



Review Article

Atypical Cases of Gastric Perforation Due to Ischemia: Case Series And Review of Literature

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Abstract

Introduction and aim: Gastric ischemia is infrequently reported in literature and underrecognized clinically and histopathologically. It has been reported in isolated case reports or small case series. Most cases are associated with vascular etiology and have high mortality rate. In addition to present a case series, we aim to define and review pathogenesis, diagnosis and outcomes of gastric ischemia from our experience along with a literature review.

Methods: A retrospective review of patients diagnosed with isolated gastric ischemia at our institution from Jan 2016, to August 2020 was performed. Demographic, clinical, endoscopic, radiologic, and outcome variables were abstracted for analysis.

Results: Three patients were included in the study with age range (24-62) years. The etiologies for gastric ischemia included: vasculopathies and local vascular causes, systemic hypoperfusion and severe sepsis. All three patients were managed surgically with explorative laparotomy with total or partial gastrectomy with Roux-En-Y reconstruction. Mortality rate was 67% (2 out of three) which was a sequelae of gastric ischemia as it was associated with generalized vascular complications like septic shock and hypoperfusion status and complications secondary to pancreatitis like ARDS and infection.

Conclusion: Although uncommon, gastric ischemia is associated with significant morbidity and mortality. Early diagnosis with endoscopy and CT scan is essential to make an early diagnosis. The management is dictated by the severity of the presentation and the associated co-morbidities. Identifying and treating the underlying etiology. Gastric perforation as a result of gastric ischemia carries high morbidity and mortality.

Keywords: CT Scan; Gastric Ischemia; Gastric Perforation; Vascular Compromise

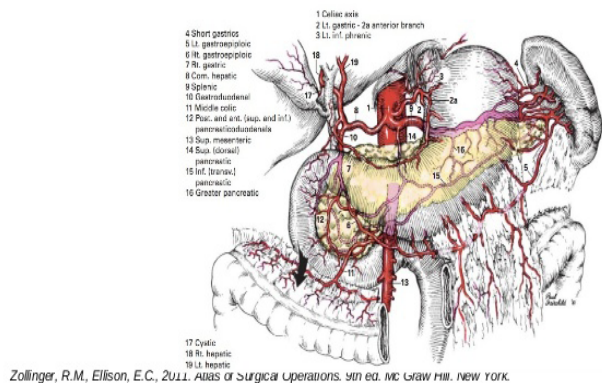
Introduction

Gastric perforation due to ischemia is infrequent due to rich blood supply of the stomach. The stomach is vascularized from the celiac trunk and its branches, which include the left gastric, splenic, and common hepatic arteries (Supplementary Figure 1).

Ischemic gastritis was first described by Cohen in 1951 when he reported 3 cases of complete and 1 case of partial gastric ischemia in an autopsy series of 24,000 patients. Since then, it has been described in case reports and small case series [1]. The clinical presentation is variable depending on the acuity and the extent of the ischemia. The symptoms vary from vague abdominal pain and nausea or vomiting to GI bleeding and gastric perforation or sepsis. Only a handful of cases of gastric ischemia have been reported in the literature in patients with predisposing factors,

such as atherosclerosis, vasculitis, paraoesophageal hernia, gastric volvulus, gastric dilation, disseminated intravascular coagulation, shock, and postoperatively [2].

VASCULARISATION



initial signs and symptoms of AMI [3]. Haruna et al. presented a case report about acute mesenteric ischemia and duodenal ulcer perforation as a double pathology. In their article; they hypothesized that their patient developed the dual pathology due to stress related duodenal ulcer post ischaemic bowel infarction and eventually this ulcer perforated. The cause of the ischaemia is likely due to arterial thrombosis with a background of severe atherosclerotic disease caused by the patient's long history of hypertension and smoking [4].

Gastric ischemia can be managed conservatively, however; if it is complicated by perforation or bleeding then surgery is usually required [1]. For example; one of our patients in this case series was an adult male that presented with ischemic gastric perforation associated also with small bowel ischemia, diagnosis was preoperative and confirmed by the intraoperative findings and post-operative histopathology, resection of ischemic segment of small bowel and distal gastrectomy with Roux-en-Y reconstruction were performed, and the post-operative course was uneventful. We report this case in line with the updated Consensus-Based Surgical Case Report (SCARE) guidelines [2]. We aim in this article to define and review the pathogenesis, diagnosis and management of gastric ischemia.

