Review Article

The Role of Interventional Radiology in Esophageal Varices and Hematemesis: Review Article

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Abstract

Esophageal varices and hematemesis in severe cases can lead to significant morbidity and mortality without correct treatment. Esophageal varices and hematemesis can be brought on by a variety of causes, including infection, vascular abnormalities, inflammatory illnesses, trauma, and cancer. Its location, severity, and causes all influence how it is diagnosed, managed, and treated. The field of radiology especially interventional radiology offers quick, efficient, and successful endovascular therapeutic procedures as well as diagnostic imaging tests. Nuclear scintigraphy, computed tomography angiography, and endovascular angiography can all help to pinpoint the cause of bleeding and give the interventional radiologist important information for managing the case. Examples of minimally invasive procedures that have been suggested for the treatment of variceal bleeding include endoscopic sclerotherapy (ES), variceal ligation (EVL), transjugular intrahepatic portosystemic shunt (TIPS), percutaneous trans-hepatic embolization of gastroesophageal varices (PEGV), and other combined therapies. TIPS had a significant drawback in that it might aggravate liver failure and roughly increase the likelihood of encephalopathy while being quite successful in decreasing portal pressure and preventing rebleeding.

Keywords: Esophageal varices, Hematemesis, Radiology, Interventional radiology, Bleeding, Portal pressure

Introduction

The rise of interventional radiology's (IR) function as a substitute for medical, endoscopic, or surgical procedures is due to IR's minimally invasive character, which is backed by advances in imaging and advancements in endovascular techniques. Several medical, oncological, and surgical practice guidelines presently include IR procedures; nevertheless, the strength of the recommendations is primarily based on low-quality research that lacks long-term outcomes and comparisons to alternative treatments [1].

When it comes to esophageal varices, the dilated submucosal distal esophageal veins that join the portal and systemic circulations are known as esophageal varices. This is brought on by increased portal venous blood influx, resistance to portal blood flow, and portal hypertension, which is most frequently caused by cirrhosis. Variceal rupture is the most common fatal cirrhosis complication; the likelihood of bleeding and the existence of varices are correlated with the degree of liver disease [2-5].

Blood circulates through the portal vein at a rate of over 1500 ml/min, and if there is an obstruction, the portal venous pressure rises as a result. Collaterals emerge as the body's reaction to the elevated venous pressure. These portosystemic collaterals direct blood to the inferior and superior vena cava from the portal venous system, sometimes known as uphill varices, this term describes the upward blood flow to the superior vena cava. Downhill varices are situated in the proximal esophagus and have a retrograde blood flow as opposed to uphill varices. Downhill varices are uncommon and typically brought on by mediastinal malignancies, bronchogenic carcinoma, and other superior vena cava obstructions [6, 7]. The gastroesophageal collaterals, which drain into the azygos vein and cause esophageal varices, are another crucial system operating concurrently. These varices can expand to the point where they rupture, resulting in serious bleeding. After duodenal and gastric ulcers, esophageal varices are the third most frequent cause of upper GI hemorrhage.

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Portal hypertension causes include: (A) Prehepatic: Massive splenomegaly with increased splenic vein blood flow or portal vein obstruction (EHPVO), (B) Constrictive pericarditis, severe right-sided heart failure, and hepatic vein obstruction are all posthepatic symptoms (Budd-Chiari syndrome), (C) Intrahepatic: The majority of portal hypertension cases are caused by cirrhosis. Schistosomiasis, significant fatty change, disorders affecting the portal microcirculation, such as nodular regenerative hyperplasia, and diffuse fibrosing granulomatous disease, such as sarcoidosis, are less common causes. Among the more unusual causes of portal hypertension are Wilson disease, alpha-1 antitrypsin deficiency, primary biliary cirrhosis, tuberculosis, and constrictive pericarditis. Interventional radiology as percutaneous transhepatic embolization is used in managing esophageal varices and hematemesis [8]. An interprofessional team composed of a gastroenterologist, internist, surgeon, invasive radiologist, and intensivist manages esophageal varices.

