Primary Care Disease- Type II Diabetes

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Introduction

Type II diabetes is a chronic condition that is mostly associated with adults and is viewed as adult-onset diabetes; however currently there are even young people who are diagnosed with the disorder become of the rising number of children with diabetes. Currently, there is no cure for Type II diabetes. There are measures recommended by doctors which entail being able to eat well, doing regular exercises and losing weight as among the major ways of helping with the management of the condition. In the event that diet and exercise do not work for the patient, one will require to undergo a medicine or insulin and insulin therapy. One of the reasons for primary care is because this is a chronic condition which affects the way that the human body will metabolize glucose, which is a very important aspect of the human body. In any case, the human body is going to resist the effect of the insulin, which is a hormone in the human body that regulates the movement of the glucose or sugar on the cells, or when it does not produce the required insulin to maintain the levels of glucose.

Evidence-Based Facts about Type II Diabetes

Type II diabetes was formerly referred to as a non-insulin-dependent or adult-onset, which is as a result of the ineffective use of insulin by the body. Type II diabetes, according to the world health organization, is evident all over the world and is mostly as a result of the excess weight of the body and the physical inactivity (WHO, 2016). In most cases, symptoms are similar as those of type 1 diabetes which are usually less marked, and as a result, this may be diagnosed in several years after the onset of the condition after the complications are already realized (WHO, 2018). Diabetes II is mostly seen in adults but also occurs in children.

According to the world health report of 2018, the number of people diagnosed with diabetes has risen from 108 million to 422 million by 2014 (WHO, 2018). The prevalence of diabetes in the world among adults over 18 year has also risen from 4, 7% in 1980s to 8.5% in 2014 (WHO, 2018). The prevalence of diabetes has continued to rise more rapidly over the years especially in the middle and low income countries. Diabetes is also reported to be one of the major causes of kidney failure, blindness, heart attacks, lower limb amputation and stroke (WHO, 2018). By 2016 it was estimated that about 1.6 million people died directly due to diabetes-related complications (Need Reference). Another 2.2 million deaths were also reported due to high level of sugar in the body (WHO, 2018). Diabetes II often affect older people were about half of all deaths are related to high blood glucose before people reach age 70 (WHO, 2018). It is also estimated by the world health organization that diabetes is the seventh leading cause of death around the globe (Need Reference). According to the Center for Disease Control, there are more than 30 million in the United States that have been diagnosed with diabetes where 1 in 4 of them know that they have the condition (CDC, 2019). There are also more than 84 million adults in the United States and over a third are pre-conditioned to diabetes (Need Reference). However, 90% are not aware that they have it. Reports also indicate that diabetes is the 7th leading causes of death in the United States, and this could even be worse. In most cases, Type II diabetes is going to account for 90 to 95% of all cases diagnosed while Type I diabetes only accounts for 5% (CDC, 2019). Statistics by the CDC indicate that in the last two decades, the number of adults diagnosed with diabetes has doubled due to the ageing population in the United States (Need Reference).

Diabetes Pathophysiology

The pathophysiology of diabetes is usually related to the insulin levels in the body and the ability of that body to make use of the insulin. When it comes to Diabetes I there is a lack of insulin (Moini, 2019). However, when it comes to diabetes II, it looks at the ability or inability to resist the effects of the insulin. In the normal situation, the pancreatic beta cells are going to release insulin because of the increase in blood sugar concentration since the brain required enough sugar to work well (Moini, 2019). This calls for the interaction of different hormones with the liver and the renal function, which is often difficult to pinpoint and is different for various patients (Moini, 2019). Despite the cause of the condition, it will eventually lead to a decrease in glucose uptake. The resistance of the insulin is often mediated by the predisposing genetic factors, which also is presented as abdominal obesity. There is a strong relationship between obesity and diabetes II where eight per cent of Type II diabetic patients are obese where excess fat is carried in the upper area (Everett, Medunjanin, & Frithsen, 2018). In most case, interventions to the condition are going to vary with the degree of control and the level of insulin resistance or then noted insufficiency. Also, the expected outcomes will widely vary.

Current Evidence-based Practice

According to recent studies, as compared to the normal type of care, among the best interventions for type II, diabetes is a lifestyle, especially during pre-diabetes. This can lower the risk of developing diabetes at the population level, as a group-based program is said to have low incremental or medial impact ration within the health systems (Golden et al., 2017). According to various multi-randomize, controlled trials around the world have indicated that there is the efficacy of modification of lifestyle in relation to the prevention of diabetes II among the highrisk population in the united states and around the globe. Other interventions include weight loss, physical therapy and drug therapy.

