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Case Report

Sudden Death Due To Cardiac Tamponade- A Case Report

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ABSTRACT

Forensic experts deal not only with criminal, accidental and suicidal deaths, but also with wide range of natural deaths especially if they had occurred suddenly in apparently healthy individuals. Many of these deaths are sudden, unexpected, clinically unexplained or obscure even though there may not be any criminal element in their causation. Suspicion usually arises when an individual is found dead without anyone having witnessed the death. Investigation of sudden and unexpected death is a challenge for forensic expert

The causes of sudden cardiac death are diverse, and are a function of age. Death due to cardiac tamponade is one of the causes of sudden cardiac death. Since it occurs very rapidly and without any prior symptoms, which has a highly variable clinical presentation, the diagnosis is frequently made at autopsy. In this article, we report the death of a healthy male due to cardiac tamponade following rupture of a dissecting thoracic aortic aneurysm.

Key words: Dissecting aneurysm; Cardiac tamponade; Sudden cardiac death; Autopsy.

INTRODUCTION

An aneurysm is a pathological dilatation of the lumen of a vessel. Degeneration of the medial layer of the aortic wall leads to weakening of the wall, resulting in progressive dilatation of the wall, leading to the formation of an aneurysm. Thoracic aortic aneurysms are unfortunately a well-known and frequent leading cause of sudden death, when complicated by dissection and/or rupture. Since most patients are often asymptomatic or manifestation of diverse and non-specific symptoms or present in the late course of their disease. Thus, in many cases, sudden death is the first manifestation of the aortic pathology, hence in these unexplained, sudden deaths; medico-legal autopsy is often requested. The most common cause of death in cases of thoracic aortic aneurysm rupture is cardiac tamponade.

Increase in intra pericardial fluid pressure that exceeds a trial venous pressure, thereby impeding venous return to the heart. Rapidly evolving (200 to 300 ml) is more likely to cause death from cardiac tamponade than slowly evolving accumulation (500 to 2000 ml). [1]

CASE REPORT

A 45 year old male who was a cook by profession was not seen at home in the early morning hours and was later found dead on the terrace of his residence. The body was found face down on the floor. The circumstances of discovery of his body aroused suspicion among the relatives. He had no prior history of any co morbid conditions like hypertension or diabetes.

AUTOPSY FINDINGS:

External examination revealed postmortem staining over the front of chest and abdomen and was fixed. Rigor mortis present all over. No demonstrable external injuries. On opening the pericardial cavity it contained 207g of blood clots and 100 ml of blood. Heart weighed 514 g with left ventricular thickness being 2 cm and right measured 1.2 cm. Black discoloration was noted around the aorta with an intimal tear measuring 1cm across at the root of the aorta. Left anterior descending artery showed a plaque causing thickening of the wall and occlusion of 70-80%. Left circumflex coronary artery and right coronary artery were also showed 80-90% luminal occlusion. The lungs were edematous. All the other internal organs were intact.

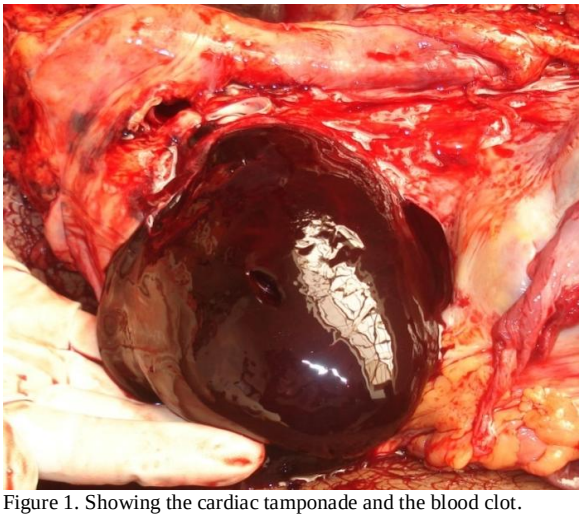

Figure 1. Showing the cardiac tamponade and the blood clot.

Histopathology

Biventricular hypertrophy, dissecting aneurysm of aorta –Type II and 80-90% luminal occlusion of all the three major coronary vessels.

