Coronary artery dissection as cause of sudden death

Harald Jung 1, Alexandra Lostun1*, Dumitru Bucur Matei 1

Abstract: Spontaneous coronary dissection is a rare condition, more frequently appearing in young people and that may determine stable or instable angina, heart attack, cardiogenic shock or sudden death. Women are more affected than men, especially the ones in the peripartum period. The pathologic mechanism is not at all known but it seems that atherosclerosis, connective tissue conditions and the peri-delivery period have been associated with spontaneous coronary dissection. The anterior descending artery is more often the site of the lesion in women while in men it is the right coronary artery. Taking into consideration the localization, dissection extension and the associated pathology a more effective treatment can be ruled, such as coronary by-pass, PTCA, stenting and drug treatment. Thanks to the modern cardiology investigation possibilities, the number of survivors is rising.

We present a case of spontaneous coronary dissection that was identified in the circumstances of a sudden death. A 38 years old man deceased suddenly on the street, apparently in perfect health condition. During autopsy a rupture of an atheromatous plaque located on the ascending aorta determined the dissection of the wall that further continued on the left main coronary artery and on the descendent coronary artery. The histopathologic examination mentioned a false lumen in the outer third of the media, hyperemia of the vasa vasorum and minimal coronary atherosclerosis.

Key Words: sudden death, spontaneous coronary dissection, men, anterior descending artery.

Coronary artery dissection (CAD) is a rare condition [1, 2] characterized by separation of the arterial wall layers, which may occur between media and adventitia or between media and intimae. It can cause stable or unstable angina, myocardial infarction [2, 3], cardiac shock [4] or sudden death [5], which is the most common clinical manifestation [6]. The classical histological finding is that of a large haematoma occupying the outer third of the media resulting in complete compression of the true lumen [5].

Pretty described the first case in the literature in 1931, an aneurismal coronary dissection in a Caucasian woman aged 42 who accused chest pain and then suddenly died [7, 8]. Since then there have been described around 300 cases, most found postmortem, but the number might be higher because many coronary artery dissections were presented as sudden death [5].

Coronary dissection can be spontaneous or secondary. The secondary type can occur iatrogenically (by representing a complication of cardiac catheterism, coronary angiography, coronary angioplasty after coronary or thoracic surgery), extension of an aortic dissection. [9, 10]

Spontaneous coronary artery dissection (SCAD) can occur during the post delivery time, immune system diseases, after physical exercise, thoracic trauma or idiopathic [1].

SCAD is a heterogeneous condition determined by many associated pathologies [10] such as atherosclerosis, Kawasaki syndrome, pregnancy, idiopathic, this residing the necessity for at least one clear therapeutic strategy [11]. The triggers might be physical exercises (aerobics, soccer, rugby), cocaine abuse [12, 13] or even prolonged sneezing as all three of them have in common
the increased coronary perfusion pressure [14].

Spontaneous coronary artery dissection is a very rare cause of acute coronary syndrome in younger patients, otherwise with no associated co-morbidities, with a higher incidence for young women [1], especially during peri-partum or for those using contraceptives [2].

The survival rate for these patients is rising thanks to the progress and large scale utilisation of the coronary angiography techniques [2].

Seat-belt restricted vehicle occupants involved in traffic accidents are exposed to lethal traumatic coronary dissection and in these cases the conclusion is of violent death.

**MATERIAL AND METHOD**

We present a case of sudden cardiac death occurred in a middle-aged man who collapsed on the street, based on emergency medical records review, macroscopic examination of all organs during medico-legal autopsy, histopathological examination of relevant fragment of organs formalin-fixed and paraffin-embedded, sections cut at five microns and stained with hematoxyline-eosine. All slides were analyzed on a Leica DM 1000 optical microscope, digital images were acquired and processed with Leica Application Suite V4.0 program running under Windows XP Professional. Macroscopic lesions have been photographed using a Canon SX 100 IS digital camera.

