EDITORIALS

Acute Myocardial Infarction without Coronary Arteriographic Abnormalities

Angina pectoris, as well as acute myocardial infarction and left ventricular aneurysm, may all occur in the absence of obstruction, as demonstrated on high-quality coronary arteriograms. While obstructive disease in the coronary arteries may indeed be missed on coronary arteriograms, studies of necropsies have demonstrated that myocardial infarction may occur with minimal or no luminal reduction due to atherosclerosis. We have recently reported the findings in 13 patients who had normal or near normal coronary arteriograms following a documented myocardial infarction 2 to 48 months earlier. Nine patients were 30 years of age or less. Eight of these nine were male patients, suggesting to us the occurrence of this syndrome predominantly in otherwise healthy young men. Four of these eight male patients had recently participated in strenuous exertion or had experienced moderate trauma.

The etiology of the syndrome of myocardial infarction without abnormal coronary arteriograms is, of course, unclear. Coronary embolism or coronary arterial spasm is a possibility. Pad-like cellular structures which bulge into the lumen of the left coronary artery and appear to be coronary arterial sphincter apparatus have been described in man. Functional disorders of this apparently normal vascular structure may be related to coronary arterial spasm and may predispose to acute myocardial infarction. Also, cushion-like lesions may occur in human intramyocardial arteries. While the pathogenesis of these latter lesions is not clear, hemodynamic trauma or aggregates of platelets, or both, may be involved in the initial injury. Dissolution or recanalization of induced coronary arterial thrombosis has been demonstrated experimentally.

Platelets may aggregate as part of the blood's response to injury, and this response may be initiated by extravascular, as well as intravascular, stimuli, including adenosine diphosphate. Aggregation of platelets may be induced by adenosine diphosphate released from injured cells or from platelets themselves. Such aggregates may then embolize from the site of origin. Aggregates of platelets in humans may cause functional disturbances or injury to tissue by impeding the microcirculation. Experimentally, infusions of adenosine diphosphate into the myocardial circulation of swine has been demonstrated to cause transient circulatory collapse, as well as electrocardiographic evidence of myocardial ischemia. In swine allowed to live two hours or more, gross, as well as light-microscopic and electron-microscopic, evidence of myocardial infarction was present.

Whether the recent excessive physical exertion or moderate trauma present in the four patients with acute myocardial infarction in our series could have played a role by initiating a chain of events similar to that described herein is entirely unknown; however, such a possibility with acute coronary embolism by aggregates of platelets, with subsequent lysis, retraction, or recanalization, would be consistent with the hypothesis advanced by Arnett and Roberts in consideration of this syndrome.

While there was slight, but measurable, depression of cardiac function, as judged by the left ventricular ejection fraction, in five patients in our series, the prognosis for the group seemed favorable for the follow-up period from 2 to 99 months. Investigation of patients with unusual clinical circumstances or presentation of acute myocardial infarction may provide useful clues in understanding the more common illness and its precipitating events and thus hopefully lead to its possible prevention.

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Additional references are available from the authors.

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Alveolitis

A Functional Tip for the Morphologic Crutch

When Averill Liebow (Providence shine her light upon him!) first marched us from unlettered barbarism to horizons of pulmonary vision, the truth seemed imminent, even immanent. Digestion of the diagnostic alphabet soup of BIP, DIP, GIP, LIP, and UIP would provide histopathologic guile and enlighten us about prognosis and therapy. From that promontory, Liebow’s disciples and critics have swarmed among a thousand islands of controversy, where dwell the pigeon-fanciers, grain-stokers, industrial snifters and snufflers, and the intensive care-ologists who (wiser than their leech-setting predecessors a bicentennial ago) provide abundant oxygen tensions to men, women, and babes in need. Meanwhile, Western culture progresses in the public and private airs of our times. “Interstitial” disease, whatever obscurity it was, has become an ascending medical problem.

But what is interstitial disease? In fact, most pulmonary diseases, cancers included, are largely “interstitial” in their manifestation. Bacterial pneumonias nowadays organize there, immunopathology is expressed there, and so are viruses, Mycoplasma, hyaline membrane disease, “shock lung,” and radiation. With the ingenious concept that desquamated intra-alveolar cells represent interstitial disease, one is left with two main kinds of pulmonary problems: (1) obstructive or (2) “interstitial.” The Liebovian lamp is brighter than first we thought! But how may one decide if “desquamative” disease is an illuminating concept?

Liebow, an astute tactician, invented the “interstitial” game rules but, like all pathologists, could only view innings and not whole games. From his one-inning slides, he surmised the score. Considering desquamation as a type (his italics), rather than a phase, of reaction, Liebow discovered that patients with desquamative interstitial pneumonitis played a better game than those with usual interstitial pneumonitis. But for desquamation to be a type (figuratively, a prototype), its morphologic and clinical pattern should be distinctly identifiable. Alas! Enter controversy. No, not so, says the team from Boston and New Haven, Conn, on the American Atlantic coast. Eastern detailed clinicopathologic correlation says that desquamative interstitial pneumonitis exists; however, along the British Atlantic coast, desquamative interstitial pneumonitis has never been a game. Instead, their sport is called “alveolitis” and is played with a Pepys, a Scadding, and divers other implements. One can see in the Clevelanders’ title a certain reluctance to say whose game they are playing! In any event, British prognosis is gloomy, with or without desquamation, while Cleveland finds room for optimism or at least steroid therapy in either case, and New England views desquamative interstitial pneumonitis as a tradition of sorts. Maybe we’d best take a look at the rules to see that we are all looking at the same game. Meanwhile, score some points for Cleveland.

Tubbs and colleagues propose “rigid” cytotlogic criteria for desquamation, so that a plethora of macrophages might be separated from one of type 2 cells. Turnabout, and they have soft criteria for desquamation which allow for multifocal, as well as uniform, diffuse exfoliation. Yet, although they give some thrill to cross-correlation, they don’t show the wealth of detail to which Gaensler, Car-