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Case Report

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SUDDEN DEATH DUE TO VENTRICULAR FREE WALL RUPTURE FOLLOWING ACUTE MYOCARDIAL INFARCTION: THREE CASE REPORTS

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ABSTRACT

Ruptured heart is the most common cause of a haemopericardium and cardiac tamponade, the rupture always occurring through an infarct in non-traumatic cases. In these reports, the authors present three separate rare incidents of left ventricular free wall rupture and subsequent haemopericardium which were never suspected and detected only after meticulous autopsy. In all three cases, pericardial cavity contained clotted blood weighing 150 grams or more and site of rupture was free wall of left ventricle through myocardial infarct, 3 to 7 days old. These cases are of interest due to scarcity of such reports and they emphasize that high index of suspicion is necessary for diagnosis of such cases.

KEYWORDS: tamponade, autopsy, haemopericardium, sudden death.

INTRODUCTION

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. Investigation of sudden and unexpected death is a challenge for forensic expert.

According to World Health Organisation, death is said to be sudden or unexpected when a person not known to have been suffering from any dangerous disease, injury or poisoning is found dead or dies within 24 hours after the onset of terminal illness. Diseases of the cardiovascular system are responsible for 45-50% cases of sudden natural deaths.[1] From medicolegal point of view, unexpected aspect of death is more significant than its rapidity. It is the striking invasion of the unexpected that is often responsible for arousing the suspicion of violence.

The presence of blood in the pericardial cavity is known as haemopericardium. The accumulation of fluid in the pericardium in a quantity sufficient to cause serious obstruction to the inflow of blood to the ventricles results in cardiac tamponade.[2] If small amount of fluid blood is present, it may be largely resorbed. Blood not resorbed will clot and become organised. As little as 150 to 250 ml of blood, rapidly filling the pericardial cavity can cause cardiac tamponade and death. Ruptured heart is the most common cause of haemopericardium and cardiac tamponade. The incidence of rupture in patients with acute infarct varies in different reports from 5.5% to 24%. [3]

CASE REPORTS

Case 1- case presentation

A 55 years male was working as technician in a private engineering workshop. On the day of incidence, during his visit to the workshop he had sudden breathlessness while going down from stairs and fell unconscious. The nearby passerby took him to a private hospital where he was declared brought in dead condition by treating doctor. His body was sent to our medicolegal centre for post-mortem examination. No past history of hypertension or heart disease noted. Nothing suspicious was found at the crime scene.

Autopsy findings

On external examination, the deceased was averagely built with well developed rigor mortis in the whole body. Post-mortem lividity was seen over back and buttocks, fixed. Grease stains were present over both hands and feet. No evidence of external injuries was found over surface of body. On internal examination, 300 grams of clotted blood was present in pericardial cavity covering heart. Heart was enlarged weighing 375 grams. Tear with

irregular margins of size 2 cm x 0.5 cm present over anterior wall of left ventricle. Area of infarct was present over anterior wall of left ventricle, having hyperaemic border of size 3cm x 2 cm, surrounding the tear. Left anterior descending coronary artery was 90% blocked and right coronary artery was 80% blocked by atherosclerosis.

Other visceral organs were intact and pale without any gross trauma or pathology. Stomach was empty with no peculiar odour, mucosa was pale. Toxicological analysis did not reveal any poison or drugs. Histopathological examination revealed disintegration of dead myofibres with few neutrophils and abundant mononuclear infiltration suggestive of myocardial infarction, 3 to 7 days old (figure 1).

Based on above findings, we concluded that the victim might have died because of cardiac tamponade due to left ventricular free wall rupture as a result of myocardial infarction.

Case 2- case presentation

A 39 years old male was working as a salesman in private company. On the fateful day, at about 5.30 p.m. he suddenly felt breathless in his office. He was rushed to the nearby private hospital by his colleagues where he died after 45 minutes of resuscitation. Body was brought for post-mortem examination at our medicolegal centre. No past history of hypertension or any other cardiac disease was obtained from relatives.

