MAK HILL Publications Research Journal of Medical Sciences

Research Article
doi: 10.59218/makrjms.2023.662.665

Prevalence and Treatment of Acid Peptic Disease in Patients with Sickle Cell Disease a Hospital Based Cross Sectional Study

[1]Neha Banseria, [2]Amit Jain, [3]Manish Rathor and [4]Hemant Kumar Jain

[1]Department of Pathology, Government Medical College, Ratlam, Madhya Pradesh, India

[2]Department of ENT, Gajra Raja Medical College, Gwalior, Madhya Pradesh, India

[3]Department of Pediatrics, Government Medical College, Ratlam, Madhya Pradesh, India

[4]Department of General Medicine, Government Medical College, Datia, Madhya

ABSTRACT

Sickle cell disease (SCD) is a chronic hemolytic disorder characterized by the presence of crescent-shaped red blood cells. In adults with SCD, abdominal pain is a common presenting symptom. Among patients not in crisis, it is estimated that one-third of individuals with homozygous SCD and chronic recurrent epigastric pain exhibit endoscopic evidence of peptic ulcer disease. This study aimed to analyze the incidence and management of acid peptic disorders among SCD patients in Central India. A prospective study was conducted, involving 68 homozygous SCD patients. All patients underwent upper gastrointestinal endoscopy (UGIE) with gastric antral biopsy and rapid urease test (RUT). Cases with Helicobacter pylori were administered a 14 days course of a three-drug regimen of anti H. pylori drugs and were followed up for 6 weeks from the initiation of treatment with repeat UGIE and RUT. Out of the 68 cases, 14 cases (20.59%) were diagnosed with duodenal ulcer, which was significantly higher than the number of patients with gastric ulcer (11.76%). Other findings included gastroesophageal reflux disease and gastritis. Majority of patients tested negative for RUT after receiving anti H. pylori medication. SCD patients with acid peptic disorders form a considerable proportion of patients attending the surgical outpatient department and indoor department of surgery. Conservative treatment with lifestyle modification can effectively improve the symptoms of acid peptic disease.

OPEN ACCESS

Key Words

Peptic ulcer, helicobacter pylori, sickle cell anemia, gastroesophageal reflux

Corresponding Author

Hemant Kumar Jain,
Department of General Medicine, Government Medical College, Datia, Madhya, India

Received: 3 July 2023
Accepted: 28 July 2023
Published: 2 August 2023

Citation: Neha Banseria , Amit Jain, Manish Rathor and Hemant Kumar Jain, 2023. Prevalence and treatment of acid peptic disease in patients with sickle cell disease a hospital based cross sectional study. Res. J. Med. Sci., 17: 662-665, doi: 10.59218/makrjms.2023.662.665

INTRODUCTION

Sickle cell disease (SCD) stands as the most prevalent hereditary hematological disorder affecting the human population[1,2]. Upon deoxygenation, red blood cells containing sickle hemoglobin undergo intracellular polymerization, adopting a sickle shape. This alteration renders these cells more fragile and susceptible to hemolysis[1]. Additionally, the deformed cells contribute to microvasculature occlusion, leading to ischemia in various organs and resulting in distinct clinical manifestations.

Globally, sickle cell disorders constitute approximately 70% of all hemoglobin disorders[3]. In Western Odisha, India, SCD holds a high prevalence across all castes and communities. Notably, in 1965, Nanda *et al*.[4] reported the occurrence of sickle cell hemoglobinopathy in the state of Odisha. Subsequently, Kar *et al*.[5] conducted a comprehensive study on the distribution and prevalence of SCD among individuals of different castes in Western Odisha. They identified a gene frequency of about 15% in the general population and noted its prevalence in specific cases.

In contrast, acid peptic disease is characterized as a condition affecting the gastric and duodenal mucosal barrier due to either excessive or insufficient secretion of acid and pepsin into the gastric juice. This process leads to the detrimental erosion of the mucosal and muscular layers of the stomach and duodenum[6]. Acid peptic disease encompasses a range of conditions, such as gastroesophageal reflux disease (GERD), gastritis, gastric ulcer, duodenal ulcer, esophageal ulcer, Zollinger Ellison syndrome and Meckel's diverticulitis.

Among the various clinical manifestations of SCD, abdominal pain frequently presents as a common symptom in adults. Potential underlying etiologies for this symptom encompass acid peptic disease, splenic sequestration, acute chest syndrome, ischemic colitis, hepatobiliary pathology, acute pancreatitis or appendicitis. Studies on the incidence of acid peptic disorders in SCD patients have been undertaken, yielding diverse outcomes[7-9]. It is crucial to note that most of these investigations were conducted before the 1990 and predominantly in Western countries. Considering the limited literature available on acid peptic disorders in SCD patients from the Indian population, the current study was devised to explore the incidence of acid peptic disorders among SCD patients in Central India and to propose a possible management protocol for such cases.

