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Spontaneous Coronary Atheroembolism with Multiple Infarctlets.

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Atherosclerosis is a disease of elastic and the larger muscular arteries. In the heart it affects only the epicardial branches of the coronary arteries. Pathology shows the deposition of toxically altered light density lipoprotein (LDL) in the intima (inner lining) of the vessel wall. The toxically altered LDL is taken up scavenger macrophages and phenotypically altered smooth muscle cells. The presence of the lipid within the cytoplasm causes the formation of vacuoles when the tissue is processed in alcohol, and the lipid is dissolved out. This gives a foamy appearance to the cytoplasm and these cells are called “foam cells”. If there is no resolution of the lesion, the foam cells are killed by the toxic LDL, and liberate their content of lipid is released into the extracellular matrix, giving rise to the formation of a lipid core. The scavenger macrophages release many proteolytic enzymes, which break down the tissue, leading to weakening of the vessel wall and the smooth muscle cells lay down fibrous tissue as a fibrous cap over the lesion. Subsequent events depend upon the number of smooth cells, the relative strength of the fibrous cap, the extent of the lipid core and the number of macrophages, releasing proteolytic enzymes. In the event of there being a thin fibrous cap (less than 65 microns a large lipid core and more than 25 macrophages per high power field, the fibrous cap can rupture, with release of the lipid, atheromatous material into the lumen of the vessel, where it can be carried with the blood flow and impact downstream, in another vessel-this is the entity of atheroembolism. It is a well known complication of ulceration of atheromatous lesions in the aorta, with the kidney being a common site for atheroembolism . Coronary atheroembolism may occur as a complication of modern intravascular procedures such as percutaneous angioplasties, but, spontaneous coronary atheroembolism is rare. To the best of our knowledge, this is the first documented case of it.

Going through archival tissue, predating 1967, we were interested to find a case of coronary atheroembolism with multiple infarctlets (small foci of myocardial necorosis). Figure 1 shows one such intramyocardial vessel. The lumen is completely occluded. It shows the presence of cholesterol clefts, surrounded by an inflammatory reaction, which includes giant cells. There is one small focus of revascularization. Figure 2 shows multiple subendocardial, irregular areas with loss of myofibers and their replacement by young fibrous tissue-granulation tissue. These are organizing microscopic infarcts – infarctlets-, which are about a week old.

