AN OVERVIEW OF RISK FACTORS FOR CORONARY HEART DISEASE AND PREVENTION STRATEGIES

* M .A. Siddiqui

In developed countries, coronary heart disease (CHD) accounts for 40-50% of all deaths, while 15-35% of all cardiac admissions in hospitals in our country are due to coronary heart disease[1]. The prevalence of CHD in our country varies from 1.7% to 6.5%[2] and is rising, affecting young and middle aged persons in prime of their life and career [3a] and it is said that CHD will replace infectious diseases as the major killer in India by year 2015 A.D.[3b]. Indian immigrants settled abroad have higher incidence and higher mortality as compared to native population. [4a,b]. The famous cardiologist Paul Dudley White has said "Heart disease before the age 80 is not God's will but due to our own faults" and it is the same message as given by Charaka about 30 centuries back, in 1000 B.C., who emphasized the role of overeating, heavy and fatty meals , worries and sedentary habits in causation of heart disease[5]. The human and economic costs of such a situation are very heavy.

Atherosclerosis, the term introduced by Marchand in 1904, is the lipid rich lesion in the innermost layer of the arteries which is responsible for the majority of cases of myocardial and cerebral infarction. The endothelial lining of the artery is in direct and continuous contact with blood and interaction of blood cells & other constituents with the endothelium gives rise to atherosclerosis. This lesion presents as either fatty streak which is made up of foam cells, lipids & T lymphocytes and occurs early, commonly in children or fibrous plaque consisting of dense connective tissue, lipid droplets, smooth muscle cells, macrophages & T lymphocytes. Platelets adhere and aggregate to ulcerated or fissured plaques.[6]

The `risk factor' concept evolved from the epidemiologic and prospective studies of incidence & progression of CHD in U.S.A. and Europe. The non-modifiable risk factors like age, sex, and family history and modifiable risk factors like hyperlipidemia, (and dyslipidemia), cigarette smoking, hypertension, diabetes mellitus, obesity (BMI & waist-hip ratio),sedentary habits , water softness, type A personality , stress, iron overload , coffee drinking , CMV infection of arterial wall, genetic factors have all been studied in detail but the first two have been taken up with greater emphasis for preventive modification[7]. Abnormalities of apolipoproteins esp. Apo a and E play an important role in atherogenesis and increased incidence of CHD. These apolipoproteins are embedded in the surface of lipoprotein particles and are encoded by the genes on chromosomes 1,2,6,11 and 19[8]. Framingham and other studies have clearly demonstrated higher incidence of atherosclerosis and higher CHD mortality with high serum cholesterol, LDL, VLDL and triglycerides and the beneficial effect of lowering these in prevention of coronary artery disease; while elevated HDL is inversely related to incidence of atherosclerosis and its sequelae. Lower levels of HDL are found in menopausal females, cigarette smokers, obese, diabetics and those with sedentary habits. Regular aerobic physical exercise, individualized, benefits lipid profile and apolipoproteins [9a,b] besides improving blood viscosity and angiogenesis[10]. Various lipid lowering drug therapies are beneficial and of these-HMG CoA reductase inhibitors (Lovostatin) appear most promising [11]. Reversal of experimental atherosclerosis by cholesterol lowering drugs [12], calcium channel blockers (Nifedipine)[13a], probucol [13b], and omega - 3 fatty acids have been tried[13C].

Cellular molecular biology and genetic encoding studies[14] have given a newer insight to the understanding of the fundamental abnormalities causing atherosclerosis, and the role of prostacyclin (PGI_2), c - AMP, endothelium derived relaxation factor (EDRF) [which appears to be nitric oxide (NO)] have been studied extensively. From such studies newer concepts in preventive aspect are being evovled[15].

An interesting observation was made in Norway that all the 64 patients who died of premature

* *MD., FRCP (Lond.); FRCP (Edin.); FCCP (USA);*
Professor of Medicine, Ex - Dean & Principal,
J.N. Medical College, Aligarh Muslim University; Aligarh - 202002

CHD had markedly elevated levels of amino-acid homocysteine. Since then more than 50 papers have been published confirming the findings that elevated homocysteine level appears to be another risk factor making the patient 4 1/2 times more prone to death[16a,b]. Excess of homocysteine causes aggregation of blood platelets, damage of intima, and deposition of lipids in the damaged area. Low levels of Vitamin B_6 , B_{12} and folic acid decrease the breakdown of circulating homocysteine, thereby giving another prevention strategy.

Prevention of CHD is cheaper and a better strategy could be:-

1) **Primordial prevention:** which involves prevention of development of coronary risk factors in population / person not known to have the risk factors of the disease.
2) **Primary prevention:** to prevent the development of CHD in persons having risk factors.
3) **Secondary prevention:** to prevent the progression and complications in patients of known CHD.
4) **Tertiary prevention:** to treat the complications and sequelae of CHD.

