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EDITORIAL TEAM

CASE SERIES

Surgery for Life Threatening Upper GI Bleeding Caused by Portal Hypertension After Failed Endoscopic Options: Single GI Surgical Unit Experience

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ABSTRACT

Portal hypertension can give rise to varices mainly involving the esophagus and gastric fundus. Rupture of these varices can result in life-threatening gastrointestinal (GI) bleeding. The mainstay of treatment involves resuscitation, medical management with vasoactive agents, and endoscopic therapies to obliterate bleeding varices. Radiological interventions, such as trans-jugular intrahepatic portosystemic shunt (TIPS), have become increasingly popular. However, surgery, including shunts or devascularization procedures, remains an option when other measures fail to control recurrent bleeding, particularly when there is no access to radiological interventional procedures (Trans jugular intrahepatic portosystemic shunts - TIPS).

This case series discusses the management of five patients who underwent surgical procedures for recurrent variceal bleeding, their outcomes, and the key factors to consider when selecting a surgical approach.

Keywords: Portal hypertension, variceal bleeding, shunting procedures, devascularization procedures

INTRODUCTION

Portal hypertension (PHT) though most commonly caused by cirrhosis of the liver, extra hepatic obstruction due to portal vein (PV) thrombosis (PVT) or cavernous formation of the PV can lead to life-threatening Upper GI bleeding (UGIB). (1,2). Advances in medical and endoscopic therapies, such as sclerotherapy and band ligation, have improved initial management. However, a subset of patients experiences recurrent or uncontrolled bleeding despite these interventions (3,4).

Current treatment strategies focus on hemodynamic stabilization, vasoactive drugs (e.g., terlipressin, octreotide), and endoscopic therapy (5,6). TIPS is often employed in refractory cases, but its feasibility depends on factors such as liver function and risk of hepatic encephalopathy (7,8).

Surgical procedures, including portosystemic shunt procedures (e.g., mesocaval, distal splenorenal shunt) or esophagogastric devascularization, remain viable options when endoscopic and radiological approaches fail (9-11).

Although surgical interventions carry inherent risks, particularly in patients with advanced liver disease, they remain an essential component of management, especially in settings where TIPS or liver transplantation is not readily available (12-14). Understanding the role of surgery in managing severe GI bleeding in portal hypertension is crucial to improving patient outcomes.

This case series presents the experience of a single GI surgical unit in managing severe GI bleeding secondary to portal hypertension through surgical means. By analyzing patient outcomes, perioperative complications, and survival rates, this study provides insights into the role of surgery in this high-risk patient population.

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CASE PRESENTATION

Between 2003 and 2005, five patients with portal hypertension underwent surgical procedures due to uncontrolled gastrointestinal bleeding. All five patients were young, and age between10 to 35 years, and included two females and three males. In all five cases, cavernous formation of the portal vein most likely resulted from PV thrombosis was identified as the underlying cause of portal hypertension.

These patients presented with recurrent episodes of acute upper gastrointestinal bleeding. Despite receiving multiple sessions of endoscopic therapy, including sclerotherapy and band ligation, they continued to experience recurrent and severe bleeding. During these bleeding episodes, all patients required significant transfusion support, with a minimum of five red cell packs administered per episode. Imaging and endoscopic evaluation revealed cavernous transformation of the portal vein, a complication of chronic thrombosis that leading to the portal hypertension.

Despite the aggressive endoscopic and medical management severity and frequent recurrences of the UGIB could not ne adequately controlled. Therefore, all five patients underventelective surgical procedures aimed at reducing portal pressure and preventing further episodes of life-threatening bleeding. Four of them underwent mesocaval shunt surgery using a reverse saphenous vein graft. This procedure was selected based on its ability to effectively divert blood flow and decompress the hypertensive portal circulation.

The remaining patient, a female who had previously undergone distal leno-renal shunt in a another center several years ago continued to experience persistent bleeding and was therefore subjected to a Sigura's procedure where devascularization of the gastric fundus followed by transection of the Gastro-esophageal junction using a circular stapler combined with splenectomy was carried out. In addition this patient was found to have an aneurysm of the splenic arterywhich was under run prior to the splenectomy, highlighting the complexity of managing varices in patients with recurrent hemorrhage despite prior shunting.

Postoperatively, there were no mortalities among the five patients. The patient, who underwent splenectomy and stapling of the gastroesophageal junction, developed intra-abdominal bleeding on the third postoperative day. She required a re-laparotomy, during which a bleeding vein in the splenic bed was identified and ligated. Following this intervention, her postoperative recovery was uneventful.

During follow-up, three of the patients remained under long-term monitoring, and none of them experienced recurrent gastrointestinal bleeding within a three-year period. Two patients, however, were lost to long-term follow-up, limiting the ability to assess their outcomes beyond this period.

One patient male patient developed cirrhosis during follow-up, suggesting ongoing progression of liver disease despite successful control of variceal bleeding. The female patient who underwent Sigura procedure is married and having two children and she was encountered accidently by the author recently and she never had a bleeding episode over the last 18 years.

These findings underscore the potential for surgical interventions to provide durable bleeding control in patients with severe portal hypertension while also highlighting the importance of continued monitoring for long-term complications such as cirrhosis or shunt dysfunction.

DISCUSSION

Portal hypertension secondary to PVT presents a significant challenge, particularly in young patients, as it predisposes them to life-threatening GI bleeding. The patients in this series continued to experience recurrent and severe hemorrhage despite the attempt to control the bleeding with multiplesessions of endoscopic therapy (sclerotherapy and band ligation). The failure of these endoscopic measures necessitated a more radical approach to contain the recurrent bleeding. In such cases, surgical interventions remain an important option, especially when TIPS is unavailable or contraindicated due to the underlying vascular anatomy or concerns regarding hepatic encephalopathy (15,16).

Summary of patients underwent surgery to control bleeding due to portal hypertension

Year	Patient Ref. no.	Age	Sex	Cause for Portal hypertension	Surgical access	Surgical Procedure	Immediate post-op complications	Post-op Mortality	Follow up
2003	1	35	Male	Portal vein	Open	Mesocaval shunt	None	No	Cirrhosis
2004	2	29	Female	Portal vein	Open	Splenectomy + Stapling of GO junction	Post-op bleeding	No	No bleeding
2004	3	20	Male	Portal vein	Open	Mesocaval shunt	None	No	No bleeding
2005	4	16	Female	Portal vein	Open	Mesocaval shunt	None	No	No follow-up data
2005	5	10	Male	Portal vein	Open	Mesocaval shunt	None	No	No follow-up data

The presence of cavernous transformation of the portal vein in all patients further complicated their management. This transformation results from chronic thrombosis and leads to the development of a network of small collateral vessels, which can make both endoscopic and radiological interventions technically challenging and less effective (17). Given these anatomical and clinical constraints especially the porto-caval shunt is not possible.Therefore, we had to resort to a more distal procedure such as meso-caval shunts.Devascularization perhaps remain the only available options for bleeding control when there is a recurrence following a previous shunt procedure.

Among the five patients, four underwent mesocaval shunting with a reverse saphenous vein graft. Shunting procedures are designed to decompress the portal system and reduce pressure within the varices, thereby preventing further episodes of bleeding (18).

The success of the shunting procedures in these patients, with no episodes of recurrent bleeding at three years of follow-up, highlights their effectiveness in selected cases. The patient who had already undergone a leno-renal shunt required devascularization procedure due to continued gastric variceal bleeding. This underscores the fact that while shunting is generally effective, certain patients, particularly those with prominent gastric varices, may require a combination of surgical approaches to achieve optimal bleeding control (19).

Long-term follow-up data further reinforce the benefits of surgical intervention. At three years, none of the patients who were followed up had experienced recurrent GI bleeding. However, one patient did develop cirrhosis, indicating that while surgical procedures effectively control bleeding, they do not halt the progression of liver fibrosis most probably due poor perfusion of the liver. This finding highlights the importance of continued hepatology follow-up, even in patients who have undergone successful surgical treatment for portal hypertension. Liver disease progression, shunt dysfunction, or the development of new varices remain long-term concerns that necessitate ongoing surveillance (20,21).

In the context of resource-limited settings, where access to radiological interventions such as TIPS is restricted, the findings of this study suggest that surgical procedures continue to hold an important role. The technically demanding nature of shunt surgery and devascularization procedures requires careful patient selection and a skilled surgical team, but when performed appropriately, these interventions can provide effective and durable bleeding control.

CONCLUSION

In this case series, mesocaval shunting and devascularization procedures were found to be effective in controlling recurrent variceal bleeding in young patients with portal hypertension due to PVT. The patients who were followed long-term did not experience further bleeding episodes, demonstrating the durability of these interventions. Though technically demanding, these procedures remain valuable treatment options, particularly in settings where TIPS is not available. However, the risk of disease progression highlights the importance of continued hepatology follow-up.

