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Coronary risk factors in South Asians: A prevalence study in an urban populace of Eastern India

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Abstract
Aim: This study examined the prevalence of coronary risk factors and significant predictors of coronary artery disease (CAD) in one of the poorest states of Eastern India among a unique ethnic urban population that is experiencing changing lifestyle patterns.
Methods: A multi-stage probability sampling from a sampling frame of 37 electoral wards geographically representative of the urban population of Berhampur, with a population of 307,724 in 2001, was based on an estimated sample of 1200 with adequate power. One thousand one hundred and seventy eight subjects (590 males; 588 females) ⩾20 years of age were finally selected. In addition to socio-demographic characteristics, physiological, behavioral, anthropometric and biochemical parameters were ascertained using interviewer-completed questionnaires and appropriate clinical examinations. Both descriptive and multivariable logistic regression analyses were performed.
Results: The overall prevalence of CAD was 10%. The main coronary risk factor prevalence rates were: hypertension (37%); smoking (27%); hypercholesterolemia (23%); diabetes (16%); central obesity (49%); physical inactivity levels (34%); and 47% had low HDL levels. Overall, age, central obesity, hypertension (adjusted odds ratio: 2.2; 95% confidence interval: 1.4; 3.4), physical inactivity levels and diabetes in females alone were significant predictors of CAD.
Conclusions: A high CAD prevalence of 10%, with higher rates of some classical cardiovascular risk factors such as diabetes, hypertension and physical inactivity levels, reinforce the need for a comprehensive CAD prevention and control program. This is the first study conducted in one of the poorest states within the fold of an emerging economy, clearly suggesting the ubiquitous nature of the CAD epidemic.
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1. Introduction

Cardiovascular disorders are among the most significant causes of disease, disability and premature death in South Asians facing insurmountable challenges [1—4]. South Asians that include Bangladeshis, Indians, Nepalese, Pakistani and Sri Lankans have the highest incidence of Coronary Artery Disease (CAD) among all the ethnic groups, irrespective of their religious affiliations, life style, diet or the country of residence [5,6]. Cardiovascular diseases (CVD) are predicted to increase rapidly in India and will comprise half the global CVD burden over the next 15 years [7]. It is also well established that this population experiences CAD in a younger age [8].

INTERHEART [9] study indicated that conventional CVD risk factors are associated with South Asians, but evidence elsewhere suggests that these risk factors do not fully account for the excess incidence of CAD in South Asians [10—12]. It is possible that distinctive mechanistic pathways in South Asians contribute to ethnic susceptibility and risk multiplication by both conventional and non-conventional cardiovascular risk factors [5,6].

Furthermore, a reversal of socio-economic gradient in cardiovascular risk factors is observed in South Asians. [4,13]. Significant differences exist for anthropometric, metabolic, and blood pressure variables between rural and urban areas [14—16]. Such differences may be due to diet, body weight, physical activity, diverse life styles and social structure [17] which necessitates studies from multiple regions of the countries in developing national strategies for CVD prevention. Primary prevention based on population-based risk reduction programmes is the most cost-effective method to control the rising epidemic of CVD [18].

Moreover 80% of the global burden of CVD occurs in low-income and middle-income countries, but knowledge of the importance of risk factors is largely derived from studies of Caucasians of European origin [19]. Therefore, the effect of such factors on risk of CAD in most regions is unknown. Importantly, there is no evidence on CAD and coronary risk factors in areas with increased poverty within the South Asian population groups. Because of the uniqueness of the above two factors- poverty and South Asians' increased susceptibility to coronary risk factors, we undertook this study to determine the prevalence of CAD and coronary risk factor profile in a representative sample of an urban populace of Orissa, one of the poorest states in Eastern India.

2. Methods

2.1. Study design and setting

A cross-sectional study was designed in Berhampur Urban population, the study site, with an estimated population of 307,724 in 2001.

2.2. Sample size calculation

Based on an estimated prevalence of 25% hypertensive patients [20], the required sample size was approximately 1200 persons as quantified from the following formula of Sample size = $4pq/l^2$, where 'q' is equal to 100-p and 'l' is an allowable error [21] i.e. 10% of factor 'p' and 'p' is the prevalence of hypertension, namely, 25%.

