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Coronary Heart Disease in a Remote Area

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Abstract

A study on coronary heart disease (CHD) in a remote area, Ceningan Island,was conducted.The nutrition states based on BMI were underweight,19.3%; normoweight, 54.9%; and overweight at risk, 13.3%; obese I, 9.5%; and obese II, 3.0%. The prevalence of impaired fasting glycemia (IFG), 15.1%; diabetes mellitus (DM), 6.9%; metabolic syndrome (MetS), 6.8%; and CHD, 11.5%. Age, systolic and diastolic blood pressure, levels of total cholesterol; low-density lipoprotein (LDL) cholesterol, apolipoprotein (Apo)A, ApoB, Apo B/A ratio were found higher among subjects with old myocardial infarction (OMI) than subjects without CHD; while BMI and waist circumference were lower among subjects with OMI than subjects without CHD. Conclusion: traditional risk factors such as age, blood pressure, and cholesterol, and Apo were related to CHD in the remote area.

Keywords: Coronary heart disease; Remote area

Introduction

Coronary heart disease (CHD) is one of the main causes of mortality in the most countries in the world. Althought a decreasing trend has been noted in many developed countries the incidence of CHD in many developing countries tends to increase. A report by American Heart Association, based on data in 2005, revealed that overall death rate from cardiovascular diseases (CVD) was 278.9 per 100000, nearly 2400 American die of CVD each day, CHD caused 1 of every 5 death, each year about 795000 people experience a new or recurrent stroke [1]. A report from Japan, based on longitudinal community-based study (1964-2003) showed a significant increase in the incidence of CHD in Asia; adopting more Westernized diets and lifestyle might be the underlying reason for the increasing trends [2].

Coronary heart disease is a complex disorder, with many risk factors involve in its pathogenesis. In general, risk factors for CHD can be devided into two main groups i.e. traditional and non-traditional risk factors. Traditional risk factors include age (older than 40 years for men, 45 years for women), male sex, family history of coronary heart disease, smoking, hypertension, diabetes, central obesity, unhealthy cholesterol levels (high total cholesterol, low high-density lipoprotein [HDL] cholesterol, high low-density lipoprotein [LDL] cholesterol, high triglycerides), and low physical activity[3,4]. The non-traditional risk factors and its impact on CHD have been reviewed [3,5,6]. The impact of each risk factor on CHD event have been described to vary widely in the various study.

The objectives of the study were to know the prevalence and some risk factors related to coronary heart disease inpopulation of a remote area, Ceningan island.

Methods

A cross sectional field study was conducted at the population of Ceningan Island, one of three small adjucent islands located, called the Nusa Penida Islands, located at the south-eastern part of Bali (Figure 1). In the past, populations of these islands live relatively isolated and had much lower finacial incomes as compared with those of the general population of Bali. Nusa Penida islands are consist of 3 sister islands, i.e. Penida island, Lembongan island, and Ceningan island. Apart from the other two sister islands, the population of Ceningan island has remained practically untouched and isolated from the booming neighbouring tourism industry of the island of Bali.

Three hundred five (means of age was 43 [14-100] years; male/female, 148/157) out of 888 total populationin the island were recruited by simple random sampling. Variables measured in the study included age (year), body weight (kg), body mass index (BMI[kg/m^2]), waist circumference (cm), systolic blood pressure (mmHg), diastolic blood pressure (mmHg), fasting blood glucose (mg/dl), total-cholesterol

Figure 1: Map of Penida (Penida, Lembongan and Ceningan) Islands.

