Letter to the Editors

Why is atherosclerosis non-existent in human intramyocardial coronary arteries?

To the Editor

In explaining the 'neglected' phenomenon of absence of atherosclerosis in human intramyocardial (or intramural) coronary arteries, Scher advanced the theory of 'protective' action of myocardial contraction on the transfer phase of the development of atherosclerosis [1]. He also cited as supporting evidence that atherosclerosis is rare beneath myocardial bridges in superficial coronary arteries [2] where myocardial contraction plays a significant role but common in venous bypass grafts [3] which are superficially located and therefore not subject to the effects of myocardial contraction. If contraction of the myocardium is the sole or principal mechanism in preventing atherosclerosis in human intramyocardial coronary arteries, then a paradox exists in hypertrophic cardiomyopathy. In the latter condition where strong myocardial contraction is the sine qua non, atherosclerosis of intramyocardial coronary arteries is common, existing in more than 80% of the patients studied at necropsy [4–6], although the epicardial coronary arteries are typically free of obstructive ather sclerotic disease [7]. Although abnormal intramyocardial coronary arteries which were found in three of the eight infants with hypertrophic cardiomyopathy [5] could represent a congenital component of the underlying cardiomyopathic process [5,8], this finding still poses a strong challenge to the theory that myocardial contraction per se is 'protective' against atherosclerosis. Perhaps it is the altered flow pattern that results from myocardial contraction rather than myocardial contraction per se that is mainly responsible for absence of atherosclerosis in human intramyocardial coronary arteries. In the case of myocardial bridges, flow under the bridges is laminar and therefore less intimal thickening is produced there, whereas the intimal thickening proximal to the bridging is striking due to greater force from arterial blood flow at the boundary area [2]. This also explains why coronary arteriosclerotic plaque formation is enhanced just proximal to branching points with relative sparing of the flow dividing regions [9].

References


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