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Coronary atherosclerosis in relation to body fatness and its distribution

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Summary

In a cross-sectional study of 226 patients (160 men and 66 women) who underwent coronary angiography, the relationships between total body fatness and abdominal fat distribution, and angiographically assessed coronary artery disease (CAD) were examined. Two different scoring systems were used to quantify the degree of CAD: an 'extent score' and a 'myocardial score'. The extent score provides an estimate of the extent of coronary atherosclerosis and the myocardial score provides an assessment of the amount of myocardium threatened by coronary lesions. Total body fatness was estimated using the body mass index (BMI) and the waist-to-hip circumference ratio (WHR) was used to assess abdominal fat distribution. The weight and height were obtained by questionnaire at the time of angiography, and self-reported waist and hip circumference measurements were used to calculate WHR. The BMI and WHR were associated with several coronary heart disease (CHD) risk factors. However, BMI was not significantly associated with either of the CAD scores. The WHR was positively associated with both the extent score ($r_s = 0.18$; $P < 0.05$) and the myocardial score ($r_s = 0.17$; $P < 0.05$) for men and women together, and positively associated with the myocardial score for women aged 40 to 70 years ($r_s = 0.32$; $P < 0.05$). The associations between WHR and the CAD scores were not significant after adjusting for several risk factors for CHD. These results indicate that other risk factors for CHD may be involved in the associations between WHR and CAD.

Keywords: obesity, body mass index, body fat distribution, waist-to-hip ratio, angiography, coronary atherosclerosis, coronary artery disease

Introduction

Prospective studies have shown a positive association between body fatness, which is often estimated using body mass index (BMI), and CHD incidence.[1–3] The BMI has also been positively associated with both severe coronary artery stenosis, assessed angiographically, and incident myocardial infarction.[4] These results suggest that obesity is related to coronary events through atherosclerosis. However, an angiographic study[5] and several autopsy studies[6–8] have failed to find a positive association between total body fatness and atherosclerosis.

The relationship between body fat distribution and CHD incidence has also been examined in several prospective studies.[9–13] These studies have found that abdominal obesity, which can be estimated using waist-to-hip circumference ratio (WHR), is positively associated with CHD incidence. Angiographic studies also provide evidence for a relationship between WHR and CAD.[5,14] However, data from autopsy studies on the relationship between abdominal obesity and either CAD or atherosclerosis are lacking.

In the present study, two different measurements of the degree of CAD were used, one of which provides an estimate of the extent of coronary atherosclerosis. Although the available evidence is suggestive of a relationship between WHR and coronary atherosclerosis,[5,14] an endpoint which provides an estimate of the extent of coronary atherosclerosis has not been used previously. The relationship between BMI and coronary atherosclerosis is also not clear. The purpose of this study was to examine the relationships between both BMI and WHR and the degree of CAD assessed angiographically.

Methods

Patient sample

All patients underwent coronary angiography. The patients were on the routine cardiac catheterization list for investigation of chest pain thought to be due to either CAD (96%), valvular heart disease (3%), or both (1%). Consecutive patients, totalling 226 (160 males and 66 females), were enrolled.

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Table 1 Patient characteristics

Characteristic	*Total (n = 226) (%)*	*Men (n = 160) (%)*	*Women (n = 66) (%)*
Angina	97	98	95
Previous myocardial infarction	36	39	27
Valvular heart disease	3.8	2.0	8.2
Aspirin use	62	67	49
Nitrate use	54	58	44
Calcium antagonist use	46	48	42
Beta-blocker use	41	41	39
ACE* inhibitor use	19	16	29
History of hypertension	47	41	63
Smokers	13	14	11
Ex-smokers	58	58	58
Never smoked	29	28	31
Diabetes mellitus	8.8	8.7	9.1

* *angiotensin-converting enzyme.*

Assessment

The patient characteristics are presented in Table 1. Clinical details such as age, diabetic status and smoking history were gathered using a questionnaire administered at the time of angiography. Data on history of angina, myocardial infarction, valvular heart disease, hypertension and medication use were collected from medical records. To obtain a continuous variable for cigarette smoking, the number of cigarettes smoked per day was multiplied by the duration of time that the patient had smoked, to give an estimate of total number of cigarettes smoked. Coronary angiography was performed according to the Judkins technique and recorded on 35 mm ciné film. Two different scoring systems were used to quantify the degree of CAD: an 'extent score'[15] and a 'myocardial score'.[16]

