Sudden Death Due to Dilated Cardiomyopathy: A Case Report

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ABSTRACT

The sudden death in apparently healthy young individuals is always a devastating and shocking event. The incidence of sudden deaths in young due to various cardiac pathologies has wide variations in different parts of the world. Dilated cardiomyopathy is a form of cardiomyopathy characterized by progressive cardiac dilation and contractile (systolic) dysfunction, usually with concomitant hypertrophy. It is sometimes called congestive cardiomyopathy. Although it is recognized that approximately 25% to 35% of individuals with DCM have a familial (genetic) form, DCM can result from a number of acquired myocardial insults that ultimately yield a similar clinicopathologic pattern.

In the present case, dilated cardiomyopathy (DCM) was found during autopsy in an 18 year old male who had chest pain, breathlessness and died suddenly. He was a known case of epilepsy and was regularly taking oxcarbamazepine tablets. There was no history of hypertension or any other disease. There was no history of alcohol abuse. There was no family history of any congenital heart disease. Heart valves and coronaries were also normal.

INTRODUCTION

Death is said to be sudden or unexpected when a person not known to have been suffering from any dangerous disease, injury or poisoning is found dead or dies within 24 hours after the onset of terminal illness.1,2

Sudden cardiac death is most commonly defined as unexpected death from cardiac causes either without symptoms, or within 1 to 24 hours of symptom onset (different authors use different criteria).3

The causes of sudden cardiac death differ greatly among various age groups. In individuals > 40 years old, atherosclerotic coronary heart disease is the most common cause. Between 1 to 40 years of age, the causes of sudden cardiac death are commonly hypertrophic cardiomyopathy, myocarditis, congenital heart disease, arrhythmogenie right ventricular dysplasia/ cardiomyopathy etc.4

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The Cardiac Muscle is a specialized muscle cell (myocyte) with one or two centrally placed nuclei, having an extensive sarcoplasm and numerous large mitochondria. These myocytes are having branches. Adjoining cardiac myocytes are joined together by transverse lines called intercalated discs which contain numerous desmosomes, gap junctions and tight junctions. This gives the appearance of a continuous three dimensional network known as a syncytium.5,6

Dilated cardiomyopathy is a form of cardiomyopathy characterized by progressive cardiac dilation and contractile (systolic) dysfunction, usually with concomitant hypertrophy. It is sometimes called congestive cardiomyopathy. Although it is recognized that approximately 25% to 35% of individuals with DCM have a familial (genetic) form, DCM can result from a number of acquired myocardial insults that ultimately yield a similar clinicopathologic pattern. These include toxicities (including chronic alcoholism, a history of which can be elicited in 10% to 20% of patients), myocarditis (an inflammatory disorder that precedes the development of cardiomyopathy in at least some cases, as documented by endomyocardial biopsy), and pregnancy-associated nutritional deficiency or immunologic reaction. In some patients, the cause of DCM is unknown; such cases are appropriately designated as idiopathic dilated cardiomyopathy.

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In DCM, the heart is usually heavy, often weighing two to three times normal, and large and flabby, with dilation of all chambers. Nevertheless, because of the wall thinning that accompanies dilation, the ventricular thickness may be less than, equal to, or greater than normal. Mural thrombi are common and may be a source of thromboemboli. There are no primary valvular alterations, and mitral or tricuspid regurgitation, when present, results from left ventricular chamber dilation (functional regurgitation). The coronary arteries are usually free of significant narrowing.

Case report

An 18 years old male who was an electrician by occupation suddenly had chest pain and breathlessness at home, collapsed after sometime. He was taken to the hospital where he was declared brought dead. He was a known case of epilepsy and was regularly taking oxcarbazepine tablets. There was no history of hypertension or any other disease. There was no history of alcohol abuse. There was no family history of any congenital heart disease. Autopsy was conducted at the mortuary of Victoria hospital, Bangalore Medical College and Research Institute, Bangalore. On external examination the dead body measured about 166 cm in length, moderately built and nourished. Rigor mortis was present all over the body. Post mortem staining was seen over the back of the body. There were no external injuries on the body. On internal examination, Heart weighed 270 gms. Grossly, the heart was enlarged and globular in shape. Petechial haemorrhages present over surface of the heart at places. Both atria and both ventricle chambers were dilated. The thickness of free wall of both ventricles was normal. Right ventricle wall thickness measured 0.3 cm. Left ventricle wall thickness measured 1.3 cm. Interventricular septum thickness measured 1.2 cm. Heart valves and coronaries were also normal and patent. Lungs showed congestion and edema. Other internal organs were congested. On histopathological examination of the heart, both ventricles showed disorganized and closed packed cardiac muscle bundles. The myofibrils showed hypertrophy and enlarged nucleus. Myocardial fibers were thinned out at places. Areas of minimal interstitial fibrosis were also seen.

