Curing Peptic Ulcer Disease (PUD) with One Month Mono-therapy Course of Very Dilute Food-grade Hydrogen Peroxide: Fourteen Case Reports

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Summary

Peptic ulcer disease (PUD) is a global public health challenge. Untreated, it could be complicated by gastric or duodenal perforation, bleeding and gastric cancer. A major risk factor discovered is *Helicobacter* pylori (*H.* pylori) infection, for which reason antibiotics have been added to the treatment regime. Regrettably, the currently recommended triple or quadruple treatment regiments for the disease are becoming ineffective, on account of emergence of new strains of *H.* pylori resistant to

antibiotics used. Against this background, we present an initial proof of principle of efficacy and safety experimental study involving the administration of low-dose food-grade hydrogen peroxide (FGHP) to fourteen (14) consenting adult patients with up to thirty nine years history of PUD unresponsive to conventional therapies. *H.* pylori test was positive in ten (10) of these patients before therapy. The other four (4) in the study had not tested for *H.* pylori.

Each patient was placed on a mono-therapy of 40 ml of 0.5% food-grade hydrogen peroxide (FGHP) three times daily for four weeks. All had relief from clinical symptoms of PUD by the end of four weeks. Five (5) of the 10 patients who tested positive for *H*. pylori before the study, repeated the test after treatment; all were negative. Aside a report of brief nausea experienced by one patient, no adverse reports to treatment were reported. None of the treated patients has had a relapse or had been on conventional PUD therapy since FGHP mono-therapy.

These clinical outcomes suggest efficacy and safety of low-dose FGHP mono-therapy in managing PUD.

Background:

Peptic ulcer disease (PUD) is characterized by mucosal damage of the gastro-duodenum greater than 3-5mm, extending to the submucosa and even to the muscular layers. There are two main types of this disease, a gastric ulcer that occurs in the stomach, and a duodenal ulcer that occurs in the proximal duodenum (the first part of the small intestine). PUD constitutes a major global public health challenge. It had been estimated that 8.1 million people had PUD globally in 2019. This disease can be complicated by bleeding, perforation, or by acute obstruction. Nearly 6 million disability-adjusted-life years (DALYs) were attributable to PUD, with an agestandardized rate of 74.40 DALYs per 100,000 population in 2019.

Among the 21 global burden of disease (GBD) regions in the world, Oceania (7.2) and Western sub-Saharan Africa (6.7) had the highest age-standardized mortality rate of PUD per 100,000 population.³ Our country, Ghana, situated in Western sub-Saharan Africa, is in the region with the second highest age-standardized mortality from PUD.⁴

The discovery of H. pylori as an aetiologic factor in PUD by Marshall JR and Warren BJ 5 , was a major breakthrough in medical science. Chronic inflammation is initiated when this bacterium invades the mucosa of the stomach or the proximal duodenum. 6 When the protective mucosal layer is damaged, the acidic environment of the stomach hastens the formation of the ulcer. Most H. pylori infections are asymptomatic, but some experience a spectrum of outcomes, ranging from benign conditions such as dyspepsia, gastritis and peptic ulcers, to malignant lesions of gastric cancer and mucosa-associated lymphoid tissue (MALT) lymphoma. 7,8,9

H. pylori prevalence is relatively high in Africa, being 70.1% averagely, with 87.7% in Nigeria, the highest rate in the world. In a study of ulcers at the Korle Bu Teaching Hospital in Ghana, *H.* pylori were linked to 82% of gastric ulcers, to 88% of duodenal ulcers, and to 65% of gastric cancer.

The discovery of H. pylori association with PUD also revolutionized the treatment of this otherwise highly debilitating disease, with the addition of antibiotics to the therapeutic regime.⁵ Currently, effective treatment requires a quadruple therapy, consisting of a proton pump inhibitor, acid reducing bismuth, tetracycline or amoxicillin, and the addition of metronidazole for 14 days,¹¹ with associated side effects. Rapid development of antibiotic resistance to H. pylori, however, is now posing a major challenge to the management of PUD.^{12,13} There is, therefore, an urgent need for a more effective treatment regimen that is inexpensive and can readily cure PUD with little or no adverse effects, and without the risk of becoming ineffective in managing resistant strains of H. pylori.

The thesis of this current study rests on hydrogen peroxide (HP) which immune cells naturally produce to rid the body of invading pathogens such as fungi, bacteria, and viruses. These pathogens are predisposed to the Fenton reaction because of their richiron content; HP reacts with the ferrous ion in the pathogen to form the powerful oxidizing free hydroxyl radical to destroy the pathogen. In recently published articles on low-dose FGHP treatment of onychomycosis, it was established that increasing molecules of HP through orally administered low-doses of FGHP (0.5 and 1%), aids in breaching the keratin barrier of nails to reach and destroy fungi that have defied anti-mycotic agents for up to 52 years.

