e-ISSN: 0975-1556, p-ISSN:2820-2643

Available online on www.ijpcr.com

International Journal of Pharmaceutical and Clinical Research 2023; 15 (12); 1893-1899

Original Research Article

Analysis on Risk Factors of Premature Coronary Artery Diseases in Tertiary Care Centre

Md. Shaukat Ali[1], Vijay Trehan[2]

[1]DM Cardiology, GB Pant Hospital, New Delhi
[2]Professor, GB Pant Hospital, New Delhi

Received: 30-09-2023 / Revised: 20-10-2023 / Accepted: 13-11-2023
Corresponding author: Dr. Md. Shaukat Ali
Conflict of interest: Nil

Abstract:

Background: These days, heart attacks and cardiac fatalities are so regular that when an elderly person passes away, it's hardly noticed because it seems predictable and natural. The risk of coronary artery disease in Indians is substantially correlated with conventional risk factors, as it is in all other populations. The purpose of this study was to assess the association between traditional risk factors and early coronary artery disease by examining the levels of triglycerides, lipoproteins, and HbA1c.

Methods: For three years, from 2018 to 2020, the Department of Cardiology at GB Pant Hospital, New Delhi, was the place of this study. 180 patients, 116 males and 64 females, underwent angiography which we analyzed. Three stages of analysis were conducted on the study population: first, their demographic representation according to risk factors; second, the presence and type of angiographic findings; third, a case control type analysis to demonstrate the independent significance of risk factors in relation to CAD; and finally, a risk factor analysis based on the severity of the angiographic findings. Calculations were made for weight, BMI, HbA1c, pulse, systolic and diastolic blood pressure, LDL, VLDL, HDL, total cholesterol, and triglycerides.

Result: The controls had a mean age of 51±6.3 years while the patients had a mean age of 50±7.3 years, with a male preponderance [3.5:1]. The most common conventional risk factors found in the entire study sample were alcohol use (43%), tobacco use (38%), dyslipidemia (41%), family history (32%), hypertension (53%), diabetes (50%), and dyslipidemia (41%); in contrast, the most common conventional risk factors found in cases of CAD were alcohol use (60%), tobacco use (53%), dyslipidemia (62%), and family history (73%). The following risk factors were statistically significant: total cholesterol, triglycerides, LDL, BMI, and HbA1c. Dyslipidemia and the severity and development of CAD were 20% related.

Conclusion: Additionally, multifactorial causation of CAD is demonstrated by our study, with a higher number of risk variables in cases and a lower number in controls. In 2% of cases, there were eight risk factors, while in 31% of cases, there were six.

Keywords: Angiographic Severity of Disease, Conventional Risk Factors, Premature coronary artery disease [CAD].

Introduction

These days, heart attacks and cardiac fatalities are so regular that when an elderly person passes away, it's hardly noticed because it seems predictable and natural. However, there could be serious repercussions if young people who are in the best of health are affected.

Similar to the elderly, atherosclerosis is the primary cause of coronary artery disease (CAD) in young people. It is responsible for 80% of heart attacks, with 60% of cases being caused by a single CAD. About 4 percent of heart attacks in young adults are due to inborn anomalies in the structure of the coronary arteries. [1] Coronary heart disease is on the rise, particularly in India. According to the Registrar General of India, CAD caused 17% of all deaths and 26% of adult fatalities in 2001–2003. From 2010–2013, total and adult deaths increased by 6% in comparison to the previous years. Following a review of research, WHO India has observed rising rates of CAD over the past 60 years, rising from less than 1% to 6% in rural areas and from 1% to 10% in urban areas. [2] With age standardization used, the prevalence rate of CAD is 11.0% overall.

Diabetes and reduced glucose tolerance are two major risk factors for the high occurrence of CAD. Triglycerides (TG), total cholesterol, low-density lipoprotein (LDL) cholesterol, and the ratio of total cholesterol to high-density lipoprotein (HDL) all rise with an increase in the prevalence of coronary

artery disease (CAD). [3] The fact that atherosclerosis begins in childhood and advances gradually for decades is well known. Angina, an advanced warning symptom of heart disease, appears in only one-third of patients. If not, it typically appears as a heart attack or unexpected death that occurs without any prior warning symptoms. [4] Over the past 50 years, the Framingham Heart Study has consistently validated the notion of cardiovascular risk factors. [5] These risk variables, which are now divided into standard and non-traditional risk factors, have been better understood because to advancements in medical research. The likelihood of developing cardiovascular disease rises with the number or intensity of risk factors. [6]

