Case Report

Variceal Banding: Experience with Two Cases in NAUTH, Nnewi – A Resource-Limited Environment

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Abstract

Esophageal varices can present with life-threatening hemorrhage and can be managed by surgery or medically. Banding is an endoscopic treatment for variceal bleeding and is a known management option for variceal bleeding but is not widely available in Nigeria due to lack of expertise and equipment. Previously, patients relied on medical management and surgery for treatment; however, these were not effective as mortality rates were high. Few centers in the country are known to offer this procedure to their patients and none has been reported in Southeast Nigeria, hence the justification for reporting our experience managing two cases of bleeding esophageal varices. We report two cases of variceal bleeding on the background of decompensated liver cirrhosis of alcohol and viral etiology, respectively. The first patient was on propranolol, but it was not effective in preventing variceal bleeding. The second patient was not on prophylaxis. Both the patients had variceal banding in NAUTH, Nnewi, and no complications were reported. Variceal banding is a safe procedure and is an effective measure in control of bleeding varices.

Keywords: Banding, bleeding, treatment, variceal

Introduction

Variceal banding is one of the endoscopic methods employed in the treatment of variceal bleeding.[1] Variceal bleeding is one of the manifestations of portal hypertension occurring usually in the setting of decompensated liver cirrhosis of varying etiology.[2] Varices arise due to dilatation of the submucosal venous plexus resulting in elevated intravariceal pressure and wall tension. Portal hypertension is associated with both increased portal inflow and increased outflow resistance. Hepatic venous catheterization is the most common technique to determine the portal pressure. Wedged hepatic venous pressure (WHVP) reflects sinusoidal pressures, and hepatic venous pressure gradient (HVPG) is the difference between WHVP and free hepatic venous pressure and is a good predictor of the severity of portal hypertension. The normal HVPG is usually between 5 and 10 mmHg. The risk of developing esophageal varices increases when HVPG reaches a minimum pressure of 10–12 mmHg.[3] Variceal bleeding typically manifests as vomiting of fresh blood or coffee grounds and it is usually vigorous and difficult to control.[1] It is a life-threatening condition and results in high rates of mortality. The mortality associated with variceal bleeding is about 30% and is higher in severe liver disease (Child–Pugh C).[1] Mortality occurs due to the fact that patients with liver disease have background clotting disorder, poor nutrition, and increased risk for hepatic encephalopathy.[2] Following the initial episode of bleeding, the incidence of re-bleeding is up to 70% and usually occurs within 6 weeks of the initial bleeding.[4]

Variceal banding is usually employed in the emergency management of variceal bleeding as well as in both primary prophylaxis and secondary prophylaxis to prevent variceal bleeding. The complications are rare and include bleeding, ulcers, and rarely, obstruction.[3]

There is little in literature about variceal banding in Nigeria, especially in the Southeast; however, it is known that few

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centers have both the workforce, expertise, and equipments needed to offer this procedure and NAUTH offers the procedure. We report two cases of variceal banding done in NAUTH, Nnewi, between February 2019 and June 2019.

Case Reports

Case 1

A 58-year-old man presented to the accident and emergency department with vomiting of blood of 24-h duration. Vomitus initially contained fresh and later altered blood clots. The estimated blood loss was about 850 ml. He also had a history of passage of melena and he had mild dizziness. He had been on management for decompensated alcoholic liver cirrhosis for about 11 months and had been on low-dose propranolol (20 mg daily) because of bradycardia. There was no history suggestive of cardiac disease, recent use of nonsteroidal anti-inflammatory drugs (NSAIDS), and alcohol or herbal use. He is not a known peptic ulcer disease patient.

The patient was anxious, afebrile, pale, dehydrated with palmer erythema and bilateral gynecomastia. He had no leg edema or other stigmata of chronic liver disease. The abdomen was distended with an enlarged liver of 5 cm below the subcostal margin, firm, smooth, nontender with a blunt edge. The liver span was 16 cm. The spleen was not enlarged and the kidneys were not palpable. There were mild ascites and normal bowel sounds.

Digital rectal examination revealed melena and no other significant findings.

Urgent packed cell volume (PCV) was 28%. He was negative to hepatitis B, hepatitis C and HIV. His abdominal ultrasound revealed an enlarged liver with micronodular outline. His liver function test, serum electrolytes , urea and creatinine were essentially normal. He had reduced protein and albumin levels.

His Child–Pugh score was 8 (Class B).

He was stabilized using intravenous terlipressin, blood transfusion, intravenous fluids, and antibiotics and was placed on nil per oral.

Forty-eight hour later, he was taken for upper gastrointestinal (GI) endoscopy which revealed large varices involving the lower and middle third of the esophagus (Grade 3). He subsequently had variceal banding and discharged. He subsequently had two repeated banding sessions at 4-week interval. He has been having variceal banding although irregularly due to financial and logistic reasons for 1 year now, he has not bled since.

At present, the patient is stable and has been regular with his clinic visits. He is currently on tablets propranolol 20 mg nocte, tablets rabeprazole 20 mg bd, tablets eplerenone 50 mg daily, capsules livolin forte 1 bd, and tablets thiamine 100 mg bd.

Case 2

A 56-year-old man was referred to the accident and emergency department with 5-h history of vomiting of blood and passage of blood in the stool. There was no history suggestive of hemodynamic compromise. He was not a known peptic ulcer disease patient; he had no history of ingestion of NSAIDs, no significant history of alcohol intake. He was being managed in a peripheral hospital for chronic liver disease.

