Case Report

An Autopsy Twist: Natural Hidden Beneath Unnatural

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Abstract

There is a general belief that most cases sent for autopsy are all sinister unnatural deaths. This, along with overburdened work, constrains of time, disinclination towards tedious detailed autopsies, has led to heavy reliance on the garbled history given by the relatives, friends or police personnel accompanying the body. A happily married, well off, young farmer had gone to the market, where he drank tea, and within minutes of doing so, collapsed and died.

He was rushed to Sri Aurobindo hospital where he was declared dead and sent for autopsy. The cause of death, from the stomach and intestinal findings and corroborating it with the history of alleged consumption of some unknown substance with tea was thought to be poisoning but examination of the heart suggested hypertrophic cardiomyopathy which on histopathology was concluded as a case of myocardial infarction. Forensic experts, many times get biased by the history given by attendants or police, which may lead to misrepresentation and hence inevitably causes mistake in the form of mislabeled opinion of cause of death as unnatural deaths. This calls for a meticulous autopsy supported by ancillary investigations.

Key Words: Sudden death, Hypertrophic cardiomyopathy, Histopathology, Myocardial infarction

Introduction:

There is a general belief that most cases sent for autopsy are all sinister unnatural deaths. This, along with overburden of work, constrains of time, disinclination towards tedious detailed autopsies, has led to heavy reliance on the garbled history given by the relatives, friends or police personnel accompanying the body.

Here we discuss one such case of sudden death. [1, 6] Sudden death is defined by WHO as death within 24 hours of onset of symptoms. In sudden death, the immediate cause is almost always found in cardiovascular system, even though topographically the lesion is not in the heart or great vessels.

Sudden deaths from cardiac disease comprises, most commonly of coronary atherosclerosis, cardiomyopathic enlargement, hypertensive heart disease.

Other common causes are aortic valve disease, anomalies of coronary circulation, congenital heart disease, and other coronary artery disease such as polyarteritis.

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Case Report:

A 34 yrs old male had gone to the market on 10 July 2012 at around 11 AM to purchase vegetables. After making the purchases he drank a cup of tea and some unknown substance and within minutes of doing so, he fell unconscious.

He was immediately brought to Sri Aurobindo hospital where his vitals were not recordable. CPR was performed on him, but despite all best efforts to resuscitate, the patient was declared dead. There was no past history of any illness, hospitalization, or treatment or similar family history.

Patient was apparently a healthy asymptomatic farmer, was happily living with his wife and children and had even recently profited monetarily the patient was then shifted to mortuary as a case of sudden death.

Autopsy Findings:

On external examination the body was of a thin built male of average height, BMI being 19.45 with no signs of any external injury.

On internal examination all vital organs were congested and stomach contained water like fluid mixed with blood, around 100 ml which had a disagreeable smell and the mucous membrane was congested and bright red. [2] The heart was grossly enlarged with a size of 15 cm x 11.3 cm and weight of 380 gms.

The left ventricular wall showed hypertrophy with thickness of anterior wall – 1.7

cm, lateral wall- 2.4 cm, posterior wall – 1.4 cm and interventricular septum- 2.0 cm with hypertrophied papillary muscles and prominent trabeculated surface. (Fig. 1)

Multiple (2 to 3-visible grossly) hyperaemic zones of around 2 cm x 1 cm to 1cm x 0.5 cm were present in the posterior wall of the left ventricle involving the endocardium and up to 2/3rd myocardium.

The right coronary artery showed 10-15% of eccentric stenosis with the rest of the coronary arteries being patent. (Fig. 4)

Histopathology of hyperemic areas from left ventricle posterior wall showed myocardial muscle with degenerative changes, hypereosinophillic cytoplasm and pyknotic changes in nuclei with oedema and separation of muscle fibers in central area. (Fig. 2, 4)

Blood vessels were congested. It was concluded as Left ventricular wall hypertrophy with early infarction changes (less than 12 hours) in left ventricular posterior wall.

Discussion:

Initially based on the history given by the attendants, the congested organs and disagreeable smell from the stomach contents, poisoning (unnatural cause of death) was thought to be the cause of death but after the gross examination of the heart which showed left ventricular hypertrophy, together with, sudden death in the young individual raised suspicion of hypertrophic cardiomyopathy (natural cause of death) but histopathological features of HCM as were not seen which negated its possibility.

Finally, based on the histopathology of hyperemic areas from left ventricle posterior wall that showed myocardial muscle with degenerative changes, hypereosinophillic cytoplasm, pyknotic changes in nuclei with oedema, separation of muscle fibres in central area and congested blood vessels, it was concluded as myocardial infarction which may have lead to the death in this case.

Some of the poisons which cause sudden death due to cardiac arrest are: potassium, calcium channel blocker, carbon monoxide, cyanide, cocaine, morphine, aconite, quinine, digitalis, beta adrenergic agonist, antiarrhythmic drugs, potassium sparing diuretics etc. [5]

Cardiomyopathy means disease of the myocardium. [3, 7] Hypertrophic cardiomyopathy ooccurs between ages of 25-50yrs and is an autosomal dominant trait involving the heavy chain of beta myosin. It is one of the most common causes of sudden unexplained deaths in young athletes.

In chronic cases symptoms may include, dyspnea on exertion, harsh systolic ejection murmur and clinical features of CHF. Myocardial ischemia can occur in this even in the absence of concomitant coronary artery disease.

Ventricular arrhythmias are also common in hypertrophic cardiomyopathy. Grossly, the heart is enlarged in size, there is increase in weight, left ventricular hypertrophy, asymmetrical septal hypertrophy which may be obstructive causing sub-aortic stenosis or non-obstructive as in apical septal hypertrophy and microscopically there is haphazard arrangement of abnormally branching hypertrophied myocytes.

