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Original Research Article

Death due to Cardiac Tamponade: An Autopsy-Based Study at a Tertiary Care Facility in India

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Abstract

Introduction: Cardiac tamponade is a condition produced by a sudden increase in intra-pericardial pressure above the venous pressure due to fluid, which impairs diastolic filling by compressing the heart and leads to cardiac dysfunction and potentially cardiac collapse. Given its potential for sudden death, medico-legal autopsies are essential for accurate diagnosis. **Aim:** This study aims to analyse the incidence, prevalence, demographics, mechanism and manner of death due to cardiac tamponade based on autopsy findings at a tertiary care centre in western Maharashtra, India over six years. **Materials and Methods:** A retrospective study was conducted from 2018 to 2023 on deaths due to cardiac tamponade autopsied at a tertiary care facility in western Maharashtra, India. Data were extracted from inquest and admission papers and analysed for incidence, demographics, cause of death, mechanism and manner of incident. **Results:** Out of 5751 medicolegal autopsies, 36 cases (0.62%) were due to cardiac tamponade. The majority of cases occurred in males (74.3%), with the highest incidence in the 51-60 age group (41.66%). Non-traumatic origins accounted for 33 cases, primarily due to left ventricular wall rupture associated with post-myocardial infarction and involving rare case of pseudoaneurysm rupture of left ventricle, and one case was traumatic cardiac tamponade involving a child. **Conclusion:** Cardiac tamponade, often undiagnosed in emergency situations, emerges as a significant contributor to sudden cardiac deaths. Recognizing spontaneous cardiac tamponade promptly in the emergency department has the potential to save lives through timely interventions in this life-threatening condition.

1. Introduction

'Cardiac tamponade is a condition produced by a sudden increase in intra-pericardial pressure above the venous pressure due to fluid, which impairs diastolic filling by compressing the heart and leads to cardiac dysfunction and potentially cardiac collapse.¹⁻³

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Cardiac tamponade results from the accumulation of pericardial fluid—whether exudate, transudate, or blood—leading to compression of the heart. Its causes broadly include pericardial effusion and hemopericardium. Pericardial effusion may arise from malignancy, myocarditis, bacterial or tuberculous pericarditis, rheumatologic diseases, or conditions such as myxoedema, hypothyroidism, and radiation therapy. Hemopericardium, the presence of blood within the pericardial cavity, can occur due to trauma, surgical procedures, myocardial infarction, rupture of intrapericardial vessels, or spontaneously. Non-traumatic cases most often involve rupture of the ventricular wall following myocardial infarction or spontaneous aortic rupture, commonly at the lateral wall of the left ventricle, whereas traumatic causes include road traffic accidents, falls from height, and penetrating chest injuries.⁴⁻⁸

Most cases of cardiac tamponade present as sudden and unexpected fatalities. Such sudden deaths hold considerable medico legal importance, as they commonly arouse suspicion of foul play, while cardiovascular pathology being the predominant underlying contributor.^{9,10, 11} The unexpected death of an apparently healthy individual without significant medical history often raises suspicion, thereby necessitating a meticulous post-mortem examination to establish the precise cause of death.

2. Material and method

The study protocol was reviewed and approved by the Institutional Clinical Ethics Committee (ICEC). A six-year retrospective study was conducted on deaths due to cardiac tamponade in medico-legal autopsies performed between 2018 and 2023 at a tertiary care centre in the western Maharashtra region. The study included all medico-legal autopsies conducted during the study period in which the cause of death was solely attributed to cardiac tamponade, irrespective of age, gender, or traumatic/non-traumatic origin, while cases with other conditions contributing to the immediate cause of death in addition to cardiac tamponade were excluded. Data collection involved a detailed analysis of postmortem reports, along with a review of inquest papers, relevant medical history or past clinical records provided by relatives prior to postmortem as recorded by the autopsy surgeon and histopathology reports. Relevant parameters such as incidence, age and gender distribution, cause of death, mechanism of fluid accumulation, and manner of incident were systematically evaluated.

