INTRODUCTION

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, arrhythmogenic right ventricular dysplasia/ cardiomyopathy etc. The sudden death in apparently healthy young individuals is always a devastating and shocking event. The incidence of sudden deaths as tho

OUTLINE

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1. 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. 

2. MATERIALS AND METHODS

In myocarditis, by contrast, the inflammatory process is seen as a secondary response in conditions such as ischemic injury. In myocarditis, by contrast, the inflammatory process is the cause of rather than a response to myocardial injury.

3. RESULTS

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). Coronary artery disease is the leading cause of sudden cardiac death, responsible for 80% to 90% of cases.

4. DISCUSSION

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

5. CONCLUSION

The causes of myocarditis are as follows:

6. ACKNOWLEDGMENTS

All forms of myocarditis can be associated with sudden death.

7. REFERENCES

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**Infectious:** Viruses: Coxsackie virus, ECHO, Influenza, HIV, CMV; Bacteria: Corynebacterium diphtheria, Neisseria, Meningococcus, Borrelia (Lyme disease); Rickettsiae: R. typhi, Typhus fever; Fungi: Candida; Helminths: Trichinosis.

**Immune mediat ed:** Allergens, Alloantigens, Autoantigens: Chagas disease, Chlamydia pneumonia, Sarcoidosis, SLE, Scleroderma.

**Toxic myocarditis:** Drugs: Ethanol, Anthracyclines; Heavy metals; Physical agents; and miscellaneous.

**Case series**

**Case 1**

A 28 years old female who was a teacher had fever since morning at home. After sometimes she developed chest pain and breathlessness. She was admitted in a hospital where she died in the night of same day during the course of treatment. Autopsy was conducted at the mortuary of Victoria hospital, Bangalore Medical College and Research Institute, Bangalore. On external examination the dead body measured 160 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, Pericardial cavity contained about 50 ml of blood. Heart weighed 270 grams. Surface of the heart showed haemorrhagic patches at places and multiple irregular superficial ruptures were present over left lateral border of heart. Right ventricle wall thickness measured 0.5 cm. Left ventricle wall thickness measured 1.7 cm. Interventricular septum thickness measured 1.8 cm. Coronaries were patent. Lungs showed congestion, edema and patchy consolidation. Other internal organs were congested. On histopathological examination of heart, the myocardium showed mononuclear cells infiltration in the stroma and myocytolysis suggestive of Viral myocarditis.

**Case 2**

A 31 years old female who was a BAMS doctor suddenly had chest pain and breathlessness at home. After sometimes she collapsed and taken to a hospital where she was declared brought dead. She was a known case of Bronchial asthma and used to take medicines for it. Autopsy was conducted at the mortuary of Victoria hospital, Bangalore Medical College and Research Institute, Bangalore. On external examination the dead body measured 156 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 200 grams. Right ventricle wall thickness measured 0.4 cm. Left ventricle wall thickness measured 1.5 cm and showed haemorrhagic areas in myocardium at places. Interventricular septum thickness measured 1.6 cm. Coronaries were patent. Lungs showed congestion, edema and were hyperinflated. Other internal organs were congested. On histopathological examination of heart, the myocardium of left ventricle showed eosinophils and edema in the stroma suggestive of Eosinophilic myocarditis.

**Case 3**

A 25 years old young male who was event manager by occupation suddenly had chest pain and breathlessness. After
sometimes he collapsed and taken to a hospital where he was declared brought dead. On further asking the relatives told that the deceased person used to do physical exercises regularly at Gym and used to take nutrition supplement powder on the advice of Gym trainer. Family history of Diabetes mellitus and hypertension was also present. Autopsy was conducted at the mortuary of Victoria hospital, Bangalore Medical College and Research Institute, Bangalore. On external examination the dead body measured 179 cm in length, well 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 370 grams. Greyish-white patches were present over surface of heart at places. Right ventricle wall thickness measured 0.5 cm. Left ventricle wall thickness measured 1.5 cm. Interventricular septum thickness measured 1.6 cm. Myocardium of left ventricle and interventricular septum showed haemorrhagic areas at places. Left coronary artery, Left circumflex artery and Right coronary artery showed atherosclerosis and about 10-20% luminal narrowing. Left anterior descending artery also showed atherosclerosis and about 30-40% luminal narrowing. Lungs showed congestion and edema. Other internal organs were congested. On histopathological examination of heart, the myocardium of left ventricle and interventricular septum showed multinucleate giant cells and lymphocytes suggestive of Giant cell myocarditis.

**DISCUSSION**

Dallas criteria for the histologic diagnosis of myocarditis were introduced in 1986. Endomyocardial biopsy specimens were considered diagnostic of active myocarditis if routine light microscopy revealed infiltrating lymphocytes and myocytolysis. The specimens indicated borderline or ongoing myocarditis if myocytolysis was not present, despite lymphocytic infiltration. The biopsy was considered negative if both myocytolysis and lymphocytes were absent.  

To establish the diagnosis, at least two inflammatory cell foci, with at least 5 to 10 inflammatory cells associated with individual myocyte necrosis, per histologic section should be seen. During the active phase of myocarditis, the heart may appear normal or dilated. The lesions may be diffuse or patchy. The ventricular myocardium is typically flabby and often mottled by either pale foci or minute hemorrhagic lesions. If the patient survives the acute phase of myocarditis, the inflammatory lesions either resolve or heal by progressive fibrosis. Because myocarditis can be rapidly progressive disease, the cardiac muscle may not be hypertrophied. Thus the heart often appears relatively normal on gross inspection. An increased risk of malignant arrhythmia is associated with regions of injured myocardium, both during active myocarditis and during healing. The fully healed region and surrounding tissues may contribute to electric instability.

**Conclusion**

In this case series 3 young individuals died suddenly. During autopsy and on histopathological examination of heart, the cause of death was due to viral myocarditis, eosinophilic myocarditis and giant cell myocarditis respectively. This case series emphasises the requirement of essential investigations to diagnose myocarditis early and start treatment to prevent sudden cardiac death in young individuals. This case series also highlights the role of meticulous autopsy and histopathological examination to detect them.
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References

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