Editorial

RISK FACTORS FOR CORONARY ARTERY DISEASE

Although cardiac care is available in Saudi Arabia, it has been noted that coronary artery disease has been increasing in frequency. However, there are no studies that describe the prevalence and incidence rates of the disease in this country.

Recently, King Abdulaziz City for Science and Technology considered, as one of its priorities in the 14th annual grants program for applied research, to study coronary artery and blood circulation diseases among Saudis. The objectives were to identify the rate of occurrence and prevalence of coronary artery and blood circulation diseases in the society, to identify the risk factors leading to these diseases, and to suggest recommendations that might lead to the reduction of these diseases.

We are, in this part of the world, very fortunate to make use of the available data and knowledge from the rest of the world regarding coronary artery disease. The declining mortality of this disease that has been observed over the past four decades in western countries has been partly attributed to lifestyle changes, especially to a decrease in serum cholesterol and cigarette smoking.[1,2]

Several controlled clinical trials of the lipid hypothesis have shown that reducing serum cholesterol will reduce the overall coronary artery risk. Yusuf and colleagues[3] combined the results of 22 randomized trials that involved more than 40,000 subjects. In these trials, various means were used to alter serum cholesterol. The duration of these trials ranged from 1 to 7 years. In the overall analysis, the treated subjects had 23% fewer coronary events than the controlled subjects, a highly significant difference. Rossouw and colleagues[4] performed a meta-analysis of the results of four primary prevention trials. They found a 10% decrease in the total cholesterol mean reduction of 25%, 12%, and 22% in the number of nonfatal, fatal, and all myocardial infarction cases, respectively.

Brown and co-workers[5] showed regression of coronary artery disease as a result of intensive lipid-lowering therapy in men with high levels of apolipoprotein B. The patients were followed up for 2.5 years and then underwent repeat angioplasty. Quantitative angioplasty also showed atherosclerotic lesion regression with treatment (versus lesion progression) in a control group conducted by the University of California at San Francisco arteriosclerotic specialized center for research intervention trials.[6] It is therefore very evident from these previous trials that modification of lipids will reflect on the general public with reduction in coronary artery events.

With regard to tobacco use, Goldman and Cook[2] attributed as much as 24% of coronary artery disease reduction to smoking cessation. Despite the theoretical attractiveness of low nicotine and low carbon monoxide cigarettes, clinical studies have not shown that low nicotine cigarettes reduce the incidence of myocardial infarction.[7]

Diabetes mellitus is a well-established risk factor for coronary artery disease.[8] A number of epidemiological and clinical studies implicate hyperinsulinemia and insulin resistance in an increased frequency of coronary artery disease.[9] Insulin resistance decreases lipoprotein lipase levels which lead to increased lipolysis in adipose tissue, increased free fatty acid level to the liver, and enhanced triglyceride and very low-density lipoprotein secretion.[10] On the other hand, weight loss with normalization of glucose enhances the activity of lipoprotein lipase with reduction of very low-density lipoprotein and an increase in high-density lipoprotein level.

The declining rate of fatal cerebrovascular events apparently reflects the growing awareness of hypertension as a cardiovascular risk factor as well as the improved availability and effectiveness of well-tolerated antihypertensive agents. However, these advances against hypertension have not translated into fewer deaths from coronary artery disease. This was attributed to modifying other risk factors, such as hypercholesterolemia,

while using the antihypertensive agents. The new antihypertensive agents have a neutral effect on the level of cholesterol that may reflect improvement on the outcome of patients with coronary artery disease. While conducting our national study, we need to consider the major factors for coronary artery disease and the minor factors such as the hypercoagulability status[11] which include the fibrinogen level, antithrombin III, the interaction between lipoproteins and alpha-2 antiplasmin, and others.[12]

The objectives of the national study as stated by King Abdulaziz City for Science and Technology are to prevent coronary artery disease and to have the public practice positive health behaviors. However, if we now direct our attention only to managing the small number of established cases, we would be wasting our efforts because these cases reflect only a small sector of the population. An example of this would be studying the role of interventional cardiology such as angioplasty, atherectomy, laser therapy, and angioscopy. To concentrate our efforts on these very specialized areas will only satisfy the desire of a few individuals.

Therefore, it should be clear from the outset that we need to determine whether the risks for coronary artery disease in Saudi Arabia are the same as in other countries. We also need to emphasize the preventive aspects of coronary artery disease in addition to studying methods for treating individual patients.

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References

1. American Heart Association. Heart and stroke facts. Dallas: American Heart Association, 1991.
2. Goldman L, Cook EF. The decline in ischemic heart disease mortality rates: an analysis of the comparative effects of medical intervention and changes in lifestyle. Ann Intern Med 1984;101:825-36.
3. Yusuf S, Wittes J, Friedman L. Overview of results of randomized clinical trials in heart disease with aspirin and risk factor modification. JAMA 1988;260:2254-9.
4. Rossouw JE, Lewis B, Rifkind BM. The value of lowering cholesterol after myocardial infarction. N Engl J Med 1990;323:1112-9.
5. Brown G, Albers JJ, Fisher LD, et al. Regression of coronary artery disease as a result of intensive lipid-lowering therapy in men with high levels of apoliprotein B. N Engl J Med 1990;323:1289-98.
6. Kane JP, Malloy MJ, Ports TA, et al. Regression of coronary atherosclerosis during treatment of familial hypercholesterolemia with combined drug regimens. JAMA 1990;264:3007-12.
7. Palmer JR, Rosenberg L, Shapiro S. "Low yield" cigarettes and the risk of nonfatal myocardial infarction in women. N Engl J Med 1989;320;1569-73.
8. Garcia MJ, McNamara PM, Gordon T, et al. Sixteen-year follow-up study. Morbidity and mortality in diabetes in the Framingham population. Diabetes 1976;23: 105-12.
9. Stolar MW. Atherosclerosis in diabetes: the role of hyperinsulinemia. Metabolism 1988;37(2 Suppl 1):1-9.
10. Reaven GM. Insulin resistance and compensatory hyperinsulinemia: role in hypertension, dyslipidemia and coronary artery disease. Am Heart J 1991;121:1283-9.
11. Bronze GJ Jr. Endothelial injury, coagulation and atherosclerosis. Coronary Artery Dis 1991;2:131-40.
12. Small M, Lowe GD, Beattie JM. Severity of coronary artery disease and basal fibrinolysis. Haemostasis 1987;17:305-11.