3. Results

During the study period, of the 5,751 medicolegal autopsies conducted, cardiac tamponade accounted for 0.62% (36 cases) (Table 1). All individuals were either brought dead to the hospital or succumbed within a few minutes of arrival in casualty. Of these, 26 were males (74.3%) and 10 were females (25.7%), yielding a male-to-female ratio of 2.88:1. The mean age for males was 54.57 +/- 5.67, whereas the mean age for females was 67.33 +/- 11.79. (Table 2). Out of 36 cases of cardiac tamponade, two cases were attributed to non-hemorrhagic pericardial effusion while the remaining 34 cases were associated with hemopericardium or hemorrhagic cardiac tamponade.

Table 1: Year wise data for the total number of medico legal autopsies conducted and cases of cardiac tamponade.

Year	Total No. Of Medicolegal Autopsies	Cardiac Tamponade
2018	839	5
2019	977	3
2020	767	4
2021	850	3
2022	998	7
2023	1320	14
Total	5751	36

Table 2: Age and sex wise distribution of cases of death due to cardiac tamponade.

Age in years	Male	Female	Total
<10	0	1	1
11 - 20	0	0	0
21- 30	2	0	2
31 - 40	1	1	2
41 -50	4	0	4
51 - 60	15	0	15
61 - 70	2	4	6
71 - 80	1	3	4
81 - 90	1	1	2
91-100	0	0	0
Total	26	10	36

Notably, out of the 34 cases of haemorrhagic cardiac tamponade, only one had a history of trauma, involving a female child aged 1.5 years. In the remaining 33 non-traumatic haemorrhagic cardiac tamponade cases, rupture of the left ventricle was identified in 31 cases, including pseudoaneurysm

Figure 1(a) and (b): (1a) Characteristic tense pericardium with bluish-black discoloration. (1b) Cut section of pericardial sac reveals the presence of a frank blood and clot on opening pericardium.

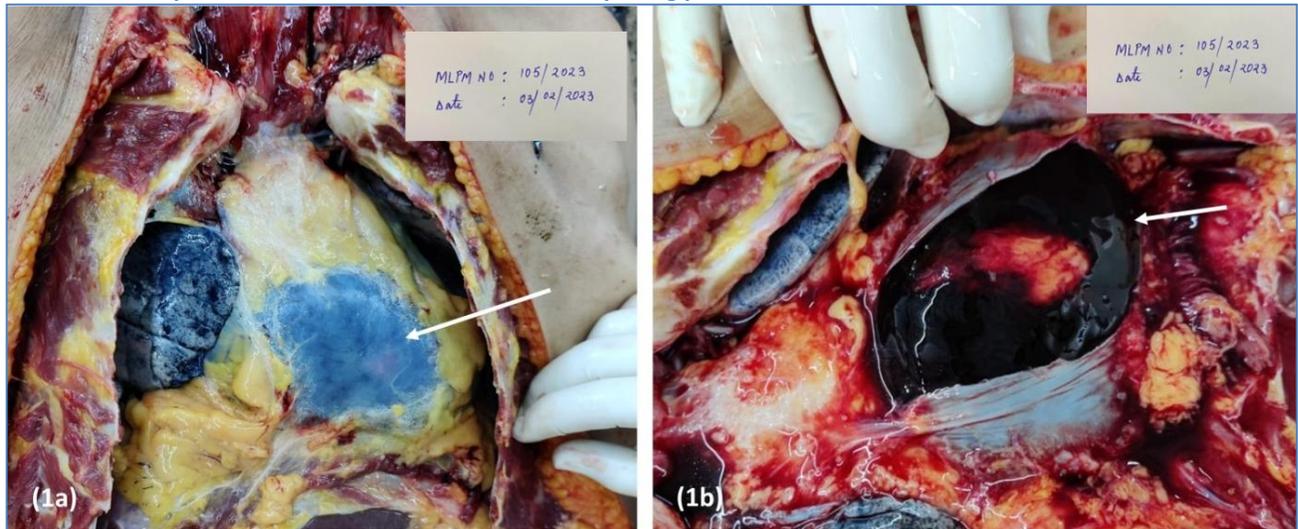


Figure 2(a) and (b): (2a) Rupture site of left ventricular wall (2b) Transmural rupture of left ventricular wall

