Late Dumping Syndrome without Surgery

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Dumping syndrome is a complication of gastric drainage resulting in rapid movement of undigested food material to move from stomach into small intestine. It is group of postprandial symptoms which arise because of rapid gastric emptying. It commonly occur after gastric surgery resulting from gastric bypass or denervation procedure. Dumping syndrome can present as early or late dumping syndrome. Late dumping syndrome comprise only 25% of dumping syndrome. Symptom severity varies from mild to severe. Mostly patients have mild symptoms but 1 - 5% can have severe symptoms.

Late dumping syndrome occurs because of rapid gastric emptying of carbohydrate rich content into proximal small intestine resulting rapid glucose absorption and high serum glucose level leading to increase release of glucagon-like peptide 1 (GLP-1) and reactive exaggerated (above normal) insulin release and resultant late reactive hypoglycemia [1,2]. It occurs 1 - 3hr after meal and present as symptoms of neuroglycopenia (weakness, fatigue, confusion, hunger, dizziness) and autonomic/adrenergic response (sweating, tremulousness, palpitation, irritability). Physical examination may reveal orthostatic tachycardia or hypotension. Severe symptoms may lead to sitophobia and weight loss.

Late dumping syndrome mostly occurs secondary due to esophageal (vagal nerve injury), gastric or bariatric surgery and vagotomy but it can be found in patient with idiopathic rapid gastric emptying (in patient without history of surgery) who is otherwise healthy subject. Disturbed gastric motor function i.e. increased amplitude of gastric contraction is postulated mechanism for idiopathic rapid gastric emptying [3-6].

Diagnosis of late dumping syndrome is based on postprandial symptoms of hypoglycemia related to neuroglycopenia and autonomic disturbances. Laboratory test shows hypoalbuminemia and anemia in patient of long standing severe symptoms. Sigstad score is a battery of symptoms and signs are used to identify dumpers. Score of > 7 suggests presence of dumping syndrome and score less than 4 favors against dumping syndrome. This score is especially useful in monitoring response to treatment [7]. Oral glucose tolerance test: 50 gm glucose with water ingested after 10 hours fasting and symptoms, heart rate and blood glucose measured at 30 minutes interval till 3 hours. A rise in plasma glucose level in first hour of ingestion followed by fall in serum glucose to < 60 mg/dl in 1 - 2 hours present in patient with late dumping syndrome. A rise in heart rate > 10 beat/min within first hour of ingestion is diagnostic of early dumping syndrome [8]. Gastric scintigraphy study will reveals features of rapid gastric emptying in most. Delayed gastric emptying is labelled when gastric retention is > 90% at 1 hour, > 60% at 2 hours, and > 10% at 4 hours. Rapid gastric emptying is said when gastric retention is < 70% at 30 minutes and < 30% retention at 1 hour [9]. Upper GI endoscopy is important to rule out ulcer or obstruction as symptoms may mimic other diseases.

First line of management is dietary modification followed by oral glucosidase inhibitor. If refractory, then octreotide can be used to delay gastric emptying and prevent reactive insulin release. Small frequent meals containing low carbohydrate content should be used. Increasing food material containing proteins and high fibre diet in diet helps to reduce postprandial symptoms. Avoiding fluids contain-
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ing sugar until 30 minutes after meals [10]. Acarbose, an oral glucosidase inhibitor, helps to lower risk of dumping syndrome by reducing absorption of glucose. Octreotide, a subcutaneous injection delays gastric emptying and inhibiting the release of GI hormones. Surgical re-intervention if symptoms are severe and not responding to medical management.

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Bibliography


