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Diabetic Gastroparesis

Noreen Burris Welch

Otterbein University, noreen.burriswelch@otterbein.edu

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Burris Welch, Noreen, "Diabetic Gastroparesis" (2015). *Nursing Student Class Projects (Formerly MSN)*. 108.

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Diabetic Gastroparesis

Noreen Burris Welch, BSN, RN, ACM

Otterbein University, Westerville, Ohio

Introduction

According to the 2014 Centers for Disease Control (CDC) 9.3% of the United States Population have diabetes (National diabetes statistics report, “2014, p.1). One of the complications of diabetes is gastroparesis. Gastroparesis is “a delay in the emptying of ingested food in the absence of mechanical obstruction of the stomach or duodenum ” (Koch & Calles-Escandon, 2015, p. 40). Mortality in the diabetic patient with gastroparesis is higher than the diabetic patient without gastroparesis (Koch & Calles-Escandon, 2015). Patients with either type 1 diabetes (T1DM) or type 2 diabetes (T2DM) may experience he clinical symptoms of early satiety, prolonged fullness, nausea, and vomiting, and have difficulties with management of blood glucose levels, nutritional issues, and other drug absorption issues (Koch & Calles-Escandon, 2015). Health care providers may see patients in a variety of settings, with and without known diagnosis of gastroparesis and must be aware of the symptoms, the potential diagnosis, the appropriate testing, and the options for treatment. Advances in the treatment of gastroparesis surround the increased understanding of the pathophysiology behind gastroparesis.

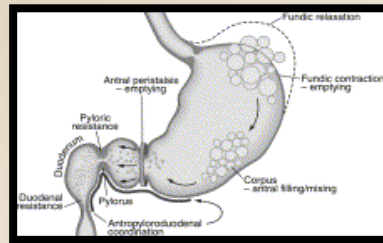
Signs & symptoms

- nausea
- early satiety
- bloating
- abdominal or epigastric pain
- impaired glycemic control
- malnutrition
- poor drug absorption
- poor quality of life
- frequent hospitalizations (Shin & Camilleri, 2013)

Pathophysiology

The normal activity in gastric motility involves three areas of action.

- The fundus, which relaxes with a volume of food ingested and requires an intact vagus nerve and enteric neurons (Koch & Calles-Escandon, 2015)
- The corpus and antrum, which produce recurrent waves at a frequency of 3 cycles per minute, turning digested food solids into finer material called chyme. These peristaltic waves are controlled by gastric pacemaker cells call Interstitial cells of cajal (ICCs) (Koch & Calles-Escandon, 2015).
- The emptying of chyme through the pylorus into the duodenum. The pylorus regulates the volume and particle size of chyme and is affected by catecholamine and peptide releases (Koch & Calles-Escandon, 2015).



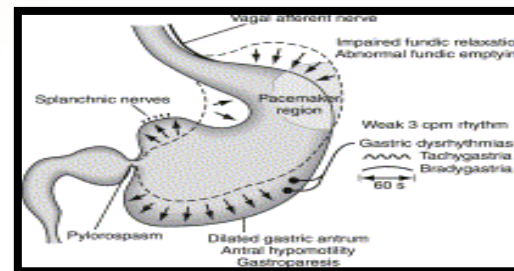
Normal gastric neuromuscular function (Koch & Calles-Escandon, 2015)

There are several alterations suspected in gastroparesis.

- The most common abnormality is loss or depletion of ICCs . ICCs regulate contractility and impaired ICCs results in gastric dysrhythmias (Farrugia, 2015).
- Damage to the vagus nerve innervation to the stomach, may lead to motor abnormalities and abnormal relaxation of the pylorus (Farrugia ,2015).
- Evidence has shown smooth muscle degeneration and end stage evidence may include fibrosis with eosinophilic inclusion bodies (Farrugia, 2015).
- Loss of neurotransmitters, such as neuronal nitric oxide synthase (nNOS) have been seen and are thought to be reversible (Farrugia, 2015). Nitregic neuron loss and ICC loss is thought to account of the poor fundic relaxation and decreased gastric capacity that is seen in gastroparesis (Koch & Calles-Escandon, 2015).
- Fibroblast-like cells (FLCs), similar to ICCs are involved in neurotransmission, and may be decreased in gastroparesis (Farrugia, 2015).
- Alterations in the balance of pro-inflammatory M1 macrophages and anti-inflammatory M2 macrophages of the stomach wall muscle are suspected to become disrupted in gastroparesis (Farrugia, 2015). M1 macrophages are associated with the development of delayed gastric emptying (Farrugia, 2015).

