

Gastric Perforation in Ndola- Zambia: A Case Report

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Abstract

It is known that there is problem of gastric perforation (GP) in Africa, in the Japanese and the Western countries. However it occurs much less frequently [1,2]. Some cases have been free perforation and some have been major bleeding in patients with gastric cancer. Perforations are serious conditions with potentially dangerous effects however they are rare [3]. There is also evidence that neonatal gastric perforation (GP) does occur but it is a very rare event. It is associated with a poor prognosis and there is still much debate about its etiology [4]. It is believed that the perforation occurs in the lesser curvature and anterior wall of the stomach. Some authors think that gastric Perforation (GP), is caused by a mechanical rupture of the stomach wall, as a result of increased intragastric pressure, in adults, full-term and premature infants [5]. There is also a theoretical concern that the capnoperitoneum (Increased intra-abdominal pressure 10-12mmHg), may aggravate peritonitis and induce septic shock. It is associated with a poor prognosis [6]. The Gastric perforation is also seen that one of four ulcer perforations can be attributed to the use of nonsteroidal anti-inflammatory drugs, a risk factor of particular in the elderly [2].

In peptic ulcer or gastric perforation, Endoscopy and or Laparoscopy are being increasingly used in conditions even though there is complication of peritonitis [7-9].

Keywords: Cancer, Doudenum, Elderly, Endoscopy, Gastric, Laparoscopy, Neonates, Perforation, Poor prognosis

Introduction

There are some conditions that lead to the perforation of the Gastrium. However these are not that common. There also have been cases of gastric perforation occurring during endoscopic resection for early gastric cancer when a surgical treatment generally is performed. This too is rare; for example, from 1987 to 2004, 121 of 2460 patients who underwent gastric Electronic Medical Record (EMR), at the National Cancer Center Hospital, had gastric perforation of 4.9%. Other workers of Japan also reported the fact that free perforation of gastric carcinoma accounts for less than 1% as an incidence of acute abdominal crisis in Japan, they went on to say that this problem occurs much less frequently in Western countries. To clarify the characteristics of patients with perforated gastric carcinoma (PGC) and to investigate a treatment of choice, they reviewed the data of Japanese patients with PGC. They found a total of 155 cases of PGC reported in the Japanese literature from 1985 to 1994, [1,6]. Some

cases have been free perforation and major bleeding in patients with gastric cancer, these are rare, but have serious conditions with potentially dangerous effects. In these cases, patients with gastric cancers who undergo emergency gastrectomy have a poor prognosis [3].

There is also evidence that neonatal gastric perforation (GP) is also a very rare event. It is associated with a poor prognosis too. There is still much debate about its etiology, recent findings suggest that the lack of the interstitial cells of Cajal (ICC) may be implicated in the etiology of idiopathic GP in neonates. Most historical reports describe GP in the neonatal population as “spontaneous”; however, several variables, including prematurity and nasal ventilation, have recently been implicated as contributing factors [4]. Sultan Kara, Zekeriya I, et al reviewed the records of all newborns admitted to their hospital between 1978 and 2001 with regard to gender, age at admission, site of perforation, type of operation, associated anomalies, contributing factors, and outcome. Their findings were that Gastric perforation was diagnosed in 13 neonates: 11 (85%)

boys and 2 (15%) girls. The mean body weight was 2375 g (range 990–3100 g). There were 4 (30.7%) preterm infants and gestational age ranged from 29 to 35 weeks. The mean age at admission was 3.2 days (range 1–7 days). The initial clinical findings were sudden-onset hemorrhage and “coffee-ground” vomiting in 5 of the 9 full-term neonates, and abdominal distension and vomiting in the other four [4]. It revealed their experience of treating 13 neonates with gastric perforation (GP) over the past 23 years. This is not a frequent occurrence.

