Waist Girth Does Not Predict Metabolic Complications in Severely Obese Men

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The epidemic of obesity has received considerable attention because of its increasing prevalence and its deleterious impact on health (1–6). In this regard, the metabolic syndrome has been recognized as a prevalent cause of cardiovascular disease, and the National Cholesterol Education Program Adult Treatment Panel III guidelines (7) have proposed clinical tools for the identification of individuals characterized by this syndrome. However, there is considerable metabolic heterogeneity among equally overweight/obese individuals. While some patients show a relatively "normal" metabolic risk profile despite being obese, others who are moderately overweight can nevertheless be characterized by metabolic complications (8,9).

Thus, it is not uncommon to find severely obese patients with minimal changes in their metabolic risk profile, suggesting that they may be at lower cardiovascular disease risk than what could be expected from their massive obesity. Therefore, the aim of the present study was to examine the relationships between selected features of the metabolic syndrome and waist circumference as a crude marker of abdominal obesity in moderately and severely obese men.

RESEARCH DESIGN AND METHODS

— Informed written consent was obtained from all patients, and studies were conducted according to the Helsinki Declaration. The reference group consisted of moderately obese men (n = 97) of the Québec Health Survey (10,11) with BMI values between 30 and 40 kg/m^2. A group of 84 men with BMI ≥40 kg/m^2 underwent biliopancreatic diversion for severe obesity. Patients with hepatotoxic medication or exposure, history of hepatitis, prior weight loss surgery, or elevated consumption of alcohol were excluded from the present study.

For moderately obese men, anthropometric measurements were obtained according to standardized procedures. A fasting lipid profile was performed at the St. Michael's Lipid Research Laboratory in Toronto. Fasting glycemia was determined using the glucose oxidase assay.

Severely obese patients were weighed either on a detectomedical or on an electronic Toledo scale (weight < or ≥136-kg). Waist girth was measured at the widest circumference of the umbilicus level. The fasting lipid profile and glycemia were determined according to routine methods of the Laval Hospital.

In moderately obese men, blood pressure was recorded with an appropriate cuff size but with a large cuff in severely obese men.

Pearson correlation coefficients were calculated to quantify the univariate associations among variables. Analyses were performed with SAS.

RESULTS

— The prevalence of the metabolic syndrome was similar in moderately and severely obese men (29 and 32%, respectively). Although characterized by greater adiposity indexes, severely obese men displayed a better plasma lipoprotein-lipid profile than moderately obese men. Plasma cholesterol and LDL cholesterol concentrations were lower in severely obese men despite a comparable use of hypolipidemic medication in the two groups. However, systolic and diastolic blood pressures were higher in men with severe obesity ($P \leq 0.02$), although a greater proportion of severely obese men (27.4 vs. 13.7%, $P \leq 0.02$) was on antihypertensive medication. Significant relationships between waist circumference and HDL cholesterol concentrations ($r = -0.33$, $P \leq 0.001$) or the total-to-HDL cholesterol ratio ($r = 0.21$, $P \leq 0.04$) were only found in moderately obese men (Fig. 1). A positive association was found between waist girth and systolic blood pressure only in the severely obese group ($r = 0.27$, $P \leq 0.02$).

CONCLUSIONS

— The prevalence of the metabolic syndrome among severely obese men is not as high as expected from their large adipose mass. This finding corroborates previous findings observed in morbidly obese individuals (12). In patients with elevated BMI values (34–77 kg/m^2), no relationship was found between obesity and lipid levels (13). The prevalence of dyslipidemia and of metabolic syndrome also appears to be independent of BMI in patients displaying a high degree of obesity (14). Finally, a low prevalence of the metabolic syndrome has been reported in severely obese pre- and postmenopausal women (15).

