G120 Plaque Morphology in Subjects With Coronary Disease Who Suddenly Died

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After attending this presentation, attendees will be able to better understand the main pathological features of atherosclerotic plaque and pathogenesis of artery thrombosis in coronary sudden death.

This presentation will impact the forensic science community by increasing awareness that Sudden Coronary Death (SCD) depends frequently on active atherosclerotic lesions.

Most of Acute Coronary Syndromes (ACS) are precipitated by luminal thrombi, which arise from three different plaque morphologies: rupture, erosion and calcified nodules. Of these, plaque rupture is the most frequent, accounting for 60 – 75% of cases. The purpose of the present study was to determine the frequency of active and inactive coronary lesions and Myocardial Infarction (MI) in people with SCD.

SCD is defined as a sudden unexpected death within one hour of onset of acute symptoms from a stable medical condition or the death of a person who had been seen in stable condition less than 24 hours antemortem. No other potentially lethal cardiac or noncardiac cause of death could be present, including toxicology screening. The presence of acute thrombosis (collections of platelets, fibrin, and trapped erythrocytes and white blood cells) and disrupted coronary plaques (disruption of the luminal fibrous cap with fissure or rupture into a lipid core) was noted. An active coronary lesion was defined by the presence of a disrupted coronary plaque, luminal thrombus, or both (luminal thrombus in the area of a ruptured plaque). An inactive lesion had a luminal stenosis ≥75%, but lacked both plaque disruption and thrombus. Organized thrombi consisted of granulation tissue and recannalized channels within the arterial lumen with or without fibrin. Healed MI was identified by focal macroscopic replacement of the myocardium by scarring, with histological confirmation. Acute MI was diagnosed by the presence of coagulation necrosis with or without an associated inflammatory infiltrate.

The distribution of coronary lesions for the entire group of hearts (166 cases) was: acute thrombosis in 87 (53%), disrupted coronary plaque (with or without acute thrombosis) in 54 (32%), and organized thrombus in 25 (15%). There were also active coronary lesions in 49 cases (30%). The association of thrombosis and disrupted coronary plaque was as follows: acute thrombosis with plaque disruption in 49 cases (30%) and acute thrombosis without plaque disruption in 38 (23%). Disrupted plaque without acute thrombosis was identified in 5 cases (3%) among the 72 coronary lesions with ≥75% luminal stenosis. Plaque hemorrhage of any size was present in 96 cases (58%); of these, plaque hemorrhage occupying ≥25% of total plaque area was identified in 89 (54%). Thus, SCD could be attributed to active coronary lesions (thrombus, disrupted plaque, or both) in 141 cases (85%). There were 22 cases (13%) without an active coronary lesion which had an acute (two cases) or healed (20 cases) MI. In the 34 cases without an acute or a healed MI, 17 (50%) had only inactive coronary artery lesions such as severe atherosclerosis without acute coronary thrombosis or plaque disruption. The examination of the myocardium revealed acute MI only in 16 hearts (10%), both acute and healed MI in 18 (11%), healed MI in 68 (41%), and the absence of MI in 63 (38%). In hearts with acute MI, the infarct was identified macroscopically in 9 of 27 cases. Of these 27, there was a transmural infarct in 20 cases and subendocardial in seven cases. Platelet-fibrin emboli in small intramyocardial coronary arteries were found in 13 cases (8%) and only in cases with an acute coronary thrombosis.

Acute changes in coronary plaque morphology (thrombus, plaque disruption, or both) were found in 85% of SCD cases. In hearts with myocardial scars and no acute infarction, active coronary lesions were identified in 31 (46%) cases. Neither myocardial infarction (acute or healed) nor active coronary lesion was present in 38 (53%) cases.

Sudden Death, Atheroscler Plaque, Myocardial Infarction

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