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(mg/dl), LDL-cholesterol (mg/dl), HDL-cholesterol (mg/dl), triglyceride (mg/dl), apolipoprotein (Apo)A (mg/dl), ApoB (mg/dl), total-/HDL-choletserol ratio, LDL-cholesterol/ApoB ratio, Apo B/A ratio, serum creatinine (mg/dl), uric acid (md/dl), macroalbuminuria (negative or positive by routine urinalysis), excercise (no or never , yes [sometimes to regularly]), alcohol drink (no drink, yes [sometimes to regularly]), smoking habit (no smoking, yes [sometimes to regularly]). All biochemical variables were measured in fasting state. Diagnosis of left ventricular hypertrophy, myocardial ischemia (ST-segment depression and T wave inversion) and old myocardial infarction (Q wave) were based on electrocardiogarphy (ECG) examination [7]. Coronary heart disease was defined by using ECG tracings which were consistent with myocardial ischemia and old myocardial infarction (OMI). Classification of nutrition states by BMI and central obesity by waist circumference were defined by WHO criteria for Asia Pacific population (2000) [8]. Diagnosis of impaired fasting glycemia (IFG) and diabetes mellitus (DM) was confirmed by using ADA (2009) criteria, i.e. IFG if fasting blood glucose levels was ≥100 mg/dL and <126 mg/dL; and DM if fasting blood glucose levels was ≥126 mg/dL [9]. Diagnosis of metabolic syndrome (MetS) was defined by criteria of A Joint Interim Statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for theStudy of Obesity (2009). Metabolic syndrome is diagnosed if 3 out of 5 below criteria are found: elevated waist circumference (male >90 cm, femal >80 cm); elevated triglycerides (drug treatmentfor elevated triglycerides is analternate indicator; ≥150 mg/dL); reduced HDL-cholesterol (drug treatment forreduced HDL-cholesterol is an alternateindicator; ≤40 mg/dL inmales, and ≤50 mg/dL infemales); elevated blood pressure(antihypertensive drug treatment in apatient with a history of hypertensionis an alternate indicator; systolic ≥130 and/or diastolic ≥85 mm Hg); elevated fasting glucose (drugtreatment of elevated glucose is analternate indicator; ≥100 mg/dL) [10]. Defining cut-off points of some risk factors for CHD were as follows:older-age (male ≥45 year and female ≥55 year), systolic hypertension (systolic blood pressure ≥140 mmHg), diastolic hypertension (diastolic blood pressure ≥90 mmHg), high total-cholesterol (≥240 mg/dl), high LDL-cholesterol (≥160 mg/dl), low HDL-cholesterol (<40 mg/dl), high triglyceride (≥200 mg/dl)were based on ATP-III criteria (2001) [11]. Small-dense LDL was represented by LDL-cholesterol/Apo B ratio.

Statistical methods used for data analysis include descriptive presentation, independent t-test and cross-tab (chi square); with each significant value were confirmed at $p<0.05$.

Results

The levels and prevalence of almostall measured variables are within normal range; but some variables such as body weight, total-/HDL-cholesterol ratio, apolipoprotein B/A ratio, serum creatinine and uric acid levels were higher and HDL cholesterol, apolipoprotein A levels, LDL-cholesterol/Apo B ratio were lower in the male than in the female. The prevalence of excercise, alcohol drink and smoking were higher in the male (Table 1). In the population, the prevalence of underweight was actually relatively high (19.3%) and was higher than prevalence of obesity in both gender (11.5%). The prevalence of DM was moderate (6.9%), nevertheless the prevalence of CHD in this area were relatively high (11.5%). The prevalence of central obesity were significantly higher in the female as compared with that in the male (7.2 vs. 28.0, respectively; $p<0.001$); and althought any tends higher prevalence of obesity by BMI in women but it was not different significantly ($p = 0.068$). Female have higher prevalence of central obesity, but this was not associated with higher prevalence of MetS significantly (7.2% in male and 12.0% in female; $p = 0.180$). The prevalence of glucose intolerance (IFG [12.8% in male and 17.2% in female] and DM [8.8% in male and 5.1% in female]; $p = 0.291$) in both sexes was comparable. The prevalence of left ventricular hypertrophy was higher in the male (8.8% in male and 2.5% in female; $p = 0.018$), but the prevalence of myocardial ischemia (2.0% in male and 5.1 in female) and OMI (9.5 in male and 6.4 in female) was comparable in both sexes ($p = 0.233$) (Table 2).

