Devascularization for Portal Hypertension not Obsolete: Technical Details and Experience of 104 Cases

Authors
Sadaf Ali*, Omar J Shah, K Sudarshan Reddy, Faroze Khan, Ajaz A Wani
Department of Surgical Gastroenterology, Sher I Kashmir Institute of Medical Sciences, Soura, Srinagar, Jammu and Kashmir, India, 190011
Corresponding Authors
Dr Sadaf Ali
Email: sadafskkims@gmail.com, +91-9797555599

Abstract
Background - Portal hypertension of various etiologies results in various complications, of which most common and dreaded complication is esophago-gastric varices and its bleed. Devascularisation with our modified technique has a vital role in the management of esophago-gastric vertices, it's not obsolete procedure.

Methods - A Prospective study of 104 patients underwent our modified Devascularization procedure over a period of 8 years from January 2006 to December 2014 in surgical gastroenterology department, SKIMS, INDIA. These patients had a follow up of 8 years to minimum of 1 year. The study patients were compiled in 2015.

Results - In our study with our Devascularisation technique, the operative time was 150 mins+/− 30 min, operative blood loss was 300 ml +/- 50 ml, during post operative follow up there was no esophageal leak or stenosis, no residual varices (0%) and noencephalopathy observed. In12 patients developed recurrent varices (12.53%), rebleeding occurred in 8(7.69%) patients and 9 patients were died (8.65%), all of them were Child C.

Conclusion - In failed medical and endoscopic management of esophagogastric varices our devascularisation technique has an important role, with less post operative morbidity and mortality and its role will only continue to evolve.

Keywords - Esophago-gastricvarices, our devascularisation technique, portosystemic shunts, recurrent varices, rebleed, encephalopathy, mortality.

Introduction
Normal hepatic venous pressure gradient (HVPG) is 3-5 mm Hg. HVPG 6 mm Hg or more is defined as portal hypertension(1). Varices generally will not develop at HVPG of< 10 mm Hg. However, once the HVPG rises to 12 mm Hg or greater complications can arise from portal hypertension of any cause in the form of varices leading to bleed and other issues of liver decompensation, hypersplenism, ascites and spontaneous bacterial peritonitis. Out of these clinical end results of portal hypertension bleed is commonest and usual cause of death(2). Causes of the portal hypertension are pre hepatic, hepatic and post
hepatic diseases\(^3\) of which cirrhosis, extra hepatic portal venous obstruction (EHPVO) and non cirrhotic portal fibrosis (NCPF) or idiopathic portal hypertension (IPH) are common causes. There are various treatments for esophago-gastric varices, such as endoscopic treatment, interventional radiology, and surgical procedure. Devascularization and shunting are the two known and accepted methods of surgical treatment for bleeding or post bleeding varices\(^4,5\). Liver transplantation is the curative treatment of chronic liver disease with portal hypertension and related complications\(^6-9\). Though China and Japan do have proponents of various devascularization options rest of the world is finding it more of an obsolete procedure in the era of endotherapy\(^{10,11,12}\). We here at a tertiary health care center in Kashmir valley of India come across management of all types of portal hypertension. Knowing and carrying the art of doing shunt surgery and devascularization procedures in patients with EHPVO, NCPF and early cirrhosis; we strongly believe that devascularization surgeries still has its own place and indication in management of portal hypertension. Authors share an experience of 104 cases of devascularization surgery done at our center.

**Material and Methods**

An experience of 104 cases of Devascularization done over a period of 8 years from January 2006 to December 2014 was evaluated for demographic details, indication, early and late outcome of surgery. These patients had a follow up of 8 years to minimum of 1 year. The study patients were compiled in 2015. Patients selected for Devascularization (inclusion criteria) were:

- Technical reasons of vessels unavailable for shunt in EHPVO, NCPF, Childs A/ early B cirrhosis
- In elderly comorobant patients with poor performance status where a prolonged shunt surgery wouldn’t be tolerated.
- Asan emergency procedure when medical and endoscopic treatment failed and TIPS had failed or not available.
- As an elective surgery 1) where failure of long term medical and endoscopic treatment (life-threatening rebleed on regular followup) 2) In patients noncompliance or inability to follow up for regular endoscopic variceal therapy.
- In bleeding fundal gastric varices where endoscopic treatment most of the times unsuccessful.
- In cirrhotic and NCPF patients required splenectomy for symptomatic hypersplenism, and also had high risk esophageal or gastroesophageal varices (primary prophylaxis).
- Cirrhotic patient with recurrent variceal bleed, who were candidate for liver transplantation (LT) – as long term bridge to LT.