Angiographic scores

Extent score. An angiographic scoring system which was designed to reflect the proportion of the coronary endothelial surface area affected by atheroma has been developed as an estimate of the extent of coronary atherosclerosis.[15] The proportion of the coronary arterial tree with detectable atheroma, identified as luminal irregularity, was scored out of a maximum of ten. A score of zero indicates that no coronary atheroma was detected, and a score of ten means that 100% of the coronary arteries visualized showed detectable atheroma.

Myocardial score. A myocardial scoring system which takes into account the degree of stenosis of any number of arterial branches, and their relative importance in terms of the amount of myocardium supplied, has been developed.[16] This scoring system takes into account severity as well as location of coronary lesions. A score of zero to 15 can be given.

Body mass index and waist-to-hip circumference ratio

The body mass index (BMI) was calculated by dividing weight in kilograms by the square of height in metres. The weight and height were obtained by questionnaire, which was administered by nursing staff at the time of angiography. The waist-to-hip circumference ratio (WHR) was calculated by dividing the waist circumference, taken at the level of the umbilicus, by the hip circumference, measured as the maximum gluteal diameter. Self-reported waist and hip circumference measurements were used. To obtain these measurements, a questionnaire, together with a tape measure, and instructions and diagrams indicating the correct technique to use when taking these measurements, were sent to each patient.

Lipid and apolipoprotein measurements

Blood was drawn from the femoral artery immediately prior to cardiac catheterization and placed into evacuated glass tubes. The blood was untreated and was allowed to clot. Serum and plasma were separated by centrifugation. Total cholesterol, triglycerides and high density lipoprotein (HDL) cholesterol were measured in fresh serum. Total cholesterol and triglycerides were measured enzymatically with commercial kits (kits 13225 and 22203, respectively;

Table 2 Descriptive statistics for the study population

Characteristic	*Total*		*Men*		*Women*	
	n	*mean ± s.d.*	*n*	*mean ± s.d.*	*n*	*mean ± s.d.*
Extent score (out of 10)	225	4.1 ± 2.1	159	4.3 ± 1.9	66	3.4 ± 2.3
Myocardial score (out of 15)	225	7.3 ± 3.8	159	7.7 ± 3.6	66	6.4 ± 4.3
Age (years)	225	59 ± 11	159	59 ± 11	66	61 ± 10
Height (m)	189	1.68 ± 0.096	132	1.72 ± 0.072	57	1.59 ± 0.070
Weight (kg)	193	72.5 ± 10.0	134	76.5 ± 10.0	59	63.5 ± 11.0
Body mass index (kg/m^2)	189	25.6 ± 3.20	132	25.7 ± 2.59	57	25.2 ± 4.30
Waist-to-hip ratio	158	0.95 ± 0.067	108	0.96 ± 0.045	50	0.92 ± 0.091
Total cholesterol (mmol/l)	226	5.9 ± 1.0	159	5.8 ± 0.93	66	6.4 ± 1.1
LDL cholesterol (mmol/l)	221	4.1 ± 0.92	154	4.0 ± 0.86	66	4.4 ± 1.0
HDL cholesterol (mmol/l)	222	1.06 ± 0.35	155	0.98 ± 0.28	66	1.25 ± 0.42
Triglycerides (mmol/l)	226	1.7 ± 0.90	159	1.8 ± 0.96	66	1.7 ± 0.77
Apolipoprotein B (mg/l)	221	1.27 ± 0.27	156	1.27 ± 0.25	64	1.27 ± 0.30
Apolipoprotein A-1 (mg/l)	221	1.14 ± 0.23	156	1.09 ± 0.19	64	1.27 ± 0.28
Number of cigarettes smoked in lifetime*	186	623 ± 739	129	741 ± 760	57	344 ± 606

* *Cigarettes smoked during the smoking years: includes smokers, ex-smokers and non-smokers.*

Trace Scientific Pty Ltd, Clayton, Victoria, Australia). High density lipoprotein cholesterol was measured enzymatically as for total cholesterol following the precipitation of apolipoprotein B-containing lipoproteins using equal volumes of 20% polyethylene glycol 6000 and serum. Low density lipoprotein (LDL) cholesterol was derived by calculation using the Friedewald formula adapted to SI units.[17] Cholesterol and triglyceride measurements were performed on a KONE Progress selective chemistry analyser (KONE Instruments Corporation, Espoo, Finland).