2.3. Sampling design

The study population was selected using a multi-stage random sampling technique. The sampling frame constituted 37 electoral wards spread across the urban population of Berhampur. Thirty wards were selected randomly to identify the sampling unit, a household. Each ward of the town is divided into 12—14 streets and each street is spread in two rows of households. Two rows of households were randomly selected and the sampling unit household was selected by simple random sampling to enroll approximately 40 who are $\geqslant$20 years of age from each ward. A total of 1178 subjects who are $\geqslant$20 years of age were finally recruited for this study. Socio-economic details of the population in these wards were available from the Voters' Lists.

2.4. Survey methods

The survey methodology adopted was the step-wise approach of the World Health Organization [22,23], namely, questionnaire based survey for behavioral risk factors, anthropometric measurements and biochemical measurements and ECG examination [22,23]. The local community leaders and the local Lady Health Visitors (Anganwadi workers) were involved in the participatory phase of the survey to increase compliance. A piloted questionnaire in English back translated into the regional language was finally administered with the help of six previously trained interviewers.

2.5. Ethical approval

Institutional ethical committee approval was obtained from the Kalinga Institute of Medical Sciences, Bhubaneshwar, prior to the start of study and informed consent was obtained from all the study subjects [24].

2.6. Questionnaire

In addition to demographic, socio-economic, and self-reported behavioral information (smoking, alcohol, physical activity, diet), objective measures of anthropometry (height, weight, waist and hip circumferences), biochemical (plasma glucose, total cholesterol, triglycerides, HDL cholesterol levels) and electrocardiographic readings were also collected.

Health conditions were documented based on a self-reported history of diabetes, hypertension and cardiovascular disease (chest pain, heart attack or stroke). *Family history* for all of the above conditions was also collected. Details pertaining to *psychosocial aspects* such as depression, stress and hopelessness were obtained. *Physical activity* was assessed by using a standard questionnaire, which was validated earlier in population studies from both South and North India [25,26]. Details on anthropometric measurements and the standard definitions of the several coronary risk factors studied are presented in Appendix A and B, respectively.

2.7. Statistical analysis

Statistical analyses were performed using SPSS windows version 11.0 software (SPSS Inc., Chicago, Illinois). All continuous variables were reported as Means ± Standard deviation. Significant predictors of CAD were also estimated applying backward elimination of multivariate logistic regression technique using SAS statistical package (version 9.1), SAS, Cary, North Carolina (US).

3. Results

A total of 1178 subjects out of 1200 invited subjects participated in this study. The response rate was 98.16%, with 50% males ($n = 590$). A majority of the respondents were between 31 and 50 years of age (25%), college-educated (38%), and three-fourths from middle-class family background. Although 13% ($n = 152$) had a family history of CAD, only 3.5% ($n = 41$) were on treatment for CAD. The total CAD prevalence in the study population was 10.0% (118/1178) based on a definite medical history, ''Q'' wave changes as well as ST-T changes. (Table 1).

Table 1 shows the sex distribution of the coronary risk factors studied. In general males have a higher prevalence of CAD risk factors but females have a significantly higher prevalence of both general and central obesity ($p < 0.05$). The lipid profiles are worse in females when compared to males (a significantly lower HDL; higher LDL and higher total cholesterol levels in females) (Table 1). Almost 37% ($n = 431$) of the respondents were hypertensive and 11.5% ($n = 136$) were known diabetics, 32% ($n = 379$) and 24% ($n = 283$) reported a family history of hypertension and diabetes, respectively. There were 10.4% alcoholics, 61.2% had low or no fruit intake, but 67% were nonvegetarians (Table 1).

Table 2 shows that all the mean values of CAD risk factors are within the normal range. Table 3 shows the age-distribution of the coronary risk factors studied. In general, there is a significant positive age-gradient of coronary risk factors. However, young adults (20—30 years of age) have higher rates than the average of low/no fruit intake, higher rates of high LDL and a high rate of low HDL (not statistically significant age-gradient). There are some gender differences by age groups. CAD prevalence peaked in those above 70 years of age in females alone. Obesity (both central and general) and low HDL levels in general are higher across all age groups in females. Although smoking levels are lower in females across all age-groups, the levels peaked in the age-group 51—60 years unlike in males.