Methods

This was a retrospective case series study, where we looked at all gastrectomies operated in Hamad General hospital between 2016-2020. We included adult patients (≥ 18 years) diagnosed with gastric ischemia based on the presence of at least 2 features: (1) clinical (e.g, abdominal pain, GI bleeding) and (2) radiographic (e.g, gastric pneumatosis, portal venous gas). We excluded patients with discrete gastric ulcers secondary to causes such as *Helicobacter pylori* and non-steroidal anti-inflammatory drugs, patients with mesenteric ischemia without gastric involvement, and those with symptomatic paraesophageal hernias with no endoscopic signs of gastric ischemia.

We abstracted various demographic and clinical variables, including age, sex, Body Mass Index (BMI), clinical features, and putative risk factors (e.g, thromboembolic disease, vasculitis, septic or hemorrhagic shock). We noted radiological findings (e.g, portal venous air, gastric pneumatosis, intra-abdominal collection), as well as histopathological findings. Specific management details; including medications [e.g, proton pump inhibitor (PPI), antibiotics], nasogastric (NG) tube decompression, nutritional support, radiologic and/or surgical intervention, were recorded, as well as morbidity and mortality data at last follow-up. Descriptive statistics were used for the study.

We performed a literature review on PubMed looking for all cases series and case reports published in literature and it is summarized in Table 1. Two researches independently reviewed

Supplementary Figure 1: A: anatomy and vascular blood supply of the stomach. B: diagram for etiology of mesenteric ischemia.

Acute Mesenteric Ischemia (AMI) comprises a group of pathophysiologic processes that have a common end point-bowel necrosis. The survival rate has not improved substantially during the past 70 years, and the major reason is the continued difficulty in recognizing the condition before bowel infarction occurs [3]. Clinical presentation is nonspecific in most cases and can be characterized by an initial discrepancy between severe abdominal pain and minimal clinical findings. In general, patients with AMI have an acute onset of symptoms and a rapid deterioration in their clinical condition. Complications such as ileus, peritonitis, pancreatitis, and gastrointestinal bleeding may also mask the

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published articles in pubmed using the following keywords: gastric ischemia, surgical management of gastric ischemia, gastric perforation, sepsis, atherosclerosis and gastric ischemia. Exclusion criteria were articles not published in English, articles published prior to 1960, and case reports.

Study	N of pt	Etiology	Treatment	Outcome	Follow up
Sharma et al, 2017 [5]	17	1-Local vascular causes (n=8) 2-systemic hypoperfusion (n = 4) 3-mechanical obstruction (n = 5)	1-Gastric resection (n=6) 2-Radiological stent 3-placement (n-3) 4-Conservative (n=8)	Mortality rate: 35% (n-6)	21-116 months
Elwir et al, 2016 [6]	12	1-Celiac access stenosis (n=2) 2-Embolization (n=2) 3-vasculitis(n=2) 4-Unknown (n=6)	1-Steroids (for vasculitis) 2-IR revascularization (for celiac access stenosis) 3- gastrectomy (n=6) 4- medical treatment (n=4)	Mortality rate: 33% at 30 days and 41% at 1 year	1 year

Herman et al, 2011 [7]	14 (7 gastric and 7 duodenal)	1-stress ulcer(n=4) 2-TACE (n=2) 3- vasculitis (n=1) 4- hypotension (n=1) 5- atherosclerosis (n=1) 6-unknown (n=3) 7- mesenteric ischemia(n=1) 8- portal hypertension (n=1)	1-medical therapy (n=12) 2-surgery (n=2), one with SMA thrombectomy and one with small bowel resection	Mortality rate (29%) (n=4)	100 days
Chambon et al, 2012 [8]	5	Celiac artery stenosis and atherosclerosis	1-aortohepatic bypass (N.=1), 2-renohepatic bypass (N.=1), 3-retrograde iliosuperior mesenteric bypass (N.=2) 4-celiac artery angioplasty (N.=1) and celiac 50superior mesenteric artery angioplasty (N.=1)	Mortality rate 60% (n=3)	1-2 years
Casey et al, 1993 [9]	7	Celiac and SMA occlusion	Surgery (gastrectomy n=6)	Mortality rate 28% (n=2)	12-18 months

Babu et al, 1993 [10]	6	5 patients: celiac, SMA, and IMA high-grade stenosis/occlusion; 1 patient: celiomesenteric trunk stenosis	Temporary revascularization, followed by surgical bypass graft: 5/6 (bowel resection in 1 case); 1 patient unfit for treatment	Mortality rate 83% (n=5)	6 months
Liberski et al, 1990 [11]	2	Celiac, SMA, and IMA occlusion	Surgery (gastrectomy)	All alive	N/A
Cherry et al, 1986 [12]	8	1, celiac and IMA occlusion, SMA stenosis; 2, celiac, SMA, and IMA occlusion; 3, celiac, SMA, and IMA stenosis; 4, celiac, SMA, and IMA stenosis	1, percutaneous balloon dilation; 2, surgery; 3, percutaneous treatment failure, successful surgery; 4, percutaneous treatment failure, successful surgery	All alive	N/A

Table 1: literature review of case series looking at gastric ischemia.