Cause of Death

The cause of death was opined to be due to "Cardiac tamponade as a result of rupture of dissecting aneurysm of thoracic aorta".

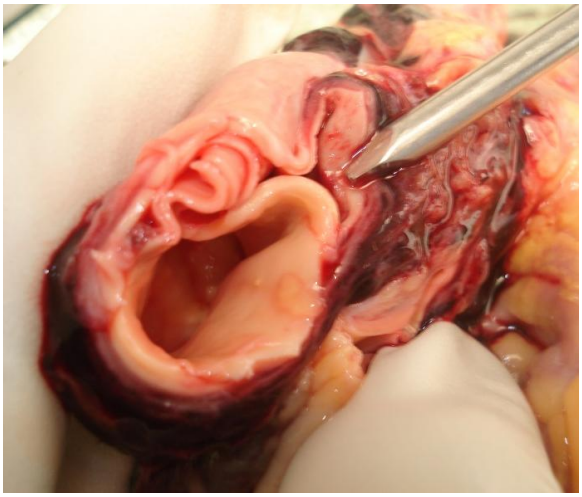

Figure 2. Showing the dissecting aneurysm in the ascending aorta.

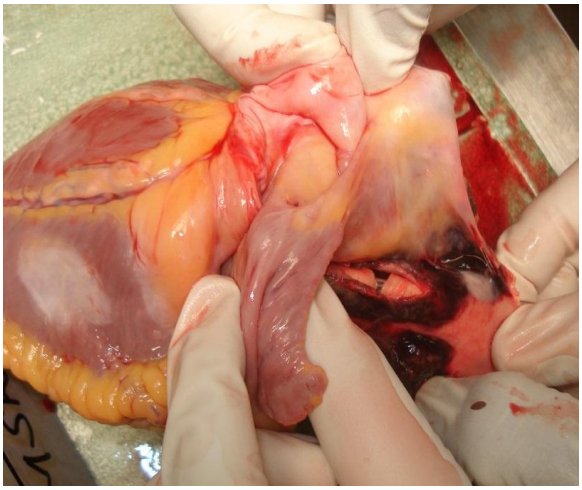

Figure 3.Showing the tear in the ascending aorta measuring 2.5 cm in length.

DISCUSSION

Aortic dissection, first described in 1761 remains one of the most severe acute vascular disorders; its low prevalence and

great symptoms diversity makes it hard to be diagnosed in the emergency room without a high level clinical suspicion.

The incidence of aortic dissection is estimated to 5-30/million people/year with a prevalence about 100-250 lower than coronary artery disease. Untreated, aortic dissection has a mortality of 25% at 24 hours, 50% at 48 hours and 80% in two weeks Main causes of death in aortic dissection are: rupture of the aneurysmal sac 80 -86% usually in the pericardial sac, 70% in acute cases, 20% in sub acute cases and 25% in chronic cases, hemorrhage in left (more often) or right pleural cavity, mediastinum, retro peritoneum, peritoneal cavity, gastrointestinal tract, congestive heart failure (often in chronic aortic dissection), coronary artery involvement/acute myocardial infarction, occlusion of abdominal aorta or it's terminal branches, gut malperfusion, carotid hemiplegic, mycotic dissecting aneurysms renal or spinal mal perfusion; mortality due to aortic dissection is highest in patients with no back/thoracic pain at initial presentation, hypotension/shock or aortic branches involvement - "the deadly triad". [2]

Aortic dissection occurs when a tear in the inner wall of the aorta causes blood to flow between the layers of the wall of the aorta and force the layers apart. Aortic dissection is a medical emergency and can quickly lead to death, even with optimal treatment. If the dissection tears the aorta completely open (through all three layers), massive and rapid blood loss occurs. Aortic dissections resulting in rupture have an 80% mortality rate, and 50% of patients die before they even reach the hospital. The vast majority of aortic dissections originate with an intimal tear in either the ascending aorta (65%), the aortic arch (10%), or just distal to the ligamentum arteriosum in the descending thoracic aorta (20%).

Classification systems to describe aortic dissections may be discussed under following headings.

