

Myocardial dissection as rare complication of pseudoaneurysm

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65-year male patient presented to emergency department complaining of progressive short of breathing for the last three months was preceded by attack of chest pain. Patient not asked for medical advice at that time. Transthoracic Echocardiography done reveal pseudoaneurysm of left ventricle at the basal segment of infero-posterior segment with contained pericardial effusion and Patient admitted coronary angiography done and reveal total occluded right coronary artery. Unfortunately, patient refused cardiac surgery and insisted for discharge due to family commitment. After one-month patient presented in emergency department in cardiogenic shock Transthoracic ECHO repeated reveal intramyocardial dissection involve inferior wall and inferior septum. After discussion with cardiac surgeon patient shifted to operation room for emergency surgery aim to control this fatal complication. Intraoperative TEE showed rapidly increasing large extensive intraventricular dissection involve the inferolateral wall and interventricular septum progressing compressing of left ventricular cavity with blood flow at site of insertion of posterior papillary muscle.

Our case displayed an unusual form of cardiac disease secondary to myocardial infarction: ID coexisting with LVA. During the wait for cardiac surgery, the patient experienced deterioration of the ID, which caused a rare form of VSR.

ID is a rare complication of AMI. Dellborg, et al. reported that among 3960 AMI cases, sub-acute cardiac rupture with concomitant ID accounted for approximately 1% of patients. The pathogenesis of ID involves the rupture of intramyocardial vessels in the extracellular matrix, which increases tissue fragility and decreases tensile force in the infarcted area. In cases of microcirculatory obstruction, ischemic reperfusion increases coronary capillary perfusion pressure. ID can appear in the left ventricular free wall, ventricular septum, or right ventricle, but most cases occur at the ventricular septum next to the left ventricular free wall.

Prior to the availability of non-invasive imaging techniques, ID was a postmortem diagnosis. However, even with newer diagnostic tools, definitive diagnosis usually involves more than one test. Angiography of the coronary arteries and left ventricle is considered the gold standard for diagnosing coronary lesions, guiding CABG, and identifying dissecting sacs in the myocardium and/or silhouettes of ventricular aneurysms. However, during angiography, contrast agent is rapidly injected into the left ventricle under high pressure, which increases the possibility of expanding the dissection and inducing cardiac rupture. TTE is a reasonable first examination because it is non-invasive, inexpensive, and portable. Color Doppler echocardiography can indicate a low-velocity flow profile in the abnormal route, sometimes with a bidirectional to-and-fro pattern. Transesophageal echocardiography can evaluate the various myocardial ventricular segments with higher resolution, particularly ID located on the septal and/or right ventricular segments. ID is prone to further extension and rupture and may be in an evolutionary period before cardiac rupture and formation of a pseudoaneurysm, as described in our report and others. Therefore, it is essential to correct ID secondary to AMI. Harpaz, et al. reported that the mortality rate among medically treated patients was 90% and that none of the surgically treated patients died. However, another small case series suggested that of 15 cases, four of six patients died undergoing surgery with simultaneous CABG and ID repair, while in five patients receiving pharmacological treatment, only one died. Sari, et al. also reported a case in which ID resolved spontaneously. Because the data were scattered, derived from case reports and small case series studies, and because of the lack of systemically retrospective reviews of large numbers of consecutive cases, the actual incidence and mortality of ID is not well known. Nonetheless, considering the irreversible progression of coronary arteriosclerosis and the underlying ischemic cardiomyopathy, we emphasize the significance of coronary revascularization at the time of ID repair. In our case, the simultaneous ID and LVA complications of

AMI greatly increased the risk of cardiac rupture, heart failure, and the frequency of ventricular arrhythmias related to the LVA. Therefore, our team successfully performed CABG accompanied by ID repair. More importantly, the surgical repair was conducted with a beating heart, differentiating our study from procedures performed under cardiac arrest in other reports. The beating-heart technique not only avoided aortic cross-clamping, but also facilitated identification of the non-contractile zone of the LVA, avoiding excessive reduction of the residual ventricular volume and insufficient removal of abnormal contractions compared with on-pump LVA linear closure.

In conclusion, with rapid developments in percutaneous coronary artery intervention, the incidence of LVA and ID has declined; however, this creates a new challenge for surgeons confronted with more severe coronary disease. Nevertheless, operative risks are also substantial, and comprehensive evaluation of patients' physical status and appropriate surgical procedures are essential. We believe that complete revascularization combined with surgical correction of concomitant abnormalities is indicated for all patients with a confirmed diagnosis unless there are obvious operative contraindications.

Conclusion: Left ventricular intramyocardial dissection considered as rare complication of myocardial infarction usually of limited size due to rupture of small intramyocardial vessel.

In our case dissection initiated at the site of insertion of posterior papillary muscle with the neck of pseudoaneurysm which could be explained by the tow antagonizing forces during systolic phase the inward force of contracting papillary muscle against the outward pushing of the free wall of the pseudoaneurysm by the high intraventricular pressure during systole. These two opposing forces causes dissection at the friable spiral muscle with more dissection causes disruption of intramyocardial small vessel causes more progression of the dissection

Dissection of myocardium as complication of myocardial infarction at basal segment between the site of insertion of posterior papillary muscle and base of heart in setting of pseudoaneurysm is very rare fatal complication and not reported before.