The traditional risk factors include mental stress, a sedentary lifestyle, alcoholism, smoking, diabetes, obesity, hypertension, hyperlipidemia, and family history. A balanced diet, frequent exercise, and abstaining from tobacco use can prevent at least 80% of CAD. These days, early risk assessment and mitigation in the high-risk adult population is the hallmark of preventive care. [7]

A renewed sense of urgency for primordial and primary prevention has been sparked by an increased understanding of the consequences of prolonged exposure to risk factors and worry about the epidemic of CAD in the younger population. Research into new pathophysiological variables causing CVD has increased recently. These cardiovascular disease (CVD) factors have the potential to improve clinical risk management by aiding in the diagnosis, evaluation, and prognosis of atherosclerotic disease. [12]

The risk of CAD in Asian Indians is substantially correlated with conventional risk factors, as it is in all other ethnicities. Asian Indians are more likely to have low HDL, high TG, central obesity, and glucose intolerance than Caucasians are to have low cholesterol, hypertension, and smoking. [8]

Finally, in light of the several risk factors that are present concurrently with the severity of the disease, glycated hemoglobin (HbA1c), lipoproteins, and triglycerides will be investigated. [9] Because, in addition to other known risk factors, diabetes and dyslipidemia must be considered as significant risk factors when it comes to more serious disease.

It will be useful to deduce that, particularly in the presence of additional risk factors, early interventions are necessary to manage the level of triglycerides and lipids with tighter glycemic control. The current study also seeks to demonstrate the necessity of multifactorial analysis in order to correlate with the effects of different combinations of risk variables that are present at a given period.

Material and Methods

Over a period of three years, from 2018 to 2020, the study was carried out at the Department of Cardiology, GB Pant Hospital, New Delhi. 180 patients, of both sexes, underwent angiography and were investigated. The three stages of the study population analysis were: their demographic representation according to risk factors; the presence and type of angiographic findings; an independent significance analysis of risk factors in relation to CAD using case control type analysis; and an analysis of risk factors based on the severity of the angiographic findings. LDL, VLDL, HDL, total cholesterol, triglycerides, pulse, weight, BMI, HbA1c, and diastolic and systolic blood pressure were all calculated.

Results

Table 1: Demographic distribution of cases and control

		Group		Total
		Cases	Controls	
Age	<= 35	2(2%)	2(2%)	4
	36 – 45	24(27%)	12(13%)	36
	46 - 55	52(58%)	60(67%)	112
	56+	12(13%)	16(18%)	28
Gender	Male		116	64.44%
	Female		64	35.56%

Table 1 show that 27% of cases and 13% of controls, respectively, were in the 36–45 age range. In the 46–55 age range, 58% of cases and 67% of controls were present. The cases' average age was fifty years, with a 7.3-year standard deviation (SD). The controls had a mean age of 51 years and an SD of 6.3 years.

Table 2: Risk factor significance analysis compiled group statistics

Risk Factor	Cases		Controls		P Value (t-test)
	Mean	SD	Mean	SD	
Weight	69.31	9.278	67.51	10.304	0.386
BMI	25.51	3.109	24.20	2.573	0.032
Pulse	78.31	10.146	83.00	11.897	0.047
SBP	128.00	17.787	126.44	13.341	0.640

DBP	82.44	10.478	82.00	8.146	0.823
Serum Creatinine	0.94	0.203	0.97	0.224	0.589
HBAIC	7.58	1.486	5.72	0.813	<0.001
Triglyceride	257.38	68.106	114.20	37.099	<0.001
HDL	35.89	10.456	39.09	7.713	0.102
LDL	164.69	83.433	84.96	27.111	<0.001
VLDL	32.87	20.677	28.62	13.359	0.251
Total Cholesterol	234.31	81.908	152.27	33.452	<0.001

The final section of the research examined the substantial causal relationship between the presence of different risk variables and the various angiographically diagnosed categories of CAD. ANOVA was used to statistically evaluate weight, BMI, HbA1c, and other lipid profile components, such as triglycerides, HDL, LDL, VLDL, and total cholesterol. The p value was then calculated to determine statistical significance. [Table 2]

Table 3: Group characteristics and risk factor significance analysis of angiographicseverity

