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Acid Peptic Disorders in Sickle Cell Disease: Incidence and Management Strategies

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Abstract
Sickle cell disease (SCD) is a chronic hemolytic condition marked by the presence of crescent-shaped red blood cells. Abdominal pain is a frequent presenting symptom in adults with SCD. Among non-crisis patients, it is estimated that one-third of individuals with homozygous SCD who experience 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. A prospective study was conducted involving 108 homozygous SCD patients. Each patient underwent upper gastrointestinal endoscopy (UGIE) with gastric antral biopsy and a rapid urease test (RUT). Patients with Helicobacter pylori infection were treated with a 14-day regimen of triple therapy for H. pylori and were re-evaluated six weeks after starting treatment with repeat UGIE and RUT. Among the SCD cases, patients had duodenal ulcers, a significantly higher incidence compared to gastric ulcers. Additional findings included gastroesophageal reflux disease and gastritis. Approximately 94% of patients tested negative for H. pylori on the RUT after completing the medication regimen. A substantial proportion of SCD patients presenting to surgical outpatient and inpatient departments suffer from acid peptic disorders. Conservative treatment combined with lifestyle modifications can effectively alleviate the symptoms of acid peptic disease.

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Key Words
Sickle cell disease, acid peptic disorder, helicobacter pylori, management

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INTRODUCTION

Sickle cell disease (SCD) is the most prevalent hereditary hematological condition affecting humans. When red blood cells contain sickle hemoglobin, they assume a sickle shape due to intracellular hemoglobin polymerization upon deoxygenation. These sickle-shaped cells are more fragile and prone to hemolysis. Additionally, they obstruct microvasculature, leading to ischemia in various organs and resulting in diverse clinical symptoms. Worldwide, sickle cell disorders represent approximately 70% of hemoglobin disorders[1,2].

SCD exhibits a high prevalence across social strata and communities in India. In 1965, Nanda *et al*. first reported sickle cell hemoglobinopathy[3]. Subsequently, Kar *et al*. reported on the distribution and prevalence of SCD among different castes. They noted a gene frequency of about 15% in the general population, with certain groups showing a higher prevalence[4].

Acid peptic disease refers to a condition where the gastric and duodenal mucosal barrier is disrupted due to either excessive or reduced secretion of acid and pepsin into the gastric juice, leading to damage to the mucosal and muscular layers of the stomach and duodenum[5]. This encompasses various conditions such as gastro esophageal reflux disease (GERD), gastritis, gastric ulcer, duodenal ulcer, esophageal ulcer, Zollinger-Ellison syndrome and Meckel's diverticulitis.

Among the different clinical manifestations of SCD, abdominal pain is a common presenting symptom in adults. Possible causes include acid peptic disease, splenic sequestration, acute chest syndrome, ischemic colitis, hepato-biliary pathology, acute pancreatitis, or appendicitis. Several studies have explored the incidence of acid peptic disorders in SCD patients, yielding varying results[6-8]. However, most of these studies were conducted in Western countries. Given the limited literature on acid peptic disorders in SCD patients within the Indian population, this current study aims to investigate the prevalence of acid peptic disorders in SCD patients from India and to propose potential management strategies for such patients.

MATERIAL AND METHODS

The study included 108 SCD patients of both sexes and all age groups who presented to the outpatient department (OPD) or were admitted with complaints of dyspepsia and recurrent upper abdominal pain. Patients in vaso-occlusive crisis or those who had taken anti-secretory or antibiotic drugs within the past four weeks were excluded. Additionally, patients with peptic perforation or complicated cases requiring emergency surgery that could not undergo prior endoscopy were not included.

Consent was obtained from all participants and 2 mL of venous blood was collected from each patient for SCD diagnosis. The diagnosis was confirmed using the sickling slide test, followed by the Variant-II hemoglobin testing system, according to the manufacturer's instructions. All diagnosed SCD patients underwent a thorough clinical history, comprehensive clinical examination and relevant investigations, including upper gastrointestinal endoscopy (UGIE). Gastric antral biopsy specimens were collected within 2-3cm from the pylorus and subjected to a rapid urease test (RUT) using a commercial kit.

Patients were initially counseled on lifestyle modifications, including reducing NSAID use and avoiding smoking. Depending on the severity of symptoms, H2 receptor antagonists (H2RAs), proton pump inhibitors (PPIs) and antacids were administered either alone or in combination. Patients with dyspepsia symptoms associated with Helicobacter pylori infection (RUT positive) received a 14-day course of a triple-drug regimen for H. pylori. Some patients were hospitalized for close monitoring and management of other associated symptoms. Follow-up evaluations, including repeat UGIE and RUT, were conducted six weeks after initiating treatment. Patients with persistent pathological features or no symptom improvement were considered for surgical intervention.

