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Original Research Article

Peptic ulcer disease in school children aged 2-11 years in Southeast Nigeria

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ABSTRACT

Background: A peptic ulcer is a sore on the inner lining of the stomach or duodenum, caused mainly by non-steroidal anti-inflammatory drugs and Helicobacter pylori infection. In childhood, peptic ulcer disease (PUD) is an uncommon disease, with an estimated frequency of 1 case in 2, 500 hospital admissions in the United States. Data for developing countries, including Nigeria, are scarce, although peptic ulceration is being increasingly recognized in children in the developing world now. For this reason, this study was designed to investigate PUD in school children in Southeast Nigeria.
Methods: 264 children, aged 2-11 years, who attended Cottage Hospital Inyi in 2020 with complaints of abdominal pain were investigated for PUD with stool antigen test. Data collected for a period of one year were analysed as proportions and Chi square, using MaxStat (version 3.60) statistical software. P≤0.05 was considered significant.
Results: The prevalence of PUD was 16%; 8.3% in females and 7.6% in males. In the 2-6 years age group prevalence was 7%, while in the 7-11 years age group it was 9%. Association between sex and prevalence of PUD was not significant (p=0.62), but very significant for that with age (p=0.0003).
Conclusions: Prevalence of PUD was 16%. This increased with age (7% in the 2-6 years age group and 9% in the 7-11 years group). To address the rising prevalence of PUD in these children, there is need to monitor their feeding habits in school, besides teaching them the importance of personal hygiene.

Keywords: Peptic, ulcer, Disease, *H. pylori*, Childhood, Nigeria

INTRODUCTION

Simply put, a peptic ulcer (caused by peptic acid) is a sore on the inner lining of the stomach or duodenum.[1] However, histologically, it is defined as a breach in the mucosa that extends through the muscularis mucosae into the submucosa or deeper layer.[2] Anatomically, a peptic ulcer is represented by a mucosal break greater than 3-5 cm in the stomach or duodenum with a visible depth. And so, PUD refers to ulcerative disorders of the lower oesophagus, upper abdomen and lower portion of the stomach.[3] Under normal conditions, there exists a physiologic balance between gastric acid secretion and gastroduodenal defense. Disruption of this equilibrium between the aggressive factors and defensive mechanisms can lead to ulcer formation. The well-known aggressive factors include acid, pepsin and *Helicobacter pylori*. On the other hand, the defensive factors are gastric mucus, bicarbonate ions and prostaglandins, along with innate resistance of mucosal cells.[4] Even though the use of non-steroidal anti-inflammatory drugs (NSAIDs) and H. pylori infection have been identified as the two main risk-factors for peptic ulcer, relatively few people with *H. pylori* infection who are taking NSAIDs develop PUD, suggesting that personal susceptibility to bacterial virulence and drug toxicity may be essential to the initiation of mucosal damage.[5] The main mechanism of NSAID-associated damage of the gastroduodenal mucosa

is the systemic inhibition of constitutively expressed cyclooxygenase-1 (COX-1), which is responsible for prostaglandin synthesis, and is associated with decreased mucosal blood flow, low mucus and bicarbonate secretion, in addition to the inhibition of cell proliferation.[2]

The second factor, *H. pylori*, is a common infection worldwide. The mechanism by which it induces the development of different types of lesions in the gastroduodenal mucosa is not yet fully explained. Although more than 70% of individuals with PUD are infected by H. pylori, fewer than 20% of these individuals eventually develop peptic ulcer. The aetiology of primary PUD is mainly attributable to infection by *H. pylori* which is usually acquired in childhood in an environment of unsanitary conditions and crowding, mostly in countries with lower socio-economic status. Furthermore, data from sero-prevalence surveys indicate that *H. pylori* incidence increases with the age of the children (before the age of 10), the household size and with lower socio-economic status. In addition, it has been found that the incidence varies with ethnicity.[6,7] Also, a positive family history in first degree relatives of children with PUD has been found in one quarter to two thirds of cases.[8] This may be accounted for by *H. pylori* infection which is known to cluster within families and other close knit communities. A higher frequency of *H. pylori* infection is found in spouses and offspring of *H. pylori*-infected adult cases, probably suggesting that genetic factors are less important than environmental ones.[8]

