Perforated gastric and duodenal ulcers in an urban African population

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Summary

Background: **Perforations of the stomach and duodenum are frequent causes of acute generalized peritonitis in our environment. This is a prospective study of 331 cases of gastric and duodenal perforations.**

Study design: **A consecutive series of adult patients admitted and treated for acute generalized peritonitis due to peptic ulcer perforations was studied prospectively from January 1998 to June 2004. A specially designed proforma was used to record patient characteristics, clinical and operative findings and outcome. Additional information on self-medication and other relevant social habits was obtained and recorded. The data was analysed and the ratio of gastric to duodenal perforations as well as factors associated with perforations determined.**

Results: **More males were affected by a ratio of 3.3:1. For all the patients the median age was 55.0 and the mean age was 52.2 ± 17.5 years. The mean age of the perforated gastric ulcer patients was 47.7 ± 17.1 years but higher for perforated duodenal ulcer patients at 64.8 ± 11.4 years. There were 244 gastric and 87 duodenal perforations giving a ratio of 2.8:1. Most of these perforations were associated with the intake of Non Steroidal Anti Inflammatory Drugs (NSAIDS) 158 (47.7%), habitual ingestion of herbal medicines or concoctions 81 (24.5%) and prolonged fasting for religious reasons, 18 (5.4%).**
Acute renal failure 32 patients (31.1%), continuing or persistent peritonitis 12 patients (11.6%), wound infection 25 patients (24.3%), and chest infection 12 patients (11.6%), were some of the 103 post - operative complications seen. The overall mortality was 73(22.1%). The death rate from perforated duodenal ulcer was 27 (32.2%) and that from perforated gastric ulcer was 46 (20.1%).

Conclusion: **In our community perforated gastric ulcer is seen more often than perforated duodenal ulcer. These gastric perforations are related to the widespread use of NSAIDS and herbal medicines.**

Key-words: ***Gastroduodenal perforations, Patterns, NSAIDS abuse, Herbal medicines, Concoctions, Mortality.***

Résumé

***Introduction*:-** Perforations d'estomac et du duodénum sont des causes fréquentes de la péritonite généralisée aigue dans notre milieu. Il s'agit d'une étude en perspective de 331 cas des perforations gastriques et duodénals.

***Plan d'étude*:** Une série consécutive des patients adultes admis et traités pour la péritonite généralisée aigue provoquée par des perforations peptiques d'ulcère était étudiée prospectivement à partir du janvier 1998 jusqu' en juin 2004. Un proforma était spécialement préparé et utilisé pour noter des caractéristiques des patients, des résultats chimiques et opératoires. Des informations suplémentaires sur le médicament de soimême et d'autres habitudes sociales et pertinentes ont été obtenues et notées. Les données ont été analysées et la proportion de la gastrique par rapport à des perforations duodénales de même que des facteurs liés avec des perforations déterminées.

***Résultats*:-** Beaucoup plus du sexe masculin ont été touchés d'une proportion de 3,3 : 1. Pour tous les patients l'âge médian était 55,0 et l'âge moyen était 52,2±17,5 ans. L'âge moyen des patients atteints d'ulcère gastrique perforé était 47,7± 17,1 ans mais plus élevé chez des patients atteints d'ulcère duodénal perforé en 64,8± 11,4 ans. Il y avait des cas de 244 gastriques et 87 duodénaux perforés ce qui donne une proportion de 2,8 : 1. La plupart de ces perforations étaient liées à la consommation des drogues nonstériodales Anti inflammatoire (DSNSAI). 158 soit 47,7%, ingestion habituelle des médecines par les plantes, phytothérapie ou concoctions 81 soit 24,5% et jeûne prolongé pour des raisons religieuse, 18 soit 5,4%. Insuffisance rénale aigue chez 32 soit 31,1% des sujets, péritonite continuelle ou incessante 12 soit 11,6% des sujets, infection à travers la blessure 25 soit 24,3% des sujets, e t infection dans les poitrines 12 soit 11,6% des sujets, étaient certaines d'entre 103 complications post-opératoires vues.
Dans l'ensemble , la mortalité était 73 soit 22,1% taux des morts provoquéées par l'ulcère duodénal perforé était 27 soit 32,2% et celui d'ulcère gastrique perforé était 46 soit 20,1%.

