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AN OVERVIEW OF NON-TRAUMATIC CARDIAC TAMPONADE: A CAUSE OF SUDDEN AND UNEXPECTED DEATH – CASE SERIES.

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ABSTRACT Cardiac tamponade – a medical emergency where excess amounts of fluid accumulate in the pericardial sac compressing the heart, reducing cardiac output, and leading to cardiogenic shock, can present as sudden death if not diagnosed and treated promptly. Cardiac tamponade has varied etiology, with traumatic causes and non-traumatic origins like idiopathic pericarditis, neoplastic disease, tuberculosis, rupture of acute myocardial infarction, rupture of aortic dissection, cardiac operation, or treatment with anticoagulants. As little as 200 ml of blood can produce cardiac tamponade, when the fluid develops rapidly, in rupture of acute myocardial infarction or acute type A aortic dissection. This study investigates three cases of sudden death where an autopsy revealed cardiac tamponade as the primary cause of death. Detailed autopsies performed to identify the underlying cause of cardiac tamponade revealed rupture of acute myocardial infarction, and acute type A aneurysmal aortic dissection, along with another notable finding of atherosclerotic plaques in coronary artery and aorta.

KEYWORDS : Cardiac tamponade, non-traumatic hemopericardium, rupture of acute myocardial infarction, aneurysmal rupture of aorta, sudden death, autopsy.

INTRODUCTION

The World Health Organization defines sudden and unexpected or unexplained deaths as natural deaths within 24 hours from the onset of symptoms in an individual who is not known to be suffering from any disease, injury, or poisoning.(1,2) To better align with the needs of clinicians and pathologists, some authors suggest a more concise time duration of 6 hours or even 1 hour from the onset of symptoms.(2)

Sudden death often brings patients to the emergency department, where cardiovascular causes are identified as the leading contributors to sudden death, accounting for approximately 45-50% of such cases.(2) Within this category, cardiac tamponade – a medical emergency where excess amounts of fluid accumulate in the pericardial sac compressing the heart, reducing cardiac output, and leading to cardiogenic shock – represents 20-30% of the cases.(3)

Less than 50 mL of thin, clear, straw-coloured fluid, normally fills the pericardial sac, which cushions and protects the heart, However, significant fluid collection in the pericardial cavity can occur in a number of pathological conditions. This fluid may include – serous fluid (pericardial effusion), transudate (hydropericardium), blood (hemopericardium), chyle (chylo-pericardium), pus (pyo-pericardium or purulent pericarditis), or air (pneumopericardium).(5) While the mere accumulation of fluid or blood in the pericardial cavity is not necessarily life-threatening, it becomes critical when the volume increases to a level that exerts significant pressure on the heart chambers. This pressure impairs diastolic filling and reduces cardiac output, leading to a fatal condition known as cardiac tamponade.(4,6) Hemopericardium, specifically, refers to the accumulation of blood within the pericardial space, and is attributed to traumatic and non-traumatic causes. Traumatic events like, road traffic accidents and stab injuries to the chest, wherein blunt or penetrating thoracic trauma leads to laceration of the myocardium or aorta, resulting in acute bleeding into the pericardial cavity.(1) Additionally, hemopericardium can result from various non-traumatic causes, like free wall rupture following acute myocardial infarction, dissecting aortic aneurysms, retrograde bleeding into the pericardial sac after aortic root (type A) dissection, complications from any invasive cardiac procedure, central venous catheterization, and anticoagulation therapy.(7,8)

Hemopericardium has been described as a complication of 5% to 10% of patients with Acute Myocardial Infarction (AMI).(9,10) Ruptures of the left ventricular free wall, ventricular septum, or the papillary muscle are examples of post-AMI myocardial rupture. In the ventricle, the lateral wall is thought to be the most frequently ruptured free wall following an infarction.(12) However, the lateral and inferior aspects of the left ventricle have been reported as equally susceptible to post-infarction rupture.(10) Risk factors for these ruptures include age over 60 yrs, female gender, pre-existing hypertension and left ventricular wall hypertrophy etc.(12)

In cases where fluid accumulates slowly, such as in infections (e.g., tuberculosis), autoimmune diseases, neoplasms, uraemia, and inflammatory conditions (e.g., pericarditis), the pericardium can adapt to accommodate larger volumes up to 500 mL, without significantly restricting cardiac function. Conversely, rapid accumulation of even modest volumes of fluid (200 to 300 mL), as in traumatic or non-traumatic hemopericardium, can have significant clinical effects. This swift buildup of fluid can exert pressure on the thin-walled atria, venae cavae, or ventricles, impairing cardiac filling and potentially leading to life-threatening cardiac tamponade.(12) The prevalence of cardiac tamponade ranges from 25% to 30% among cases of large pericardial effusions.(8,13)

CASE SERIES

Rapid onset cases like haemorrhagic cardiac tamponade, often present as sudden deaths and are typically diagnosed during autopsy.(3) We came across a few cases within a brief period, wherein there was no history of any traumatic events or any sign of external/internal chest injury; however, cardiac tamponade was revealed on autopsy.

