. Braunwald, E. (Ed.). New York: WB Saunders. 756–779.

Ogunlade, O. (2011). Sudden cardiac death in Nigeria: A health challenge. *Inter. J. Health Res.;* 4(4): 163-168.

Park, K. (2007). Web of Causation. Concept of Health and Disease. Park's Textbook of Preventive and Social Medicine. 19th Edition. Prem Nagar Jabalpur. M/s banarsidas Bhanot Publishers. 31.

Rotimi, O., Fatusi, A.O. and Odesanmi, W.O. (2004). Sudden cardiac death in Nigerians - The Ile-Ife experience. *West Afr. J. Med.;* 23(1):27-31.

Seeley, R., VanPutte, C., Regan, J. and Russo, A. (2011). Fatty Acids and blood clotting. Nutrition, metabolism and temperature regulation. Seeley's Anatomy and Physiology. Ninth Edition. New York. McGraw Hill Companies Inc. 926.

Siri-Tarino, W.P., Sun, Q., Hu, B.F. and Krauss, R.M. (2010). Saturated fatty acids and risk of coronary heart disease: Modulation by replacement nutrients. *Curr. Atheroscler. Rep.;* 12(6): 384-396.

de Vreede-Swagemakers J.J.M., Gorgels A.P.M. and Dubois-Arbouw, W.I. (1997): Out-of-hospital cardiac arrest in the 1990s: a population-based study in the Maastricht area on incidence, characteristics and survival. *J. Am. Coll. Cardiol.;* 30(6):1500–1505.

Yalta, K., Turgut O.O., Yilmaz, M.B., Yilmaz, A. and Tandogan, I. (2010). Genetic basis of sudden cardiac death due to emotional trauma in apparently healthy individuals. *Int. J. Cardiol.;* 145(3): 518–519.

Zipes, D.P. and Wellen, H.J.J. (1998). Sudden cardiac death. *Circulation*; 98: 2334-2351.

AUTHOR(S) CONTRIBUTION

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