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Case Report

Resource-constrained Management of Portal Hypertension: A Case Series Evaluating Alternative Therapies for Gastric Varices

Yusuf Musa[1*], Habib Tijjani Saleh[1], Nuruddeen Muhammad Olalekan[1], Dada Idowu[2], Abubakar Sadiq Aminu[1], Hafiz Abdullahi Zubairu[1] and Adamu Alhaji Sama'ila[3]

[1]*Gastroenterology Unit, Department of Internal Medicine, Federal Teaching Hospital, Katsina, Nigeria;* [2]*Gastroenterology Unit, Department of Internal Medicine, Federal Medical Centre Ebuta Meta, Lagos, Nigeria;* [3]*Gastroenterology Unit, Department of Internal Medicine, Aminu Kano Teaching Hospital, Kano, Nigeria*

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Abstract

Portal hypertension can cause serious complications such as upper gastrointestinal bleeding, primarily due to esophageal and gastric varices. The risk of mortality from variceal hemorrhage is significant, particularly when the hepatic venous pressure gradient exceeds 12 mmHg. Established treatments generally include endoscopic variceal band ligation and cyanoacrylate glue for gastric varices; however, challenges such as limited availability and a lack of technical expertise can hinder the use of glue, leading to preventable complications. This study investigates the efficacy of using a 50% glucose solution for injection sclerotherapy in cases of gastric varices. We present three unique patient cases. The first case involves a 21-year-old with persistent upper gastrointestinal bleeding and a portal vein thrombus, who experienced temporary relief after receiving injection sclerotherapy but tragically succumbed to significant bleeding later. The second case describes a 24-year-old who successfully managed his bleeding with the same treatment but was subsequently lost to follow-up. Lastly, a 72-year-old patient with recurrent painless hematemesis remained free of symptoms following injection sclerotherapy. Overall, while cyanoacrylate glue remains the preferred treatment, injection sclerotherapy with 50% dextrose shows promise as an effective alternative, particularly in settings where conventional treatments are not readily available, potentially reducing the risks associated with untreated variceal bleeding.

Introduction

Portal hypertension is an increase in pressure within the portal venous system, defined as exceeding 5 mmHg, and becomes clinically significant when the pressure gradient surpasses 10 mmHg. This condition leads to the formation of collateral veins, such as gastroesophageal varices, which pose a high risk of gastrointestinal bleeding.[1] Factors contributing to variceal rupture include wall tension, transmural pressure, and the size and thickness of the varices.[2] Portal hypertension and its complications significantly impact morbidity and mortality in patients with chronic liver disease, with esophageal varices affecting up to 60% of individuals with cirrhosis, while gastric varices are less common, occurring in 5–33% of cases.[3–5] The annual incidence of varices is approximately 9%, with a mortality rate of up to 25% following a variceal hemorrhage.[3]

Endoscopic techniques for managing varices have advanced considerably, with methods such as endoscopic variceal ligation (EVL) and endoscopic variceal sclerotherapy (EVS) playing vital roles in treatment. EVS involves injecting a sclerosant into or around the varices to induce thrombosis and fibrosis, thereby reducing blood flow and preventing rupture.[6] This method has proven effective in controlling acute bleeding and reducing recurrence risk, particularly in resource-limited settings where access to interventions, such as transjugular intrahepatic portosystemic shunt, may be limited. Although EVL is often the preferred treatment for esophageal varices, EVS remains essential when EVL is unfeasible or unavailable. Primary indications for EVS include gastric varices associated with esophageal varices and anatomical

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[*]**Correspondence to:** Yusuf Musa, Gastroenterology Unit, Department of Internal Medicine, Federal Teaching Hospital, Katsina 820101, Nigeria. ORCID: https://orcid.org/0000-0001-7283-8310. Tel: +234-8060661115, E-mail: yusuf.musa@npmcn.edu.ng

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challenges that preclude the use of EVL.[1,7]

In sub-Saharan Africa, the incidence of esophageal and gastric varices is significant due to the high prevalence of chronic liver diseases and schistosomiasis. However, practicing endoscopic sclerotherapy faces numerous challenges, including high costs and the limited availability of standard sclerosants, which range from 50,000 to 130,000 Nigerian Naira per session (approximately 30–78 USD as of December 2024).[8] Additional barriers include a shortage of trained specialists, poor access to necessary equipment, and infrastructure limitations such as unreliable electricity and insufficient sterilization facilities.[9,10] Given these constraints, exploring affordable alternatives to conventional sclerosants is essential. One promising option is a 50% dextrose solution, which preliminary studies suggest may induce variceal sclerosis through osmotic injury, making it a potential low-cost sclerosant for use in resource-limited environments.[8,9] This case series aimed to evaluate the safety and efficacy of using 50% dextrose for managing esophagogastric varices, addressing a critical need for accessible treatment options in these regions.

