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Mini Review

Diabetic Gastroparesis - An Undiagnosed Disorder which is Mismanaged among the Common Medical Practitioner

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Abstract

This article focuses more towards Diabetic Gastroparesis pathogenesis, its diagnostic criteria's and various modalities of treatment offered for it. Above all it highlights the necessity to differentiate Peptic ulcer disease with Diabetic Gastroparesis, since there is surge every year in diabetic population, and escalating misconception which has been quite prevalent among the common medical practitioner in their routine life.

Keywords: *Diabetic Gastroparesis; Peptic Ulcer Disease; Treatment; Common Medical Practitioner*

Introduction

The word Gastroparesis (Gp) explicate, clinical syndrome which constitutes upper abdomen pain, bloating, nausea, vomiting and post prandial fullness. Along with objective evidence of delayed gastric emptying in absence of mechanical obstruction. Though the term Gp has been coined long back, yet it remains an unexplored disorder in our society owing to its identical symptoms of the peptic ulcer disease. The uniqueness of the Gp is that most of the patient affected from this disorder falls in either type 1 or type 2 diabetes mellitus which is evident from most of the epidemiological studies. Therefore, there is a considerable agreement on the naked truth that Gp are still underdiagnosed by the common medical practitioners in their contemporary medical practise.

Pathogenesis of diabetic gastroparesis

There are numerous mechanism which connects diabetes to gastric motor dysfunction, like autonomic neuropathy, acute fluctuations in blood glucose, enteric neuropathy including excitatory and inhibitory nerves, pathological intrinsic cells of cajal (ICC), incretin based drugs used to normalise postprandial blood sugar and psychosomatic factors via autonomic mechanisms.

Mechanism of major elements

Autonomic neuropathy

Among diabetes patients there are less number of cells in motor vagal and sensory sympathetic ganglia and structural changes (e.g. segmental demyelination and axonal degeneration) of vagal nerve fibers both within myenteric and submucosal plexi and outside of the GI tract. The loss of nerve fibers is often multifocal, indicating of ischemic origin. In humans, GI vagal abnormality is indicated by reduction in plasma polypeptide levels during sham feeding. Though a subnormal pancreatic polypeptide response is associated with cardiovascular vagal dysfunction, a threshold increase of 20 pg/mL was 100% specific but only 45% sensitive for cardiac vagal dysfunction. Above all, hyperglycemia is associated with greater fasting levels of plasma pancreatic polypeptide and a reduced response to sham feeding, which affect the interpretation of the test in DM. Based on the premise that vagal neuropathy is generally a length-dependent axonopathy, impaired cardiovascular vagal responses, such as reduced heart rate responses to deep breathing and the Valsalva maneuver, are used as surrogate markers of GI vagal dysfunction.

Intrinsic mechanism

Interstitial Cells of Cajal are responsible for spontaneous pacemaker activity, Loss of those cells activity is the most common enteric abnormality in Diabetic Gp and idiopathic gastroparesis. From the cardia to pylorus stomach shows distinct regional variations in the distribution of subtypes of ICC, whereas the small intestine and colon both seem to retain nearly the similar pattern of subtypes of ICC throughout each organ. In DGp, pathways can damage ICC by various mechanisms, such as insulinopenia, IGF-1 deficiency, and oxidative stress, dominate. Deficiency of ICC survival factors (insulin and IGF-1 promote the production of smooth muscle cell produced stem cell factor, an important ICC survival factor) is detrimental to ICC. Above all, in diabetes, mechanisms that normally counteract increased oxidative stress, such as upregulation of HO-1 (heme xygenase), are impaired, leading to loss of ICC and subsequent delay in gastric emptying. Upregulation of HO-1 by hemin increases ICC and nNOS and normalizes delayed gastric emptying. The protective effects of HO-1 are said to be mediated by one of its products-carbon monoxide (CO).

Common clinical features of diabetic gastroparesis

- Upper abdominal pain 90%
- Nausea 80%
- Vomiting 74%
- Abdominal bloating 65%
- Early satiety 60%
- Delayed gastric emptying 33%
- Rapid gastric emptying 12%
- Depression 05% (via gut - brain axis).

Diagnosis

Diagnosis is often picked up from DM patients who has upper GI symptoms and further established by documenting delayed GE in the absence of gastric outlet obstruction, predominantly by upper GI Endoscopy in order to be more evident. Other factors to be considered are, drugs which causes delayed or rapid GE (i.e. anticholinergic drugs, GLP-1 analogues, metoclopramide, domperidone, erythromycin)

which should be stopped 48 - 72hrs before the test. In regard to diagnostic approach, 99Technetium scintigraphy is currently considered to be the gold standard for Gp. An alternative to above is, C13 breath testing (rate of gastric emptying of the 13C substrate incorporated in a solid meal is reflected by breath excretion of $^{13}CO_2$. Capsule Endoscopy is an another favourable diagnostic tool, but owing to its cost and scarce availability in many countries, remains dormant. Other diagnostic approach are: 1. MRI using gadolinium which accurately measure semi solid gastric emptying and accommodation, which interpreted by notable reduction in velocity of antral propagation waves. 2. Electrogastrography and adjunctive diagnostic test, here pre and post prandial myoelectrical activity of the gastric waves are recorded for a period of 45-60mins and the values are interpreted for disordered gastric function.

Management of diabetic gastroparesis

Diabetic Gastroparesis management is a multi-disciplinary approach owing to the need of multiple factor control starting from life style modification, nutritional support, glycaemic control, obesity reduction, pharmacotherapy for the GI symptoms and psychological support.

Medical management

Among the major factors listed above, blood sugar control should be extremely important as it plays a pivotal role in reducing the GI symptoms. Furthermore, the role of Prokinetics medications has a significant role in controlling the GI symptoms. Metoclopramide, Levosulpiride and Domperidone being common drug administered for the DGp, while the former one being the most commonly used due to its availability as oral, IV and IM types which is started as a low dose later titrated further keeping in the mind about its black box warning effects of extra pyramidal symptoms.

With combination of vitamin B12 and Pregabalin, many studies shows there is better electrophysiological activity which evident that DGp patient will benefit more.

Drugs like Cisapride, Tegaserod, Bethanecol, Low dose Tricyclic Antidepressants and Phenothiazines has shown only minimal efficacy and not used predominantly due to their over-whelming various side effects.

Endoscopic therapies

- Intrapyloric injection of botulinum toxins is still being a vague line of treatment in DGp due to its inadequate efficacy among various trials.
- Other: Venting gastrostomy, Jejunostomy, Transpyloric stenting, Gastric per oral Myotomy.

Surgical therapies

Though the involvement of surgical therapies in DGp patients are quite rare due to the current excellent multidisciplinary approach, the options left for Gp are Gastrectomy and Pyloroplasty [1-6].

Conclusion

To summarise, though the Diabetic Gastroparesis condition mimics like a peptic ulcer disease, it needs special attention as the management for this condition typically relies on multidisciplinary approach to be specific more on diabetic control, as its adverse effects carries very high morbidity which left untreated can be fatal. Henceforth, this article emphasis on, to differentiate between a common peptic ulcer disease and diabetic gastroparesis by a common medical practitioner in his/her routine clinical practise.

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