***Conclusion*:-** Dans notre communauté ulcère gastrique perforé est vu le plus souvent plus que l'ulcère duodénal perforé. Ces perforations gastriques sont attribuables à la consommation généralement admise de DSNSAI et médecine par les plantes.

Introduction

Acute perforations of the stomach and duodenum continue to cause significant morbidity and mortality in Ghana and elsewhere[1-2]. These perforations are usually considered mostly as complications of peptic ulcer disease[2-3]. There are wide variations in the prevalence of peptic ulcer disease in different populations especially in Africa[4]. Duodenal ulceration for instance is reported to have a high incidence in the coastal region of West Africa and the Bile/Congo water shed, but a low incidence in the northern savannah of West Africa [5]. Gastric ulcer is considered a rare disease in Africa being 6-30 times less com-

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mon than duodenal ulcer [6-9]. Records from our upper gastrointestinal endoscopies reveal an approximate ratio of 20 times more duodenal than gastric ulcers. It was therefore unusual to notice in our clinical workload, more gastric than duodenal perforations. Many of these gastric perforations were found in patients less than 30 years old. It is against this background that this study was planned and designed. The aim is to report the high incidence of gastric perforations seen in our hospital and to highlight some of the patient - related factors that are associated with these gastric perforations.

Patients and methods

This prospective study was done at the Komfo Anokye Teaching Hospital serving the city of Kumasi, Ghana, and which has a population of over one million people; mostly artisans, traders and farmers. The study covered a period of 6 ½ years - January 1988 to June 2004. A consecutive series of patients with a clinical diagnosis of generalized peritonitis, and the identification of a perforation in the stomach or duodenum at laparotomy was studied. Patients with generalized peritonitis from other causes were excluded.

Baseline and routine investigations were performed and resuscitative measures established. Gastric perforations patients were treated by excision of the edge of the perforation and simple direct suture closure with vicryl 3/0. Perforated duodenal ulcers were treated by the standard Grahams technique of closure with an omental patch. The abdomen was lavaged with warm saline and closed with No.2 nylon. Gastric (antral) biopsies were not taken to determine the *Helicobacter pylori* infection rate either by the antibody test, culture or histology. The patients were not specifically treated for *Helicobacter pylori.* Antibiotic therapy administered for the peritonitis was expected to have eradicated any associated *Helicobacter pylori* present. Intravenous omeprazole was started immediately post – operative until the peritonitis improved. With the return of bowel activity and the resumption of oral intake of fluids, the omeprazole was changed to the oral preparation and given for a total of 14 days post – operative. Post-operative upper GI endoscopy was arranged only for patients with persistent dyspepsia after a maximum of 6 weeks on omeprazole.

Data collection

Data was collected using a specially designed proforma and included the patients demography, clinical symptoms and signs, past medical history, drug history including self-medication habits, the use of NSAIDS and herbal concoctions; as well as operative findings, operative procedures, post-operative complications, length of hospital stay and follow up information. A special questionnaire was completed as soon as the patient was considered well enough to do so after the operation.

Statistical analysis

All the data collected was entered into an IBM compatible personal computer and analysed using the SPSS version 11 software. Average values are given as mean ± SD

Results

There were 331 patients with gastro duodenal perforations representing 24.3% of the 1358 acute abdominal admissions during the period. These patients 'included 234 (70.7%) men and 97 (29.3%) women – M/F ratio of 2.4:1. The ages range from 17 to 95 years with a median age of 55.0 and mean age of 52.2 ± 17.5 S.D years.(Table 2)

There were 224 gastric perforations 162 in men and 82 in women male/female ratio of 2.1 ; 1 The ages ranged from 17 to 81 years with a mean of 47± 17.9 years .

