We present Mr M.A. a 27 year old Nigerian of the Yoruba tribe who has been having recurrent dyspeptic symptoms for 5 years but not previously endoscopically evaluated for peptic ulcer disease who now presented with six days history of abdominal pain which was initially epigastric but later became generalized associated with four episodes of vomiting and a day history of abdominal distension and fever. He was acutely ill-looking and dehydrated. The Abdomen was moderately distended, does not move with respiration, generalized tenderness with guarding and rebound tenderness, intra-abdominal organs were difficult to palpate due to the guarding. Bowel sound was absent. Examination of the other systems was not remarkable. A provisional diagnosis of Generalized peritonitis likely secondary to perforated peptic ulcer was made. He had an emergency exploratory laparotomy under general anaesthesia. He was commenced on intravenous fluids and parenteral proton pump inhibitor (PPI) and antibiotics. Intra-op findings include 2 liters of bilious peritoneal fluid, Multiple fibrous adhesions, a 2.5 cm x 2 cm gastric perforation at the anterior wall of the antrum and a grossly normal bowel. The surgical operation performed was a Graham Omental patch closure of the gastric defect with Bilateral Truncal Vagotomy and Peritoneal lavage. Post-operative state was satisfactory and he was later discharged. He presented at the gastroenterology out-patient clinic eight months later with complaints of early satiety, feeling of indigestion and episodes of vomiting. Vomitus is usually offensive / foul smelling and contains undigested or partially digested stale food substances. Abdominal examination revealed a positive succussion splash. Other clinical examination findings were not remarkable. A Provisional Diagnosis of suspected Gastric outlet obstruction probably secondary to a chronic duodenal ulcer was made. Endoscopy findings revealed copious fluid and food debris in the stomach cavity which had an offensive smell with remnants of partially digested stale food substances seen. There was poor peristaltic activity and the stomach was poorly distensible. There were multiple areas of mucosa erosions seen in the gastric cardia and corpus. No corporal or antral lesion seen. The pyloric ring was normal and the duodenum was normal. Diagnosis- (1) Endoscopic features of Gastroparesis likely secondary to previous Bilateral Truncal Vagotomy. No evidence of mechanical obstruction of the Gastric outlet. (2) Gastric Cardia and Corporal mucosa lesions likely secondary to Stasis Gastritis. Histology of biopsy specimens showed acute on chronic non-specific gastritis and acute duodenitis. He was commenced on empirical first line Helicobacter pylori eradication triple therapy (PPI, Amoxicillin and Clarithromycin) and a prokinetic drug (Domperidone). He had made
significant clinical improvement with resolution of the symptoms by the time he was seen in the clinic three weeks later. He was tolerating oral food intake adequately and he had no new complaints. He was counselled on dietary modification and placed on long term prokinetic agent (Domperidone) and long-term PPI therapy with regular clinic follow-up. The initial management of post-vagotomy gastroparesis should be conservative as many symptoms resolve with time, this occurs possibly because the enteric nervous system is able to adapt to the loss of vagal input. Modification of dietary habits, such as regular intake of small meals, and use of prokinetic drugs bring about symptomatic relief in most patients.

**Keywords:** Post-vagotomy, Gastroparesis, Prokinetic, Endoscopy

**Introduction**

Gastroparesis is defined as a syndrome of objectively delayed gastric emptying in the absence of mechanical obstruction and presence of cardinal symptoms including early satiety, postprandial fullness, nausea, vomiting, bloating, and upper abdominal pain.\(^1,2\) The majority of cases is idiopathic consisting of about 35-40\% \(^3,4\) and long-standing diabetes mellitus is responsible for about 25-30\% \(^3,5,6\) of cases. Postsurgical gastroparesis (PSG), often with vagotomy or vagus nerve injury, represents the third most common etiology of gastroparesis consisting of about 15-20\%.\(^7,8\) The treatment of peptic ulcers is largely medical but surgical therapy may be indicated when ulcers are refractory or become complicated.\(^8\) There are different surgical options in such instances including vagotomy. Types of vagotomy procedures include: Truncal vagotomy and drainage, Selective vagotomy, Highly selective vagotomy and Posterior Truncal vagotomy with anterior seromyotomy.\(^8\) Truncal Vagotomy removes the cephalic stimulus to the parietal/oxyntic cells and the motor stimulus to the stomach thereby causing significant reduction in acid secretion and impaired gastric motility. Post-vagotomy gastroparesis consequently develops.\(^2,8\) Such individuals will present with nausea, vomiting, postprandial bloating, early satiety and abdominal discomfort.\(^2,9\) We report a case of symptomatic Post-vagotomy gastroparesis who was managed conservatively with significant resolution of symptoms.

