

Central obesity, hypertension and coronary artery disease: The seed and soil hypothesis

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Author contributions: Dwivedi S and Aggarwal A contributed equally to this work, including analyzing the data and designing and writing the paper.

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Received: October 11, 2010 **Revised:** January 7, 2011

Accepted: January 14, 2011

Published online: January 26, 2011

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Key words: Pedigree chart; Coronary artery disease; Seed and soil

Peer reviewer: Khalid Rahman, BSc (Hons), PhD, Professor of Physiological Biochemistry, Academic Recruitment Co-ordinator, School of Pharmacy and Biomolecular Sciences, Max Perutz Building, Byrom Street, Liverpool, L3 3AF, United Kingdom

Dwivedi S, Aggarwal A. Central obesity, hypertension and coronary artery disease: The seed and soil hypothesis. *World J Cardiol* 2011; 3(1): 40-42 Available from: URL: <http://www.wjgnet.com/1949-8462/full/v3/i1/40.htm> DOI: <http://dx.doi.org/10.4330/wjc.v3.i1.40>

Abstract

Coronary artery disease (CAD) is a multifactorial disease wherein hereditary and environmental factors play a major role. Our hypothesis is that an individual's genetic profile functions as soil while various environmental factors such as physical inactivity, smoking, stress etc. act as seeds in the etiopathogenesis of CAD. Much of the information regarding genetic and environmental factors can be determined in a pedigree chart by taking a history of the index patient, including details of major risk factors such as age, sex, smoking, hypertension, diabetes, coronary artery disease and stroke in the family. Preparing such a chart is a cost-effective way of initiating primary preventive measures in patients in a developing economy. The advantage of a detailed pedigree chart is to provide a snapshot view of the evident and underlying risk factors in the family as a whole, and not to merely study conventional risk factors. It elucidates the hidden stressors and hereditary factors responsible for cardiovascular disease in the family. We report herein an illustrative pedigree chart which exemplifies our above hypothesis.

INTRODUCTION

Coronary artery disease (CAD) is a multifactorial disease wherein hereditary and environmental factors play a major role. Our hypothesis is that an individual's genetic profile functions as soil while various environmental factors such as physical inactivity, smoking, stress etc. act as seeds in the etiopathogenesis of CAD. Much of the information regarding genetic and environmental factors can be determined in a pedigree chart by taking a history of the index patient, including details of major risk factors such as age, sex, smoking, hypertension, diabetes, coronary artery disease and stroke in the family^[1]. Preparing such a chart is a cost-effective way of initiating primary preventive measures in patients in a developing economy. We report herein an illustrative pedigree chart which exemplifies our above hypothesis.

We had the opportunity to observe and assess three generations of a family over a period of 15 years. We collected information by verbal interview^[2] of individuals and made detailed cardiovascular assessments in both parents of the index case, in 8 of his siblings and in one child of