REFERENCES

1.García-Tsao, G., Abraldes, J. G., Berzigotti, A., & Bosch, J. (2017). Portal hypertensive bleeding in cirrhosis: Risk stratification, diagnosis, and management: 2016 practice guidance by the American Association for the Study of Liver Diseases. Hepatology, 65(1), 310-335. doi:10.1002/hep.28906

2.Bosch, J., &Abraldes, J. G. (2021). The pathophysiology of portal hypertension: Current concepts. Gastroenterology, 160(6), 1770-1785. doi:10.1053/j.gastro.2020.10.045

3.de Franchis, R. (2020). Expanding consensus in portal hypertension: Report of the Baveno VI Consensus Workshop: Stratifying risk and individualizing care for portal hypertension. Journal of Hepatology, 63(3), 743-752. doi:10.1016/j.jhep.2015.05.022

4.Villanueva, C., Colomo, A., Bosch, A., &Abraldes, J. G. (2015). Transfusion strategies for acute upper gastrointestinal bleeding. New England Journal of Medicine, 372(1), 11-22. doi:10.1056/NEJMra1411312

5.Thabut, D., & Rudler, M. (2019). Transjugular intrahepatic portosystemic shunt in the management of portal hypertension. Nature Reviews Gastroenterology & Hepatology, 16(4), 173-186. doi:10.1038/s41575-018-0089-7

6.Boyer, T. D., Haskal, Z. J., & American Association for the Study of Liver Diseases. (2010). The role of transjugular intrahepatic portosystemic shunt (TIPS) in the management of portal hypertension: Update 2009. Hepatology, 51(1), 306-328. doi:10.1002/hep.23383

7.Clavien, P. A., Petrowsky, H., DeOliveira, M. L., & Graf, R. (2007). Strategies for safer liver surgery and partial liver transplantation. New England Journal of Medicine, 356(15), 1545-1559. doi:10.1056/ NEJMra 065156

8.Malinchoc, M., Kamath, P. S., Gordon, F. D., Peine, C. J., Rank, J., & Ter Borg, P. C. (2000). A model to predict poor survival in patients undergoing transjugular intrahepatic portosystemic shunts. Hepatology, 31(4), 864-871. doi:10.1053/he.2000.5852

9.Sarin, S. K., & Kumar, A. (2017). Management of portal hypertension: Consensus statement of the Asian Pacific Association for the Study of the Liver (APASL). Hepatology International, 11(4), 317-332. doi:10.1007/s12072-017-9817-6

10.Perarnau, J. M., Leclercq, C., Bercoff, J. P., & Hillaire, S. (2012). Early TIPS in cirrhosis with variceal bleeding: Are we ready for broader indications? Journal of Hepatology, 56(5), 1206-1211. doi:10.1016/j.jhep.2011.10.010

11.Monescillo, A., Martínez-Lagares, F., Ruiz-Del-Arbol, L., Sierra, A., Nicolau, C., Guevara, C., ... & Rincón, D. (2004). Influence of portal pressure reduction on the outcome of variceal bleeding in patients with cirrhosis. Hepatology, 40(2), 355-362. doi:10.1002/hep.20284

12.Sauerbruch, T., Mengel, M., Dollinger, M., Zipprich, A., Rössle, M., Panther, E., & Gotthardt, D. (2015). Prevention of rebleeding from esophageal varices in patients with cirrhosis receiving small-diameter covered TIPS: A randomized controlled trial. Gastroenterology, 149(3), 660-668. doi:10.1053/j.gastro.2015.05.005

13.D'Amico, G., De Franchis, R. (2003). Upper digestive bleeding in cirrhosis: Post-therapeutic outcome and prognostic indicators. Hepatology, 38(3), 599-612. doi:10.1053/jhep.2003.50385

14.Funakoshi, N., Duny, Y., Ségalas-Largey, F., Flori, N., Bismuth, M., Fumery, M., & Blanc, P. (2017). Meta-analysis: Beta-blockers versus endoscopic band ligation for primary prophylaxis of esophageal variceal bleeding. European Journal of Gastroenterology & Hepatology, 29(2), 101-107. doi:10.1097/MEG.0000000000000765

15.Jalan, R., Elton, R. A., Redhead, D. N., Finlayson, N. D. C., & Hayes, P. C. (1995). Analysis of prognostic variables in the prediction of mortality, shunt failure, and encephalopathy following transjugular intrahepatic portosystemic stent-shunt (TIPS). Journal of Hepatology, 23(2), 123-128. doi:10.1016/0168-8278(95)80480-4

16.Fuster, J., Garcia-Valdecasas, J. C., Grande, L., Tabet, J., & Andreu, H. (1997). Mesocaval shunts in portal hypertension. Surgery, 122(2), 135-141. doi:10.1016/S0039-6060(97)90239-5

17.Boyer, T. D., &Haskal, Z. J. (2005). The role of transjugular intrahepatic portosystemic shunt in the management of portal hypertension. Hepatology, 41(2), 386-400. doi:10.1002/hep.20665

18.Liu, F., Du, C., Zhou, X., Xia, Y., & Zhang, X. (2020). Comparison of surgical shunts and TIPS for variceal bleeding in noncirrhotic portal hypertension. Journal of Vascular and Interventional Radiology, 31(1), 97-104. doi:10.1016/j.jvir.2019.08.025

19.Orloff, M. J., Isenberg, J. I., Wheeler, H. O., Hayward, G. W., &Jinich, H. (1994). Randomized trial of emergency portacaval shunt and endoscopic sclerotherapy for acutely bleeding esophageal varices in cirrhosis. New England Journal of Medicine, 330(15), 1227-1233. doi:10.1056/NEJM199404143301501

20.Henderson, J. M., Kutner, M. H., Millikan, W. J., & Warren, W. D. (1982). Prophylactic portacaval shunt: A randomized controlled trial. Annals of Surgery, 196(4), 442-449. doi:10.1097/00000658-198210000-00008

REVIEW ARTICLE

Proton Pump Inhibitors: A Review on Uses and Misuses

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ABSTRACT

Proton pump inhibitors (PPI) are a medication that is invaluable in treating patients with gastroesophageal reflux disease (GERD), erosive esophagitis, Barrett esophagus,eosinophilic esophagitis, peptic ulcer disease etc. Since it's a medication that is used by health care providers at every level in addition to its use as an over the counter medication, clear awareness on definite indications and attempts to de prescribe by all means whenever necessary is essential to maintain a rational clinical practice in our health care setting.

Concernsare raised on possible adverse effects of long-term use of PPI such as acute interstitial nephritis, chronic kidney disease, dementia, consequences of acid inhibition including gastrointestinal infections, pneumonia, nutrient deficiencies, fractures, spontaneous bacterial peritonitis, and small intestinal bacterial overgrowth. Education of both health care provider and patients on appropriate use of PPI is the key to maintain the quality of an excellent health care system. This article is aimed at improving awareness among health care providers on appropriate uses of PPI.

Key Words: Proton pump inhibitors, Esophagitis

BACKGROUND

Introduction of Proton pump inhibitors (PPI) to the clinical setting transformed the management of patients with dyspeptic symptoms due to the benefits over the traditional anti secretory medications previously used such as H2 receptor blockers (H2RB) due to their efficacy, duration of action and minimal effects on tolerance. Endless expansion of prescriptions of PPIs raised the concerns on cost for both patients and governments and the potential risk for iatrogenic harm due to potential adverse effects related to these medications.

Proton pump inhibitors (PPIs) are commonly used both by clinicians and as over the counter medication. PPI use is on the rise estimating 7%–15% of patients use these medications at any time, and accounting for 40% for patients 70 years or older. (1)

Although PPIs are the first line option for the management of acidmediated upper gastrointestinal (GI) disorders, such as erosive esophagitis and peptic ulcer disease, they are being used excessively for less clear indications and for indeterminate durations.(1)

INDICATIONS FOR PPI

Clinician should always be responsible to justify the decision on starting PPI for a valid indication and regularly review the patient with regards to the need for ongoing use.

