Case Report

Hemopericardium with Subdural Hemorrhage: A Rare Case

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Abstract

The incidence of simultaneous occurrence of intracranial hemorrhage and hemopericardium due to the rupture of a dissecting aneurysm is very rare. We describe an autopsy case of a 52-year-old male with hypertension who died suddenly. He had been treated for hypertension. During post-mortem examination we found pericardial cavity filled with blood and blood clots. This hemopericardium was due to rupture of dissecting aortic aneurysm. Ascending aorta shows ruptured dissecting aortic aneurysm [De Bakey type 2]. Intramural hematoma present in the wall of ascending aorta. Histologically, the wall of the aneurysm revealed cystic medial necrosis, which appears to idiopathic in nature. There was also evidence of subdural hemorrhage. After analyzing the findings the opinion as to the cause of death was "Hemopericardium due to rupture of dissecting aortic aneurysm along with intracranial bleeding." As per our view in present case the subdural hemorrhage was occurred after the rupture of dissecting aortic aneurysm when the venous pressure increases, leading to the rupture of bridging vein in subdural space.

Key Words: Intracranial Hemorrhage, Hemopericardium, Post-mortem, Aortic Aneurysm

Introduction:

Hemopericardium is a rare but important cause of sudden death. The etiology may be traumatic or spontaneous. In spontaneous occurrence there may be rupture of heart dissecting aortic aneurysm. The incidence of aortic dissection is estimated to be 2.9 to 3.5 per 100,000 person-years. Approximately two-thirds of those with an aortic dissection are males. [1] The mortality from aortic dissection ranged between 0.5 and 2.7% per 100,000 people from 1950 to 1981. [2] Males are more frequently affected from aortic dissection than females. [3]

Acute aortic dissection results from a tear in the intima and media of the aortic wall, with the subsequent creation of a false lumen in the outer half of the media and elongation of this channel by pulsatile blood flow. Dissection of the aorta is associated with a high degree of morbidity and mortality despite continuing improvements in techniques. [4] Hypertension is present as the most common cause in 70–90% of patients with aortic dissection. [5] We describe an autopsy case of a 55-year-old male with hypertension who died suddenly.

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Case History:

A 55 years old male was brought by relatives to a private hospital with history of a syncopal attack. The patient had one episode of vomiting followed immediately by loss of consciousness. He was immediately taken to the hospital, examined and subjected to routine investigations. His B.P. was 90/60mmHg. ECG findings suggestive of ischemic heart disease and changes of left ventricular hypertrophy.

CT scan of head was done. The CT scan did not reveal any abnormality. He had to be intubated and placed on ventilatory support.

His B.P. did not improve even with IV fluids and dopamine. He succumbed within a period of 24 hours and was referred for post-mortem examination.

Autopsy Findings:

External Examination:

Dead body of male person was averagely built, cold. Postmortem lividity present over back and buttocks except over pressure points, fixed. Rigor mortis well marked present in whole body. Therapeutic intravenous injection mark present on dorsum of both hands.

Internal Examination:

Subdural hemorrhage present about 90 ml, and red. Subarachnoid hemorrhage was present all over brain surface as a red thin blood film. Brain congested. Pericardial sac filled with blood and blood clots~500ml. Heart congested, weight 460 gms. Ascending aorta shows ruptured dissecting aortic aneurysm [De Bakey type 2]. Intramural hematoma present in the wall

of ascending aorta. Both kidneys small contracted. Cortical surface is granular, on cut section architecture of cortex & medulla are slightly obscured. Other organs congested.

Histopathological Findings:

Sections from the wall of the ascending aorta showed myxoid degeneration in the media. There was elastic tissue fragmentation and separation of the fibromuscular and elastic elements of the media by numerous cystic cleft like spaces in the media containing basophilic amorphous extracellular matrix/ground substance. There was increased fibrosis of the media. These findings are consistent with Cystic Medial Necrosis.

After analyzing the findings the opinion as to the cause of death was **"Hemopericardium due to rupture of dissecting aortic aneurysm along with intracranial bleeding."**

Discussion:

Dissection of the aorta is associated with a high degree of morbidity and mortality. [4] The highly lethal nature of acute aortic dissection was confirmed by studies analyzing both, proximal and distal untreated dissection published during the following decades. 30% of patients were dead within 24 hours and a rate of 50% mortality at 48 hours and about 95% at one month was reported. The reported deaths were related in three fourth to dissection into the mediastinum, pleural cavity and pericardium. [6]

We found ruptured aortic aneurysm which is limited only up to the ascending aorta.

Here the ruptured site at the external surface. In case of aortic aneurysm there is also intimal tear. But in this case there is no evidence of intimal tear. Studies have shown that in such cases when there is no evidence of intimal tear, the medial cystic necrosis is considered to be abnormality responsible for dissecting aortic aneurysm. Many patients with ascending thoracic aortic aneurysms appear to have nothing more than idiopathic cystic medial degeneration, where it is unclear as to what specifically predisposes to the development of medial degeneration.

