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Diabetic Gastroparesis: A Risk Factor for Perioperative Aspiration

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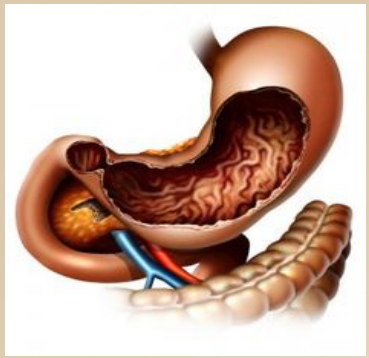
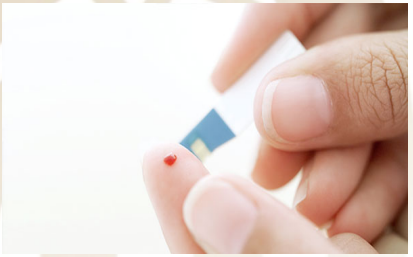


Introduction

Aspiration of gastric contents represents the most significant cause of airway-related death in the perioperative setting. Depending on the risk factors, perioperative aspiration effects as many as 1 in 900 patients, often leading to severe respiratory complications, surgical airway, prolonged hospital stay or death (Robinson & Davidson, 2014). Each anesthetic plan should address aspiration risk factors, requiring knowledge of conditions known to predispose patients to aspiration of gastric contents. Delayed gastric emptying, also known as gastroparesis, is one such condition. As the incidence of diabetes mellitus continues to rise, diabetic gastroparesis has become a common, predisposing risk factor for perioperative aspiration (Farmer & Aziz, 2012). According to the World Health Organization, 171 million people suffer from diabetes with 12% of those reporting symptoms consistent with gastroparesis (2012). As such, knowledge of diabetic gastroparesis and its complications should be part of the preoperative decision-making process.



Underlying Pathophysiology

Diabetic neuropathies, including autonomic neuropathy, are long-term complications of impaired insulin secretion, insulin resistance and excess glucose production (Stoelting & Miller, 2011). Although the exact mechanism of diabetic neuropathy is unknown, vagal dysfunction has been contributed to chronic exposure to high blood glucose levels, resulting in damage to the capillaries responsible for supplying nutrients and oxygen to nerves. Studies also suggest deposition of sorbitol within nerves, lack of nitric oxide secondary to excess glucose metabolism and free radical production secondary to chronic inflammation may also cause neurovascular damage (Bielefeldt, 2012). Co-morbidities associated with diabetes mellitus also contribute to neurovascular damage including hypertension, peripheral vascular disease and hyperlipidemia.

Pathophysiological Processes

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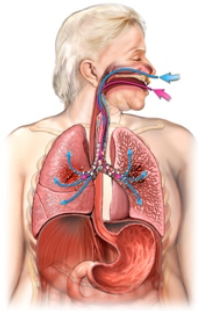
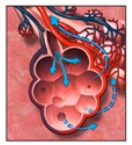
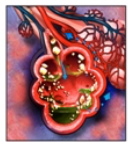
Diabetic gastroparesis is primarily due to autonomic neuropathy of the gastrointestinal tract, namely the vagus nerve. The vagus nerve is responsible for parasympathetic control of the digestive tract as well as communication between the central nervous system and enteric nervous system (Vanormelingen, Tack & Andrews, 2013). In the presence of an intact vagus nerve, the fundus of the stomach will relax and distend in response to a food bolus. Vagal nerve stimulation also aids in the propulsion and grinding of food followed by coordinated emptying of gastric contents into the duodenum (2013). Damage to the vagus nerve secondary to diabetes mellitus disrupts these mechanisms, resulting in delayed gastric emptying.

