Obesity and Type 2 Diabetes: Preventing Associated Complications

Editorial
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Editorial

Type 2 Diabetes (T2D) obesity go hand in hand. Many of the complications in both diseases are due to underlying primary drivers, insulin resistance, and metabolic syndrome. In this millennium, two diseases have reached a state of global epidemics [1]. In addition, obesity and T2D have become major public health issues, threatening the well-being of millions of people in both industrialized countries and emerging economies. In those who are genetically susceptible, taking in more energy than is necessary favors the development of glucose intolerance and subsequent development of T2D and associated complications [2].

Like other diseases, obesity has

I. a cause (caloric imbalance, availability and abundance of food, and consumption of low-nutritious, high-caloric food);

II. pathology (adipocyte-mediated excessive production of inflammatory cytokines and hormones and genetic suceptibility);

III. pathophysiology (an environmentally and psychologically inducible dysregulation of appetite, low physical activity levels, body fat distribution, in favor of viceral fat, psychological issues, and deranged body-weight–controlling mechanisms) [3], and

IV. a disease that can be treated with anti-obesity medication.

However, not all obese patients experience complications such as T2D, Cardio Vascular Disease (CVD), and strokes [4,5]. However, there are no specific, sensitive, and cost-effective markers or tests available for separating those who are vulnerable to developing complications from those who have low risks. Obesity is much more complicated than being just a lifestyle issue. It also involves abnormalities of the mitochondria, which play central roles in energy expenditure and energy balance, and also has genetic predispositions [6]. Meanwhile, obesity is becoming the leading preventable cause of cancer [7-9].

Instead of blaming junk food and lack of exercise alone, the emergence of a worldwide epidemic of obesity and T2D can also be considered as a results of adversely altering intestinal microbiota that can supress satiety. Adherece to alteration of fat intakeand high caloric sugery diets, in combination with inappropriate and excessive use of antibiotics, and succesful control of infections, not only increase the caloric intake and appetite, but also slow down the energy metabolism.

Although there are common causes, each individual has a different set of risk factors that lead to the development of overweight, obesity, and T2D; if the issue of excess weight is unattended and untreated, these can lead to serious complications [10-12]. Excess visceral adiposity is positively correlated with the insulin resistance, T2D, certain cancers, and premature deaths [13]. An effective strategy to control this necessitates both, preventing individuals from becoming overweight and early identification of the cause(s) of obesity and interventions. With such knowledge, clinicians should then could individualize care and treatment plans to target weight loss, particularly to lose intra-abdominal fat (using waist size as a surrogate marker), Such root-caused driven approache would improve patient quality of life and generate cost savings in the long run.

Different people have different causes and underlying genetic susceptibilities for accumulating fat and experiencing of T2D and obesity [14-15]. Thus, one management plan will not work for everyone [16]. To be successful, the management approaches need to be individualized and cause-oriented (elimination of root causes in a given person) [6], proactive and systematic, and use cost-effetive methods to identify those who are at high-rish and prone to develop complications. In addition, treatment strategies must be timely, appropriate, affordable, acceptable, and cost-effective, and patients must be able to adhere to the interventions.

By the law of conservation of energy, obesity is the result of an imbalance between energy intake and energy expenditure; fat content increases when energy intake is consistently greater than energy expenditure. Because of inherent difficulties with energy expenditure and alteration of mitochondrial activity, , older people accumulate greater amounts of visceral fat more easily than do younger individuals [17]. Similarly, a greater amount of visceral fat accumulates in women during the perimenopausal period [18], in part because of relative estrogen deficiency [19].

The increase in body weight [fat] associated with overconsumption of calories and engaging in less-than-optimal physical activity [20,21], This scenario is influenced by socio-economic status, beliefs and social, cultural, and behavioral factors. However, the stigma associated with the obesity

hindering the successful outcome of many [22]. The health risks associated with obesity also depend on the distribution of body fat; accumulation of intra-abdominal fat being the worst. For example, people with less visceral fat but higher amounts of peripheral subcutaneous fat, even though may have higher BMI, can be healthy [23] and may experience less obesity-associated complications [14].

In white Caucasians, the use of the Body Mass Index (BMI) is a sensitive tool for categorizing those who are overweight versus those who are obese [24]. Although BMI alone is useful for white Caucasians, because of its lack of sensitivity, by itself, is not useful for most other ethnic groups, such as Asians, blacks, Hispanics, and Pacific Islanders, for the identification of obesity or assessment of future CVD risks [25]. However, the sensitivity of BMI improves when it use in combination with waist circumference measurement. The latter can be easily done in each biannual clinic visit and noted in medical records for decision-making and follow-up.