Results

Three patients matched our criteria and were included in the study as summarized in Table 2. All patients were male with age range from 24-62 (median of 35 and average of 39.5). Two patients had preexisting risk factors including DM and cigarettes smoking. Patient A had multiple comorbidities including; dyslipidemia, hypertension and peripheral arterial disease. The most common presentation was melena and hemodynamic instability in patients B and C while patient A presented with abdominal pain and fever that rapidly progressed to septic shock. Patients A and C were stabilized and underwent CT abdomen as shown in Figure 1. Some of the common features on CT abdomen included; thickening of the gastric wall, fat stranding and air loculi around the stomach. For patient A; upon revision of the pre-operative CT scan; there was extensive atherosclerosis in the aorta with an atheroma which was also seen in CT angiogram with no presence of any vascular

anomalies. Figure 2 shows the CT angiogram with the atheroma and extensive atherosclerosis in the abdominal aorta and its branches. Patients B and C had upper endoscopy pre-operatively; as shown in Figure 3. For patient C; OGD showed two fundal ulcers and unhealthy mucosa of the stomach. On the other hand; patient B OGD did not provide valuable information as he was critically ill post the massive myocardial infarction; it mainly showed the stomach to be filled with blood with diffuse necrotic walls and sloughing of the mucosa. In addition to the involvement of the stomach, there was a segment of ischemic small bowel in patient A that showed extensive ischemia on histopathology. As in Haruna et al case report; they reported a single case with duodenal ulcer perforation with segment of ischemic small bowel [4]. Figure 4 shows the distal stomach after resection with the perforation and the friable edges in the posterior wall of the stomach. For all three patients the stomach pathology proven presence of hemorrhagic ischemia.

Pt	Age/ Sex	BMI	Comor- bidities/risk factors	Pre- sent- ing Symp- tom	Radiology: CT findings, Xray	OGD	Etiology	Acute/ Chronic	Treatment	Site of the perfo- ration	Outcome
A	62/ M	42	DM, HTN, peripheral vascu- lar disease, dyslipidemia, cigarettes smoking	Ab- domi- nal pain, fever	CT: ill-defi- nition of the gastric wall with collection within the lesser sac and pneu- moperitoneum. atheromatous calcification noted in the aorta and its major branches.	None	Vascu- lopathy and athero- sclerosis in celiac artery	Acute on chronic	exploration laparotomy + distal gastrec- tomy with Roux-en-Y gastrojeju- nostomy + small bowel resection	4 cm perfo- ration in the posteri- or wall gastric body	Death after 30 days (sudden cardiac arrest due to massive MI)
B	24/M	26	None	GI Bleed	X-ray abdomen: pneumoperito- neum	stomach filled with blood, could not assess properly	Post cardiac arrest (myocar- ditis) on ECMO and hypoten- sion	Acute	Explorative laparotomy with total gastrectomy and Roux en Y esoph- agojejunos- tomy	Perfo- ration in the antrum and ischemic fundus	Alive 14 months after surgery
C	36/M	34	DM, cigarettes smoking	GI bleed	CT: significant mesenteric fat stranding and peripancreatic collections in the lesser sac and inferior to the stomach, and there is free fluid in the up- per abdomen	Large D1 ulcer with partial hemo- stasis achieved with adrena- line injection, 2 clean base fundal ulcers. Stomach with un- healthy mucosa	Hypo- tension/ sepsis (necrotiz- ing hem- orrhagic pancre- atitis)	Acute	Explorative laparotomy with total gastrec- tomy and Roux en Y esophagoje- junostomy+ feeding jejunostomy tube	Ne- crotic gastric wall with trans- mural perfo- ration and diffuse ischemic gastric wall with patchy hyper- emia	Death after 20 days , due to ARDS and septic shock with MODS secondary to pancre- atitis

Table 2: Patient Characteristics, Presentations, and Outcomes.