**Case Presentation**

We present Mr M.A. a 27year old Nigerian of the Yoruba tribe who presented with six days history of abdominal pain associated with four episodes of vomiting and a day history of abdominal distension and fever. He was apparently well until days prior to presentation when he developed a sudden onset abdominal pain. The pain was located at the epigastric region. It was described as a sharp pain, aggravated by movement and does not radiate to any part of the body, no similar episode in the past. The pain later became generalized about three days before presentation. The patient subsequently had about 4 episodes of vomiting which was described as post-prandial in nature, non-bilious, non-bloody. There was a progressive generalized abdominal distension which he noticed a day prior to presentation, also has associated anorexia. Last bowel movement was a day prior to presentation. No hematochezia, no
passage of dark tarry stool. Patient also had fever a day prior to presentation which was low grade intermittent and was relieved by tepid sponging.

No headache, no sore throat or ear discharge, no dysuria or frequency, no diarrhoea, no cough or chest pain. No trauma to the abdomen, no preceding history of NSAID use. He has been having recurrent dyspeptic symptoms for 5 years but not previously endoscopically evaluated for peptic ulcer disease. He has not been admitted at any hospital before. No history of blood transfusion in the past. He is not a known patient with systemic hypertension or diabetic mellitus. He does not drink alcohol nor smoke cigarette.

Clinical examination revealed a young man, conscious, acutely ill-looking, not pale, anicteric, afebrile (Temp – 36.3°C), dehydrated, no significant peripheral lymph node enlargement, no pedal oedema and no asterixis. The Abdomen was moderately distended, does not move with respiration, generalized tenderness with guarding and rebound tenderness, intra-abdominal organs were difficult to palpate due to the guarding. Bowel sound was absent. Examination of the other systems was not remarkable. A provisional diagnosis of Generalized peritonitis likely secondary to perforated peptic ulcer was made. A Nasogastric tube was passed which was draining copious efflux. He was reviewed by the General surgery unit and was immediately worked-up for an emergency exploratory laparotomy under general anaesthesia. He was commenced on intravenous fluids and parenteral proton pump inhibitor(PPI) and antibiotics. Intra-op findings include 2 liters of bilious peritoneal fluid, Multiple fibrous adhesions, a 2.5cm x 2cm gastric perforation at the anterior wall of the antrum and a grossly normal bowel. The surgical operation performed was a Graham Omental patch closure of the gastric defect with Bilateral Truncal Vagotomy and Peritoneal lavage. Specimens of the vagus nerve and gastric tissue were sent for histology. There were no complications. The immediate post-operative state was satisfactory.

Patient sustained a remarkable clinical improvement. He was commenced on sips of water and then graded oral feeds on fifth day Post-Op following the return of bowel sounds. The operation site apposed very well, sutures were removed and he was subsequently discharged nine days Post-Op on oral antibiotics and PPI.

**Histology**

Macroscopy: Specimen consist of 2 tiny pieces of firm greyish tissue. One was tagged and measure 1.0cm x 0.5cm while the untagged one (vagus nerve) measure 0.4cm x 0.4cm.

Microscopy:- Histologic section of gastric mucosa show wide areas of ulceration, haemorrhage and adjacent to this are granulation tissue with foci of fibrinous exudates. Other sections show nerve bundles within a loose, vascularized fibro-collagenous stroma. Diagnosis – Gastric Biopsy – Edge of an ulcer.

He was subsequently seen at the general surgery out-patient clinic one week after he was discharged with no new complaints. The operation site had completely healed. He was counselled and discharged from the General surgery clinic.

Patient presented at the gastroenterology out-patient clinic eight months later with complaints of early satiety, feeling of indigestion and episodes of vomiting. Vomitus is usually offensive / foul smelling and contains undigested or partially digested stale food substances. Abdominal examination revealed a positive succussion splash. Other clinical examination findings were not remarkable. A Provisional Diagnosis of suspected Gastric outlet obstruction probably secondary to a chronic duodenal ulcer was made. He was scheduled for an upper GI endoscopy.

Endoscopy findings revealed copious fluid and food debris in the stomach cavity which had an offensive smell with remnants of partially digested stale food substances seen. There was poor peristaltic activity and the stomach was poorly distensible. There were multiple areas of
Figure 1: An Endoscopic image of the Gastric corpus showing copious food debris and fluid collection within it.