He was chronically ill-looking, pale, anicteric, dehydrated, with digital clubbing and loss of small muscles of the hand. He had bilateral pitting leg edema up to the mid leg.

The abdomen was distended with a shrunken liver (7 cm). The spleen was about 4 cm enlarged and the kidneys were not ballotable. Ascites was demonstrated by shifting dullness. Bowel sounds were normoactive.

Digital rectal examination revealed melena and no other significant findings.

Investigation results showed a PCV = 0.25%, hepatitis B surface antigen was reactive, full blood count showed absolute leukocytosis with neutrophilia, abdominal ultrasound revealed shrunken liver with moderate ascites. He had hypoproteinemia and hypoalbuminemia.

Other investigation results were not remarkable.

He was stabilized with intravenous fluids, blood transfusions, terlipressin, and intravenous antibiotics.

Subsequently, he had upper GI endoscopy which revealed columns of varices (Grade 3). He had a first session of variceal banding and is currently on follow-up and having variceal banding roughly every month and has been followed up to a year. His medications include tablets spironolactone, frusemide, rabeprazole, propranolol nocte, livolin forte, and tenofovir.

Discussion

Variceal band ligation is a recognized and safe method for the treatment of variceal bleeding.[1] Endoscopic variceal ligation is considered the first line of endoscopic treatment for the management of bleeding esophageal varices.[5] It has a better hemostasis, lower rate of side effects (ulcer and stricture), reduced rate of early re-bleeding, and a lower rate of early mortality compared to sclerotherapy.[5]

The goal of treatment for patients with esophageal varices is to reduce portal hypertension. Before the era of variceal banding, nonselective beta-blockers were essentially used for primary prophylaxis. This works by reducing the HVPG.[3] Nonselective beta-blockers such as propranolol are readily available and not expensive. Others approved include nadolol and carvedilol.[6] They are effective in primary prophylaxis due to their action on both beta-1 and 2 blockers. However, they have some contraindications which make them unsuitable for use in patients with asthma, chronic obstructive pulmonary disease, atrioventricular block, intermittent claudication, and psychosis. It also has some adverse effects such as light headedness, fatigue, dyspnea on exertion, bronchospasm, insomnia, impotence, and apathy.[7] The use of nonselective beta-blockers

is limited by the fact that portal pressure measurement which determines portal hypertension is invasive and cannot readily be measured leaving the use of 25% reduction in basal pulse rate as a fair guide to monitoring treatment. Even with treatment using beta-blockers, only 38% of patients respond, and there is no way to know who will respond unless HVPG measurement is done which is invasive.[8] In the event of acute variceal bleeding, Sengstaken–Blakemore tubes could be temporarily used to maintain hemostasis but not in severe cases due to the risk of aspiration, esophageal ulceration, and rupture. It cannot be left in place for more than 24 h.[9] The tube is also not routinely found.

Vasopressors came into use and in some cases, they were used as a stopgap until the patient was able to have endoscopic treatment. Vasopressors like terlipresssin have also been shown to improve mortality. In some other cases, patients had to undergo open surgery with various kinds of shunt surgery which were usually not effective and also resulted in high morbidity and mortality.[1]

Combination therapy with nonselective beta-blockers and endoscopic variceal ligation is the first line of treatment for secondary prophylaxis with a goal to eradicate varices and prevent recurrent bleeding.[10]

The first patient presented (case A) had been on low-dose propranolol since diagnosis; however, it was not able to prevent him from having variceal bleeding. This may be from inadequate dose or the appropriate dose could not be reached due to adverse effects of nonselective beta-blockers. Had he done endoscopy earlier for screening of varices, he would have been offered the choice of banding, thereby preventing the morbidity and mortality associated with variceal bleeding.

The second patient was previously managed in a peripheral hospital and was not placed on beta blockers, neither was he evaluated for hepatitis B. He was not screened for varices at the time of diagnosis, thereby predisposing him to variceal bleeding.

There were no complications noted during or after the procedure in the four sessions of variceal banding. All were done under mild sedation.

A study carried out in North Central Nigeria among patients who presented with upper GI bleeding showed that 53 of 83 patients (63.9%) had Grade 4 varices, 20 (24.1%) had Grade 3 varices, and 10 (12.0%) had Grade 2 varices. The high proportion of patients with Grade 4 varices suggests that it is the more common finding in patients with portal hypertension presenting with variceal bleeding. The study also revealed that there was no complication during all the sessions of variceal banding, further stressing the safety profile of the procedure.[11]

Other studies done elsewhere have also confirmed the safety profile of this procedure placing it higher than injection sclerotherapy, which is another form of endoscopic treatment available. Furthermore, far fewer sessions were required with banding to achieve variceal obliteration when compared with injection sclerotherapy.[12,13]

The endoscopic treatment options are becoming more widely available worldwide and are easy to perform with training and are safer for the patient. The two endoscopic treatment options available are injection sclerotherapy using a variety of agents that are injected either intravariceally or perivariceally and endoscopic variceal band ligation. Of the two methods, rubber band ligation is far safer since it is associated with lower re-bleeding rates and fewer procedure-related complications such as esophageal strictures and ulcers and required fewer sessions to achieve variceal obliteration.[14-16]

We therefore recommend routine screening for patients at risk of variceal hemorrhage such as decompensated liver cirrhosis patients quite early in the diagnosis and, if varices are found, advice for banding.

Conclusion

Patients who have portal hypertension are at risk of having variceal bleeding. There should be a regular screening of such patients. Variceal banding is a safe procedure and can be employed in patients with bleeding varices. This will help to reduce mortality associated with this condition.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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