Apolipoproteins A-1 and B were measured in plasma using a nephelometric method, on the Behring Nephelometer (Behring Werke, Marburg, Germany). Antibodies to apolipoproteins A-1 and B, standards, control materials, supplementary reagent, reaction buffer and dilution buffer were obtained from Behring Diagnostics (Australia Pty Ltd, Sydney, Australia). The method for measurement of the apolipoproteins was programmed into the Behring Nephelometer. The sample volume used for apolipoprotein A-1 was 10 μl, and for apolipoprotein B the sample volume was 20 μl. The sample was diluted 1:20 with dilution buffer. For the measurement of apolipoprotein B, a second reagent was also added: 10 μl of supplementary reagent. Apolipoprotein A-1 or apolipoprotein B antibodies (40 μl) were mixed with the sample along with 160 μl of reaction buffer. Measurements of light scatter were then taken after 10 seconds and again after 6 minutes. The concentration of the apolipoprotein was read off a standard curve.

Data analysis

The data analysis package used for all statistical analyses performed was SAS.[18,19] At a univariate level, Spearman's rank correlation coefficient (r_s) was used to determine the degree and direction of association between two variables. To control for covariates, the PARTIAL option was used. The Wilcoxon rank sum test was used to test whether there were differences between two population means.

Results

The descriptive statistics for the study population are presented in Table 2. Sixty-six women and 160 men, aged between 16 and 80 years, were enrolled. The extent score ranged from zero to 8.5, with a mean of 4.3 for men and 3.4 for women. The mean extent score was significantly higher for men ($P < 0.0001$). The myocardial score ranged from zero to 15, with a mean of 7.7 for men and 6.4 for women. The mean myocardial score was also significantly higher for men ($P < 0.0001$). Nineteen patients (8.4%) had no detectable CAD, with both extent and myocardial scores of zero. The degree of correlation between the two scores was high ($r_s = 0.72$; $P < 0.0001$). The frequency distributions of the two CAD scores are presented in Figure 1. The BMI ranged between 20.0 and 33.1 kg/m^2 for men with a mean of 25.7 kg/m^2, and between 16.4 and 34.3 kg/m^2 for women, with a mean of 25.2 kg/m^2. There was no significant difference between the mean BMI for men and women. The WHR ranged from 0.82 to 1.06 for men, with a mean of 0.96, and between 0.76 and 1.17 for women, with a mean of 0.92. The mean WHR was significantly higher for men than for women ($P < 0.0001$).

Correlations between BMI and WHR and risk factors for CHD are presented in Table 3. Several significant correlations were found. For men, BMI was positively associated with total cholesterol, apolipoprotein B and triglycerides, and inversely associated with age. For women, BMI was

