An Epidemiological Study of Cardiovascular Disease Risk Factors

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ABSTRACT Coronary heart disease (CHD) is the largest killer in the developed countries and is rapidly assuming a similar trend in developing countries. The prevalence of this disease and its associated risk factors are more in urban setting than in rural areas. To determine the urban-rural differences of various risk factors, a population based study was conducted among males of Reddis, an endogamous population of Andhra Pradesh. A total of 110 and 102 males were studies from urban and rural areas, respectively. The prevalence of risk factors (urban vs rural) were: obesity (38% vs 21%), truncal obesity (38% vs 15%), hypertension (23% vs 16%), hypercholesterolemia (14% vs 10%), low levels of HDL cholesterol (16% vs 10%), smoking (27% vs 33%) and low physical activity (49% vs 25%). The comparison of $\chi^2$-values showed that the prevalence of obesity, truncal obesity and sedentary lifestyle were significantly greater in urban Reddis but no significant difference was found for hypertension, hypercholesterolemia and low HDL cholesterol. Though the risk factors were greater in urban Reddis, they are showing a rising trend among rural Reddis also. Based on the results of the present study, not only the urban Reddis but the rural ones also are showing a rising trend in developing coronary heart disease risk factors. This might be due to the role of environmental factors besides genetic constitution of the Reddy population.

INTRODUCTION

Cardiovascular disease is a major public health problem in both developed and in developing countries like India. Cardiovascular disease is now on the increase in India possibly due to the changing lifestyle and is causing a grave concern. It has been predicted that cardiovascular diseases will be the most important cause of mortality in India by the year 2015 (Enas et al., 1992; Reddy, 1993). The WHO (1959) Expert committee on cardiovascular disease and hypertension recommended epidemiological surveys in as many countries as possible to analyse the risk factors and to establish the prevalence of the disease in different countries. During the past three decades, a few epidemiological studies have been carried out in India for evaluating the prevalence of CHD and its risk factors. Most of them are hospital based and on select population groups.

A major cause of cardiovascular diseases is atherosclerosis or arteriosclerosis, meaning sclerotic thickening of the arterial wall (Rao and White, 1993; Gopinath et al., 1994). Various risk factors are responsible for this disease. Major risk factors are smoking, hypertension, hypercholesterolemia, diabetes and obesity (Enas et al., 1992). Other factors which are considered important are truncal obesity, insulin resistance, hypertriglyceridemia, hyperfibrinogenemia and mental stress. Lifestyle risk factors such as the type of dietary fat, lack of education, lack of social and family support and lack of mental and physical relaxing activities may also be important (Bhatia, 1991; Enas et al., 1992; Reddy, 1993; Gupta et al., 1994). Studies carried out in India indicated urban-rural differences in disease prevalence and the risk factors.

The available data in India on urban-rural differences shows that the prevalence is more in urban setting and is associated with greater prevalence of risk factors (hypertension, obesity, diabetes, truncal obesity, higher total cholesterol and LDL cholesterol levels) than the rural counterparts. The results of the studies conducted on urban-rural differences are available from Delhi and its surrounding rural areas (Chadha, 1998), Jaipur and its surrounding rural areas (Gupta and Gupta, 1998) and in Tiruvananthapuram (Begom and Singh, 1995) and its surrounding rural areas (Ramankutti et al., 1993), revealed that urban population is having more risk. Studies, particularly, community based are lacking in Indian subcontinent. Vasan and Kartha (1998) have emphasised the need and importance of community based studies on
CHD and its risk factors in Indian context. Hence, we have undertaken an epidemiological study among ‘Reddis’, an endogamous population of Andhra Pradesh. The Reddis are one of the land owning caste groups of Andhra Pradesh and sound in economy. The practice of consanguinity is more prevalent among this population (Surendranatha Reddy, 1993). Now-a-days most of the Reddis are living in urban areas due to their business/employment. Epidemiological studies have indicated both genetic and environment play an important role in causing the CVD. So, we have taken up a study to examine whether any perceived differences would occur between urban and rural populations within the endogamous populations. Our objective was to understand the magnitude of urban-rural differences in risk factors of CHD among Reddis.