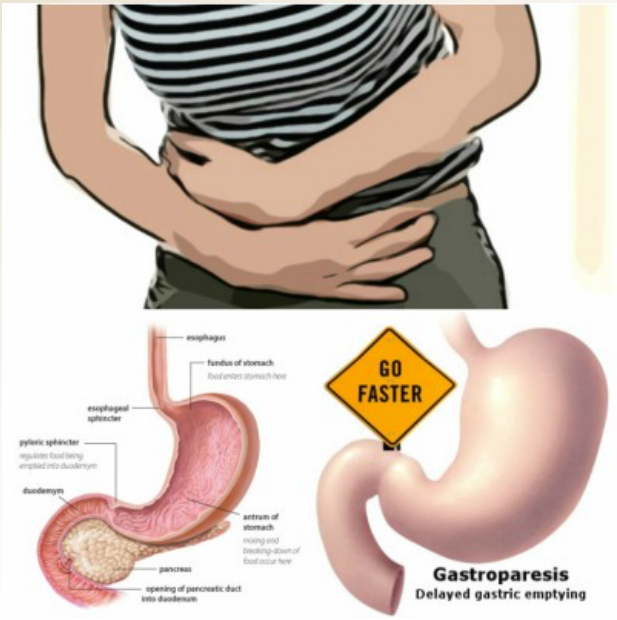






Implications for Nursing Care

Diabetic gastroparesis is characterized by prolonged feelings of fullness and increased gastric volumes following the ingestion of solid food (Farmer & Aziz, 2012). The resulting "full stomach" in conjunction with loss of protective airway reflexes during anesthesia predisposes these patients to perioperative aspiration even when strict NPO guidelines are followed. As such, knowledge of diabetic gastroparesis and its complications should be part of the preoperative decision-making process. Perioperative treatment options include administration of histamine-2 receptor antagonists, proton pump inhibitors, gastric pro-kinetics and/or non-particulate antacids (Nagelhout & Plaus, 2014). Supraglottic airways are usually contraindicated in patients with gastroparesis due to increased risk of aspiration. Rapid sequence intubation or awake fiberoptic intubation are known to decrease the risk of aspiration in patients with a presumed full stomach.

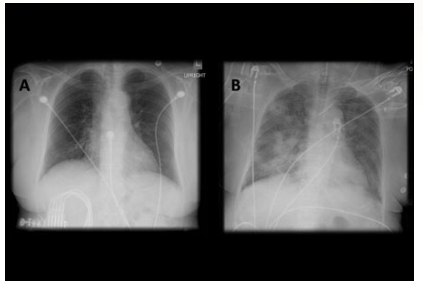


Conclusion

Diabetic gastroparesis is one of several long-term complications of diabetes mellitus. It is characterized by delayed gastric emptying secondary to autonomic neuropathy. As the incidence of diabetes mellitus continues to rise, it becomes even more important for anesthetists to address the risk of aspiration posed by diabetic gastroparesis. Currently, aspiration of gastric contents accounts for nearly half of all major airway complications in the operating room, often due to incomplete assessment of risk factors (Robinson & Davidson, 2013). Understanding the pathophysiology is the first step in recognizing diabetic gastroparesis as a significant risk factor for aspiration. Every effort should be made to mitigate perioperative aspiration, starting with the ability to identify risk factors where they exist.

Significance of Pathophysiology

Diabetes represents a significant underlying cause of morbidity and mortality inside and outside of the operating room. It is also the most common endocrine disorder seen in the operative setting (Nagelhout & Plaus, 2014). Furthermore, diabetic gastroparesis is significant in the perioperative setting where increased residual gastric volumes increase the risk of pulmonary aspiration. Understanding the pathophysiological mechanism behind diabetic gastroparesis also aids in the understanding of other serious, long-term complications of poor glucose control. Because neurovascular damage is not limited to the gastrointestinal tract, those suffering from diabetic gastroparesis may also exhibit arterial damage, cataracts, sensory and peripheral neuropathies and infection (2014). Thus, the pathophysiology of diabetic gastroparesis is significant because it suggests the presence of other life-threatening conditions.



Signs & Symptoms

In the early stages, diabetic gastroparesis may be asymptomatic. As the degree of autonomic neuropathy progresses, symptoms often include post-prandial fullness, abdominal distension, early satiety, nausea, vomiting, diarrhea and abdominal pain. A delay in gastric emptying can also result in poor glycemic control secondary to unpredictable increases in blood glucose levels (Vanormelingen, Tack & Andrews, 2013). These patients may have difficulty timing insulin administration as food absorption is delayed. Additional symptoms associated with autonomic neuropathy include orthostatic hypotension, resting tachycardia, prolonged QT interval, bowel and bladder dysfunction and impotence. Myocardial infarction and signs of myocardial ischemia may also be painless in patients with autonomic neuropathy (2013). Autonomic neuropathy tends to occur in conjunction with other forms of neuropathy including peripheral and retinal.

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