Journal of Surgery

Ali MM, et al. J Surg 9: 11093 www.doi.org/10.29011/2575-9760.011093 www.gavinpublishers.com







Gastroparesis: A Unique Case Leading to Acute Limb Ischemia with Literature Review

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Citation: Mahmood Ali M, Al-Doseri M, Elbarbary SA, Zidan TAM, Haider Ali A (2024) Gastroparesis: A Unique Case Leading to Acute Limb Ischemia with Literature Review. J Surg 9: 11093 DOI: 10.29011/2575-9760.11093

Received Date: 18 July 2024; Accepted Date: 23 July 2024; Published Date: 25 July 2024

Abstract

Gastrointestinal symptoms are a common presentation in the Emergency Department. Ranging from abdominal pain to nausea and vomiting, most patients do not require a full work-up for a diagnosis to be made. Gastroparesis is the delay in gastric emptying and motility leading to the retention of gastric fluids. It may appear as abdominal pain, distention, nausea, and vomiting. Multiple etiologies may present, including idiopathic, medications, and neuromuscular disease. Complications from gastric distention such as acute limb ischemia may occur due to the pressure effect on the iliac artery. Most management focuses on symptomatic management with surgical treatment being for extreme cases. This case follows a 54-year-old female who presented with abdominal pain and distention. The patient developed acute limb ischemia which subsided after decompression of the stomach. The patient had gastric by-pass surgery to subside the symptoms of gastroparesis.

Keywords: Acute Limb Ischemia; Drug-induced; Emergency Medicine; Gastrointestinal; Gastroparesis

Introduction

Gastrointestinal complaints are common in the Emergency Department (ED). It was noted that more than 11% of presentations to the ED consist of gastrointestinal complaints, with over 16% of such cases being admitted through the ED between 2016 and 2019 [1]. Furthermore, gastrointestinal complaints come from a spectrum of illnesses that may include medical or surgical causes. Patients may present with abdominal pain, nausea, vomiting, constipation, diarrhea, blood-stained stool, and more, which

opens a differential of causes for the ED physician. This requires proper history taking, examination, and appropriate investigation to understand the underlying pathology and the treatment course. Of such pathologies is gastroparesis, a disorder characterized by the delay in gastric emptying leading to symptoms such as vomiting, bloating, and abdominal distention [2]. Although the underlying mechanism is poorly understood, it is considered a part of a larger spectrum of disorders noted as neuromuscular disorders of the gastrointestinal tract [2]. This case report will discuss a presentation of a 54-year-old female with complaints of abdominal distention and vomiting due to gastroparesis with a complication of acute limb ischemia due to the extreme distention of the stomach.

Volume 09; Issue 10

J Surg, an open access journal ISSN: 2575-9760

Case

A 54-year-old Middle Eastern Female, with a history of Hypertension and Schizophrenia, came with complaints of severe abdominal pain, diffuse, associated with multiple episodes of non-bilious, non-bloody vomiting, for one week. The patient was not tolerating any oral feeds, including solids and liquids. The patient was conscious, alert, and oriented, with no features of psychosis. Vitals recorded at the time showed a blood pressure of 84\45, Heart Rate of 130, Respiratory Rate of 25, Oxygen Saturation of 100% on Room Air, and a Temperature of 37.5 degrees. The patient was shifted into Level One Canadian Triage System Bay. On examination, the patient has severe abdominal distention, associated with diffuse tenderness. According to the relatives, the distention was progressive, and the patient had not passed stool or gas in two days. A venous blood gas showed a pH of 7.00, associated with a lactic acid of 8 and a Bicarbonate of 14. The patient is currently on a regimen of Atypical Anti Psychotics (clozapine, 300mg, once daily), Selective Serotonin Reuptake Inhibitors (escitalopram, 10mg, once daily), and Angiotensin Receptor Blockers (Valsartan, 80mg, once daily). Upon further assessment, a Nasogastric Tube (NGT) was inserted after Computed Tomography (CT) of the Abdomen was done. During insertion, the patient develops right lower limb pain. On examination, the right lower limb was pale and moldy, and the dorsalis pedis artery pulse was not felt. A Doppler ultrasound was done showing no flow and vascular surgery was called straight away. The NGT was inserted and drained over five liters initially. As aspiration was done, the right lower limb started to become red and the pulse returned. Upon repeat Doppler, all pulses were heard, and vasculature was intact. A computed tomography (Figure 1) and laboratory investigations (Table 1) were ordered urgently.