For primordial and primary prevention, WHO [17a,b,c], American Heart Association[18], British Cardiac Society [19] and European Atherosclerotic Society[20], have all made a set of recommendations which include no cigarette smoking, control of factors which can give rise to hypertension and diabetes mellitus, regular moderate physical exercise, proper diet (containing lot of green leafy vegetables, whole gram cereals, fresh fruits, dried beans & peas), maintenance of ideal body weight, and avoidance of stress (& relaxation techniques). It aims at community adoption of healthy life style, improving social and environmental conditions in which development of risk factors is minimized, if not eliminated.

People with risk factors are identified by medical check-ups, who are then prescribed individualized programmes directed at reducing or eliminating the risk factors and thus, preventing or at least delaying the development of CHD[20].

In secondary and tertiary prevention, those persons who already are affected by CHD, the programme is designed to lessen the mortality and morbidity. This includes avoidance of cigarette smoking, physical exercise, lowering of lipids by drugs (& exercise), control of hypertension and diabetes mellitus, low dose aspirin [21,22 a,b], appropriate drug regimens, useof β blockers (Metoprolol), [23] psychological rehabilitation, yoga & biofeed back and surgical interventions like angioplasty, coronary artery bypass grafting, transmyocardial revascularization etc., if required, are underaken. Prevention of coronary bypass graft occlusion is possible with the use of statin group of drugs [24], omega - 3 fatty acids [25], and anti-platelet therapy [26,27].

Although the clinical manifestations of atherosclerosis appear in fourth decade or later, the pathological process usually starts in childhood. Therefore, the preventive programme directed at risk factors should start early and continue through adulthood and later years for effective prevention of CHD.

REFERENCES:

1. Wasir, H.S., Vijay Kumar, M., Reddy, K.S., (ed)., Cardiovascular disease in India, The magnitude of problem and its changing pattern, Preventive Cardiology, An introduction, H.S. Wasir (Ed.); Vikas Publishing House Pvt. Ltd., New Delhi, 1991, 49.
2. Gupta, S.P., Malhotra, K.C., Urban and rural trends in epidemiology of coronary heart disease, J.Assoc. Phys. India, 23: 1975, 883.

3a. Mittal, M.C., The changing trend in health and coronary artery disease. J.I. Assoc. of Prev. & Soc. Medicine, March 1975.

3b. Reddy, K.S., Cardiovascular diseases in India., WHO. Stat. Q. 46, 1993, 101-7.

4a. McKeigue, P.M., Miller, G.J., Marmot, M.G., Coronary heart disease in South Asian overseas. A review, J. Clin. Epidemiol. 42, 1989, 597.

4b. Fox, K.M., Shapiro, L.M., Heart disease in Asians in Britain commoner than in Europeans, but why ?; Br. Med. J. 297, 1988, 311.

5. Bhatia, M.L. (Ed.), in Preventive Cardiology, An

introduction; H.S. Wasir (Ed.)., Vikas Publishing House Pvt. Ltd., New Delhi;,1991, 133.

6. Kaul, U., Dogra, B., Manchanda, S.C., Wasir, H.S., Rajani, M., Bhatia, M.L., Myocardial infarction in young Indian patients. Risk factors and coronary arteriographic profile, Am. Heart J. 112, 1986, 75.

7. Arntzemius, A.C., Kromhout D., Barth, J.D. et. al., Diet, lipoprotein and the progression of coronary atherosclerosis. The Leiden intervention trial., New Eng. J. Med., 312, 1985, 805.

8. Thompson, W.G., Apoprotenis, lipoprotein subfractions and the risk of coronary of artery disease., South Med. J., 86,1993, 194.

9a. Godon, D.J., Rifkind, B.M: HDL - The clinical implications of recent studies., New Eng. J. Med., 321,1990, 1311.

9b. Brown, B.G., Alberts, J.J., Fisher, L.D. et. al., Treatment study. A randomized trial demonstrating coronary artery disease regression and clinical benefits from lipid altering therapy among men with high apolipoprotein B., N. Eng. J. Med., 323,1990 1287.

10. Hespel, P., Lijmen, P., Fapard, R., Changes in plasma lipids and apoproteins associated with physical training in middle aged sedentary men: Amer. Heart. J., 115, 1988,786.

11. Dev, V., Wasir, H.S., (Ed.) in Preventive Cardiology, An introduction., H.S. Wasir (Ed.)., Vikas Publishing House Pvt. Ltd., New Delhi., 1991,229.