CASE 1

A 57-year-old male, suddenly collapsed at his workplace, he was taken to the Casualty of the hospital where he was declared 'Brought Dead'. The body was then moved to the mortuary for medico-legal autopsy. The deceased was obese with a Body Mass Index (BMI) of 30.64. The face and nailbeds of both hands were cyanosed with congestion of both conjunctiva. Postmortem hypostasis was present over dependent areas of the back of the body except for the sites of contact pressure areas and was found to be fixed. Rigor mortis was well-developed over all major joints of the body. There was no external injury on the body, including the chest. The pericardium was intact and showed bluish discolouration. The pericardial space contained a reddish firm clot with intervening whitish fibrinous strands weighing 316 grams along

with 100 ml of non-clotted blood. The heart, weighing 370 grams, showed a recent haemorrhagic infarct, measuring 5.1 cm x 2.9 cm x 1.4 cm in the apex, along with a Y- shaped transmural rupture measuring 2.3 cm x 0.9 cm x cavity deep, in the anterior aspect of left ventricle, with its lower end situated 1.9 cm from the apex. Left anterior descending artery showed calcified atherosclerotic narrowing, with around 50% of luminal blockage across a length of about 2.4 cm just distal from its origin and subsequently the distal segments showed around 80% luminal blockage across its complete length. Left circumflex artery and right coronary artery showed about 40% of calcified atherosclerotic luminal narrowing. All other internal organs were found to be congested.

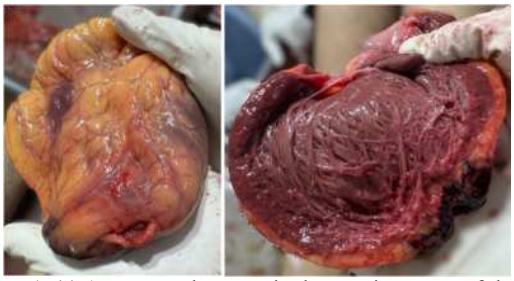

Figure 1. (a) A transmural rupture in the anterior aspect of the left ventricle. (b) A recent haemorrhagic infarct in the apex of the heart.

CASE 2

A 31-year-old male, suddenly lost his consciousness and was found in gasping state, for which he was taken to the hospital where he expired after three and half hour of hospital stay. During autopsy, it was revealed that the deceased was of average built and nourishment. Both corneas were hazy. Postmortem hypostasis was present over dependent areas of the back of the body except for the sites of contact pressure areas and was found to be fixed. Rigor mortis was well-developed over all major joints of the body. There was no external injury on the body, including the chest. The pericardium was intact. However, the pericardial cavity contained approximately 600 ml of fluid mixed with clotted blood. The heart was significantly enlarged, weighing 1200 grams. Left anterior descending artery was hardened, calcified and showed antemortem thrombus causing 100% luminal blockage over a length of 1.0 cm situated 0.8 cm distal to its origin. The distal segment of this artery showed about 80% atherosclerotic luminal narrowing. Left circumflex artery and right coronary artery showed about 90% of calcified atherosclerotic luminal narrowing. A transmural rupture, measuring 2.5 cm x 0.2 cm x cavity deep, was present on the posterior surface of left ventricle, situated 2.5 cm above the apex of heart. The myocardium surrounding the rupture displayed a soft, edematous, and pale area with hyperaemic border measuring 2.5 cm x 1.5 cm x transmural thick, indicative of recent myocardial infarction.. Additionally, multiple areas of whitish fibrosis in the left ventricular wall were observed, suggesting old, healed infarctions. All other internal organs were found to be congested and edematous.

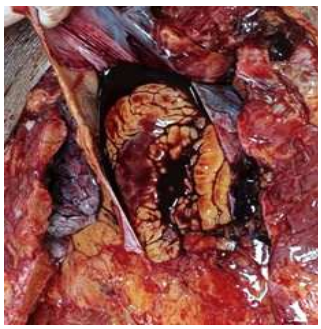

Figure 2. Fluid and clotted blood in the pericardial space.