Author would like to share the technical details of the modification of Devascularization being performed at our center.

**Technical details:** This is the single stage tranabdominal procedure is performed via a left subcostal or upper midline incision.

1. If severe hypersplenism is a concern then first step is splenic artery ligation (fig.1). We routinely perform a splenectomy first unless in set up of massive bleed while doing a resuscitative surgery where we ligate the left gastric vein first. Removal of the spleen allows for better exposure for paraesophageal gastric devascularization.
2. The proximal two third of the stomach is devascularized by ligating short gastric, retro gastric vessels and left gastric artery and veins (fig.2) These veins were ligated very close to the wall of the stomach, thereby saving the vagal innervations to the pylorus, obviating the need to perform a drainage procedure. Under running of fundal gastric varices done if present.
3. The left lobe of the liver is retracted and the oesophagogastric junction identified; Mobilization of the left lobe may be necessary at times. After division of the oesophagogastric reflection of peritoneum and mobilization, the distal 7 to 8 cm of esophagus is devascularized by ligating paraesophageal perforating veins (fig.3). Difficulty may be experienced with this mobilization in patients who had undergone injection sclerotherapy.

4. We usually preserve the vagus supply unless it’s necessary to sacrifice it.

5. Then interrupted transmural suturing of the GE junction is done over the guidance of Ryle’s tube placed early during surgery (fig.4). These sutures are taken 2 cm above the gastroesophageal junction circumferentially.

For last one decade we have been using this modifica-fied Sugiura’s procedure which obviating the complications of esophageal transaction and reanastomosis but simultaneously serving the purpose.

**Observation**

**Demographic & Disease Pattern**

One hundred and four cases of devascularization were performed over a period of eight years for portal hypertension of various etiologies. There was slight preponderance of female patient undergoing devascularization in our series (43 male and 61 females). As far as timing of surgery is concerned 58 patients had elective procedures and 46 patients underwent emergency devascularization after failed endoscopic intervention. The etiology of portal hypertension was Extra hepatic portal vein obstruction (EHPVO) in 50 patients, Non cirrhotic portal vein obstruction(NCPF) in 32 patients, Cirrhosis in 22 cases (Table 1).

**Table 1:** Etiology of portal hypertension in patients undergoing Devascularization

| Etiology   | No of patients |
|------------|----------------|
| EHPVO      | 50 (48.07%)    |
| NCPF       | 32 (30.76%)    |
| Cirrhosis  | 22 (21.15%)    |

**Preoperative Work Up**

Majority of the patients had significant Hypersplenism. Perioperative workup (Table: 2) revealed a deranged liver function (LFT) in 12 cases, an associated coagulopathy in 10 cases and significant hypersplenism in 66 cases. Upper GI Endoscopy was done in all patients revealed grade 1varices in 10 cases, grade 2varices in 42 patients and grade 3 varices in 40 patients and gastroesophageal varices in 12 patients. Sixty four slices CT portovenogram was done in most of the cases (93/104) except for few emergent patients. Most of the patients were of either Child A or B grade and only 13 patients were of Child grade C. Color Doppler was done only in 30 cases.

**Table no: 2:** Preoperative work up

| Investigations                       | findings  |
|--------------------------------------|-----------|
| Deranged LFT                         | 12/104 (11.53%) |
| Deranged Coagulogram                 | 10/104 (9.69%)  |
| Hypersplenism                        | 66/104 (66.46%) |
| Grade 1varices                       | 10/104 (9.61%)  |
| Grade 2varices                       | 42/104 (40.38%) |
| Grade 3 varices                      | 40/104 (38.46%) |
| Gastroesophagealvarices              | 12/104 (11.53%) |
| 64 slice CT splenoportovenogram      | 93/104 (89.42%) |
| Complimentary color doppler          | 30/104 (28.84%) |

**Operative Details**

It was observed that the average time taken for our modified devascularization procedure was 150 minutes with an average blood loss of 300 ml. natural shunts (Table: 3) were observed in 20 cases which was preserved in all. Splenic artery aneurysm was found in 8 cases, associated chronic pancreatitis was in 2 cases and retroperitoneal fibrosis was seen in one patient.
Table no: 3: Operative Details

| Operative Details               | Value                      |
|---------------------------------|----------------------------|
| Average time                    | 150 mins +/- 30 mins       |
| Average blood loss              | 300 ml +/- 50 ml           |
| Presence of natural shunt       | 20 cases (19.23%)           |
| Associated Splenic artery aneurysm | 8 cases (7.69%)        |
| Presence of chronic pancreatitis | 2 cases (1.92%)           |
| Presence of retroperitoneal fibrosis | 1 case (0.09%)     |