A total of 87 duodenal perforations were studied. There were 66 men and 21 women M: F ratio of 3:1.The ages varied, ranging from 35 to 95 years with a mean of 64.8 ± 11.6 years.

Medication habits

Gastric perforations: Of the 224 patients treated for gastric perforations 136 (55.7%) admitted to the use of NSAIDS and were actually taking these drugs at the time of the perforation. A history of ingestion of herbal medicines and concoctions was obtained from 73 (29.9%) patients. The other patient – related factors associated with gastric perforations are shown in table 1.

***Duodenal perforations*:** Duodenal perforations associated with NSAIDS use were 22/87 (25.3%) and ingestion of herbal medicines or concoctions 8 /87 (9.2%).Table 1.

In both groups of patients, the traditional medicines were taken to treat impotence, infertility, fever and many other ailments similar to those for which NSAIDS were used. Table 1 summarizes the patient-related factors associated with the gastroduodenal perforations.

Other associated risk factors

Table 1 Patient-related factors associated with 331 gastro-duodenal perforations

Ulcer type	Number (%)						
	Regular NSAIDS users >12 weeks	Brief NSAIDS users <7 days	Fasting >3 days	Dyspepsia	Herbal medicine > 7 days	Without history of associated factors	Total
Gastric	90(36.9)	46 (18.9)	12(4.9)	7 (2.9)	73(29.9)	16(6.6)	244 (100)
Duodenal	12(13.8)	10 (11.5)	6 (6.9)	44 (50.6)	8 (9.2)	7 (8.0)	87 (100)
Total	102 (31.1)	56(16.9)	18(5.4)	51 (15.4)	81(24.5	23 (6.9)	331(100)

Table 2 Associated factors, age and sex distribution of gastroduodenal perforations

Patient group	Ulcer type	Male	Female	Total	Mean age (S.D.)
Regular NSAIDS	G.U.	54	36	90	55.2 (15.8)
User's> 12 weeks	D.U.	8	4	12	65.2 (7.0)
	Total	62	40	102	
Brief NSAIDS Users	G.U.	35	11	46	28.8 (10.0)
<7 days	D.U.	6	4	10	52.0 (5.8)
	Total	41	15	56	
Herbal Treatment	G.U.	50	23	73	51.5 (14.7)
	D.U.	5	3	8	54.1 (16.5)
	Total	55	26	81	
Fasting	G.U.	7	5	12	
<3 days	D.U.	4	2	6	45.2 (14.7)
	Total	11	7	18	52.1 (13.5)
Previous Dyspepsia/ulcer	G.U.	5	2	7	52.1 (13.4)
>3 months	D.U.	25	19	44	65.1 (12.5)
	Total	30	21	51	
No association	G.U.	11	5	16	52.0 (9.9)
Noted	D.U.	4	3	7	56 (5.0)
	Total	15	8	23	
All perforations	G.U.	162	82	244	47.0 (17.9)
	D.U.	66	21	87	64.8 (11.6)
	Total	228	103	331	55.5 (12.3)