Table 4 shows the univariate analyses of risk factors: past histories of hypertension and diabetes were significant individual co-variates. However, on multivariable backward elimination logistic regression analyses (Table 5), age, central obesity, hypertension and physical inactivity were significant independent predictors overall of CAD in this study population. Age was the strongest predictor, those above 64 years of age were six times more likely to develop CAD (Adjusted Odds Ratios [AOR]: 6.1; 95% CI: 3.2; 11.8). Those with a history of hypertension were more than two-fold at risk of developing CAD (AOR: 2.2; 95% CI: 1.4; 3.4). Hypertension and diabetes remained independent significant predictors of males and females, respectively. Those with a history of no diabetes were 57% less likely to develop CAD among females only (AOR: 0.43; 95% CI: 0.12; 0.99). History of hypertension remained an independent risk factor of CAD among males only (AOR: 2.4; 95% CI: 1.3; 4.4).

4. Discussion

This cross-sectional study of adequate statistical power and representativeness ($n = 1178$) conducted for the first time among an apparently urban healthy population in Eastern India, a region with unique lifestyles and culture, has three distinct findings. First, a very high 10% overall CAD prevalence solely based on objective clinical parameters is slowly approaching the levels among migrant South Asian populations in the Western world. Second, a very high prevalence

Table 1 Prevalence of CAD and coronary risk factors.

Variables	Males = 590	Females = 588	Total = 1178
Age (years)	47 ± 14.46	44.21 ± 13.26	45.92 ± 13.9
CAD prevalence	59(10.0)	59(10.0)	118(10.0)
Smoking	247(41.9)	73(12.4)	320(27.0)*
Physical inactivity	222(37.6)	178(30.3)	400(34.0)*
Low/no fruit intake	369(62.5)	352(59.9)	721(61.2)
Hypertension (history or SBP ⩾ 140 or DBP ⩾ 90 mmHg)	227(38.5)	204(34.7)	431(36.6)
Diabetes (history or FBS ⩾ 126 mg/dl or PGBS ⩾ 200 mg/dl)	105(17.8)	80(13.6)	185(15.7)
General obesity BMI ⩾ 25 kg/m^2	234(39.7)	282(48.0)	516(43.8)*
Central obesity (WC males ⩾ 90 cm females ⩾ 80 cm)	247(41.9)	329(56.0)	576(48.9)*
Hypercholesterolemia ⩾ 200 mg/dl	128(21.7)	145(24.7)	273(23.2)
Hypertriglyceridemia ⩾ 150 mg/dl	232(39.9)	212(36.1)	444(37.7)
High LDL ⩾ 130 mg/dl	127(21.5)	138(23.5)	265(22.5)
Low HDL (males < 40 mg/dl, females < 50 mg/dl)	56(9.5)	497(84.5)	553(46.9)**

Numbers in parenthesis indicate percentages.
* $P < 0.05$.
** $P < 0.01$.

Table 2 Mean and Standard Deviation (SD) of CAD risk factors studied ($n = 1178$).

	N	Minimum	Maximum	Mean	SD
Syotolic BP mmHg	1178	100	198	131.89	14.462
Diastolic BP mmHg	1178	64	120	84.09	8.514
Blood sugar	1178	50	507	97.07	35.706
Blood sugar post glucose	1178	76	706	137.23	57.051
Cholesterol mg%	1178	103	309	182.10	35.502
Triglyceride mg%	1178	60	574	157.80	60.305
LDL Chol mg%	1178	34	211	104.28	31.350
HDL in mg%	1178	33	64	46.12	4.458
Valid N (listwise)	1178				

Table 3 Age-specific prevalence of coronary risk factors.

