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Editorial Open Access

Visceral Fat and Association with Metabolic Risk Factors

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Editorial

Cardiovascular disease (CVD) is the leading cause of morbidity and mortality in the world. Globally, more than one billion adults are overweight (BMI≥25 kg/m2). At least 300 million are obese (BMI≥30 kg/m2) [1]. The visceral fat (VF) is a risk factor for multiple CVD risk factors, including endothelial dysfunction, hypertension, dyslipidemia, diabetes, impaired glucose metabolism, insulin resistance, metabolic syndrome (MetS), liver insulin resistance, non-alcoholic fatty liver disease, sleep apnea, increased predisposition to cancers of the colon, breast and prostate, and it is associated with prolonged hospital stays, increased incidence of infections and non-infectious complications, and increased mortality in hospital [2-5]. Age, gender, genetics, and ethnicity are factors contributing to variation in visceral adipose tissue accumulation. Specific mechanisms responsible for proportionally increased visceral fat storage may involve sex hormones, local cortisol production in abdominal adipose tissues, dietary fructose endocannabinoids and growth hormone. Physiological characteristics of abdominal adipose tissues such as adipocyte size and number, lipolytic responsiveness, lipid storagecapacity, and inflammatory cytokine production are significant correlates and even possible determinants of the increased cardiometabolic risk associated with visceral fat [6].

Abdominal obesity itself is an independent component of MetS and VF accumulation also determines a comprehensive cardiovascular risk profile and increases the susceptibility to ischaemic heart disease and arterial hypertension [7-8].

The VF releases different bioactive molecules and hormones, such as adiponectin, leptin, tumour necrosis factor(TNF-α), resistin and interleutin (IL-6, IL-1β)), resistin, adipocyte fatty acid binding protein (A-FABP), omentin-1, and chemerin. Adipokines contribute to the modulation of adipogenesis, immune cell migration into adipose tissue, adipocyte metabolism and function. Adiponectin is inversely correlated with the amount of VF, while decreased concentrations of adiponectin are associated with hypertension, type 2 diabetes, elevated glucose levels, cardiovascular disease and certain malignancies. Hypertriglyceridemia is a central correlate of visceral obesity. It is caused by a combination of increased liver VLDL triglyceride production and impaired clearance from the circulation [9-13].

An important contribution to evaluation of the influence of obesity on cardiovascular risk is the InterHeart Study, which shows evidence that abdominal obesity makes a higher contribution than BMI to the probability of these events[14]. Yusuf et al. defining the results more accurately, showing that the association between abdominal adiposity and coronary heart disease risk is highly significant in all geographical areas in which InterHeart Study data were collected [15].

In a population of 1,498 Caucasian, already showed that there was a strong independent association with VF and both cardiovascular (odd ratio (95% CI):2.45 (1.52–3.95)) and cerebrovascular events (odd ratio (95% CI):1.63 (1.06–2.50)); in the same study, a (ROC) analysis proved greater sensitivity and specificity of VF, compared to its individual components (WC, BMI, HDL, and TG) with regard to cardiovascular and cerebrovascular events [16]. In a large case-control study, a high VF is associated with elevated risk of CHD in Chinese men and women [17]. As visceral obesity is associated with poor prognosis, metabolic disturbances and degree of pathology also occur in several chronic diseases. The VF is metabolically active and associated with insulin resistance, atherosclerosis, metabolic syndrome and CVD. To develop novel aetiology based strategies for the prevention and treatment of these diseases, a better understanding of the molecular mechanisms underlying obesity and its relationship to metabolic and cardiovascular diseases is essential.

Therefore, the identification of VF that regulates the atherosclerotic process might provide new opportunities for developing more effective approaches for preventing and treatment of cardiovascular disease.

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