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MYSTERY BEHIND A SUDDEN DEATH – A CASE REPORT

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Abstract: Sudden death is a mysterious death which is difficult to diagnose. As per WHO, it is defined as death within 24 hours from the onset of symptoms. Diseases of the cardiovascular system account for about 45% of sudden deaths. Among diseases of the cardiovascular system, occlusive coronary heart disease is the most important cause of sudden death. Occasionally sudden death may result from hypertrophic obstructive cardiomyopathy in which a portion of myocardium is hypertrophied. It is perhaps known as a leading cause of sudden cardiac death in young athletes. Younger people are likely to have a more severe form of hypertrophic cardiomyopathy. One of the other commonest causes of cardiac hypertrophy is athletic heart syndrome. Differentiation as to cause behind hypertrophy needs to be done in such cases. Herby we present a case of a 20 year old male who was found struggling in the bed in the morning hours and was brought dead to the hospital. As it was a sudden death, necropsy was done.

Key words: sudden death, hypertrophic obstructive cardiomyopathy, younger people

Introduction

Sudden deaths are more frequently seen in older individuals and take place under such circumstances which doesn't arouse any suspicion. But similar deaths in young are likely to cause suspicion.¹ It is essential to rule out trauma and poisoning before conclusion regarding the cause of death in these cases. Sudden death can result from various reasons like gradual effect on a vital organ, rupture of diseased vessel, fulminating infectious diseases etc. Amongst the diseases, the diseases involving cardiovascular system account for 45% of sudden deaths and acute Myocardial infarction is the leading entity.¹

According to McCaffry et al hypertrophic cardiomyopathy is the leading cause of sudden cardiac death in athletes.²

Sometimes these deaths can occur in persons without any predisposing factor or symptoms being present. Such cases pose a challenging task to the forensic pathologist while ascertaining the cause of death after autopsy.

In any case of sudden death emphasis has to be given to unexpected character than suddenness of death. Age, past medical history and the signs and symptoms preceding death would be helpful in directing the autopsy.

Case Report

History:

Deceased was an apparently healthy male, aged 20 years and was a known football player. He had returned from his

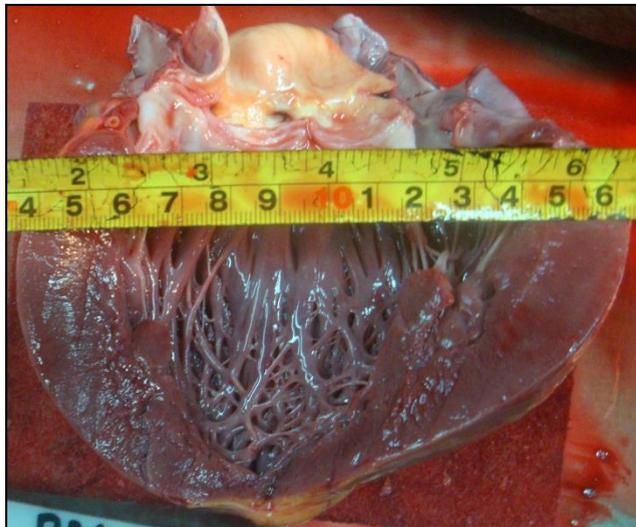


Figure 1: Left ventricular hypertrophy

Home by flight at midnight. He was found struggling in the bed by his friend in the Morning hours and was brought dead to the hospital. As it was a sudden death, necropsy was done.

Findings in autopsy:

On External examination, the face and conjunctiva were congested. Oral mucosa was bluish in colour and there was bluish discoloration of all the fingernails. No external injuries were present except for the therapeutic artifacts.

On Internal examination, Cerebral odema was present. Whitish froth was noted in trachea. Pulmonary oedema was present

and blood stained froth oozed out on cut section of both the lungs. All the other organs were congested.

Heart revealed left ventricular wall thickness to be 2.4 cm as seen in figure 1. Mitral valve was incompetent. Multiple athermanous Streaks were present in the lumen of all the coronary arteries at several places.

