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

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Abstract

Dumping syndrome is a set of gastrointestinal symptoms initially explored in individuals after peptic ulcer surgery. Currently, it is more common among individuals who have undergone upper gastrointestinal cancer, bariatric or anti-reflux surgery. The symptomatology includes early as well as late dumping symptoms. Symptoms happening after 15 minutes to 1 hour after of meal ingestion in early dumping syndrome are primarily gastrointestinal and result from osmotically induced fluid changes from the bloodstream to the intestinal lumen. The symptoms of late dumping syndrome occurring 1 to 3 hours postprandially are primarily vasomotor in nature. They result from reactive hypoglycemia triggered by high insulin production that compensates excessively for the glucose influx into the portal circulation.

Current guidelines advocate for the assessment of heart rate, doing laboratory analyses, and juxtaposing these results with diagnostic standards through a provocation test utilizing a glucose solution. Dumping syndrome is diagnosed through symptoms that worsen postprandially, provocation tests, laboratory evaluations, gastric surgery history, or inherent conditions of rapid stomach…emptying. The majority of dumping syndrome cases are effectively managed with dietary modifications. If dietary modifications are ineffective, the healthcare professional may suggest medical or surgical procedures; however, the efficacy of these approaches is not well proven.

Keywords: Dumping syndrome, Gastrointestinal, Gastrectomy, Somatostatin .

Introduction

Dumping syndrome (DS) refers to specific symptoms that manifest postprandially, resulting from the accelerated transit of gastric contents through the stomach, a condition identified and named more than a century ago following gastric surgery. It was carefully examined because to the comparatively elevated

incidence of peptic ulcer-related gastrectomy during the 20th century. The significance of DS has resurfaced in recent decades due to the increase in bariatric surgery. Nonetheless, it remains not well delimited and insufficiently comprehended [1].

Dumping syndrome arises from alterations in the gastric-emptying process, resulting from anatomical changes in the gastrointestinal tract and disruption of its innervation. This results in the swift transit of food into the small intestine. [2].

Patients exhibit a combination of gastrointestinal symptoms (such as pain in the abdomen, nausea, bloating, and diarrhea) alongside vasomotor manifestations (including sweating, palpitations, flushing, tachycardia, hypotension, dizziness, and, infrequently, syncope) [3].

The current therapy for dumping syndrome involves dietary supplements, pharmaceutical and surgical procedures, as well as dietary monitoring. Patients ought to limit their food intake during each meal and postpone fluid consumption for no less than 30 minutes post-meal. [4]. Food high in protein and fiber, as well as fruits and vegetables, are also advised. [5].

Clinical Features of Dumping Syndrome

the clinical picture of Dumping syndrome shows two kinds: early and late dumping, differentiated by the timing of symptom onset after meals and their fundamental pathophysiology. Approximately 70% of patients having dumping syndrome experience early dumping exclusively, while 30% show both early and late dumping. A limited subset of patients has solely late dumping.[6].

(1). Early dumping happens during one hour following food consumption. Early dumping symptoms are categorized into abdominal and systemic manifestations. Abdominal symptoms include borborygmi, fullness, distension, discomfort, nausea, and diarrhea [7].

Systemic signs involving tachycardia, palpitations, weariness, a propensity to recline postprandially, diaphoresis, flushing or pallor, lightheadedness, hypotension, headache, and even syncope [8].

2) Late dumping begins 1 to 3 hours postprandially. The symptoms of late dumping syndrome primarily reflect reactive (neuro) hypoglycemia, characterized by fatigue, weakness, confusion, hunger, syncope, and loss of consciousness, as well as autonomic reactivity, showed by palpitations, perspiration, tremors, and irritability. [9,10].

Causes

Typically, 25–50% of gastric surgery patients present symptoms of Dumping Syndrome (DS) postoperatively; however, only around 10% of these instances are persistent and severe enough to need a diagnosis of Dumping Syndrome [11]. Antrectomy and V&P result in dumping syndrome roughly 10% of the time; in pediatric patients, the predominant cause is Nissen fundoplication for gastroesophageal reflux disease [8]. In current adults, gastric bypass surgery is the predominant cause, with the frequency of (DS) reaching as high as 75% [12].

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].

Post-surgery, the gastric remnant is diminutive and therefore incapable of accommodating substantial intakes, Sleeve gastrectomy is an alternative bariatric surgical procedure that entails shaping the stomach into a tubular form that aligns with the smaller curvature, Dumping Syndrome has been linked to diabetes

mellitus, as well as several idiopathic instances. Diabetes mellitus may be linked to accelerated stomach emptying. This phenomenon is particularly observed in type II diabetes mellitus, when early Wallerian degeneration and first vagal nerve impairment are postulated [8]

pathophysiological

The pathophysiological mechanisms generating these occurrences are inadequately comprehended. The current belief posits that early dumping results from the fast passage of food into the small intestine, leading to osmotic fluid changes from the intravascular zone to the gut [3].