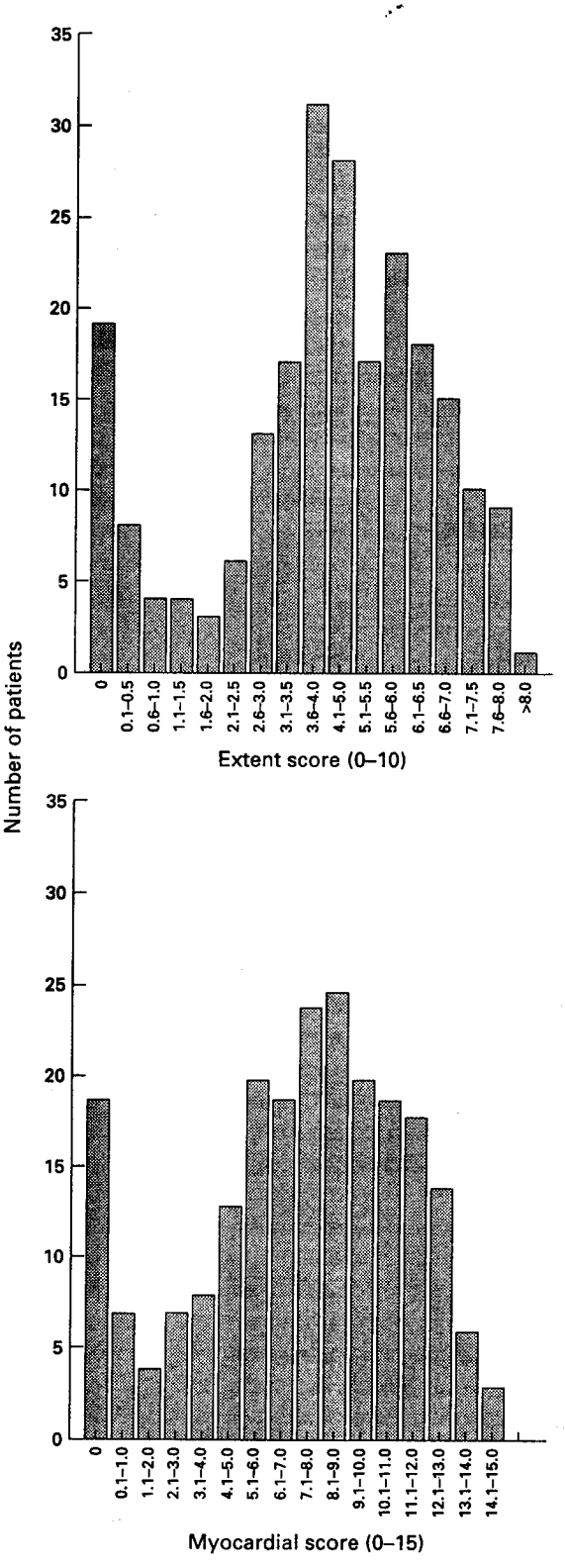

Figure 1 Frequency distribution, in number of patients, of the extent score and the myocardial score.

Table 3 Correlations (r_s) between BMI, WHR and CHD risk factors

	BMI			*WHR*		
Risk factors	*Total*	*Men*	*Women*	*Total*	*Men*	*Women*
Age	−0.18*	−0.19*	−0.17	0.10	0.04	0.27*
Cigarette smoking	0.09	−0.02	0.11	0.12	0.12	−0.13
Total cholesterol	0.14*	0.21*	0.12	−0.06	0.04	0.00
LDL cholesterol	0.09	0.14	0.06	−0.06	0.02	−0.01
HDL cholesterol	−0.21**	−0.17	−0.19	−0.22**	−0.05	−0.14
Apolipoprotein B	0.22**	0.30***	0.08	−0.07	0.02	−0.25
Apolipoprotein A-1	−0.10	−0.03	−0.10	−0.19*	0.06	−0.22
Trigycerides	0.32†	0.33†	0.31*	0.07	0.10	−0.04
WHR	0.17*	0.24*	0.07			

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$; † $P < 0.0001$.

Table 4 Correlations (r_s) between CAD scores and BMI, WHR and CHD risk factors

	Extent score			*Myocardial score*		
Risk factors	*Total*	*Men*	*Women*	*Total*	*Men*	*Women*
Age	0.29†	0.29***	0.39***	0.40†	0.42†	0.41***
Cigarette smoking	0.19*	0.09	0.29*	0.23**	0.21*	0.18
Total cholesterol	0.00	0.03	0.13	0.02	0.05	0.10
LDL cholesterol	0.07	0.10	0.15	0.09	0.11	0.13
HDL cholesterol	−0.13*	−0.05	−0.14	−0.16*	−0.05	−0.24*
Triglycerides	0.03	−0.03	0.19	0.10	0.03	0.27*
Apolipoprotein B	0.09	0.03	0.24	0.10	0.05	0.21
Apolipoprotein A-1	−0.13*	−0.04	−0.17	−0.15*	−0.05	−0.22
BMI	−0.03	−0.16	0.12	−0.05	−0.01	0.12
WHR	0.18*	0.12	0.08	0.17*	0.10	0.16

