Guest Editorial

Coronary Artery Disease: A Pediatric Perspective

Atherosclerosis is a process which leads to the developments of obstructive coronary artery disease. It is a complex pathologic process which takes shape over an extended period of time. Endothelial factors and plasma constituents such as cholesterol in its various structural forms have been implicated. Why cholesterol accumulates in the body in pathologic levels, i.e. essential hypercholesterolaemia has been elucidated by discovery of hepatic cholesterol binding receptors sites, and, its genetic linkage has also been elucidated. The risk factors such as smoking, diabetes, hyperlipidemia, and hypertension have no direct cause and effect relationship to atherosclerotic coronary artery lesions, although a significant statistical association to coronary artery atherosclerotic lesions has been shown. Atherosclerosis is a slow process and takes years to develop. Recent evidence cited by Berenson et al (NEJM 1998; 338:1650) has shown that the extent of fatty streaks and fibrous plaques (hallmark of established atherosclerosis) in aorta and especially coronary artery increase with age. Furthermore, risk factor such as smoking, body mass index, systolic and diastolic blood pressure, serum concentration of total cholesterol, LDL and HDL cholesterol singly but especially when combined were strongly associated with extent of lesions within the coronary arteries of children. The striking observation cited was that coronary arteries are involved in children as young as two years age and by teenage prevalence increased substantially. It seems reasonable to conclude that atherosclerosis starts during childhood with consequences of unchecked progress of plaques resulting in angina, myocardial infarction and death in adult life.

There is strong evidence to suggest that risk factors can be identified during childhood and furthermore risk factors tend to persist from childhood to adult life so that hypertensive or hypercholesterolaemic child is very likely to become hypertensive and hypercholesterolaemic adult. Therefore, it is tempting to speculate that childhood lesions in coronary arteries will regress with modification of the risk factors. This speculation is based on the observation that established lesions in the adults tend to regress or stabilize with cholesterol reducing therapies, i.e. diet and life style modification. Therefore, unless a key is discovered in the 21st century which would unlock the genetic and environmental secrets that produce atherosclerosis, we would continue the preventive strategy of risk factors modification.

The method of risk factor identification and modification obviously needs to be developed into general recommendations which can be applied, starting from early childhood at an individual, community or national level. Our aim should be to reduce, if not eliminate, coronary artery disease by the middle of the 21st century.

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