We present a report on 14 patients to show that low-dose of FGHP alone as monotherapy is sufficient, effective and safe for clinical cure of symptomatic PUD. All participants in the study volunteered and consented to taking the diluted FGHP after being informed of its risks and benefits. None of these patients was on any conventional ulcer medication at the time of the therapy. The studies complied with the Helsinki Declaration on Human Experimentation 2013, updated in 2024.

FOURTEEN CASE REPORTS OF 0.5% FGHP CLINICALLY CURING PUD

Case Report 1:

In July 2022, we met a 94-year-old lady, a close friend and wife of one of our professors of medicine in Ghana. She had been in agony from diagnosed peptic ulceration for over 20 years. Characteristically, she experienced epigastric pain sometimes before a meal, and often 15 to 20 minutes after a meal. That pain also woke her up in the early hours of the morning - between 1 am and 2 am. Stool *H.* pylori tests had been positive.

We shared with her the possible effect of low-dose FGHP on peptic ulcer and how that might help cure her. We also informed her concerning the possible side effects of this medication. She voluntarily agreed to try it. As we had 0.5% FGHP solution on

hand, we administered the first dose to her on an empty stomach, alerting her that she might experience epigastric pain soon after. True to that prediction, she experienced severe pain 20 minutes after. A glass of water drank to dilute further the FGHP solution brought relief and the pain subsided. We asked her to continue with the 40 ml of the 0.5% FGHP three times daily, each time on an empty stomach, drinking water to relieve pain when it occurred. By the fourth day, her daughter informed us that the pain experienced soon after ingesting the FGHP was subsiding. Generally, she felt better; the pain no longer woke her up in the early hours of the morning. By the beginning of the second week, the pain was almost gone. From the third week, the pain disappeared completely. The therapy ended immediately after the fourth week. Stool *H.* pylori test was repeated in the fifth week and it was negative.

This patient has not had any pain associated with food since; she has also not been on any PUD medication after the FGHP therapy; she has been free of symptoms of PUD to date. In the four-week period of the FGHP mono-therapy, she did not experience nausea or vomiting.

Case Report 2:

In 2020, a 44-year lady with history of extreme social stress and COVID-19, was diagnosed with peptic ulcer, confirmed by a positive stool H. pylori test and endoscopy. She had been treated with antibiotics and antacids at different hospitals without cure. From time-to-time, classic symptoms of peptic ulcer including epigastric pain associated with meals and which interrupted her sleep in the early hours of the morning, reappeared. In January 2024, she met a member of the peroxide research team who shared with her the benefits and risks of using low-dose foodgrade hydrogen peroxide and our observations on its intervention in a few other patients with peptic ulcer. She readily consented to trying it. Stool H. pylori test was repeated and it was positive. She was placed on 40 ml of 0.5% FGHP three times daily for one month, ingesting it on an empty stomach each time. Within twenty minutes after the first dose, she reported severe epigastric pain, as she had been told to expect. A glass of water helped. By the end of the first week of therapy, the pain had subsided. A week later, the epigastric pain was gone. She continued the treatment till the end of the fourth week. Stool H. pylori test done in the fifth week was negative. She has been free of epigastric pain and other PUD symptoms ever since. She did not experience nausea or vomiting over the period of the study.

Case Report 3:

A 66-year-old woman and close associate of a member of the hydrogen peroxide research team, had struggled with peptic ulcer since 1984. In March 2023, she called to seek advice on a prescription for Nexium she had for the disease at a private hospital. Two gastric endoscopies and H. pylori tests had been done to confirm PUD. Just before the Nexium prescription, two stool H-pylori tests had yielded different results within a week, the first being positive and the second negative. Previous treatment, mainly with omeprazole, had not provided relief. The mid-night epigastric pain had become unbearable and distressing. She readily agreed to try the low-dose

food-grade hydrogen peroxide after its benefits and possible side effects had been explained to her. Within one month, she was completely PUD-symptom-free, and there were no side effects. She re-tested for *H*. pylori in May 2023; it was negative. For the first time since 1984, she has been free of epigastric pain and other PUD symptoms and has remained symptom free for over two years.

Case Report 4:

A 76-year-old woman who had had mild ulcer symptoms for about five (5) years and was being managed at a private clinic for *H*. pylori-*confirmed* PUD, offered to try the FGHP therapy, as she had not had permanent cure for the disease. After four weeks of low-dose FGHP therapy in August 2023, all symptoms she had experienced from the PUD disappeared. She did not complain of any side effects of the therapy, and there has been no relapse since.

Case Report 5:

A 43-year-old woman with 30-year history of peptic ulceration, was placed on the low-dose FGHP therapy in April 2023 when she volunteered to try it after the benefits and risks had been explained to her. *H.* pylori test was positive before treatment and negative after. Severe epigastric pain experienced after the first ingestions of FGHP disappeared within a week. She had no adverse effect from the treatment. To date, she has not had a relapse of the peptic ulcer.

