G57 Non-Arteriosclerotic Myocardial Infarction in a 38-Year-Old Female

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After attending this presentation, attendees will be able to develop a differential diagnosis for the underlying etiology of myocardial infarction, particularly as it relates to spontaneous dissection of epicardial coronary arteries.

This presentation will impact the forensic science community by increasing awareness of the recognition of conditions and risk factors for non-arteriosclerotic myocardial infarction, particularly in women, aiding in proper diagnosis and accurate death certification. Additionally, identification of hereditable thrombophilias followed by appropriate referral of family members embodies our public health function.

A 38-year-old female presented to the emergency department two consecutive days with complaints of chest pain and nausea. On second presentation, she sustained a seizure on transfer to a stretcher. ST elevation and elevated troponin were documented before death. The decedent did not smoke or use drugs recreationally. At autopsy, the heart was 340 grams and showed transmural infarction with perforation of the posterolateral free wall near the apex, associated with an apparent occlusive, long segment thrombus in proximal circumflex coronary artery. Analysis of antemortem blood from the referring hospital was negative for Factor V Leiden and Prothrombin G20210A mutations. Functional assays for antithrombin and Protein C were normal; however, Protein S activity was 3%. Histological examination of the left circumflex artery showed dissection within the media with reactive inflammation within the adventitia.

Although myocardial infarction is twice as common in men, heart disease is the number one cause of death in women in the United States. The risk of myocardial infarctions in women is often underestimated and symptoms often go unrecognized. There are also studies that indicate that females are less likely to survive a myocardial infarction, particularly younger women. Spontaneous Coronary Artery Dissection (SCAD) is a rare cause of myocardial infarction. Since the condition was first described in 1931, fewer than 200 cases have been reported in the medical literature. There are currently no known direct causes of this condition, although some correlations have been noted. Many patients are women (73% of cases) in the peripartum period or of childbearing age (mean age: 39 years), with few or no risk factors for coronary artery disease. Other associations include contraceptive use and connective tissue disorders, Ehlers-Danlos and Marfan syndromes, and autoimmune vasculitides such as polyarteritis nodosa, Churg-Strauss Syndrome, systemic lupus erythematosus, and antiphospholipid syndrome. Most of the reported dissections have occurred in the left anterior descending coronary artery.

The most common presentation is sudden cardiac death (60% to 80%). SCAD also causes angina, myocardial infarction, arrhythmias, and pump failure. To be classified as spontaneous, dissection must occur in the absence of trauma, previous surgery or catheterization, or extension of an aortic dissection. Coronary artery dissection, while having some morphological and mechanistic similarities to aortic dissection, seems to be pathogenetically distinct. Unlike aortic dissections, no association with hypertension has been noted. Many of the proposed pathogenetic mechanisms for SCAD pertain to changes in vascular wall properties leading to weakening of the media. These include changes in smooth-muscle-cell metabolism, the effect of proteases released from eosinophilic infiltrates, and pregnancy-related changes in connective tissue. The striking predilection for women and histopathological findings suggest changes in the arterial wall due to hormonal influences.

Protein S serves as a cofactor for Protein C to inactivate factor Va and factor VIIIa in the coagulation cascade. Low Protein S activity is associated with acquired conditions such as liver disease, contraceptive and menopausal hormone use, pregnancy, acute and chronic inflammatory illness, sepsis, consumption by acute thrombosis, or concurrent warfarin therapy. Deficiencies of Protein S, Protein C, and antithrombin are associated primarily with an increased risk for venous thrombosis, and seem to play little or no role in development of arterial thrombosis, leading to heart attack and stroke. However, a recent study suggests that Protein C and Protein S deficiencies may be associated with an increased risk for arterial thrombosis in people younger than 55 years of age.

Myocardial Infarction, Coronary Artery Dissection, Hereditary Thrombophilia

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