A STUDY ON RELATIONSHIP OF STATURE TO CORONARY ARTERY DISEASE RISK FACTORS IN CASES AND CONTROLS

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ABSTRACT

Background: Several studies have observed an inverse association between height and risk for coronary disease, although the reasons for the association remain unclear. Data on the relation between stature and coronary artery disease (CAD) is more limited.

Objective: To elucidate the relationship of simple anthropometric index (height) with coronary artery disease

Materials and Methods: Two hundred subjects (100 cases and 100 controls) aged between 20-60 years were included in this study. Height was measured using a graduated tape attached to the wall. Subjects who have been diagnosed to have CAD in any form were selected in the study as cases (Group I). To diagnose CAD, a supportive investigation like lipid profile was taken. Subjects who have attended general outpatient department but without proven CAD were selected in the study as controls (Group II).

Results: Age distribution was non-significantly more in Group I when compared to Group II with $p=0.431$. Males were significantly more in Group I when compared to Group II with $p = 0.048$. Height distribution was significantly less in Group I when compared to Group II with $p=0.001$. Abnormal lipid profile was significantly more in Group I compared to Group II with $p = <0.001$.

Conclusion: Anthropometric variable height in both genders was significantly affected in subjects with coronary risk factors. The relationship between short stature and CAD appears to be more prominent.

KEY WORDS: Height, coronary artery disease, short stature.

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INTRODUCTION

For the past several decades stature has been thought to be predictor for many lethal diseases of which coronary artery disease (CAD) also one [1]. In past several studies it was found that stature has an inverse association with CAD [2-4]. It is one of the leading cause of death and disability universally and one of the most common chronic illness in the developing world. It has absorbed large share of time and wealth

on research all over the world [2]. There are many modifiable and non-modifiable risk factors associated with CAD which may be generalized or systemic [5]. Behavioral risk factors such as smoking, lack of exercise, high fat containing diet, stress, hyperlipidemic status, alcohol intake are well known modifiable risk factors, whereas age, male sex and positive family history are non-modifiable risk factors of cardiovascular disease. A combination

of risk factors has been shown to increase the risk of occurrence of these diseases [6].

Simple anthropometric index like height plays an important role in clinical practice in predicting the development of CAD risk factors. Despite the modern techniques, anthropometric measurements are traditionally important methods to study the genetic structure and prediction of risk factors of many complex diseases in human health. Both height and weight are influenced by genetic, environmental and socio economic factors and are associated with other coronary risk factors [7]. Dates back to 19th century an inverse relationship has been demonstrated between height and incidence of CAD, by both case and controlled and cohort studies.

In the ageing population of industrialized nations, increasing burden of CAD has an enormous impact on population health, the health care system and the economy. The need for a better understanding of how to achieve "healthy ageing", how to slow down the process of cardiovascular disease generation and progression and how to improve preventive and therapeutic strategies is obvious in society with a steady rising life expectancy. Therefore the present study was done to elucidate the relationship of simple anthropometric index (height) with coronary artery disease; it may help to facilitate enhanced screening for disease risk.

MATERIALS AND METHODS

A case control study was done from January to December 2012 involving 100 cases and 100 controls of both gender between the age of 20 and 60 years. The study population was selected from Basaweshwar teaching and general hospital attached to HKE's M.R. Medical College Kalburgi, Karnataka, India. The study was approved by Institutional Ethics Committee of HKE's M.R. Medical College Kalburgi, Karnataka. Detailed and signed informed consent was obtained from the subjects in the presence of witness, after oral reading of the protocol in verbatim and explaining it in the subject's own language.

Cases (Group I): One hundred patients between 20 and 60 years who have been diagnosed to have coronary artery disease (CAD) in any form were selected in the study. To diagnose CAD,

electrocardiography of patients along with supportive investigations like lipid profile was taken.