Case Report 6:

A wife of a medical doctor friend overheard discussions on FGHP and its benefits in managing various diseases including peptic ulcer at her 80th birthday celebration in January, 2025. Excited about that, she asked to try the therapy, as she had not had permanent relief from conventional treatment for *H*. pylori-confirmed PUD she had had for about five years. Her ulcer symptoms had generally been mild, though, taking antacids and regulating her diet to ameliorate them. *H*. pylori positive test was established again before therapy. After one week, the ulcer symptoms disappeared. She did not experience nausea or vomiting taking the FGHP solution. Repeat stool *H*-pylori test in the fifth week was negative. She has been well since.

Case Report 7: 37-year-old man with 2-year history of peptic ulcer (with *H*. pylori positivity), unresolved with courses of antibiotics and other medications for the disease at a faith-based hospital in Ghana, voluntarily offered to try the FGHP therapy in August 2025. His brother, who knows about the hydrogen peroxide study, asked him to try the HP remedy. The treatment protocol and possible adverse effects were shared with him. After the first dose, he called to complain of severe epigastric pain as he had been asked to expect. That resolved with a glass of water. The pain diminished and disappeared by the end of the second week. The patient was encouraged to continue the therapy till the end of the fourth week. The patient did not go for stool *H*. pylori test after treatment. There was no nausea or other symptoms related to the therapy.

Case Reports 8-14

Seven (7) other patients, three (3) males and four (4) females, with peptic ulcer also volunteered to try the FGHP therapy. Apart from two (both female) aged 75 and 86, the rest were in their forties. All of them had had the disease for more than three (3) years. Three (3) of them had confirmatory H. pylori positive stool tests before therapy but did not repeat the test after the treatment. Four (4) were treated based only on clinical history of unresolved peptic ulcer. All of them were cured clinically of their PUD, after low-dose FGHP had been administered for four weeks. One of them had brief nausea at the beginning of the FGHP therapy.

Discussion:

Fourteen patients with chronic peptic ulcer disease (PUD) were clinically cured of symptoms of the disease, after ingesting 40 ml of low-dose (0.5%) food-grade hydrogen peroxide (FGHP) three times daily for four weeks. They included ten (10) patients with stool H. pylori positive test before therapy, two of whom also had endoscopy confirmation. Of these ten (10) patients, five (5) re-tested for H. pylori after therapy and were negative. The rest did not follow with laboratory tests to confirm elimination of H. pylori.

Interestingly, the clinical sequence of response to FGHP treatment in each of the 14 patients was the same. Each progressed from epigastric pain after the first doses of FGHP, to relief from pain by the second week, and to clinical cure by the fourth week. Also, aside a brief nausea experienced by one patient, there were no reports of adverse effects from ingesting the low-dose FGHP. Again, although the study period has been relatively short, covering three (3) years, there has been no report of relapse of the disease in all the 14 patients placed on the FGHP mono-therapy. It is to be noted that before this low-dose FGHP mono-therapy, some of these patients had received conventional PUD treatment, including antibiotics, histamine H2 receptor antagonists, and antacids variously without permanent relief. This present study, therefore, shows that chronic PUD can be managed with low-dose FGHP only.

That all these patients did not have permanent relief from conventional and herbal therapies before low-dose FGHP therapy was administered is not surprising. *H.* pylori which are the most common bacteria in the human body, spreading from the oral cavity to the gastro-intestinal tract, ¹⁷ are becoming resistant to the commonly used antibiotics for peptic ulcers. ^{9,11,12} In the study in Ghana, no *H.* pylori sensitivity was recorded for metronidazole, amoxicillin, clarithromycin, and amoxicillin-clavulanic acid against the gastric antrum isolates of *H.* pylori; resistance to levofloxacin was 40%, and 20% for either tetracycline or ciprofloxacin. ¹⁰ Unfortunately, it appears we are losing the battle against *H.* pylori as the frequent mutations in their genes encoding the target proteins of antibiotics continue unabated, ¹⁸ thus highlighting the importance of the present case reports.

That all 14 patients have remained free of ulcer symptoms, some even after 3 years, also suggests that this mono-therapy low-dose (0.5%) FGHP directly inactivates H.

pylori in ulcers, including those with antibiotic-resistant genes. Most likely, deeply buried H. pylori pathogens are also indirectly destroyed through the Fenton reaction.

Limitations of this study are the lack of endoscopy investigations in all 14 subjects to directly confirm ulceration and healing before and after the FGHP therapy, and the inability to get all patients also to do the *H*. pylori test before and after treatment with FGHP. Further studies which address these limitations will improve our knowledge on FGHP and its public health role in the prevention and treatment of PUD.

In conclusion, evidence is provided that low-dose FGHP mono-therapy for four weeks is effective and safe for clinical cure of PUD that had not responded adequately to conventional modes of treatment. Also, though inconclusive, the study reveals that all H. pylori present in the ulcer, including the gene-modified ones resistant to antibiotics used in the triple and quadruple therapy for PUD, were inactivated by low-dose FGHP. The findings of this study suggest that relatively inexpensive low-dose FGHP alone can cure PUD within one month with little or no adverse effects. This is of great public health importance, especially for patients in the poorest regions of the world, including Western sub-Saharan Africa which also has a high H. pylori burden.

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