In most obese patients, the metabolic damage is due to excess visceral (abdominal) fat. Visceral obesity is strongly linked with insulin resistance and the metabolic syndrome [26,27]. Metabolic studies conducted in several laboratories have confirmed that among equally obese patients, subjects with excess visceral adipose tissue have the metabolic profile with the highest risks [28,29], including insulin resistance and compensatory hyperinsulinemia. Furthermore, atherogenic dyslipidaemia and insulin resistance frequently present in patients with abdominal obesity [10,15,30].

However, there are no direct, practical, and cost-effective methods of measuring the severity of metabolic syndrome or determining who is at high risk for experiencing complications [14,31]. The currently available biochemical methodologies are not specific enough to determine who is at risk versus who is not. Nevertheless, expensive high-tech testing is available for quantifying visceral fat to identify those who are at risk of CVD. Because of the lack of cost-effectiveness, it is hard to justify the use of these imaging techniques and non-invasive methods for routine investigation and assessment of obesity and for cardiovascular risks.

Therefore, the use of a simple anthropometric measurements, such as the waist circumference and/or waist-to-hip ratio, together with basic blood lipid profile scan be cost-effectively in identifying those who are at a higher risk for complications and for monitoring the progress [16,32]. In obese patients, measurement of abdominal girth is the most cost-effective (but underutilized) and the easiest way to identify and monitor patients who are at a high risk for CVD.

Despite medical advances and the availability of a series of new pharmaceutical agents, lifestyle changes, healthy eating, and increased physical activity (if adhered to advice and treatments by patients) are still the most cost-effective approach and the fundamental basis of preventing and managing obesity and T2D. Even for those obese persons who would benefit from pharmacotherapy or bariatric surgery, positive lifestyle changes are essential for the longer-term success in weight maintenance and the disease control [6,33].

The combination of sustainable lifestyle changes and pharmacotherapy (or bariatric surgery in highly selected patients) not only would maintain a patient's weight at a lower physiological set point but also would minimize the long-term complications of T2D and obesity [34]. However, because of the poor compliance with instructions and medications, the overall effectiveness of lifestyle changes and obesity therapies for weight loss is limited. However, encouraging and close monitoring in a motivated person, set goals can be achieve successfully.

Evidence-based research on lifestyle interventions have demonstrated that effectiveness of reducing the body weight by 5 to 10% in improving insulin resistance [35], and decreasing the incidence of CVD, heart failure, stroke, cancer, diabetes, and all-cause mortality [36-38]. These alone should encourage overweight and obese patients to consider losing weight.

In addition, such patients should be offer advice and guidance not only on healthy life styles and education, but also about the causes leading to excess weight (i.e., to empower patients) assisting them to adhere to a weight-reducing diet, a reasonable physical activity regimen, and periodically monitoring the patient's progress. Medications and bariatric surgery are effective but not the first set of options. Even when such treatments offered, lifestyle and behavioral changes need to be complementary [39].

It is easier to prevent obesity than to lose weight; one needs to identify risk factors in individual patients at the earliest possible time. Those who are overweight or obese with excess visceral fat, and engaged in smoking, drinking excessive amounts of alcohol, partaking of the least amount of physical activities, and having an adverse CVD family history are at the highest risk for insulin resistance, T2D, CVD, and premature death [40-42].

In these high-risk populations, timely and effective interventions (goal of achieving approximately, 10% weight loss) would significantly reduce the human and financial costs in themajority of patients, as well as productivity lost from complications associated with metabolic syndrome, T2D and obesity [26,43]. Since these diseases and associated complications and cost are predict to triple during the next two decades, policy makers must pay attention to this progressive, insidious epidemic, and proactively determine the right path. However, tackling T2D, obesity, and metabolic syndrome, requires prioritization of resources, combined approaches, and paradigm shift in thinking.

Obesity is more than simply a thermodynamic, genetic, or a metabolic problem of handling calories; it is also an inflammatory disease, impairment of the neuro hormonal system, and a behavioral disorder leading to dysregulation of metabolism and energy balance that results in accumulation of visceral fat leading to serious multi-system complications. In addition to reasonable weight reduction programs, controlling the obesity-associated complications requires coordinated, individualized, cause-driven, and complication-centric approaches. Understanding the cause of obesity in each patient would greatly facilitate the development of an individualized, sustainable treatment plan that patients can adhere to, that would lead to successful outcomes.

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