MATERIALS AND METHODS

This prospective analytical study was conducted at the Department of Surgery in a tertiary healthcare facility in Central India. The study included patients with SCD of both sexes and all age groups, who presented with dyspepsia and recurrent pain in the upper abdomen. Patients in vaso-occlusive crisis and those on anti-secretory or antibiotic drugs within the preceding 4 weeks were excluded. Additionally, patients with peptic perforation and complicated cases requiring emergency surgery without prior endoscopy were also not considered for the study.

Diagnosis of SCD was confirmed through sickling slide test followed by Variant-II hemoglobin testing system as per the manufacturer's instructions. After confirming the diagnosis, thorough clinical history, clinical examination and relevant investigations were conducted for all patients. UGIE was performed on all recruited patients and gastric antral biopsy specimens were collected from each patient within 2-3 cm from the pylorus. The collected specimens underwent rapid urease test (RUT).

Patients were counseled to adopt lifestyle changes, reduce the use of nonsteroidal anti-inflammatory drugs (NSAIDs) and avoid smoking. Treatment with H_2 receptor antagonists (H2RAs), proton pump inhibitors (PPIs) and antacids was administered either individually or in combination based on the severity of symptoms. Patients with dyspepsia associated with Helicobacter pylori (RUT positive) were given a 14 days course of a three-drug regimen of anti-H. pylori drugs. Hospitalization was considered for some patients to closely monitor their condition and manage associated symptoms. Follow-up was scheduled after 6 weeks of treatment initiation, during which repeat upper GI endoscopy and RUT were performed. Patients with persistent pathological features or no improvement in symptoms were considered for surgical interventions.

Statistical analysis was performed by recording all findings in individual case sheets and entering them into a pre-designed Excel sheet for Windows. The data were presented in terms of numbers, percentages, means and standard deviations. Categorical data were analyzed using the Chi-square test and all statistical analyses were conducted using SPSS v19.

RESULTS

The hospital records were thoroughly reviewed, revealing a total of 357 SCD patients attending the surgery OPD, representing 4.1% of the patient population. Among these SCD patients, 19.04% (68 out of 357) reported gastroduodenal symptoms. The most commonly reported symptom was recurrent pain in the abdome, followed by chest pain. Acid peptic disorders were more prevalent in male patients with a male-to-female ratio of 2:1. Majority of patients belonged to the low socioeconomic status.

In dyspeptic patients who underwent UGIE, the findings were normal in 50% of cases. Among, the

Table 1: Endoscopic findings in SCD patients

UGIE finding	No.	Percentage (%)
Normal	33	50.00
Duodenal ulcer	14	20.59
Gastric ulcer	9	13.23
Gastroesophageal reflux disease (GERD)	8	11.76
Gastritis	4	5.88
Total	68	100.00

Table 2: Age wise UGI endoscopic findings in SCD patients

	Age group (years)						
UGIE findings	11-20	21-30	31-40	41-50	51-60	61-70	71-80
Normal	4	7	11	5	4	2	0
Duodenal ulcer	1	4	3	3	2	1	0
Gastric ulcer	0	3	3	1	1	0	1
Gastroesophageal reflux disease (GERD)	0	1	1	3	1	2	0
Gastritis	0	1	1	2	0	0	0
Total	5	16	19	14	8	5	1

Table 3: Ulcer healing rate (%) after management by UGI ndoscopy

UGIE finding	No. of patients	Healing (n)	Healing rate (%)
Normal	33	-	-
Duodenal ulcer	14	10	71.43
Gastric ulcer	9	6	66.67
Gastroesophageal reflux disease (GERD)	8	8	100.00
Gastritis	4	4	100.00

pathological findings observed, duodenal ulcer was the most prevalent, which was significantly higher than gastric ulcer. Furthermore, 11.76% of the dyspeptic patients exhibited symptoms consistent with gastroesophageal reflux disease (GERD), while 5.88% showed signs of gastritis Table 1. Age wise UGI Endoscopic findings in SCD patients are shown in Table 2.

In the 21-30 year age group, the majority of patients (88.8%) in our study were found to have H. pylori infection. However, there was a gradual decrease in the trend of H. pylori positivity as the age groups increased. Approximately, 26.1% of patients in the study had a habit of tobacco consumption, with a significant proportion of duodenal ulcer (DU) patients (36.8%) reporting a history of tobacco addiction. Additionally, around 53.4% of patients had a habit of using nonsteroidal anti-inflammatory drugs (NSAIDs) to manage their painful episodes. Following medical management and lifestyle modifications, all patients with heartburn and reflux symptoms showed improvement. On repeat UGIE, it was observed that all patients with gastritis and GERD had no endoscopic pathology. About 71% of patients with DU showed healing with conservative treatment. The overall healing rate among all patients was approximately 90%. However, despite conservative treatment, about 15% of patients with recurrent abdominal pain remained symptomatic Table 3.

DISCUSSIONS

In this study, 68 patients with SCD presented with gastroduodenal symptoms, with a majority of them being male. UGIE findings revealed various pathologies like duodenal ulcer, gastric ulcer, GERD and gastritis. These endoscopic observations align with previous studies, such as that by Lee *et al*.[9] who found approximately one third of SCD patients with chronic recurrent epigastric pain to have endoscopic evidence of peptic ulcer, with a higher prevalence of duodenal ulcers over gastric ulcers. Serjeant *et al*.[8] also reported a prevalence of 5% of duodenal ulcers in SCD patients over 25 years of age with the SS genotype.