Case presentation

Study design

This case series was conducted at the Federal Teaching Hospital, Katsina, among patients with gastroesophageal varices. Inclusion criteria included patients presenting with a history of upper gastrointestinal bleeding from gastric varices confirmed by endoscopy.

Definition of terms

Esophageal varices classification

Esophageal varices were graded using the Modified Paquet classification as follows[11]:

- Grade 0: No varices present;
- Grade I: Varices extend just above the mucosal level;
- Grade II: Varices project up to one-third of the luminal diameter and cannot be compressed by air insufflation;
- Grade III: Varices project up to 50% of the luminal diameter or are in contact with each other.

Gastric varices classification

Gastric varices were classified using the Sarin classification into[12]:

- Oesophago-gastric varix (GOV) Type 1 or Type 2;
- Isolated gastric varix Type 1 or Type 2.

Signs of bleeding

Signs indicating potential bleeding include the red wale sign and the white nipple sign.[13]

Bleeding risk assessment

The risk of bleeding from varices was categorized as[13]:

- Mild risk;
- Moderate risk;
- High risk.

Intervention protocol

Preparation

In Nigeria, many hemostatic interventions are often unavailable, placing a significant financial burden on patients who must pay out of pocket. Furthermore, if active bleeding is encountered during endoscopy, the necessary equipment to manage and halt bleeding is frequently lacking. As a result, endoscopy is typically performed only when funds are readily available and there is no clinical evidence of ongoing bleeding. Patients generally underwent endoscopy within 24–48 h of presentation, following stabilization with intravenous fluids, vasoactive agents (such as Terlipressin or Octreotide), and blood transfusions.

Technique

Under direct endoscopic visualization, approximately 30–40 mL of 50% dextrose was slowly injected into the gastric varices over 10 m using a 23G sclerotherapy needle. Injections were performed in sessions repeated at two- to four-week intervals, with the goal of achieving complete variceal obliteration.

Follow-up

Patients were monitored for at least 24–48 h post-procedure before discharge. Outcomes were assessed during hospitalization and at outpatient follow-up visits within 30 days.

Case 1

De-identified demographics: A 21-year-old male patient.

Chief complaints

The patient presented with a two-year history of recurrent hematemesis, melena, hematochezia, and a left upper abdominal mass.

Medical, family, and psychosocial history

No family history of liver disease or bleeding disorders; no relevant psychosocial issues reported.

Relevant past interventions and outcomes

The patient had received multiple blood transfusions and was evaluated for tropical splenomegaly syndrome and myelodysplastic syndrome by hematologists, with no significant findings.

Significant examination findings

Physical examination revealed massive splenomegaly without peripheral stigmata of liver cirrhosis.

Hospitalization and diagnostic evaluation

The patient was hospitalized for approximately 70 days and underwent several sessions of endoscopic variceal band ligation (EVBL). Despite these interventions, bleeding persisted, resulting in approximately 30 units of blood being transfused during his two-month stay. He also received multiple doses of Terlipressin.

Diagnostic testing

- Liver function tests were within normal limits;
- Serologic tests for hepatitis B virus (HBV), hepatitis C virus (HCV), and Human immune deficiency virus were negative;
- Abdominal ultrasound showed splenomegaly and portal vein thrombosis;
- Liver elastography indicated F2 fibrosis;
- Upper gastrointestinal endoscopy revealed high-risk esophageal varices (Grade III with stigmata of bleeding), fundal extension: GOV II, and portal hypertensive gastropathy.

Diagnostic challenges

The patient's caregivers faced significant financial constraints, limiting access to basic investigations, including packed cell vol-

ume monitoring and payment of bed fees. The managing team contributed funds for medications and tests, and procedures were performed at no charge. Despite multiple EVBL sessions, upper gastrointestinal (GI) bleeding remained uncontrolled, prompting consideration of dextrose sclerotherapy.