It is believed that the Perforation occurs in the lesser curvature and anterior wall in 23% infants, at the greater curvature and anterior wall in 15.4% infants, in necrosis of anterior wall in 7.7%, at the esophageal junction and posterior wall in 15.4%, at the lesser curvature and posterior wall in 7.7%, at the lesser curvature and esophageal junction in 7.7%, and the site was not specified in 23%. Twelve patients were treated with gastrorrhaphy and drainage, and 1 was treated with gastrorrhaphy alone. Three patients required additional gastrotomy. Mortality was 53.8% [5]. In gastric Perforation, some authors think that the perforation is caused by a mechanical rupture of the stomach wall, as a result of increased intragastric pressure, in adults, full-term and premature infants [5].

Bloechle C , Emmermann A , Strate T, et al. have demonstrated in recent cases that Laparoscopy is increasingly used in conditions complicated by peritonitis, e.g., peptic ulcer perforation. The theoretical concern is the capnoperitoneum, which may aggravate peritonitis and induce septic shock due to increased intra abdominal pressure and distension of the peritoneum. An animal study was devised to analyze the effectiveness of laparoscopic versus traditional open repair of gastric perforation and abdominal lavage for associated peritonitis. Critical appraisal of laparoscopic surgery is warranted in conditions associated with severe, longstanding peritonitis [6].

For peptic ulcers, Cecilie Svanes puts it as follows: “After increasing steeply at the beginning of the twentieth century, ulcer perforation incidence during the last decades has declined in the young and in men, and it has risen among the elderly and in women”. These changes can be attributed to a cohort phenomenon: It means that it affects certain groups every time of the years. Most ulcer perforations among subjects less than 75 years of age can be attributed to smoking [10]. We know that subjects with a history of ulcer perforation have poorer long-term survival than the general population. About one of four ulcer perforations can be attributed to the use of non-steroidal anti-inflammatory drugs, a risk factor of particular in the elderly.

There are certain measures we have come to know: Ulcer perforation was frequently treated by gastric resection in the past, but from 1887 the first method of choice introduced was suture, It is the method of choice today says Cecilie Svanes [10]. History has it that the introduction of antibiotics improved the prognosis of ulcer perforation surgery greatly, this too has remained the same. We have come to know also that the lethality is higher in the elderly and is higher after gastric than after duodenal perforation.

The actual event was as follows: During the nineteenth century ulcer perforation was a rare disease that occurred mainly in young women, with the perforations located near the cardia of the stomach. During the first decades of the twentieth century ulcer perforation incidence increased greatly, and there was an epidemic of ulcer perforations situated in the duodenum of middle-aged men. In our time, ulcer

perforation incidence has become stable or tends to decline, and most patients with ulcer perforations are elderly men and women, with perforations in the prepyloric and pyloric areas as frequent as perforations in the duodenum in the western world. In Zambia we still see large perforations in the Duodenal region. The Western world also has an epidemic of duodenal perforations among young men but for them it seems to be waning. In both areas the Gastric perforations are still uncommon. The past Ulcer perforation was a lethal disease until surgical treatment was introduced at the end of the century. We were trained that Mikulicz sutured a perforated gastric ulcer for the first time in 1880 and we have come to realize that suture is still the most common treatment for ulcer perforation.

The revolution in ulcer treatment that occurred with discovery of the *Helicobacter pylori* has not yet led to any detectable changes in incidence or treatment of ulcer perforation. We know up to now that the ulcer perforation is still a surgical disease for which the possibility for improvement in prognosis is in the general advances of acute surgery. The potential for prevention lies in better understanding of causal factors, which have not been known. Since then Ulcer perforation incidence has been studied over an extended period in western Scotland (1924–1963) the United Kingdom (1958–1983) and western Norway (1935–1990). Still there is a lot to study. We still do not fully understand.

