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Do parental coronary heart disease risk factors (non-modifiable) effect their young ones?

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ABSTRACT

Objective: To study the differences between the lipid profiles of the subjects whose parents are having known non-modifiable risk factors such as obesity, hypertension (HTN), myocardial infarction and diabetes, and compare them with the lipid profiles of the subjects whose parents are not having those risk factors.

Methods: A total of 402 subjects were recruited to this study. A detailed questionnaire which included information on the past medical history, height, weight, blood pressure, physical activity, smoke, alcohol, family history of coronary heart disease, HTN, diabetics and obesity. Basic demographic data and dietary habits were completed by all participants. Blood samples were obtained from all subjects after 14 h. Lipid profiles were analyzed using automated analyzer. The results were analyzed using SPSS software packages.

Results: The mean body mass index of the population was well below the cut-off value of obesity (>24.5 kg/m^2) and high risk of future cardiovascular disorder (CVD) events in this age group. The mean levels of total cholesterol (TC), triglycerides (TG) and TC/high density lipoprotein (HDL) were less than the risk levels indicative of future CVD events according to the ATP III cut-off values. However the mean HDL level in our population was slightly greater than the cut-off value while the mean low density lipoprotein level was almost similar to the risk level. Differences were observed when the subjects without history of maternal obesity were compared with subjects with history of maternal obesity. The greater percentage of subjects who are having risk levels of body mass index, TC, low density lipoprotein, TG, and TC/HDL indicated that maternal obesity contributed to the greater susceptibility of developing CVD risk in their offspring.

Conclusions: Advancing age may result in changes that could be atherogenic in the future. Such atherogenic changes have already initiated when the subjects are about 21 years old. The incidence of atherogenic changes is far greater when mothers who are having any of the risk factors such as obesity, diabetes, HTN and myocardial infarction than that fathers who are having similar risk factors.

1. Introduction

Coronary heart disease (CHD) is still the leading cause of morbidity in the general population[1]. In 2008, five out of the top ten causes for mortality worldwide were non-communicable diseases (NCDs), which have figured out to be seven out of ten by the year 2030 and majority resides in the developing countries. It is projected that, by the year 2030, about 76% of the deaths in the world would be due to NCDs[2]. India topped the world with 1.5 million cardiovascular disease (CVD) related diseases deaths in 2002.

According to the World Health Organization, CVD is the leading cause of death in India now, accounting for 29% of all deaths in 2005 with almost 100 million people affected. India is set to become the heart disease capital of the world[3]. Almost 2 000 people die of strokes every day. This alarming increase implies a future increase in the financial burden associated with this disease and raises the possibility that age-adjusted death rates from coronary artery

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disease would be increased[4].

Even the worldwide obesity levels have drastically increased over the couple of decades. As the recent World Health Organization estimates over one billion and nearly 300 million adults are overweight and obese respectively[5]. In many developed countries, the prevalence of obesity has reached epidemic levels and is associated with NCDs including diabetes, hypertension (HTN), dyslipidemia and CHD[6]. In South Asia, the major health problems were infectious diseases and under-nutrition, and very little attention was paid to obesity by healthcare workers, policy makers or researchers[7]. However, attention is being paid with the rapid emergence of the obesity epidemic in South Asian countries now.

The term risk factor includes modifiable life styles and biochemical and physiological characteristics as well as non modifiable personal characteristics such as age, gender and family history of early-onset of CVD found in healthy individuals, which are independently related to the subsequent occurrence of CHD. The effects of risk factors in adults are additive, *i.e*. the greater the number of high risk factors present, the greater the risk of CVD[8].

Autopsy studies have shown that early coronary atherosclerosis often begins in childhood and adolescence. Further, hypercholesterolaemia in adolescents correlated positively with changes in vasculature predictive of later CVD[9]. There is a tendency to persistence in ranks (tracking) for total serum and β-lipoprotein cholesterol with age[10].