Diagnosis

Non-Cirrhotic Portal Hypertension, likely secondary to Portal Vein Thrombosis.

Intervention

Interventions included:
- Blood transfusions: Over 30 units transfused during hospitalization;
- Medications: Vasopressors (Terlipressin), anticoagulants, and beta-blockers (prescribed at discharge);
- Endoscopic treatments: Multiple sessions of EVBL.

Change in therapeutic intervention

After serial EVBL failed to control bleeding, endoscopic injection sclerotherapy using 50% dextrose was performed. In the first session, 30–40 mL of 50% dextrose was steadily injected into the fundal varices over 20–30 m. A follow-up session was conducted four weeks later, resulting in a six-month bleeding-free period.

Outcome

Unfortunately, the patient was lost to follow-up and later re-presented to the emergency department with massive upper gastrointestinal bleeding. Tragically, he passed away before any intervention could be performed.

Case 2

De-identified demographics: A 24-year-old male student.

Chief complaints

The patient presented in mid-2022 with recurrent painless hematemesis and melena.

Medical, family, and psychosocial history

There was no family history of liver disease or bleeding disorders.

Relevant past interventions and outcomes

The patient exhibited no clinical signs of liver cirrhosis. He had experienced several episodes of bleeding prior to the current presentation and had received approximately five units of blood transfusions.

Significant examination findings

Clinical examination revealed massive splenomegaly with no peripheral stigmata of liver cirrhosis.

Historical and current information

The patient was admitted for approximately two weeks, during which he underwent resuscitation and necessary evaluations.

Diagnostic testing

- Serologies for HBV and HCV were negative;
- Abdominal ultrasound showed a normal-sized liver with uniform echogenicity and massive splenomegaly;
- Liver elastography indicated F2 fibrosis;
- Upper gastrointestinal endoscopy revealed Grade 2 esophageal varices (with stigmata of bleeding), fundal extension GOV II, and portal hypertensive gastropathy.

Diagnostic challenges

Financial constraints caused delays in obtaining basic investigations and endoscopic procedures. Elastography was offered free of charge, but the patient could not afford an abdominal computed tomography scan or liver biopsy.

Diagnosis

Non-Cirrhotic Portal Hypertension, likely secondary to hepatosplenic schistosomiasis.

Interventions given

Types of interventions:
- Blood transfusions: Approximately five units;
- Endoscopic interventions: A previous upper GI endoscopy with EVBL did not improve bleeding.

Changes in therapeutic intervention

The patient underwent injection sclerotherapy with 30–40 mL of 50% glucose solution over 20–30 m, which resolved the gastrointestinal bleeding.

Follow-up

The patient was lost to follow-up in late 2022 and re-presented in June 2024 with severe anemia and melena, but no hematemesis. A repeat upper GI endoscopy revealed similar findings of GOV II varix.

Case 3

De-identified demographics: A 72-year-old retired public servant.

Chief complaints

The patient presented with a two-year history of recurrent painless hematemesis.

Medical, family, and psychosocial history

No family history of liver disease or bleeding disorders.

Relevant past interventions and outcomes

The patient received 4 units of blood transfusions.

Examination findings

No peripheral stigmata of liver cirrhosis or abdominal organ enlargement were observed during examination.

Historical and current information

The patient had experienced recurrent bleeding and was frequently hospitalized, but there had been no thorough evaluation or specialist consultation.

Diagnostic testing

- Serologies for HBV and HCV were negative;
- Abdominal ultrasound and computer tomography scans confirmed a normal liver and pancreas with no ascites;
- Liver elastography indicated F0 (< 5 kPa);
- Upper gastrointestinal endoscopy revealed an isolated gastric varix type I (IGV I) with portal hypertensive gastropathy.

Diagnostic challenges

The patient refused further evaluation, repeat endoscopies, and medications after the initial session.