All patients without an indication for long term PPI should be considered for de prescribing. Table 1 shows definite and conditional indications for PPI in short and long term use.(1)

ADVERSE EFFECTS

Studies have shown associations between long term use of PPIand numerous side effects such as intestinal infections, pneumonia, osteoporosis, stomach cancer, chronic kidney disease, vitamin and mineral deficiencies, ischaemic heart disease, strokes, dementia, and early death. Most of these adverse effects are studied on the basis of its acid suppression causing a rise in luminal pH. However, high quality studies have not shown definitive cause-and -effect relationship between PPI and these adverse reactions except intestinal infections.(2)

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Table 1 : Indications for PPI

Indications for long-term use		
Definitely indicated for long-term use (>8 wk)	**Conditionally indicated for long-term use**	**Not indicated for long-term use**
Barrett's esophagus Clinically significant (Los Angeles Classification grade C/D) erosive esophagitis Esophageal strictures from GERD (ie, peptic strictures) Zollinger-Ellison syndrome Eosinophilic esophagitis Gastroprotection in users of ASA/ nonsteroidal anti-inflammatory drug at highrisk for GI bleeding Prevention of progression of idiopathic pulmonary fibrosis	PPI-responsive endoscopy-negative reflux disease, with recurrence on PPI cessation PPI-responsive functional dyspepsia, with recurrence on PPI cessation PPI-responsive upper airway symptoms ascribed to laryngopharyngeal reflux, with recurrence on PPI cessation Refractory steatorrhea in chronic pancreatic insufficiency with enzyme replacement Secondary prevention of gastric and duodenal peptic ulcers with no concomitant antiplatelet drugs	Symptoms of nonerosive reflux disease with no sustained response to high-dose PPI therapy Functional dyspepsia with no sustained response to PPI therapy Steroid therapy in the absence of ASA/ nonsteroidal anti-inflammatory drug therapy Prevention of recurrent upper GI bleeding from causes other than: Peptic ulcer disease, including gastric and duodenal erosions Erosive esophagitis

Indications for short-term use		
Definitely indicated for acute/ short-term use (<8 wk)	**Conditionally indicated for acute/ short-term use**	**Not indicated for acute/ short-term use**
Helicobacter pylori Eradication Stress ulcer prophylaxis for ICU patients with risk Factors Uninvestigated GERD/ Dyspepsia Treatment of NSAID-related gastric and duodenal peptic ulcers	Initial or on-demand treatment of endoscopy- negative reflux disease Initial treatment of functional Dyspepsia Uninvestigated dyspepsia Ulcer prevention after sclerotherapy or band ligation treatment of esophageal varices Prevention of rebleeding from Mallory-Weiss tears	Empiric treatment of laryngopharyngeal symptomatology Acute undifferentiated abdominal pain Acute nausea and vomiting not believed to be related to GERD/ esophagitis Any isolated lower GI Symptomatology

Nevertheless, it is too early to disregard the above adverse effects completely since further research on this aspect is essential. Generally, well established benefits of PPIs far outweigh the theoretical adverse effects. Minor side effects such as headache, abdominal pain, nausea, vomiting, diarrhea, constipation, and flatulence could be managed with switching to a different PPI. (2) Table 2 shows evidence and clinical recommendations regarding potential risks of PPIs.

DEPRESCRIBING

Current evidence suggest a potential result of prolonged PPI therapy with increased risk of long-term hypergastrinemia, Enterochromaffin-like cell hyperplasia, and parietal cell hypertrophy.

Rebound acid hypersecretion is thought to be the cause for symptomatic dyspepsia once PPI therapy is withheld.(3) Therefore, patients should be made aware that there's a possibility of transient worsening of dyspeptic symptoms after withdrawal of PPI due to this rebound acid hyper secretion.(1)

Most patients with an indication for PPI and on double dose PPI could be considered for once daily dosing. However, for patients with severe erosive esophagitis, esophageal ulcer, peptic stricture, Barretts oesophaus, eosinophilic oesophagitis with satisfactory response to PPI or idiopathic pulmonary fibrosis should not be considered for a trial of de prescribing.

Deprescribing PPI could be attempted by stepping down to a lower dose or to an intermittent/on demand course or by replacing with a less potent anti secretory medication, such as H2 receptor antagonists. (4)Furthermore, discontinuation of PPI should be based mainly on the lack of an indication for PPI use, and not considering the PPIassociated adverse events.(3)

CONCLUSION

Introduction of PPI to the market has transformed the practice of managing patients with various acid related disorders. When considering the adverse effects and burden of cost to the health system, decision on prescription should be universal and rational.

Table 2:Evidence and clinical recommendations regarding potential risks of PPIs (3)

Potential risk	Evidence	Clinical recommendation
Infections Enteric infections	More than twofold risk of development of Clostridium difficile related diarrhoea	Prescribe weighing risks and benefits
Community-acquired Pneumonia	Minimal increase in risk	Withholding PPIs in pulmonary disease is not recommended
Bone fracture	Conflicting results hip fractures	Consider risks and benefits in aging patients and those at risk for osteoporosis
Drug interactions Clopidogrel	Inconsistent results	Consider risks and benefits on individualized basis
Nutritional deficiencies Vitamin B12	Higher risk in elderly, malnourished and post gastric by pass	Consider screening patients at higher risk
Iron	Paucity in data	Routine screening not recommended
Magnesium	Paucity in data	Consider screening patients at higher risk
Pregnancy	Most studies are limited to omeprazole; no significant risk	Omeprazole is safe in pregnancy

This article is expected to shed some light on health care providers to enhance the quality of practice with regards to uses of PPI.

REFERENCES

1.Targownik LE, Fisher DA, Saini SD. AGA Clinical Practice Update on De-Prescribing of Proton Pump Inhibitors: Expert Review. Gastroenterology. 2022 Apr;162(4):1334-1342. doi: 10.1053/j.gastro.2021.12.247. Epub 2022 Feb 17. PMID: 35183361.

2.Katz PO, Dunbar KB, Schnoll-Sussman FH, Greer KB, Yadlapati R, Spechler SJ. ACG Clinical Guideline for the Diagnosis and Management of Gastroesophageal Reflux Disease. Am J Gastroenterol. 2022 Jan 1;117(1):27-56. doi: 10.14309/ajg.0000000000001538. PMID: 34807007; PMCID: PMC8754510.

3.Heidelbaugh JJ, Kim AH, Chang R, Walker PC. Overutilization of proton-pump inhibitors: what the clinician needs to know. Therap Adv Gastroenterol. 2012 Jul;5(4):219-32. doi: 10.1177/1756283X12437358. PMID: 22778788; PMCID: PMC3388523.

4.Vincenzo Savarino, Elisa Marabotto, Patrizia Zentilin, Manuele Furnari, Giorgia Bodini, Costanza De Maria, Gaia Pellegatta, Claudia Coppo & Edoardo Savarino (2018): Proton pump inhibitors: use and misuse in the clinical setting., Expert Review of Clinical Pharmacology, DOI: 10.1080/17512433.2018.1531703

CASE REPORT

Acute Pancreatitis Revealing An Underlying Gastric Lymphoma: A Case Report Highlighting A Diagnostic Pitfall

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ABSTRACT

Gastric lymphoma is a rare malignancy of the stomach, often presenting with nonspecific gastrointestinal symptoms. Presentation as acute pancreatitis is extremely uncommon and can obscure the underlying diagnosis. A 17-year-old male presented with epigastric pain and vomiting. Investigations revealed elevated serum amylase and imaging findings suggestive of acute pancreatitis. During admission, he developed obstructive jaundice and varicella-zoster infection. Persistent symptoms prompted endoscopic retrograde cholangiopancreatography (ERCP), which revealed an ulcerated stomach. Biopsy confirmed high-grade B-cell lymphoma. The patient's condition deteriorated rapidly, and he succumbed before chemotherapy could be initiated. Gastric lymphoma can rarely mimic acute pancreatitis. Clinicians should maintain a high index of suspicion in atypical cases and consider early endoscopic evaluation to avoid delays in diagnosis and treatment.

Keywords: Gastric lymphoma, Acute pancreatitis, Varicella-zoster

INTRODUCTION

Primary gastric lymphoma accounts for one to five percent of all gastric malignancies, with the stomach being the most common site of extranodal lymphoma [1,2]. Most cases are of B-cell lineage. Clinical presentation is often nonspecific and may include abdominal pain, nausea, vomiting, anorexia, and dyspepsia [3]. Constitutional “B” symptoms such as fever, night sweats, and weight loss are less common [3]. Presentation as acute pancreatitis is extremely rare [4,5], particularly in adolescent patients. Awareness of this atypical manifestation is critical for timely recognition, accurate diagnosis, and prompt initiation of therapy.

CASE REPORT

A 17-year-old previously healthy male presented with three weeks of epigastric pain radiating to the back. The pain was aggravated by meals and relieved by leaning forward. He reported postprandial nonbilious vomiting, anorexia, and weight loss, without any changes in bowel habits.