**MATERIAL AND METHODS**

The data were collected on adult males (20-60 years) from an endogamous population known as ‘Reddis’ from Chittoor district of Andhra Pradesh. A simple random house-to-house survey was employed in selecting the subjects. Only co-operative individuals were included in the study. A total of 110 and 102 males, respectively from urban and rural areas were interviewed and the data were collected. Strict precautions were taken to avoid related individuals. The urban sample was drawn from Tirupati town and the rural sample in a cluster of three villages 100 km away from Tirupati town.

The data on anthropometry includes-height (cm), weight (kg) and abdominal circumference (cm). Height was measured with a standard anthropometer rod and weight was measured with weighing machine. By using standard nonelastic tape abdominal circumference was measured at the umbilicus. Standard landmarks and methodology were followed in recording the anthropometric measurements (Lohman et al., 1988). The body mass index (BMI) was calculated to estimate overall obesity by dividing body-weight in kilograms by height in metres squared (kg/m²). Conicity index (CI), the most sensitive index to determine abdominal obesity was used (Valdez., 1993). The CI was calculated by using the formula CI=abdominal girth

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(0.109 \ W/H) \text{, where abdominal girth in meters, weight (W) in kgs, and height (H) in meters. Obesity was identified when the body mass index was } \geq 25 \text{ kg/m}^2. \text{ No standard values are available for conicity index to identify abdominal obesity. The 75th percentile value of CI in our study population was 1.33. Hence this value was taken as cut-off point to determine the abdominal obesity in the present population.}
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For each individual the data on age, smoking habit and physical activity were also collected. The smoking habit has been categorised into smokers and non-smokers. Current smokers, past smokers and users of all forms of tobacco were pooled together as smokers. Physical activity was classified into three levels. Low/Sedentary activity refers to people involved in office work, research, teaching, business and land owners; medium activity was attributed to dual jobs and land owners who involve in agriculture work; and high activity includes farmers actively involved in the field of agriculture labours.

Systolic and diastolic blood pressures were measured by recording the appearance and disappearance of Korotkoff’s sounds to the nearest 2 mmHg on a seated subject. Subjects were identified as hypertensive based on the United States Fifth Joint National Committee recommendations (1993) as blood pressure of or more than 140 mmHg systolic or 90 mmHg diastolic. Also we have classified the population as hypertensive based on WHO (1962) classification as SBP ≥160 mmHg and/or DBP ≥95 mmHg.

A sample of about 5 ml of fasting blood was collected into vacutainers containing EDTA. Plasma was separated on centrifugation of the blood samples at 2500 rpm for 20 minutes. Total cholesterol and HDL cholesterol were estimated using standard protocols (Zlatkis et al., 1953 and Burstein et al., 1970, respectively). Hypercholesterolemia and low high density cholesterol levels were determined according to definitions of United States National Cholesterol Education Programme (1994). Based on this criteria, high serum total cholesterol was taken as ≥200 mg/dl and low HDL cholesterol was taken as <35 mg/dl.

**Statistical Analysis:** The data were analysed using SPSS (7.5v) package. The difference
between urban and rural populations was tested by t-test and χ² test. Two tailed P values less than 0.05 were considered significant.

**RESULTS**

Table 1 shows the descriptive statistics for anthropometry, blood pressure, lipid and lipoprotein levels. The only statistically insignificant difference between urban and rural was found in DBP but in remaining all variables significantly higher mean values were observed in urban compared to rural ones. The difference in prevalence of various risk factors between urban and rural Reddis were presented in table 2. The results showed that the prevalence of obesity, truncal obesity, hypertension, hypercholesterolemia and sedentary life style were more in urban men than in the rural ones. But the smoking habit was more in rural Reddis. The comparison of χ²-values showed that the prevalence of obesity, truncal obesity and sedentary lifestyle were significantly greater in urban Reddis but no significant difference was found for hypertension, hypercholesterolemia and low HDL cholesterol.