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$; † $P < 0.0001$.

positively associated with triglycerides. For the total population, BMI was positively associated with total cholesterol, apolipoprotein B and triglycerides, and inversely associated with age and HDL cholesterol. The WHR was positively associated with age for women, and inversely associated with HDL cholesterol and apolipoprotein A-1 for the total population. The BMI and WHR were not strongly correlated. There was a significant positive association for men, but not for women. The relationships between diabetes and hypertension, as well as use of the various medication types, and BMI and WHR were also determined. Nitrate use was associated with a significantly higher WHR for men. Calcium antagonist use was associated with a significantly lower WHR, and beta-blocker use was associated with a significantly higher BMI for women. No other significant associations were found.

Table 4 presents correlations between both BMI and WHR and the CAD scores. The BMI was not significantly associated with either CAD score. The WHR was positively associated with both the extent score and the myocardial score for the total population. However, when the study population was separated into men and women, significance was not reached. Table 4 also presents correlations between several CHD risk factors and the CAD scores.

The relationships between WHR and the CAD scores were examined further by adjusting for covariates. These results are presented in Table 5. The nature of associations between WHR and either the extent score or the myocardial score was modified when several established risk factors for CHD were considered. The WHR was not significantly associated with the extent score after adjusting for apolipoprotein A-1 or several covariates combined. After adjusting for either age, BMI, cigarette smoking, HDL cholesterol or apolipoprotein A-1, or several covariates combined, the association between WHR and the myocardial score was not significant.

The associations between WHR and the CAD scores for patients aged from 40 to 70 years were also examined. For men and women together, WHR was positively associated

Table 5 Correlations (r_s) between the CAD scores and WHR after adjusting for CHD risk factors (total population)

	Extent score	*Myocardial score*
WHR adjusting for the following risk factors individually		
Age	0.16*	0.15
BMI	0.18*	0.12
Cigarette smoking	0.17*	0.13
Total cholesterol	0.18*	0.18*
LDL cholesterol	0.19*	0.19*
HDL cholesterol	0.16*	0.14
Apolipoprotein B	0.19*	0.19*
Apolipoprotein A-1	0.15	0.15
Triglycerides	0.17*	0.17*
All the above CHD risk factors combined	0.17	0.05

* $P < 0.05$.

with both the extent score (r_s = 0.19; $P < 0.05$) and the myocardial score (r_s = 0.23; $P < 0.05$). After adjusting several other variables, including age, cigarette smoking, lipid and apolipoprotein measurements and BMI, WHR was not significantly associated with either CAD score (r_s = 0.19; $P > 0.05$ (extent score), r_s = 0.13; $P > 0.05$ (myocardial score)). The WHR was also positively associated with the myocardial score for women (r_s = 0.32; $P < 0.05$). After adjusting for covariates, this association was not significant (r_s = 0.27; $P > 0.05$). No other associations reached significance.

Discussion

The myocardial score takes into account both severity and location of coronary lesions, and provides an estimate of the amount of myocardium threatened by lesions. The extent score is an estimate of the aggregate degree of atherosclerosis in the coronary arteries. The two CAD scores were significantly associated, but the degree of association suggests that different aspects of CAD are being assessed. The CAD scores were not normally distributed (Figure 1). Non-parametric statistics were therefore used for the analyses.

Self-reported waist and hip circumference measurements were used for the calculation of WHR. Self-reporting of waist and hip circumference measurements has been used previously, and it was found that self-reported measurements were highly correlated with measurements taken on the same individuals by technicians.[20,21] Self-reporting of waist and hip circumference measurements would therefore seem to be a valid method for obtaining these measurements. However, the error is likely to be greater than for measurements taken by a technician.

