Aortic Dissection in a Case of Unidentified Intimal Tear

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Abstract

The aorta is the largest artery of our body. In an average lifetime, this large and remarkably tough vessel must absorb the impact of 2.3 to 3 billion heartbeats while carrying roughly 200 millions litres of blood through the body. The occurrence of aortic dissection is a rare condition. During autopsy, 13% cases of aortic dissection do not show an identifiable intimal tear. A diagnosis of aortic dissection is rarely made during life because it generally leads to cardiogenic shock and sudden death. We report a case of 47 years old male patient, without any preexisting cardiac abnormalities, hypertension or any cardiovascular risk factors which was brought dead to casualty with history of sudden chest pain and collapse at home.

Keywords: *Sudden Death, Aortic dissection, autopsy*

Introduction

The aorta is the largest artery of our body. The aorta is ultimate conductance vessel. In an average lifetime, it absorbs the impact of 2.3 to 3 billion heartbeats while carrying roughly 200 million liters of blood through the body.[1]

The aorta is composed of three layers: a thin inner layer or intima, a thick middle layer or media and a rather thin outer layer, the adventitia. The strength of the aorta lies in the media which is composed of laminated but interwining sheets of elastic tissue arranged in a spiral manner that affords maximum tensile strength.[2]

Acute aortic dissection is an uncommon but potentially fatal condition. Aortic dissection is to be believed to begin with the formation of a tear in the aortic intima that directly exposes an underlying diseased medial layer to the driving force of the intraluminal blood. This blood penetrates the diseased medial layer and cleaves the laminar plane of the media in two, thus dissecting the aortic wall.[1]

We present a case of sudden death as a consequence of acute aortic dissection with unidentified leak in intimal layer of aorta.

Case report

A case of 47 years old male patient, without any preexisting cardiac abnormalities, hypertension or any cardiovascular risk factors, brought dead to casualty with history of sudden chest pain and collapse at home. The weight and length of the dead body were 156 cm and 88 kg repectively.

On postmortem examination, pericardial sac was enlarged and tense. With the help of arge bore needle blood was aspirated from pericardial sac. Vertical cut was made from apex of heart to upper part of heart and sac was opened. On opening a bright red coloured clot of around 190 gms was found. On insitu examination heart was enlalged along with aortic vessels. After careful dissection heart with all the major blood vessels were removed.

There was spindle shaped dilatation in aorta extending from its origin of around 8.3 cm length with maximum width of 7.2 cm. After vertical opening of the chamber of heart and aorta there was no morphological abnormalities of cardiac muscles. The heart was enlarged in size and weighed around 475 grams. Coronary atherosclerosis was moderate with atherosclerotic changes in the aorta,

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and there was no calcification. From the origin of the aorta, there was clear separation of walls by bright red colour clots, which were uniformly spread (Fig. 1). On handlens examination of walls no any injury was found in the intima.

The left and right lung weights were 437 grams and 573 grams respectively. Congestive oedema was observed.

The perinephric capsule was firmly adherent to the kidneys. Bilateral kidneys show multiple cortical cysts.

Microscopic examination of the aorta showed tunica intima lined by endothelial cells and underlying collagenous tissue showed foamy macrophages with clear vacuoles showing atherosclerotic changes. Histopathogy of aorta showed dissecting aneurysm with atherosclerotic change (Fig. 2 & 3). No specific pathology was found in any other organs of the body.

After review of gross and microscopic findings cause of death was given as death caused due to cardiac tamponade as a result of acute aortic dissection.

Fig. 1 : Formalin fixed specimen of heart showing blood in the wall of aorta

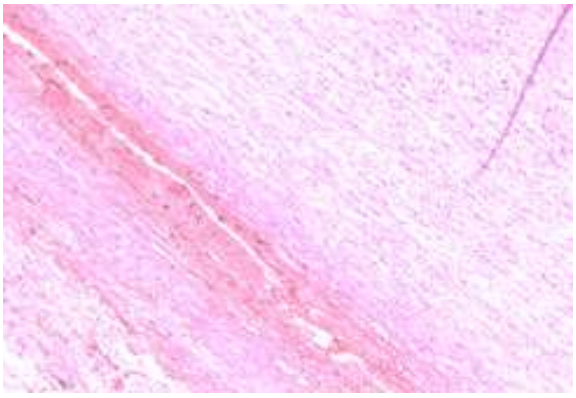

Fig. 2 : Presence of RBCs and inflammatory cells in the media of aorta (x100)