Upper gastrointestinal (GI) surgery can diminish gastric size, eliminate the pyloric barrier function, or influence gastric motility through vagal denervation. these factors may result in quick passage of food to the small intestine. The hyperosmolar inside the small intestine chyme can induce a fluid shift in the vascular cavity to the intestinal lumen, leading to hypotension and, in some cases, syncope. The fluid shift may induce bowel distension and produce abdominal symptoms including fullness, pain, diarrhea, and distension. The manifestation of this fluid shift has been previously validated by an increase in hematocrit levels within the first hour post-meal consumption or while performing dumping provocation tests [14].

A secondary mechanism at play is the augmented secretion of diverse gastro intestinal hormones, leading to

(1) the triggering of gastrointestinal secretion and motility and

(2) the increased release of vasoactive substances that influence on systemic and abdominal flow of blood and vasomotor activity.

In recent decades, much focus has been directed into incretin hormones released from the small intestine, which are known to promote insulin secretion following a hyperglycemic spike, leading to reactive hypoglycemia [15,17].

Epidemiology

The reported prevalence and incidence of dumping syndrome varies significantly depending on (1) the definitions and diagnostic criteria employed, and (2) the type and extent of surgical intervention conducted. Approximately 20% of patients who undergo vagotomy with pyloroplasty exhibit dumping symptoms, while this figure rises to 40% in individuals following Roux-en-Y gastric bypass or sleeve gastrectomy, and may reach 50% in patients post-esophagectomy. [6]. The rapid expansion in the use of bariatric interventions by gastrectomy or RYGB has led to an increasing number of patients with dumping syndrome worldwide [18].

Diagnosis

Dumping syndrome is diagnosed through symptoms that worsen postprandially, provocation tests, laboratory assessments, and underlying conditions of rapid emptying of the stomach. Scales and scoring systems for dumping syndrome have been developed, including Sigstad’s score [19]. Post-Gastrectomy Syndrome Assessment Scale (PGSAS)-45 [20-22]. Dumping Symptom Rating Scale (DSRS) [23].

The diagnosis is validated through a provocative test, followed by incremental therapies that commence with dietary modifications, the incorporation of meal viscosity enhancers or glycosidase inhibitors, and the addition of somatostatin analogues in refractory instances. Several novel therapies aimed at intestinal

motility, peptide hormone influences, and hypoglycemic occurrences are currently being assessed [24].

A clinical evaluation became feasible only post-1970 Sigstad [25]. Diagnostic questionnaires, including the Sigstad Score Scale and the Arts Dumping Questionnaire, were developed to identify clinically significant symptoms.

The Sigstad scoring system is predicated on the manifestation of diverse symptoms indicative of the syndrome. Scores of seven or higher, following glucose consumption, are deemed diagnostic [26].

The oral glucose challenge serves as an effective diagnostic assessment for DS. It is employed to identify symptoms of Down syndrome and validate the diagnosis. Patients undergo a 10-hour overnight fast followed by the consumption of 50 grams of glucose. Blood pressure, hematocrit, pulse, and blood glucose are assessed every 30 minutes for a duration of 3 hours. An elevation in hematocrit by 3% and/or a pulse increase by 10 beats per minute indicates early dumping syndrome, whereas the onset of hypoglycemia indicates late dumping syndrome [27].

Laboratory and radiologic assessments are instrumental in diagnosing patients with late dumping syndrome, encompassing plasma glucose, GLP-1postprandially insulin and gastric emptying scintigraphy evaluations [28].

Treatment Options

Treatment modalities for dumping syndrome encompass dietary alterations, pharmacological therapies, and potentially surgical re-intervention or continuous enteral feeding. Certain treatments are exclusively indicated for late dumping (e.g., acarbose), while others may be advantageous for both early and late dumping (e.g., somatostatin analogues) [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

Acarbose is a medication that stops the body from breaking down complex carbohydrates into simple sugars too quickly. Consequently, postprandial glucose absorption diminishes, leading to a reduced secretion of insulin and gastrointestinal hormones, thereby alleviating symptoms, particularly those associated with late dumping syndrome [31].

Somatostatin Analogues

Somatostatin analogues represent a viable therapeutic option for patients with confirmed dumping syndrome who do not respond to or cannot tolerate initial dietary modifications and acarbose therapy. Somatostatin analogues address multiple aspects of the pathophysiology of DS, such as prolonging gastric emptying, slowing transit through the small intestine, suppressing the release of gastrointestinal hormones, inhibiting insulin secretion, and reducing postprandial vasodilation, thereby alleviating both early and late dumping symptoms [32].

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