The associations between BMI and WHR were weak, or not significant, depending on gender. This suggests that total body fatness is not a strong determinant of an abdominal fat distribution. The relatively weak associations between BMI and WHR may have been due in part to the highly selected patient population and to the increased error involved in self-reporting of the variables used to calculate BMI and WHR. Several significant correlations between either BMI or WHR and CHD risk factors were observed. These associations may offer explanations for relationships between body fatness and its distribution and CAD.

Prospective studies have found a positive association between BMI and CHD incidence.[1–3] However, whether BMI is associated with CHD incidence through atherosclerosis or through other processes which influence CHD events is unclear. Although one study has produced results which suggest that obesity is linked to CHD events through atherosclerosis,[4] a number of other studies have failed to find an association between BMI and either angiographically-assessed CAD[5] or atherosclerosis at autopsy.[6–8] The BMI was not significantly associated with either of the two CAD scores in the present study. Also, no significant associations were found between BMI and CAD when men and women were analysed separately. The CAD scores used in this study relate more to atherosclerosis, and the extent score in particular was developed as a measure of coronary atherosclerosis. Although CAD and CHD events are linked, particular risk factors, such as BMI, may be related to processes which influence CHD events, rather than atherosclerosis. Much of the evidence presently available indicates that total body fatness is not an important direct determinant of coronary atherosclerosis.[5–8] The results from this study support this suggestion.

Abdominal fat distribution has been positively associated with CHD incidence, independently of BMI, in several studies.[9–13] Abdominal fat distribution has also been associated with various risk factors for atherosclerosis and CHD.[22–24] In the present study, the WHR was inversely associated with HDL cholesterol and apolipoprotein A-1 in men, and positively associated with age in women. An association between WHR and angiographically-assessed CAD has been observed previously in women but not in men in a study which included both men and women,[14] and in men in a study which included only men.[5] Both of these studies had a case-control type design where those with and without significant CAD were compared. They provided the opportunity to assess the relationships between WHR and presence of significant CAD, which is presumably an assessment of coronary atherosclerosis. The present study provided the opportunity to assess the relationships between WHR and measurements of the degree of CAD, including an estimate of the extent of coronary atherosclerosis. The WHR was positively associated with both CAD scores for the total population. The positive association between WHR and the extent score is suggestive of a relationship between WHR and coronary atherosclerosis. However, WHR was not associated with either CAD score for men and women analysed separately. These results indicate that the positive association between WHR and CAD, observed for the total population, was due, at least in part, to gender differences. There were two gender differences which may have contributed to this association. Firstly, despite the fact that over 95% of both men and women involved in this study were thought to have CAD prior to angiographic assessment of CAD, men had significantly more CAD than women. Secondly, there were gender differences in the distribution of fat. Men had a significantly higher WHR than women, indicating higher levels of abdominal fatness in men.

Apart from gender differences, relationships between abdominal fatness and other risk factors for CHD might also have contributed to the positive associations between WHR and CAD for men and women together. For example, after adjusting for apolipoprotein A-1, which was inversely associated with WHR, the associations between WHR and both the extent and myocardial scores were no longer significant (Table 5). Several factors, including gender, may be related to the observed positive associations between WHR and CAD. The associations between WHR and CHD risk factors indicate possible pathways for the associations between WHR and CAD.

The age of patients involved in this study ranged from 16 to 80 years. However, most of the patients (80%) were aged from 40 to 70 years. In patients less than 40 years of age, a strong genetic component for CAD would be suspected. Coronary artery disease in those aged 40 to 70 years may still be regarded as premature. Within this age group, WHR

was positively associated with both CAD scores for men and women together, and positively associated with the myocardial score for women. None of these associations were significant after adjusting for other risk factors for CHD. The positive association between WHR and the myocardial score for women in the 40 to 70 years age group, which was not present for all women, indicates that WHR is a stronger risk factor within this patient population for women from 40 to 70 years than for the total age range.

In conclusion, it was found that BMI was not associated with CAD. This is consistent with several earlier studies which suggest that total body fatness may not directly influence the process of atherosclerosis.[5–8] The WHR was positively associated with CAD for men and women together, and positively associated with the myocardial score for women aged 40 to 70 years. These associations could be explained by several risk factors for CHD, indicating that other risk factors for CHD may mediate the associations between WHR and CAD.

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