**CASE PRESENTATION**

We bring into attention the case of a 38-year-old male, from Tîrgu-Mureş, with no previous medical history (according to wife’s statement during the interview for relevant antecedents) who deceased on the street during morning hours while returning home from the market. He unexpectedly complained of acute chest pain and collapsed. The medical emergency rescue team arrived in ten minutes but cardio-respiratory resuscitation was ineffective.

The forensic pathology report (no. 666/2012 Institute of Legal Medicine Tîrgu Mures) mentions: 167 cm height, hypersthenic constitution, external and internal hydrocephaly, cerebral edema. The right lung weights 600 grams and the left one 540 grams; pleural asphyxic petechiae and pulmonary stasis are visible.

The heart has 10 cm on its longitudinal axis, 14 cm on its transverse diameter and is 7.5 cm thick; it weighs 560 grams. On the posterior wall of the left ventricle a hyperemia zone of 1,5/1,5/2,5 cm can be seen. The left ventricle has a thickness of 20 mm, the interventricular septum is 18 mm and the right ventricle 3 mm. On the thoracic aorta the rupture of an atheromatous plaque of 2.5 x 0.3 cm is visible, together with hemorrhagic dissection towards the left coronary ostium (Figure 1).

**Figure 1.** Aortic plaque tearing with hemorrhagic dissection prolonged towards the left coronary.

**Figure 2.** Left coronary artery dissection.
Figure 3. False coronary lumen in the outer third of the media.

Figure 4. Minimal coronary atherosclerosis.
The arterial wall dissection between the media and the adventitia is extended further on the left coronary artery for 1.5 cm and on the anterior descendent artery for 1 cm (Figure 2). Other findings were: incipient atherosclerosis on the thoracic and abdominal aorta, liver distrophy and a hyperemic area on the mediastinum zone of both the kidneys.

The histopathological examination of the coronary artery (Hpt. 33380, IML Tg Mures) showed a slot-like false lumen at the level of the media layer (which indicates a wall dissection – Figure 3).

Hyperemia of the vasa vasorum is obvious (Figure 3) and atherosclerotic changes in the intima are minimal (Figure 4). We concluded that the death of L.M., 38 years old was non-violent, the medical cause of the death being the acute cardio-respiratory failure following coronary dissection determined by the rupture of an atheromatous aortic plate.

**DISCUSSION**

The average age for CAD is 35-40 years with higher frequency for females [3], with a ratio of approximately 3 to 1 [15]; females have the lowest survival rate [16]. In women the left anterior descending artery is most commonly affected (80% of the cases) while in men the right coronary artery is more often the site of the dissection [17].

CAD is a very rare condition with an incidence from 0.28 to 1.1% in candidates for coronary angiography [5]. The left anterior descending artery is affected in most cases [18] followed by the right coronary artery and the circumflex artery on the incidence scale [19, 20]. In over 60% of the cases, the proximal part of the artery is described to have this condition [11]. In about 20% of the cases, more than one artery is affected [14], with only 6 cases described to have all three arteries bearing this pathology [21].

The mechanism in thoracic trauma is believed to be wall shearing causing intima torn. Then platelet aggregation follows with consecutive intra-coronary thrombosis [20]. Traumatic coronary dissection may occur in belted car drivers involved in traffic accidents due to sudden chest compression combined with impact shearing forces and myocardial infarction may develop [22].

Aortic dissection can also lead to CAD [23] as the dissection is prolonged via the coronary vessels. Aortic dissection is often associated with arterial hypertension, congenital conditions of the conjunctive tissue (Marfan syndrome, Ehlers – Danlos syndrome), vasculitis, congenital malformations of the aortic valve (double cuspis, unicomissural) [24], myxoid / mucoid degeneration of the aortic media with focal disarrangements of smooth muscle fibers and decreased number of collagen fibers [25], inside the wall haematoma, iatrogenically (aortic catheterisation, intraaortic counterpulsation balloon) atherosclerosis [26], aortic penetrating ulcer [27]. There are two classifications of the aortic dissections – Stanford and DeBakey and they are used to divide the dissections into two main categories: the ones needing surgery and the ones with drug management indication [24]. Therefore, DeBakey type II and Stanford type A which means the ascending aorta and the aortic arch are the place for the lesion are advised for surgical treatment while DeBakey type III and Stanford type B (the ascending aorta is the place of the lesion) are advised for drug treatment [24, 27].