Risk factor	20–30	31–40	41–50	51–60	61–70	71–80	Total
CHD (%)	8(4.4)	8(2.8)	20(7.0)	40(16.7)	20(15.4)	22(40.7)	118(10.0)**
Smoking	34(18.8)	63(22.0)	93(32.5)	71(29.6)	40(30.8)	19(35.2)	320(27.0)**
Physical activity	35(19.3)	71(24.7)	93(32.5)	89(37.1)	68(52.3)	44(81.5)	400(34.0)**
No/low fruit intake	111(61.3)	156(54.4)	167(58.4)	168(70.0)	83(63.8)	36(66.7)	721(61.2)
Hypertension	18(9.9)	50(17.4)	115(40.2)	132(55.4)	82(63.1)	34(63.0)	431(36.6)**
Diabetes	4(2.2)	19(6.6)	35(12.2)	69(28.8)	43(33.1)	15(27.8)	185(15.7)**
General obesity	34(18.8)	116(40.4)	147(51.4)	124(51.7)	75(57.7)	20(37.0)	516(43.8)**
Central obesity	41(22.7)	117(40.8)	157(54.9)	148(61.7)	87(66.9)	26(48.1)	576(48.9)**
High chol	26(14.4)	54(18.8)	73(25.5)	74(30.8)	32(24.6)	14(25.9)	273(23.2)**
High TG	31(17.1)	80(27.9)	121(42.3)	124(51.7)	65(50.0)	23(42.6)	444(37.7)**
High LDL	44(24.3)	53(18.5)	65(22.7)	62(25.8)	29(22.3)	12(22.2)	265(22.5)
Low HDL	80(44.2)	154(53.7)	142(49.7)	105(43.8)	51(39.2)	21(38.9)	553(46.9)

Numbers in parenthesis indicate percentages.
** $P < 0.01$.

Table 4 Univariate analyses for risk factors affecting CAD.

Risk factors	Chi-square	*p*-value	Odds Ratio	95% C.I	
Hypertension	41.1	0.00**	3.44	2.32	5.12
Diabetes	5.10	0.024**	1.69	1.068	2.68
General Obesity	0.42	0.52	1.13	0.77	1.66
Central Obesity	0.41	0.52	1.13	0.77	1.66
High cholesterol	2.34	0.13	1.39	0.91	2.12
High TG	6.29	0.012**	1.625	1.10	2.38
High LDL	0.65	0.42	1.19	0.77	1.856
Low HDL	1.65	0.19	1.28	0.87	1.87
Smoking	0.05	0.81	0.95	0.62	1.46
Alcoholic	0.15	0.69	0.88	0.46	1.68
Low/no fruit intake	3.79	0.05**	1.50	0.99	2.26
Diet	6.44	0.01**	1.70	1.12	2.58
Fam Hx of CAD	3.24	0.07	0.53	0.26	1.06
History of hypertension	44.95	0.00**	3.55	2.40	5.23
History of diabetes	5.02	0.02**	1.78	1.07	2.96

** $P < 0.01$.

rate of hypertension (37%) was reported that raises important clinical and public health policy implications, despite the availability of cost-effective anti-hypertensive drugs. Finally, conventional risk factors showed distinct gender variations, as significant predictors of CAD following multivariable logistic regression analyses.

Orissa is one of the poorest states in India but Berhampur, the study site, experienced rapid urbanization in recent years. India has also the dubious distinction of being the diabetes capital, and an equally high prevalence of almost 16% diabetics was also reported in this study population which is higher than an earlier estimate of 11%. Such high estimates

Table 5 Significant predictors of CAD among the general population in South Orissa (backward elimination logistic regression modeling) by gender.

Variables	Adjusted Odds Ratios (AOR)	95% Confidence intervals (CI)	
(1) Overall (n = 1178)			
Age (in years)			$p < 0.0001$
<45	Reference		
45—64	3.74	2.13—6.59	
>64	6.12	3.18—11.8	
Hypertension			$p = 0.0005$
No	Reference		
Yes	2.19	1.41—3.40	
Physical inactivity			$p = 0.003$
Yes	Reference		
No	0.53	0.35—0.81	
Central obesity			$p = 0.036$
Yes	Reference		
No	0.64	0.42—0.97	
(2) Males (n = 590)			
Age (in years)			p = 0.002
<45	Reference		
45—64	2.89	1.31—6.39	
>64	4.89	2.05—11.7	
Hypertension			$p = 0.004$
No	Reference		
Yes	2.43	1.34—4.41	
Physical inactivity			$p = 0.02$
Yes	Reference		
No	0.50	0.28—0.89	
(3) Females (n = 588)			
Age (in years)			$p < 0.0001$
<45	Reference		
45—64	5.69	2.64—12.3	
>64	11.6	4.24—31.6	
Diabetes			$p = 0.046$
Yes	Reference		
No	0.43	0.19—0.98	
Physical inactivity			$p = 0.03$
Yes	Reference		
No	0.49	0.26—0.94	

reflect the fast changing lifestyle patterns consistent with the fact that significant levels of physical inactivity were observed compounded by high levels of both central and general obesity. The total prevalence of CAD in the present study is 10.0% which is lower than in the Tirupathi study [27] (12.63%) in 2006, in the Panjim study [28] (13.21%) in 2004, and in the Chennai study [15] (11%) in 2001 but higher than in the Jaipur study [29] (8.12%) in 2002 JHW-2. It is evident that CAD prevalence has increased ten-fold since the early 1960s which is a matter of great concern. Comprehensive nationwide CAD prevention and control programs are patchy and are yet to have an impact on CAD morbidity and mortality at the population level.