DeBakey: The DeBakey system, named after surgeon and aortic dissection sufferer Michael E. DeBake, is an anatomical description of the aortic dissection. It categorizes the dissection based on where the original intimal tear is located and the extent of the dissection localized to either the ascending aorta or descending aorta, or involves both the ascending and descending aorta. [3]

Type I – Originates in ascending aorta, propagates at least to the aortic arch and often beyond it distally.

Type II – Originates in and is confined to the ascending aorta.

Type III – Originates in descending aorta, rarely extends proximally but will extend distally.

Stanford: The Stanford classification is divided into 2 groups; A and B depending on whether the ascending aorta is involved. [4]

A – Involves the ascending aorta and/or aortic arch, and possibly the descending aorta. The tear can originate in the ascending aorta, the aortic arch, or, more rarely, in the descending aorta. It includes DeBakey type I, II and retrograde type III (dissection originating in the descending aorta or aortic arch but extending into the ascending aorta).

B – Involves the descending aorta (distal to left subclavian artery origin), without involvement of the ascending aorta or aortic arch. It includes DeBakey type III without retrograde extension into the ascending aorta.

Sign and symptoms in about 96% of individuals with aortic dissection present with severe pain that had a sudden onset. It may be described as tearing in nature, or stabbing or sharp in character. [5] 17% of individuals will feel the pain migrates as the

dissection extends down the aorta. The location of pain is associated with the location of the dissection. Less common symptoms that maybe seen in the setting of aortic dissection include congestive heart failure (7%), syncope (9%), cerebro vascular accident (3-6%), ischemic peripheral neuropathy, paraplegia, cardiac arrest, and sudden death. If the individual had a syncopal episode, about half the time it is due to hemorrhage into the pericardium leading to pericardial tamponade, as seen in our case.

Aortic dissection is associated with hypertension (high blood pressure) and many connective tissue disorders. Vasculitis (inflammation of an artery) is rarely associated with aortic dissection. It can also be the result of chest trauma. 72 to 80% of individuals who present with an aortic dissection have a previous history of hypertension. The highest incidence of aortic dissection is in individuals who are 50 to 70 years old. The incidence is twice as high in males as in females (male-to-female ratio is 2:1). Half of dissections in females before age 40 occur during pregnancy (typically in the 3rd trimester or early postpartum period). A bicuspid aortic valve (a type of congenital heart disease involving the aortic valve) is found in 7–14% of individuals who have an aortic dissection. Marfan syndrome is noted in 5–9% of individuals who suffer from aortic dissection.

Turner syndrome also increases the risk of aortic dissection, by aortic root dilatation. Chest trauma, cardiac surgery, aortic insufficiency and syphilis are also associated with aortic dissection. The risk of death is high in untreated aortic dissection. While the risk is very high in the first 24 hours of the event, those that survive the initial event still have an elevated mortality compared to age- and sex-matched controls. 75% of those with ascending aortic dissection who are not treated die within 2 weeks. [6] With aggressive treatment 30-day survival for thoracic dissections may be as high as 90%. [7]

Rupture is more likely at sites of aneurysms, because of the reduced thickness and hence greater weakness of the wall, and because of the "Laplace's law", according to which tension acting on the wall of a vessel is greater where the luminal diameter is greater. Hence, a much lesser degree of trauma would be required to rupture an aneurysmal aorta as compared to a normal one, as in the instant case.

According to the osseous pinch mechanism / the aorta is crushed or pinched between the vertebral column and the inner surface of the manubrium, first rib and the clavicles during antero–posterior thoracic compressive deformation. [8]

Sudden deceleration also contributes to trauma to the aorta. During sudden deceleration, traction and torsion forces are placed on the aorta at points of anatomic fixation, i.e. isthmus and supravalvular ascending aorta. This is called the mechanism of "differential deceleration". During falls, the primary stress is longitudinal traction, with the weight of the heart producing greatest traction on the aortic root. Also, a sudden increase in intraluminal aortic pressure occurs with the force of impact. [9]

CONCLUSION

Cardiac tamponade due to dissecting thoracic aortic aneurysm is an uncommon but life threatening condition. It can mimic other conditions such as hypovolaemia particularly after trauma or injury. Due to its rapid and sudden presentation, it is most commonly diagnosed only at autopsy. Hence a high index of suspicion must be retained if it is to be diagnosed and treated effectively.

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