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Dumping syndrome Following Gastrointestinal Surgery: A Review of Pathophysiology Diagnosis and Management

***Saja nabeel taqi*[1]. *Hawraa Dheyaa Rasool*[2], *Ameer Mohammed Ridhab*[3]**

[1.2.3]*Department of Laboratory and Clinical Sciences , Faculty of Pharmacy, Jabir Ibn Hayyan University for Medical and Pharmaceutical Sciences, Al-Najaf, Iraq.*

Abstract

Dumping syndrome originated as a group of gastrointestinal symptoms experienced by individuals who had undergone surgery for peptic ulcer. It is now seen quite frequently in those who have had surgery for upper gastrointestinal (GI) cancer, bariatric surgery, or anti-reflux surgery. Early and late dumping syndrome have distinct symptom patterns. Symptoms that happen less than 1 hour after eating are considered early dumping syndrome and consist mainly of gastrointestinal symptoms resulting from osmotic fluid shifts in the intestinal lumen. Late dumping syndrome symptoms occur between 1-3 hours after eating and consist primarily of vasomotor symptoms due to reactive hypoglycemia from the over-secretion of insulin in response to the influx of glucose into the portal circulation.

According to the existing guidelines, evaluation of HR; running laboratory tests; and comparing results to diagnostic criteria for postprandial dumping syndrome with a glucose challenge test are recommended assessments for the diagnosis of dumping syndrome. Dumping syndrome is diagnosed based on Worsening symptoms after meals, results of provocation tests, lab investigations, an individual's history of gastric surgery or an inherent condition of rapid gastric emptying (i.e.: dumping syndrome).

Most cases of Dumping Syndrome can be treated with changes in diet. If diet does not help, then medical or surgical options can be recommended by a healthcare service provider, but there is little evidence to support the effectiveness of either option.

Keywords: Dumping syndrome, Gastrointestinal, Gastrectomy, Somatostatin .

Introduction

Dumping syndrome (DS) describes a unique set of signs or symptoms experienced after eating due to rapid passage of food from the stomach, a syndrome that was first diagnosed and named over 100 years ago as the result of gastric surgery. DS was thoroughly studied due to the relatively high prevalence of gastrectomies for peptic ulcer diseases in the 20th century. Recently, the significance of DS is being

recognized again, as there has been a proliferation of bariatric surgery in recent years. However, the condition continues to lack precise definition and is still inadequately understood [1].

The phenomenon of dumping syndrome involves changes occurring within both the gastric-emptying process resulting from alterations to both the alimentary canal (the tract through which food travels) and neurological control (innervated) of that process will occur. Alterations to the anatomy produce early gastric emptying and food is rapidly transported to the small intestine 【2】. Symptoms are experienced as a mix of both GI symptoms (abdominal pain/Nausea/abdominal distention/diarrhea) as well as vasomotor symptoms (sweating/heartracing/redness/palpitations/tachycardia/low blood pressure/lightheadedness/rarely loss of consciousness) 【3】. Currently, treatment for dumping syndrome includes dietary supplementation, medication/surgery or lifestyle changes. Patients should eat smaller amounts per meal and wait at least 30 minutes after eating before drinking anything. It is also recommended that patients eat foods that are primarily made up of protein and fiber, and to include plenty of fruits and vegetables. [5].

Clinical Features of Dumping Syndrome

Both types, early and late types of dumping syndrome, have different timings of symptom onset that will occur following meals as well as different underlying pathophysiological mechanisms. 70% of individuals with dumping syndrome have only the early type, while less than 30% have both types. Furthermore, only a small percentage will develop late dumping syndrome [6].

(1.) Early dumping occurs 1 hr after stomach emptying, resulting in an emptying syndrome with two different types of symptoms: systemic symptoms and abdominal symptoms. Abdominal symptoms include borborygmi, bloating, nausea, pain, retching, and diarrhea. Systemic symptoms may include tachycardia, palpitations, fatigue, a feeling to lie down after eating, headache, flushing/pallor, lightheadedness, hypotension, sweating, and possibly syncope.

After eating, late dumping syndrome generally begins within 1 to 3 hours. Both the autonomic reactivity (palpitations, sweating, tremors, irritability) and reactive (neuro) hypoglycemia (confusion, weakness, tiredness, hunger, syncope) are the symptoms for late dumping syndrome. [9, 10].

Causes

Dumping syndrome (DS) occurs in anywhere from 25% to 50% of patients following gastric surgery, but only in about 10% of those cases is the dumping syndrome severe or persistent enough to warrant a diagnosis of dumping syndrome. Antrectomy and V & P usually cause dumping syndrome about 10% of the time; with the primary cause being Nissen fundoplication for gastro-esophageal reflux disease in pediatric patients. The most common cause of DS in current adults is gastric bypass surgery, with DS rates of as much as 75%.

Roux-en-Y gastric bypass, resulting in a gastrojejunostomy, was previously the predominant kind of weight loss procedure in the USA and is therefore a significant contributor to DS [13].

The small remnant after gastric bypass surgery limits the volume of food that can be consumed after surgery. A sleeve gastrectomy is a different type of bariatric surgery where the stomach is shaped into a tube that is congruent with the smaller curvature of the stomach. Dumping syndrome has been associated with diabetes mellitus and some idiopathic cases of dumping syndrome. There may be a connection between diabetes mellitus and rapid gastric emptying; this has been especially true for patients with type II diabetes who experience a long history of early Wallerian degeneration and have evidence of damage

or dysfunction of their first vagus nerve. [8]

pathophysiological

The pathophysiological mechanisms behind these occurrences are not well understood; however, it is theorized that early dumping occurs due to rapid movement of food into the small intestine and accompanying shifts in osmotic fluid between the vessels (intravascular zone) and intestines [3].