Study Objective

The study aims to summarize current evidence regarding the role of interventional radiology in esophageal varices and hematemesis.

Materials and Methods

Study Design

Integrative Literature Review (ILR).

Study Duration

Data was collected during the period from 1– 29 May 2022.

ILR is a technique for compiling previously published studies to synthesize suggestions on a subject; it is frequently utilized in the health disciplines to identify novelties and then discover new approaches to health services, enabling the utilization of evidence-based health care, guaranteeing high-quality services, and enhancing patient welfare and safety. Six sequential stages require to be followed: Explanation of the study issue; inclusion and exclusion criteria; sample definition; evaluation of included studies; findings interpretation; and presentation of the ILR synthesis

The papers that had the same goal as our study was reviewed in depth after searching and defining the sample.

Due to their reputation as reliable databases, PubMed and EBSCO Information Services were preferred as the exploration databases for the papers utilized in the study. One of the biggest online digital libraries, PubMed was created by the National Center for Biotechnology Information (NCBI), a division of the National Library of Medicine of the United States. The article was created using subjects relating to the role of interventional radiology in esophageal varices and hematemesis. The topics and summaries of the established papers were scrutinized.

The subjects were selected for addition founded on their applicability to the research, which must have at least one of the following subjects; esophageal varices, hematemesis, radiology, interventional radiology, bleeding, and portal pressure.

Exclusion Criteria

All additional papers, recurring research, and reviews of research that do not possess one of these themes as their major end were disregarded.

Analysis of Statistics

The data didn't be analyzed by any program. The information was taken from a specified form that included the research topic, author's designation, aim, executive summary, results, conclusions, and outcomes. To guarantee rationality in addition to reducing errors, the results of each affiliate were double-revised.

To ensure that the research we contained within the study applies to the goal line of our study and to avert or diminish slips in the results, studies were double-reviewed during the article selection process.

Esophageal Varices and Variceal Bleeding Epidemiology

Incidence of varices and bleeding, varices affect 30% of cirrhotic patients at diagnosis, but 90% of them do so after 10 years, whereas 5% of tiny varices and 15% of large varices experience their first variceal bleeding within a year. Males experience it more frequently than females do. Esophageal varices are associated with a 50% chance of bleeding in patients. In the six weeks after an incident of variceal hemorrhage, fatality rates range from 10% to 20% [9]. Alcohol and viral hepatitis are the two main causes of portal hypertension in the West. Schistosomiasis and hepatitis B/C are the most prevalent causes of portal hypertension in Asia and Africa.

Pathophysiology

Portocaval anastomosis forms as a result of portal hypertension to decompress portal circulation. In the absence of a portal obstruction, the pressure may reach as high as 15-20 mmHg, which is higher than the normal range of 5–10 mmHg. Due to the absence of valves in the portal venous system, any level of resistance between the splanchnic arteries and the right side of the heart causes retrograde flow and increased pressure. The collaterals gradually grow and join the portal venous system to the systemic circulation. As a result, the distal esophagus develops a congested submucosal venous plexus with tortuous dilated veins over time. Hemorrhage results from variceal rupture [9].

Intrahepatic vasoconstriction due to decreased nitric oxide generation and increased release of endothelin-1 (ET-1), angiotensinogen, and eicosanoids and disruption of blood flow via sinusoidal remodeling are the main contributors to

increased portal flow resistance at the level of the hepatic sinusoids.

Prognosis and Complications

A patient's chance of experiencing another variceal bleeding episode after the first one is 70%. At least 30% of rebleeding events result in death. Most fatalities take place within the first several days following the bleed. Acute variceal hemorrhage and surgical intervention both increase the risk of death. Complications of variceal bleeding include Aspiration, multiorgan failure, encephalopathy, perforation of the esophagus, and death [9].

Management

It's possible that video capsule endoscopy screening will replace traditional endoscopy as a diagnostic procedure.