Classes of Drugs

Metformin (Change this to Biguanides drug class and please discuss about the biguanides below)

Metformin is one of the drugs considered as a first-line agent in the elimination of type II diabetes (Inzucchi et al., 2015). Metformin works by altering the structure of gut microbiota and activating mucosal AMP-activated protein-kinase (AMPK) that is fundamental in maintaining the integrity of the intestinal barrier. The effect coupled with activating AMPK is the primary mechanism used by metformin to reduce the levels of lipopolysaccharide (LPS) circulating the body and in the liver (An & He, 2016). When it arrives in the liver, metformin uses four different mechanisms to inhibit gluconeogenesis. These include (1) use of liver-kinase B1 to activate AMPK and decrease energy; (2) blocking adenyl cyclase that inhibits the production of glucagon-induced cAMP; (3) inhibiting the functions of NADH coenzyme oxidoreductase in the electron transport of mitochondrion that is critical in reducing the levels of ATP and increase the ratio of AMP/ATP. The increased rate is instrumental in activating AMPK; and (4) prohibiting the functions of mitochondrial glycerol phosphate dehydrogenase (MG3PDH) (An & He, 2016). This process affects the transportation of NADH from the cytoplasm to the mitochondria, which suppresses gluconeogenesis process from lactate. Metformin is critical for the treatment of patients with type II diabetes because it reduces blood sugar glucose by 20% and HbA1c by approximately 1.5% (Need Reference).

Insulin Secretagogues: Sulfonylureas and Meglitinides

These are two different classes of drugs that are administered orally but possess a similar mechanism of action. Meglitinides and sulfonylureas are both involved in the stimulation of pancreatic beta cells that produces insulin. Sulfonylureas are either considered as a first or

second-line therapy in the treatment of patients with type II diabetes (Genuth, 2015). The drug has been widely applied in the clinical setting since its introduction in the early 1950s (Need Reference). Meglitinides stimulate the release of insulin through a similar mechanism as sulfonylureas but use a different subunit binding site that has more rapid stimuli and more rapid absorption of the secreted insulin. The mechanism of action of meglitinide and sulfonylureas is based on the secretion of insulin, a process which is controlled by ATP-sensitive potassium channels that are found in the membrane of the beta cells of the pancreas. Although the two medications have a different binding site for the receptor, they are both involved in inducing cell depolarization and channel closure that is essential in increasing the level of calcium in the cytoplasm leading to secretion of insulin.

Alpha-glucosidase Inhibitors

Examples of drugs in this class include acarbose, voglibose, and miglitol. These medications help in reducing the level of postprandial triglycerides. However, the effect of alpha-glucosidase on fast-acting triglycerides and HDL and LDL cholesterol levels is insignificant (Ferrannini, & DeFronzo, 2015). This class of drugs rarely induces hypoglycemia and does not help in the stimulation of insulin either does it assist in lowering the weight of the body. The alpha-glucosidase inhibitors are similar in structure to oligosaccharides. This class of drugs produces a reversible inhibition of alpha-glucoside hydrolase enzymes that are bound in the membranes of the intestines (Abe et al., 2011). The process leads to delay in the absorption of carbohydrates and digestion of food and leads to a decrease in postprandial hyperglycemia. The carbohydrates that have not undergone digestion in the lower region of the small intestine increases the levels of plasma RA-GLP1. Alpha-glucosidase inhibitors do not facilitate the secretion of insulin because of the reduced concentration of blood glucose (Abe et al., 2011).

Thiazolidinediones (TZD)

The two main types of TZD currently available in the market include pioglitazone and rosiglitazone. TZD increases the sensitivity of insulin by acting on liver and muscle adipose tissue to increase the utilization of glucose and decrease the production of glucose. The drug binds to perisome proliferator-activated receptors (PPARs). PPAR- Υ is located in the central nervous system, adipose tissue, macrophages, pancreatic beta-cells, and vascular endothelium. The concentration of PPAR- Υ is high in the skeletal muscle of people with obesity and type II diabetes (Ryan et al., 2011). Rosiglitazone is purely PPAR- Υ agonist while pioglitazone has both PPAR- Υ and PPAR- α effects meaning they exert different effects on lipids. Pioglitazone leads to the production of a more favourable lipid profile, i.e. the LDL cholesterol remains constant during therapy administration, whereas rosiglitazone increases them (Ryan et al., 2011).

Conclusion

In conclusion, Type II diabetes is one of the most dangerous chronic conditions that require primary care from both the patient and the nurse of physicians to ensure positive results and prolong the life of the individual diagnosed with the condition. Since diabetes is mostly considered as a lifestyle condition, one can easily manage it with proper education on insulin intake, physical exercise and change of lifestyle. With a better diet, one is also able to reduce the weight and control of sugar intake to ensure that the glucose level does not rise or does not go lower than what the body and the human brain required. Even though there has been a lot of development in regard to medical therapy, it is important that health professionals insist in ensuring that patients and the overall population learn the importance of the decision they make about their health is this is also one of the primary interventions.

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