Estimation of serum lipid profile: Serum was prepared with 2-3mL of rat blood collected in centrifuge tube and kept for centrifugation (2500rpm x 30min) to estimate the serum cholesterol (TC), serum triglycerides (TG) and serum high density lipoprotein cholesterol (HDL-C) with a semi-automated biochemical analyzer (ERBA Star 21 plus) by using a commercial kit (Erba Diagnostics, Mannheim, GmbH, Germany) according to manufacturer's protocol.]

Serum LDL-C level was calculated by using the following Friedewald's equation

$$\text{LDL-Cholesterol} = \text{Total Cholesterol} - \frac{\text{Triglyceride}}{5} - \text{HDL-Cholesterol}$$

very low density lipoprotein was determined by using the following equation:

$$\text{VLDL} = \text{TC} - (\text{HDL} + \text{LDL})$$

Controls (Group II): One hundred subjects between 20 and 60 years who attended general outpatient department but without proven CAD were selected in the study.

Inclusion Criteria: Patients with coronary artery disease of age group of 20 and 60 years were selected for the study as cases. Subjects between 20 to 60 years male or female attending general outpatient department for any other complaint were selected for study as controls. At the onset, the study protocol was briefed and those who came forward voluntarily to participate were included.

Exclusion Criteria: Subjects less than 20 years and more than 60 years were not included in the study. Patients who are proved not to have CAD either clinically or by any diagnostic aid are not included as cases.

Measurement of Height: Height was measured barefoot with head in horizontal plane to the nearest 0.1cm using graduated tape attached to the wall (in centimeters).

Statistical Analysis: Descriptive and inferential statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean $\pm$ SD and results on categorical measurements were presented

in number (%). Significance was assessed at 5% level of significance. The following assumptions on data were made, Assumption 1: Dependant variables should be normally distributed, Assumption 2: Samples drawn from the population should be random, cases of the samples should be independent. Student t test (two tailed, independent) has been used to find the significance of study parameters. Fisher exact test has been used to find the homogeneity of samples on categorical scale.

RESULTS

Mean age in cases was 50.16 ± 6.39 years and mean age in controls was 49.75 ± 6.87 years. Age distribution was non-significantly more in Group I when compared to Group II with $p=0.431$ (Tab. 1).

Male subjects in Group I (cases) was 70 and in Group II (controls) was 56 in numbers. Males were significantly more in Group I when compared to Group II with $p = 0.048$ (Figure 1). These results depicts males were more significantly affected.

Mean height in Group I (cases) was 155.01 ± 7.70 cm and mean height in Group II (controls) was 165.38 ± 9.80 cm. Height distribution was significantly less in Group I when compared to Group II with $p=0.001$ (Table 2). Our results showed height had inverse association with CAD.

Abnormal lipid profile in Group I (cases) was 70 and in Group II (controls) was 41 in numbers. Abnormal lipid profile was significantly more in Group I compared to Group II with $p = <0.001$ (Figure 2). This study showed that lipid profile is a strong predictor of CAD.

Table 1: Relationship between Age Distributions among Study Groups.

Age (in years)	Group I		Group II	
	No	%	No	%
20-30	0	0	1	1
31-40	9	9	10	10
41-50	52	52	50	50
51-60	38	38	39	39
Total	100	100	100	100
Mean $\pm$ SD	50.16 ± 6.39		49.75 ± 6.87^{NS}	

No: number of subjects; %: percentage; SD: Standard deviation. **Group I:** coronary artery disease cases; **Group II:** subjects without CAD (controls).

Age distribution non-significantly (NS) differ from respective group ($p<0.05$) (Paired t- test done)

Table 2: Relationship between Height Distributions among Study Groups.

Height (in centimeters)	Group I		Group II	
	No	%	No	%
<150	45	45	37	37
151-160	30	30	17	17
161-170	22	22	36	36
>171	3	3	10	10
Total	100	100	100	100
Mean $\pm$ SD	155.01 ± 7.70		$165.38 \pm 9.80^*$	

No: number of subjects; %: percentage; SD: Standard deviation. **Group I:** coronary artery disease cases; **Group II:** subjects without CAD (controls). Height distribution significantly differ (*) from respective group ($p<0.05$) (Paired t- test done).