CASE 3

A 51-year-old male with a history of hypertension experienced an episode of transient weakness in his left lower limb, which resolved spontaneously within a few hours without residual effects. Subsequently, while eating, he suffered a seizure-like episode and was taken to the hospital, where he was declared dead upon arrival. The deceased was of average built. During autopsy, the face and nailbeds of both hands were found cyanosed. Postmortem hypostasis was present over dependent areas of the back of the body except for the sites of contact pressure areas and was found to be fixed. Rigor mortis was well-developed over all major joints of the body. There was no external injury on the body, including the chest. The pericardial was intact, but the pericardial cavity contained 300 grams of reddish firm clotted blood along with 80 ml of non-clotted blood, with multiple fibrous adhesions between posterior aspect of heart and pericardium. The heart weighed 385 grams, and no gross abnormalities were present in the valves, endocardium, or myocardium. Left anterior descending artery showed hardened calcified atherosclerotic narrowing, with about 30% of luminal blockage in a segment of 0.3 cm just distal to its origin from left coronary artery. Brachiocephalic trunk and all segments of aorta i.e., ascending aorta, arch of aorta and descending thoracis aorta showed yellowish hardened calcified plaque of atherosclerosis. An aneurysmal dilation of lumen along with thinning of vessel wall was observed in both ascending and descending thoracic aorta, where it also exhibited a double lumen, suggestive of Acute type A aortic dissection (ATAAD). A transmural rupture, measuring 2.6 cm x 1.4 cm x lumen deep, was observed on the posterior aspect of the aneurysmal dilated part of the ascending aorta, with blood infiltration into the surrounding vessel wall. The heart weighed 385 grams; no gross abnormality was seen in the valves, endocardium, and myocardium. All other internal organs were found to be congested.

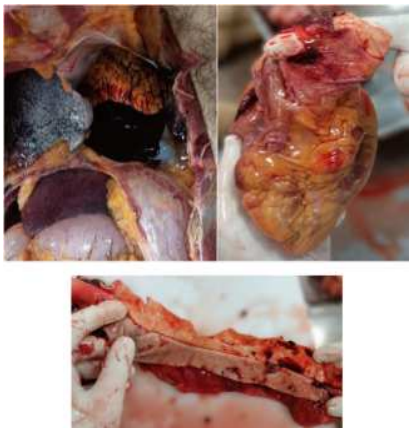

Figure 3. (a) Clotted blood in the pericardial space. (b) A transmural rupture in the posterior aspect of the aneurysmal dilated part of the ascending aorta. (c) A double lumen in the aneurysmal dilated part of the ascending aorta – Acute type A aortic dissection (ATAAD).

DISCUSSION

Hemopericardium is defined as accumulation of blood in the pericardial space.(12) When this accumulated blood reaches a level that disrupts inflow of blood into the ventricles, and reduces cardiac output, then it results in a medical emergency called cardiac tamponade.(14) In less drastic circumstances, patients may present with severe shortness of breath, chest tightness and dizziness. However, tamponade may occur so suddenly that the patient does not complain of symptoms. If it is not recognized and treated promptly, then the patient may present as sudden and unexpected death, and being brought dead at the hospital.(14,15)

Cardiac tamponade has varied aetiology, with traumatic causes leading the list,(8,14) alongside non-traumatic origins like idiopathic pericarditis and pericarditis secondary to neoplastic disease, tuberculosis, or bleeding into the pericardial space after leakage from an aortic dissection, cardiac operation, or treatment with anticoagulants.(14)

The quantity of fluid necessary to produce cardiac tamponade may be as small as 200 mL when the fluid develops rapidly, like in traumatic (penetrative or blunt force trauma to the chest) and non-traumatic (rupture of AMI or ATAAD) conditions, or be as much as >2000 mL in slowly developing effusions, as in neoplasms, infections, autoimmune diseases, uraemia, and inflammatory conditions, where the pericardium has had the opportunity to stretch and adapt to an increasing volume.(12,14) However, as little as 150 mL of blood is also known to cause death.(16)

In non-traumatic hemopericardium, bleeding into the pericardial sac may arise from the heart's surface, its cavities, or from the intrapericardial segments of the roots of the great vessels, especially the aorta and pulmonary artery. Rupture of acute myocardial infarct is the most common cause of a hemopericardium and cardiac tamponade