SCD patients are more susceptible to avascular damage in various parts of the body and ischemia may play a role in their susceptibility to peptic ulcers. Notably, a study by Worsornu and-Ahulu[7] found that gastric acid responses in SCD patients were normal, indicating that the pathogenesis of ulcers in SCD might differ from the general population. The use of nonsteroidal anti-inflammatory drugs (NSAIDs) by SCD patients during pain crises poses a significant health problem, as NSAID-induced gastropathy is prevalent. NSAIDs reduce gastric mucosal secretion, creating a suitable environment for H. pylori colonization[10]. Helicobacter pylori infection was found in some SCD patients with recurrent upper abdominal pain and treatment with Helicobacter pylori eradication led to symptom resolution[11].

Patients diagnosed with gastritis and GERD in this study did not show any endoscopic pathology. Approximately, 71% of patients with duodenal ulcers healed with conservative treatment, resulting in an overall healing rate of about 90%. The NICE guidelines indicate the effectiveness of various interventions for duodenal and gastric ulcers and emphasize the importance of combining acid suppression with eradication therapy to improve healing rates and reduce relapses[12].

In patients taking NSAIDs, H. pylori eradication did not improve the ulcer healing rate but it did decrease the number of endoscopically proven ulcers at 6 months post-treatment.

CONCLUSION

Among the acid peptic disorders diagnosed in this study, a significant majority of patients were found to have duodenal ulcer. While plausible associations have

been established between H. pylori infection, stress, NSAID usage and smoking habits with the causation of acid peptic disorders, it is important to consider the potential role of altered local mucosal hemodynamics in steady-state SCD patients as well. This factor cannot be discounted as a contributing factor in the development of such disorders. In-depth long-term follow-up studies are imperative to accurately assess the outcomes of managing acid peptic disorders in SCD patients, both during sickling crisis and in steady state conditions. Separate evaluation of these two distinct phases will help to better understand the disease course and the efficacy of management strategies in addressing acid peptic disorders in SCD patients.

REFERENCES

1. Bishi, P., B. Mishra, P. Singh, J. Das and B. Rath, 2022. Incidence and management of acid peptic disorders in sickle cell disease patients in western odisha. Nat. J. Physiol. Pharm. Pharmacol., 13: 350-353.
2. Hahn, E.V., 1927. Sickle cell anemia. Report of a case greatly improved by splenectomy. Experimental study of sickle cell formation. JAMA Internal Med., 39: 233-254.
3. Angastiniotis, M., B. Modell, P. Englezos and V. Boulyjenkov, 1995. Prevention and control of haemoglobinopathies. Bull World Health Organ., 73: 375-386.
4. Nanda, B.K., G.K. Panda, U.P. Naik and C.N. Nanda, 1965. Abstracts book. In: 15th Annual Meeting, Nanda, B.K., G.K. Panda, U.P. Naik and C.N. Nanda, (Eds.)., Indian Association of Pathologists, India, pp: 37.
5. Kar, B.C., A.E. Kulozik, S. Sirr, R.K. Satapathy, M. Kulozik, B.E. Serjeant and G.R. Serjeant, 1986. Sickle cell disease in Orissa state, India. Lancet, 2: 1198-1201.
6. Tortora, G.J. and B. Derrickson, 2006. Peptic Ulcer Disease. In: Principles of Anatomy and Physiology, Tortora, G.J. and B. Derrickson, (Eds.)., Wiley Publication, USA, pp: 942-943.
7. Wosornu, L. and F.I.D.K. Ahulu, 1971. Gastric acid secretion in sickle-cell anaemia. Gut, 12: 197-199.
8. Serjeant, G.R., H. May, A. Patrick and E.D. Slifer, 1973. Duodenal ulceration in sickle cell anaemia. Trans. Royal Soc. Trop. Med. Hyg., 67: 59-63.
9. Lee, M.G., C.H. Thirumalai, S.I. Terry and G.R. Serjeant, 1989. Endoscopic and gastric acid studies in homozygous sickle cell disease and upper abdominal pain. Gut, 30: 569-572.
10. Gisbert, J.P., J. Legido, I.G. Sanz and J.M. Pajares, 2004. Helicobacter pylori and perforated peptic ulcer prevalence of the infection and role of non-steroidal anti-inflammatory drugs. Dig. Liver Dis., 36: 116-120.
11. Woods, K.F., A. Onuoha, R.R. Schade and A. Kutlar, 2000. Helicobacter pylori infection in sickle cell disease. J. Natl. Med. Assoc., 92: 361-36.5
12. Scottish Intercollegiate Guidelines Network 2008. Management of acute upper and lower gastrointestinal bleeding: A national clinical guideline., https://heeoe.hee.nhs.uk/sites/default/files/upper_and_lower_gi_bleeding_full_sign_2008_0.pdf