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Case Report

Acute myocardial ischemia due to retrograde aortic dissection in a man with history of prolong opioid drug abuse: a forensic case report

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Abstract

Aortic dissection is urgent, serious, life-threatening condition which without medical intervention in most cases has a fatal outcome. Most often it is a complication of poorly controlled hypertension. We present a rare case of acute dissection of the ascending aorta that led to sudden death due to retrograde involvement of both left and right coronary arteries and development of acute myocardial ischemia in a man with history of prolonged opioid drug abuse. A full forensic examination was performed – anamnestic, medical and criminology data was collected, a toxicological analysis of blood and urine samples was made, as well as comparison of the macroscopic findings and the histological changes in the affected organs.

Keywords: Acute myocardial ischemia, Acute aortic dissection, Forensic analysis, Opioid drug abuse, Sudden death

INTRODUCTION

Aortic dissection is characterized by incursion of blood in the vessel wall, causing longitudinal split in the aortic media and forming a channel filled with blood (dissecans intramural hematoma). This channel can re-enter the aortic lumen or lead to external rupture of the aortic wall with subsequent massive hemorrhage. Along the dissection the aorta is slightly to moderately enlarged, but true aneurysms most often is not observed, therefore the term "dissected aneurysm", used until recently, should be replaced with "acute dissection of the aorta".
Sometimes the dissecting channel can be directed in the opposite direction of the blood flow to the aortic root – in such cases the process is called "retrograde dissection." It can lead to acute failure of the aortic valve, stenosis and occlusions of the coronary arteries with subsequent severe myocardial ischemia and sudden death (Hagan et al.,2000).

Case Report

We present a case of a 53 year-old man who according to witnesses traveled in public transport vehicle and suddenly fell down and died. Forensic autopsy was carried out, with a complete external and internal examination of the body. During the external examination very low body weight was observed as well as presence of numerous traces of needle marks along the lower extremities - groin, thighs and legs, with purple, purple-brown, green-brown colored bruising around them (fresh

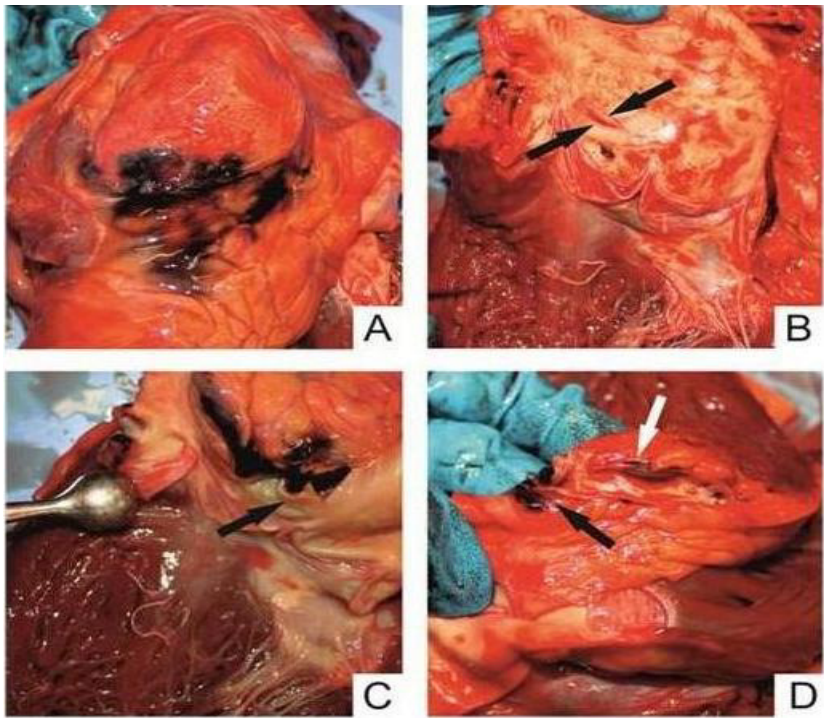

Figure 1. A- Subepicardial hemorrhages around the right coronary artery; B – Transvers rupture of the aortic intima (indicated by arrows); C - Hemorrhage around the right coronary artery; D - Hemorrhage around the ostium of the left coronary artery (indicated by black arrow), subepicardial hemorrhage pressing the descending branch of the left coronary artery (indicated by white arrow).

and old needle marks) and numerous scars with irregular or rounded shapes, sometimes below the surrounding skin or with hyperpigmentation.