The noteworthy clinical and biochemical features of subjects with CHD were older age (40.5 years in subjects without CHD, 58.5 years in subjects with myocaridal ischemia, and 65.0 years in subjects with OMI), lower body weight (54.6 kg in subjects without CHD, 45.2 kg in subjects with myocaridal ischemia, and 48.3 kg in subjects with OMI),

Variables	Male (N=148)	Female (N=157)	Total (N=305)	P Male vs. Female
Age (year)	44.0 ± 17.0	42.2 ± 17.5	43.0 ± 17.7	0.367
Body weight (kg)	56.3 ± 9.4	51.2 ± 10.8	53.7 ± 10.4	<0.001
BMI (kg/m^2)	21.0 ± 2.8	21.6 ± 4.0	21.3 ± 3.4	0.130
Waist circumference (cm)	75.1 ± 8.9	75.3 ± 10.3	75.2 ± 9.3	0.870
Systolic blood pressure (mmHg)	118.0 ± 20.0	117.6 ± 18.7	117.8 ± 19.3	0.844
Diastolic blood pressure (mmHg)	74.6 ± 10.8	74.4 ± 10.7	74.5 ± 10.7	0.866
Fasting blood glucose (mg/dl)	101.3 ± 43.0	95.6 ± 34.9	98.4 ± 39.0	0.202
Total-cholesterol (mg/dl)	193.9 ± 34.7	204.9 ± 41.4	199.5 ± 38.6	0.013
LDL-cholesterol (mg/dl)	125.1 ± 29.6	129.2 ± 33.3	127.2 ± 31.6	0.266
HDL-cholesterol (mg/dl)	53.9 ± 10.3	61.7 ± 12.3	57.9 ± 12.0	<0.001
Triglyceride (mg/dl)	106.9 ± 53.0	96.1 ± 45.0	101.3 ± 49.7	0.055
Apolipoprotein A (mg/dl)	119.4 ± 17.1	129.8 ± 17.8	124.8 ± 18.2	<0.001
Apolipoprotein B (mg/dl)	89.0 ± 21.0	88.7 ± 23.2	88.8 ± 22.1	0.926
Total-/HDL-choletserol ratio	3.7 ± 0.8	3.4 ± 0.8	3.5 ± 0.8	0.003
LDL-cholesterol/Apolipoprotein B ratio	1.41 ± 0.1	1.46 ± 0.1	1.4 ± 0.1	<0.001
Apolipoprotein B/A ratio	0.76 ± 0.21	0.70 ± 0.19	0.72 ± 0.20	0.004
Serum creatinine (mg/dl)	1.1 ± 0.2	0.9 ± 0.1	1.0 ± 0.2	<0.001
Uric acid (md/dl)	6.2 ± 1.3	4.7 ± 1.0	5.4 ± 1.3	<0.001
Macroalbuminuria	8.2%	14.8%	11.6%	0.070
Exercise*	30.8%	11.1%	21.2%	<0.001
Alcohol drink*	19.6%	0.0%	9.6%	<0.001
Smoking habit*	35.3%	0.0%	17.4%	<0.001

*Sometimes to regularly

Table 1: Characteristics of subjects by gender.

