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Type II Diabetes and its Treatment

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Signs and Symptoms.

1) Polyuria (McCulloch, Nathan, Wolfsdorf, & Mulder, 2015)
2) Polydipsia (McCulloch, et al., 2015)
3) Elevated glycated hemoglobin A1C
   A. A1C greater than 6.1% (McCulloch, et al. , 2015)
4) Increased BMI (McCulloch, et al.,2015)
5) Increased fasting glucose (McCulloch, et al.,2015)
6) Glycosuria (McCulloch, et al.,2015)
7) Acute changes in vision (McCulloch, et al.,2015)

Complications

1) Diabetic Retinopathy-Diabetes is one of the major causes of blindness (CDC, 1991).Chronic hyperglycemia causes increased capillary permeability and the capillaries become less functional (CDC, 1991). Due the this fact, microaneurysms form, retinal capillaries close, a formation of fibrous tissue and new vesselsare created, hemorrhages and/or retinal detachment are all signs of diabetic retinopathy (CDC, 1991).
   A. Glaucoma, cataracts and macular edema are complications resulting from poorly managed DM2
2) Diabetic Nephropathy-DM2's effect on the kidney is still widely unknown (CDC, 1991). Hyperglycemia and hypertension contribute to diabetic glomerulosclerosis, increased thickening of the glomerular basement membrane and mesangial expansion (CDC, 1991). Increased blood flow to the kidney from hyperglycemia cause an increased size and weight of the kidney (CDC,1991). Microalbuminuria (30 to 300 mg/day)is usually one of the first notable signs of kidney disease (CDC,1991).
3) Cardiovascular disease-patients with DM2 are 2 to 3 times more likely to have cardiovascular disease (CDC, 1991). Coronary artery disease and stroke are common complications as a result from chronic hyperglycemia. Atherosclerosis contributing to these complications is caused from abnormal platelet function, hyperinsulinemia, impaired blood flow and coagulopathies (CDC, 1991) Cardiovascular disease is the leading cause of death for patients with DM2 (CDC, 1991).
4) Diabetic Neuropathy-The three types are distal symmetrical polyneuropathy, focal neuropathy, and autonomic neuropathy. They result from poorly controlled blood sugar and decreased blood flow to the nerves (CDC, 1991).

Complications Cont'd

Distal symmetrical neuropathy is characterized by dysesthesia and paresthesia in the lower extremities (CDC, 1991). As the neuropathy progresses with poor glycemic control, varying degrees and pain and sensation loss occur (CDC, 1991). Focal neuropathy is an uncommon form associated a blood vessel occlusion. Symptoms are usually unsymmetrical in nature and have a sudden, acute onset (CDC, 1991). Autonomic neuropathy affects multiple body systems. The symptoms include: orthostatic hypotension, gastroparesis, constipation, diarrhea, fecal incontinence, bladder dysfunction and sexual dysfunction such as impotence and dyspareunia (CDC, 1991).

1)Lower extremity ulcers and amputations-they are a result of diabetic neuropathy and cardiovascular disease caused by DM2 (CDC, 1991). Due to the loss of sensation deformities can occur which lead to ulcerations (CDC, 1991). The ulcers are slow to heal due to the poor blood flow from peripheral artery disease (CDC, 1991). Unhealed ulcers become infected leading to gangrene and osteomyelitis (CDC, !991). These conditions often require amputation of part or all of the affect area (CDC, 1991). 50% of the nontraumatic amputations in the United States are a result of diabetes (CDC, 1991).

Dietary and Exercise Recommendations

Weight reduction through dietary modification can help improve glycemic control by decreasing insulin resistance and increase insulin secretion (McCulloch, Nathan, & Mulder, 2015). 30-60 minutes of moderate intensity aerobic activity is recommended most days of the week (McCulloch, Nathan, & Mulder, 2015). Weight lifting should be done at least twice per week (McCulloch, Nathan, & Mulder, 2015

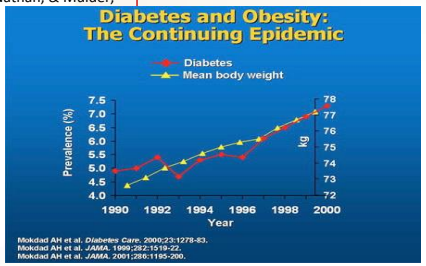
Dietary and Exercise Recommendations Cont'd

1) Carbohydrates-50% of a patients caloric intake should consist of carbohydrates; but of that, less that 10% should be made up of sugars (Bussell, 2014).
2) Sugar-it is recommended that less than 90g of sugar be consumed per day. However, these sugars should be naturally found in food such as honey, fruit, and milk (Bussell, 2014).
3) Fats-total fat intake should not exceed 70g per day. Mono-unsaturated fats are recommended such as oily fish, peanuts, avocado, olive oil etc. (Bussell, 2014).
4) Fiber- 24g per day are recommended. Soluble fiber has been shown to reduce cholesterol and improve blood sugar control (Bussell, 2014).
5) Proteins-should make up 25% of a patients caloric intake. It helps increase the feeling of satiety allowing the patient to go longer periods without feeling hungry (Bussell, 2014).

