Sudden cardiac death due to triple myocardial bridging associated with atypical coronary topography

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Abstract
Myocardial bridging (MB) is defined as the presence of an intramural course of a coronary artery, most likely caused by a defect in resorption of the musculature that encircles the epicardial arteries during morphogenesis [1]. Even tough it is usually without clinical consequences, the presence of a hemodynamically significant myocardial bridging (HSMB), characterized by some particular morphological characteristics (width, angulation, position, etc.) [2], or the association with young age and hypertrophic cardiomyopathy can cause a wide array of cardiac dysfunctions like angina pectoris, ischemic preconditioning, stunning, hibernation (chronic ischemia), second window of protection (against stunning or infarction), acute myocardial infarction, reperfusion effects, remodeling, chronic ischemic heart disease, ischemic cardiomyopathy [3–5].

We present a case of the young man who died suddenly while playing professional football and whose cause of death was acute myocardial infarction associated with multiple myocardial bridges.

Case Report
A male patient, 21-year-old, suddenly collapsed while playing football professionally. The ambulance, which came to the field, found the patient to be in ventricular fibrillation and started cardiopulmonary resuscitation with two 200 J biphasic, asynchronous shocks and adrenaline. After 40 minutes, the patient presented electromechanical dissociation and after another 17 minutes was pronounced dead. Past history didn’t reveal any cardiovascular pathologies. He didn’t fell very well the day before the game, but wanted to play, and most likely had hidden the severity of the symptoms.

Gross pathology examination during autopsy revealed subconjunctival petechial hemorrhages, cerebral and meningeal stasis and edema, a spongy appearance of the thyroid, pulmonary edema, stasis and fibrosis, a small hemorrhage in the posterior gastric mucosa, hepatic and renal dystrophy. Heart was 12.5/11/8 cm, the pericardium was smooth, whitish, with about 38 mL of slightly pinkish pericardial fluid.

The left coronary artery (LCA) had a normal origin and branched in anterior in terventricular (AIVA) and circumflex (LCx) arteries. The AIVA had a subepicardial trajectory for 2.5 cm in the left anterior groove, after which it entered beneath a myocardial bridge for 1.8 cm (with a thickness of 0.3 cm) (Figure 1A). After reemerging in the left anterior groove, it descended toward the right
The main supplier of the diaphragmatic surface of the left ventricle was the left marginal artery as on the diaphragmatic surface of heart, the LCx continued beneath the left atrial myocardium without sending off left ventricular branches and the RCA did not distribute on the left side of the crux cords.

On the inner surface, the AIVA had a circumferential atheroma proximal to the bridging. The AIVA and LCx had discontinuous atheroma plaques that did not diminish significantly the arterial lumen. The myocardium was brown-red, firm, with a violet-red, pale are of 4.5/3.5/1 cm, having a lower consistency compared to the surrounding myocardium, on the posterior side of the left ventricle, with an oblique, intramural position. Myocardial fibrosis was present at the apex and in the postero-lateral area of the left ventricle.

**Histopathology investigation**

Specimens of myocardial tissue from different areas of the anterior and posterior wall of the left ventricle and the right ventricle were taken for histopathology investigation, as specimens from the left coronary artery (two segments from the left anterior descending artery – LAD) and right coronary artery (RCA). Other samples were taken from brain, lungs, liver, kidney, pancreas, spleen, thyroid and adrenal glands. The selected tissue samples were fixed in 10% neutral buffered formalin (pH 7) for 24–48 hours and paraffin embedded. Sections were cut at 5 μm and stained with standard HE and van Gieson, and special stains – Weigert’s elastic, Mallory’s Phosphotungstic Acid–Hematoxylin (PTAH) and Lie. To ensure the reliability of the experimental study, internal quality control was performed as a part of an implemented and certified quality assurance system (ISO 9001/2008). All slides were examined and photographed on a Zeiss AxioImager microscope (Göttingen, Germany). Digital images acquired with Zeiss Axio Vision program have been processed and analyzed with ACDSee Pro.
Results

A wide subendocardial infarct (of about 7–10 days), extended to the subjacent myocardium was noticed in the middle third of the posterior and lateral wall of the left ventricle (Figures 1D, 2a and 2b). Cardiomyocytes with contraction band necrosis were also noticed within ischemic perinecrotic area (Figure 2c).