12. Malinow, M.R., Regression of atherosclerosis in humans. Fact or myth? Circulation, 64 (1), 1981, 1-3.

13a. Lichtlen, P.R., Hugenhoptz, P.G., Rafflenbent, W., Heike, M., Jost, T., Dieters, J.W., Retardation of angiographic presence of coronary disease by Nifedipine, Intact study Lancet., 335, 1990, 1109.

13b. Yammamoto, A., Matsuzawa, Y., Yokoyamma, S., Funahashi, T., Yamamiora, T., Kichino, B., Effects of Probucal on Xanthonia regression in familial hypercholesterolemia., Am. J. Cardio., 57,1986, 29.

13c. Davis, H.R., Bridenstine, R.T., Vesselimovich, D. et al., Fish oil inhibits development of atherosclerosis in rhesus monkeys. Artero sclerosis, 7;1987, 441 - 449.

14. Chien, K. R., Molecular advances in cardiovascular biology, Science, 260,1993, 916.

15. Nadal - Ginard, B., Mahdavi, V., General principles of cardiovascular cellular and molecular biology in heart diseases, 4th Ed; E. Braunwald (ed.); Saunders, Philadelphia, 1992.

16a. Clark, R., Hyperhomocysteinemia as an independent risk factor for CHD., N. Eng. J. Med., 324, 1991, 1149.

16b. Robinson, K., Homocysteine and coronary heart disease, Clev. Clin. J. Med., 61,1994, 438.

17a. Prevention of coronary heart disease, WHO Technical Report Series, 678, 1982

17b. Primary prevention of coronary heart disease. Euro Reports and Studies, 98, 1985

17c. Community prevention and control of cardiovascular diseases, WHO Technical Report Series, 732, 1986.

18. Grundy, S.M., Greenland, P., Herd, A., Cardiovascular and risk factor evaluation of healthy American adults. A statement for physician by an adhoc committee appointed by the steering committee of American Heart Association, Circulation, 75: 1339A, 1987.

19. The British Cardiac Society working group on coronary prevention, conclusions and recommendations., Br. Heart J., 57, 1987, 188.

20. Study group of European Atherosclerotic Society: Strategies for the prevention of coronary heart disease. A policy statement, Eur. Heart J. 8, 1987, 77.

20a. Atherosclerosis study group; Optional resources for primary prevention of atherosclerotic disease. Circulation, 70: 157A; 1984.

21. Harker, L.A.: Clinical trials evaluating platelet modifying drugs in patients with atherosclerotic cardiovascular disease and thrombosis, Circulation, 73, 1986, 206.

22a. Peto, R., Grey, R., Collins, R. et al., Randomised trial of prophylactic daily aspirin in British male doctors. Br. Med. J., 296, 1988.

22b. Willard, J.E., Lang, R. A., Hillis, L.D., The use of aspirin in ischaemic heart disease. N. Eng. J. Med. 327, 1992, 175.

23. Yusuf, S., Sleight, P., Held, P., McMohan, S.: Routine medical management of acute myocardial infarction. Lessons from overviews of recent randomized controlled trials. Circulation 82 (Suppl II): 1990, 11 - 117.

24. Blankenhorn, D.H., Nessim, S.A., Johnson, R.H., Sanmarco, M.E., Azen, S.P, Cashin - Hemphill, L., Beneficial effect of combined colestipol - niacin therapy on coronary atherosclerosis and coronary venous bypass graft, JAMA., 257,1987, 3233.
25. Komori, K., Shimokawa, H., Vanhoutte, P.N., Endothelium dependant relaxation in response to aggregating platelets in porcine femoral veins and its modulation by diet. Circulation, 80, 1989, 401.
26. Lorenz, R.L., Schacky, C.V., Weber, M, et al: Improved aortocoronay bypass patency by low dose aspirin (100mg daily), Effect on platelet aggregation and thromboxane formation, Lancet. 1, 1984, 1261.
27. Fuster, V., Chesebro, J.H., Role of platelets and platelets inhibition in aortocoronary artery - vein graft disease. Ciculation, 73, 1986, 232.

Electronic Contraceptive

The world's first electronic contraceptive will go on sale after being tested by 1500 women in England, Irish Republic & Germany. The contraceptive is said to be a suitable alternative to pills with no health risk. Its reliability falls between that of condoms (88%) and pills (92.8%). The system provides computer guided advice, displaying a green light, when it is safe to have sex. Since it is not a physical device, it has been welcomed by the Catholic Church as well. it can accurately indicate time of fertility.

SOURCE: Sunday Observer

Contributed by:
Dr. M. Athar Ansari,
Junior Resident - 3
Deptt. of Community Medicine,
J.N. Medical College,
A.M.U., Aligarh - 202002