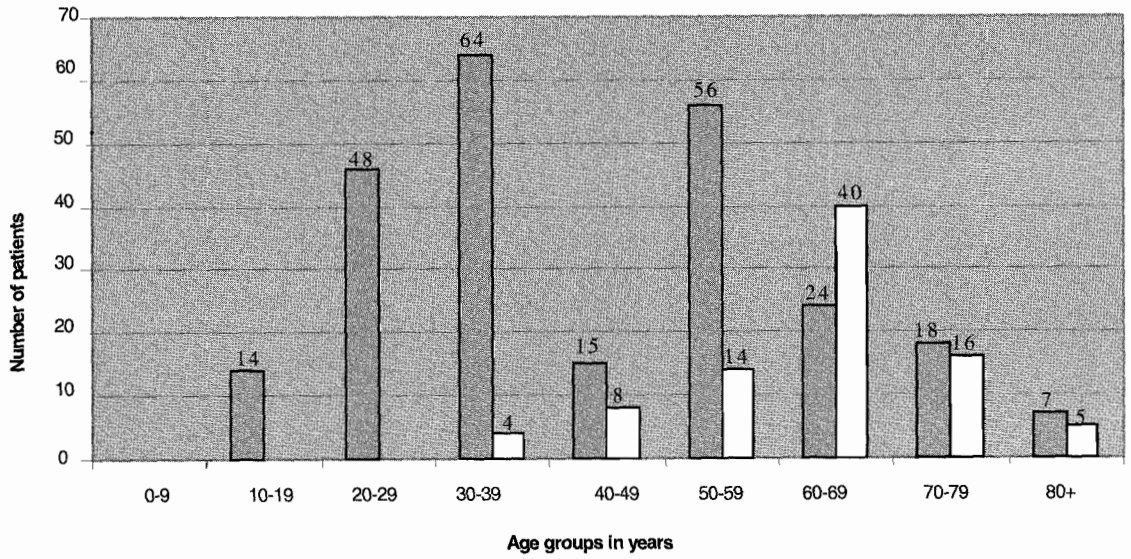

Figure 1 *Age distribution of 331 patients with gastro-duodenal perforations*

Table 3 Causes of death in 46 gastric and 27 duodenal perforations.

Cause of death	Ulcer type	No. of patients	%
Acute renal failure	G.U.	19	
	D.U.	7	
	Total	**26**	**35.6**
Hypovolaemic shock	G.U.	11	
	D.U.	4	
	Total	**15**	**20.1**
End toxic shock	G.U.	7	
	D.U.	3	
	Total	**10**	**13.6**
Persistent peritonitis	G.U.	3	
	D.U.	3	
	Total	**6**	**8.2**
Bilateral pneumonias	G.U.	3	
	D.U.	2	
	Total	**5**	**6.8**
Haematemesis	G.U.	0	
	D.U.	3	
	Total	**3**	**4.1**
Aspiration of gastric contents	G.U.	1	
	D.U.	2	
	Total	**3**	**4.1**
Acute pulmonary oedema due to fluid	GU	**0**	0
	DU	4	0
	Total	4	5.5
Gastric fistula	GU	1	
	DU	0	
	Total	1	1.3
All deaths	GU	46	20.1
	DU	27	31.0
	Total	73	22.1

Gastric perforations

There were 12 patients (4.9%) who had been fasting for more than 3 days at the time of the perforation. Only 7 patients (2.9 %) had a previous history of dyspepsia or endoscopic diagnosis of peptic ulcer. There were 16 patients with no history of a risk factor (Table 1).

Duodenal perforations

Of the 87 patients with duodenal perforations, the largest group were those who had a previous history of dyspepsia or known peptic ulcer disease 44 (50.6 %) (Table 1). Six patients (6.9%) have been fasting for more than 3 days. In 7 patients no history of an associated risk factor was obtained. (Table 1).

Diagnosis

The clinical diagnosis of a perforated ulcer with diffused peritonitis was confirmed by chest radiographs which revealed variable amount of gas under the diaphragm in all the patients. No abdominal CT scans were done.

Operative findings

Gastric perforations

The 244 perforations in the stomach were located in the anterior antrum/ pyloric area in 156 (63.9 %), the anterior body in 65 (26.6 %) , the greater curvature in 10 (4.5%) , the lesser curvature in 7(3.1%) and the cardia in 6 (2.7%). The size of the perforations ranged from 0.3cm – 3.5.cm with a mean of 2.3 $\pm$ 1.5 SD cm. Acute ulcer perforations

were closed in (209)80%) and chronic ulcers in 35 (20%). Perforated gastric cancers were excluded from this report.

Duodenal perforations

Eighty-five perforations were on the anterior aspect of first part of the duodenum. There were two perforations on the anterior aspect of the second part of the duodenum. Posteriorly located penetrating ulcers were excluded from this study. These perforations were larger than the gastric ones ranging from 1.0 – 3.5cm with a mean of 2.5 ± 0.5 SD cm. There were 55 (63.2%) chronic and 32 (36.8%) acute duodenal ulcers.