The internal examination of body showed severe cerebral and pulmonary edema and acute venous congestion in the internal organs (cyanosis). During the section of the heart and surrounding structures an enlarged heart was found, at the expense of the left half, with 20mm wall thickness of the left ventricle, measured directly beneath the mitral valve. In addition the myocardium was wiry, with brownish-red color, and bundle-like structure, among which there are numerous whitish fields with diameter from 1 mm to 3 mm (myocardial fibrosis). On the back wall of the left ventricle located at the hearts base, a paler field of the myocardium was observed with irregular shape and size of about 20/15mm. The valves were smooth and shiny, transparent and flexible with preserved shape and diameter of the valve rings. Endocardium was smooth and shiny. The aorta was enlarged in the area of the aortic bulbus, with normal width in its other parts; the intima was with a yellow-red color, with multiple white-yellowish atherosclerotic plaques. Directly above the ostium of the right coronary artery, 20 mm distal of the aortic valve, a transverse (to the longitudinal axis of the vessel) rupture of the aortic wall was observed with approximately 12mm of length. There was subsequent separation of the layers of the wall from the formed "false" lumen on one hand towards the aortic arc along 35 mm and on the other, to the opposite direction - the aortic valve, with the involvement of both ostia of the left and right coronary arteries. The heart has right type of blood supply. The section of the right coronary artery showed subepicardial massive bleeding which applied pressure from outside to inside on the vessel wall. In a limited area around the ostium of the left coronary artery similar changes were noticed (Figure 1A-D).

The histological examination of the aorta, myocardium and lungs revealed: Wall of an artery of elastic type (aorta) with the presence of fibrolipid plaque in the area of which, including the inner layers of the media was found increased amount of extracellular matrix containing mucoid substance (acidic mucopolysaccharides – glycosaminoglycans), which gave a positive reaction in the histochemical staining with Alcian Blue; manifestation of partial fragmentation of elastic fibers in all layers of the media, with mild reduction of smooth muscle cells and formation of lacunas filled with amorphous, Alcian blue-positive matrix; increased accumulation of collagen in the