Risk Factors		**No. of patients**	**Mean**	**SD**	**P Value**
Weight	Normal Coronary	32	66.13	11.407	0.543
	Mild CAD	58	68.28	9.768	
	SVD	24	66.50	9.709	
	DVD	38	69.26	9.544	
	TVD	28	71.79	8.460	
BMI Raw	Normal Coronary	32	24.38	2.156	0.286
	Mild CAD	58	24.10	2.807	
	SVD	24	25.08	3.175	
	DVD	38	25.58	3.372	
	TVD	28	25.79	2.860	
HB/IC	Normal Coronary	32	5.706250	0.5397144	<0.001
	Mild CAD	58	5.734483	0.9393295	
	SVD	24	6.983333	1.1983575	
	DVD	38	7.105263	1.2176816	
	TVD	28	8.735714	1.4505209	
Triglyceride	Normal Coronary	32	104.63	40.013	<0.001
	Mild CAD	58	119.48	34.982	
	SVD	24	289.42	55.801	
	DVD	38	230.79	79.167	
	TVD	28	266.00	48.229	
HDL	Normal Coronary	32	37.75	7.716	0.418
	Mild CAD	58	39.83	7.746	
	SVD	24	37.67	14.355	
	DVD	38	34.68	8.226	
	TVD	28	36.00	9.845	
LDL	Normal Coronary	32	83.19	16.558	<0.001
	Mild CAD	58	85.93	31.707	
	SVD	24	119.33	51.215	
	DVD	38	175.58	96.455	
	TVD	28	188.79	75.906	
VLDL	Normal Coronary	32	26.13	11.419	0.688
	Mild CAD	58	30.00	14.320	
	SVD	24	30.33	20.847	
	DVD	38	32.89	14.529	
	TVD	28	35.00	27.860	
Total	Normal Coronary	32	147.00	25.602	<0.001
	Mild CAD	58	155.17	37.185	
	SVD	24	188.17	42.697	
	DVD	38	244.68	99.233	
	TVD	28	259.79	68.617	

It was discovered that triglycerides, LDL, and HbA1c were significantly significant ($p<0.01$). When statistically examined in five angiographic categories for significance, HDL and VLDL were determined to be risk factors; nevertheless, the results were not significant ($p>0.05$). The trend of

total cholesterol is trending upward as disease severity increases. The statistical analysis revealed a statistically significant result with a p value of less than 0.0.1. [Table 3]

Discussion

According to demographic profiling of the study population, the age distribution of the samples revealed that 62% of them belonged to the 46–55 age range, while only 2% were under 35. The age range of these 27% cases and 13% controls was 36–45 years old. Based on distribution, 58% of cases and 67% of controls were found in the 46–55 age group. The cases' average age was 50 years, with a 7.3-year standard deviation. The controls had a mean age of 51 years and a standard deviation of 6.3 years. This aligns with the Gajanan D. al [3] study, which found that the elder age group averaged 55.39 years. Since all age groups were covered, the study by Bhattacharyya et al. (2006) had a mean age of 57.3 years and a standard deviation of 8.7 years. The mean age of the Sriharibabu et al. (2013) study was 54.5 years, which is consistent with our findings. Their investigation revealed that, when the age distribution was adjusted for our age criterion, 1.87% of the population was under 35, 20.78% was between 35 and 45, and 48.9% was between 45 and 55 years old. These results are nearly identical to the graphical depiction of our study's age distribution. This finding was consistent with prior research suggesting that Indians experience CAD and CAD-related mortality at a younger age. [14] Iyanger et al [19] also observed mean age in their study to be 49 years which is consistent with our study.

Analysis of the gender distribution revealed that the gender of a man is a statistically significant risk factor ($p<0.01$). Men made up 78% of cases and 51% of controls, whereas women made up just 22% of cases and 49% of controls. M:F ratio in these situations was 3.5:1. 64% of the sample as a whole was male, and the study's M:F ratio was 1.81:1. It is consistent with what Gajanan D. al [3] found. There were 67.6% males in their study, and the M:F ratio was 2.09:1. In a comprehensive study conducted in Andhra Pradesh by Sriharibabu et al. (2013), it was 1.91:1.