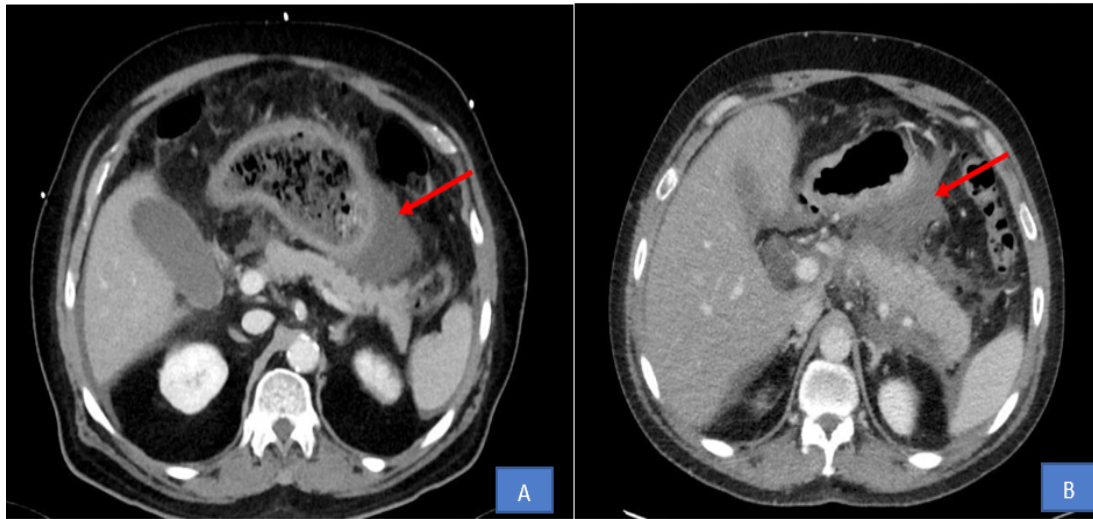


Figure 1: Pre-operative CT abdomen with IV contrast. Images **A:** showing fat stranding and thickening of the gastric wall, with free fluid seen in the lesser sac. **B:** thickened gastric wall with edematous pancreas (Arrow points to the collection).



Figure 2: 3D reconstruction CT angiogram: both images showing extensive chronic calcifications in the abdominal aorta and iliac vessels as well as in the superior mesenteric artery and inferior mesenteric artery. For patient 1.