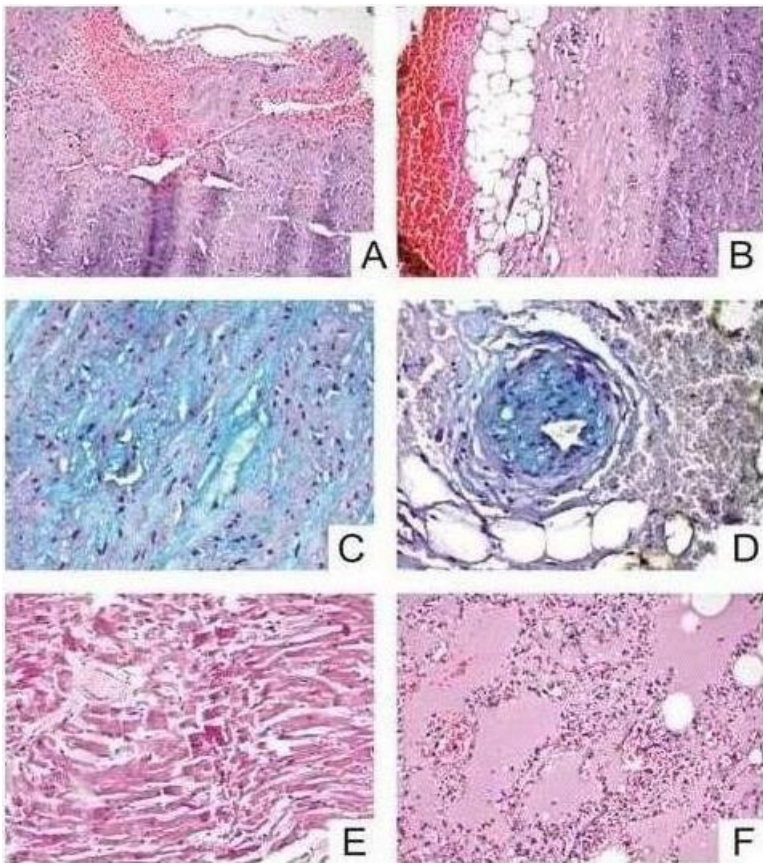

Figure 2. A - Delaminating hemorrhage in the area between the middle and outer layer of the aortic media (H and E); B - Increased collagen deposition in the outer layer of the media and periadventitial hemorrhage (H and E); C - Cystic degeneration of the aortic media by deposition of Alcian blue-positive amorphous material (Alcian blue); D – Segmental thickening of the wall of blood vessel from the system of vasa vasorum, with increased deposition of Alcian blue-positive extracellular matrix (Alcian blue); E - Focal eosinophilia and fragmentation of cardiomyocytes (H and E); F - Interstitial and intraalveolar pulmonary oedema (H and E).

outermost layers of the media; section with delaminated hemorrhage between the middle and the outer layer of the media which in large areas involves the adventitia too; segmental uneven thickening of arterial vessels from vasa vasorum system, with eccentric intimal proliferation and stratification of their internal elastic membrane; hyalinosis of arterioles located mainly in the adventitia, but also of single ones in the outer layer of the media; fresh massive adventitial hemorrhages; scarce chronic inflammatory infiltrates around the vessels (Figure 2 A-D).

The described morphological changes are consistent with acute aortic dissection of the type seen in hypertensive patients with signs of so-called "cystic medionecrosis" or cystic degeneration of the media. Myocardium: fragmentation of cardiomyocytes focal eosinophilia with signs of karyopyknosis; perivasal interstitial fibrosis (Figure 2E). Lungs: interstitial and intra alveolar edema, fresh and old (with presence of hemosiderophages) intra alveolar bleeding (Figure 2F).

The toxicological analysis of blood and urine samples analyzed with the technique of thin layer chromatography, UV spectrophotometry and immune-assay has proven presence of morphine, methadone and tetrahydrocannabinol (Table 1).

DISSCUSSION

First described by Morgagni more than 200 years, acute aortic dissection remains a challenge for emergency medicine (Hagan et al., 2000). The first surgical

Table 1. Toxicological report of blood and urine samples.

Sample	Morphine	Methadone	Tetrahydrocannabinol
Blood	0,0025mg%	0,0030mg%	Traces
Urine	0,0085mg%	0,0060mg%	Traces

Table 2. Classifications of dissection of the aorta by DeBakey and Stanford.

DeBakey	Stanford
Type I –involves ascending aorta, aortic arc and descending aorta	Type A involves the ascending aorta (DeBakey тип I и II)
Type II – involves ascending aorta	
Type III – involves descending aorta distal of a. subclavia sinistra. - IIIa – proximal and distal propagation, but in most cases only above the diaphragm - IIIb –distal propagation, including under the diaphragm	Type B does not involve the ascending aorta (DeBakey тип III)

correction of aortic aneurysms is associated with the name of American cardiac surgeon Michael DeBakey, who survived himself aortic dissection in 2005 when he was 97 years old. Nowadays the classification of DeBakey's types of aortic dissection is still in use, along with the Stanford classification (Table 2).

Causes of aortic dissection can vary (Бояджиев, 2012; Начев, 2011). The disease develops most frequently in men with increased blood pressure between 40 and 60 years of age (90% of the cases). Also affected are young patients with Marfan or Ehlers-Danlos syndromes. In other cases it is a congenital bicuspid aortic valve, coarctation of the aorta, as well as some inherited metabolic conditions such as homocystinuria and familial hypercholesterolemia. The risk is increased in pregnancy and vasculitis, especially luetic mesaortitis, developing during the tertiary stage of syphilis. There are cases of delamination of the aortic wall caused by cocaine (Singh et al., 2010), as well as iatrogenic cases during cardiac catheterization (Perez-Castellano et al., 1998), cannulation for inclusion in an extracorporeal circulation or during thoracic endovascular aortic repair (Khoynezhad et al., 2013).