The cause of medial cystic necrosis is not known. It is considered to be non specific changes in the aorta in response to hemodynamic stresses. Some authors believe that hypertension, accelerates the development of medial cystic necrosis. Histologically, as in the instant case, cystic medial necrosis is characterized by elastic tissue fragmentation and separation of the fibromuscular and elastic elements of the tunica media by small cleft like spaces where the normal elastic tissue is lost, these areas are filled with the amorphous extracellular matrix of connective tissue. Hypertension is a common risk factor. Other risk factors are Toxemia, nicotine, hyperadrenalism, infectious aortitis, great vessel arteritis. [7-9]

Aoyagi S et al has reported a case of spontaneous non-traumatic rupture of the thoracic aorta in a hypertensive patient. The clinical findings suggested acute aortic dissection, and a large pericardial effusion was detected by echocardiography. Autopsy revealed a longitudinal intimal tear and a rupture in the postero-lateral aspect of the ascending aorta. [10] Dettmeyer R et al in their study have presented two rare cases of sudden deaths.

Autopsy and morphological examination in these cases revealed a dissection of the aorta. In both cases mucoid deposits in all layers of the media and rarefactions of the elastic fibers were found, rendering cystic medionecrosis as the cause of the aortic dissection. [11]

The simultaneous occurrence of cerebral hemorrhage and hemopericardium due to the rupture of a dissecting aneurysm has been previously reported only in one case.

The authors described an autopsy case of a 45-year-old man with hypertension who died suddenly following both of these conditions. He had been treated for hypertension.

Because rupture of the aorta invariably causes a marked fall in blood pressure, the occurrence of cerebral hemorrhage following hemopericardium is highly unlikely. [12] Hemopericardium leads to Cardiac tamponade.

Cardiac tamponade is a clinical syndrome caused by the accumulation of fluid in the pericardial space, resulting in reduced ventricular filling and subsequent hemodynamic compromise. When there is no laceration of the pericardium, there is no escape route for the blood from the pericardial sac. When sufficient blood accumulates, the pressure in the pericardial sac increases and begins to prevent the passive filling of the atria during diastole.

The cardiac output falls, as does the systemic blood pressure and the venous pressure rises. If unrelieved, death follows. [13]

Subdural hemorrhage results from bleeding of the subdural portion of bridging veins, which has been identified as more fragile than the subarachnoid portion of the vein.

Cerebral atrophy, from ageing or alcoholism, accentuates the degree of traction on the bridging veins. In the absence of trauma, subdural bleeding might instead result from sudden increase in intravenous pressure. [14]

On anatomical and physiological grounds alone subdural hemorrhage could occur when raised intra-thoracic or intra-abdominal pressure is transmitted to intracranial circulation. [15] Yamushima & Frieds concluded that not only sudden acceleration or deccelaration of head, but also sudden increase in venous pressure can lead to augmentation of tension especially subdural portion of bridging veins, thus inducing subdural bleed. [16]

In present study we found both ruptured dissecting aortic aneurysm [De Bekey type 2] and subdural hemorrhage. The simultaneous occurrence of cerebral hemorrhage and hemopericardium due to the rupture of a dissecting aneurysm has been previously reported only in one case. [9] They presumed that rupture of the aorta occurred after the cerebral hemorrhage. But as per our view in present case the subdural hemorrhage was occurred after the rupture of dissecting aortic aneurysm when the venous pressure increases leading to the rupture of bridging vein in subdural space.

Conclusion:

A careful clinical examination and proper investigation is helpful for diagnosis of such cases. A detailed post mortem examination is indispensable to confirm the cause of death.

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Fig. 1: Hemopericardium

Fig.2: Aortic Aneurysm

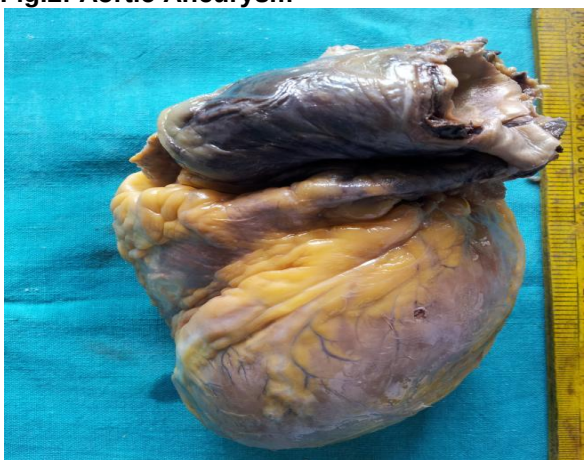

Fig. 3: Intramural Hematoma

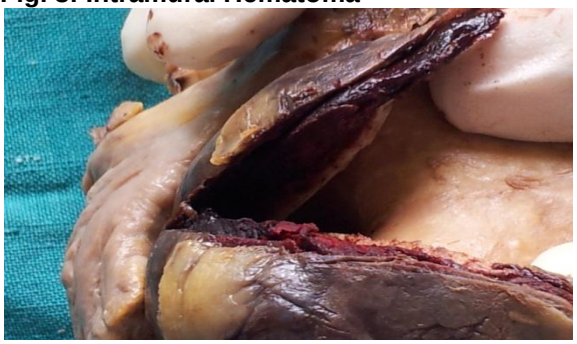

Fig. 4: Heart after Post-mortem Dissection