During hospitalization, the patient developed obstructive jaundice. Examination revealed icterus and a tender epigastric mass. Laboratory investigations demonstrated a serum amylase of 440 U/L, C-reactive protein of 35 mg/L, total bilirubin of 10 mg/dL with a direct fraction of 6.5 mg/dL, gamma-glutamyl transferase of 1500 U/L, and alkaline phosphatase of 510 U/L. Abdominal ultrasonography suggested acute pancreatitis with biliary dilatation. Computed tomography of the abdomen revealed diffuse pancreatic enlargement with multiple mildly enhancing areas and diffuse gastric wall thickening(Figure 01).

The patient was initially managed conservatively for acute pancreatitis. ERCP, planned to evaluate the cause of obstructive jaundice, was delayed by one week due to varicella-zoster infection. During ERCP, the stomach appeared edematous and ulcerated(Figure 2), and biopsies were obtained.

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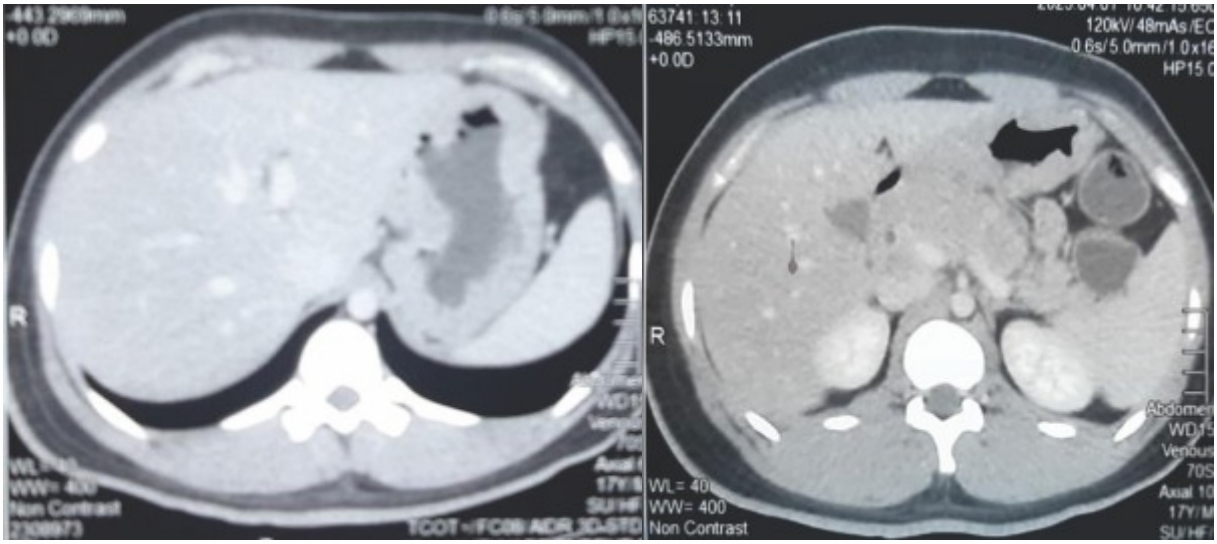

Figure 01- Contrast CT abdomen showing thickened gastric wall and oedematous pancreatic head

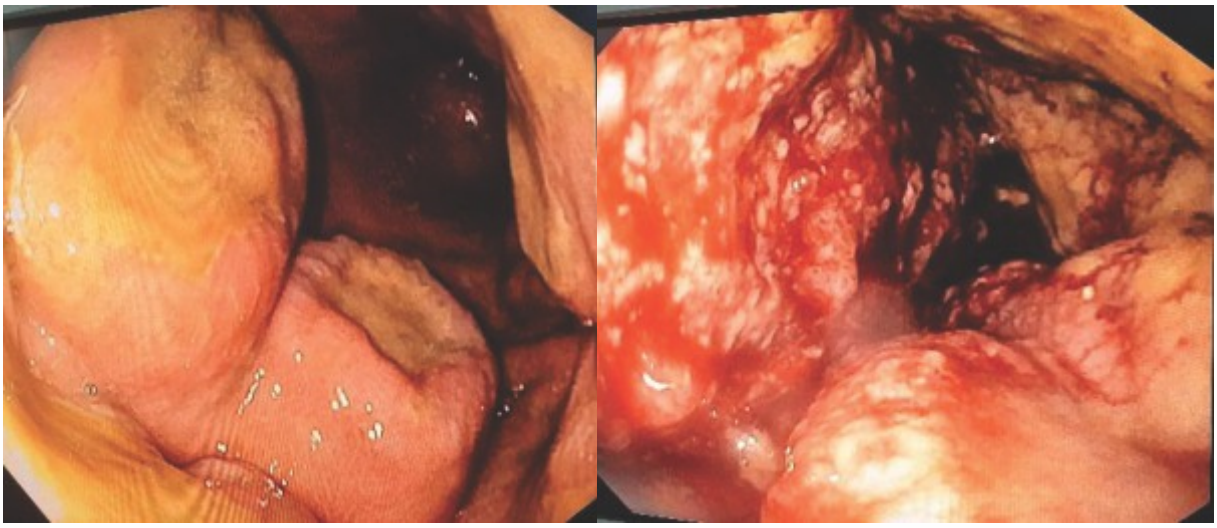

Figure 02- Upper gastrointestinal endoscopy showing diffusely edematous and ulcerated gastric mucosa with loss of normal folds

Histopathological analysis confirmed high-grade B-cell lymphoma. Subsequently, the patient developed bilateral pleural effusions and gross ascites, leading to respiratory compromise. He was admitted to the intensive care unit but could not commence chemotherapy and unfortunately passed away.

DISCUSSION

Primary gastric lymphoma frequently presents with nonspecific gastrointestinal symptoms, which can result in delayed diagnosis [3]. Abdominal pain, nausea, vomiting, and dyspepsia are common manifestations, whereas constitutional "B" symptoms such as fever, night sweats, and weight loss are less frequent [3]. Presentation as acute pancreatitis is exceedingly rare [4,5], and atypical presentation can obscure the underlying diagnosis. In this patient, initial laboratory and imaging findings, including elevated serum amylase and diffuse pancreatic enlargement on computed tomography, were consistent with acute pancreatitis, which initially reinforced that diagnosis and delayed consideration of an underlying malignancy.

The pathophysiology of pancreatitis in gastric lymphoma is not fully understood. It may result from mechanical obstruction of the pancreatic duct by the tumor mass, direct infiltration of lymphoma into

pancreatic tissue causing local inflammation, or periampullary and biliary involvement leading to cholestasis and secondary pancreatic injury [4,5]. In this patient, the subsequent development of obstructive jaundice and persistent symptoms were key indicators that prompted further investigation. Imaging had already demonstrated diffuse thickening of the gastric wall, which, in retrospect, should have prompted earlier upper gastrointestinal endoscopy with biopsy, potentially allowing timely diagnosis and initiation of chemotherapy.

The patient's clinical course was further complicated by varicella-zoster infection, which delayed definitive ERCP and biopsy. Immunosuppression associated with lymphoma increases susceptibility to opportunistic infections such as varicella-zoster, adding complexity to patient management and contributing to morbidity and mortality [4].

Few cases of gastric lymphoma presenting as acute pancreatitis have been described in the literature, most in adults, with pediatric and adolescent cases being exceptionally rare [4,5]. Reporting such cases is important to raise awareness that atypical presentations of pancreatitis, particularly when prolonged or associated with biliary obstruction, may indicate an underlying malignancy.

Clinicians should maintain a high index of suspicion in atypical cases and consider early endoscopic evaluation when imaging demonstrates gastric wall thickening or when symptoms do not follow the expected course. Awareness of these rare presentations can prevent diagnostic delays, allow timely therapeutic intervention, and improve patient outcomes.

CONCLUSION

Gastric lymphoma may rarely present as acute pancreatitis. Clinicians should maintain vigilance in atypical cases, pursue early endoscopic evaluation, and recognize secondary infections that may complicate management. Timely recognition is essential for improving outcomes in these patients.

REFERENCES

1.Raj M, Ghoshal UC, Choudhuri G, Mohindra S. Gastric lymphoma presenting as acute pancreatitis: a case report. JOP. 2013;14:1186–9.

2.Ghani S, Sarhani A, Benzzoubeir N, Chahed Ouazzani L, Errabih I. Primary gastric lymphoma: Clinical presentation and management. PAMJ-CM. 2020;18(3):58.

3.Psyrri A, Papageorgiou S, Economopoulos T. Primary extranodal lymphomas of the stomach: Clinical presentation, diagnostic pitfalls, and management. Ann Oncol. 2008;19:1992–1999.

4.Radhika M, Ghoshal UC, Choudhuri G. Gastric lymphoma presenting as acute pancreatitis: a rare case. JOP. 2013;14:1186–9.

