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Biomarkers of dysfunctional visceral fat

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Abstract

Dysfunctional visceral fat plays a key role in the initiation and maintenance of chronic inflammation, liver steatosis and subsequent systemic insulin resistance that primes the body for development of metabolic syndrome. These changes, occurring with or without obesity, lead to type 2 diabetes. In this chapter, we first provide a brief overview of the factors that lead to dysfunctional visceral fat and their relative importance. Adipose tissue has a great plasticity which allows for cell hypertrophy and, when needed, angiogenesis to sustain hypertrophy. Due to the prevalence of inexpensive and widely available "junk food," i.e., those enriched in fat, carbohydrate and sugar, this response becomes maladaptive. Hypertrophied adipocytes become hypoxic. Some undergo necrosis which induces macrophage recruitment forming crown structures wherein macrophages and leukocytes surround injured adipocytes. This leads to the ominous triad: inflammation, fibrosis (extracellular matrix hypertrophy) and impaired angiogenesis as well as consequent unresolved hypoxia. Adipokines and cytokines secreted by these crown structures as well as the palmitate fluxes due to excessive lipolysis are released from visceral adipose tissue to portal blood. They inundate the liver causing insulin resistance. In this review we explore the actions of adipokines, proteins and macrophage cytokines (adiponectin, leptin, FABP4, resistin, PAI-1, ANGPT3/4, IL-6 and TNF α) that normally intervene but whose action goes awry in the presence of inflammation and insulin resistance. We provide an assessment of their relative clinical utility as well as challenges associated with their use as biomarkers.

Keywords: Adipokines; Biomarkers; Cytokines; Fatty acid binding proteins; Insulin resistance; Interleukins; Lipotoxicity; Metabolic syndrome; Visceral fat.

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