Outcome

Post operative complications occurred in a total of 103 patients (31.1%:103/331).

Gastric perforations

Sixty – four patients (26.2%) (64/244) developed complications after closure of 244 gastric perforations. These complications included acute renal failure 21 patients (32.8%) (21/64), wound infections 17 patients (26.6%) (17/64), shock 11 patients (17.2%) (11/64) and chest infections 7 (10.9%) (7/64). The other complications noted and the numbers of patients affected were persistent peritonitis 4, burst abdomen 2, and 2 cases of gastric fistula.

Duodenal perforations

Thirty-nine post operative complications were noted in this group of patients (44.8%), (39/87). The most common was acute renal failure seen in 11 patients (11/39) (28.2 %). The other complications were persistent peritonitis in 8 patients (20.5%) (8/39), wound infection 8 patients (20.5%) (8/39), chest infection 5 (12.8%), shock and burst abdomen with 4 and 3 patients respectively.

There were a total of 73 deaths from 46 gastric and 27 duodenal perforations – an over all mortality of 22.1%. All the deaths were post-operative. Thirty - one deaths (42.5%) occurred 48 hours and 42 deaths (57.5%) occurred 6-7 days after the operation. The causes of death are shown in Table 3. The mortality rate was higher in the duodenal perforation group 31.1% (27/87) than in gastric perforation patients 20.1 % (46/224).Also mortality was age related with more deaths recorded in patients aged 60 years or over.

The length of hospital stay varied from 8 to 42 days with a median of 16 days. Follow up was possible for 270 patients (81.6%) at two weeks, but only for 103 (30.8%) at 6 weeks. There were no cases of re- perforation in the post operative or the follow - up periods.

Discussion

Gastric perforations out-numbered duodenal ones by a ratio of 2.8:1 (Table 1), affected younger patients (Fig. 1) and were associated with a high mortality (Table 3). It is noted that these findings are unlike those of most major previous reports where duodenal perforations are more often seen [2-4]

It has been suggested that perforated peptic ulcer may have a different pathogenesis from the uncomplicated chronic disease, and that the first should not be regarded simply as a complication of the second [10]. Furthermore gastro-duodenal perforations are now regarded as a heterogeneous group associated with interacting but also independent risk factors including smoking,[11] alcohol, NSAIDS use, age and history of peptic ulcer [12-13]. These observations from previous reports may explain the pattern of perforations in this series.

The large numbers of the gastric perforations, the very wide age distribution(S D of 17.5) , the location of the perforations (mainly antrum/ body) and the associated mortality are findings that reflect more on the widespread use and abuse of NSAIDS and herbal medicines in both sexes and all age groups (table 2) than on the prevalence of chronic gastric ulcers, which are rare in Africa [6-9]. Other studies have reported a high proportion of NSAIDS use in patients with peptic ulcer perforations [13].

HelicobacterPylori infection is widespread in Africa [14-18] and in our communities especially in patients with dyspepsia [18]. It is however known that perforated gastric ulcers have been less frequently associated with *Helicobacter pylon* infection occurring more in the *H. pylori*-negative stomach [12,19].This may partly explain the finding that less than 5% of the gastric series were associated with previous dyspepsia.

Duodenal perforations affected mostly patients with dyspepsia or known peptic ulcer disease. This may be explained by the documented high incidence of duodenal ulcers in our sub-region [5] and which are known to be associated with *H. pylori* infection in the majority of cases [18]. However NSAIDS and herbal concoction use were also associated with about a third of the duodenal perforations (34.5%) further demonstrating the heterogeneous aetiology of gastroduodenal perforations. [10, 12, 13]

Risk factors

It is nearly 100 years now that was, postulated the concept of acid / pepsin digestion versus mucosal resistance to explain peptic ulcer disease. Many factors have since been considered to reduce mucosal resistance and thus facilitate ulceration and perforation [22-23].

In this series 6.6% of the gastro-duodenal perforations gave a history of fasting for more than 3 days preceding the illness In 8.4% of the cases, the perforations were not associated with a definite event.

