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Type II Diabetes Mellitus

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Introduction

Type II Diabetes Mellitus (DMII) affects an "estimated 8.3% of the adult population or 382 million people worldwide" (Cornell, 2015, p. 621). The researcher is currently an emergency department registered nurse and future advanced practice provider of family medicine. DMII was chosen by the researcher as the primary topic to gain a better knowledge and understanding of the pathophysiology and implications of DMII, thus the future APP will be able to provide the best treatment and provide the best quality of life for patients with DMII.

- DMII is a metabolic disorder that causes glucose to accumulate in the blood rather than being used as fuel by the cells in the body (Waddell, 2017)
- DMII is the result of insulin resistance, in which insulin production is significantly increased initially, but subsequently decreases as a result of beta cell failure (Mayo, 2016)

Criteria for the diagnosis of diabetes is of the following:

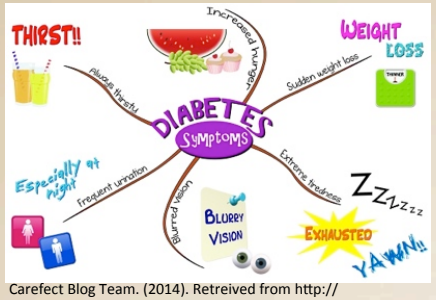
- Hemoglobin A1C (HbA1C) of 6.5% or greater
- 8-hour fasting plasma glucose (FPG) of 126 mg/dL or greater
- 75-g oral glucose tolerance test with 2-hour plasma glucose level of 200 mg/dL or greater in a patient presenting with hyperglycemic symptoms (Waddell, 2017)

Signs and Symptoms

Signs and symptoms of DMII include:

- Polyuria
- Polydipsia
- Polyphagia
- Glycosuria
- Fatigue or tiredness
- Blurred vision

(Mayo, 2016)



Carefect Blog Team. (2014). Retreived from http://www.carefecthomecareservices.com/blog/warning-signs-type-2-diabetes/

Risk Factors

Risk Factors include (Mayo, 2016):

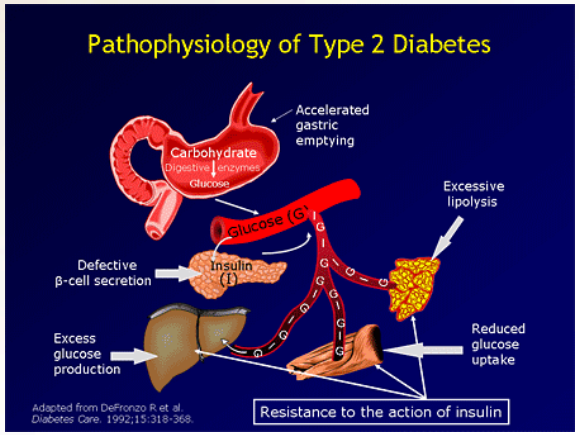
- Family history of DM
- Overweight or obesity
- Ethnicity: African American, Alaska Native, Native American, Asian American, Hispanic, native Hawaiian, or pacific islander
- Age 45 or over
- Family history of DM
- Prehypertension or hypertension
- Low levels of high-density lipoprotein, elevated triglyceride levels
- History of gestational diabetes or gave birth to a baby weighing 9 pound or more
- History of cardiovascular disease
- Depression
- Polycystic ovary syndrome
- Physical inactivity
- Smoking
- > 5 hours of sleep / day
- Exposure to environmental pollutants

Presentation of Diabetes Mellitus II

Mr. B presents to his PCP's office with complaints of increase fatigue, increase thirst, and numbness in his toes. Mr. B is a 53 year-old Native American male. Mr. B's past medical history is reviewed and his height, weight, and vital signs are obtained, the abnormal values as followed: BMI 31, BP 190/10. Furthermore into Mr. B's exam, a fasting blood glucose test and lab value known as a HbA1C is ordered. Results: Blood glucose 209, HgA1C 8.3%. Mr. B has met the criteria of the diagnosis of type 2 diabetes. Mr. B is advised to be active for 30 minutes a day, and to have make some dietary changes for a healthful diet. Mr. B will also be started on oral antiglycemic medication, such as Metformin, to lower and manage his blood glucose levels. Mr. B is also started on an antihypertensive medicine, as well, as a antihyperlipidemia medication related to his physical exam findings and lab value results. Mr. B is to have a follow-up with his PCP in 3 months for repeated blood work (Kajal, Cadet, Hirani, & Thomas, 2018).