Figure 2: An Endoscopic image of the Gastric fundus showing food debris and fluid collection within it.
Figure 3: An Endoscopic image of the Gastric antrum showing food debris and some fluid collection within it. The pylorus appear oedematous but there is no mechanical obstruction in the gastric outlet.

Figure 4: An Endoscopic image of the Duodenum showing normal appearance.
mucosa erosions seen in the gastric cardia and corpus. No corporal or antral mass lesion seen. The pyloric ring was normal, not deformed but slightly oedematous. No strictures or luminal narrowing or mass seen involving the pylorus. The Duodenal Bulb and Post bulbar segments were grossly intact and normal. No duodenal ulcer, stricture, mass or luminal narrowing seen. Diagnosis- (1) Endoscopic features of Gastroparesis likely secondary to previous Bilateral Truncal Vagotomy. No evidence of mechanical obstruction of the Gastric outlet. (2) Gastric Cardia and Corporal mucosa lesions likely secondary to Stasis Gastritis.

Histology

Macroscopy

(1) Gastric cardia: Received 3 tiny pieces of firm greyish white tissue each measuring 0.2cm x 0.2cm.

(2) Gastric corpus: Received 4 tiny pieces of firm greyish white tissue each measuring 0.2cm x 0.2cm.

(3) Gastric antrum: Received 3 tiny pieces of firm greyish white tissue each measuring 0.2cm x 0.2cm.

(4) Duodenum: Received 3 tiny pieces of firm greyish white tissue each measuring 0.2cm x 0.2cm.

Microscopy

Gastric Cardia and Corpus:

Histology sections show intact mucosal epithelial lining. There is infiltration of the submucosa and mucosa by mixed inflammatory cells consisting of neutrophils, lymphocytes, plasma cells and macrophages. No metaplasia, atrophy or dysplasia and no Helicobacter pylori-like organism seen. Conclusion – Acute on chronic non-specific gastritis.

Gastric Antrum:

Histologic sections show focal mucosal epithelial ulceration and infiltration of the submucosa by neutrophils, plasma cells, lymphocytes and macrophages. There is submucosa fibrosis and hypertrophy of the muscularis mucosae. No metaplasia no atrophy or dysplasia and no Helicobacter pylori-like organism seen. Conclusion: Acute on chronic non-specific gastritis.

Duodenum:

Histologic sections show infiltration of the intact mucosal epithelium by neutrophils. No dysplasia or atrophy seen.

Conclusion: Acute duodenitis.

He was commenced on empirical first line Helicobacter pylori eradication triple therapy (PPI, Amoxycillin and Clarithromycin) and a prokinetic drug (Domperidone). He had made significant clinical improvement with resolution of the symptoms by the time he was seen in the clinic three weeks later. He was tolerating oral food intake adequately. Succussion splash was also absent and he had no new complaints. He was counselled on modification of dietary habits, such as regular intake of small meals. He was also placed on long term prokinetic agent (Domperidone) and long-term PPI therapy with regular clinic follow-up.

Conclusion

Post-vagotomy gastroparesis has been documented in the literature and should be anticipated in patients who have undergone vagotomy. The symptoms of gastroparesis include nausea, vomiting, bloating, early satiety and discomfort. Weight loss, dehydration, electrolyte disturbances and malnutrition may develop in severe cases. The initial management of post-vagotomy gastroparesis should be conservative as many symptoms resolve with time, this occurs possibly because the enteric nervous system is able to adapt to the loss of vagal input. Conservative measures include modification of dietary habits with regular intake of small meals; psychological support; and drug therapy. Prokinetic agents (erythromycin, domperidone, metoclopramide) and antiemetics (phenothiazines, serotonin antagonists, butyrophenones) are the most widely used medications. Dietary measures
and prokinetic drugs bring symptomatic relief in most patients.⁹ Some patients with severe nausea and vomiting will require antiemetic medications. Injection of botulinum toxin into the pyloric sphincter and Gastric electrical stimulation are novel therapies that results in faster gastric emptying and symptom alleviation.²,⁸ Few patients will fail medical therapy and will continue to have debilitating symptoms of gastroparesis; management of such patients will require a multidisciplinary team approach. Such patients may benefit from a venting gastrostomy or a jejunostomy placed surgically, endoscopically, or fluoroscopically.⁹

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Conflict of interest disclosure
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