Nursing Implications

Education, preventative care, symptom recognition and appropriate treatment are imperative for patients at risk and/or patients with type II diabetes (CDC, 1991). Nurses are the front line when it comes helping patient manage DM2. They must educate patients on everything from proper diet and exercise to when and how to take their medications (CDC, 1991). Nurses must be knowledgeable on preventative care to help patients monitor and avoid the many complications associated with DM2 (CDC, 1991). Education on blood sugar management and if the prescribed treatment is properly controlling the patient's blood sugar is largely on the nurses shoulders (CDC, 1991). Most importantly, nurses must recognize the signs and symptoms of uncontrolled diabetes in an undiagnosed patient (CDC, 1991).

Graphic retrieved from www.salemfreemedclinic.org



Introduction

Approximately 28.9 million Americans have been diagnosed with type II diabetes (Jia-Haur Hu, Lin, Miller, Nguyen, & Nguyen, 2014). It is a disease that can cause or potentiate numerous comorbidities that negatively affects multiple body systems if left untreated (Jia-Haur Hu, et al., 2014). In 2012, the total estimated cost for treatment of type II diabetes in America was an astronomical $245 billion (Jia-Haur Hu, et al., 2014).

Type II diabetes is a result of many cellular processes that lead to insulin resistance resulting in increased blood glucose levels (Jia-Haur Hu, et al., 2014). Treatments for type II diabetes include several different classifications of oral hypoglycemic medications, insulin injections, exercise, and a modified diet (Jia-Haur Hu, et al., 2014). With the number of Americans being diagnosed with type II diabetes on the rise, a strong understanding of the pathophysiology of the disease and its treatments cannot be disregarded.

Pathogenesis

Type II diabetes (DM2) can result from genetic and environmental influences or both. Making it difficult to determine the specific cause in each patient (McCulloch, Robertson, Nathan, & Mulder, 2014). However, DM2 is characterized by hyperglycemia, insulin resistance, and relative impairment in insulin secretion (McCulluch, et al., 2014). Hyperglycemia can impair pancreatic beta cell function thus perpetuating the body's insulin resistence leading to an even worse metabolic state (McCulluch, et al., 2014). Insulin secretion helps move glucose into the cell, which is mediated by glucose transporter 2 (GLUT-2) (McCulluch, et al., 2014). The alteration of GLUT-2 can be linked to a high- fat diet in a mouse model decreasing insulin secretion in those mice (McCulluch, et al., 2014). A large majority of DM2 patients have shown to have genetic susceptibility to insulin resistance (McCulluch, et al., 2014). This fact could be attributed to idea that insulin resistance becomes worse with increasing age and body weight (McCulluch, et al., 2014). Individuals with the genetic predisposition for DM2 so signs of increased insulin resistance causing hyperglycemia. In a cyclic manner, that hyperglycemia contributes to future weight gain in those patients (McCulluch, et al., 2014). Adipose tissue releases leptin, adiponectin, Tumor necrosis factor alpha, and resistin, which relate to increasing insulin resistance (McCulluch, et al., 2014).

The dysregulation of fatty acid metabolism and decreased glycogen synthesis can be an inherited defect than contributes to insulin resistance (McCulluch, et al., 2014). The process of converting proinsulin to insulin is shown to be impaired in the beta cells of DM2 patients. Increased amounts of proinsulin are still produced in DM2 to account for increasing demands but are not able to be converted into insulin due to this beta cell dysfunction (McCulluch, et al., 2014). Amylin is secreted along with insulin in the pancreatic beta cells. Increased amounts of amylin reduce glucose uptake and inhibit insulin secretion (McCulluch, et al., 2014). Amylin concentrations are found to be extremely low in patients with DM2 patients (McCulluch, et al., 2014). Other genetic factors for DM2 include ethnicity and having familial history of DM2. African americans are up to six times more likely to have DM2 (McCulluch, et al., 2014). Having a first-degree relative can make a patient up to ten times more susceptible to DM2 (McCulluch, et al., 2014). Maturity onset of diabetes of the young or MODY is an autosomal dominate form of insulin resistance and impaired insulin secretion from a mutation on chromosome 7 effecting glucokinase genes (McCulluch, et al., 2014). Inflammation markers such as C reactive protein, IL-6, plasminogen activator inhibitor-1, tumor necrosis factor alpha, and white cell count can all be linked between obesity and DM2, which correlates to insulin resistance (McCulluch, et al., 2014).

