Obesity: A Crucial Risk Factor for Underlying Cause of Cardiovascular Diseases

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Introduction

Obesity has become more common around the world, which is a cause for concern because the detrimental e ects of obesity can begin as early as childhood. e body mass index (BMI) is the most extensively used anthropometric technique to determine relative weight and de ne obesity; BMI alone indicates a U- or J-shaped relationship with clinical outcomes and death. Such an inverse association has sparked a debate in the literature known as the 'obesity paradox,' which claims that individuals with high BMI and chronic conditions have a better survival rate and have fewer cardiovascular (CV) events than non-obese patients. BMI, on the other hand, is unable to distinguish between an increased body weight caused by high levels of lean vs. fat body mass. In general, metabolic problems are more typically related with an excess of body fat (BF) than with a high amount of lean body mass.

Adipose tissue is now thought to be a key organ in the fate of excess dietary lipids, determining whether body homeostasis is maintained (metabolically healthy obesity) or a state of in ammation/insulin resistance is produced, both of which have negative cardiovascular consequences. Obesity, particularly visceral obesity, causes a number of anatomical changes in the structure and function of the cardiovascular system. Adipose tissue is currently thought of as an endocrine organ that orchestrates critical interactions with important organs and tissues such the brain, liver, skeletal muscle, heart, and blood vessels [1].

Obesity and CVD: Obesity has a number of negative e ects on the CV system. Excess body fat accumulates over time, causing a number of metabolic changes that increase the incidence of CVD risk factors while also altering systems that control in ammation. Obesity increases changes in other intermediate risk factors such as dyslipidemia, hypertension, glucose intolerance, in ammation, obstructive sleep apnea/hypoventilation, and a prothrombotic state, as well as possibly many more unknown processes.

Cardiac adaptations to obesity: e CV system adapts to maintain whole-body homeostasis as a result of chronic excessive body fat buildup. In this adaptative condition, increased cardiac output and decreased peripheral resistance are important. e increase in circulating blood volume increases stroke volume, which is a primary predictor of higher cardiac output in obese patients. Increased heart

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spillover into typically lean tissues such as the liver, muscles, and intra-abdominal or visceral adipose depots. e saturation of lipid storage capacity in subcutaneous adipose tissue, as well as the ectopic fat deposition that results, causes in ammation and insulin resistance. Adipo(cyto)kines are also implicated in modifying processes that promote atherosclerosis, such as endothelial vasomotor dysfunction, hypercoagulability, and dyslipidemia, and are released by adipose tissue. Obesity changes the levels of several in ammatory mediators. First, levels of circulating Creactive protein (CRP) and tumour necrosis factor (TNF) (produced by adipose tissue) are elevated, but other mediators (such as Il-6 and 1B, and monocyte chemoattractant protein 1) and hormones (such as adiponectin and leptin) are also known to play a role in the in ammatory pro le seen in obesity, particularly abdominal obesity [5].

Raised blood supply is required for excessive adipose tissue growth, as seen in obesity, and total adipose tissue blood ow is increased globally. Perfusion per unit of adipose tissue, on the other hand, diminishes as adiposity rises. When an obese person is compared to a nonobese control, the di erence in perfusion may imply a 35 percent reduction in relative perfusion. is misalignment in perfusion reduces the oxygen supply to adipocytes, contributing to cellular hypoxia, organ stress and malfunction, pro-in ammatory responses, and metabolic illness.

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