Table 1. Summary of cases

S/N	Age (Yrs.)	Presentations	Duration	Other investigations	Endoscopy/ interventions	No of sessions	Follow-up/outcome
1	21	Hematemesis, melena, abdominal swelling, repeated transfusions	Two years	Elastography (F2); HBsAg = negative; HCV = negative; abdominal USS = splenomegaly	GOV II: 40 mL of 50% glucose injected into the gastric varix	3	Lost to follow up. Re-presented with massive bleeding and died before intervention
2	24	Recurrent hematemesis and melena, multiple transfusions, splenomegaly on examination	18 months	Elastography = F2; abdominal USS = normal liver with splenomegaly; HBsAg and anti HCV were negative	GOV II: 40 mL of 50% glucose injected into the gastric varix	2	Lost to follow-up in 2022. Re-present two years later (June 2024) with severe anemia and melena
3	72	Recurrent painless hematemesis. Transfused with 4 units of blood	Two years	Abdominal USS and CT scans = normal liver. FibroScan = F0 (< 5 kPa), HBV, and HCV were negative	IGV I: 40 mL of 50% glucose injected into the gastric varix	1	The patient was asymptomatic since February 2024 (10 months)

CT, computer tomography; F0, no fibrosis; F2, moderate fibrosis; HBsAg, hepatitis B surface antigen; HBV, hepatitis B virus; HCV, hepatitis C virus; IGV, isolated gastric varix; GOV II, oesophago-gastric varix II; USS, ultrasound scan.

Intervention

Endoscopic injection sclerotherapy was performed, injecting 30–40 mL of 50% dextrose into the fundal varices over 20–30 m.

Outcome

The patient remained asymptomatic and bleeding-free since February 2024 (10 months) but declined any further endoscopic evaluation or intervention.

Summary of presentations

Table 1 below summarizes the cases, while Figure 1 shows endoscopic images of the gastric varices.

Discussion

These cases illustrate the complexity of managing portal hypertension and the financial burden faced by many patients in low-resource settings. The patients' recurrent upper gastrointestinal bleeding highlights the importance of timely interventions and the need for better access to medical resources. Using 50% dextrose in the endoscopic treatment of gastric fundal varices represents an innovative and promising approach to achieving hemostasis in the management of these challenging vascular lesions. Gastric varices, particularly fundal varices, pose significant clinical challenges due to their high propensity for severe bleeding and the associated morbidity and mortality.[14,15] Traditional sclerosants such as cyanoacrylate and ethanolamine have been widely used, but their application is often limited by cost, availability, and the potential for systemic complications.

In this case series, 50% dextrose demonstrated its efficacy by consistently inducing thrombosis and obliterating the variceal lumen, leading to immediate hemostasis in all treated cases. Its hyperosmolarity contributes to endothelial damage, protein denaturation, and vascular fibrosis, critical mechanisms in variceal obliteration.[16] Importantly, these effects were achieved with minimal complications, underscoring the favorable safety profile of 50% dextrose.

One of the major advantages of 50% dextrose lies in its accessibility and affordability. As a readily available solution, it provides an effective alternative in resource-limited settings where conven-

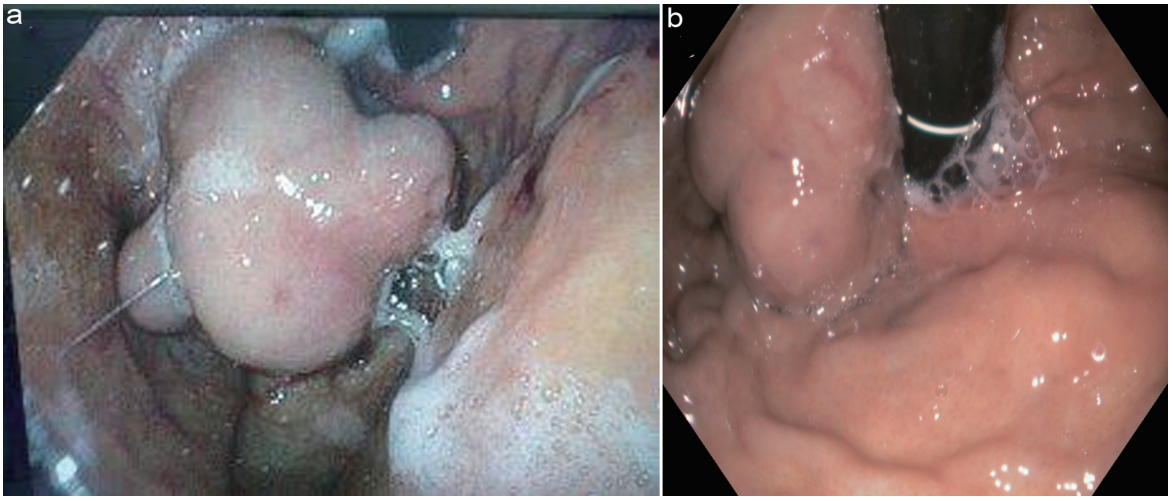

Fig. 1. Original endoscopic images of gastric varices from case 1 (a) and 2 (b).