Consistent with earlier findings, age is also a strong independent predictor of CAD in this study population. Although those above 65 years of age had the greatest risk of developing CAD, individuals above 45 years of age were also at a threefold increased risk of developing CAD. Previous evidence that relatively young individuals of Indian ethnic origin develop CAD is also reflected in our study population. Nevertheless, conventional risk factors are also significant predictors of CAD in this study population indicating that lifestyle and environmental risk factors still play a major role in contributing to the rising incidence of CAD in India, in addition to a unique genetic pool. Such findings strongly suggest that lifestyle factors are cornerstones for any comprehensive prevention and control programs to reduce this emerging burden of CAD, which is further worsened due to rising hypertension and diabetes prevalence rates.

In our study total prevalence rates of CAD in both sexes are similar although gender variations were reported earlier (Tirupathi study [27]; the Chennai study [15] and in Trivandrum [30]). However, distinct gender variations were observed

in the conventional risk factors. For example, hypertension and diabetes were independent significant predictors in males and females, respectively, and the reasons are unclear. It is well known that Asian Indians have low HDL cholesterol levels, which could be one of the risk factors for premature CAD among this ethnic group. The fact that very significant low levels of HDL are reported among females in this study population and the overall mean HDL (46 mg/dl) is also low, might suggest a significant increased risk of diabetes among females. Nevertheless, long follow-up studies are imperative to explore such possibilities among Asian Indians before embarking on a sex-specific Non-Communicable Disease (NCD) Prevention and Control Program in India and elsewhere.

Although a thrombogenetic risk factor was suggested to support an increased risk of CAD among South Asians, our study did not show significant effects of cholesterol or LDL levels on CAD. Lipid lowering drugs might have influenced such findings, but the uptake levels of such drugs would be very minimal in such low-resource settings. Also, the overall prevalence of hypertension in our study is much higher 36.6% (38.5% in males and 34.7% in females), while the Tirupathi study [27] reported 26.06% (27.64% in males and 25.20% in females) and in the Jaipur study [31](JHW-4) it was 53.3% (57.9% in males and 48.9% in females). Such observations indirectly indicate low compliance and poor uptake of anti-hypertensive drugs. In addition, the lack of adherance to a strict hypertension treatment guideline might be contributing to such a high prevalence of hypertension.

Although serum triglyceride was significantly associated with CAD in the univariate analyses, such effects were not observed following multivariate analyses. Nonetheless, the role of serum triglyceride as a risk factor for CAD remains controversial. Likewise, smoking was not a significant predictor which might be due to reporting bias or the lack of objective measurements, and a greater use of smokeless products might be indirectly contributing to the dilution of effects of smoking on CAD risk in this ethnic population. Compared to previous studies, the present study clearly shows an increasing prevalence of smoking (from 10% in South India and 20% in the Western India in 2007 to the current estimate of almost 27%).

5. Study limitations

This study is a cross-sectional study and therefore no causal inferences can be drawn. However, the study is adequately powered and is representative of an apparently healthy urban population of Southern Odisha because of a probability sampling technique. Recall bias for self-reported behavioral risk factors is a possibility and observer bias such as measurement error from using a conventional sphygmomanometer cannot be ruled out. However, as all readings were taken by the same observer and two readings were taken and averaged, we believe observer bias was reduced to a certain extent.

Prospective longitudinal follow-up studies are required to throw light on the true risk factors of CAD, especially to tease out the gene-environment interactions among South Asian ethnic populations.

6. Conclusions

In conclusion, this cross-sectional study among an ethnic urban population undergoing urbanization and changing lifestyles showed elevated levels of risk of CAD, despite the study being conducted in one of the poorest states in India. In addition, high prevalence rates of some classical cardiovascular risk factors such as diabetes, hypertension, smoking and physical inactivity levels were reported. Such observations reinforce the need for a comprehensive targeted CVD control and prevention program that is sustained, well resourced and effective. Nonetheless, prospective longitudinal follow-up studies are imperative to draw conclusive evidence about ethnic-specific genetic or environmental risk factors contributing to premature CAD and high diabetes and hypertension prevalence rates in ethnic South Asians.

