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Relation between obesity and myocardial infarction

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Abstract:

Overweight and obesity have become increasingly common; worldwide, at least 1.1 billion adults are overweight and 312 million are obese, when overweight and obesity are defined conventionally as having a body mass index (BMI) of $>25 \text{ kg/m}^2$ and $>30 \text{ kg/m}^2$, respectively.^{1,2} In the general population, overweight and obesity are associated with increased risk of developing cardiovascular disease,^[3,4] and thus it is not surprising that in cohorts of patients with prevalent ischemic heart disease or acute coronary events, well over 50% are overweight or obese.^[5,6] Despite the association between obesity and cardiovascular risk in the general population, a multitude of studies have described an inverse correlation between BMI and mortality in patients with coronary artery disease (CAD), including post-coronary revascularization patients and those with acute myocardial infarction (MI); the association between elevated BMI and improved survival has been termed the obesity paradox. The aim of this study was to evaluate the associations of overweight and obesity with myocardial infarction (MI).

Introduction:

Overweight and obesity are defined by the World Health Organization as abnormal or excessive fat that accumulate and present a risk to health. Obesity is measured in body mass index (BMI), which is a person's weight (in kilograms) divided by the square of his or her height (in meters). A person with a BMI of 30 or more is generally considered obese. A person with a BMI equal to or more than 25 is considered overweight.

Cardiovascular disease (CVD) mortality and morbidity has been shown to be elevated in individuals who are overweight, particularly with central deposition of adipose tissues.⁴ Abdominal obesity has been shown to be a risk factor for CVD worldwide.⁸ Obesity may be associated with hypertension, dyslipidemia, diabetes, or insulin resistance, and elevated levels of fibrinogen and C-reactive protein, all of which increase the risk of CVD events.^[6]

In addition to CVD, obesity has been shown to increase the risk of high blood pressure (HBP). Persistent hypertension is one of the risk factors for stroke, myocardial infarction (MI), heart failure, and arterial aneurysm, and is a leading cause of chronic kidney failure. Moderate elevation of arterial blood pressure leads to shortened life expectancy, which also increases the risk of heart diseases.

Discussion:

myocardial infarction (MI) is a common disease that could lead to high mortality. In 2012, the WHO reported that 7,200,000 people died of ischemic heart disease with AMI as the major contributor.^[7, 8] The AMI is fatal and often occurs in the prime of life, which brings a heavy burden for individuals and families. The risk factors of smoking, cholesterol, diabetes, obesity, left ventricular hypertrophy, and elevated triglycerides are high in AMI. These findings would be meaningful for preventing AMI.

In recent years, obesity and overweight have raised more and more concerns. The incidence of overweight and obesity is increasing and has been reported to be associated with type II diabetes mellitus, metabolic syndrome, cancer, hypertension and cardiovascular disease. But the relation of overweight and obesity with AMI is still controversial. Overweight and obesity are associated with AMI in some studies showed an independent relationship between them.

study in the United States showed that among males younger than 65, BMI had a significant positive association with the CHD risk, but the association was much weaker among older males

aged 65 years or more.^[9] In contrast, another case – control analysis of people aged 30–60 years in India indicated no association between BMI and AMI. A recent report indicated a significant relationship between obesity and coronary atherosclerosis in young male adults, particularly in those with central obesity, and little relationship between them in young female adults.^[9] Still other recent clinical studies have also shown that obesity and its metabolic impairment (metabolic syndrome) are associated with impaired endothelial function and early atherosclerotic change in adolescents. However, there are no data regarding the association of obesity with atherosclerosis and CHD in Japanese young adults. Recently, we showed that young Japanese patients with AMI have a higher BMI than older patients and in the present report, we have demonstrated for the first time that obesity is an independent AMI risk factor in young and middle-aged Japanese males. In the present report, the susceptibility to AMI from the risk of obesity or smoking differed between males and females. A previous study has shown that the relative CHD risk associated with obesity in middle-aged and older females is slightly lower than that for males.^[10] The sex difference in the effect of obesity on CHD might be derived from the pattern of fat distribution. Although at an equivalent BMI, females at all ages have a larger percentage of body fat than male,^[11] males are more prone to have a central (visceral) pattern of fat distribution.^[12] Recent overwhelming evidence has indicated that adverse effects of obesity, such as a variety of metabolic disorders, cardiovascular morbidity, and mortality, are more tightly associated with central rather than peripheral obesity. Another possible explanation is the protective effects of female hormones against CHD progression in the premenopausal woman. In the present study, AMI cases had a significantly greater BMI than control subjects in the older female group, but there was no significant difference in BMI between AMI cases and control subjects among middle-aged females, we cannot rule out the possibility that an increased prevalence of obesity after menopause, together with hypercholesterolemia, hypertension, and diabetes mellitus might also contribute, in part, to the etiology of CHD in older women. Our results have shown that obesity is an independent risk for AMI in young and middle-aged males, not older or very old males. The reason for this age-related variability in the effect of obesity on AMI remains unclear. Older obese men may have a higher prevalence of hypertension or diabetes mellitus than younger obese ones. Thus, in the present study, adjusting for other risk factors might make the AMI risk associated with obesity more difficult to detect in the older men. Another possible explanation is that the increasing prevalence of obesity might make the risk easier to detect in young adults with CHD. The other possible explanation is that metabolic syndrome, accompanied by obesity, particularly visceral obesity, might play a central role in the pathogenesis of AMI in young male adults with a lower incidence of comorbid diseases such as diabetes mellitus and hypertension. Findings from previous reports and our present study support the current emphasis on control of obesity, particularly visceral obesity, to prevent CHD in young adult males.

Conclusion:

The present study provides evidence that obesity is an independent risk for AMI in young and middle-aged males, suggesting that it is necessary to correct obesity for primary prevention of AMI in young male adults. However, the relatively small sample size is a major limitation and a larger study should be performed to confirm our findings

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