The third category of peptic ulcers, idiopathic ulcers, caused by neither *H. pylori* infection nor the use of NSAIDs, can be diagnosed in about one-fifth of cases of all ulcers.[9] Presently, the pathogenetic mechanisms behind the development of idiopathic peptic ulcers are still unknown.[10] Nonetheless, a Danish study has shown that psychological stress could increase the incidence of idiopathic ulcers.[11] Studies have also shown that the prevalence of idiopathic ulcers has been increasing in recent years.[12] Furthermore, a multicenter study in France has found that 22% of patients with duodenal or gastric ulcer were neither infected by *H. pylori*, nor using ulcerogenic drugs.[9] Between 20% and 50% of duodenal ulcers in the USA and 3-12% in Europe have been found to be negative for *H. pylori*.[13] Since an idiopathic ulcer is a diagnosis of exclusion, before designating an ulcer as one, it is important that all other risk factors be excluded.

Among other symptoms, a dull or burning pain in the stomach, anywhere between the umbilicus and the breast bone, is the most common in PUD. The diagnosis of PUD is based on medical history, physical examination, laboratory tests, upper gastrointestinal endoscopy and CT scan.

Laboratory tests are the most commonly used methods of investigation in PUD, especially in resource limited settings. They include serological tests, urea breath tests and stool antigen tests.

Serological tests detect immunoglobulin G specific to H. pylori in serum, but cannot distinguish between active infection and a past infection. Urea breath test (UBT) requires the ingestion of urea labelled with the nonradioactive isotope carbon 13 or carbon 14. Stool monoclonal antigen test, just like UBT, detects only active infection and can be used as a test of cure. When in doubt, endoscopy with biopsy is recommended to rule out cancer and other serious causes in patients 55 years or older, or with more alarm symptoms.

Untreated or poorly treated PUD can lead to some of its complications. The commonly known complications include bleeding, perforation, penetration into a surrounding organ, and obstruction from fibrotic stricturing (usually in the pyloric region). Therefore, it becomes imperative that all patients with PUD are treated for *H. pylori*, in order to prevent these complications.[14] Standard therapy is a reasonable initial therapy where clarithromycin resistance is low.[15] First line therapy includes the use of proton pump inhibitor (PPI) and two antibiotics (clarithromycin and metronidazole or amoxicillin for 7-14 days. Current data suggest that increasing the duration of therapy to 14 days significantly increases the eradication rate of *H. pylori*.

Prevention of PUD consists in proper environmental sanitation to reduce or eradicate the transmission of H. pylori within the population and judicious use of NSAIDs. It also includes employment of ‘test and treat’ strategy for *H. pylori* in patients under 60 years with suspected PUD who have no symptoms.[16]

Geographic prevalence of *H. pylori* varies worldwide, with a prevalence of less than 40% in developed countries and more than 80% in developing ones, especially in Africa, Central America, Central Asia and Eastern Europe.[17,18] About 3 million people are suffering from peptic ulcers (duodenal and gastric) and 310, 000 new cases are being diagnosed each year, with a male-to-female ratio of about 3:1.[19] Around 4,500 people die from PUD yearly.

PUD is an uncommon disease of childhood, with an estimated frequency of 1 case in 2,500 hospital admissions in the United States. Although data for developing countries, especially from Africa, are scarce, nonetheless, peptic ulceration is being increasingly recognized in children in the developing world.

In Nigeria, studies on the prevalence of PUD in children are scanty. One Nigerian study found an overall sero-prevalence rate of 30.9% among the children seen in a tertiary hospital in Uyo, Southern Nigeria.[20] The need to contribute to data on PUD from Nigeria, especially the southeastern part, was the main thrust of this study.