5.Rossi E, Cavanna L, Di Russo G, et al. Gastric lymphoma presenting as acute pancreatitis: Review of the literature and case report. Tumori. 2005;91:344–6.

CASE REPORT

A Possible Case of Warfarin-Induced Acute Liver Failure in a Patient with Severe Heart Failure with Reduced Ejection Fraction: A Case Report

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ABSTRACT

Warfarin is a commonly used oral anticoagulant in the prevention and management of thromboembolic conditions (3). It has a very narrow therapeutic index and need regular monitoring on INR. It is well known to cause common side effect such as bleeding while hepatotoxicity is a relatively uncommon and frequently over looked. We present the case of a 71-year-old male patient with heart failure with reduced ejection fraction (HFrEF) (1,2)who experienced acute liver damage following commencement of Warfarin(4). Prompt recognition, removal of the cause, and proper care led to complete liver recovery. This case underscores the significance of maintaining a heightened index of suspicion for warfarin-induced liver damage (7), especially in patients with existing heart conditions that make them more susceptible to liver failure.

Key Words: Warfarin, Acute Liver Failure, Heart Failure with Reduced Ejection Fraction

INTRODUCTION

Heart failure with reduced ejection fraction (HFrEF) (1) is a major global health problem associated with significant morbidity and mortality. Management relies on pharmacological and lifestyle interventions, with warfarin, a vitamin K antagonist (3), commonly prescribed in HFrEF patients (1,2) at high thrombotic risk. Warfarin inhibits vitamin K–dependent clotting factors (8) but has a narrow therapeutic index. Although bleeding is the most frequent complication, hepatotoxicity is rare. Warfarin-related liver injury ranges from mild enzyme elevation to acute liver failure, an exceptionally uncommon but life-threatening condition (6).

This report describes a 71-year-old man with severe left ventricular failure (1) who developed acute liver failure following warfarin initiation.

CASE REPORT

A 71-year-old male with severe left heart failure and an ejection fraction of 20% presented to the emergency department with progressively worsening jaundice, abdominal distension, and extreme fatigue of one-week duration. His medical history included heart failure with reduced ejection fraction (New York Heart Association [NYHA] class IV), hypertension, and stage 3 chronic kidney disease.

His heart failure was managed according to guideline-directed medical therapy, including an angiotensin-converting enzyme inhibitor, beta-blocker, mineralocorticoid receptor antagonist, SGLT2 inhibitor, and anticoagulation with warfarin. Warfarin 3 mg had been initiated two weeks earlier following detection of severe left ventricular dysfunction and enlargement on echocardiography. His international normalized ratio (INR) had been closely monitored and maintained within the therapeutic range of 2.0–3.0. He denied alcohol use, illicit drug exposure, recent travel, or contact with viral hepatitis, and had no family history of liver disease.

Two days prior to admission, he developed worsening fatigue, increasing abdominal distension, and progressive disorientation, culminating in reduced responsiveness that prompted his family to seek emergency care.

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On examination, the patient was afebrile, jaundiced, and disoriented to time and place, with a Glasgow Coma Scale (GCS) score of 13. He had mild scleral pallor, bilateral pitting peripheral edema, and right-sided abdominal tenderness with minimal free fluid. There were no stigmata of chronic liver disease such as clubbing or spider naevi. Cardiovascular examination revealed a heart rate of 88 beats per minute, blood pressure of 110/70 mmHg, and bibasal fine crepitations on lung auscultation.

An urgent non-contrast CT scan of the brain showed no acute intracranial pathology. Laboratory investigations demonstrated markedly elevated liver enzymes, with aspartate aminotransferase (AST) of 672 U/L and alanine aminotransferase (ALT) of 1328 U/L. Alkaline phosphatase was elevated at 190 U/L. Total bilirubin was 46.4 µmol/L, predominantly unconjugated (32.9 µmol/L). Prothrombin time was significantly prolonged, with an INR of 4.76, indicating impaired hepatic synthetic function. Serum albumin was 34 g/L, consistent with an acute hepatic insult. Serum creatinine remained stable at 204 µmol/L, in keeping with his baseline renal impairment. The full blood count was unremarkable.

Further investigations included serological testing for hepatitis A, B, and C, cytomegalovirus, Epstein–Barr virus, and HIV, all of which were negative. Autoimmune markers, including antinuclear antibodies, immunoglobulin G levels, and anti-smooth muscle antibodies, were also negative. A toxicology screen detected no hepatotoxic substances other than warfarin. Abdominal ultrasound demonstrated a liver of uniform echogenicity without focal lesions, biliary dilatation, or fatty infiltration. Doppler studies of hepatic vessels were normal. The kidneys showed features consistent with early chronic parenchymal disease, with no other significant abdominal abnormalities.

Based on the rapidly progressive jaundice, markedly elevated transaminases, prolonged INR, altered mental status, and exclusion of common etiologies, a diagnosis of acute liver failure was established. Given the temporal relationship between symptom onset and warfarin initiation, along with the absence of alternative causes, possible warfarin-induced acute liver failure was considered the most likely diagnosis. Although the INR was supratherapeutic, it was not thought to solely account for the degree of hepatic injury.

Ischemic hepatitis related to heart failure was considered in the differential diagnosis; however, the magnitude and pattern of enzyme elevation were greater than typically observed in ischemic injury.

Other drug-induced causes were unlikely based on medication history and toxicology results. Viral, autoimmune, and biliary causes were excluded through appropriate investigations.

Warfarin was immediately discontinued, and the patient was admitted to the intensive care unit for close monitoring and supportive management. Intravenous vitamin K was administered to correct coagulopathy, and lactulose was initiated for hepatic encephalopathy. Fluid intake was restricted, and empirical third-generation cephalosporin therapy was commenced. His heart failure medications were continued with careful monitoring for potential hepatic effects.

Liver function tests and blood glucose levels were monitored daily. Initially, liver enzymes and bilirubin continued to rise but gradually improved following cessation of warfarin and supportive care. The INR normalized with vitamin K therapy, which was continued until stabilization within the therapeutic range to minimize thromboembolic risk.

After one week in intensive care, the patient showed steady clinical and biochemical improvement, with resolution of encephalopathy and gradual clearance of jaundice. He was transferred to a general medical ward for further observation and rehabilitation. Repeat liver function tests demonstrated significant reductions in transaminases and bilirubin. Warfarin was permanently discontinued due to the high likelihood of drug-induced liver injury. Given his severe left ventricular dysfunction and thrombotic risk, anticoagulation was reinitiated with apixaban following careful risk–benefit assessment.

The patient was discharged after a three-week hospital stay. Follow-up evaluations showed continued improvement and eventual normalization of liver function tests. His heart failure remained well controlled, and he tolerated the direct oral anticoagulant without complications. At six-month follow-up, he remained asymptomatic with stable cardiac function and normal liver biochemistry.

DISCUSSION

This case report highlights a rare but potentially life-threatening adverse effect of warfarin therapy, namely acute liver failure. Warfarin (4) is a widely used anticoagulant and is generally considered safe; however, it has been associated with infrequent yet clinically significant hepatotoxicity. The precise mechanism underlying possible warfarin-induced liver injury (6) remains unclear. Proposed mechanisms include hypersensitivity reactions, direct hepatocellular toxicity, and idiosyncratic metabolic responses.

Hypersensitivity-related liver injury typically presents with fever, rash, eosinophilia, and elevated liver enzymes. Although our patient did not exhibit fever or rash, this does not entirely exclude a hypersensitivity-mediated process. Direct hepatotoxicity from warfarin is considered uncommon, as the drug is administered in low doses and there is no clear dose–response relationship. Individual susceptibility related to genetic or metabolic variation may therefore play an important role in the development of liver injury.

A review of the literature reveals very few reported cases of acute liver failure attributable to warfarin(7). Similar cases often describe rapid onset of jaundice with marked transaminase elevation in patients receiving warfarin, following exclusion of more common causes of liver injury. The onset of hepatotoxicity may range from weeks to several years after initiation of therapy. In our patient, liver injury developed after three weeks of treatment. Diagnosis is largely one of exclusion, with particular attention to the temporal relationship between drug exposure and liver dysfunction. The Roussel Uclaf Causality Assessment Method (RUCAM), which considers factors such as timing, clinical course, risk factors, concomitant drugs, and exclusion of alternative causes, would likely categorize this case as probable or very likely warfarin-induced acute liver failure.

Management centers on immediate discontinuation of warfarin and supportive care. Vitamin K, with or without fresh frozen plasma, may be required to correct coagulopathy, while liver transplantation remains a consideration in refractory cases. In this patient, withdrawal of warfarin and supportive treatment resulted in gradual and complete recovery (8-10).

