EDITORIAL

Abdominal Subcutaneous Adipose Tissue, Diet, and Risk of Cardiovascular Disease: What do we Know?

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Editorial referring to the article: Association between Deep Subcutaneous Adipose Tissue Estimated by DAAT Index and Dietary Intake in Patients with Acute Coronary Syndrome

Excess adiposity increases the risk of cardiovascular disease (CVD) due to dyslipidemia, systemic inflammation and other risk factors.[1] The distribution of body fat depots, and central adiposity in particular, is related to cardiometabolic diseases.[2] The different adipose tissue depots in the body, ie, epicardial adipose tissue, visceral adipose tissue (VAT) and subcutaneous adipose tissue (SAT), have different biochemical characteristics and produce unique biologically active molecules that affect normal and pathological processes related to CVD risk.[2,3] Abdominal SAT is classified as superficial or deep. Deep subcutaneous adipose tissue (DSAT) has a higher ratio of saturated to monounsaturated fatty acids, and, consequently, is more saturated than superficial subcutaneous adipose tissue (SSAT).[4] Despite being in the subcutaneous compartment, DSAT differs from VAT because of differences in gene transcription, having different metabolic functions than SSAT.[5]

In Acute Coronary Syndrome (ACS) patients, coronary plaque instability is related to abnormal abdominal fat distribution. Analyses of the abdominal fat distribution of ACS patients have demonstrated that both VAT and SAT are positively correlated with vessel and plaque volumes, but not with plaque tissue components, the fibrous and lipid content of plaque.[6] However, considering coronary plaque characteristics, a higher VAT/SAT ratio was independently associated with a higher percentage of plaque lipid content, with a lower percentage of fibrous volumes and a thinner fibrous cap thickness,[6] characteristics that define greater plaque rupture vulnerability and a high risk for coronary events.

Subcutaneous adipocytes express higher adiponectin and lower pro-inflammatory adipokines, as well as increased adipogenesis.[3] However, most studies have not evaluated abdominal fat depots, ie, DSAT and SSAT. In patients recently diagnosed with type 2 diabetes mellitus, muscle insulin sensitivity and metabolic flexibility (the ability of insulin-sensitive individuals to switch from lipid to carbohydrate oxidation in response to insulin) were negatively associated with DSAT thickness, which was also related to impaired suppression of lipolysis and liver fat.[7] These results suggest that DSAT may play a deleterious role in mechanisms related to cardiometabolic health.

Diet plays a crucial role in abdominal fat accumulation. In a longitudinal analysis of the Framingham Heart Study, the lowest diet quality, evaluated by adherence to a Mediterranean-style diet score, was associated with the greatest abdominal SAT.[8] In addition, in a systematic review of observational and controlled intervention studies,[9] the quantity and quality of carbohydrates, protein, fat, and dietary fiber, as well as the intake of calcium, alcohol, phytochemicals, probiotics, cereal-based foods, sweets, sugar-sweetened beverages, between-meal snacks, and dietary patterns, were related to VAT and/or SAT. Thus, while it is clear that dietary exposure is related to abdominal fat depots, the relationship among diet, abdominal adiposity, and CVD outcomes is poorly understood.

Keywords

Abdominal Subcutaneous Fat; Acute Coronary Syndrome; Diet; Obesity, Abdominal; Inflammation; Metabolic Syndrome; Risk Factors/prevention and control.

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In the current issue, Stein et al.,[10] investigated the relationship between the dietary intake of ACS patients and DSAT estimated by the Deep-Abdominal-Adipose-Tissue (DAAT) index. In a cross-sectional analysis of data from 138 Brazilians, no association was found between dietary intake, as assessed through a quantitative food frequency questionnaire validated for the population, and DSAT. The results of the multivariate analysis demonstrated a positive relationship between DSAT and sedentary lifestyle and a negative relationship between DSAT and female sex. In addition, women had a higher waist circumference and waist-to-hip ratio, and lower DSAT levels than men. Although the DAAT index is not the gold standard for estimating DSAT, it can be easily calculated in clinical settings, and might encourage more clinicians to assess their patients' body fat distribution during office visits so that appropriate interventions, especially healthy lifestyle practices, can be prescribed. Based on Stein et al., increased physical activity would benefit ACS patients, but following a healthy diet would also be expected to promote weight loss and decrease adiposity and DSAT.

Finally, more research is needed to answer some remaining questions. First, what roles do adipose tissue and the different fat depots play as an endocrine organ by releasing inflammatory and/or anti-inflammatory molecules that may influence a coronary patient's metabolic response? Second, how does diet affect this relationship? Third, the way that diet can affect abdominal SAT, especially SSAT and DSAT, and their relationship with CVD outcomes are poorly understood. In summary, future studies should focus on clarifying the relationship between dietary intake, abdominal fat depots, including SSAT and DSAT, and cardiovascular health.

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