Conflicts of interests

None declared.

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Appendix A

A.1. Anthropometric measurements: [32,33]

Height was measured with a tape to the nearest cm. Subjects were requested to stand upright without shoes with their back against the wall, heels together and eyes directed forward.

Weight was measured with a traditional spring balance that was kept on a firm horizontal surface. The scale was checked every day and calibration was done with ''known'' weights. Subjects were asked to wear light clothing and weight was recorded to the nearest 0.5 kg.

Body mass index (BMI) was calculated using the formula: observed weight divided by height squared (kg/m2).

Waist circumference: Waist was measured using a non-stretchable measuring tape. The subjects were asked to stand erect in a relaxed position with both feet together on a flat surface; one layer of clothing was accepted. Waist girth was measured as the smallest horizontal girth between the costal margins and the iliac crests at minimal respiration.

Hip Circumference: taken as the greatest circumference at the level of the greater trochanter (the widest portion of the hip) on both sides. Measurements were made to the nearest centimeter.

Waist and hip ratio (WHR): was calculated by dividing the waist circumference (cm) by the hip circumference (cm).

Table B1 Details on the definitions of coronary risk factors are presented in the following table.

Definition of coronary risk factors	
Risk factors	Criterion
Hypertension [43]	Drug treated or SBP $\geqslant$ 140 or DBP $\geqslant$ 90 mm Hg
Diabetes [44]	Drug treated or FBS $\geqslant$ 126 mg/dl or PGBS 2 h $\geqslant$ 200 mg/dl
Hypercholesterolemia [45]	Serum Cholesterol $\geqslant$ 200 mg/dl
Hypertriglyceridemia [45]	Serum Triglycerides $\geqslant$ 150 mg/dl
Low HDL cholesterol [45]	Males $\leqslant$ 40 mg/dl, females $\leqslant$ 50 mg/dl
Abnormal LDL cholesterol [45]	LDL $\geqslant$ 130 mg/dl
General obesity [46]	BMI $\geqslant$ 25
Central obesity [46]	Males $\geqslant$ 90 cm; females $\geqslant$ 80 cm
Socioeconomic status [47]	Kuppuswamy socioeconomic status scale-updating for 2007 based on educational status, occupation and per capita income.
Definition of CAD for prevalence studies [48—50]	Definite history or ECG changes suggestive of ST-segment depression (Minnesota 4—1 to 4—2) or Q wave changes (Minnesota codes 1—1—1 to 1—1—7) or T wave changes (Minnesota codes 5—1 to 5—3).
Smoker (past and present) [13,22]	Users of all types of tobacco products
Low/no fruit and vegetable intake [6,18]	Intake of <5 servings a day
Physical activity [25,26]	Both work related and leisure time activities

Blood pressure [34] was recorded in the sitting position in the right arm to the nearest 2mmHg using a mercury sphygmomanometer. Two readings were taken 5 minutes apart and the mean of two was taken as the blood pressure.

A.2. Biochemical Investigations

A fasting blood sample was collected after an overnight fast of at least 10 hours for biochemical investigations. All biochemical parameters were performed using enzymatic kits as per the following methods; plasma glucose by GOD/POD Method (Trinder, 1969) [35], total cholesterol by CHOD/PAP Method (Allain *et al*. 1974) [36], triglycerides by GPO/PAP Method(Fossati and Prencipe, 1982) [37] and HDL by PEG/CHOD-PAP Method (Donald and smith, 1985) [38], LDL and VLDL were calculated using formulae of Freidewald *et al*. (1972) [39] and Wilson and Spiger (1973) [40]. Non-HDL cholesterol was calculated using formula (total cholesterol - HDL cholesterol).

A.3. Electrocardiogram

The recommended procedure for recording a resting ECG, and the technical requirements for a suitable electrocardiograph, are described in detail in the reference manual for the Minnesota code [41] and the same was followed for all the participants.

Appendix B

B.1. Definition of Coronary risk factors

The diagnostic criteria for coronary risk factors have been advised by the WHO [22] and American College of Cardiology Clinical Data Standards [42].

See Table B1

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