

Coronary Risk Factors in Malaysia: Precious Little is Known, ... Still

K Yusoff, FRCP Edin, Professor of Medicine and Consultant Cardiologist, Dean, Faculty of Medicine, Universiti Kebangsaan Malaysia, Jalan Yaacob Latif, Cheras, 56000 Kuala Lumpur

Cardiovascular disease, in particular coronary artery and other atherosclerotic diseases, is the main cause of death in many developed countries. In developing countries such as Malaysia, cardiovascular diseases may not be the number one cause of deaths at the moment. However, it has been estimated that by the year 2020 there will be tremendous changes in disease patterns and demography that cardiovascular disease will be the main cause of death in these countries and will remain the main cause of death in developed countries¹. Over the past three to four decades, there has been a phenomenal progress in the understanding of this disease due to the considerable rapport between basic scientists, clinicians, clinician scientists, statisticians, epidemiologists, communities as well as governments in an effort to overcome this modern scourge of the human race. This has led to remarkable successes in not only treating the disease but also preventing it altogether or controlling its natural history². However, the battle is not yet over³ and for some countries it is perhaps just beginning⁴.

The underlying pathogenesis for coronary artery disease is undeniably the atherosclerotic plaque. This process which is initiated very early in life, perhaps as early as the second decade⁵, may manifest itself clinically as either stable angina or one of the acute coronary syndromes. Heart failure, arrhythmias and premature deaths are well known consequences. Whilst genes provide the soil, environmental factors called coronary risk factors play a pivotal role in the pathogenetic mechanisms of this disease. Numerous coronary risk

factors have been identified - some modifiable, others not. No doubt many more will be identified in the coming years as the current milieu of coronary risk factors cannot entirely account for the occurrence of coronary artery disease. Their identification in specific communities is necessary as their eradication can prevent the onslaught of the disease, or their control can reduce the consequences of the disease on individual patients. Identification of coronary risk factors cannot be a finished business. Yet in Malaysia, perhaps caught on the onslaught of the disease, too much attention is given to treating the disease (sometimes with minimal scientific basis) and too little to prevention. This is fire-fighting, with no strategic planning.

It is in this setting that the unwavering efforts by the National Heart Foundation of Malaysia through its Heart Week programmes deserve applause. These programmes serve to arouse the awareness and perhaps interest in the population on their health, in particular in relation to heart disease. Further, it has succeeded in bringing out two reports, the latest for 1995 - 1997 is as authored by Khoo et al.⁶ in this issue of the Journal. Blood pressure, body mass index, heart rate, blood glucose and cholesterol levels for 6858 Malaysians of either gender and of diverse ethnic backgrounds who attended the Heart Week programmes are reported. Although the number of subjects is large, there are a number of methodological problems inherent in such surveys. The question of whether the participants are truly representative of the Malaysian population

requires some thought. It is quite likely that those who attend the Heart Weeks were more health conscious than those who did not. Some, though interested, might not have the means nor capacity to present themselves to these programmes. The setting in which the data was collected was not standardised and only non-fasted bloods were available. Further, some communities were rather under-represented and some had very few numbers (e.g. Ibans and Kadazans). Further, the authors did not perform age- and gender-standardised comparisons when comparing data between ethnic groups. Despite these short-comings, the message is quite clear: that the prevalence and levels of adverse coronary risk factors are high and in some instances (such as diabetes and serum cholesterol levels) are getting higher. This is not just one more reminder; it serves to raise the red flag, demanding more rigorous data on trends of coronary risk factors in this country.

Familial hypercholesterolaemia is an important condition illustrating the impact of raised serum lipids on the incidence of coronary artery disease; the monozygotes tend to get coronary artery disease early with myocardial infarctions in their teens or early twenties. The underlying abnormality is a defect in the LDL receptors⁷. A number of mutations of the gene coding for the LDL receptor proteins have been identified⁸. There appears to be some differences in the mutations between certain ethnic group. Khoo et al.⁹ reported in this issue of the Journal their findings on the LDL receptor mutations in 86 patients with familial hypercholesterolaemia. The authors did not find significant differences in the pattern of mutations

between patients of various ethnic backgrounds. There was a predilection for mutations in the ligand binding domain in this cohort of patients. Interestingly in their cohort of 86 patients, none was found to have Apo B-3500 gene mutation. The effort by the authors to delineate the genetic basis of familial hypercholesterolaemia is laudable. However the clinical application of this knowledge awaits further studies.

Interest on coronary risk factors is now focussed on the younger age group. Appropriate promotion of healthy life style from this age group is expected to prevent coronary artery disease in later years. Thus the work by Chan et al.¹⁰ on blood pressure values in healthy Malaysian children reported in this issue of the Journal is timely. The data though expected is important as these are obtained from Malaysian children. It would do well if the authors continue to consolidate their data by increasing the size of the study sample as in the present report, no age group has more than 200 children.

I believe that all three articles add to our sum of knowledge on coronary risk factors in Malaysia. However, as pointed out above, more work is required to better equip us to manage the onslaught of atherosclerotic diseases in the country. Agencies responsible to promote research should not refrain from supporting studies into risk factors, from basic science, epidemiology and interventional studies. At the moment, the bottom-line is: Precious little is known about coronary risk factors in Malaysia, ..., yet.

References

1. Murray CJL and Lopez AD. Alternate projections of mortality and disability by cause 1990 - 2020: Global Burden of Disease Study. *Lancet* 1997; 349: 1498 -504.
2. Marmot MG. Interpretation of trends in coronary heart disease mortality. *Acta Med. Scand.* 1985; 58 : 701 (Suppl).
3. Braunwald E. Shattuck Lecture - Cardiovascular medicine at the turn of the millenium: Thriumph, concerns and opportunities. *New Engl J Med* 1997; 337: 1360 - 9.
4. Yusoff, K. *Kardiologi di Alaf Baru: Cabaran, Peluang dan Persiapan*. Bangi: UKM Publications, 2000.
5. McGill HC, Greer JC, Strong JP. Natural history of human atherosclerotic lesions. In: Sandler M, Bourne GH,

- Eds. *Atherosclerosis and Its Origins*. New York: Academic Press; 1963: 43 - 52.
6. Khoo KL, Tan H, Liew YM, et al. Blood pressure, body mass index, heart rate and levels of blood cholesterol and glucose of volunteers during National Heart Weeks 1995 - 1997. *Med J Mal*, 2000; 55: 439-50.
 7. Brown MS, Goldstein JL. A receptor-mediated pathway for cholesterol homeostasis. *Science* 1986; 232: 34 - 47.
 8. Hobbs HH, Brown MS, Goldstein JL. Molecular genetics of the LDL receptor gene in familial hypercholesterolaemia. *Hum Mutat* 1992; 1: 445 - 6.
 9. Khoo KL, Van Acker P, Tan H and Deslypere JP. Genetic causes of familial hypercholesterolaemia in a Malaysian population. *Med J Mal* 2000; 55: 409-18.
 10. Chan PWK, Cheong B, Lai BH, et al. Blood pressure values in healthy Malaysian children aged 6 - 12 years. *Med J Mal* 2000; 55: 493-96.