Discussion

Dilated cardiomyopathy (DCM) is a syndrome of impaired systolic function characterized by many hemodynamic, neurohormonal, and electrical derangements that are the end point of a diverse array of pathologic processes not related to coronary artery disease (CAD). With an estimated 2-year survival rate of ≤50%, total mortality in DCM is dismal. As expected, many deaths are secondary to progressive pump

![Fig 1](image1.png) The photographs showing Large “Globular” shaped heart in case of Dilated Cardiomyopathy.

![Fig 2](image2.png) The photograph of the dissected heart showing dilated left ventricle chamber in case of Dilated Cardiomyopathy.

![Fig 3](image3.png) The photomicrograph of Dilated Cardiomyopathy showing disorganized thinned out myocardial fibers (H & E, 10X).
failure, but even more alarming is the large proportion of patients who die suddenly or unexpectedly. Despite advances in medical management that reduce overall mortality associated with congestive heart failure (CHF), the mortality rate from sudden death has remained unchanged at 30 to 40% of all deaths.8-10

In the study conducted by Eckart RE et al, in America, over a period of 10 years, in 902 cases of adjudicated unanticipated sudden cardiac death, they found that out of total 298 cases of <35 years of age idiopathic dilated cardiomyopathy (DCM) was seen in 14 (4.7%) cases while out of 604 cases of age ≥35 years DCM was seen in 21 (3.5%) cases.11

Wu AH et al in his review article searched MEDLINE database from 1985 to 1999 and concluded that a large proportion of patients with DCM die suddenly, most secondary to ventricular arrhythmia and a smaller proportion due to bradyarrhythmia.12 Bulbanat B et al, in 2014 reported a case of sudden cardiac death diagnosed with dilated cardiomyopathy in a Kuwaiti family implying a genetic susceptibility factor associated with it.13

Sathirareuangchai S, described a case of Thai adult male who was found dead in his bed. The autopsy finding revealed the cause of death to be dilated cardiomyopathy (DCM). There was no history of medical condition or any hereditary disease in the family.14

Chen X et al, conducted an autopsy study from 1997 to 2003, to probe into myocardial connexin (Cx) 43 expression in the cases of sudden death due to dilated cardiomyopathy (DCM) and relationship between Cx43 expression and sudden death. Myocardial Cx43 was detected with immunohistochemical staining in the cases of 11 sudden death caused by DCM and 14 cases of control group who died of violent reasons and other diseases. He concluded that myocardial Cx43 expression was reduced in the patients with DCM who die suddenly. The alteration of quantity and distribution of myocardial Cx43 expression was probably related to sudden death of the patients with DCM.15

In the present case, dilated cardiomyopathy (DCM) was found during autopsy in an 18 year old male who had chest pain, breathlessness and died suddenly. He was a known case of epilepsy and was regularly taking oxcarbazepine tablets. There was no history of hypertension or any other disease. There was no history of alcohol abuse. There was no family history of any congenital heart disease. Heart valves and coronaries were also normal.

CONCLUSION

In this case report a young male who was a known epileptic and regularly taking oxcarbazepine tablets, had chest pain and breathlessness. He died suddenly. During autopsy the heart was found enlarged and globular in shape. Both atria and both ventricle chambers were dilated. It was diagnosed as Dilated cardiomyopathy on histopathological examination of the heart. There was no family history of any congenital heart disease. This case emphasises the requirement of essential investigations and preventive measures to prevent sudden cardiac death in young individuals and their family members due to inheritable cardiac pathologies like this. It also highlights the role of meticulous autopsy and histopathological examination to detect this condition.

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References