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Relationship between obesity and atherosclerosis

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Date of submission: 14\4\2018

Abstract

Obesity related to visceral fat accumulation, is known to increase the risk of various adult diseases, especially cardiovascular disease. This study was conducted to clarify the relationship between obesity and atherosclerosis.

Although several risk factors for heart disease including high blood pressure, diabetes mellitus, lipid and lipoprotein abnormalities are associated with obesity, obesity is not consistently associated with coronary heart disease risk. Some prospective studies of white men (life insurance cohorts, airline pilots, cancer study volunteers, and the Framingham population) have shown a positive linear relationship of weight to coronary heart disease. Other epidemiologic studies show a negative association, no association.[1]

Introduction

Obesity is a medical condition in which excess body fat has accumulated to the extent that it may have a negative effect on health.[2] People are generally considered obese when their body mass index (BMI), a measurement obtained by dividing a person's weight by the square of the person's height, with the range 25–30 kg/m^2 defined as overweight. Some East Asian countries use lower values[3]. Obesity increases the likelihood of various diseases, especially heart disease[4]

Obesity is most commonly caused by a combination of excessive food intake, lack of physical activity, and genetic susceptibility. A few cases are caused primarily by genes, endocrine disorders, medications, or mental illness. The view that obese people eat little yet gain weight due to a slow metabolism is not generally supported. [5]

Atherosclerosis, or hardening of the arteries, is a condition in which plaque builds up inside the arteries. Plaque is made of cholesterol, fatty substances, cellular waste products, calcium and fibrin (a clotting material in the blood).

Atherosclerosis is a type of arteriosclerosis. Arteriosclerosis is a general term for the thickening and hardening of arteries.

Plaque may partially or totally block the blood's flow through an artery in the heart, brain, pelvis, legs, arms or kidneys. Some of the diseases that may develop as a result of atherosclerosis include coronary heart disease, angina (chest pain), carotid artery disease, peripheral artery disease (PAD) and chronic kidney disease.

The introduction sets the scene for the main body of the report. The background knowledge, objectives of the report should be explained, and any necessary background history should be included.[6]

Discussion

The relationship between obesity and atherosclerosis (and coronary heart disease [CHD]) has been a subject of some dispute for many years. Data from early investigations suggested that obesity is not an important contributing cause of coronary atherosclerosis and CHD. For example, results of the Seven Countries Study revealed little correlation between body weight and incidence of CHD. Moreover, in the massive autopsy study called "The Geographic Pathology of Atherosclerosis," edited by Henry C. McGill, Jr" the relationship between body weight and atherosclerosis was weak at best. This study included a detailed examination of arteries from a large number of autopsies carried out in New Orleans, Sao Paulo, Puerto Rico, Lima, and Santiago.[7]

In spite of these earlier negative findings, the Framingham Heart Study in the United States has consistently shown that increasing degrees of obesity are accompanied by greater rates of atherosclerosis. Even so, multivariate analysis of Framingham data strongly suggests that most of the relationship between body weight and atherosclerosis risk is mediated through the standard, major risk factors, i.e., blood pressure, total cholesterol, HDL cholesterol, and diabetes. Their own data led Framingham investigators to question whether obesity is truly an independent risk factor for atherosclerosis. This is not to say that obesity is not a causative risk factor for atherosclerosis, certainly if obesity is a contributing cause of risk factors that are directly atherogenic, then obesity must belong in the chain of causality. In fact, if obesity induces several major risk factors, it could be a more significant cause of atherosclerotic disease than an individual risk factor.[8]

The present issue of *Circulation* contains an article by McGill, in which body fat was correlated with the severity of coronary atherosclerosis in the Pathological Determinants of Atherosclerosis in Youth (PDAY) study. This study included autopsies carried out in approximately 3000 persons aged 15 to 34 years dying of external causes. Gross atherosclerotic lesions were graded in the right coronary artery and in the left anterior descending coronary artery. Adiposity was estimated by body mass index (BMI) and by thickness of the panniculus adiposus. In young men, BMI was positively associated with both fatty streaks and raised atherosclerotic lesions in both coronary arteries. A thick panniculus adiposus was also significantly associated with greater lesions of the right coronary artery when BMI was >30 kg/m^2. Thus, obesity appeared to be a contributor to coronary atherosclerosis in adolescent and young adult men. In young women, BMI was not associated with coronary atherosclerosis, but in those with a thick panniculus adiposus, there was a trend toward greater coronary lesions.[9]

One example of a difference in susceptibility appears to exist between men and women in the United States. As the PDAY study showed, adiposity has a greater effect on atherogenesis in men than in women. It is well known that men develop coronary atherosclerosis more rapidly than women. This difference in atherogenesis rates may be related in part to differences in body fat distribution. Men are more prone to abdominal obesity than are women; and abdominal obesity is widely held to have more impact on risk factors than does gluteofemoral adiposity, which is common in women. Whether abdominal obesity is a direct cause of risk factors or a

reflection of an underlying metabolic abnormality of a more fundamental type is not certain, but the association with cardiovascular risk factors certainly is present.[10]

Conclusions

Two important questions must be asked. First, why did the Framingham Heart Study find a positive correlation between BMI and atherosclerosis risk, whereas no such relationship was found in the Seven Countries Study? And second, why did the PDAY study find a positive correlation between adiposity and coronary atherosclerosis in men, whereas the large Geographic Pathology of Atherosclerosis did not? Regarding both questions, the Seven Countries Study and the Geographic Pathology Study may have included populations in which susceptibility to the adverse effects of obesity may have been too variable to detect a definite relationship. If some populations are less sensitive to the influence of adiposity, then an adverse effect in other populations may have been missed in the analysis. This possibility raises the issue of differences in susceptibility in different groups. This issue is important because it has implications for the approach to the problem of obesity in different populations.

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