Hypercholesterolemia in childhood is common in westernized countries with high rates of CHD[11]. India is a classical example to illustrate drastic changes that occur in societies during the industrialization and urbanization or the westernization[12]. Thus incidence of chronic diseases due to changes in the dietary patterns and physical activity are on the rise among Indians.

The prevalence of cardiovascular risk factors such as HTN, diabetes and obesity are very high among Indian adults in the age group of 30-65 years old[13]. Furthermore, 12.6% of hypercholesterolemia, 18.4% of body mass index (BMI) (>24 kg/m^2) and 5.8% of diabetic were reported among 975 middle aged males of 35-39 years old[14]. Therefore the effects of parental CVD risk factors on the future risk of CVD in adolescents should be investigated.

The parental obesity influences childhood obesity through a mixture of genetic and environmental mechanisms[15]. Children with obese family members are several folds more likely to be obese than children whose family members are lean, due to the reason that the former has increased preferences for high fat foods but decreased physical activity[16]. Furthermore, adolescents with extreme obesity (BMI>40 kg/m^2) have significantly heavier parents than those with Class I or II obesity (BMI<40 kg/m^2)[17].

A substantial amount of weight gain occurs in the transition from adolescence to young adulthood with strong tracking from adolescence into adulthood[18-21]. It is also suggested that adults who become obese in childhood and remained obese into adulthood are at higher metabolic risk than those with adult-onset obesity[22]. It has been found that maternal obesity was the most significant predictor of obesity during childhood[23]. Thus the combination of having an obese mother and an earlier onset of obesity translates into higher BMI and weight at young adulthood, especially in black children as compared with white children[22,23].

Furthermore, adolescent obesity is associated with increased mortality and morbidity related to a variety of chronic diseases later in life, and reversal of obesity is associated with decreased metabolic risk in adulthood[24,25].

According to the fetal insulin hypothesis, non-insulin-dependent diabetes in parents is associated with lower birth weight among their offsprings[26].

Data on increased risk of non-insulin dependent diabetes for the fathers of children with low birth weight confirm that diabetes in fathers and the birth weight of their offsprings are strongly associated[27]. On the other hand, diabetes in the mother increase the birth weight of offspring, which is likely to reflect immediate effects of the mother's metabolic control, possibly masking genetic effects operating in the opposite direction[24,28].

There are very few studies on the effects of history of parental HTN on young adults. In such a study of 315 black and white students, on the effects of parental HTN on children's BMI and cardiovascular reactivity over time[29], it was concluded that parental HTN independently predicted children's BMI, BMI z score, resting blood pressure, and blood pressure reactivity.

Family history of premature CHD is an independent risk factor for cardiovascular events in the offspring due to an increased susceptibility to atherosclerosis, and an increased tendency for thrombosis or other factors[30].

It is important to elucidate the mechanism on offspring of parents with premature CHD which differs from offspring of parents without premature CHD because it would be beneficial to asymptomatic adults with a positive family history in determining the need for primary preventive therapies[31].

Increased intima-media thickness, which is a heritable trait, is associated with prevalent CHD, peripheral vascular disease and incidents of cardiovascular events[30]. It was reported that CHD family risk score was correlated with mean increased intima-media thickness in whites, but not African-Americans.

In the Framingham offspring study of 1 662 subjects who underwent carotid ultrasonography, it has been found that subclinical atherosclerosis, assessed in the carotid arteries, is more prevalent in individuals with a family history of CHD. Early-onset parental CHD, in particular, identifies offspring with a strong familial predisposition to atherosclerosis. This confirms the concept that premature age of parental disease identifies a subgroup with a strong familial predisposition to vascular disease[30].

It is evident that the intervention to prevent CVD in adulthood may be difficult, particularly in the middle age, of being too late in terms of the atherosclerotic process. Therefore, early intervention, perhaps starting in childhood, may be more appropriate. It is only in the 1980s that adult epidemiological risk factor models have been extended to children.