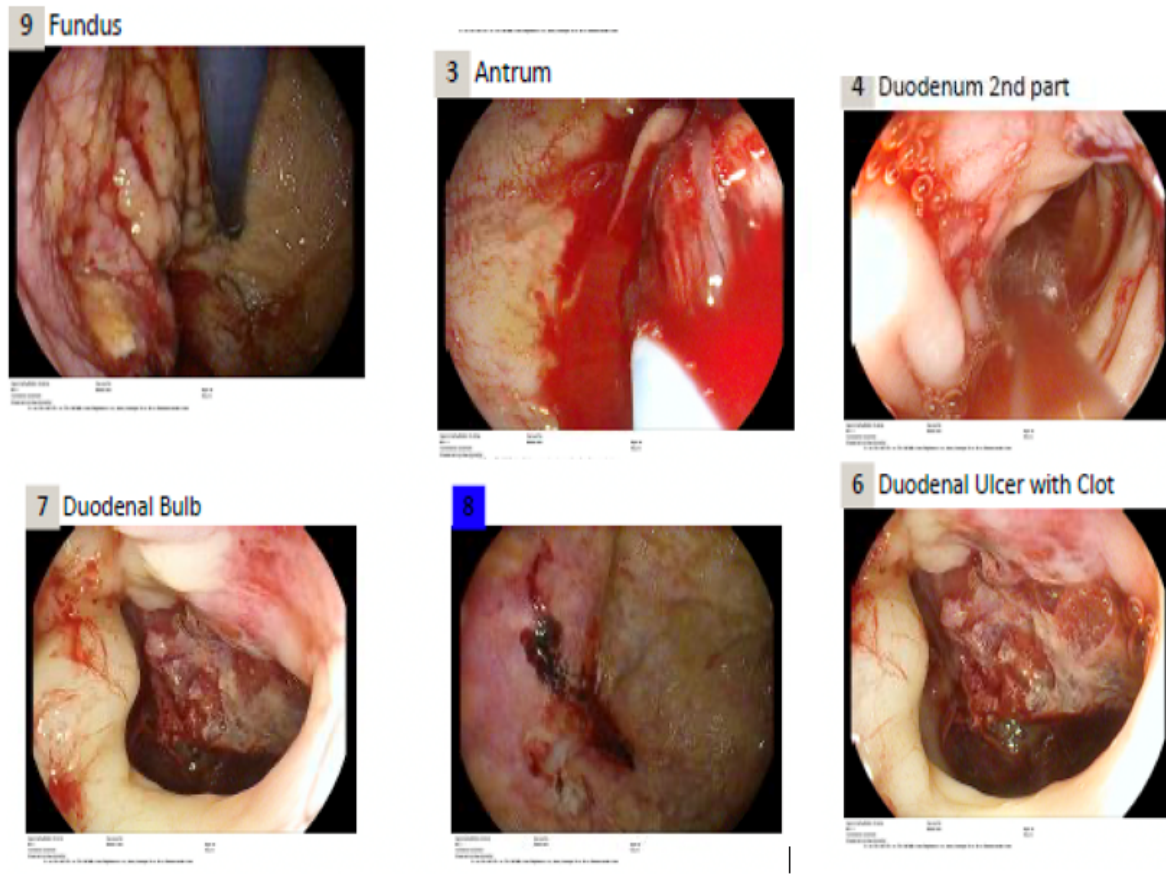


Figure 3: pre op endoscopy for patient 3: unhealthy mucosa.

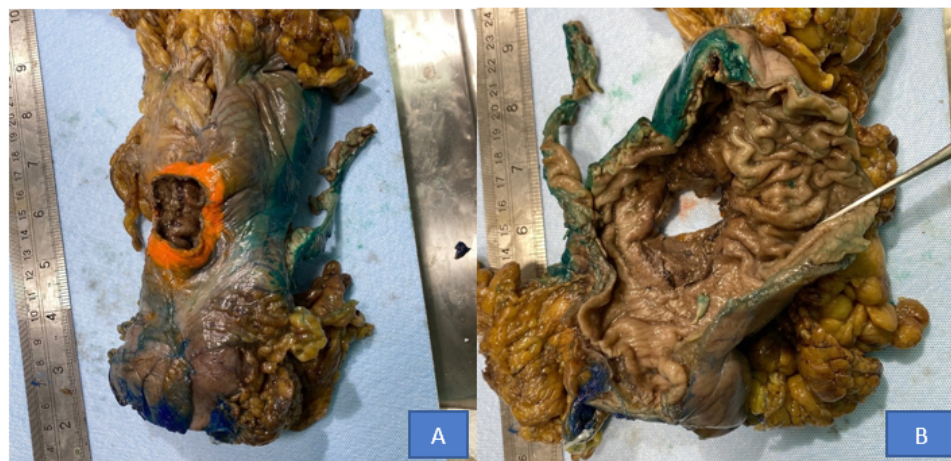


Figure 4: **A:** posterior view: post-operative specimen, with posterior wall of the stomach at the body with large perforation. **B:** anterior view, showing the anterior wall of the body of the stomach opened with the perforation in the posterior wall with friable edges.

Etiology and risk factor: The etiologies for gastric ischemia included: vasculopathies and local vascular causes, systemic hypoperfusion and sepsis. For patient A; he had CT angiogram showing atheroma and stenosis in the celiac trunk, while for the other two patients the main etiology was hypoperfusion and sepsis.

Treatment and outcome: All three patients were initially managed with NGT decompression of the stomach with PPI. All three patients needed surgical intervention which was explorative laparotomy with total or partial gastrectomy and Roux en Y reconstruction. Patients A and C passed away within 20-30 days from the surgery while patient B is still alive 14 months after his surgery. The median duration of hospitalization for our patient cohort was 30 days range (20-65).

Discussion

The rich blood supply that the stomach receives makes it less susceptible to ischemic injury compared with other organs [1]. Gastric ischemia usually occurs due to decrease in gastric blood flow by vascular insufficiency or by reperfusion injury [13]. Vascular insufficiency caused by different etiologies that may be classified by systemic hypoperfusion (such as shock or sepsis) or splanchnic vessel hypoperfusion secondary to gastric volvulus, acute gastric dilatation, stenosis, thrombosis, embolism, vasculitis or vasoconstriction [1,7].

Factors such as advancing age, smoking, atherosclerosis, diabetes, and hypertension predispose to ischemia by causing a decrease in gastric and splanchnic blood flow [5,9,14-16]. Hypertension, diabetes, and hyperlipidemia accelerate the atherosclerotic process, which may affect the celiac axis leading to reduced gastric blood flow [3]. As shown in Table 1; the most common etiology for ischemic gastritis was local vascular causes (75% in sharma et al, 100% in Chambon et al, 100% in Casey et al, 100% in Babu et al), stress ulcer (50% in Herman et al), and hypoperfusion (10-25% in Herman et al, 24% in Sharma et al).