Early Postoperative Complications

Early postoperative period was uneventful in majority of patients. There was one on table death in a massively bleeding patient shifted directly from endotherapy suite and was Child C. Morbidity included (Table 4) one case of gastric fistula, one case of recurrent unexplained pain and 5 patients complained of dysphagia which settled with time and prokinetic drugs. There was one case of rebleed in early postoperative period in a cirrhosis patient who was operated as emergency.

Late Post Operative Complications

All patients follow up post operatively with UGIE on 1st and 3rd month later yearly. After devascularisation there was complete obliteration of varices, no residual varices, and during follow up 12 cases had recurrent varices of which 7 patients presented with rebleed. All but one case of recurrent bleeder had minor bleed were managed with endotherapy. One case required surgical re-intervention in whom a splenorenal shunt was performed.

One patient presented with mesenteric vein thrombosis with gangrene of small bowel, was underwent resection of bowel with ileostomy; None of the patients presented with encephalopathy in early or late post operative period. During follow up of eight years with minimum follow up of 11 months 8 patients were died (7.69%), all of them were Child C. All of them were died after 18 months of surgery, 5 of them died due to liver failure or 3 of them died due to septic shock but none died because of variceal bleed.
Discussion

Portal venous pressure gradient more than 10 mmHg leads to gastroesophageal varices and other portosystemic collateral formation, when these portal venous pressure gradient is 12 mm Hg or more will leads to variceal bleeding \(^{(1,13)}\). Acute variceal bleeding is the lethal complication of the portal hypertension, it is associated with a 15% to 20% mortality rate at 6 weeks \(^{(14,15)}\). Hepatic functional reserve is the main determinant of variceal bleeding related deaths, a patient’s Child class at time of onset of bleeding closely related to both early and late mortality following variceal bleeding \(^{(16-20)}\). Initial management of acute esophagogastric variceal bleed is endoscopy (endoscopic sclerotherapy /banding) & pharmacotherapy. In patient with bleeding refractory to endoscopy intervention and pharmacotherapy next line of management will be Portosystemic shunts followed by gastroesophageal devascularisation. Surgical treatment remains the most effective mode of portal hypertension management because of a lower rebleeding rate compared with other forms of treatment \(^{(21)}\).

Shunt and devascularization is an effective procedure for the treatment of portal hypertension, as indicated in the several systematic reviews \(^{(22,23,24)}\). To date, several studies, including randomized controlled trials (RCTs), have compared the outcomes of devascularization and shunt \(^{(25-28)}\). However, The role of shunt and devascularization in portal hypertension still a topic of debate \(^{(29)}\). These surgical shunts have their own limitations like availability of the veins, requires longer operative time than devascularisation which is main concern in hemodynamically compromised patients, surgical expertise, preoperative angiography to declineate anatomy abdominal vessels and higher risk of shunt thrombosis \(^{(30-33)}\). Particularly in non-selective shunts there is risk of postoperative encephalopathy and detoriation of liver function due to diversion of portal blood flow.

TIPS is a non selective shunt, has its own limitations like shunt occlusion, stenosis, availability and vascular anatomy. TIPS require intense post procedure endoscopic surveillance and it is temporary short term measure \(^{(34,35)}\). Esopagogastric devascularisation has prime role in the management of emergency and elective variceal bleeding, it’s indication already mentioned in the material and methods \(^{(30,36)}\).

Liver transplantation is the curative treatment of chronic liver disease with portal hypertension and related complications \(^{(6-9)}\).