Lie stain revealed frequent residual perivascular ischemic cardiomyocytes near the infarcted area (Figure 2d). Coronary bridging was observed on both LAD and RCA. In the lung, foci of hemorrhagic infarcts with subpleural extension were found, in association with partial thrombosis of the medium size vessels, on a background of marked congestion and edema (Figure 3).

The other examined organs were within normal histological limits.

Immunobiology

One-step, rapid, immunochromatographic test for the qualitative detection of Poxvirus was carried out and was positive (Figure 4). The ELISA method for detection of IgG antibodies against cytomegalovirus in centrifuged blood serum was done and was also positive, whilst other viral tests yielded negative results (including hepatitis B and C and HIV).
Toxicological screening

Toxicological screening was negative for alcohol or other drugs of abuse.

Thanatochemical analyses

Thanatochemical analyses revealed: total T3=2.56 ng/mL (normal values 0.58–1.59), total T4=9.15 μg/dL (4.87–11.72), TSH=6.725 IU/mL (0.35–4.94), CK-MB in the pericardial fluid 7756.54 IU/L (105–154), troponin – positive, myoglobin – positive.

Discussion

Our case had an atypical combination of myocardial bridging on all three main coronary arteries, associated with a wide hypovascular area on the diaphragmatic and inferior septal area. The presence of atherosclerosis on the LCx, associated with bridging on both the LCx and RCA led to a significant perfusion deficit on the left marginal artery territory, exactly the area in which the myocardial infarction was identified.

The presence of a hemodynamically significant static (atherosclerosis, fibromuscular dysplasia [6]) or dynamic (MB) arterial obstruction, affects the normal blood flow by the formation of an atypical distribution of atherosclerosis and distal coronary hypoplasia [7, 8]. In our case, the distribution of atherosclerosis was preeminent on the LAD, where, proximally from the bridged area was present circumferentially whilst distal hypoplasia was preeminent on the RCA, where its distal part, located after an intramyocardial course was rudimentary, thus needing additional supply from the postero-inferior recurrent artery.

HSMB can lead to sudden cardiac death throughout two main mechanisms: primary ischemic effect (acute myocardial ischemia) or a primary electrical effect [8]. Acute myocardial ischemia is associated with the presence of an additional factor like vasospasm [9, 10], acute thrombosis on another coronary artery [11] tachycardia [12, 13], coronary hypoplasia [14], proximal intimal hyperplasia [6, 15, 16], proximal atherosclerosis [17–25], etc. In our case, the myocardial infarction was about a week old, and had as favoring factors tachycardia (trainings before games), coronary hypoplasia, and proximal atherosclerosis. During the game additional hypoxia and tachycardia caused the development of a malignant arrhythmia, which caused the death.

Normally the posterior infarction is of a lower severity compared to anterior or anterolateral. However, the presence of multiple myocardial bridges led to a significantly increased oxygen consumption caused by compensatory hypercontraction of the anterior wall in response to the reduced postero-inferior wall motion determined by the infarction. This in turn caused severe, global myocardial ischemia, and the augmentation of the infarcted area.

The patient was a professional athlete, with numerous cardiovascular examinations in years preceding his death. However, all were within normal limits, and therefore haven’t raised any suspicion regarding the presence of severe cardiac anomalies.

Conclusions

Myocardial bridging is difficult to diagnose clinically, needing expensive and invasive techniques (intravascular Doppler echography, nitroglycerin enhanced angiography, multislice CT, etc.), not readily available in sports medicine. This may lead to potentially severe MBs to remain undiagnosed, with possible fatal consequences, especially in professional athletes.

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