Most of these risk factors applied to patient A who had multiple risk factors for developing gastric ischemia. He was elderly (62 years) old male with long standing history of diabetes, hypertensive and had atherosclerosis. More importantly; upon revision of his pre-operative CT, there was atherosclerosis and atheroma in the abdominal aorta. Gastric ischemia can be also associated with mesenteric ischemia which has been previously reported in the literature [17]. In line with the prior statement, our patient had ischemic segment of proximal jejunum which suggests an element of mesenteric vessels involvement or embolic phenomena with the resultant acute ischemia. To date; we couldn't find any article with coexisting gastric and small bowel ischemia like the presentation of this patient.

Hypotension is another important predisposing factor for gastric ischemia as for the other two patients in this cohort (patients B and C). Herman et al and sharma et al have looked at hypotension as risk factor for ischemic gastritis [7,18]. Systemic hypoperfusion was observed in 25% of our patients, presenting predominantly as septic shock [5].

CT imaging plays a pivotal role in the assessment of gastric ischemia, with the most common suggestive findings being gastric wall pneumatosis and portal venous gas [17]. In case of gastric perforation; extraluminal free air is the most common and consistent finding although it may be absent especially at the onset of the symptoms [18]. Free gas is usually abundant and noted around the liver and the stomach [19]. If the free air is found in the lesser sac, then the likely site of perforation is the posterior wall of the stomach, gastric perforation is seldom correlated with air trapped in the mesenteric or sigmoid recess, in contrast to perforation of the colon and small bowel [20]. Patients A and C had collections in the lesser sac which correlated with ischemic gastritis and perforation in the posterior wall of the stomach.

The medical management of gastric ischemia includes fluid resuscitation, aggressive acid reduction therapy with intravenous proton pump inhibitors, nasogastric tube placement, and selective use of broad-spectrum antibiotics if sepsis or gastric pneumatosis is present [1]. Gastric ischemia impairs motility of the stomach causing fluid and food accumulation leading to stomach distension and worsening of ischemia, thus nasogastric tube decompression alleviates gastric distention and may reduce the extent and effect of ischemia [21]. In managing acute gastric ischemia, supportive measures are generally enough once the initial vascular compromise is reverted, whereas in chronic gastric ischemia, a specific vascular or anatomic variant is usually present and corrective intervention is needed. In patients with severe gastric ischemia or gastric volvulus, surgical consultation and multidisciplinary management are indicated. Surgery is recommended for gastric perforation, as in all our cases, in gangrenous and/or necrotizing gastritis that are not responding to supportive care and broad-spectrum antibiotic therapy [1].

Gastric ischemia is associated with a poor prognosis, and early diagnosis is critical to guide potential interventions. In our literature review; we did not find any patients with small bowel ischemia and/or perforation in association with gastric ischemia, except for Haruna et al case report on duodenal perforation with small bowel ischemia [13]. As it was concluded from most studies Sharma et al looked at mortality rate as 6 out of the 17 (35%) patients in their studies died during follow up: 4 died secondary to gastric ischemia and sepsis or multiorgan failure, whereas 2 died from progression of underlying malignancy [5,22]. From Table 1 we can determine the mortality rate to range from 29% up to 83% from the studies included.

Despite the inherited limitations to retrospective studies; our cohort unintentionally displayed severe presentation of ischemic gastritis and the pivotal role of early diagnosis and prompt surgical intervention to improve the outcomes of the patients. In the two death cases there was 2-5 days delay in reaching the diagnosis which led to this high mortality rate. Another reason was the severity of the sepsis and vasculopathy that might have played a role in the poor outcome for the patients.

It is important to differentiate between patients who develop ischemia acutely and those who have a more chronic course. Acute ischemia will often lead to necrosis and perforation; these patients will present as an acute abdomen and will commonly require surgery

Conclusion

Gastric perforation as a result of gastric ischemia carries high morbidity and mortality and early diagnosis is critical to guide potential interventions. Cross-sectional imaging and endoscopy play an important role in the assessment of ischemic gastropathy and help to guide the type of intervention required. In cases of advanced or complicated disease; early diagnosis and intervention improves the outcomes of patients. Gastric perforation as a result of gastric ischemia carries high morbidity and mortality.

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