H106 Postpartum Non-Atherosclerotic Spontaneous Coronary Artery Dissection (NA-SCAD) Recurrence in Subsequent Pregnancies: A Case Report

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After attending this presentation, attendees will be aware of the risk of NA-SCAD in subsequent pregnancies, the associated histological findings of SCAD, and the current etiological thinking behind this rare entity.

This presentation will impact the forensic science community by introducing the concept of recurrent SCAD in subsequent pregnancies, a previously unreported phenomenon.

NA-SCAD, or separation of the layers of the coronary arterial wall, is a rare cause of sudden cardiac death predominantly seen in young, healthy women. One retrospective single-center cohort study of 87 patients with angiographically-confirmed NA-SCAD identified a predilection for women (82%) with the most common female association being postpartum status (18%). The mean age of postpartum patients was 33 years and the mean postpartum time frame was 38 days. Of the 87-patient cohort, 91% presented with chest pain, 49% had ST-elevation myocardial infarction, and 14% had a life-threatening ventricular dysrhythmia. The left anterior descending coronary artery was the most common location for dissection (71%).

While intimal tears have been suggested as the inciting event of NA-SCAD, these tears are rarely found at autopsy. Another proposed model is supported by the location of the plane of dissection, which is commonly between the tunica media and tunica adventitia. In this model, disruption and bleeding of the vasa vasorum leads to intra-medial hemorrhage and hematoma formation. Axial propagation occurs due to ongoing hemorrhage and clot formation in the absence of an intimal tear. Luminal collapse ensues. Originally considered a reactive phenomenon, the presence of eosinophilic infiltrates in 50% of NA-SCAD, with their collagenase-containing granules, suggests their primary, or at least propagating, role in dissection.

Several mechanisms have been proposed for increased peripartum risk of SCAD. Progesterone excess has been associated with elastic fiber disarray and loss of acid mucopolysaccharide ground substance, while estrogen-associated release of matrix metalloproteinase has been shown to result in cystic medial necrosis. These combined hormonal effects have been proposed to result in a loss of structural support of the vasa vasorum with resultant susceptibility to rupture, especially in the setting of pregnancy-induced hemodynamic stress.

A 30-year-old Hispanic female with a past medical history significant only for two cesarean sections was driving with her two children, ages three months and three years, when her car was seen to slowly drift off the road and come to a stop. A bystander found the woman slumped in the driver's seat with no signs of trauma. Her two children were secured in the back seat. Emergency medical services responded quickly, found the woman to be in ventricular fibrillation, and performed cardiac defibrillation without success. The woman was transported to the closest regional medical center where additional unsuccessful attempts at cardiac resuscitation were performed. In addition to being three months postpartum, the decedent had complained of chest pain one week prior to death. She did not smoke or use prescription or illicit drugs.

External exam revealed a well-nourished, Hispanic female (BMI 22.9kg/m2). Autopsy revealed a heart of normal shape and size (230 grams) with 90%-99% luminal reduction of the Right main Coronary Artery (RCA) beginning just distal to the right marginal branch and continuing through the distal posterior descending branch. Close inspection of the RCA revealed a pinpoint lumen surrounded by clotted blood. Sectioning of the heart revealed an extensive, white fibrous infarction of the anterior left ventricle and interventricular septum extending from the apex to the level of the mitral valve cusps in the distribution of the Left Anterior Descending (LAD) coronary artery. The LAD itself had no grossly identifiable abnormalities and there was no coronary or other arterial atherosclerosis.

Histological examination of the heart confirmed extensive replacement of the left ventricular free wall and adjacent interventricular septum by a dense collagen scar. Examination of the RCA revealed near-complete luminal compression by an extra-luminal, antemortem thrombus associated with extensive adventitial destruction. Eosinophilic infiltration of the tunica adventitia with areas of adventitial necrosis, myxoid change, and adjacent perivascular fat necrosis was prominent. There was also scattered early formation of fibrovascular granulation tissue at the outer margins of adventitial dissection.

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The remote myocardial infarction described in this patient is the result of NA-SCAD of the LAD in the peripartum period of the first pregnancy and it is proposed that this is the first case of recurrent SCAD in the setting of subsequent pregnancy. Therefore, monitoring survivors of pregnancy-associated NA-SCAD is strongly recommended. In the setting of autopsy, Magnetic Resonance Imaging (MRI) should be considered in the evaluation of young, healthy, and especially peripartum females who present with sudden, unexplained death.

Coronary, Dissection, Pregnancy