In addition to all this we are seeing some very interesting gastric perforation in our practice. Carlos Serra, Aniceto Baltasar et al report an interesting case of a gastric perforation. A 27-year old woman who underwent laparoscopic Rouxen-Y gastric bypass. She had a retrocolic-retrogastric herniation of most of the small bowel and later a gastric perforation due to internal hernia at the mesenteric defect of the jejuno-jejunostomy which occurred [11]. Lehnert T, et al. report that the gastric perforation represents a severe complication of gastric cancer. Because it is rare, only few data are available regarding treatment and prognosis of these patients. In their view, the gastric perforation of the stomach should raise suspicion of malignancy, particularly in elderly patients [12]. In their view, they go on to say that their “observations and a review of the literature confirm that perforation of gastric cancer does not preclude long-term survival *per se* in a substantial number of patients”.

It is also now known that the Rapunzel syndrome (RS), though a rare form of gastric trichobezoar extending throughout the bowel can also produce a gastric perforation. Nuran Pul and Mehmet Pul report their case of gastric perforation; a patient with RS causing gastric perforation. They discussed the aetiology, pathogenesis, diagnosis and treatment of this unusual syndrome. In their, view RS is found characteristically in girls with varying gastro-intestinal symptoms. They recommended surgery for the treatment for large or complicated trichobezoars [13]. In fact Nadko G. Velitchkov, Georgi I. Grigorov et al report the fact that Ingested foreign bodies (FBs) present a common clinical problem. Foreign body ingestion is frequently seen in prisoners, psychiatric patients, alcoholics, and senile patients. It is well known that most of them pass uninterrupted through the gastrointestinal tract. However, 10% to 20% must be removed endoscopically, and about 1% to 14% require operative removal. The patients’ ages usually range from 15 to 82 years (mean 26.4 years) No perforations of the gastric stomach or pylorus were encountered at operation. Four patients presented with peritonitis due to perforation of the small intestine – the ileocecal region through appendicostomy [7].

So, many things can cause gastric issues even as much as Gastric perforation. For example there been reports of a complication that can arise from ventriculo-peritoneal (VP) shunting [14]

The new methods of managing Gastric Perforation

It is also known that Gastric perforation is a rare. It is usually a life-threatening condition in neonates and adults. To avoid deterioration, prompt surgical treatment is mandatory [15]. Sylvia Glüer, Annika I. Schmidt et al made a report on two neonates (1 and 8 days old) with feeding tube associated gastric perforation in which they managed laparoscopically by single layer suture repair. Both children suffered from severe peritonitis. Operative time was 60 minutes in both cases. Oral feeding was started on postoperative day 3 and 7, respectively. No complications regarding the gastric perforation were encountered on follow-up (11 and 8 months, respectively) in both cases [16]. They represented the fact that laparoscopic suture repair is a safe and feasible method for surgical treatment of gastric perforation in neonates. This is also essential in adults as was suggested by Kazuki Sumiyama, Christopher J Gostout, et al. [17]. In fact Laparoscopic repair of perforated peptic ulcer has also began to be gaining popularity in recent years, but there are few data that exist to support the superiority of the laparoscopic approach over open repair. Evidence suggests that laparoscopic repair of perforated peptic ulcer confers superior short-term benefits in terms of postoperative pain and wound morbidity. This approach is as safe and effective as open repair [8].

Endoscopy is also being greatly introduced. Kiyoshi Hashiba, Ademar Margonari Carvalho et al have strongly suggested that treatment for gastric perforation should be Endoscopically done, not onlt laparoscopy. In their view the omental patch is a standard surgical treatment of gastroduodenal ulcer perforation. In their report of an experimental method for endoscopic repair of anterior gastric perforations with an omental patch developed by using a porcine model was carried out and their view is that endoscopic repair with an omental patch appears to be an effective procedure for closure of gastric perforations [9].

We present one case of gastric perforation. It occurred in March 2018 at the Ndola Teaching hospital in Ndola Zamb.

Our case presentation

DC was a 44 years old man. He was referred to Ndola Teaching hospital from Mpongwe district hospital.

History of presenting complaints

He was a 44-year old man and was admitted on March 26. The major complaint was that he vomited every meal he ate or water he drank. He denied any history of violence. As a result he had wasted his body weight. The history was that he had differed with is fourth wife and she left him and went to her parents. He was very depressed that he decided to kill himself by taking acid. The patient did not die soon after drinking Acid fluid but he developed difficulty in swallowing water and food.