higher blood pressure (116.0/73.8 mmHg in subjects without CHD, 124.8/75.4 mmHg in subjects with myocaridal ischemia, and 132.4/81.0 mmHg in subjects with OMI), higher levels of total-cholesterol levels (196.5 mg/dl in subjects without CHD, 216.4 mg/dl in subjects with myocaridal ischemia, and 226.3 mg/dl kg in subjects with OMI), LDL-cholesterol levels (125.2 mg/dl in subjects without CHD, 137.4 mg/dl in subjects with myocaridal ischemia, and 145.1 mg/dl in subjects with OMI), Apo A levels (123.8 mg/dl in subjects without CHD, 130.6 mg/dl in subjects with myocaridal ischemia, and 132.8 mg/dl in subjects with OMI), Apo B (87.4 mg/dl in subjects without CHD, 95.0 mg/dl in subjects with myocaridal ischemia, and 102.5 mg/dl in subjects with OMI), Apo B/A ratio (0.71 in subjects without CHD, 0.74 in subjects with myocaridal ischemia, and 0.80 in subjects with OMI), and creatinine serum levels (1.0 mg/dl in subjects without CHD, 0.9 mg/dl in subjects with myocaridal ischemia, and 1.1 mg/dl in subjects with OMI) than in subjects without CHD (Table 3).

Discussion

Overall, means values or levels of all measured variables were likely within normal range (Table 1). Nevertheless, by further analysis, obesity appeared to be less frequent found than underweight (12.8% *vs.* 18.9%). The prevalence of DM and obesity were moderate, 6.9% and 11.5%, respectively. Some traditional risk factors for CHD were measured in the study. Age, underweight, systolic blood pressure, diastolic blood pressure, levels of total-cholesterol, LDL-cholesterol, Apo A, Apo B, Apo B/A ratio wererelated with CHD event. Smoking habit and alcohol drink were not significantly seen as important risk factors, this might be due to the frequencies and quantity of smoking and alcohol intake being not accurately recorded and thus not analyzed. Similar limitation were also observed in our data on the exercise.

Study by Daviest et al. (2007) in United Kingdom showed that from 1996 to 2005 there was decreasing incidence of coronary heart disease (CHD) over 35 years by 2.2% in men and 2.3% in women per year; and decreased all-caused mortality among those with CHD by 4.5% in men and 3.4% in women per year. Inversely, the prevalence of CHD increased by 1.3% in men and 1.7% in women; the results might be due to improvement in survival among people with CHD [12]. A study among African Americans aged 45 to 64 years revealed that incidence rate of CHD was 4.9 in black women, 2.9 in white women, 9.2 in black men, and 7.9 in white men. By multivariable analysis, hypertension was a strong risk factor in black women, DM was somewhat more predictive in white women, LDL-cholesterol was predictive in all race-sex groups, and HDL-cholesterol seemed somewhat more predictive in white than in black persons [13]. Some reviews stressed that age is the strongest risk factor for CVD. Age itself may be an independent risk factor or may have other risk factors related aging or exposure to risk factors during their life [14-16]. The prevalence of central obesity and MetS in this study were relatively low, but the prevalence of DM and CHD were relatively high for population who live in a traditional way and being far from excessive calory intake and modern life amenity. Very similar to other findings that some traditional risk factors, especially age, were also related to CHD, except underweight which appeared to be a risk factor for CHD in this study. Race and genetics factors may account for the differences in findings among the various studies.

Variables	Male (%)	Female (%)	Total (%)	p Male vs. Female
Nutrition state				
Underweight (BMI<18.5 kg/m^2)	17.4	21.2	19.3	0.068
Normoweight (BMI = 18.5-22.9 kg/m^2)	62.1	47.7	54.9	
Overweight at risk (BMI = 23-24.9 kg/m^2)	12.1	14.4	13.3	
Obesity I (BMI = 25-29.9 kg/m^2)	7.6	11.4	9.5	
Obesity II (BMI≥30 kg/m^2)	0.8	5.3	3.0	
Central obesity	7.2	28.0	17.7	<0.001
Metabolic syndrome	7.2	12.0	9.6	0.180
Glucose intolerance				
Impaired fasting glycemia	12.8	17.2	15.1	0.291
Diabetes mellitus	8.8	5.1	6.9	
Left ventricular hypertrophy	8.8	2.5	5.6	0.018
Coronary heart disease				
Myocardial ischemia	2.0	5.1	3.6	0.233
Old myocardial infarction	9.5	6.4	7.9	

Table 2: Prevalence of nutrition state and obesity, glucose intolerance, metabolic syndrome, and coronary heart disease by gender.