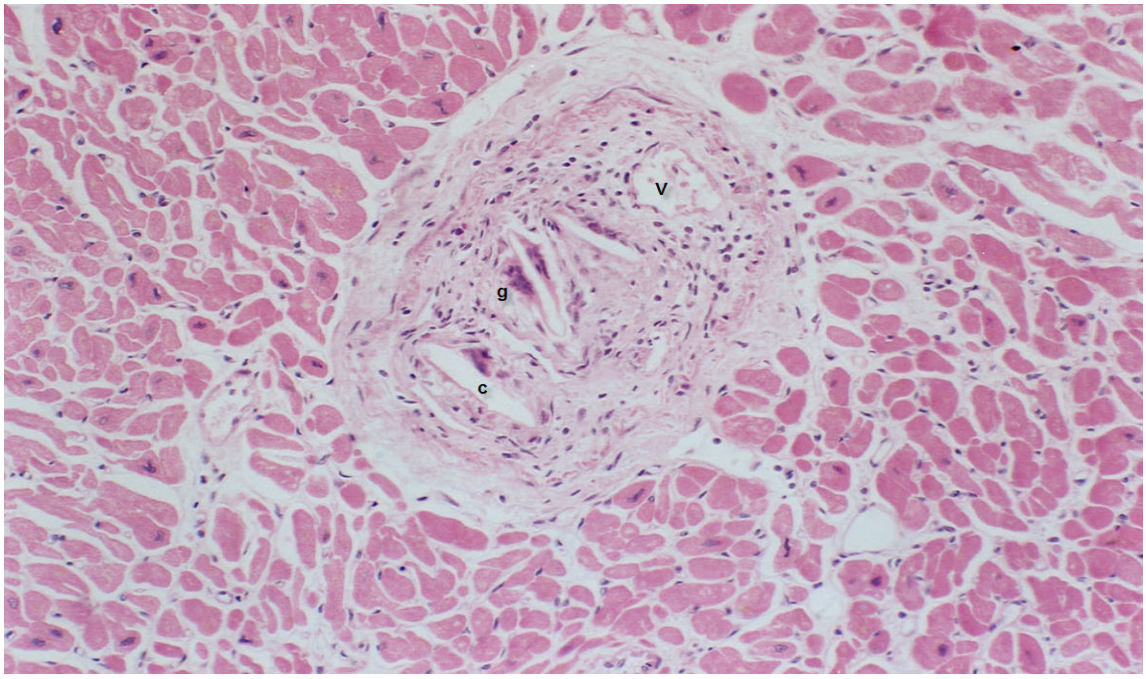

FIG. 1. Atheroembolism of intramyocardial vessel: -c-cholesterol cleft; g-giant cell; v-revascularization (Original magnification 200x)

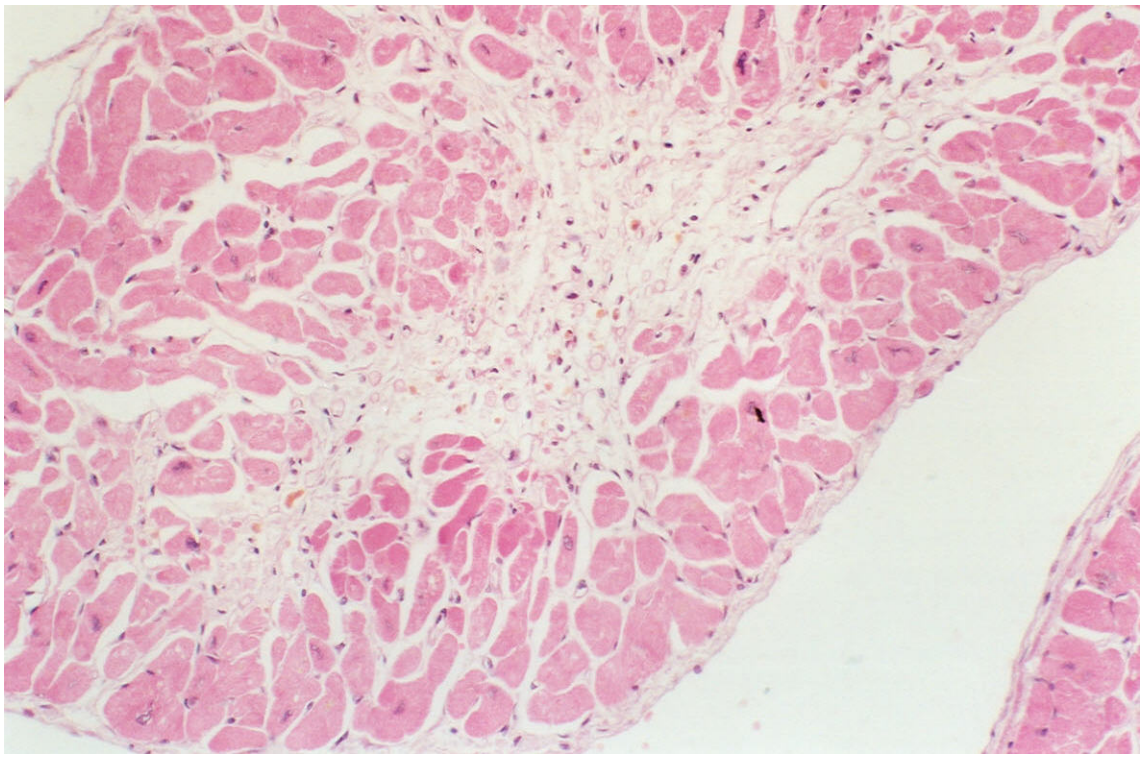

FIG 2 Organization of infarctlet by ingrowth of granulation tissue (Original magnification 200x)

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