Male gender has been shown to be one of the most reliable and well-documented risk factors for coronary atherosclerosis. Previous epidemiologic investigations have revealed the clear preventive benefits of estrogens in avoiding atherosclerosis. [2] The male to female ratio in a study [3] that examined young patients with acute MI was 20:1, whereas the Sricharan et al. study1 found that ratio to be 9:1. Still, the pattern is the same. Similar to our work, Iyanger et al. [19] found that the M:F ratio in their investigation was 2.12:1, indicating a clear male preponderance.

The risk factor for the presence of hypertension was statistically significant ($p<0.01$). Of the cases, 73% had known hypertension and were all receiving therapy. In addition, smoking, diabetes, and hypertension were found to be the most common risk factors in all CAD cases by Sriharibabu et al [13]. The study conducted by Gajanan D. et al [3] produced similar results. In India, the prevalence of hypertension varies from 12% to 17% in rural areas and from 20% to 40% in metropolitan areas. [25] In India, 28.9% of the population has an increased risk of acute myocardial infarction as a result of hypertension. [16] Additionally, Bhasin et al. found that 33% of the study population had hypertension, which nearly half the percentage is found in our investigation. It can be explained by patients coming to our hospital are for hypertension follow-up.

On statistical significance testing weight, HDL, and VLDL were not found to be significant while BMI, HbA1c, LDL, triglyceride and total cholesterol were found to be significantly associated with CAD causation.

The risk factor of diabetes mellitus was statistically significant ($p<0.01$). In all, 50% of research participants had diabetes. Just 24% of controls had diabetes, compared to 76% of cases. This demonstrates that diabetes does not cause CAD on its own. Diabetes has been shown in previous research to be a substantial risk factor for the genesis of CAD.4. [23,26,32,29] This makes sense in light of our research. Diabetes prevalence was reported as 44% and 48.5%, respectively, by Iyanger et al. [19] and Gajanan D. et al. (2003). In the Chennai Urban Population Study (CUPS), the prevalence rates of type 2 diabetes were 21.4% and 9.1%, respectively, among persons without coronary artery disease. In the Inter Heart Study, the diabetes-related attributable risk for myocardial infarction was 20.5%. [16]

In our investigation, the risk factor HbA1c was statistically very significant ($p<0.01$). With a mean of 7.58, HbA1c was abnormal in 69% of cases compared to 20% of controls. Test values more than 6.5 indicate that it is disturbed; 44% of the sample as a whole had deranged HbA1c levels. The results align with the study conducted by Bhasinet al. (2017). Upon statistical analysis of HbA1c as a risk factor across five angiographic categories, it was determined to be very significant ($p<0.01$). According to research by Ravipati et al [8], diabetics' HbA1c level dramatically rose as the number of arteries impacted by CAD grew.

The severity and advancement of coronary atherosclerosis were found to be correlated with

fasting blood glucose, HbA1c, and the existence of diabetes, according to Berry et al [5]. They came to the conclusion that in patients with abnormal glucose tolerance or diabetes, improved glycemic control positively improves CAD. The results of Bhattacharya et al. [6] are in line with the current investigation, which identifies HbA1c as a separate risk for CAD severity. Gong [9] investigated the relationship between glycemic fluctuation and CAD severity and presence.

Our results imply that when traditional risk variables were assessed in patients with coronary artery disease, diabetes showed up as an independent predictor of the severity of obstructive CAD. Diabetes has repeatedly been found to be an independent predictor of severity or disease progression in previous studies of the angiographic progression of CAD employing serial quantitative angiography to determine predictors of severity or progression of disease burden, including CASS registry [10]. Compared to people without diabetes, diabetic patients have more severe and widespread atherosclerosis in their coronary arteries. [9]

When the lipid profile of our study was compared to that of Bhasin et al. (2017) and Gajanan et al. (2003), it was discovered that although HDL and VLDL were variable, triglycerides, LDL, and total cholesterol were significantly higher in our study as well. All of these studies, however, as well as the one we conducted, demonstrate statistically significant trends and a relationship between dyslipidemia and elevated total cholesterol, elevated LDL, elevated triglycerides, and decreased HDL. Nonetheless, a very variable inter-study range in mean values was observed. Of the samples, 62.7% had abnormal lipid profiles, and 41% had known dyslipidemias. Bhasinet al. (2017) reported similar results, with 70.9% of patients having dyslipidemia.