All findings were recorded on individual case sheets and entered into a predesigned Excel spreadsheet. Data were presented as numbers, percentages, means and standard deviations. Categorical data were analyzed using the Chi-square test. All statistical analyses were performed using EpiInfo Software.

RESULTS AND DISCUSSIONS

(Table 1) presents the endoscopic findings in SCD cases. Duodenal ulcers were the most common finding

Fig. 1: Age distribution and endoscopic findings in SCD cases

Fig. 2: Endoscopic healing rates after management

Table 1: Endoscopic findings in SCD cases

Endoscopic finding	n	Percentage
Normal	53	49.07
Duodenal Ulcer	23	21.3
Gastric Ulcer	15	13.89
GERD	11	10.19
Gastritis	6	5.56
Total	108	100

followed by Gastric ulcers. Also, cases of GERD and gastritis were found.

(Fig. 1) depicts the age distribution and endoscopic findings in SCD cases. The majority of duodenal ulcers were observed in the 21-30 age group, whereas gastric ulcers were more prevalent in the 31-40 age group.

(Fig. 2) illustrates the endoscopic healing rates after management. GERD and gastritis cases exhibited the highest healing rates, reaching 100%, while duodenal ulcers showed the lowest healing rates at 74%.

In this prospective study, 88 patients diagnosed with Sickle Cell Disease (SCD) experienced gastroduodenal symptoms, with a predominance of male patients. Upon upper gastrointestinal endoscopy (UGIE), duodenal ulcer, gastric ulcer, gastroesophageal reflux disease (GERD) and gastritis were detected. Our endoscopic findings align with those of Lee *et al.,* who noted that about one-third of SCD patients with chronic recurrent epigastric pain had endoscopic evidence of peptic ulcers, with duodenal ulcers (27%) being more common than gastric ulcers (8%)[8]. Serjeant *et al*. reported a 5% prevalence of duodenal ulcers in SCD patients over 25 years old with homozygous sickle cell anemia (SS genotype), with an overall clinic prevalence of 7%, consistent with our observations[7].

SCD patients are particularly susceptible to avascular damage across various body sites, contributing to their vulnerability to peptic ulcers. Worsornu and Konotey-Ahulu investigated gastric acid response in 115 Ghanaian patients with duodenal ulceration, including 45 with concurrent SCD, noting that patients without SCD but with duodenal ulcers secreted significantly more acid than those with SCD. Gastric acid responses in SCD patients were found to be within normal ranges[6].

In the general population, about half of NSAID users experience gastric erosions and 10-30% develop gastric ulcers. Given that our study participants frequently use NSAIDs during pain crises, NSAID-induced gastropathy poses a significant health concern in this population. NSAIDs reduce gastric mucosal secretion by modulating the arachidonic pathway, creating a conducive environment for H. pylori colonization[9].

Woods *et al.* reported eight cases of H. pylori infection in adult SCD patients presenting with recurrent upper abdominal pain, of which three had gastric or duodenal ulcers. Treatment of H. pylori resulted in complete symptom resolution within four weeks. Although H. pylori is prevalent in developing countries' endemic areas, data on its prevalence and eradication in SCD patients are scarce[10].

Patients with gastritis and GERD did not exhibit any endoscopic abnormalities. Approximately 75% of duodenal ulcers healed with conservative treatment, yielding an overall healing rate of 92%. According to NICE guidelines[11,12], acid suppression for 4-8 weeks led to ulcer healing in 70% of patients, with an additional 6.4% improvement with eradication therapy. Relapse rates at 3-12 months were 40% after short-term acid suppression alone, but eradication raised this by 52-91%. While acid suppression supplemented with eradication did not enhance gastric ulcer healing rates, it reduced relapse rates, with 46% ulcer-free at 3-12 months post-treatment solely with acid suppression, increasing to 32-77% with eradication. Eradication did not improve ulcer healing rates in NSAID users but halved the incidence of endoscopically proven ulcers six months later, from 18%-9%[13-15].

CONCLUSION

This study findings indicate that duodenal ulcers are the most common acid peptic disorder in this population. While H. pylori infection, stress, NSAID use, and smoking habits are plausible contributing factors, the role of altered local mucosal hemodynamics cannot be discounted even in patients in a steady state. Long-term follow-up studies are necessary to accurately assess the outcomes of managing acid peptic disorders in SCD patients both during sickling crises and in steady state conditions.

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