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Treatments

1) Metformin-is the most widely used oral DM2 medication on the market. It decreases hepatic insulin production, decreases reabsorption of intestinal glucose and decreases insulin resistance (McCulluch, Nathan, Mulder, 2014). Metformin can be used in conjunction with other DM2 medications or insulin therapy. Combination therapy with DPP-4 has shown to decrease patient's HgA1c more than just treatment with Metformin alone (Liu & Wu, 2014).
2) DDP-4 inhibitors-The inhibit the enzyme GLP-1, which decreases fasting plasma glucose, post prandial glucose, and HbA1C. The common side effects for this class is nausea, headache, renal and hepatic dysfunction (Eyadeh, & Jennings, 2015)
   A. This class consists of Sitagliptin, Saxagliptin, Linagliptin, Alogliptin and Vildagliptin (Eyadeh, & Jennings, 2015).
3) SGLT inhibitors- they inhibit renal absorption of glucose so that in is dumped in the urine (Eyadeh, & Jennings, 2015). The SGLT inhibitors contribute to lower blood pressure, weight loss, decreased lipids and decreased uric acid (Eyadeh, & Jennings, 2015).
   A. This class consists of Dapagliflozin, Canagliflozin, Empagliflozin (Eyadeh, & Jennings, 2015).
4) GLP-1 agonists-incretion secreted from the intestinal mucosa, which is released into circulation with the ingestion of food (Eyadeh, & Jennings, 2015). GLP-1 agonist mimic incretion thus stimulation the secretion of insulin (Eyadeh, & Jennings, 2015). Nausea is the main side effect for this class medication (Eyadeh, & Jennings, 2015).
   A. This class consists of Exenatide, Liraglutide, Lixisenatide, and Albiglutide (Eyadeh, & Jennings, 2015).
5) Alpha glucosidase inhibitors-slow the absorption of intestinal glucose causing a slower rise in postprandial blood sugars (McCulluch, Nathan, Mulder, 2014). However, they are usually poorly tolerated due to their GI side effects (McCulluch, Nathan, Mulder, 2014).
6) Insulin- It is usually added when oral agents become less effective and lifestyle changes are no longer appropriately controlling blood sugar. It can be added as an adjunct to oral medication or used as primary therapy. Disadvantages for the use of insulin therapy include weight gain and hypoglycemia (McCulloch, Nathan, Mulder, 2015)
   A. Basal insulin- intermediate to long acting insulin such as NPH, NPL, glargine, or detemir. It is given once or twice a day to suppress hepatic glucose production in a fasting state. (McCulloch, Nathan, Mulder, 2015).
      i. Onset-2 hours
      ii. Peak
         a. NPH (4-12 hours)
         b. NPL (6 hours)
         c. Glargine (no peak)
         d. Detemir (3-9 hours)
      iii. Duration
         a. NPH (18-28 hours)
         b. NPL (15 hours)
         c. Glargine (20-greater than 24 hours)
         d. Detemir (6-24 hours)
   B. Prandial dose insulin- short acting or rapid acting insulin such as regular, lispro, aspart, and glulisine given after meals to counteract food absorption (McCulloch, Nathan, Mulder, 2015).
      i. Onset
      a. Regular (30 minutes)
      b. Lispro, apart and glulisine (5-15 minutes)
      ii. Peak
      a. Regular (2-4 hours)
      b. Lispro, apart and glulisine (45-75 minutes)
      iii. Duration
      a. Regular (5-8 Hours)
      b. Lispro, apart and glulisine (2-4)
      C. Afrezza-is a recombinant regular human insulin inhalation powder (Jia-Haur Hu, et al., 2014). It is market as the alternative to injectable short acting insulin therapy (Jia-Haur Hu, et al., 2014).
7) Bariatric surgery-this is not a primary treatment for DM2 but helps treat an underlying cause of DM2. The reduction of weight is associated with a decrease in insulin resistance. More than 50% of the patients who undergo bariatric surgery decrease their HgA1c within normal limits without medication (Wilding, 2015)

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