Prolong fasting by keeping the stomach empty and allowing prolong contact between acid/pepsin and the mucosa may lead (in theory) to acute mucosa injury and perforations; a situation similar to acute perforations and bleeding in high risk patients in Intensive Care Units ICU [21]. Thus the association between ulcer perforation and prolong fasting seems biologically plausible [11]

The standard surgical treatment of perforated gastroduodenal ulcer is the omental patch repair (Graham) with peritoneal lavage. [24-25] However simple suture closure after excision of the ulcer edge to expose tissue with good vascularisation and peritoneal lavage has produced satisfactory results in many institutions.[26] In our series the simple closure technique was used for the gastric and the omental patch for the duodenal perforations. This approach resulted from our findings that the gastric perforations

were mostly acute (80%) and the edges were easier to approximate. Many of the duodenal perforations were due to chronic ulceration (63.2%) associated with chronic inflammation and fibrotic edges. It was much easier and safer to close these perforations with an omental patch.

Over the past years there have been some new developments in the diagnosis and management of perforated gastroduodenal ulcers. The detection of pneumoperitomum using the standard plain chest X- ray has been the mainstay of the diagnosis of perforated peptic ulcer.[27-28] Today contrast – enhanced thin sliced spiral CT scans are now considered the most reliable method of detecting a small pneumoperitoneum before surgery and the gold standard for the diagnosis of a perforation[29] Ultrasonography has also been found to be superior to plain radiographs in the diagnosis of free intra – peritoneal air[29].

In this series we relied on plain radiographs of the chest to establish the cause of peritonitis in our patients as perforations. The reported incidence of pneumoperitoneum following a perforated peptic ulcer - using the standard horizontal X-ray beam plain chest radiograph - has varied between 50% and 85%[21, 27, 29]. This variable incidence may be related to the site, size and duration of the perforation[21]. In many instances pneumoperitoneum may be detected much earlier in gastric than in duodenal perforations. Long standing perforations may lead to the accumulation of large amounts of intra- peritoneal air. In our series there were more gastric than duodenal perforations; most perforations were anterior, and many of the patients had fully established diffused peritonitis. These findings may explain the detection of free intra – peritoneal air in all our cases. Increasing availability of ultrasonography and CT scan services on emergency basis may provide for the detection of pneumoperitoneum in suspected perforations before diffused peritonitis is established.

Laparotomy was performed for all our patients. Today laparoscopic omental patch repairs have been safely performed in many institutions[30-31].

Following the very high healing rates achieved for uncomplicated peptic ulcers with a combination of acid – suppression (omeprazole) and the eradication of *Helicobacter pylori* infection [32-33], many surgeons now consider the eradication of *Helicobacter pylori* as essential for the prevention of ulcer recurrence after simple closure of a perforated ulcer.[34] There is some evidence to show that the course of intravenous antibiotics that are routinely prescribed for patients with perforated ulcer might eradicate *H. pylori*.[34] However unlike the treatment of the uncomplicated ulcer, the ideal treatment regimes for the perforated peptic ulcer based on controlled clinical trials are yet to be established [35] especially for African populations. The post- operative acid – suppression with omeprazole for 14 days in this series probably prevented the otherwise high ulcer recurrence rates as there were no re-perforations in the post – operative or follow- up periods. In an occasional patient with persistent dyspepsia omeprazole was given for 6 weeks while waiting for an upper GI endoscopy .Multi- centre controlled clinical trials are required to determine the optimum duration of post-operative omeprazole after simple closure of a perforated peptic ulcer.

The high over- all complication rates (32.9%) in this series may reflect the severity of the peritonitis and is related to the equally high mortality of 20.1% and 31.1 %. for gastric and duodenal perforations respectively The over all mortality was 22.1%. Several factors may account for the very high mortality in this series. The patients treated for duodenal perforations were on the average 65 years or older. Acute renal failure was the most common cause of death accounting for over a third of the deaths (table 3).

Conclusion

In our community gastric perforations are more common than duodenal perforations and are mostly associated with the use and abuse of NSAIDS and herbal medicines.

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