Upper GI surgery impacts motility of the stomach in various ways, such as through vagal denervation, decreased stomach size and removal of the pyloric barrier. These factors may lead to the rapid transfer/emptying of the contents of the stomach into the small intestine after meals. A fluid shift occurs from the vascular space into the intestinal lumen due to the hyperosmolar nature of the contents of the small intestine. This fluid shift may also lead to symptoms of fullness, discomfort, diarrhea, and/or distension of the bowel, in addition to causing abdominal symptoms. An increase in hematocrit levels, either within the first hour after a meal or during a test for dumping syndrome, has been utilized to support the existence of such a fluid shift [14].

Another mechanism is that there is increased production of various gastrointestinal hormones resulting in: 1) the stimulations of the secretion and motility of the GI Tract 2) the increase in release of vasoactive substances that will have an influence on systemic and abdominal blood flow and vasomotor activity.

Much attention has been placed on incretin hormones released by the small intestine in recent years. Incretins act to stimulate insulin release after a high blood sugar level, resulting in reactive hypoglycemia. [15,17].

Epidemiology

How much surgery there was done, how complex it was categorized and how well it was diagnosed all directly impact the numbers of people with dumping syndrome. From findings based on laparoscopic and open vag VPID; about 20% of patients after vagotomy/pyloroplasty, approximately 40% of people with sleeve gastrectomy (SG) or Roux-en-y gastric bypass (RYGB) are symptomatic, and some studies show nearly 50% post-esophagectomy have had these dumping symptoms [6]. The rapid growth in bariatric surgery performed using either SG/RYGB has resulted in a larger number of individuals worldwide developing the symptoms associated with dumping. [18].

Diagnosis

Dumping syndrome is diagnosed through a number of methods, including assessments of laboratory values, conducting provocations, assessment of increasing symptoms that occur after meals and other causes that lead to rapid gastric emptying, as well as using various scales and scores for diagnosing dumping syndrome, including Sigstad Score [19], PGSAS-45 (Post-Gastrectomy Syndrome Assessment Scale) [20–22] and DSRS (Dumping Symptom Rating Scale) [23].

The diagnosis was confirmed with a provocative test, then appropriate incremental treatments commenced, beginning with diet changes; followed by adding food viscosity modifiers or glycosidase inhibitors to their diet; and then adding somatostatin analogues in severe cases. There are multiple emerging treatments that target gut motility, peptide hormonal players and the occurrence of hypoglycaemic episodes that are under evaluation [24]. Clinical assessment of these patients did not occur until 1970, according to Sigstad [25].

Diagnostic tests such as the Sigstad Score Scale and the Arts Dumping Questionnaire identify whether you are experiencing significant symptoms of the syndrome. According to the Sigstad scoring system,

scores of 7 or more following glucose consumption are considered diagnostic based on the presence of multiple symptoms characteristic of the syndrome [26].

According to the ADA, the oral glucose challenge is considered one of the most reliable tests to determine if a patient has Down syndrome. This test helps confirm Down syndrome by examining physical signs and confirms Down syndrome has occurred. The test involves a patient fasting overnight for 10 hours prior to taking a glucose challenge, with the patient consuming 50 grams of glucose (glucose challenge). After consuming the glucose challenge, the patient is monitored for blood pressure, hematocrit, pulse, and blood glucose every 30 minutes for 3 hours. If there is a > 3% increase in hematocrit and/or a > 10 beat per minute increase in pulse, the patient is considered to have early dumping syndrome; if a patient develops hypoglycemia, they are considered to have late dumping syndrome (27).

The use of laboratory tests, as well as x-ray imaging, is critical for the diagnosis of a patient with late dumping syndrome, including tests for plasma glucose, postprandial GLP-1 levels, postprandial insulin levels, and gastric emptying using scintigraphy. [28].

Treatment Options

Dumping syndrome can be treated with dietary changes, pharmacological therapy, and, in certain cases, surgical re-intervention or continuous enteral feeding. While certain treatments, like acarbose, are specifically licensed for late dumping, others, including somatostatin analogs, may be helpful in treating late and early dumping [3].

Diet

The primary treatment for dumping syndrome involves dietary modification. Patients are recommended to ingest modest portions of food multiple times daily. One should avoid rapidly digesting simple carbohydrates and fat-rich foods [29]. Intake of liquids through or inside 30 minutes post-meal should be discouraged to slow gastric emptying. Patients unresponsive to conservative treatments may consider pharmacological agents like acarbose and somatostatin analogues advantageous [30].

Acarbose

A drug called acarbose prevents the body from rapidly converting complex carbohydrates into simple sugars. As a result, postprandial glucose absorption decreases, which lowers insulin and gastrointestinal hormone release and relieves symptoms, especially those related to late dumping syndrome [31].

Somatostatin Analogues

For individuals with diagnosed dumping syndrome who fail to respond to or can't tolerate initial dietary changes and acarbose medication, somatostatin analogues offer a promising therapeutic option. Somatostatin analogues alleviate both early and late dumping symptoms by addressing several aspects of the pathophysiology of DS, including prolonging gastric emptying, slowing movement through the small digestive tract, reducing the release of gastrointestinal hormones, inhibiting secretion of insulin, and decreasing postprandial vasodilation. [32]

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