Autopsy findings

On external examination, deceased was averagely built. Rigor mortis was well developed in the whole body. Postmortem lividity was seen over back and buttocks. No external injuries were found over surface of body. On internal examination, pericardial cavity contained 150 grams of clotted blood completely covering the heart (figure 2). Heart was enlarged weighing 400 grams. Old healed patch of transmural myocardial infarction was present over anterior wall of left ventricle of size 3cm x 2cm (figure 3). Posterior wall of left ventricle was soft, thinned out having infarct of size 3cm x 3cm, with hyperaemic borders. Tear with irregular margins of size 1 cm x 0.5 cm was present in the centre of infarct (figure 4). On dissection of coronaries, right coronary artery was 100% blocked, left main trunk was 90% blocked and left anterior descending coronary artery was 100% blocked by atherosclerosis. Left kidney was shrunken weighing 20 grams. Right kidney was intact, pale weighing 120 grams.]

Other visceral organs were intact and pale without any gross trauma or pathology. Stomach was empty with no peculiar odour, mucosa was pale. Toxicological analysis did not reveal any poison or drugs. Histopathological examination of heart showed atherosclerotic changes in aorta, disintegration of dead myofibres, vacuolar degeneration with few neutrophils and abundant mononuclear infiltration, suggestive of myocardial infarction, 3 to 7 days old.

Histopathological examination of left kidney showed features of chronic pyelonephritis. After taking into consideration all findings, we concluded that the victim might have died because of cardiac tamponade due to left ventricular free wall rupture as a result of myocardial infarction.

Case 3- case presentation

A 65 years old female slipped while she was coming downstairs. She sustained blunt trauma to her left leg and was brought to the emergency ward of our hospital. X ray examination showed fracture of left tibia at its proximal 1/3 rd. She was known case of hypertension since 1 year and was taking tablet atenolol 50 mg once daily. On third day of admission, she complained of sudden onset breathlessness, became unconscious and died. Her body was brought for post-mortem examination at our medicolegal centre.

Autopsy findings

On external examination, deceased was averagely built. Rigor mortis was well developed in the whole body. Postmortem lividity was seen over back and buttocks. Contusion was present over front of upper 1/3 rd of left leg of size 11 cm x 2 cm, blue, underlying tibia fractured. On internal examination, pericardial cavity contained 150 grams of clotted blood completely covering the heart. Heart was weighing 250 grams, ruptured at posterior wall of left ventricle creating a tear of size 1.5 cm x 0.3 cm with irregular margins.

Area of infarct was present surrounding the site of rupture, having hyperaemic border of size 2 cm x 0.8 cm. Dissection of coronaries revealed 90% block in left anterior descending coronary artery and 100% block in right coronary artery due to atherosclerosis. Other visceral organs were intact and pale without any gross trauma or pathology. No evidence of fat embolism was found on gross or histopathological examination. On examination we concluded that the victim might have died because of cardiac tamponade due to left ventricular free wall rupture as a result of myocardial infarction.

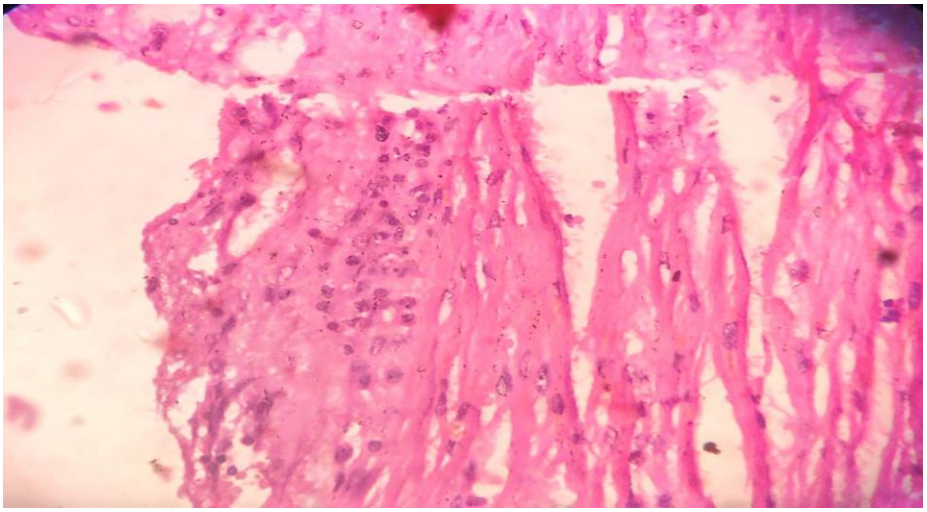

Figure 1 case 1- Histopathological slide showing myocardial infarction, 3 to 7 days old.

