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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 presentation is type II diabetes mellitus: a case of beta cell underproduction and insulin resistance (Kahn, Cooper, & Del Prato, 2014). Type II diabetes Mellitus occurs when a patient has two or more characteristic warning signs, and a level of fasting blood glucose exceeding 7.0 mmol/L (130 mg/dL) (Lupo, 2014).

Signs & Symptoms

• Signs and symptoms of type II diabetes include increased thirst, increased hunger, dry mouth, frequent urination, unexplained weight loss, fatigue, blurred vision, numbness or tingling in the hands or feet, dry and itchy skin, and slow healing of cuts and sores (Chowdhury, 2013).

• Type II diabetes can also manifest as elevated blood glucose levels in the postprandial state (Kahn et al., 2014). The combination of impaired insulin production and elevated glucagon secretion is known as insulin resistance (Kahn et al., 2014). The correct treatment also tackles the underlying condition by increasing insulin sensitivity (Kahn et al., 2014).

Patient Presentation

• Mr. R is a 59-year-old Hispanic American male presenting to the health clinic with complaints of fatigue, thirst, and increased appetite three to six months despite eating, drinking, and sleeping more than usual.

• Upon a complete history and physical examination Mr. R is diagnosed with type II diabetes mellitus (diabetes in the elderly, 2012).

• Diabetes mellitus is brought on in type II diabetics by medication underproduction, or present or past infection, resulting in sustained uncontrolled hyperglycemia (2018).

• Long-term effects of uncontrolled type II diabetes include retinopathy, neuropathy, nephropathy, elevated coronary artery disease risk, elevated cerebrovascular disease risk, and elevated peripheral arterial disease risk (Table of Hispanic Medicine, 2018).

Pathophysiology of Type II Diabetes Mellitus and Insulin Resistance.

Type II diabetes mellitus and insulin resistance are complex interrelated conditions that involve diverse types of pathophysiological abnormalities (Cersosimo, Tripodi, Sollin, Ferrarese, & Manieri, 2018). The topic I have chosen for my presentation is type II diabetes mellitus as well as the negative consequences it has on clinical outcomes (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 and have been blessed with a fantastic support system. My family has always been there to make sure I keep my A1C in the mid to upper five range.

• Working in a very active recovery unit, patients are frequently observed having poor or worsening cardiovascular and special outcomes due to uncontrolled type II diabetes.

• Type II diabetes presents itself in one of two ways (Kahn et al., 2014). First, beta cells within the pancreas are unable to produce adequate amounts of insulin to maintain blood glucose levels (Kahn et al., 2014; Lupo, 2014). Second, insulin resistance occurs where individuals who are typically overweight or obese or have been producing too much insulin due to increased sensitivity inappropriately (Kahn et al., 2014). Eventually the beta cells cannot keep up the insulin production and production declines due to decreased insulin output for a (Kahn et al., 2014). It was estimated that 29 million individuals in the United States have type II diabetes, along with an additional 86 million with prediabetes (“CDC,” 2014). A diagnosis of type II diabetes is made when a patient has two separate tests indicating an A1C of 6.5 or higher (“MayoClinic,” 2018).

• In insulin resistance the kidneys increase rates of glucose reabsorption at time to levels of 220 to 250 mg/dL before reaching a maximum reabsorptive capacity (Cersosimo et al., 2018). One reabsorptive capacity is reached elevated blood glucose occurs in peripheral circulation (Cersosimo et al., 2018). Beta cells at first are able to increase insulin production but eventually are unable to keep up with the elevated demand (Cersosimo et al., 2018). At the same time, an inappropriate release of glucagon in the alpha cells of the pancreas occurs post-prandially (Cersosimo et al., 2018). The combination of impaired insulin production and elevated glucagon secretion is known as the decreased insulin effect, existing in the gastrointestinal tract (Cersosimo et al., 2018). The central control system also plays a major role in type II diabetes as the hypothalamus determines the body’s resistance by inhibiting the body’s capacity to suppress glucose production (Cersosimo et al., 2018).

Pathophysiology continued.

The liver plays a major role in glucose regulation as it is the main organ responsible for glucose uptake after meal consumption (Cersosimo et al., 2018). Liver dysfunction is affected with type II diabetes have reduced hepatic glucose uptake post-prandially resulting in elevated glucose levels (Cersosimo et al., 2018).

Significance of Pathophysiology

With an increasingly overweight and obese population insulin resistance has become more commonplace (Kahn et al., 2014). Insulin resistance and obesity often times go hand in hand when it comes to type II diabetes (Kahn et al., 2014). There is a known link that exists between beta cells and beta cell insulin and beta cell sensitivity (Kahn et al., 2014). The beta cells facilitate the uptake of glucose, fatty acids, and proteins and provide energy to the body (Kahn et al., 2014). The cause of overweight and obese individuals becomes less sensitive to insulin increasing the demand on the beta cells (Kahn et al., 2014). The cause of overweight and obese individuals becomes less sensitive to insulin increasing the demand on the beta cells (Kahn et al., 2014). The increased risk of overweight and obese individuals will be more prominent when it comes to type II diabetes (Kahn et al., 2014).

Implications for Care

A study was conducted evaluating patients undergoing coronary surgery from the years 2004 to 2013 encompassing roughly 10.5 million individuals (Newman, Wilcox, & Berger, 2014).

The study evaluated MAEC outcomes in diabetic patients versus non-diabetic individuals. The significance of this study is that it is one of the first to apply statistical significance to the risk factors associated with diabetes (Newman, Wilcox, & Berger, 2014).

The increased risk of MAEC events in diabetic patients was supported with a p value of <0.001 (Newman et al., 2018). The study found that from 2004 to 2013 the amount of diabetic patients presenting for surgery had increased significantly from the studies inception (Newman et al., 2018).

Fatman and others found that patients presenting with ischemic stroke events were often found to have hyperglycemia and raised hemoglobin A1C than not (Fatman, Islam, & Rehman, 2017).

Conclusion

Hayward and others conducted a study indicating 1,791,520 patients with type II diabetes and randomly assigned to patients who consumed glucose control and the other half to standard therapy (Hayward et al., 2015). The results revealed that the incidence in the intensive glycemic therapy group occurred significantly less major cardiovascular events than those assigned to the standard therapy group (Hayward et al., 2015).

Several projections have shown that by the year 2030 roughly 552 million individuals will have diabetes (Lupo, 2014).

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

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