The risk for SCAD is high in women during peripartum (as soon as the ninth week of pregnancy and not later than the third month after delivery) – one third in the third semester of pregnancy and the other two thirds during the puerperal period [11]. More than one pregnancy and the elder the person provide higher risk for this pathology. It is thought that the modified concentrations of hormones play an important role for vascular structural modifications that appear by determining muscular cells hypertrophy, weakening of the intra-cellular matrix (on behalf of high levels of mucopolysaccharides) and low collagen production in the media layer. The haemodynamic transformations are also credited with an important role – risen circulatory volume, stress during delivery. [8, 28].

In approximately 30% of the cases SCAD is given by tearing of an atheromatous plaque as incipient or medium atherosclerosis is always present in all patients of both sexes that are not during peripartum [11, 17]. In our case the coronary dissection had and aortic atherosclerotic origin as well, while coronary atherosclerosis was minimal.

Coronary atherosclerosis leads to vasa vasorum density growth that may determine hemorrhage [2] or plaque tearing and further coronary dissection [11]. It is difficult to determine the grade of stenosis after coronary dissection using microscopic imaging software since fragmentation influences the measurements accuracy [29].

Conjunctive tissue diseases such as Marfan or Ehlers-Danlos syndrome can also determine SCAD [2] through a physio-pathologic mechanism that resides in medial arterial layer degeneration and thinning of the arterial wall [11]. Systemic lupus erythematosus is also a predisposing factor through generalized vasculitis and chronic inflammation of the blood vessels wall [2].

Patients with coronary dissection may suffer from Prinzmetal instable angina, acute heart attack with or without ST elevation, cardiogenic shock. Sometimes it can be diagnosed incidentally while catheterization for other cardiac conditions [7]. The golden standard for diagnosis is the coronary angiography and the false lumen is the characteristic morphology [2]. IVUS (intravascular ultrasound system) or coronary angio-CT can also be of use. The patients arriving in hospital for acute heart attack and instable angina are firstly submitted
to cardiac catheterism which quickly leads to diagnosis and treatment [7].

CAD etiology is of high importance as it gives the survival prognosis. CAD together with atherosclerosis has a good prognosis because of the collateral circulation [7]. The treatment course depends on the associated diseases and the anatomic localization and extension of the dissection, being represented by the drug treatment, surgery options or percutaneous coronary surgery. The drug therapy is the choice when the small vessels have a certain condition or when there are no signs of ischemia or hemodynamic instability [28]. This therapy includes anticoagulation (heparine), antiplatelet agents (aspirin, clopidogel), beta-blockers and nitrates but Calcium channels blockers can be also used to treat the coronary spasm. Anti platelet glicoproteine Iib/IIia can be used to treat the ischemia determined by the dissection [2].

The surgery or percutaneous procedures are used when the dissection occurs for the big vessels, with subsequent ischemia. Percutaneous procedure with stenting is the most frequently choice when only one coronary artery is affected [2]. It helps recovering the normal blood flow and eliminates the ischemia, it repairs the dissection and prevents its extension. The surgical treatment meaning by-pass uses the mammary artery and/or the radial and is the choice when more than one coronary is affected or the percutaneous techniques results are not satisfactory [2, 5].

The prognosis for the patients suffering from coronary dissection has strongly ameliorated lately thanks to the diagnosis methods – cardiac catheterization – and evolution of the treatment options [5].

**CONCLUSIONS**

The risk factor and the origin for coronary dissection in our case was represented by an atheromatous plaque located on the ascending aorta wall. On the other hand, coronary atherosclerosis was minimal. The morphological feature was an aortic dissection prolonged to the left coronary artery. Hyperemia in vasa vasorum of the affected coronary artery was an associated microscopic finding.

Cardiomegaly with left ventricle hypertrophy suggests an underlying undiagnosed hypertension that could act as a trigger factor. We haven’t identified any lesion suggestive for a traumatic coronary dissection.

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**References**