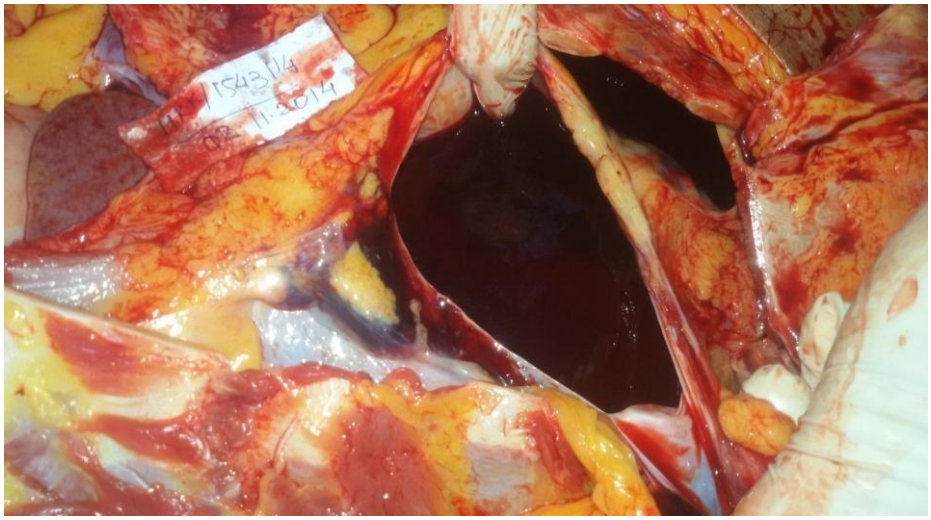

Figure 2. Case2 - Clotted blood in the pericardial cavity completely covering the heart

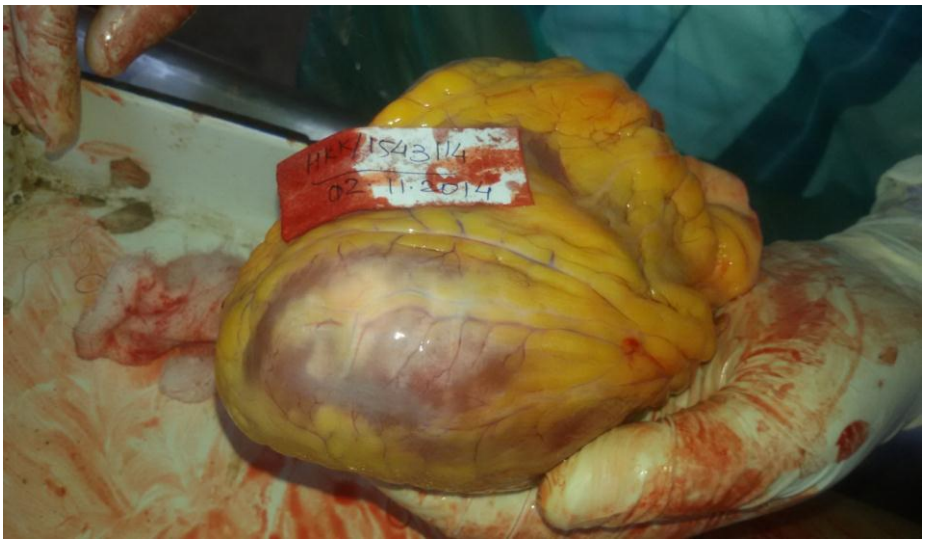

Figure 3. Case2-old healed myocardial infarction in anterior wall of left ventricle

