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Pathophysiology of Acute Myocardial Infarction

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More than 80% of acute myocardial infarcts are the result of coronary atherosclerosis with superimposed luminal thrombus. Uncommon causes of myocardial infarction include coronary spasm, coronary embolism, and thrombosis in nonatherosclerotic normal vessels. Additionally, concentric subendocardial necrosis may result from global ischemia and reperfusion in cases of prolonged cardiac arrest with resuscitation. Myocardial ischemia shares features with other types of myocyte necrosis, such as that caused by inflammation, but specific changes result from myocyte hypoxia that vary based on length of occlusion of the vessel, duration between occlusion and reperfusion, and presence of collateral circulation.

Gross pathologic findings

The earliest change that can be grossly discerned in the evolution of acute myocardial infarction is pallor of the myocardium, which occurs 12 hours or later after the onset of irreversible ischemia. The gross detection of infarction can be enhanced by the use of tetrazolium salt solutions, which form a colored precipitate on gross section of fresh heart tissue in the presence of dehydrogenase-mediated activity. Myocardial necrosis can be detected as early as 2 to 3 hours in the dog and in man by this method [1,2]. In nonreperfused infarction, the area of the infarct is well defined at 2 to 3 days with a central area of yellow discoloration that is surrounded by a thin rim of highly vascularized hyperemia (Fig. 1A–C). In a reperfused infarct the infarcted region will appear red from trapping of the red cells and hemorrhage from the rupture of the necrotic capillaries (Fig. 1D). At 5 to 7 days the regions are much more distinct, with a central soft area and depressed

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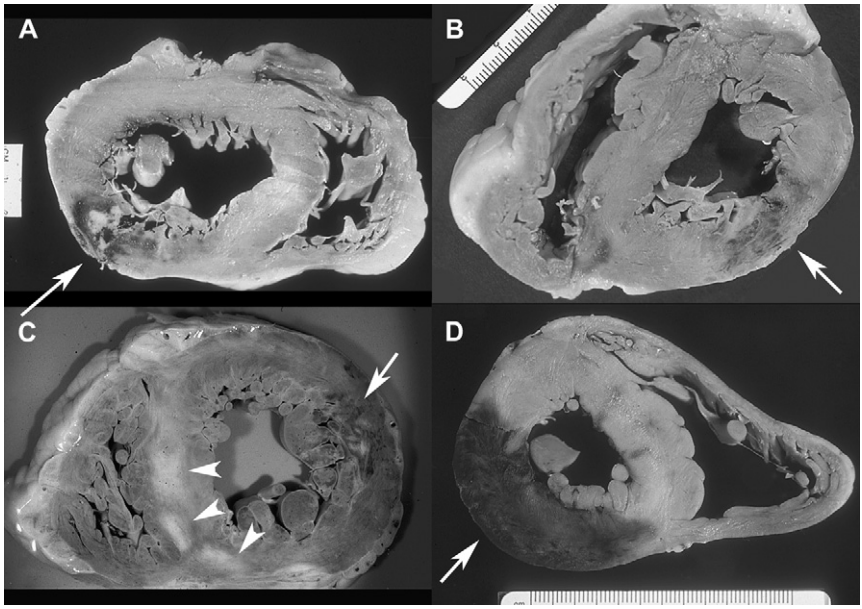

Fig. 1. Acute myocardial infarction. (*A*) Rupture of acute infarction at day 3 post symptoms (*arrow*). Note hyperemic border surrounding pale area. No reperfusion occurred. (*B*) Acute myocardial infarction, 4 days after onset of symptoms. Hemorrhagic area, with no central pallor (*arrow*). Partial reperfusion may have occurred with attempted thrombolysis up to 1 day after symptoms. (*C*) Healing myocardial infarct (*arrow*) 19 days after initial ECG changes. Note persistent pale areas in center of infarct. Older infarcts are seen (*arrowheads*) in the septum. (*D*) Acute reperfusion infarct (*arrow*). Death 2 days after thrombolysis for acute infarct. Note diffuse hemorrhage and lack of central pallor.

hyperemic border. At 1 to 2 weeks the infarct begins to be depressed (Fig. 2), especially at the margins where organization takes place, and the borders have a white hue. Healing may be complete as early as 4 to 6 weeks in small infarcts, or may take as long as 2 to 3 months when the area of infarction is large. Healed infarcts are white from the scarring and the ventricular wall may be thinned (aneurysmal), especially in transmural infarction. In general, infarcts that occupy more than 50% of the ventricular wall, from the subendocardial to the epicardial surface, are considered transmural and associated with Q-wave changes on electrocardiogram.

Light microscopic findings in nonreperfused infarction

The earliest morphologic characteristic of myocardial infarction occurs between 12 to 24 hours after onset of chest pain. Hypereosinophilia of the cytoplasm as assessed by hematoxylin–eosin staining is characteristic of myocardial ischemia (Fig. 3A). Neutrophil infiltration is present by 24 hours at the border areas. As the infarct progresses between 24 and 48 hours,