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OBESITY AND TYPE 2 DIABETES MELLITUS – THE CHICKEN EGG PROBLEM

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Obesity has become a very common public health concern in recent times. One of the main comorbidities of obesity is diabetes. Up to 30% of the obese population have diabetes and up to 85% of diabetic patients are overweight. Due to this, the common term “Diabesity” was introduced showing the strong association between these two diseases. Obesity has a complex relationship with Type 2 Diabetes Mellitus (T2DM). Adipose Tissue is considered as an endocrine organ, which releases many pro inflammatory cytokines called as adipokines which causes inflammation. Adipokines also include hormones such as leptin and adiponectin. The resulting chronic inflammatory state and the hormone leptin can lead to insulin resistance. Additionally, TNF-α also decreases the tyrosine kinase phosphorylation of insulin receptor, which can result in systemic insulin resistance. While subcutaneous fat is responsible for 80% of the leptin production, visceral fat can also be a risk factor as it’s lipolytic activity with high release of Free Fatty Acids (FFA) negatively affects the action of insulin (M.N. Amin et al. 2019).

There have been multiple studies conducted on surgical procedures for obesity such as bariatric surgery, Roux-en-Y gastric bypass (RYGB) and vertical sleeve gastrectomy (SG), one anastomosis gastric bypass (OAGB) and their effect on glycemic control of patients. A research conducted by Moradi.M et al (2022) demonstrated that from a total of 1351 patients, 675 patients (50.0%) undergoing OAGB, 475 (35.2%) RYGB, and 201 (14.9%) SG had remission of T2DM after one year. Also, weight loss induced by calorie-restricted diet has shown to significantly increase remission of T2DM. A study conducted by Yang.X et al (2023) recruited 72 random T2DM patients and allocated Chinese Medical Nutrition Therapy (CMNT). to half of the group (36 patients). On completion of 3 months of intervention and 3 more months of follow-up, it was shown that 47.2% (17/36) of participants achieved diabetes remission in the CMNT group, whereas only 2.8% (1/36) of individuals achieved remission in the control group. This further supports the fact that obesity increases the risk of T2DM.

Although Obesity is generally considered to cause insulin resistance followed by T2DM, a subset of obese people maintains their insulin sensitivity and has fewer comorbidities compared to the rest according to Al-Sulaiti et al. (2019). The underlying protective mechanisms in these people leading to their insulin sensitivity being preserved is unknown.

John I. Malone and Barbara C. Hansen (2018) views the relation between Obesity and T2DM from the opposite perspective, which says that insulin resistance develops not as a result of obesity, but rather develops primarily in the muscles of non-obese individuals who are genetically predisposed to T2DM. This insulin resistance causes increased level of hepatic glucose production and increased insulin secretion which results in excessive fat accumulation in the body leading to obesity.

Additionally, there’s growing evidence linking T2DM and Obesity as a result of mitochondrial defects. Reduced physical activity and energy demand lead to a shift in mitochondrial dynamics from interconnected networks to its fragmentation. Additional lipid supply promotes lipotoxic effects on different insulin cascade steps.

In conclusion, there is a complex relationship T2DM and Obesity, which makes it a challenge to establish a solid mechanism of its occurrence. As of recent times, T2DM and Obesity have been viewed from different perspectives, through various researches. While some researches claim that the underlying mechanism is unclear, most researches claim that inflammation is thought to be the major factor in the development of the condition.