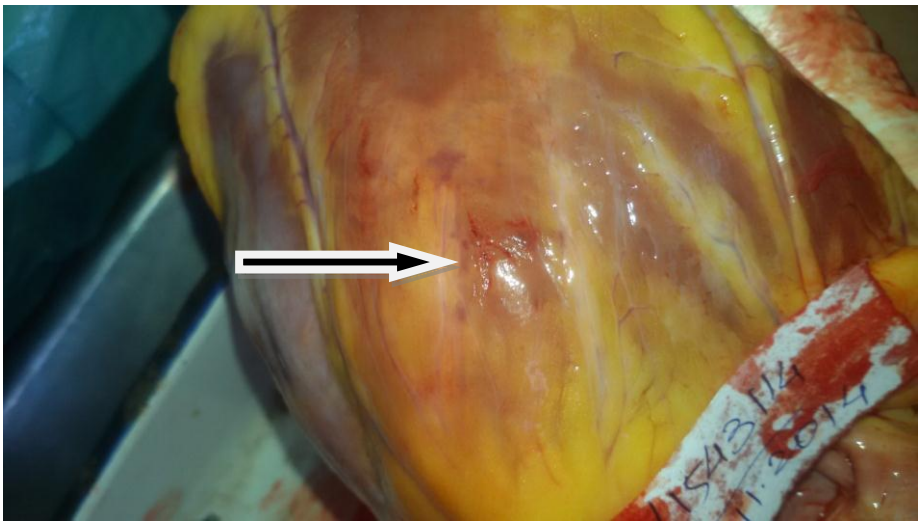

Figure 4 case 2-Arrow showing rupture of posterior wall of left ventricle

DISCUSSION

Ruptured heart is the most common cause of a haemopericardium and cardiac tamponade, the rupture always occurring through an infarct. The softened, necrotic muscle gives way from the internal pressure of the ventricular blood during systole. The blood usually tracks through tortuous channels between muscle bundles, rather than bursting a direct fistula from ventricle to pericardial sac. As the external pressure rises, the heart cannot fully expand in diastole to allow filling from the great veins. As input volume falls, so does stroke output. The venous drainage is dammed back so that congestion and cyanosis of the face and neck occur, until a fatal endpoint is reached.[4] The rupture may be either through the free wall (70%), through the ventricular septum (20%) or may be a combination of both (10%). Furthermore, a papillary muscle may rupture as a result of infarction.[3] The cardiac rupture syndrome most commonly include rupture of ventricular free wall with hemopericardium and cardiac tamponade, usually fatal.[5] Ruptures leading to hemopericardium are only found in the wall of the left ventricle.[6] The most common area for rupture is more distal part of the free wall of the left ventricle.[4] All these findings are in accordance with our observations as in all three cases, we found rupture in free wall of left ventricle. The cardiac rupture syndrome results from mechanical weakening that occurs in necrotic and subsequently inflamed myocardium.[5] Cardiac rupture may occur in the centre of infarct area, appearing as small tear in the epicardium approximately ½ to 1 inch long.[6] The rupture always occurs through an infarct.[4] These findings are consistent with our observations as rupture of heart have taken place through infarct in all three cases. Free wall rupture occurs almost any time after myocardial infarction but is most frequent in 3 to 7 days of onset.[5] The rupture usually

occurs within the first week of infarction while the necrotic region is soft.[3] Rupture of ventricular wall occurs in 2-3 days after onset of infarction, and is apparently due to rupture of the softened necrotic centre of the infarct.[6] The rupture does not take place in the early stages of a new infarct, but after a day or two when necrotic softening is well established.[4] These findings are consistent with our observations as in case 1 and case 2, histopathological examination of heart revealed features suggestive of an infarct which was 3 to 7 days old. Hypertension will increase the risk, but a more potent factor is a senile, soft myocardium, so that the elderly woman is a common victim of a ruptured heart. This by no means excludes younger men if the infarct is extensive and transmural.[4] The incidence of rupture is increased in patients with hypertension.[3] These findings are consistent with our observations where in 3rd case, 65 years old lady died due to ventricular free wall rupture having history of hypertension. In second case, though the patient was 39 years old, but the infarct was transmural.

CONCLUSION

In all three cases, pericardial cavity contained clotted blood weighing 150 grams or more and site of rupture was free wall of left ventricle through myocardial infarct, 3 to 7 days old. These reports emphasize both the significance and the necessity of the detailed evaluation, in all post-infarct patients, as it may sometimes reveal serious complications that have not been suspected on clinical grounds.

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