**DISCUSSION**

The prevalence of cardiovascular disease risk factors viz., obesity, abdominal obesity, hypertension, hypercholesterolemia were higher among urban than the rural population in the present study. However, smoking was higher in rural ones. Most of the urban populations leading a sedentary lifestyle compared to rural ones. The differences in lifestyle may be the cause for differences in obesity, abdominal obesity, hypertension, hypercholesterolemia. Epidemiological studies conducted in 1990 in Agra, Delhi and Chandigarh showed that greater prevalence of coronary heart disease and risk factors in urban subjects (Gupta and Gupta, 1996). The recent studies conducted in Delhi (Chadha, 1998) and Jaipur (Gupta and Gupta, 1998) also confirmed the same trend. In a population based study among Marwaris of Calcutta (Majumdar et al., 1994), the Marwaris had 17% of hypertensives (WHO, 1962), the mean of SBP, DBP, TC and HDL were 125.30 mmHg, 82.28, 176. 86 mg/dl and 41.06 mg/dl, respectively. The results of our study though shown similar findings when compared to the previous studies, no significant differences was observed for hypertension and smoking habit between urban and rural populations (Table 2).

Other recent studies have also shown a high prevalence of CHD and risk factors in both urban and rural populations of India. South India shows still higher rates of the disease than in other parts of the country (Begam and Singh, 1995). In urban areas, the disease is as common as in the developed countries and studies show a rising trend (Sarvatham and Berry, 1968; Gupta and Malhotra, 1975; Chadha, et al., 1990). Data from rural areas have shown a lower prevalence

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**Table 1: Mean ± S.D. values for different variables among the Reddis**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Urban (n=110)</th>
<th>Rural (n=102)</th>
<th>P-values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>47.36 ± 9.14</td>
<td>40.76 ± 14.22</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Height</td>
<td>168.26 ± 6.48</td>
<td>166.20 ± 6.41</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Weight</td>
<td>68.39 ± 11.18</td>
<td>59.87 ± 11.84</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Abdominal circumference</td>
<td>89.86 ± 10.04</td>
<td>79.86 ± 11.05</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Body mass</td>
<td>24.13 ± 3.78</td>
<td>21.62 ± 3.57</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Conicity index</td>
<td>1.30 ± 0.073</td>
<td>1.22 ± 0.087</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>128.63 ± 18.72</td>
<td>120.55 ± 14.80</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Diastolic blood pressure</td>
<td>86.77 ± 11.30</td>
<td>84.75 ± 9.94</td>
<td>n.s.</td>
</tr>
<tr>
<td>Total pressure</td>
<td>176.68 ± 45.80</td>
<td>162.97 ± 47.51</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>42.16 ± 8.68</td>
<td>46.48 ± 8.96</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

**Table 2: Differences of cardiovascular disease risk factors in urban-rural Reddis**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Urban (n=110)</th>
<th>Rural (n=102)</th>
<th>P-values</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI ≥ 25</td>
<td>42 (38.18)</td>
<td>22 (21.57)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Abdominal obesity CI ≥ 1.33</td>
<td>42 (38.18)</td>
<td>16 (15.69)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Hypertension</td>
<td>50 (45.45)</td>
<td>34 (33.33)</td>
<td>n.s.</td>
</tr>
<tr>
<td>≥ 140/90</td>
<td>26 (23.64)</td>
<td>16 (15.69)</td>
<td>n.s.</td>
</tr>
<tr>
<td>≥ 160/95</td>
<td>16 (14.55)</td>
<td>10 (9.80)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Total cholesterol ≥ 200</td>
<td>18 (16.36)</td>
<td>10 (9.80)</td>
<td>n.s.</td>
</tr>
<tr>
<td>HDL cholesterol &lt; 35</td>
<td>30 (27.27)</td>
<td>34 (33.33)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Smoking</td>
<td>54 (49.09)</td>
<td>24 (23.53)</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

**Note:** Figures in parentheses indicate percentages
as compared to cities but still rising trend is seen there as well (Ramankutty et al., 1993; Gupta et al., 1994). Based on the results of the present study, it is presumed that in parallel to urban Reddis, the risk factors for CHD are showing an increasing trend in rural Reddis also. This might be due to the role of environmental factors in addition to the genetic composition of the population.

Populations/endogamous groups showed differences/affinities with respect to anthropometry, dermatoglyphics, serology, protein polymorphism, DNA polymorphism etc. So, population differences may also be observed in obesity, hypertension, diabetes mellitus, smoking habit, physical activity and lipid and lipoprotein levels. Hence, in an anthropological perspective it is indeed necessary to carryout Genetic epidemiological studies to study the prevalence of various coronary disease risk factors among different endogamous populations of India to explore the interaction of genetic and environmental factors in causation of cardiovascular disease.

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REFERENCES