	Normal[1]	Myocardial ischemia[2]	Old myocardial infarction[3]	p (1 *vs.* 2; 1 *vs.* 3; 2 *vs.* 3)
Age (year)	40.5 ± 15.9	58.5 ± 15.1	65.0 ± 13.9	<0.001; <0.001; 0.260
Body weight (kg)	54.6 ± 10.1	45.2 ± 5.4	48.3 ± 12.0	0.003; 0.004; 0.392
BMI (kg/m^2)	21.6 ± 3.4	19.5 ± 1.9	19.8 ± 3.8	0.480; 0.018; 0.773
Waist circumference (cm)	75.8 ± 9.3	70.2 ± 6.9	71.5 ± 9.0	0.050; 0.031; 0.696
Systolic blood pressure (mmHg)	116.0 ± 18.4	124.8 ± 15.3	132.4 ± 22.6	0.132; <0.001; 0.265
Diastolic blood pressure (mmHg)	73.8 ± 10.7	75.4 ± 8.7	81.0 ± 10.6	0.619; 0.002; 0.149
Fasting blood glucose (mg/dl)	96.4 ± 34.5	118.5 ± 78.7	111.9 ± 55.6	0.064; 0.062; 0.637
Total-cholesterol (mg/dl)	196.5 ± 37.8	216.4 ± 34.4	226.3 ± 38.7	0.087; <0.001; 0.473
LDL-cholesterol (mg/dl)	125.2 ± 31.8	137.4 ± 23.0	145.1 ± 27.1	0.206; 0.003; 0.495
HDL-cholesterol (mg/dl)	57.4 ± 11.6	60.4 ± 11.7	62.4 ± 15.8	0.417; 0.052; 0.654
Triglyceride (mg/dl)	99.6 ± 50.4	104.3 ± 41.5	119.1 ± 35.8	0.759; 0.063; 0.407
Apolipoprotein A (mg/dl)	123.8 ± 17.2	130.6 ± 17.2	132.8 ± 25.7	0.219; 0.019; 0.738
Apolipoprotein B (mg/dl)	87.4 ± 22.1	95.0 ± 16.5	102.5 ± 19.4	0.257; 0.001; 0.344
Total-/HDL-choletserol ratio	3.5 ± 0.8	3.7 ± 0.8	3.8 ± 1.0	0.530; 0.125; 0.713
LDL-cholesterol/Apolipoprotein B ratio	1.4 ± 0.1	1.4 ± 0.1	1.4 ± 0.1	0.800; 0.562; 0.580
Apolipoprotein B/A ratio	0.71 ± 0.20	0.74 ± 0.19	0.80 ± 0.25	0.677; 0.046; 0.413
Serum creatinine (mg/dl)	1.0 ± 0.2	0.9 ± 0.1	1.1 ± 0.3	0.018; 0.011; 0.001
Uric acid (md/dl)	5.4 ± 1.3	4.9 ± 1.3	5.9 ± 1.6	0.207; 0.086; 0.039

[1]Normal, subjects without CHD

Table 3: Levels or ratio of risk factors for coronary heart disease.

Weaknesses of the study which might cause bias of the results was the fact that this a cross sectional study, due to technical difficulties the diagnosis of CHD was comfirmed only by using ECG tracings. Data concerning excercise, alcohol drink and smoking were grouping dichotomizedly in "yes" and "no" without further elaborating when the answer is "yes". The non-traditional risk factors for CHD were not measured in this study. Further study is needed to further elucidate the mechanisms underlying the realtionship between underweight and CHD in the population.

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Conflict of Interest

The authors declare no conflict of interest relevant to this article.

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