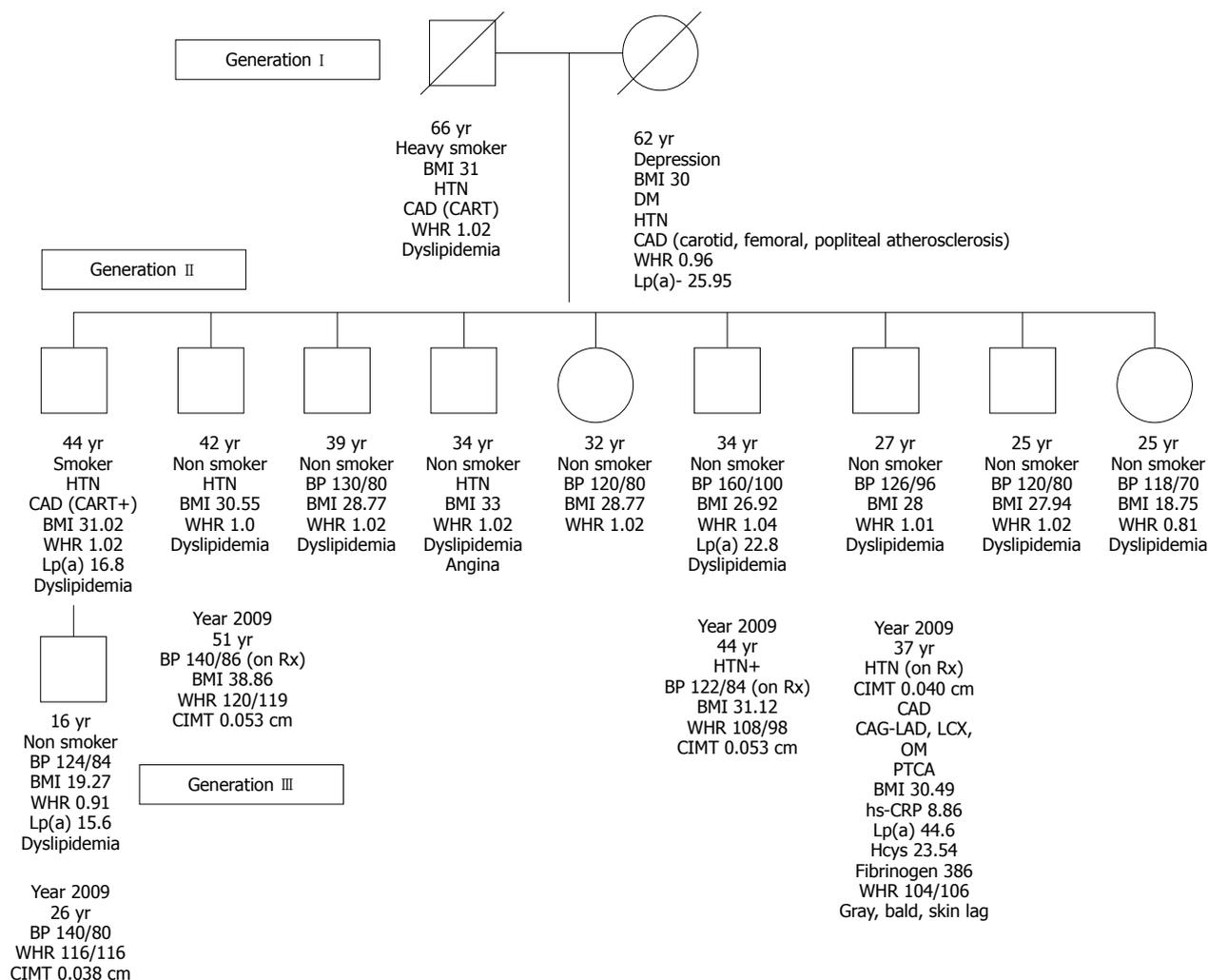


Figure 1 Pedigree chart corroborating the “seed and soil” hypothesis. Effect of environmental factors such as smoking on the eldest child, physical inactivity manifesting as central obesity in all due to a sedentary lifestyle in the second generation culminating in various sequelae of metabolic syndrome and prehypertension evolving into hypertension in the third generation. BMI: Body mass index; BP: Blood pressure; CAD: Coronary artery disease; CAG: Coronary angiography; CART: Coronary arteriography; CIMT: Carotid intima media thickness; DM: Diabetes mellitus; Dyslip: Dyslipidemia; HTN: Hypertension; Lp(a): Lipoprotein (a); TVD: Triple vessel disease; WHR: Waist hip ratio.

the index patient in the third generation. Thus a total of 12 members were examined and assessed in detail (Figure 1). Three of the siblings and the child were again assessed after 10 years. Primary preventive measures were initiated in the second generation and the effect on smoking, physical activity and diet were assessed over the years.

CASE REPORT

Initially, the 66-year-old father of the index patient presented with acute myocardial infarction. He was a chronic heavy smoker and a known hypertensive; he had manifest central obesity and dyslipidemia. A few months later, the 62-year-old mother, a known diabetic for 10 years with diabetic triopathy i.e. nephropathy, retinopathy and neuropathy, was admitted to hospital with acute thrombotic stroke and myocardial infarction. She too was obese and hypertensive. The fact that both parents suffered from CAD prematurely and had multiple cardiovascular risk

factors, led us to prepare a pedigree chart of the entire family.

It was interesting to find that even with a single high risk behavior like smoking, the eldest son of the second generation (the index patient) had developed frank CAD at a young age (44 years). This also provided us with a tool to emphasize to the entire family that they should not smoke, which fortunately was followed strictly by the siblings and progeny of the eldest son. However, the advice regarding physical activity, morning walks and a healthy diet was not followed strictly and regularly.

The son of the index patient in the third generation passed through a phase of prehypertension and, 10 years later, had developed hypertension as well as central obesity. He is thus at risk of future CAD. Another sibling of the index case had to undergo coronary revascularization at the age of 37. He had a sedentary lifestyle, central obesity and hypertension. The role of hereditary factors in this family is so obvious that we can see that children in

the second and third generations have a high prevalence of risk factors, namely central obesity, hypertension, dyslipidemia and CAD, at a much earlier age compared with the first generation parents. The explanation for the high prevalence of central obesity and hypertension in the family is that the entire family was engaged in a sedentary business with no place for any physical activity and shared a common food habit with a high fat and salt content.

Limitations of the present report are that it pertains to a single family over the years and thus the number of patients is limited.

DISCUSSION

The advantage of preparing such a detailed pedigree chart is to prepare a snapshot view of the evident and underlying risk factors in the family as a whole, and not to merely study conventional risk factors. It elucidates the hidden stressors and hereditary factors responsible for cardiovascular disease in the family. Making such a comprehen-

sive chart provides a definite basis for initiating primary preventive measures in high risk siblings and progeny of the affected patients at the earliest opportunity^[3]. We did succeed in persuading the second and third generation subjects not to smoke on the basis of this chart; however much more emphatic measures are needed to implement dietary and exercise reforms in the family. Preparing a pedigree chart of this kind is obviously a cost-effective way of detecting prospective high risk individuals thus tackling the rising trend of cardiovascular disease in a developing economy.

REFERENCES

- 1 **Dwivedi S**, Awasthy N. Familial aggregation of coronary risk. *South Asian J Prev Cardiol* 1993; **3**: 60-63
- 2 **Chandramohan D**, Maude GH, Rodrigues LC, Hayes RJ. Verbal autopsies for adult deaths: issues in their development and validation. *Int J Epidemiol* 1994; **23**: 213-222
- 3 **Dwivedi S**, Agarwal MP, Suthar CP, Dwivedi G. Migration accelerates development of metabolic syndrome--an interesting pedigree. *Indian Heart J* 2004; **56**: 258-259

S- Editor Cheng JX L- Editor Cant MR E- Editor Zheng XM