Despite the extremely low incidence of acute liver failure associated with warfarin (7), the potential severity warrants clinical vigilance. This case underscores the importance of considering drug-induced liver injury in patients receiving warfarin and supports timely recognition, cessation of the offending agent, and appropriate alternative anticoagulation.

CONCLUSION

A patient with heart failure developed acute liver failure during warfarin therapy. After exclusion of other causes, warfarin was identified as the likely trigger. Drug withdrawal and supportive care led to full recovery. This case highlights the rare but serious risk of warfarin-induced liver failure and underscores the need for vigilance. Further studies is required to gain a more complete understanding of warfarin-induced liver damage and to pinpoint potential causes of this uncommon adverse effect.

REFERENCES

1. Ponikowski P, Vasan RS, Anker SD, et al. 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: The Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC) Developed with the special contribution of the Heart Failure Association (HFA) of the ESC. Eur Heart J. 2016;37(27):2129-2200.

2. Yancy CW, Jessup M, Bozkurt B, et al. 2013 ACCF/AHA Guideline for the Management of Heart Failure: A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation. 2013;128(16):e240-e327.

3. Holbrook AM, Schulman S, Witt DM, et al. Evidence-Based Management of Anticoagulant Therapy: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. Chest. 2012;141(2 Suppl):e152S-e184S.

4. Ageno W, Gallus AS, Wittkowsky AK, et al. Oral anticoagulation therapy: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. Chest. 2012;141(2 Suppl):e44S-e88S.

5. Andrade RJ, Lucena MI, Fernández-Castañer A, et al. Drug-induced liver injury: an analysis of 461 incidences assessed by an expert panel. Gastroenterology. 2005;129(2):512-521.

6. Eckstein C, Keitel V, Kubitz R, Häussinger D. Warfarin-induced liver injury: a case report and review of the literature. Eur J Gastroenterol Hepatol. 2007;19(11):987-991.

7. Lee WM, Squires JE, Narkewicz MR, et al. Acute Liver Failure: Summary of a Workshop. Hepatology. 2008;47(4):1401-1415.

8. O'Reilly RA. Vitamin K and the oral anticoagulant drugs. Annu Rev Med. 1976;27:245-261.

9. Martin-Orozco E, Fontalba A, Romero-Gomez M. Acute liver failure associated with warfarin therapy. Gastroenterol Hepatol. 2010;33(7):531-532.

10. Danan G, Benichou C. Causality assessment of adverse reactions to drugs--I. A novel method based on the conclusions of international consensus meetings: application to drug-induced liver injuries. J Clin Epidemiol. 1993;46(11):1323-1330.

CASE REPORT

Beyond Hypersplenism: Aplastic Anemia Unmasked in A Patient with Cirrhosis - A Case Report

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ABSTRACT

The co-occurrence of aplastic anemia and cirrhosis is rare and presents a diagnostic challenge. We report the case of a 71-year-old female with compensated cirrhosis and portal hypertension who presented with symptomatic anemia and was found to have pancytopenia. The initial presumption of hypersplenism was revised after a bone marrow biopsy revealed aplastic anemia. This case underscores the importance of considering alternative causes of pancytopenia in cirrhotic patients beyond hypersplenism, particularly when the cytopenias are severe.

Key words: Aplastic anemia, hypersplenism , pancytopenia

INTRODUCTION

Aplastic anemia is a rare hematological disorder characterized by pancytopenia and a hypocellular bone marrow, which may be congenital or acquired. The acquired form is often immune-mediated. While anemia is common in liver disease due to gastrointestinal bleeding or hypersplenism, the coexistence of aplastic anemia and cirrhosis is unusual(1,10). Hepatitis-associated aplastic anemia has been described; however, the occurrence of aplastic anemia associated with other etiologies of cirrhosis, as seen in this case, remains rare.

CASE REPORT

A 71-year-old female with known cirrhosis (Child–Pugh class A), most likely secondary to metabolic-associated steatotic liver disease, presented with progressively worsening tiredness and shortness of breath for two months duration.

There were no other symptoms such as cough, chest pain, fever, abdominal distension, weight loss, melena or mucocutaneous bleeding manifestations. She had never smoked or consumed alcohol and had no history of other chronic illnesses. She was a housewife with no known exposure to medications, chemicals or toxins.

On examination, her BMI was 28 kg/m² and her temperature was 98 °F. She was pale and had mild pedal edema, but there was no icterus, clubbing, or lymphadenopathy. No petechiae or bone tenderness were noted. Abdominal examination revealed mild splenomegaly, while chest, cardiovascular, and neurological examinations were unremarkable.

Laboratory investigations (Table 1) revealed severe pancytopenia (Hb 6.3 g/dL, WBC 1,980/mm³, platelets 10,000/mm³), and the peripheral smear(Figure 1) confirmed leukopenia and thrombocytopenia.

Ultrasonography of the abdomen demonstrated cirrhosis with mild splenomegaly (14 cm), a portal vein diameter of 14 mm, and reduced Doppler flow suggestive of portal hypertension. Upper GI endoscopy revealed small oesophageal varices with evidence of portal hypertensive gastropathy.

Given the severity of pancytopenia and negative results from other investigations, bone marrow aspiration was performed, yielding a dry tap, and the subsequent biopsy (Figure 2) showed hypocellular marrow consistent with aplastic anemia. The diagnosis of compensated cirrhosis with aplastic anemia was made.

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Table 1: Laboratory Investigations

Test	Result
Hemoglobin	6.3 g/dL (11-17g/dl)
Total Leukocyte Count	980/mm³ (3000 – 15000/mm³)
Neutrophil Count	970/mm³ (1500 – 7000/mm³)
Platelet Count	10,000/mm³ (150x103 – 400 x103/mm³)
Reticulocyte Count	0.9%
MCV	90.7 fL (80 - 100 fL)
Peripheral Smear	Normocytic, normochromic RBCs, moderate leukocytopenia, severe thrombocytopenia
ESR	135 mm in first hour (0 – 20 mm in first hour)
CRP	34.7 mg/L (0 – 10 mg/L)
PT / INR	11.8 sec / 1.21 (8 – 12 sec / 0.8 – 1.2)
aPTT	21 sec (20 – 28 sec)
Total Bilirubin	29 µmol/L (0 – 17.1 µmol/L)
Direct Bilirubin	13.4 µmol/L (0 – 3 µmol/L)
AST	52 U/L (15 - 37 U/L)
ALT	40 U/L (16 - 63 U/L)
ALP	154 U/L (46 - 116 U/L)
LDH	291 U/L (120 - 246 U/L)
Albumin	23 g/L (34 – 50 g/L)
Globulin	42 g/L (22 – 48 g/L)
Creatinine	60 µmol/L (62 -115 µmol/L)
Serum Ferritin	574 ng/mL (15 – 150 ng/mL)
TSH	1.54 mIU/L (0.46 – 4.10 mIU/L)
HIV, VDRL, HBsAg, HCV Antibodies	Negative
ANA	Negative
Coombs Test	Negative
Urine Analysis	WBC 5-10/hpf, RBCs present
Blood Culture	No growth

The patient received packed red cell and platelet transfusions, after which hemoglobin improved to 7.2 g/dL and platelets to 20,000/mm³. Two doses of filgrastim (granulocyte colony-stimulating factor) were administered. Cirrhosis and portal hypertension were managed symptomatically with diuretics, beta-blockers, and supportive therapy. The patient was subsequently followed up by a hematologist for further management of aplastic anemia, in addition to ongoing hepatology clinic follow-up.

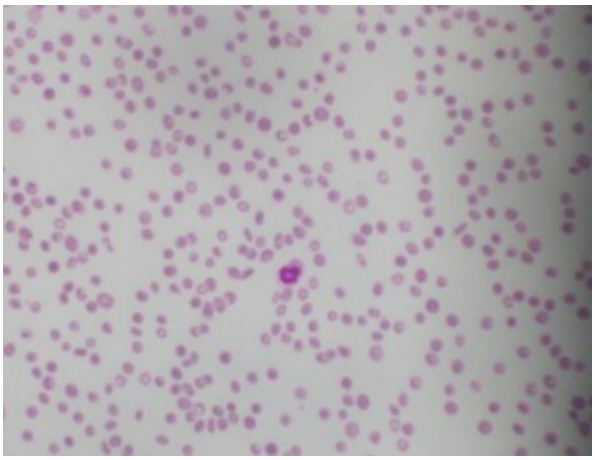

Figure 1 - Blood picture shows leucopenia and thrombocytopenia

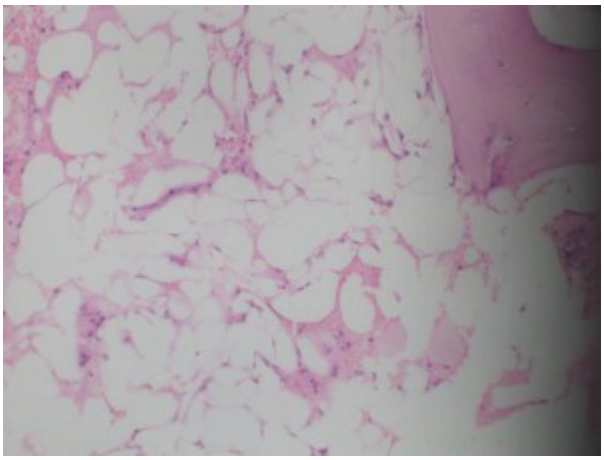

Figure 2 - Bone marrow biopsy reveals hypocellular marrow

DISCUSSION

This patient presented with pancytopenia in the context of compensated cirrhosis with portal hypertension. Although hypersplenism was initially considered, the severity of cytopenias along with only mild splenomegaly prompted further evaluation. Additionally, a low reticulocyte count despite adequate iron stores suggested impaired marrow production, which was subsequently confirmed as aplastic anemia on biopsy.