Dyslipidemia was found in 37.83% of the patients in the Pathaket al4 analysis, which is similar to the 36.3% of patients in the Kaul et al [10] study and the 41% of patients in our investigation. Dyslipidemia was found to be a statistically highly significant ($p<0.01$) risk factor. These findings suggest that lipid metabolic disorders are a major factor in the development of CAD in Indians.

In our study, the presence of a deranged lipid profile as a risk factor was statistically significant ($p<0.05$). The lipid profile test revealed abnormalities in 58% of controls and 78% of patients, which is in line with Dhadwadet al.'s findings. In the postprandial state, hypertriglyceridemia (TG >200 mg%) was present in 68% of patients, compared to 11% of patients who had hypertriglyceridemia during the fasting state. Low fasting plasma HDL cholesterol concentration and the size of the postprandial TG rise are significantly correlated, according to Iyanger et al. research [19].

A statistical analysis of triglycerides as a risk factor in five angiographic categories revealed that they are very significant ($p<0.01$), indicating a direct correlation between increasing triglycerides and the severity of coronary artery disease. With a mean triglyceride level of 257 mg%. 50% more TG rise in CAD was demonstrated by Axelson et al. theory that the rise in lipoprotein is intestinal in origin is supported by the results of the ARIC study, which included a sizable sample of both men and women. In the Dharwad et al trial, 68% of patients had hypertriglyceridemia (TG >200 mg%) in their postprandial condition, compared to 22% of patients who had hypertriglyceridemia during the fasting state. [21] These results are in line with what we investigated.

Low HDL was not found to be significant ($p>0.05$). This agrees with the inverse relation between risk of CAD and HDL level. In the PROCAM study 45% subjects who developed CHD had HDL cholesterol less than 30 mg %. [11]

Similar to the Dharwad et al. study, LDL was shown to be significantly significant ($p<0.01$) with a mean of 165 mg%. With a mean of 33%, VLDL was non-significant ($p>0.05$) and inconsistent with the research of Sekhri et al. [21] The statistical significance of hypothyroidism was not found ($p>0.05$). The lack of evidence linking hypothyroidism to CAD was likely due to the small number of hypothyroid individuals in the study population. Therefore, our research is not strong enough to identify hypothyroidism as a risk factor. The statistical significance of a positive family history of CAD was $p<0.01$, in line with the findings of earlier research by Pathaket al., [6] Kaul et al., and Kaulet al., [18]. Tobacco habit was statistically highly significant ($p<0.01$) as the INTERHEART study also observed that smoking was a greater risk factor in younger men than in women. [16]

With smoking came a progressive increase in the risk of CAD. Other Indian epidemiological studies, such the Sriharibabuetal study, which similarly point to a stronger link between smoking and CAD in younger people,8 support the consistency of our findings. [13] Krishnan et al. 20 found that 50.3% of people had smoked at some point in their lives, which is similar to our cases data of 53%.

BMI as a risk factor was statistically significant ($p<0.05$). This finding was like the study by Gajanan D. et al [3] and Bhattacharyya et al. [6]

This research is not blinded or matched. Enrollment involves people from a single hospital only. There was no statistical comparison with other studies or comparison with the reference population, which

raises the possibility of bias and correlation due to chance or selection.

Additionally, individuals with MI or a history of CAD, PCI, or CABG were not included in the trial. Only patients who had their lipid profiles and HbA1c evaluated were included.

Lower blood pressure, HbA1c, and lipids may have been a consequence of statin, OHA, and antihypertensive medication treatment for the CAD group. This could account for the lack of correlation between blood pressure, HbA1c, triglycerides, and lipids and CAD.

This study suggests that early intervention, particularly in the presence of other risk factors, can be beneficial in controlling levels of triglycerides and lipids with tighter glycemic control. In the Indian population, routine lipid profiles and HbA1c tests are necessary to identify abnormal parameters and take prompt action. To correlate the impact of different combinations of elements existing at a time, multifactorial analysis is necessary.

In addition, comprehensive multicentric investigations that represent the general population are required to determine the angiographic severity of CAD. It will take both complicated statistical analysis to evaluate multi- and inter-factorial analyses and prospective longitudinal follow-up studies to shed light on the true risk factors for various cardiovascular end goals, including death.

Conclusion

It is clear from this study that there are multiple factors that contribute to the severity and cause of CAD. The most important factors that have been linked to the severity of CAD and as independent risk factors for its causation include tobacco and alcohol use, high HbA1C, high LDL, high triglycerides, high total cholesterol, dyslipidemia, diabetes, and family history.

