Pathophysiology of Diabetes Mellitus

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Abstract

Diabetes mellitus (DM) represents a multifaceted metabolic disorder distinguished by persistent hyperglycemia caused by impairments in insulin secretion, insulin efficacy, or both mechanisms. The pathophysiology of diabetes encompasses a diverse range of factors, including genetic, environmental, and immunological influences. In Type 1 diabetes (T1D), an autoimmune mediated destruction of pancreatic β-cells culminates in a complete deficiency of insulin, whereas Type 2 diabetes (T2D) is predominantly linked to insulin resistance accompanied by a relative insufficiency of insulin (American Diabetes Association, 2020). In T2D, peripheral tissues, including muscular and adipose tissues, demonstrate diminished sensitivity to insulin, a condition often aggravated by obesity and a sedentary lifestyle (Kahn et al., 2014). This state of insulin resistance is typically characterized by compensatory hyperinsulinemia until the functionality of β-cells deteriorates, leading to hyperglycemia. Furthermore, the dysregulation of hepatic glucose production is pivotal in the pathophysiology of T2D, with the liver playing a significant role in the enhancement of gluconeogenesis (Buse, 2016).

Keywords

Diabetes mellitus, Hyperglycemia, Insulin Resistance.