Significance of Pathophysiology on Gastroparesis

- Understanding the various potential alterations that lead to gastroparesis can affect the treatment or management of symptoms and mode of testing.
- The gold standard test used for evaluation of gastric emptying is the gastric emptying study (GES), although associations are weak between abnormal results and patient symptoms due to various pathophysiology mechanisms (Shin & Camilleri, 2013).
- Alternative methods of testing exist (Shin & Camilleri, 2013).
- Assessment of symptoms and other factors, such as age, sex, comorbidities, patient preference, and test availability should contribute to the decision of mode of testing (Shin & Camilleri, 2013).
- Treatments are palliative and empiric in focus and not curative (Pasricha, 2015).
- Common treatments aimed at increasing gastric emptying (Pasricha, 2015).
- Nutritional support, fluid and electrolyte restoration, glycemic control, (Acosta & Camilleri, 2015) .
- Six or more meals or snacks a day are needed to meet nutritional needs and include restricted fat and fiber intake: tolerance is improved with liquids or ground foods (Rees Parish, 2015).
- Prokinetic medication help gastric emptying by “restoring the synchronicity between delivery of food and hormone and peptide release, but it does not target the underlying defects” (Farrugia, 2015, p. 6).
- Metoclopramide, a dopamine 2 receptor agonist, is the only approved prokinetic medication in the United States; use is limited to less than 13 weeks of treatment (Acosta & Camilleri, 2015).
- Domperidone is another dopamine 2 agonist, but use is considered investigational (Acosta & Camilleri, 2015).
- Antiemetic medications are often utilized for control of nausea, but one study suggests that antiemetics show modest benefit in comparison to non- use (Hasler, 2015).
- Opiate analgesia are often used but use could exacerbate nausea and vomiting and also slow gastric emptying (Hasler, 2015).
- Hyperglycemia treatment with insulin, may improve and normalize ICCs (Koch & Calles-Escandon, 2015).
- Gastric pacemakers provide gastric electrical stimulation aimed at controlling nausea and improving vagal function when other therapies failed ; more understanding of gastric mapping is needed (Sarosiek et al., 2015).
- A jejunostomy tube may be needed for patients experiencing gastroparesis in order to maintain hydration, nutrition, and glycemic control.
- Newer agents are being studied and developed by targeting different pathways and include serotonin receptor agonists, ghrelin agonists (Acosta & Camilleri, 2015).



Neuromuscular function in gastroparesis (Koch & Calles-Escandon, 2015)

Implications for Nursing Care

Having an understanding of the diagnosis of gastroparesis, symptom recognition, and understanding of the behind it, is important for nurses in a variety of roles. Diabetic patients are often afflicted with gastroparesis. Gastroparesis affects glycemic control and lack of glycemic control can lead to gastroparesis (Koch & Calles-Escandon, 2015). Nurses can have pivotal role in diabetic education affecting glycemic control. Dietary education in tandem with dietary professionals, can also provide symptomatic relief from those suffering from gastroparesis. Knowledge of gastroparesis and medication therapy can also benefit patients through nurse request for appropriate medication, and patient education of those drugs. Advance practice nurse knowledge of gastroparesis, can lead to improvement in patient assessments, testing, symptom management, awareness of potential electrolyte imbalance, and patient nutrition and diabetes management. Since the symptoms of gastroparesis lead to frequent medical visits and hospitalization, sensitivity to patient quality of life and screening for symptoms of depression may be important.

Conclusion

Diabetic patients often experience gastroparesis. A cure for gastroparesis is not present. Research and better understanding of the pathophysiology of gastric motility and function is needed to further advance the treatment of gastroparesis and its symptoms. Until then, knowledge of the prevalence of gastroparesis in diabetics, symptom awareness, testing, and appropriate choosing of available medical therapies is important. Support of the patient in nutrition and glycemic management is needed, as well as support of the patient’s emotional well being.

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