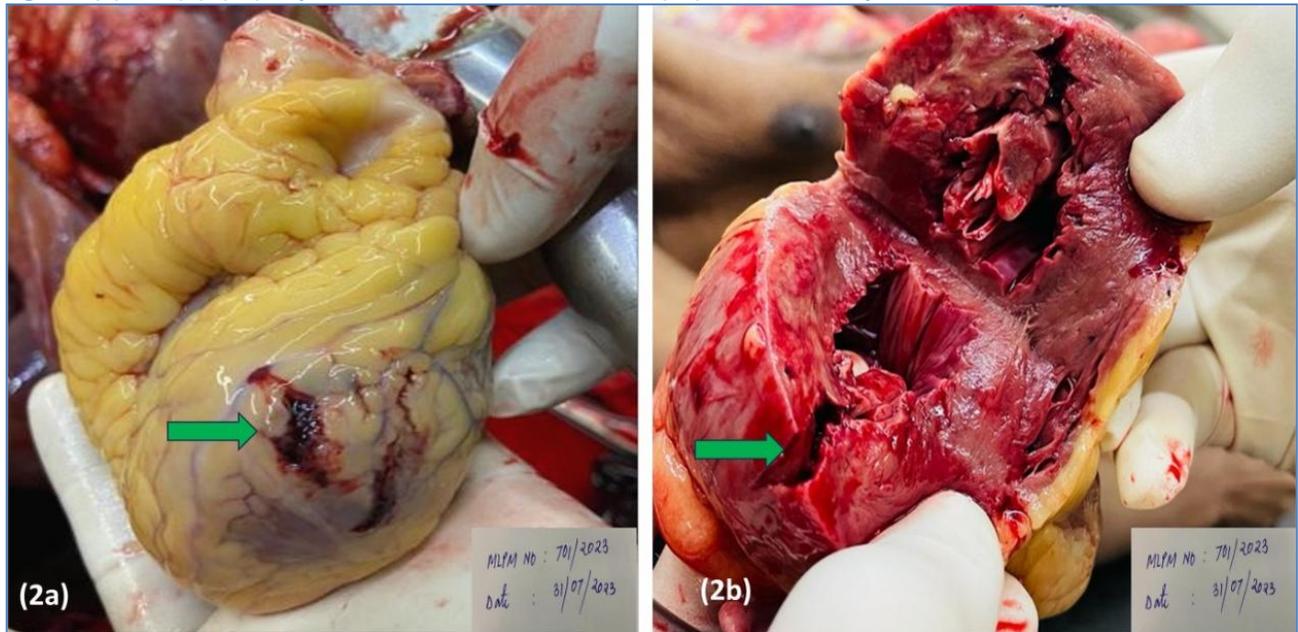
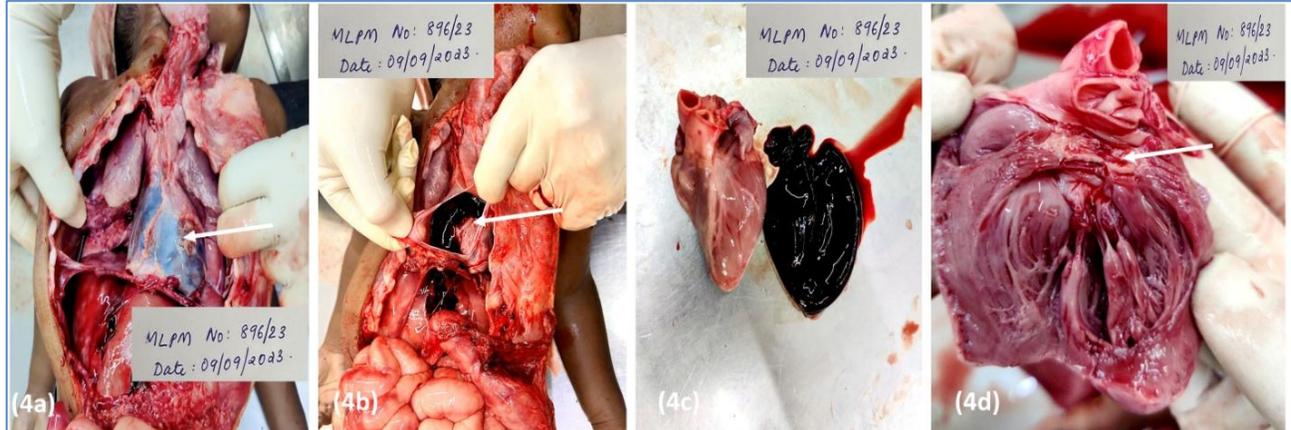


Figure 3 (a), (b) and (c): (3a) and (3b)- showing intrapericardial rupture of dissecting aneurysm of ascending aorta. (3c) Haemorrhagic area over base of aorta close to its origin.