Myocardial infarction: coronary atherosclerosis is sometimes called the *"Captain of the men of death"* and almost all myocardial infarcts are caused by atheromatous lesions and their complications. [1]

It is said that the original lumen must be reduced to 20 percent or less before ischemia in the distribution zone is sufficient to cause myocardial necrosis. But there are numerous reports of undoubted infarction in the absence of an 80 percent stenosis.

The contrary is also common: the finding of complete thrombosis of a major vessel with no sign of infarction, due to the development of an effective collateral circulation. [7]

The gross and microscopy findings seen in case of MI are:

Conclusion:

Forensic experts, many times get biased by the history given by attendants or police, which may lead to misrepresentation and hence inevitably causes mistake in the form of mislabeled opinion of cause of death as unnatural deaths.

In this case we see that, initially what we thought to be an unnatural death (poisoning) turned out to be a hidden case of natural death (MI). Hence the autopsy examination should always be meticulous and supported with ancillary investigations.

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Fig. 6: Cut Section of Left Side of the Heart

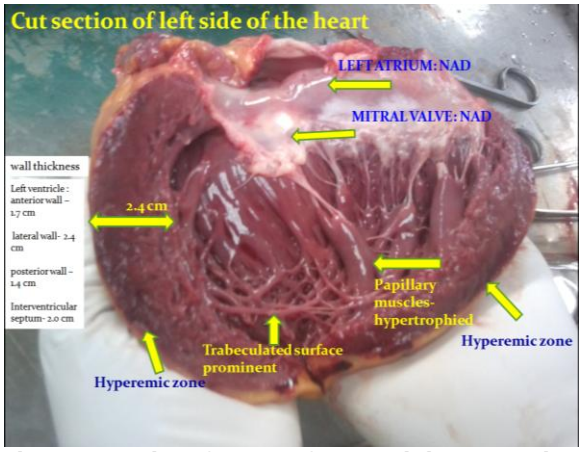

Fig. 2: Section from Left Ventricle Posterior Wall at 10x

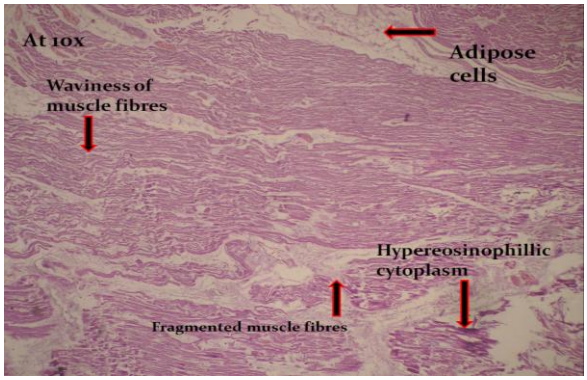

Fig. 3: Section from Left Ventricle Posterior Wall at 40x

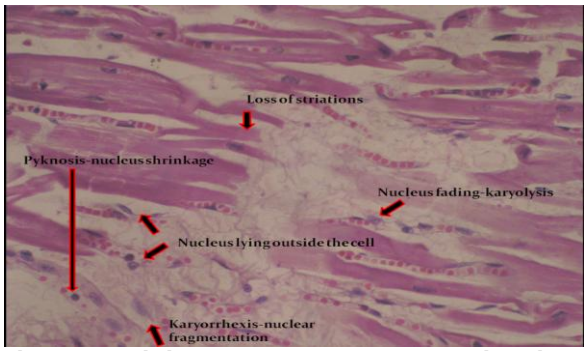

Fig. 4: Right Coronary Artery Showing Atherosclerotic Plaque

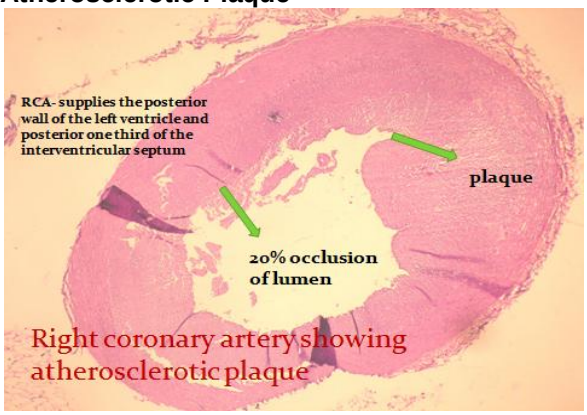

Table 1
Gross and Microscopy Findings Seen In MI

TIME	GROSS CHANGES	MICROSCOPY
0-6 hrs	No change or pale.	Stretching and waviness of fibres or no change
6- 12 hrs	-do-	Coagulative necrosis begins oedema and haemorrhages present
18-24 hrs	Cyanotic red purple areas of haemorrhage	Coagulative necrosis progresses, marginal neutrophillic infiltration,contraction band
48-72 hrs	Pale, hyperemic	Coagulative necrosis complete. Neutrophillic infiltration well developed
3- 7 days	Hyperemic border, centre yellow and soft	Neutrophils are necrosed and gradually disappear, beginning of resorption of necrosed fibres by macrophages, onset of fibrovascular response
10th day	Red purple periphery	Most of the necrosed tissue removed, fibrovascular reaction more prominent, pigmented macrophages eosinophils, lymphocytes, plasma cells present
14th day	-	Necrosed muscle mostly removed. Fibrocollagenic tissue present at the periphery
Third week	-	Necrosed muscle removed. Ingrowth of fibrocollagenic tissue
Fourth to sixth week	Thin grey white hard shrunken fibrous scar	Increased fibrocollagenic tissue, decreased vascularity, fewer pigmented macrophages lymphocytes and plasma cells