Firstly, we treat the comorbidities caused by cirrhosis [10-13]. Variceal hemorrhage is frequently complicated by hepatic encephalopathy and infection. Treating active bleeding is done through Intravenous (IV) access, and hemodynamic resuscitation; Treat coagulopathy as indicated; Record mental status, and forbid any nephrotoxic drugs; To decrease portal venous pressure give IV octreotide; Before endoscopy, give erythromycin 250 mg IV; For diagnosis and treatment, immediate upper GI endoscopy [14].

For medium- to large-sized nonbleeding varices as well as bleeding varices, variceal band ligation is recommended to sclerotherapy to reduce the risk of bleeding. Ligation had a better rate of variceal eradication, lower rates of rebleeding, fewer complications, and faster bleeding cessation [15].

Consider using peroral insertion of a Sengstaken-Blakemore-type tube for up to 24 hours to stabilize the patient for TIPS if endoscopic treatment is unsuccessful. Avoid beta-blockers when there is active bleeding since they lower blood pressure and reduce the physiological increase in heart rate that occurs during acute hemorrhage [16].

However, to prevent acute rebleeding the following is recommended: using vasoconstrictors to decrease portal pressure, endoscopic band ligation (EBL), and TIPS (transjugular intrahepatic portosystemic shunt) to lower portal pressure by establishing contact between a branch of the intrahepatic portal vein and the hepatic vein. TIPS should be referred for endoscopy, liver transplantation, and interventional radiology.

Concerning radiology, to stop variceal hemorrhage, percutaneous transhepatic embolization has been used. Its efficacy, however, is still debatable. Patients who are not candidates for surgery typically use it.

A salvage treatment called TIPS is used to stop acute variceal bleeding. However, the surgery is also linked to major side effects including encephalopathy and shunt blockage within a year. A liver transplant may be facilitated by TIPS [9].

Interventional Radiology in Disease Management

Endoscopic sclerotherapy (ES), variceal ligation (EVL), transjugular intrahepatic portosystemic shunt (TIPS), percutaneous trans-hepatic embolization of gastroesophageal varices (PEGV), and other combined therapies are examples of minimally invasive procedures that have been recommended for the treatment of variceal bleeding. By removing the varices, endoscopic therapies are successful in reducing variceal bleeding. However, they are ineffective in treating portal hypertension or other consequences, and the rate of rebleeding remains quite high despite treatment. Although TIPS was very effective in lowering portal pressure and reducing rebleeding, it had a major flaw in that it could exacerbate liver failure and nearly double the chance of encephalopathy [17-23].

TIPS is therefore suggested as a rescue therapy rather than as the first line of defense against rebleeding. Percutaneous splenic embolization (PSE) was beneficial for improving liver function and portal hypertension in addition to treating hypersplenism, but it was less successful than TIPS at lowering portal hypertension. In 70 to 90 percent of these individuals, PEGV was successful in reducing hemorrhage. However, PEGV had little effect on the underlying portal hypertension or hepatic insufficiency, and after a few months of embolization, 35 to 65 percent of patients experienced recurrent bleeding [24, 25]. Hypersplenism is also unaffected meantime. Based on the theory that combining PEGV and PSE should cure both portal hypertension and hypersplenism, preventing the rebleeding of esophageal varices, we coupled PEGV with PSE to treat patients with variceal bleeding history and hypersplenism in this investigation. It is yet unknown, though, whether PEGV and PSE together are efficient [26].

Conclusion

Surgery, interventional radiology, and endoscopic treatment are examples of pharmacological treatment options for portal hypertension with variceal hemorrhage. Many surgical techniques, including shunting and nonshunting techniques, have been developed. The limitations of these procedures are primarily their invasiveness and post-procedure complications, which include a high incidence of portal thrombosis, serious gastric mucosa damage and a delay in gastric emptying, encephalopathy, and worsening liver function. These procedures are very effective at relieving portal vein pressure and preventing rebleeding.

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