Figure 4(a), (b), (c) and (d): Traumatic cardiac tamponade in a case of 1.5-year-old child : (4a) Tense bluish-black appearance of pericardium (4b) blood and blood clots inside pericardial cavity (4c) Blood clot from pericardial cavity resembling shape of heart (4d) Hemorrhage over base of aorta showing rupture



rupture of left ventricle, while 2 cases were attributed to rupture of a dissecting aortic aneurysm. Among the 31 cases of left ventricular wall ruptures, 17 cases showed an evident fibrous scar, indicating rupture of a weakened wall as a complication of old myocardial ischemia. The gender distribution of left ventricular rupture secondary to old myocardial infarction comprised 11 males and 6 females.

4. Discussions.

Understanding the physiological changes in tamponade is critical for diagnosis and treatment. It primarily includes the heart chambers being compressed as a result of increased pressure surrounding the heart. As the pressure rises, the heart's ability to expand inside the limited space of the pericardium diminishes, affecting cardiac function. The normal pericardial fluid volume (30 to 50 ml) displays a balance between production and reabsorption. The risk of death due to cardiac tamponade is higher in cases of rapidly accumulating hemopericardium (200 to 300 ml) compared to the slower accumulation of pericardial fluid (500 to 2000 ml), which can accommodate greater volumes due to gradual distension of the pericardial sac. The rate of fluid accumulation and the effectiveness of compensatory mechanisms are key factors, as rapid hemorrhage can quickly reach the pericardium's limit, leading to tamponade, while slower fluid accumulation, such as from inflammation, can tolerate a larger volume before becoming life-threatening.^{1,5,6}

In order to ascertain the precise pathophysiological mechanism of cardiac tamponade, an extensive post-mortem examination is essential. On autopsy, hemorrhagic cardiac

tamponade is characterized by a tense pericardium with a purplish or bluish-black discoloration. On opening, the pericardial sac typically contains frank blood and clots [Figure 1(a) and (b)].

In the study conducted by Raof A et al.,⁴ over a period of 8 years, a pronounced male preponderance was evident in cardiac tamponade-related fatalities, with a male-to-female ratio of 4.6:1. Similarly, in our study, a notable male preponderance was observed with a male-to-female ratio of 2.88:1, which may reflect regional demographic and cardiovascular risk variations. The highest incidence of cases in Raof A et al.,⁴ was within the 41–50 years age group, representing 29.41% of all cases. In contrast, our study revealed the highest incidence among individuals aged 51 to 60 years corresponding to 41.66%, likely due to differences in population age distribution and cardiovascular morbidity across the populations studied. The majority of cardiac tamponade cases in both studies were of spontaneous or non-traumatic origin, with only a small fraction being attributed to trauma. In the study conducted by Raof A et al.,⁴ left ventricular rupture was identified as the most frequently reported cause, accounting for 73.53% of cases. Similarly, in our study, left ventricular rupture was also recognized as the predominant aetiology with 86.11% of cases, followed by rupture of the dissecting aortic aneurysm. Cardiac tamponade due to non-traumatic pulmonary artery rupture was observed in a single case in Raof A et al.,⁴ study but no incidence in current study, probably due to its rare presentation.

Rupture of the left ventricular wall is a complication of myocardial infarction, affecting around 4% of infarct patients and approximately 23%