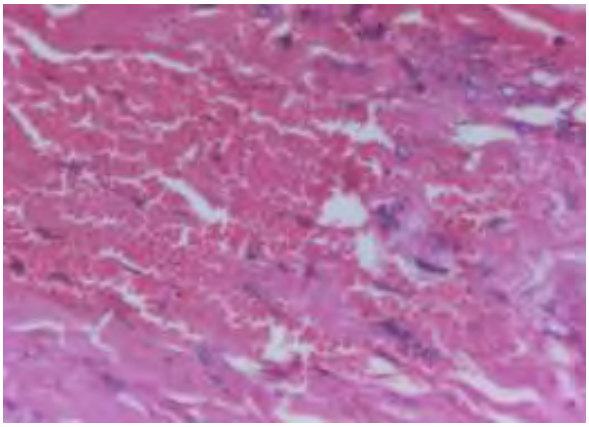

Fig. 3 : Presence of RBCs and inflammatory cells in the media of aorta (x400)

Discussion

Apparantly since sixteenth century aortic dissection has been recognized.[3] The term dissecting aneurysm was coined by Laennec in 1826 and knowledge of this entity came in 1934 with Shennan's treatise. [4]

Pathological dilatation of normal aortic lumen involving one or more segments is commonly known as aortic aneurysm. If this process is less than 14 days duration then it is labeled as a acute dissection.[5]

The process of separation was described due to many causes in literature like in Marfan's Syndrome, Turner's Syndrome[6], in autoimmune diseases[7], etc.

As per location and extent of aortic dissection there are three major classification systems (A) Debakey types I, II, III [3]; (B) Stanford types A and B [8]; (C) Anatomical catagories, proximal and distal. As in our case we don't found site of tear in intimal layer of aorta and it is limited to ascending part of aorta which fall in Type A category of Stanford classification. It is very well documented in literature that in aortic dissection may begin with rupture of vasa vasorum within the aortic media which lead to separation of layers of vessels. [1]

Bengtsson et al. [9] concluded in his study that 13 percent of aortic dissections do not show an identifiable intimal tear on postmortem examination, in present case after meticulous search we were also not able to find the site of leak.

The gross postmortem and microscopic findings in this case (cardiac hypertrophy and arteriosclerosis) strongly suggests the deceased was hypertensive, which could be a precipitating factor of the existing condition, this is well in accordance with previous researchers [10-13]

Due to dissection of aorta there was slippage of blood to the pericardial cavity resulting in formation of cardiac tamponad causing mechanical restrictions of moment of heart and death.

Conclusion

Hypertension is a precipitating cause for acute aortic dissection. Hence proper monitoring of blood pressure is required specially in middle age. If a person is diagnosed as hypertensive then proper management should be taken in order to prevent this condition.

References

1. Isselbacher EM, Eagle KA, Desanctis RW. Disease of the aorta: in Braunwald heart disease: A textbook of cardiovascular medicine, Vol-2, 5th edition, Prism books pvt Ltd, Bangalore, 1997
2. Ross MH, Reith EJ. Histology: A text and atlas. Harper & Row publichers, New York, 1985, p. 134-38.
3. Debakey ME, McCollum CH, Crawford ES, Morris GC Jr, Howell J,et al. Dissection and dissecting aneurysms of the aorta: Twenty-year follow-up of five hundred twenty seven patients treated surgically. Surgery 92:1118
4. Shennan T: Dissecting Aneurysms. Medical Research Clinical Special Report: His Majesty's Stationary Office, 1934
5. Kumar V, Cotran RS, Robbins SL. Robbins Basic Pathology. 7th edition, Elsevier, New Delhi, 2004
6. Matura LA, Ho BV, Rosing DR, Bondy CA. Aortic Dilatation and Dissection in Turner's Syndrome. Circulation. 2007;116:1663-70
7. Ohara N, Miyata T, Sato O, Oshiro H, Shigematsu H. Aortic aneurysm in patients with autoimmune diseases treated with corticosteroids. Int Angiol 2000:19:270-5
8. Daily PO, Trueblood HW, Stinson EB, et al. Management of acute aortic dissections. Ann Thorac Surg,10:237
9. Bengtsson H, Bergquist D, Sternby NH. Increasing prevalence of abdominal aortic aneurysms: A necropsy study.Eur J Surg, 158:19
10. Sabri MN, Henry D, Wechsler AS, Disciascio G, Vetrovec GW. Late complications involving the ascending aorta after cardiac surgery: recognition and management, Am Heart J 1991;121:1779-83
11. Bickerstaff LK, Pairolero PC, Hollier LH, Melton LJ, Van Peenen HJ, Cherry KJ, et al. Thoracic aortic aneurysms: a population-based study. Surgery 1982;92:1103-08
12. Spittell Jr JA, Hypertension and arterial aneurysms. J Am Coll Cardiol 1983;1:533-40
13. Takahashi M, Kondo T, Morichika M, Nakagawa K, Kuse A, et al. An autopsy case of ruptured pseudoaneurysm of the ascending aorta complicated by previous cardiac surgery for ventricular septal defect. Legal Medicine 2014;16:293-96