METHODS

This was an observational study carried out in Cottage Hospital Inyi in 2020. Inyi is one of the towns that make up Oji River LGA of Enugu State. Oji River is in the Enugu West Senatorial District of Enugu State, bounded in the north by Udi LGA, in the south by Anambra State, in the east by Awgu LGA, and in the west by Ezeagu LGA. The catchment areas of the Cottage Hospital include all the towns in Oji River LGA and adjoining towns of Anambra and Abia States.

Through purposeful sampling, all the children aged 2-11 years, who presented to the hospital with complaints of abdominal pain in 2020, were investigated for PUD using stool antigen test. Stool samples were collected from the study participants in tiny vials with the addition of specific reagents and a colour developer. A blue colour change indicated the presence of *H. pylori*, a positive result. Results were collected within 1-4 days of the test. Altogether, 264 children were recruited into the study. Data which were collected for a period of one year (from January-December 2020) were analysed as proportions and Chi-square, using MaxStat (version 3.60) statistical software. P≤0.05 was considered significant.

Inclusion criteria were clearly stated, children aged 2-11 years who presented with complaints of abdominal pain. Exclusion criteria were therefore all those children <2 years or >11 years old and all those who did not complain of abdominal pain on presentation.

Because this was an observational study done with purposeful sampling technique, all the children who satisfied the inclusion criteria during the period of the study (January-December 2020) constituted the sample size.

RESULTS

Table 1 shows the patients' demographics. 138 (52%) males and 126 (48%) females took part in the study. Patients in the 2-6 age range were 180 (68%) while those in the 7-11 age range were 32%.

Table 2 shows the prevalence of PUD. Of the 264 patients that took part in the study, 42 (16%) had PUD, while 222 (84%) did not.

Table 3 shows the age distribution of PUD. From the table, it is seen that 18 (7%) aged 2-6 years had PUD, while 24 (9%) aged 7-11 also had it. This shows that the prevalence of PUD in the study participants increased with age.

The sex distribution of PUD is shown in table 4. From the table it is evident that 20 (7.6%) males and 22 (8.3%) females had PUD. The table shows a slight male to female variation in the distribution of PUD.

Table 5 displays the association between sex and PUD. As shown in the table, the association between males and females in the prevalence of PUD was not significant (p=0.62). This shows that there was not a significant difference between males and females in the prevalence of PUD.

Table 1: Patients demographics (n=264).

Patient demographics	Number (%)	Total
Sex		
Male	138 (52)	264
Female	126 (48)	
Age (years)		
2-6	180 (68)	264
7-11	84 (32)	

Table 2: Prevalence of PUD.

Total no. of patients	No with PUD (%)
264	42 (16%)

Table 3: Prevalence of PUD according to age.

Age (years)	Prevalence
	N (%)
2-6	18/264 (7)
7-11	24264 (9)

Table 4: Prevalence of PUD according to sex.

Sex	Prevalence
	N (%)
Male	22/264 (8.3)
Female	20/264 (7.6)

Table 5: Association between sex and PUD.

Sex	+PUD	-PUD	X^2 P
Male	20	118	0.24
Female	22	104	0.62
Total	42	222	

+ PUD= with PUD, -PUD= with.

Table 6: Association between age and PUD.

Age (years)	+PUD	-PUD	$\chi 2$ P
2-6	18	162	13.41; 0.0003
7-11	24	60	
Total	42	222	

+ PUD= with PUD, -PUD= without PUD.

The association between age and PUD is shown in table 6. As seen in the table, the association was significant (p=0.0003). This indicated that the prevalence of PUD increased with age among the study participants.