tional sclerosants may be inaccessible. Additionally, its simplicity in administration makes it a practical choice for endoscopic sclerotherapy, without requiring specialized equipment or complex protocols.[17]

The cases described in this series highlight the ability of 50% dextrose to achieve hemostasis and prevent recurrent bleeding in clinical scenarios, ranging from patients with isolated fundal varices to those with refractory bleeding after other endoscopic interventions. Notably, several patients in this series achieved prolonged periods free from rebleeding, emphasizing the durability of this approach. However, the case of a young patient who experienced rebleeding and ultimately succumbed underscores the importance of long-term follow-up and the need for comprehensive management strategies, including addressing underlying portal hypertension.[18,19] Compared to other sclerosants, 50% dextrose is reported to be non-allergenic with a very good safety profile, making it a safer alternative for patients with sensitivities to other sclerosants.[17,20] This enhances its safety profile, particularly in high-risk populations, such as patients with advanced liver disease or coagulopathy.

While the results of this case series are encouraging, it is important to acknowledge its limitations. The small sample size and lack of a comparative control group preclude definitive conclusions about the superiority of 50% dextrose over other sclerosants. Moreover, long-term outcomes and the potential for variceal recurrence require further investigation in larger, prospective studies.

Conclusions

This case series provides compelling evidence for the use of 50% dextrose solution as an adjunctive treatment for gastric varices in resource-constrained settings like Nigeria, where access to cyanoacrylate glue is often limited. Our findings indicate that sclerotherapy with dextrose not only effectively manages acute variceal bleeding but also serves as a critical alternative that can significantly reduce the morbidity and mortality associated with this life-threatening condition.

Given the high incidence of portal hypertension-related complications and the associated healthcare burden, it is essential to explore and implement cost-effective strategies within local medical frameworks. The positive outcomes observed in our patients underline the necessity for broader adoption of this technique. Therefore, we advocate for the following steps moving forward:

Training and capacity building: Enhance the skills of gastroenterologists and general practitioners in using sclerotherapy with dextrose. Structured training programs can promote proficiency in endoscopic techniques and increase the availability of this intervention.

Clinical guidelines development: Formulate local clinical guidelines that include sclerotherapy with dextrose as an alternative treatment for gastric varices. This would provide physicians with a clear framework for managing cases in resource-limited settings.

Research and data collection: Conduct larger-scale studies to systematically evaluate the efficacy, safety, and long-term outcomes of 50% dextrose sclerotherapy. These studies could help establish a robust evidence base to support the practice.

Awareness and advocacy: Increase awareness among healthcare professionals and policy-makers about the burden of portal hypertension and the potential of alternative therapies. Advocacy efforts are essential to secure the necessary resources and support for implementing these strategies.

By adopting these steps, we can improve patient outcomes while mitigating the risks associated with untreated gastric varices in Nigeria and similar contexts. It is imperative to foster innovation and adaptability within our healthcare systems to ensure comprehensive and effective management of portal hypertension and its complications.

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Conflict of interest

The corresponding author, Yusuf Musa, serves at the Nigerian Medical Association, Medical and Dental Consultants Association of Nigeria, Society of Gastroenterology and Hepatology in Nigeria, and Society on Liver Disease in Africa. All the authors report no other relevant conflicts of interest for this article.

Author contributions

Conception, review, serving as a guarantor (YM), drafting of the manuscript, editing (YM, HTS, NMO, DI, ASA, HAZ, AAS). All authors have read and approved the final manuscript.

Ethical statement

The study was approved by the Federal Teaching Hospital, Katsina Health Research Ethics Committee (FTHKTNHREC.REG.24/06/22C/230), in accordance with the Declaration of Helsinki (as revised in 2024). Written informed consents were obtained from the patients for the publication of this case report and accompanying images.

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