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Letter to the Editor

OBESITY, INFLAMMATION, AND INFECTION

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INTRODUCTION

Obesity is a disease characterized by a high volume of body fat in which there is chronic low-grade inflammation. Inflammation is mediated by elevated levels of cytokines such as interleukin (IL)-1, IL-6, tumor necrosis factor (TNF), and C-reactive protein (CRP). Fat accumulation occurs especially in some specific regions, including the abdomen and visceral regions, where it creates hypoxia and death of adipocytes. Both innate and adaptive immunity are involved in obesity, which induces the activation of anti-inflammatory M1 macrophages that are transformed into inflammatory M2 types. Microbial infections are a risk factor where the inflammatory environment induced by obesity can weaken the body's immune response, with serious consequences for human health. Chronic inflammation not only predisposes individuals to infections but also contributes to the development of metabolic disorders and cardiovascular disease.

DISCUSSION

Statin therapy lowers cholesterol and reduces the incidence of death from cardiovascular disease and atherosclerosis, although some people treated with these drugs still suffer from cardiovascular diseases (1). People affected by obesity and infection present a stronger degree of inflammation, compared to the low degree inflammatory state that is present in obesity alone. Elevated lipid levels, mostly in the form of cholesterol and cholesteryl ester, are associated with obesity, atherosclerosis, and cardiovascular diseases. Obesity causes narrowing of the artery, predisposition to thrombosis, calcification, weakening of the muscles, and aneurysm dilatation.

In obesity, there is a low grade of inflammation, and in the case of infection, the production of cytokines can influence the inflammatory state induced by obesity (2). However, lipids can be lowered by diet and/or medication in most patients.

Monocytes and macrophages are innate immune inflammatory cells that respond to the excessive uptake of lipoproteins in obese patients by generating chemokines and cytokines. The adaptive immune response involves antigen-specific T cells, as well as the activation of B cells, which all produce inflammatory cytokines and chemokines. In this case, IL-12 serves as an important bridge between innate and adaptive immunity (3).

When the endothelium becomes inflamed, it expresses adhesion molecules that bind cognate ligands on leukocytes. Selectins, integrins, and chemokines mediate and favor the action of white inflammatory blood cells such as adherent leukocytes, diapedesis, migration and chemotactic stimulus. The Th1 cytokines involved in the effector T-cell response aggravate inflammation, whereas Th2 cytokines, such as IL-10 and IL-4, are anti-inflammatory and relieve

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inflammation. Leukocytes, as well as endothelial cells, secrete cytokines and growth factors that promote the migration and proliferation of smooth muscle cells (4).

Cytokines of the IL-1 family, such as IL-18, have an important function in host defense, immune regulation, and inflammation. IL-18 is an immunoregulatory cytokine which requires cleavage with caspase-1 to become active, and it was originally discovered as a factor that enhances IFN-gamma production from Th1 cells in the presence of anti-CD3 or anti-TcR Ab (5). IL-18 is essential to host defenses against severe infections and is a potent pro-inflammatory cytokine able to induce IFN-gamma, GM-CSF, TNF, and IL-1 in immunocompetent cells, and to activate killing by lymphocytes. Human pre-adipocytes of all differentiation stages spontaneously secrete IL-18, supporting the concept that adipocytes participate in innate immunity and that IL-18 mediates a fraction of the complications of obesity such as cardiovascular disease. It is known that IL-18 release from adipocytes of patients with obesity exceeds approximately 3-fold that from adipocytes of non-obese subjects (6). An augmentation of IL-18 is correlated with a significantly increased risk of developing cardiovascular diseases and type 2 diabetes. These concepts suggest that IL-18 measurement may add prognostic information to lipid and inflammatory markers in patients with or without obesity.

Inflammation is the defense response against infectious microorganisms and the low degree of inflammation that is already present in obesity can be exacerbated by infectious organisms. These reactions aggravate the pathological state that has been established in obesity.

CONCLUSIONS

Obesity is characterized by chronic, low-grade inflammation. The adipose tissue, in particular the visceral fat, secretes various pro-inflammatory cytokines such as TNF, IL-6, and CRP that promote systemic inflammation. Adipocytes and immune cells in the adipose tissue, such as macrophages, perform crucial roles in maintaining this inflammatory state. Chronic inflammation in obesity contributes to insulin resistance and cytokines interfere with insulin signaling pathways, exacerbating metabolic syndrome and type 2 diabetes.

Understanding the mechanisms that regulate inflammation in infected subjects with obesity can certainly help improve health strategies. Reducing obesity and treating the infectious state improves the inflammatory state, leading to improvements in health.

Conflict of interest

The author declares that they have no conflict of interest.

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