Hepatitis-associated aplastic anemia (HAAA) is a rare but severe complication in which an episode of acute hepatitis precedes aplastic anemia by several weeks to months.(1,2) It is most often seen in children and young adults.

The proposed mechanism involves an aberrant immune response in which cytotoxic $CD8^+$ T lymphocytes, initially directed against infected hepatocytes, cross-react with hematopoietic stem and progenitor cells. However, in this case, all major hepatotropic viruses were seronegative, and liver enzyme elevations were insufficient to support HAAA(2). Drug-induced aplastic anemia in chronic liver disease may result from impaired hepatic detoxification leading to accumulation of toxic metabolites and immune-mediated marrow injury(5-11).

Certain drugs can act as haptens, provoking a T-cell-mediated autoimmune response against hematopoietic stem cells through cytokines such as interferon-gamma and TNF-α. In this patient, there was no history of exposure to drugs, toxins, or herbal remedies.(11)

Alcohol-related marrow suppression is another possible mechanism, as ethanol and acetaldehyde are toxic to hematopoietic stem cells(5). Chronic alcohol use can induce folate and vitamin B12 deficiency, copper deficiency, and protein–calorie malnutrition, all of which impair hematopoiesis. It may also dysregulate immune function, promoting cytokine-mediated marrow injury. However, this patient was not an alcohol consumer, excluding this cause.

Autoimmune hepatitis–associated aplastic anemia has been reported, mediated by T-cell–driven destruction of both hepatocytes and hematopoietic cells. In this patient, ANA was negative and liver enzymes were not elevated, making autoimmune hepatitis unlikely(3,4,6).

Therefore, this case likely represents idiopathic immune-mediated aplastic anemia coexisting with MASLD-related cirrhosis. It highlights the importance of a comprehensive evaluation for alternative causes of pancytopenia in cirrhotic patients, particularly when cytopenias are severe and marrow response is inadequate.

CONCLUSION

Aplastic anemia in patients with chronic liver disease is uncommon, but should be considered when pancytopenia is severe and unexplained, or when hematologic recovery is inadequate. Bone marrow evaluation is crucial for establishing the diagnosis, and early recognition allows timely management and appropriate specialist referral.

REFERENCES

1. Young NS. Aplastic anemia. N Engl J Med. 2018;379(17):1643-1656.

2. Brown KE, Tisdale J, Barrett AJ, Dunbar CE, Young NS. Hepatitis-associated aplastic anemia. N Engl J Med. 1997;336(15):1059-1064

3. Oude Elferink RPJ, et al. Aplastic anemia in autoimmune hepatitis: coincidence or causal relationship? J Hepatol. 1992;14(2-3):316-318.

4. Chan YL, et al. Aplastic anaemia associated with autoimmune hepatitis. Clin Lab Haematol. 1997;19(1):27–29.

5. Qureshi K, Sarwar U, Khallafi H. Severe aplastic anemia after toxic liver injury: Case report and literature review. Case Reports Hepatol. 2014;2014:216570.

6. Cariani E, Pelizzari AM, Rodella A, Gargiulo F, Imberti L, Manca N, et al Immune-mediated hepatitis-associated aplastic anemia caused by the emergence of a mutant hepatitis B virus

7. Sun L, Zhang JC. Acute fulminant hepatitis with bone marrow failure in an adult due to parvovirus B19 infection

8. Kindmark CO, Sjölin J, Nordlinder H, et al. Aplastic anaemia in a case of hepatitis B with a high titer of hepatitis B antigen

9. Jayaweera M, Kodikaraarachchi W, Kalambarachchi H. Hepatitis-associated severe aplastic anaemia of unknown aetiology: a case report. Asian J Case Rep Med Health. 2022;5(1):110-116.

10.Vangipuram D, Singh S, Varadan S, Jayachandran D. Aplastic anaemia in a patient with chronic liver disease. J Sci Soc. 2017;44(1):55-57.

11.Ioannou S, Hatzis G, Vlahadami I, Voulgarelis M. Aplastic anaemia associated with interferon alpha-2a in a patient with chronic hepatitis C virus infection: a case report. J Med Case Rep. 2010;4:268.

CASE REPORT

EUS Guided Coil Embolization of Refractory Gastric Varices: First Case Report from Sri Lanka

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ABSTRACT

Bleeding from gastric varices (GV) carries high morbidity and mortality. Current endoscopic therapies are premised on cyanoacrylate (CYA) injection which is technically challenging with risks of embolization and possible only for intraluminal varices. We present the first case in Sri Lanka of a EUS-guided coilembolization of a refractory gastric varices. A 42-year-old female, experienced recurrent upper gastrointestinal bleeding due to portal hypertension after Whipple's surgery complicated by portal vein injury.

Post operative computed tomography (CT) of the abdomen showed portomesenteric venous occlusion. Patient needed repeated blood transfusions due to low hemoglobin and melena. Endoscopy only showed esophageal varices and few prominent gastric fundal veins.

Despite the placement of a splenorenal femoral vein interposition graft shunt patient continued to bleed needing admissions almost weekly. EUS-guided coil embolization of GV was undertaken. Initially the gastric fundal veins were identified sub-cutaneously, and the afferent vessels identified.

This vessel was packed with 02 coils and 2ml glue injected within the coils. Subsequently, she remained asymptomatic without needing further blood transfusions during the past 12 months. EUS-guided coil embolization of GV is an effective treatment modality for managing refractory GV especially when the bleeding GV are not seen within the lumen.

Key words: Gastric varices, Coil embolization, Portal Hypertension

INTRODUCTION

Gastric varices (GV) are present in approximately 20% of patients with cirrhosis and portal hypertension, typically identified during endoscopy.(1)Although GV bleed less frequently than esophageal varices (EV), when rupture occurs, it often results in more severe hemorrhage, higher mortality, and an increased risk of rebleeding following spontaneous hemostasis. (2)Historically, treatment options for bleeding GV have been limited. Even after emergency endoscopic interventions, such as glue injection, patients remain at significant risk of rebleeding and mortality.(3)

While endoscopic band ligation and sclerotherapy are well-established treatments for EV bleeding, they are less effective for fundal variceal hemorrhage. Endoscopic cyanoacrylate(CYA) injection is considered a first-line therapy when feasible, and more recently, EUS-guided angiotherapy has shown promise in managing GV bleeding.(4)The use of EUS has expanded both diagnostic and therapeutic capabilities in the management of GV, offering new treatment avenues previously unavailable.(5)EUS-guided treatments, including CYA injection and coil embolization, have been particularly effective in targeting localized gastric varices.

Among these, EUS-guided coil embolization has demonstrated advantages, such as requiring fewer procedures and presenting a lower risk of complications compared to direct CYA injection.(6)

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Although EUS-guided coil embolization has been used successfully in other countries, it had never been preformed in Sri Lanka until now. Here, we present the first successful case of EUS-guided coil embolization for the treatment of refractory GVin Sri Lanka.

CASE REPORT

A 42-year-old female (ASA II) presented with recurrent upper gastrointestinal (GI) bleeding secondary to portal hypertension, which developed following a Whipple procedure performed 10 years earlier for a solid pseudopapillary neoplasm of the pancreatic head. During the surgery, a portal vein injury was identified and repaired intraoperatively. Postoperative surveillance included regular upper GI endoscopy (UGIE) due to the risk of portal hypertension.