Pathophysiology of Type II Diabetes

The pathophysiology of DMII is complex, in that it involves multiple organs. The Islet of Langerhans located within the pancreas produces beta cells that secrete insulin, and alpha cells that secrete glucagon (McCulloch & Robertson, 2018). Although the etiology of DMII is still unclear, it is thought to be a multifactorial caused by genetic predisposition, obesity, hypertension, lack of exercise, environmental factors, and other comorbid health conditions and medications (Kahn, Cooper, & Del Prato, 2014). The release of free fatty acids and adipokines by excess adipose tissue or fat can cause inflammation that can lead to insulin resistance. (Waddell, 2017). In DMII, the response of insulin-sensitive tissues, such as liver, muscle, and adipose tissue becomes insulin resistant (McCulloch & Robertson, 2018). Initially the beta cells release insulin in response to glucose in the blood. Insulin reduces blood glucose levels by binding to the insulin receptors of insulin-sensitive tissues, when activated the insulin receptors causes the glucose transporter (GLUT) protein inside the cell to fuse to the cell membrane allowing glucose, aminoacids, and fatty acids to be transported into the cell (McCulloch & Robertson, 2018). In DMII, when the insulin-sensitive tissues do not respond to the normal amount of insulin that is secreted, the beta cells secrete more insulin in order to maintain normal blood glucose concentrations; this is done by beta cell hyperplasia and hypertrophy to secrete more insulin to maintain blood glucose homeostasis (McCulloch & Robertson, 2018). Along with insulin, beta cells also secrete islet amyloid polypeptide or amylin, overtime amylin builds up in and aggregates in the islets thus produces amyloid deposits (McCulloch & Robertson, 2018). Eventually the beta cells become dysfunctional as a result of chronic hyperglycemia and become hypoplasia and hypotrophy (McCulloch & Robertson, 2018). As a result of beta cell dysfunction, further glycaemia occurs and impaired glucose intolerance is present which leads to the development and diagnosis of DMII (Kahn, Cooper, & Del Prato, 2014).



Born et al. (2014). Retrieved from http://u.osu.edu/diabetestype2/diagnosis/

Type II Diabetes Mellitus core defects (Waddell, 2017, p. 24):

- Decreased insulin secretion by beta cells in the pancreas
- Decreased incretin effect in the gut
- Increased lipolysis
- Increased reabsorption of glucose in the kidneys
- Decreased glucose uptake in the muscles
- Neurotransmitter dysfunction
- Increased hepatic glucose production (in response to glucagon)
- Increased glucagon secretion by islet alpha cells

Significance of Pathophysiology

"Approximately 90% to 95% of newly diagnosed cases of diabetes are DMII" (Waddell, 2017, p.28). With the risking of DMII incidence amongst individuals, it it important for healthcare providers to identify, screen those who are at risk, and treat hyperglycemia and DMII early to prevent long-term complications of hyperglycemia. Chronic hyperglycemia over time damages both, micro and macro blood vessels causing vision loss, nerve damage, kidney disease, cardiovascular disease, risk of stroke, and poor blood circulation (Cornell, 2015).

Implications for Nursing Care

There are many nursing interventions and implications for DMII, the most important being to educate patients on prevention of diabetes, but also education on how to manage blood glucose with DMII. Education includes diet and lifestyle modification to prevent DMII education (Ley et al., 2016):

- Healthful diet
- BMI of 25 or less
- Exercise for at least 30 minutes/day
- Smoking cessation
- Consume alcohol in moderation
- Minimum of 7 hours of sleep/night
- Behavior support and counseling
- Glycemic control

Education for those at risk or with DMII:

- Follow up every 3-4 months with PCP for proper glycemic control and management: assess AIC levels, monitor blood glucose logs, evaluate medication dosages and adherence, screen for weight gain or loss, measure BP and lipid levels, assess foot care and nutrition, annual urinalysis, health exam, dental care, and glaucoma screening (Kajal, Cadet, Hirani, & Thomas, 2018)
- Signs & Symptoms of hypoglycemia: lethargy, confused, dizziness, sweaty, cool to touch (Palmer, 2017)
- Signs & Symptoms of HHS and DKA: "fruity" breath, confused, lethargic, tachypnea, tachycardia, ketones in urine, increase urination

Conclusion

In conclusion, DMII is a chronic illness that requires continuous medical evaluation to maintain normal blood glucose concentrations. Non-pharmacological and pharmacotherapy drug therapies are necessary for patients with DMII to maintain blood glucose levels and to prevent microvascular and macrovascular complications, and further organ complications. Effective and individualized glycemic control delays the progression and onset of associated long-term complications. The health provider's role is to screen and identify those at risk for developing DMII and to educate patients to provide self-care., such as living an active lifestyle, and consuming a healthy diet. Early identification of DMII can prevent the long-term complications that are caused by MDIII (Kajal, Cadet, Hirani, & Thomas, 2018).

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