Fig. 1: Relationship between Gender Distributions among Study Groups.

Group I: coronary artery disease cases; **Group II:** subjects without CAD (controls). Gender distribution significantly differ (*) from respective group ($p<0.05$) (Fisher exact test done).

Fig. 2: Relationship between Lipid Profile Distributions among Study Groups.

Group I: coronary artery disease cases; **Group II:** subjects without CAD (controls). Lipid profile distribution significantly differ from respective group ($p<0.05$) (Fisher exact test done).

DISCUSSION

For several decades there has been considerable interest in identifying the precursors for coronary heart disease. In addition to the traditional risk factors, several studies have examined the contribution of body habits as a risk factor for cardiovascular morbidity and mortality. An inverse linear association between height and development of coronary heart disease has been seen in many previous studies [2-4]. Today with better prevention and treatment of these risk factors height may emerge as a significant predictor of coronary disease. Due to heterogeneity of studies we cannot reliably answer the question on the critical absolute height not only differed between the articles but also between men and women and between ethnic groups. That is why in this study shortest verses tallest group setting has been used. In the present study shortest height in the cases was 147 cm and tallest was 175 cm. Out of 100 cases 45% were below 150 cm, 30% between 151-160 cm, 22% between 161-170 cm and 3% above 171 cm indicating shorter individuals are at increased risk.

Case-control studies, comparing patients with myocardial infarction with control subjects in the 1950s and 1960s noted an association of short stature in men with risk for coronary heart disease. Consistent with previous studies, inverse association with height was apparent for coronary heart disease but predominantly in males in the present study mean height was 155.01 ± 7.70 cm in cases and 165.38 ± 9.80 cm in controls. In contrast to the present study where males were predominantly affected, Framingham heart studies found no association between height and risk of myocardial infarction in men but only in women [2]. Similar to Stockholm female coronary risk study where short stature was associated with poor prognosis in women with documented coronary artery disease.

Wamala SP et al [8] found that short stature was a strong predictor of increased incidence of myocardial infarction in both sexes which was similar to the meta analysis done by Paajanen TA et al [7] who conducted that adults of both genders within shortest category had an ~50%

higher risk of coronary heart disease mortality than tall individuals. The study done by crystal Man Ying Lee [9] and Dona Parker [10] men had inverse linear association with coronary heart disease but in contrast Altan Onat et al [11] formed Turkish women to be at increased risk compared to men.

In mechanism for association between height and coronary heart disease are not well known. A proposed mechanism is that early childhood socioeconomic factors and family size may influence both pre and post-natal nutritional status, which may in turn alter the susceptibility to atherosclerosis early in life and simultaneously determine adult height [12]. According to Barker's theory, poor nutrition shows down cell division which is vital for fetal growth, thus programming the hormonal secretion, metabolic activity and organ structure (e.g. adult height).

An additional explanation is that adult height is correlated with arterial vessel caliber for short stature has been associated with narrow lumen may alter the haemodynamics within coronary vasculature leading to local adverse conditions predisposing to more severe atherosclerosis. Furthermore short stature has been associated with a lower forced expiratory volume in 1 sec (FEV-1). A reduced FEV-1 has been shown to be an early indicator of myocardial failure [8]. In the Coronary Artery Surgery Study (CASS), surgical mortality was inversely related to the average diameter of the grafted coronary arteries in both men and women [13]. It was therefore hypothesized that the physical size of the patient, including coronary artery diameter, may predict operative mortality.¹³ In recent studies using angiographic measurements, the coronary artery diameter was correlated with height and body weight [14,15]. It could be hypothesized that smaller coronary arteries may be occluded earlier in life under similar risk conditions.

CONCLUSION

The relationship between short stature and coronary artery disease indicates strong predictor in onset of disease. The possible mechanisms are complex and involve interplay between genetic factors, early childhood development, coronary arterial diameter, socioeconomic and environmental factors.

Conflicts of Interests: None

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