The aortic dissection is characterized by rupture of the inner layer of the vessel wall (intima), hemorrhage is produced in the middle layer (media) with subsequent separation of the layers of the wall under the action of the blood flow in both proximal and distal direction covering different segments of the aorta. In this process different number of the outgoing vessels of the aorta can be involved, forming highly diverse clinical picture. Leading symptoms include acute, severe pain in the chest and back, accompanied by profuse sweating, dyspnea, abdominal pain, pressure in the mediastinum, a sense of fear. The dissection can interrupt the normal blood flow to the main branching vessels of the aorta by external compression of the false lumen or by actual separation of the branch from the true lumen. In both cases, this can lead to ischemia of the supplied with blood organs or parts of the body. Retrograde propagation of the dissection into the aortic root, as in the case reported, can lead to dissection of the coronary arteries (mostly the right one) followed by myocardial ischemia and possible development of myocardial infarction (Horszczaruk et al., 2006; Tominaga et.al., 1999; Zegers et al., 2007). The aortic valve may also be affected with subsequent prolapse caused by extrusion of the aortic commissures into the aortic lumen, leading to acute valvular insufficiency. False lumen tends to rupture into the pericardium or pleura, depending on the localization of the process and development respectively of haemopericardium with cardiac tamponade and hemothorax.`

The non-specific clinical manifestations of acute aortic dissection make it difficult for differential diagnosis in terms of emergency. It is of particular importance to distinguish it from acute myocardial infarction. As the therapeutic approach to aortic dissection and myocardial infarction is radically different, timely proper diagnosis is essential to the life of these patients. Therefore cases of

acute myocardial ischemia induced by retrograde dissection with involvement of the coronary arteries commonly lead to sudden death and are diagnosed post mortem.

Aortic aneurysms and dissections result from the structural weakness of the vessel wall, with loss of smooth muscle cells or disturbances in production and / or remodeling of the extracellular matrix, due to ischemia, inflammation or genetic diseases. During histological examination of the aortic wall most often is described the so-called cystic degeneration of the media, characterized by fragmentation of the elastic fibers and the deposition of the amorphous extracellular matrix in the form of lacunas. These types of changes are expressed in the greatest extent in the Marfan's syndrome, although it is also seen in individuals with long-standing hypertension, where the degree of change is correlated with age and the control of arterial pressure. In the case described by us the cystic degeneration of the aortic media, the histological changes in the blood vessels from the vasa vasorum system, and the disseminated in various organs expressed arteriolohialinosis, prove the presence of a long, therapeutic uncontrolled systemic arterial hypertension of the deceased. The simultaneous involvement on both coronary arteries in a retrograde aortic dissection with compression from the blood accumulated around the vessels in the adventitia, resulted in the development of acute myocardial ischemia (presented morphologically by fragmentation of cardiomyocytes and focal eosinophilia with signs of karyopyknosis). It has led to severe left ventricular failure, morphologically manifested by edema of the lungs. In the performed toxicological analysis of blood and urine samples were found metabolites of heroin, methadone and marihuana. Compared with data from the external examination of many fresh and old needle marks including old scars due to multiple injections at the same place, we can make the conclusion that the deceased had a history of prolong drug abuse. Chronic, long-term use of drugs has accelerated the development of pathological changes in the cardiovascular system: cardiac hypertrophy, myocardiofibrosis, and atheros-clerosis - common finding in long-term abuse with opiates (Willoughby et al., 1993).

CONCLUSION

The described case represents interest with the rare combination between retrograde aortic dissection and acute myocardial ischemia, which is diagnostic and therapeutic problem and should be considered in people with history of drug abuse because the pain symptom can be attenuated under the influence of opiates.

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