Although the patient remained asymptomatic during the postoperative first year, a follow-up CT scan of the abdomen revealed portomesenteric venous occlusion and portal hypertension. Endoscopic evaluation confirmed the presence of esophageal varices. Since then, the patient was managed conservatively with medical therapy aimed at controlling portal hypertension and underwentendoscopic band ligation of the esophageal varices. She remained clinically stable and asymptomatic until the first episode of melena occurred six years post-surgery.

The patient presented with melena and hemoglobin of 4g/dl, prompting further investigation for upper GI bleeding. Initial UGIE identified esophageal varices and several prominent gastric cardia and body veins, but no active bleeding was detected. As a definite site of bleeding was not identified on the UGIE, a Capsule endoscopy was done which also did not identifya bleeding source but revealed a possible anastomotic stricture with ulceration.

A CT mesenteric angiogram revealed multiple varices at the gastroesophageal junction, gastric fundus, and splenic hilum. During angiography, a contrast blush was observed at the 4th jejunal branch of the superior mesenteric artery (SMA), and superselective gelfoam embolization was successfully performed. Both the celiac and inferior mesenteric artery (IMA) angiograms were normal. Post-procedural angiography confirmed successful embolization, and the patient remained asymptomatic for the next two years.

Two years later, she experienced recurrent hematemesis and melena, necessitating repeated blood transfusions. Despite initial resuscitation and transfusion of crossmatched blood products, endoscopic evaluations failed to identify the source of bleeding.

A repeat contrast-enhanced CT mesenteric angiogram demonstrated cavernous transformation of the portal vein, portal hypertension, and edematous bowel loops. Given the patient's suboptimal response to medical management for portal hypertension, a surgical intervention was pursued.

A splenorenal shunt was performed using a femoral vein interposition graft to reduce portal hypertension. Despite this, recurrent GI bleeding persisted due to shunt failure, which was confirmed on subsequent imaging. Over the next 12 months she presented with repeated episodes of upper GI Bleeding, with negative endoscopy and CT mesenteric angiograms, necessitating repeated blood transfusions.

At this point although a gastric variceal bleed was not seen, due to lack of any further treatment options, an endoscopic ultrasound assessment was done of the stomach by the third author (NF), which confirmed large submucosal GV in the body. Following hemoglobin optimization EUS-guided coil embolization of GV was performed. (Figure 1 & 2)

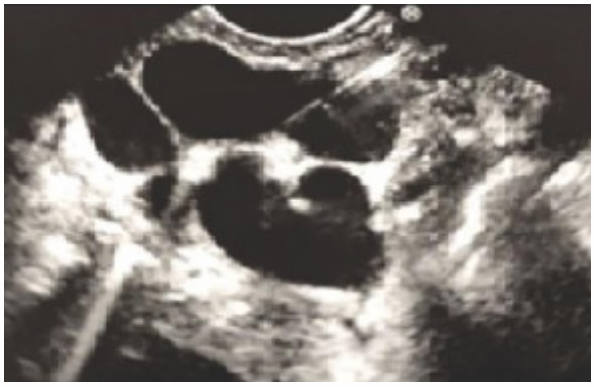

Figure -1 - EUS image showing gastric varices

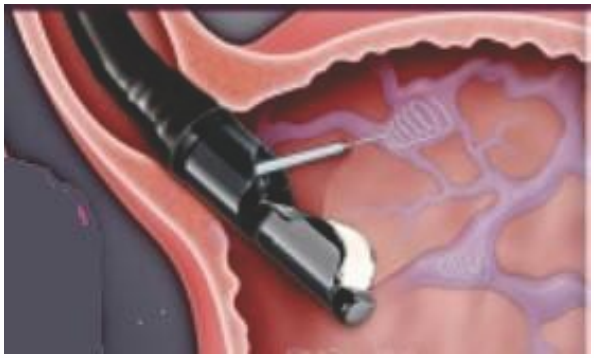

Figure - 2 - Image demonstrationg the techcnique of EUS guided coil embolisation

The gastric cardia veins were first identified submucosally, followed by localization of the afferent vessels. Two neurosurgical coils (10mm x 2) were placed within the target vessel, and 2 ml of glue injected into the coils. The patient has since remained asymptomatic and has not required any blood transfusions over the following 18 months.

DISCUSSION

GV are a serious complication of portal hypertension, often presenting management challenges, particularly when refractory to conventional treatments. This case demonstrates the complexity of GV in a patient with portal hypertension following a Whipple procedure and portal vein injury. Despite initial success with endoscopic band ligation for esophageal varices and the attempted creation of a splenorenal shunt, the patient experienced recurrent gastrointestinal bleeding, underscoring the limitations of conventional approaches.

EUS-guided interventions, particularly coil embolization, have emerged as an effective alternative for managing GV, especially when they are not seen intra luminally. Unlike EV, fundal varices cannot be managed by standard endoscopic techniques like band ligation or sclerotherapy, and need glue injection with CYA. Direct glue injection to varices can be done only if the varices are seen intra luminally. It also carries a risk of glue embolization and causing ischemic strokes.(7)

EUS provides a distinct advantage by enabling detailed visualization and precise targeting of afferent vessels feeding the varices. In this case, EUS-guided coil embolization was chosen after previous interventions failed to control bleeding, and the patient experienced recurrent episodes of bleeding requiring blood transfusions.

Coil embolization, combined with glue injection, has demonstrated better outcomes by preventing immediate rebleeding and reducing the need for repeated procedures.(8) The success of this approach in this patient, who remained asymptomatic for over 18 months, highlights its potential as a first-line therapy for refractory GV. The precision of EUS-guided coil embolization in targeting vascular structures, especially in a setting complicated by cavernous transformation of the portal vein, was critical to the favorable outcome.

This case also represents the first successful use of EUS-guided coil embolization for GV in Sri Lanka, marking an important milestone in the management of GV in a resource-limited settings. Although this procedure was effective, long-term surveillance remains essential due to the dynamic nature of portal hypertension.

CONCLUSION

EUS-guided coil embolization of GV is indeed an effective treatment option for managing refractory GV, particularly in situations where the varices are difficult to visualize endoscopically or are actively bleeding into the lumen obscuring the varices.

REFERENCES

1. Sarin SK, Lahoti D, Saxena SP, Murthy NS, Makwana UK. Prevalence, classification and natural history of gastric varices: A long-term follow-up study in 568 portal hypertension patients. Hepatology. 1992;16(6).

2. Tripathi D, Stanley AJ, Hayes PC, Patch D, Millson C, Mehrzad H, et al. U.K. guidelines on the management of variceal haemorrhage in cirrhotic patients. Gut [Internet]. 2015 Nov 1 [cited 2024 Sep 24];64(11): 1680–704. Available from: https://pubmed.ncbi.nlm.nih.gov/25887380/

3. Augustin S, Altamirano J, González A, Dot J, Abu-Suboh M, Armengol JR, et al. Effectiveness of combined pharmacologic and ligation therapy in high-risk patients with acute esophageal variceal bleeding. Am J Gastroenterol [Internet]. 2011 Oct [cited 2024 Sep 24];106(10):1787–95. Available from: https://pubmed.ncbi.nlm.nih.gov/21625271/

4. Hamerski C, Binmoeller KF, Shah JN. Hemostasis of Acute Gastric Variceal Bleeding. GI Endoscopic Emergencies [Internet]. 2016 Jan 1 [cited 2024 Sep 24];181–93. Available from: https://link.springer.com/chapter/10.1007/978-1-4939-3085-2_13

5. Zhang W, Peng C, Zhang S, Huang S, Shen S, Xu G, et al. EUS-guided portal pressure gradient measurement in patients with acute or subacute portal hypertension. Gastrointest Endosc [Internet]. 2021 Mar 1 [cited 2024 Sep 24];93(3):565–72. Available from: https://pubmed.ncbi.nlm.nih.gov/32615178/

6. Romero-Castro R, Ellrichmann M, Ortiz-Moyano C, Subtil-Inigo JC, Junquera-Florez F, Gornals JB, et al. EUS-guided coil versus cyanoacrylate therapy for the treatment of gastric varices: A multicenter study (with videos). Gastrointest Endosc. 2013;78(5).

7. Chandra S, Holm A, El Abiad RG, Gerke H. Endoscopic Cyanoacrylate Glue Injection in Management of Gastric Variceal Bleeding: US Tertiary Care Center Experience. J Clin Exp Hepatol. 2018 Jun 1;8(2):181–7.

8. Chen D, Fu S, Shen R. Efficacy and safety of EUS-guided coil embolization in combination with cyanoacrylate injection versus conventional endoscopic cyanoacrylate injection in the treatment of gastric varices with spontaneous portosystemic shunts. Gastroenterol Rep (Oxf) [Internet]. 2024 [cited 2024 Sep 25];12. Available from: https://pubmed.ncbi.nlm.nih.gov/38586537/