from natural diseases.(1) Myocardial rupture complicates only 1% to 5% of MIs, but is often fatal, rendering it a rare yet dangerous complications of acute myocardial infarction.(12,14) Left ventricular free wall rupture is most prevalent.(8,12) Cumulative figures showed anterior (42.8%) and posterior ventricular wall rupture (37.3%) being more common than apical (6%) or lateral (22.5%) ventricular wall rupture.(17) The present study also revealed myocardial ruptures present in the anterior and posterior wall of left ventricle in close proximity to the apex of the heart. Risk factors of post-AMI rupture includes age >60 years, female gender, pre-existing hypertension, atherosclerosis and left ventricular wall hypertrophy.(12) In the present study, autopsies revealed grossly visible myocardial infarction, hypertrophy of heart and atherosclerotic coronary artery diseases being associated with transmural myocardial rupture and cardiac tamponade. However, it can be observed that patients were males of varying age, i.e., both elderly (57 years) and younger (31 years) individual. It can be explained by the fact that the rupture does not take place in the early stages of infarction, but after 3 to 7 days – the time when infarcted myocardium has been converted to soft, friable granulation tissue.(2,12,18) Hence, senile and soft myocardium, seen in elderly, is a potent risk factor for post-AMI rupture. However, this by no means excludes younger males if the infarct is extensive and transmural.(1)

Angel et al. also reported a relatively young 38-year-old man, suffering from hypertension, who expired from cardiac tamponade consequent upon rupture of acute myocardial infarction.(3) Similarly, Patil A et al. documented a case of sudden death of a 50-year-old man with a history of diabetes mellitus and hypertension, who was not consistently following his prescribed treatment regimen. An autopsy revealed that the cause of death was cardiac tamponade, attributed to the occlusion of the left circumflex coronary artery.(19) Thakral S et al. discussed two incidences of deaths related to cardiac tamponade in individuals aged 60 years. Both cases exhibited significant atherosclerotic changes in the coronary arteries, underscoring the potential role of underlying cardiovascular conditions in predisposing individuals to this life-threatening emergency.(20)

Additionally, the current study revealed aneurysmal rupture of the aorta as a contributor to cardiac tamponade, which is second most frequent cause of hemopericardium and cardiac tamponade after rupture of AMI.(1) It was further compounded by male gender, elderly age of the deceased, hypertension and underlying atherosclerosis of the aorta. Ruptures of aortic dissection into pericardial space are only possible in acute type A aortic dissection, which are more common and dangerous proximal lesions involving either both the ascending and the descending aorta or the ascending aorta only (types I and II of the DeBakey classification), as compared to type B dissections.(12) Such ruptures into the pericardial cavity are relatively rare, occurring in only 2.7% of thoracic aortic aneurysm cases.(21) The incidence of cardiac tamponade was reported as between 8% and 31% in patients with acute type A aortic dissection (ATAAD),(22,23) However, cardiac tamponade is the most common cause of death in patients with ATAAD.(24)

The underlying pathology of rupture of ATAAD is a degeneration of the aortic media, called 'medio-necrosis', where the thick elastic media of the arterial wall becomes degenerate and cystic.(1) Risk factors like age over 50 years, males, and pre-existing hypertension amplifies the medial degenerative of aortic vessel wall.(12). The blood forces itself in the lumen through a tear in an atheromatous plaque. Blood enters the medial cleft under high arterial pressure, splitting the media layers and allowing the dissection to rapidly extend along the aortic wall. The dissecting haemorrhage often bursts through the outer media and adventitia into the pericardial sac, resulting in massive hemopericardium and cardiac tamponade.(1)

Hence, it can be implied that cardiac tamponade due to rupture of AMI or ATAAD is a dangerous cardiogenic cause of sudden and unexpected deaths, and are often encountered as non-traumatic sudden deaths during autopsies. Risk factors include advanced age, atherosclerotic disease, pre-existing hypertension, diabetes, and cardiac disease. Either they present as sudden deaths or with classical features of tamponade (Beck's triad). It is imperative to acknowledge that cardiac tamponade, is potentially preventable through the consistent management of hypertension or diabetes and other cardiovascular risk factors.(25,26) Immediate treatment of cardiac tamponade may be lifesaving, hence, prompt establishment of the diagnosis, usually by echocardiography, should be undertaken, followed by treatment with pericardiocentesis.(14)

CONCLUSION

Cardiac tamponade is a life-threatening condition and needs early diagnosis and intervention. In the present study, all deaths were sudden due to acute non-traumatic cardiac tamponade secondary to rupture of acute myocardial infarction and acute type aneurysmal aortic dissection of aorta. It mainly presents as sudden death, raising a high index of suspicion about the cause and manner of death. During autopsy, they present as collection of as little as 150 – 200 grams of blood with or without blood-clots in the pericardial space, which on further exploration reveals acute haemorrhagic myocardial infarct rupture or rupture of aneurysmal dissection of the proximal part of ascending aorta. Therefore, it is essential to thoroughly explore all aspects and the clinical status of patients, such as risk factors, when handling such cases.

DECLARATION OF CONFLICTING INTERESTS

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