Review of systems

The respiratory and other systems were essentially normal. However, he was weak and had wasted his body weight. The patient could swallow well but soon vomited whatever he ate or drank.

Past Medical History

There were no known serious illnesses in his life and he had no history of previous admissions to the hospital. There was no violence on him.

Family history

There was no known family history of serious illnesses or psychological problems.

Social History

Our patient never smoked but he drank his alcohol seriously.

General Examination

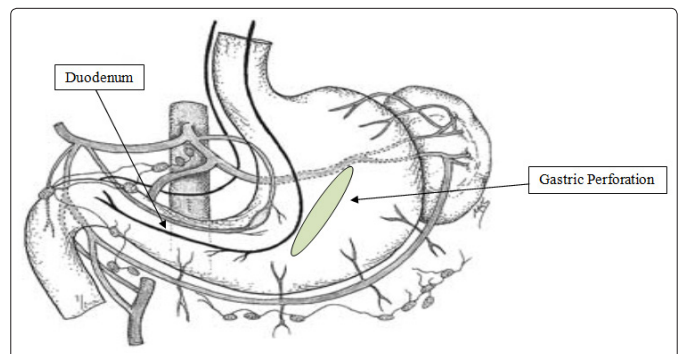
He was found to be very weak and he needed a lot of fluids and feeding. The abdomen was essentially normal. His pulse was 140 per minute and the blood pressure was by and large 100/60mmHg. Our decision was that we take him to theatre and perform a gastrostomy or a jejunostomy so that the patient could be fed and allow him to build his weight before major surgery.

Clinical findings

The Pre –operative heamogram had the following findings: The White Bell Count was $7.78 \times 10^9/L$, the Red Cell Count(RCC) was $3.44 \times 10^{12}/L$.

The Heamoglobin (HB) was 11.0g/dl and the Platelet count was $73 \times 10^9/L$.

The patient needed surgery but he was very week. Initially we started the procedure under local anesthesia but was later converted to general anesthesia due to deteriorating general condition of the patient. The estimated blood loss during the procedure was approximately 80mls. We made our decision to perform a gasrostomy or a jejunostomy. This was carried out. During laparotomy we found that the Duodenum was normal there was no perforation. In fact the Pylorus was very healthy. The perforation was in the Gastrium.



Secondly, we noted that the abdomen was clean, there was no evidence of peritonitis. The significant finding was that the whole abdominal omentum had entered the stomach through the gastric perforation and was attached to the inside of the fundus of the stomach. A lot of the omentum had become gangrenous and necrotic. We removed the attached Omentum out and fluid from the stomach started flowing out and we had to suck it out.

We used finger to feel the Pylorus and it felt entirely normal we the passed a catheter trough the Pylorus and the catheter went easily all the way to the Jejunum. This confirmed to us that this was a Gastric perforation.

The perforated area was closed using 2/0 Vycril and on top of it we attached the little omentum that was available. We created a feeding jejunostomy for the need of feeding for our patient. The abdomen was closed and the patient was taken to the ICU.

The ICU care

Patient was transferred from main operating theatre for ventilator support and close monitoring post laparotomy for the feeding jejunostomy.

The patient had been on cefotaxime and metronidazole whilst on the ward and had received local anesthesia of lignocaine 1% mixed with adrenaline 0.1mcg, later he received ketamine 100mg and 50mg of suxamethonium during conversion to general anesthesia. On examination upon arrival in the ICU