In the research sample, dyslipidemia was common; their causes were not understood. The most prevalent lipid profile abnormalities were low HDL and hypertriglyceridemia. Additionally, multifactorial causation of CAD is demonstrated by our study, with a higher number of risk variables in cases and a lower number in controls.

It has been demonstrated that HbA1c, LDL, triglycerides, and total cholesterol function as separate risk factors for disease severity.

References

1. Harvard Men's Health Watch, Premature heart disease, Nov 2009, https:// www. health. harvard.edu/heart-health/premature-heart-disease.
2. Gregory A. Roth, MD, MPH; Mark D. Huffman, MD, et al Global and Regional Patterns in Cardiovascular Mortality From 1990 to 2013 Circulation. 2015; 132:1667-1678.
3. Viswanathan Mohan, DSca Raj Deepa, Subramaniam Shanthi, et al Prevalence of coronary artery disease and its relationship to lipids in a selected population in South India: The Chennai Urban Population Study (CUPS No. 5),Journal of the American College of Cardiology, 2001, 3:9:682-687.
4. Young Mi Hong MD Atherosclerotic Cardiovascular Disease Beginning in Childhood Korean Circulation Journal 2010, 40:1: 1–9.
5. Siddharth N. Shah. API Text book of Medicine. 7th Edn. Association of Physicians of India; 2003. P-441.
6. Enas EA. How to Beat the Heart Disease Epidemic among South Asians: A Prevention and Management Guide for Asian Indians and their Doctors. Downers Grove: Advanced Heart Lipid Clinic USA; 2011.
7. Ridker PM, Brown NJ, Vaughan DE, Harrison DG, Mehta JL. Established and emerging plasma biomarkers in the prediction of first atherothrombotic events. Circulation. 2004; 109(suppl I):IV-6–IV-19.
8. Yusuf S, Hawken S, Ounpuu S, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet. Sep 11 2004; 364:9438:937-952.
9. Enas EA. How to Beat the Heart Disease Epidemic among South Asians: A Prevention and Management Guide for Asian Indians and their Doctors. Downers Grove: Advanced Heart Lipid Clinic USA; 2010.
10. Vasan RS. Biomarkers of cardiovascular disease: molecular basis and practical considerations. Circulation. 2006; 113:2335–2362.
11. Davis JE, McDonald JM Jarett L A high-performance liquid chromatography method for hemoglobin A1c.Diabetes. 1978, 27:2:102-7.
12. Justin D Pearlman, MD, ME, PhD, FACC, MA; Chief Editor: Eugene C Lin, MD Imaging in Coronary Artery Disease emedicine. medscape.com/article/349040-overview.
13. Filippo Crea and Giovanna Liuzzo Pathogenesis of Acute Coronary Syndromes Journal of the American College of Cardiology 2013, 61:1:232-34.
14. Amit Kumar, Christopher P Cannon. Mayo Clin Proc, Oct 2009; 84:10: 917–938.
15. David D. McManus, Joel Gore, Jorge Yarzebski, The American Journal of Medicine, January 2011, 124:1:40-47.

16. Floyd KC, Yarzebski J, Spencer FA, et al, A 30-year perspective (1975–2005) into the changing landscape of patients hospitalized with initial acute myocardial infarction: Worcester Heart Attack Study, Circ Cardiovasc. Qual Outcomes, 2009; 2:2:88–95.
17. Thygesen K, Alpert JS et al Third universal definition of myocardial infarction. Circulation. 2012,16:126:16-35.
18. Perski A, Olsson G, Landou C, de Faire U, Theorell T, Hamsten A. Minimum heart rate and coronary atherosclerosis: independent relations to global severity and rate of progression of angiographic lesions in men with myocardial infarction at a young age. Am Heart J. 1992; 123:609–16.
19. A.F. Person and C. Patterson, Therapeutic options for premature coronary artery disease, Curr Treat Options Cardiovasc Med. 2008, 10:294–303.
20. Singh N, Gupta M, Clinical characteristics of South Asian patients hospitalized with heart failure, Ethn Dis, 2005; 15: 615–619.
21. Palaniappan L, Wang Y, Fortmann SP, Coronary heart disease mortality for six ethnic groups in California, 1990–2000, Ann Epidemiol, 2004; 14:499–506.