DISCUSSION

Prevalence studies on PUD in developing countries, including Nigeria are scarce. This notwithstanding, peptic ulcers are now being increasingly recognized in children in the developing world. The present study found a prevalence of 16% among the study participants. This finding was very close to what had been reported for the USA (17.4%), but considerably higher than the figure for Europe (8.1%).[21] Although a Nigerian study had found a prevalence of 40.7% for *H. pylori* among children aged 6-10 years in a teaching hospital, it had been shown by some studies that only about 20% of those infected by *H. pylori* eventually develop PUD. Aside from the factors which are already known to affect the prevalence of PUD (age, the household size, lower socio-economic status and positive family history in first degree relatives), the relatively high prevalence of PUD found in this study when compared to the European figure, could be attributed to the poor feeding habits of these children at school. Hunger associated with unsupervised feeding of pupils by teachers during extension of classes beyond the normal time was suspected to tilt the equilibrium between the aggressive and defensive factors towards the side of aggressive factors. Hunger was thought to cause excessive production of hydrochloric acid which gradually destroyed the protective lining of the duodenum and the stomach, with the resultant formation of peptic ulcers. Furthermore hunger/malnutrition can contribute to depressed immunity which will predispose the children to more infection by *H. pylori*. Poor sanitation which promoted the transmission of *H. pylori* could also account for the relatively high prevalence when compared with the prevalence in Europe. Developing countries have been shown to account for greater burden of poor sanitation in the world. And children, especially in the school age, are known to have poorer personal hygiene habits (when compared to adults) because of the lack of oversight role of parents at school.

Studies have shown that sex is one of the factors which could affect the prevalence of PUD. A past study had reported a male-to-female ratio of 1.5:1 for all childhood PUD.[22] However, no such a ratio was demonstrated by the present study. There was no significant difference in the prevalence of PUD between male and female children (p=0.62). On the contrary, the female participants of this study had slightly higher prevalence (8.3%) when compared to their male counterparts (7.6%). The reason for this reversal is not known. However, difference in physiology of the female and male child could be a factor that could predispose the female child more to infection by *H. pylori* than the male counterpart.

Besides sex, age is another factor which can affect the prevalence of PUD. Some past studies have found a positive correlation between age and incidence of PUD.[6,23] Thus, as age increased, the incidence of PUD also increased. Similarly, the present study has found a positive correlation between ages of the children and the prevalence of PUD. There was a significant difference in the prevalence of PUD between the two age categories (2-6 and 7-11) (p=0.0003). This finding has lent credence to what had been reported by other researchers who demonstrated a rising incidence of PUD with age in their own studies.[6,23] The present study was also in tandem with a longitudinal follow-up which showed that prevalence of PUD increased, especially when children were less than 10 years of age.[7] This increasing incidence of PUD had been explained by the observed increasing prevalence of *H. pylori* infection with age which peaked first at 25 years and then at 64 years of age.[23]

Limitations of the study

Inability of the study to eliminate all the confounding factors such abstinence from the use of antibiotics, antacids, and ulcer drugs for two weeks before the test. The inconveniences of collecting stool samples from very young children who may not be able to produce stools when needed.

CONCLUSION

The study found a PUD prevalence of 16% among the school children, which was slightly higher for the female children (8.3%), compared to their male counterparts (7.6%). However, the difference between the two sexes was not significant statistically ((p=0.62). Rise in the prevalence of PUD with age was clearly demonstrated in the two age groups (9% in the 7-11 years age group compared to 7% in the 2-6 years age group). The difference between the two age groups was very significant statistically (p=0.0003). In view of the findings of the study, there is need for the government, through the Ministry of Education, to tackle the issue of undue prolongation of school time in order to protect the pupils from the ulcerogenic action of hunger brought about by inadequate feeding of these children resulting from inadequate supervision of their feeding habits by teachers. Furthermore, to reduce the rate of transmission of *H. pylori* among the pupils, their personal hygiene and provision of potable water for them need to be improved. This can be achieved through health education and health promotion activities which can be taught in the school.

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