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Relation between Central Obesity and Severity of Coronary Artery Disease in Patients Undergoing Coronary Angiography

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Abstract

Coronary heart disease is the most common cause of death in the general population and in patients with ESRD. The principles of cardiovascular risk assessment and management apply to both populations. Advances in noninvasive coronary artery imaging have improved early detection of subclinical disease. Coronary artery disease is a major cause of death and disability in developed countries. Obesity is an independent risk factor for atherosclerotic cardiovascular disease (ASCVD). Increasing abdominal obesity was independently associated with fatal and non-fatal heart attack, regardless of other risk factors (such as smoking, diabetes, hypertension and dyslipidemia) and secondary prevention treatments. Body mass index (BMI) is not accurately reflecting the degree of regional adiposity. Waist circumference; a direct measure of abdominal obesity, has been shown to be a better predictor of coronary artery disease than overall obesity. The abdominal obesity is considered as a part of 'metabolic syndrome' as is linked with conditions like hypertension, diabetes mellitus and dyslipidemia that accelerate atherosclerosis of the arteries and increase the incidence of cardiovascular disease.

Keywords:

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1. Introduction

Obesity is an independent risk factor for cardiovascular disease (CVD) and is associated with advanced CVD requiring procedures such as percutaneous coronary intervention (PCI), reduction in life expectancy, and a higher mortality rate. Weight loss has been associated with improvement in preexisting cardiovascular risk factors including hypertension (HTN), diabetes, and dyslipidemia and mortality [1]. Other studies have reported improved clinical outcomes in overweight and obese patients treated for CVDs compared to normal weight patients, suggesting a paradoxical survival benefit. Obesity was primarily measured using BMI in the studies. The mechanisms leading to this phenomenon, termed "obesity paradox," are unclear [2]. Obesity is a significant component of metabolic syndrome. A greater risk of CAD is associated with metabolically unhealthy people when compared to metabolically healthy subjects. Also, a paradox exists that says obese people with CAD are associated with decreased mortality [3]. Central obesity and insulin resistance are the main disorders that lead to Mets. Mets markedly increased in recent years as a result of the worldwide increase in obesity [4].

Overall obesity measures are less sensitive markers of metabolic and cardiovascular problems than measurements of central or abdominal obesity [5]. The waist circumference reflects both subcutaneous and visceral fat tissue in the abdomen. Obesity's metabolic and cardiovascular consequences are largely determined by visceral adipose tissue [6]. The quantification of coronary artery disease (CAD) severity can be captured using coronary angiography (CA). Historically CAD has been categorized as single, double, and triple vessel and left main disease, with luminal stenosis of either ≥50% (left main) or ≥70% (other major epicardial vessels) used to define significance. Scoring systems to determine the severity of CAD and prognosis were developed to address the perceived limitations associated with stratification of patients with risk level variation [7]. Different assessment modalities have developed to evaluate patients being treated for CAD and predict their outcome following therapeutic intervention. One of the most important scoring systems is the SYNTAX score [8]. SYNTAX score is a visual estimate of CAD burden and complexity.

It takes into account complex lesions including bifurcations, chronic total occlusions, thrombus, calcification, and small diffuse disease. The SYNTAX score can be calculated for all patients using software (http://www.syntaxscore.com/calc/start.htm). A higher SYNTAX score indicates a more complex condition as well as worse prognosis in patients undergoing revascularization especially with PCI [9]. The SYNTAX score is the total of the points allocated to each individual lesion in the coronary tree that has a diameter narrowing of more than 50% in

arteries larger than 1.5mm in diameter. Several various aspects of the lesions are taken into account. After that, a SYNTAX score algorithm adds up all of these features to get Degrees between the main and daughter branches. Aorto-ostial lesions are worth one point, significant vessel tortuosity is worth two points, lesion length longer than 20 mm is worth one point, heavy calcification is worth two points, thrombus is worth one point, and diffuse disease or minor vessel involvement is worth one point per segment [10]. The score varies from 0 to more than 60 in particularly complex coronary anatomical lesions.

Patients are divided into four groups based on their SYNTAX score (SS): (1) no CAD (SS=0); (2) low SS (SS 1–22); (3) intermediate SS (SS 23–32); and (4) high SS (SS>/= 33). [11]. The SYNTAX Score II combines the anatomically based SYNTAX Score with clinical variables that have shown to change the SYNTAX Score's threshold value, achieving equilibrium between CABG and PCI for long term mortality. Unprotected left main coronary artery disease, female gender, age, left ventricular ejection, creatinine clearance, and having chronic obstructive pulmonary disease fraction are among the variables [12]. Few studies have examined the association of body mass index (BMI) and CAD in patients undergoing CA. Parsa, A. F. Z., & Jahanshahi, also found that obese patients referred for CA were younger and had a lower burden for CAD; however, the authors did not find obesity to be a significant predictor for severity of CAD after adjustment for confounders suggesting that younger age may influence the obesity paradox. Others have reported an inverse relationship between BMI and severity of CAD in a cross-sectional, prospective study of 414 patients with suspected CAD [13].Both BMI and waist circumference were strongly associated with risk of CAD.