In the present series, the incidence of residual (0%) and recurrent 12 (12.53%) varices after extensive devascularization was similar to the figures reported by some authors \(^{(37,38)}\). According to reports in the literature, the rebleeding rate of patients who underwent devascularization was 7.1%-37 % \(^{(39)}\). In our study we had a rebleed in one patient (0.96%) in early post operative while in 7 (6.73%) patients in long term follow up, thus overall rebleed developed in 8 (7.69%) patients. Mathur et al. reported immediate control of bleeding was 100% and 6% of patients rebleed during follow up \(^{(40)}\). The rebleeding rate in Japan is 6%, however, in the rest of the world where there is less experience in performing this procedure, the risk of rebleeding is 20–40% \(^{(12)}\). Xie et al. reported that rebleeding rate is about 10% after a shunt but is very high to the extent of 30-40% after a devascularization procedure \(^{(41)}\). Similar results are shown by the meta-analysis done by Zong et al \(^{(42)}\). The main reason of rebleeding after shunt are stress ulcer hemorrhage and anastomotic thrombosis. Rebleeding in shunt surgery is directly proportional to shunt occlusion.
rate{Zong et al (42) In our study a low rate of rebleed because of extensive devascularization and ligation of retrogastric veins. Both the extent of the devascularization and ligation of retrogastric veins have been shown to be important factors in the prevention of recurrent varices and rebleed (43-46).

We had one patient who developed gastric fistula as a result of our aggressive devascularization. This patient settled on conservative treatment. Dysphagia developed in 5 patients may be result of esophageal edema at transmural stich site which settled with time and prokinetic drugs. One patient presented with mesenteric vein thrombosis with gangrene of small bowel, was underwent resection of bowel with ileostomy.

In gastroesophageal devascularization portomesenteric circulation will maintained and hepatic arterial blood flow will increase, only esophagogastric varices disconnected from portoazygus system (47). Where as in shunt surgeries, especially in non selective shunts diversion of the portal flow results in hepatic insufficiency and also toxic substances such as γ-aminobutyric acid, mercaptans, ammonia which metabolized and detoxicated in liver cells, enter directly into the systemic circulation leads to encephalopathy. (48,49) Rikkers et al. performed a prospective, randomized trial to evaluate the effectiveness of shunt surgeries, they found that overall postoperative encephalopathy has developed in 12% of selective shunt surgery patients and 52% in non-selective group (50). In a meta-analysis done by Zong et al. the rate of hepatic encephalopathy in the devascularization group was significantly lower compared with the shunt group (42). In our study we did not have any case of encephalopathy in post operative period or in long term follow up.

We did elective surgery in 58(55.76%) patients and emergency surgery 46 (44.23%) patients. Only one patient died during emergency procedure due to uncontrolled bleeding. During follow up of 8 years with minimum follow up of 11 months 8 patients were died (7.69%), and a total of 9(8.65%) deaths and all of them were Child C cirrhotic patients. Bernard B et al. and Gu DY et al. reported that operative mortality varies between 15- 90% depending upon the liver function (51,52). Hepatic functional reserve is the main determinant of mortality (30,40,53). In Child-Pugh grade A operative mortality rate may be as low as 15%, but in Child-Pugh grade C, it may be as high as 90%. In our studyout of 13 Child C cirrhotic patients, 9 patients died which is 69.23% which is lesser as compared to world literature. All of them were died after 18 months of surgery, 5 of them died due to liver failure or 3 of them died due to septic shock but none died because of variceal bleed. We attribute our better results partly due to our modified technique in which we apply circumferential transmural stiches instead of esophageal transaction with complete devascularisation and partly due to well preserved liver function as the majority of our patients comprise EHPVO and NCPF. We find this technique highly rewarding in terms of lesser morbidity and mortality especially in emergency settings.

Our modified esopagogastric devascularization has rebleed rate of 7.69% which is comparable to sugiura, modified sugiura and hasaab procedures which has rebleed rate of 5-26%,3.9-31%,6.2-12%, respectively (54). The rate of esophageal leakage, stenosis and chronic encephalopathy with sugiura procedure are 6-14%,2-28% and 0-7%, respectively (55). There was no esophageal leakage, stenosis and chronic encephalopathy noted in our procedure, probably due to application of circumferential transmural stiches instead of esophageal transaction with complete devascularisation. The overall mortality with our procedure was 8.65%, which is comparable to overall mortality of sugiura procedure in japan was 8.5% (55).

Our modified esopagogastric devascularization has many advantages like, it has less post operative morbidity and mortality, can be performed in both elective and emergency situations. It can be performed by any competent surgeon who may not need experience in vascular surgery and pre-operative venous imaging is not
needed. It is performed relatively faster (less operative time) than shunt procedure which is vital in hemodynamically compromised patient\(^{(36)}\). Hence we strongly believe that devascularisation is not obsolete procedure, it has important role in management of esophagogastic variceal bleeding.

No Disclosures
No Conflicts of Interest

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