of those with fatal infarctions. Rarely, this wall rupture is contained by the surrounding pericardium, leading to left ventricular pseudoaneurysm. True ventricular aneurysm develops as a gradual outpouching involving the full myocardial wall, with fibrous replacement and a wide communication to the ventricular cavity. In contrast, a pseudoaneurysm of ventricle results from ventricular wall rupture contained by pericardium and fibrous tissue, shows no myocardial elements, and has a narrow communication neck with ventricular cavity. Unlike true ventricular aneurysms, which usually rupture in the peri-infarct period, pseudoaneurysms of ventricle can rupture immediately or even years later, often resulting in sudden death from cardiac tamponade.¹²⁻¹⁶ In 2013, Dogan et al.,¹³ reported a 55-year-old woman who died suddenly 11 months after myocardial infarction, with autopsy revealing a large left ventricular pseudoaneurysm showing pericardial adhesions and organized thrombus with absence of myocardium. In comparison, our study describes two undiagnosed post-infarction cases in female patients, where rupture occurred in the lateral wall with thin fibrous tissue and organized thrombus between epicardial and pericardial adhesions. Similarities include female predominance and delayed or absent medical evaluation after infarction, while differences arise from pathological features where case report of Dogan et al.,¹³ involved a single large pseudoaneurysm resulting in heart failure, whereas our study observed smaller lesion that progressed to rupture resulting in cardiac tamponade.

Swaminathan et al.,¹⁷ reported that males are more susceptible to myocardial infarction and aortic dissection at a younger age. They found that ventricular wall rupture, primarily due to acute myocardial infarction, was the primary cause of hemopericardium with a male-to-female ratio of 1:1.6. In cases of intrapericardial rupture of dissecting ascending aortic aneurysms, although affecting a smaller group, the male-to-female ratio was 1:1.2 while our study confirms male predominance in both post-myocardial infarction and aortic dissection rupture, accounting for 6.25% of cases.¹⁸ Our autopsy findings revealed a distinctive double lumen appearance in instances of dissecting aortic aneurysm rupture, caused by an intimal tear extending from the ascending aorta. [Figure 3 (a), (b) and (c)]. Notably, the ventricular wall was the most common site of rupture in our study of the Indian population, emphasizing a higher incidence of atherosclerotic

occlusive coronary artery disease and its related complications among males. Swaminathan et al.,¹⁷ reported that 69% of 458 cases of haemorrhagic pericardial effusion were associated with post-myocardial infarction. In comparison, our study documented 34 cases of haemorrhagic cardiac tamponade, of which 54.83% were attributed to left ventricular wall rupture with a demonstrable fibrous scar indicative of post-myocardial infarction.

Murillo et al.,¹⁹ Pooniya et al.,²⁰ and Kanchan et al.²¹ documented cases of traumatic cardiac tamponade resulting from blunt chest trauma. In each of these cases, the left ventricular wall sustained rupture, emphasizing the left ventricle as the most frequently affected site in cases of traumatic rupture. In the present study, the case was involved a 1.5-year-old girl child who had been thrown from a height by her father, resulting in a traumatic rupture of the base of the aorta [Figure 4(a), (b), (c) and (d)].

5. Conclusion.

In the current study, haemorrhagic non-traumatic cardiac tamponade predominated, with rupture of the left ventricular wall as the fundamental cause. Males were more affected, likely due to their higher burden of cardiovascular risk factors such as smoking, hypertension, and dyslipidaemia. The 51–60-year age group showed the highest incidence, reflecting the cumulative impact of atherosclerosis and age-related risk exposure. Notably, most of these cases had non-traumatic origins in contrast to the limited instances of traumatic cardiac tamponade. Most cases were non-traumatic, while traumatic tamponade was rare. Left ventricular wall rupture, often following myocardial infarction, emerged as the primary aetiology, as infarcted myocardium is structurally weakened and prone to rupture, with subsequent fibrous scarring in longer survivors.

Cardiac tamponade, often undiagnosed in emergency situations, emerges as a significant contributor to sudden cardiac deaths. Recognizing spontaneous cardiac tamponade promptly in the emergency department has the potential to save lives through timely interventions in this life-threatening condition.

Limitation of the study: Post-mortem examinations of individuals who had myocardial infarction offer limited information due to a lack of detailed medical history and record availability.

Ethics approval: The study protocol was reviewed and ethically approved by the Institutional Clinical Ethics Committees (ICEC) of the study institute.

Availability of data and materials: The datasets generated and/or analyzed during our study are not publicly available since they are owned by the study institute. They can be made available from the corresponding author on reasonable request.

Competing interest: The authors declare no competing interests.

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Authors' contributions: A.N, R.P & M.G identified the problem, designed the study, interpreted data and drafted the manuscript; All authors critically reviewed the draft and approved the final manuscript.

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