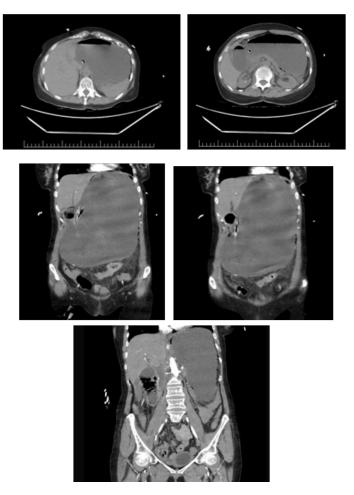


Figure 1: CT Abdomen Findings at the time of presentation.

Investigations	Findings
White Blood Cells	14.94 mg\dL.
Hemoglobin	13.70 mg\dL.
Amylase	303 U\L.
Lactic Acid	11.30 mmol\L.
Urea	283 micromol\L.
Creatinine	283 micromol\L.
Bicarbonate	15 mmol\L.
Potassium	3.6 mmol\L.
Sodium	142 mmol\L.
Urine Analysis	Unremarkable.

Table 1: Laboratory Investigations at Time of Presentation.

CT showed Significant Dilation of the stomach associated with possible stomach wall ischemia, and possible Superior Mesenteric Artery Syndrome. The patient had an increase in white blood cells associated with acute kidney injury. The patient was admitted by General Surgery. Upon admission, the gastrograffin upper gastrointestinal series was done and showed that the fluid was not passing through. Therefore, an endoscopy was ordered to exclude obstruction by malignancy. On endoscopy, no tumor obstruction was seen with normal esophagus and extremely dilated stomach. The duodenum was noted as normal. Therefore, a diagnosis of gastroparesis was established, and bypass surgery was done for the patient to reduce the risk of aspiration. The patient was well postoperatively and discharged with follow-ups.

Discussion

Normal gastric function is a complex physiological process [3]. In a processed and synchronized movement, the motility of the stomach aids in the proper passage of food within the gastrointestinal tract. In addition to the proper passage, it has certain mechanisms to aid in the decreased risk of reflux to the esophagus [3]. Furthermore, it is usually stimulated by the presence of food within its lumen, therefore lying dormant until such stimulation is present [3]. Thus, any disturbance to the rhythm will lead to the delayed emptying of the stomach content, its accumulation within the lumen, and the distention of the stomach within the abdominal cavity [3]. As such, this disturbance is the underlying pathophysiology of gastroparesis, where delayed emptying occurs without the presence of machinal obstruction, but rather a disturbance in the neuromuscular cycle within the tract [3,4]. It is noted that such disturbances can be found in 20% of adults in the United States with chronic gastrointestinal symptoms presenting to a gastroenterologist [4].

Multiple etiologies have been identified to play a role and are noted as the underlying cause of gastroparesis including [3-6]:

- Idiopathic: The most common cause of gastroparesis, where an underlying pathology is not found as seen in the case presented above. It is seen in over half of the patients presenting with gastroparesis.
- Diabetes Mellitus: It is a frequently recognized systemic cause due to its effect on musculature and neurons leading to such symptoms associated with autonomic dysfunction. It usually occurs in patients who have been diagnosed for at least five or more years. As previously stated, it is theorized that the underlying pathology is related to dysregulation of the Autonomic Nervous System (ANS).
- Viral: some viral etiologies have been noted to lead to gastric stasis such as rotavirus, Norwalk virus, and more recently, varicella zoster virus and its impact on the ANS leading to severe dysautonomia.
- Medications: medication may impact and delay gastric emptying. Examples can be noted in Table 2.
- Post-surgical: it has been noted that most cases are related to accidental vagus nerve injury during surgery [7].
- Neurological Disease: as previously stated, certain system illnesses or infections can lead to autonomic dysregulation. Therefore, neurological illness can lead to gastric dysmotility and eventually, gastroparesis. Multiple pathologies exist which include [8,9]:
- External neural control loss such as in multiple sclerosis, brainstem stroke, or tumors affects the vagus nerve or the lower thoracic spinal sympathetic outflow.
- The myenteric plexus degeneration may be seen in diffuse degenerative neurological disease.
- Medications used in the treatment of such illnesses include anticholinergic or dopaminergic medications.
- Autonomic dysfunction (has been discussed within diabetes mellitus).
- Autoimmune: regarded as autoimmune gastrointestinal dysmotility [10-12], which has been either seen idiopathetically or as a paraneoplastic syndrome, mainly small cell lung carcinoma [12]. In an interesting aspect, it is not only seen as delayed gastric emptying, but it may affect the whole tract. It has been associated with slow intestinal transient, and slow colonic transient, and may affect pelvic floor musculature [12].