Investigation:

Urine alcohol level was 58.3 mg/dl. Biochemical investigation of postmortem blood sample for cardiac markers revealed:

a) Troponin T - 2.08 ng/ml (normal range - upto 0.02 ng/ml)

b) CK-MB - 199.4 ng/ml (normal range - 0.6 - 6.3 ng/ml)

Histopathology revealed left ventricular and asymmetric septal hypertrophy (Figure 2) in the heart and pulmonary oedema of both the lungs.

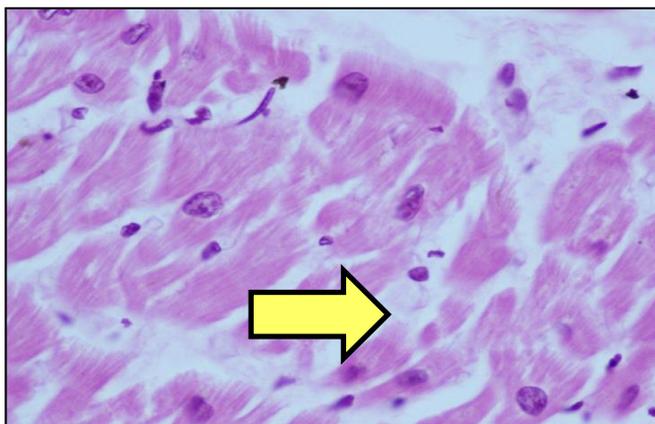


Figure 2: Septal hypertrophy

Cause of death:

Deceased died due to Pulmonary Oedema secondary to hypertrophic cardiomyopathy exacerbated by alcohol consumption.

Discussion

Deceased was a known football player with flight travel few hours before dying. Absence of external injuries with congestion of face, conjunctiva, oral mucosa and bluish discoloration of fingernails pointed towards cardiac / respiratory pathology. There were no features of Barotrauma at necropsy. Autopsy revealed and Histopathology confirmed left ventricular hypertrophy with asymmetric septal hypertrophy in heart and pulmonary oedema of both lungs. Mitral valve was incompetent. Postmortem biochemical analysis disclosed raised cardiac markers. As per literature it is said that gross and microscopic features of Myocardial Infarction requires a minimum of 6 hours survival after the attack.³ As not much of studies have been done on postmortem biochemical analysis and considering young age with patent coronaries, we kept possibility of Myocardial Infarction to be remote.

Considering the remote possibility of Myocardial Infarction, we were left with left ventricular hypertrophy and asymmetric septal hypertrophy. We now had to differentiate between hypertrophic cardiomyopathy and athletic heart syndrome.

In case of Athletic heart syndrome septal hypertrophy will be symmetric contradicting the above finding.⁴ Hence the possibility of the condition to be hypertrophic cardiomyopathy was very high.

Other possibilities were physiological adaptation or secondary to consumption of growth hormone as commonly seen in young athletes. Intensive prolonged dynamic

physical activities like aerobic exercises or static training like weight lifting would increase the chamber size of the left ventricle as well as the muscle mass and wall thickness as a physiological adaptation to counteract increased oxygen requirement to the skeletal muscles.⁵

In our case alcohol was considered to be the triggering factor causing peripheral vasodilatation and decreasing preload. Mitral valve incompetency leads to back pressure, which in turn caused pulmonary edema. Hence considering the histopathological report cause of death was given as pulmonary oedema secondary to hypertrophic cardiomyopathy exacerbated by alcohol consumption.

In our case we tried to differentiate between Myocardial Infarction, Cardiac hypertrophy and Athletic heart syndrome. We could have improved upon the case by doing growth hormone assays and thyroid hormone assays although studies pertaining to postmortem values are scarce. In future, postmortem estimation of growth hormone and thyroid hormone can be done to give a reference point.

Conclusion

This is a rare case of Hypertrophic cardiomyopathy in young. Histopathology and enzyme studies can help in diagnosis of such conditions. Hypertrophic cardiomyopathy is one of the leading causes of sudden death in young athletes.⁶ Hypertrophic cardiomyopathy usually remains asymptomatic until sudden death. Hence heart check-up of people involved in sports should be done at regular intervals to prevent these mishaps. Awareness has to be created on ill effects of alcohol in such high risk groups. It can also run in families. Familial hypertrophic cardiomyopathy is a known autosomal dominant trait and considered to be due to mutations that encode for sarcomere proteins.⁷ Hence family specific genetic testing can be done to identify relatives at risk.

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