However, WC predicts CAD risk better than does BMI among men and women 60 years of age and older [14]. A high WC increases the risk of CAD almost threefold independent of the presence of metabolic syndrome. And that abdominal fat distribution (measured by WC) may be more closely linked to metabolic risks than BMI. In the same setting, BMI showed no independent prognostic value on the future development of CAD [15]. Obesity is an accepted risk factor for CAD; therefore, it may be assumed that obese patients have poorer outcomes than nonobese patients. However, published findings contradict this supposition about relationship between BMI and mortality in patients undergoing CA for suspected CAD. The influence of BMI on extent of coronary atherosclerosis and cardiac events in a cohort of patients at risk of CAD was examined by Gregory et al. BMI was not significantly associated with extent of coronary atherosclerosis and mortality [7]. WC as a measure of abdominal obesity provides an indicator of body composition and adds critical information along with BMI. Several organizations and expert panels have recommended that WC measures be assessed along with BMI in clinical evaluations because increasing evidence supports visceral adiposity as a marker of cardiovascular risk [16].

There is a strong correlation between overall obesity and abdominal obesity; however, some individuals may be classified as having overall obesity but not abdominal obesity. The converse may occur as well with abdominal obesity in the absence of overall obesity based on the BMI definition of obesity. The presence of cardio metabolic disease and CVD in those with “normal-weight obesity” leads to under-diagnosis of CVD risk in clinical practice, particularly among patients who have excess fat but not obesity as classified by BMI. Thus, high waist circumference (WC) even in individuals with normal weight may unmask higher CVD risk because WC is an indicator of abdominal body fat, which is associated with cardio metabolic disease and CVD and is predictive of mortality [17]. Even though obesity involves enhanced risk for development of cardiovascular abnormalities, in case of an already developed disease, obesity is associated with a favorable prognosis. The phenomenon known as obesity paradox has observed in case of several cardiovascular diseases including acute and chronic heart failures, coronary artery disease, acute myocardial infarction, hypertension and atrial fibrillation [18]. In obesity, there are adipokines have pro-inflammatory effects on the cardiovascular system while other adipokines have cardio protective and anti-inflammatory impacts.

Adiponectin is one of adipokines that is released mainly by adipocytes and cardiomyocytes and has antioxidant, anti-inflammatory and vasodilator action, hence reducing the pathogenic effects of cardiovascular disease. High levels of adiponectin are mostly linked to benefit effect on cardio-metabolism. Weight loss has been found to increase adiponectin levels, which in turn increases insulin sensitivity. In some studies without strong results, adiponectin has paradoxically accompanied with elevated risk of cardiovascular disease, so higher adiponectin concentration is a marker of poor prediction. [19]. Furthermore, in the case of obese patients, cardiovascular diseases are usually diagnosed and treated earlier than in the case of thin patients plus easier titration of the dose of medications for treating associated hypertension. A possible explanation of obesity paradox is that in critical ill patients, fat which mobilized from excess adipose tissue provides energy and prevents lean tissue wasting more efficiently than exogenous nutrients [20]. According to more recent theories explaining the “obesity paradox,” obese patients have “larger blood vessels” and in the course of PCI, worse results obtained in the case of patients with narrowed blood vessels [21].

According to endotoxin-lipoprotein hypothesis, obese patients have higher cholesterol and lipoprotein levels, which reduce concentration of inflammatory agents and may thus have anti-inflammatory and probably antiarrhythmogenic effect. We noticed that the myocardial accumulation of fat enhances the density of TNF-α I and II receptors, thereby facilitating the development of an antiarrhythmogenic environment, may at the same time explain the development of the obesity paradox [22]. At any BMI or total adiposity level, there is considerable individual variation in amount of subcutaneous versus intra-abdominal or visceral adipose tissue (VAT) in the abdominal cavity. Overweight and obese individuals with low levels of VAT characterized by a more favorable CVD risk profile sometimes referred to as metabolically healthy obesity [22]. When those with metabolically healthy obesity are compared with patients with excess VAT those with excess VAT represent a subgroup of individuals at highest CVD risk regardless of BMI. Studies that have examined the relationships b/w VAT and cardiovascular outcomes have also confirmed that VAT serves as a clear health hazard [23].

2. Conclusion

- Our study concluded that body mass index (BMI) co relates with high levels of FBG, cholesterol, triglycerides, LDL and low levels of HDL.
- Waist circumference (central obesity) correlates with triglycerides and Syntax Score (severity of CAD). Waist circumference that is ≥ 130 cm has highest sensitivity (85.71%) and specificity (82.56%) to detect high Syntax Score.
- Metabolic syndrome (Mets) is associated with high levels of FBG, cholesterol, triglycerides and low levels of HDL. Also, Mets is associated with high Syntax Score (severity of CAD).

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