- He was a critically ill patient and he was cachexic, the patient was pale, he had no cyanosis and had no jaundice.
- He was hypothermic with a temperature of 35.2^o Centigrade.
- The Airway was secured with Endotracheol tube size 7.5mm, to make the airway clear.
- The ventilator assisted breathing was put on the patient. The ventilator settings were the mode-SIMV/PC (gasping respiratory pattern) FiO₂-1.0, peep 5, pressure support 20. SpO₂ was 98% and Respiratory Rate was 34 breaths/minute. On auscultation: The chest was clear, there were vesicular breath sounds bilaterally.
- The Blood Pressure was 62/38mmHg, The MAP was 46, The patient had tachycardia heart sounds. He had weak feeble peripheral pulses and the Heart Rate was 112 beats per minute. Two large bore peripheral cannulae were inserted in the patient.
- The Glasgow Coma Score (GCS) was 3T: (Best Eye response: 1. Best Verbal response 1T. Best Motor response: 1.)
- The pupils were equal in size and measuring 3mm. They were bilaterally reacting to light. The Random blood Sugar was 12.8mmol
- The abdomen was not distended. The midline incision dressings were clean and dry. The drain in situ contained approximately 100 mls of serosanguinous fluids

The total assessment in the ICU was that the patient was in Septic Shock Secondary to Necrotic omentum. He was also suspected to have developed acute kidney failure as there was poor urine output.

The plan of management was

1. Rapid infusion IV fluids -1 Litre normal saline over 30 minutes then 1 litre over 1hour then followed by fluids of 150mls/hr. Urgently cross matched 2 units whole blood. We Stopped the cefotaxime. Started the Intravascular antibiotics called cefepime. The patient was given 1gram intravenously 12 hourly. We reduced the dose of metronidazole to 500mg 12 hourly instead of 8 hourly in view of suspected renal dysfunction.
2. The Urgent investigations were carried out: Urinalysis, urea/creatinine/ serum electrolytes, repeat post operative full blood count, Liver function tests.
3. The patient Continued on mechanical ventilation which was the assisted mode.
4. He was started on adrenaline infusion at 0.3-0.5mcg/kg/min infusion (12mg in 200mls of normal saline). This had been our aim that if there was no response to fluid therapy after 2000mls of fluids.

Despite all these efforts our patient died.

Discussion

We have always known that most patients with ulcer perforations are elderly men and women. We in Zambia hardly see women developing Gastric perforation. Perforations in the prepyloric and pyloric areas are as frequent as perforations in the duodenum. In the past ulcer perforation was a lethal disease until surgical treatment was introduced at the turn of the century. Mikulicz sutured a perforated gastric ulcer for the first time in 1880 [2]. The suturing is still the most common treatment for ulcer perforation and it is said that the revolution in ulcer treatment that occurred with discovery of the role of *Helicobacter pylori* has not yet led to any detectable changes in the incidence or treatment of ulcer perforation. Ulcer perforation is still a surgical disease for which the possibility for improvement in prognosis lies with the general advances of acute surgery. The potential for prevention depends on better understanding of the causal factors, which have not been fully known until now, it is said [2]. The history suggests that during the nineteenth century, gastric ulcer perforation incidence increased greatly up to the twentieth century. After that time the Western world has seen an epidemic of duodenal perforations among young men. This is the same pattern in Zambia. Where as in the Western world this now seems to be waning. Here we have no studies to conclude. Our friends in UK have studied over an extended period in western Scotland (1924–1963), the United Kingdom (1958–1983) on Ulcer perforation incidences.

It is known that in the gastric perforation in Africa, in the Japanese and the Western countries occurs much less frequently [1,2]. Some cases have been free perforation and major bleeding in patients with gastric cancer and are serious conditions with potentially dangerous effects but they are rare [3]. In Zambia too this illness is not common.

There is also evidence that neonatal gastric perforation (GP) does occur but it is a very rare event. It is believed that the Perforation occurs in the lesser curvature and anterior wall. Some authors think that in gastric perforation(GP), the perforation is caused by a mechanical rupture of the stomach wall, as a result of increased intragastric pressure, in adults, full-term and premature infants [5]. It is associated with a poor prognosis [6]. Our patient came with a history of having drunk acid and denied soon after surgery. There was no history of violence. We expected a Pyloric stenosis in our patient but on surgery we found a gastric perforation in which the Omentum had gone into his stomach and caused him to vomit. His condition was associated with a poor prognosis. This was not a common occurrence.

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