As mentioned above, most cases have been noted as idiopathic. In the case presented, mechanical obstruction has been excluded using multiple diagnostic stools, and due to the patient being on

antidepressants doses were reduced to decrease side effects. After such an approach, no improvement was noted leading to a more idiopathic cause of her symptoms. About antipsychotics, most authors related decreased motility due to constipation induced by the medication more so than gastroparesis [13]. Therefore, antipsychotics used in the treatment of the patient's schizophrenia are unlikely to be the underlying cause in comparison to the antidepressants used [13].

Pharmacological Class	Example
Narcotics	Oxycodone and Tapentadol
Alpha 2 adrenergic agonist	Clonidine
Tricyclic Anti-Depressants	Amitriptyline
Glucagon like peptide 1 Agonists	Exenatide
Mood Stabilizers	Lithium
Ethanol	Alcohol
Anti Histamine	Diphenhydramine

Table 2: Medications leading to Gastroparesis.

In terms of clinical features, gastroparesis has a large clinical spectrum. Common symptoms include vomiting, abdominal pain, postprandial fullness, bloating, nausea, and weight loss [14]. This can be correlated with the physical examination, where in severe cases, abdominal distention and tenderness will be noted in the patient [14], as seen above. In the evaluation of such cases, the exclusion of mechanical obstruction is important. The use of Computed Tomography and endoscopy is useful in the exclusion of obstruction yet will not aid the in overall diagnosis of gastroparesis [15]. In the case presented above, the initial diagnosis was "Superior Mesenteric Artery Syndrome", where the duodenum may intersect below the artery leading to a compression effect with the aorta [16]. Yet, in this case, the patient has mainly gastric dilation as seen in Figure One. The obstruction was excluded in endoscopy where the dilation of the stomach was noted only, with a completely normal duodenal appearance, leading to a diagnosis of gastroparesis more likely. Most articles agree that assessment of gastric motility is required using Scintigraphy, which is the most cost-effective process in the diagnosis [17]. The use of solids and liquids is important to note the severity of the delay and its impact on the overall patient outcome as the risk of aspiration is high [17]. Unfortunately, it is currently not available in the Kingdom of Bahrain.

In an interesting aspect, the occurrence of Acute Limb Ischemia (ALI) was an unexpected turn. One case has been reported in an anorexia nervosa patient who had quite a similar dilation [16,17-19]. In terms of the pathophysiology, the most likely etiology is the compression of the iliac artery due to the dilation, therefore with the insertion and relief of the dilation, circulation was

returned nonsurgical. Such complications are extremely rare and not commonly seen leading to this case being an interesting presentation of such extreme dilation. It is important to note that such dilation may also impact the aorta, renal arteries, and the inferior vena cava leading to more severe complications due to the decreased prefusion to important organs and the eventual necrosis of such organs [19]. In some cases, complications of reperfusion can occur as well as surgical intervention due to decreased perfusion leading to acute necrosis of organs [19].

In terms of management, patients should be kept nil per oral (NPO), started on aggressive fluid therapy due to hypovolemia and malnutrition, and an insertion of a Nasogastric Tube (NGT) to decompress the stomach of accumulating fluid. Some literature indicates the use of prokinetics to improve gastric emptying which includes metoclopramide, domperidone, erythromycin, and cisapride. Although efficacy may vary, it is seen as a promising treatment in some cases. Finally, surgical intervention may be required depending on the severity, where some patients may require a gastrostomy insertion, gastric sleeve, or a gastric bypass due to the decreased motility of the stomach.

Conclusion

Gastroparesis is a difficult pathology in terms of diagnosis and overall management. Most patients will have long-term complications from the illness unless the cause is reversible. Most management focuses on increasing gastric motility with symptomatic management. In extreme cases, surgical management may be required.

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