A possible explanation for the low prevalence of the metabolic syndrome among severely obese men could be attributable to inherent characteristics of visceral versus subcutaneous adipose depots. A high lipolytic activity of visceral adipocytes drained by the portal vein has already been proposed to be an important factor linking intra-abdominal adiposity to metabolic complications (16,17). One could therefore suppose that severely obese men were not characterized by a greater visceral fat accumulation compared with moderately obese men. However, previous studies have shown that two visceral depots (round ligament and mesenteric adipose tissues) were charac-

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A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

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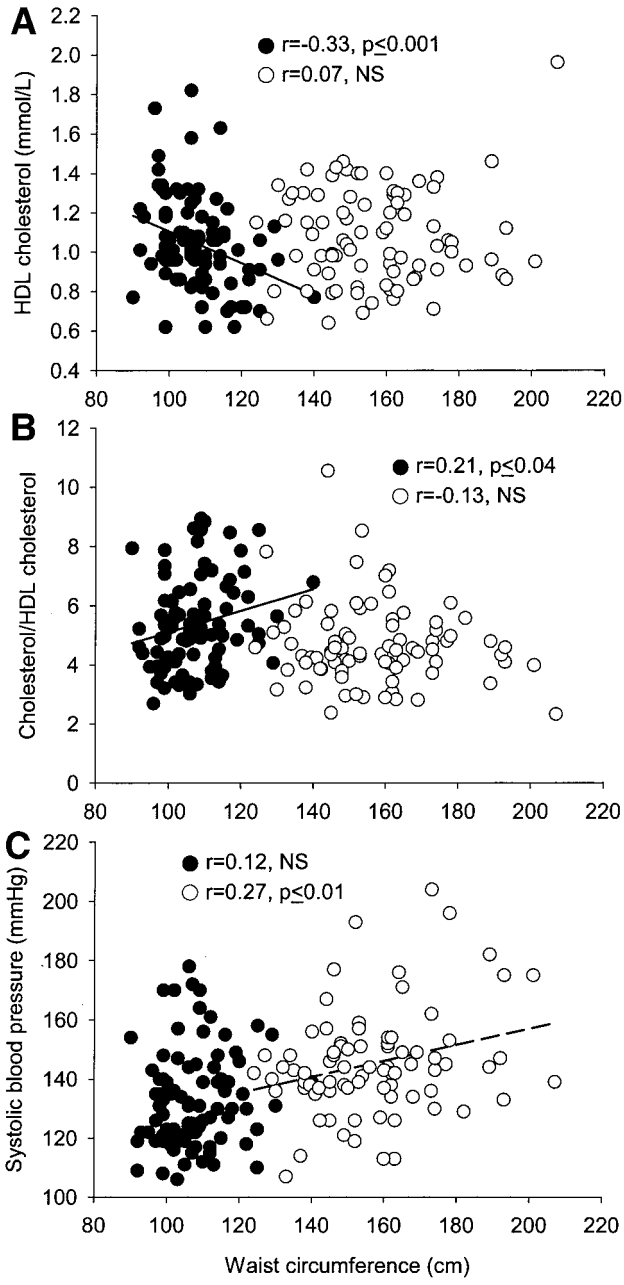

Figure 1—*Relationships of waist circumference to HDL cholesterol levels* (A)*, the total-to-HDL cholesterol ratio* (B)*, and systolic blood pressure* (C) *among moderately* (●) *and severely* (○) *obese men.*

terized by a lower fatty acid release and a greater fatty acid uptake than the omentum in severely obese women, suggesting a protective role of some intra-abdominal fat depots against obesity-related metabolic complications (18–20).

Our findings are also compatible with the notion that localization of excess fat, rather than body fatness, is a health hazard. Among subjects carefully matched for total-body fat but with high versus low visceral adipose tissue, we had previously shown that viscerally obese patients had the most disturbed metabolic risk profile (9,21). Waist circumference is the best correlation of abdominal obesity (22,23), and the higher the waist circumference, generally, the more deteriorated is the metabolic risk profile. However, the lack of relationship between waist girth and the lipid profile in severely obese men suggests that this anthropometric index is not a suitable marker of a deteriorated lipoprotein-lipid profile in severe obesity.

In summary, no difference in the prevalence of the metabolic syndrome was found between moderately and severely obese men. These results provide evidence that massive obesity is a metabolic phenotype distinct from moderate obesity. Further studies are needed to investigate whether differences in metabolic and/or secretory adipose cell capacities of distinct intra-abdominal depots could explain the absence of further deteriorations in severely obese men.

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