Pathophysiology of Insulin Resistance and Type II Diabetes Mellitus

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Type II Diabetes Mellitus

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Introduction

The topic I have chosen for my pathology presentation is type II diabetes mellitus: a case of beta cell underfunction and insulin resistance (Kahn, Cooper, & Bell, 2014).

The rationale for selecting this topic was to clearly illustrate the pathogenesis of type II diabetes mellitus as well as the negative consequences of the disease on the individual and the healthcare system. There is an obvious clinical need to discuss the subject. (Kahn et al., 2014).

I was personally motivated in selecting this topic as I am a type I diabetic patient who contracted my condition via a viral infection from a young age. I have been genetically blessed with a fantastic endocrine system but have to keep my A1C level in the mid to upper five range. Without a vaccine to combat type I diabetes, patients are frequently observed having poor or worsening clinical outcomes due to uncontrolled type II diabetes.

Type II diabetes mellitus presents itself in one of two ways (Kahn, et al., 2014). First, beta cells within the pancreases are unable to produce adequate amounts of insulin to break down blood glucose levels regulated (Kahn et al., 2014). Second, insulin sensitivity is decreased where individuals who are typically overweight or obese may have been producing too much insulin due to insulin resistance in appetite cells (Kahn et al., 2014). Eventually the beta cells cannot keep up the production and production declines despite there being an increased demand for it (Kahn et al., 2014).

It was estimated that 29 million Americans have type II diabetes mellitus in the United States (Kahn et al., 2014). It was also estimated that 20% of the additional 86 million adults afflicted with prediabetes (CDC, 2014) are at high risk for type II diabetes mellitus which is a condition where growth is not only limited to individuals with prediabetes (CDC, 2014) but also to 6.5 or higher (“MayoClinic,” 2018). The treatment of type II diabetes mellitus includes weight loss, proper diet, exercise, possible medication and insulin therapy, and frequent blood glucose monitoring (“MayoClinic,” 2018).

Signs & Symptoms

Type II diabetes mellitus and insulin resistance are complex interrelated conditions that form a feedback loop between the peripheral tissues and the beta cells (Kahn et al., 2014). Eventually the beta cells cannot produce adequate amounts of insulin due to chronic hyperglycemia (Kahn et al., 2014). As blood sugar levels go hand in hand with the beta cells, the resulting feedback loop leads to insulin resistance (Kahn et al., 2014). This is a frequent occurrence in the obese population. Insulin resistance is usually caused by the beta cells no longer being able to produce adequate amounts of insulin to maintain normal blood glucose levels (Kahn et al., 2014).

Over time the beta cells cannot keep up with the increased demand for insulin production resulting in hyperglycemia (Kahn et al., 2014). This is a frequent occurrence in obese and overweight individuals which can be rectified with weight loss (Kahn et al., 2014).

As of 2018, nearly 70% of Americans are either overweight or obese (“AHA,” 2018). Obesity is defined as being at or greater than 20% of an individual’s ideal weight (“AHA,” 2018). Currently there are nearly 13 million children and adolescents who have been classified as overweight or obese (“AHA,” 2018). The figures yield a total of 23.9 million overweight and obese patients presenting with ischemic stroke and increased ischemic and ischomic stroke event rates (Kahn et al., 2014).

The increased risk of MACCE events in diabetic patients was supported with a p value of < 0.001 (Neuman et al., 2018). The study found that from 2004 to 2013 the amount of diabetic patients presenting for surgery had increased significantly from the previous study (Neuman et al., 2018). Kubani and others found that patients presenting with ischemic stroke were more often found to have hyperglycemia and raised hemoglobin A1C than not (Kuban, almam, & Rahman, 2017).

Conclusion

Hayward and others conducted a study identifying 1,791 patients with type II diabetes and randomly assigned them to insulin-glucose control and the other half to standard therapy (Hayward et al., 2015). The results revealed that patients in the intensive glucose control group experienced significantly lower major cardiovascular events than those assigned to the standard therapy (Hayward et al., 2015).

Type II diabetes mellitus has proven that by the year 2030 roughly 552 million individuals will have diabetes (Lupo et al., 2017).

As healthcare providers, it is imperative to reverse this trend and limit the micro and macrovascular complications of type II diabetes and insulin resistance (Kahn et al., 2014).