Addressing Portal Hypertension: Exploring the Crucial Role of Partial Splenic Embolization in Critical Situations

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Abstract

Background: Esophageal varices are one of the most common symptoms of direct outcomes of portal hypertension besides ascites, hepatorenal syndrome, and hypersplenism. Nonselective beta-blockers or band ligation are currently the effective primary preventive options for variceal hemorrhage. The use of partial splenic arterial embolization for the management of variceal hemorrhage in patients with portal hypertension has been described in a small number of reports.

Case Report: A girl, 6 y.o., was hospitalized with hematemesis with hypovolemic shock. Laboratory findings show anemia and thrombocytopenia. Radiology and endoscopy show grade III esophageal varices with hepatosplenomegaly. Because of the high risk of morbidity in surgical procedures, partial splenic embolization was performed to treat portal hypertension.

Discussion: The combination of varices and a low platelet count puts these patients at high risk for catastrophic hemorrhage. Partial splenic embolization reduces splenic blood volume, resulting in a decrease in venous drainage and a reduction in portal venous flow and pressure.

Conclusion: The use of partial splenic arterial embolization to manage variceal hemorrhage in patients with portal hypertension has been described. Embolization can be used alone or in combination with other therapies, like endoscopic ligation or retrograde transvenous variceal obliteration, to treat a variety of conditions.

Keywords: Embolization, esophageal varices, splenomegaly, portal hypertension

INTRODUCTION

Esophageal varices occur in 90% to 95% of patients, and gastric varices occur in 35% to 40% of patients with cirrhosis. Seventy-nine percent of children with extrahepatic portal veins are anticipated to have at least one episode of upper GI bleeding in their lifetime. GI bleeding from a ruptured varix is the most common symptom of portal hypertension. GI bleeding may present as hematemesis or melaena; this is commonly the first symptom of previously undiagnosed portal hypertension. The risk of GI bleeding in children with portal hypertension is significant, with one study showing a risk as high as 75% over 12 years without treatment.(1,2) Variceal bleeding in children is often seen following an upper respiratory infection or fever (3).

Initial treatment for pediatric portal hypertension focuses on preventing and controlling the variceal formation and upper GI bleeding by utilizing medical therapy (e.g., nonselective β-blockers), sclerotherapy, band ligation of varices, and endotherapy. In emergency scenarios, surgical ablative procedures, transjugular intrahepatic portosystemic shunt (TIPS), or balloon-occluded retrograde transvenous obliteration (BRTO) can be done—decisions of which remain individualized due to co-morbidities, vascular disorders, or other limitations (2).

The role of partial splenic embolization is used to improve liver function, decrease
CASE REPORT

A six-year-old girl was hospitalized with hematemesis. She vomited 3 to 4 times each day with a volume as much as 2 glasses each time, which was dark red in color, and had red-to-black clot. She had a fever the day before coming to the hospital and felt better after taking paracetamol. The patient complained of abdominal distension, and pain after the fever, and then vomited without projectiles (Figure 1). Physical examination showed grade I hypovolemic shock and low hemoglobin and thrombocyte level. Thorax CT scan shows gastric varices with collateral vein dilatation to the inferior vena cava, splenomegaly, hepatomegaly, and ascites appropriate with portal hypertension (Figures 2 and 5). An endoscopic examination revealed esophageal varices grade III. Because the surgical procedure has a high risk of morbidity, partial splenic embolization was performed to reduce portal flow, which eventually may reduce variceal bleeding.

RESULT

Our patient underwent partial splenic embolization (PSE) in the distal segment of the splenic artery using 500–750-micron PVA until stasis happened and then continued to the more proximal segment of the splenic artery. Embolization with PVA at 500–750 microns to stasis and post-embolization DSA shows that about 50% of the segmental splenic arteries were occluded with the embolic material (Figure 5). Partial splenic embolization reduces splenic blood volume, resulting in a decrease in venous drainage and a reduction in portal venous flow and pressure.

Besides PSE, esophageal varices call for comprehensive management. Serial laboratory findings show improvements in Hb, platelet, and liver function (Table 1). Embolic agents may vary, including stainless...
steel coils, gelatin sponge, silk suture, and autologous blood clot and polyvinyl alcohol (PVA). The use of coils in grade III esophageal varices is avoided because the goal is distal parenchymal embolization. The infarction area of PVA embolization is located more closely to the periphery of the spleen, and the exudation that resulted from infarction should result in swelling even fluid accumulation under the adventitia of the spleen. Lower-pole and middle-pole embolization are preferred to minimize postprocedure pleurisy and pleural effusion.

Table 1 Laboratory Finding

<table>
<thead>
<tr>
<th>CBC</th>
<th>A day before PSE</th>
<th>A week after PSE</th>
<th>Unit</th>
<th>Normal score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb</td>
<td>6.0</td>
<td>10.4</td>
<td>g/dL</td>
<td>11.4–15.1</td>
</tr>
<tr>
<td>Leu</td>
<td>6010</td>
<td>15,090</td>
<td>/µL</td>
<td>4,700–11,300</td>
</tr>
<tr>
<td>HCT</td>
<td>17.6</td>
<td>30.5</td>
<td>%</td>
<td>38–42</td>
</tr>
</tbody>
</table>

Liver Function

<table>
<thead>
<tr>
<th></th>
<th>Normal score</th>
</tr>
</thead>
<tbody>
<tr>
<td>INR</td>
<td>1.15 – 1.02</td>
</tr>
<tr>
<td>SGOT</td>
<td>64 – 20 U/L</td>
</tr>
<tr>
<td>SGPT</td>
<td>78 – 20 U/L</td>
</tr>
<tr>
<td>Albumin</td>
<td>3.42 – 3.98</td>
</tr>
</tbody>
</table>

Figure 4. Partial Splenic Embolization
Partial splenic embolization was performed in the distal segment of the splenic artery using PVA at 500–750 microns, then continued to the proximal segment of the splenic artery. Post-embolization DSA showed that 50% of the segmental splenic arteries were occluded with embolic materi

Figure 5. CT scans before and after PSE
Figures 5a, 5b, 5c, and 5d show gastric varices with collateral vein dilatation to the inferior vena cava (yellow arrow), splenomegaly with dilatation of the splenic arteries (red arrow), hepatomegaly, and ascites appropriate with portal hypertension. Figures 5e and 5f show partial splenic infarction on the middle- and lower-pole of spleen, as a satisfactory result.

In the follow-up after embolization, the patient may suffer from post-embolization
syndromes such as fever, nausea, and pain in the left upper quadrant. Narcotics and antiemetics are commonly used to treat these symptoms. A week after embolization, the patient never exhibits bloody vomiting with better CBC results. Two months after embolization, the patient’s abdominal CT showed mid-lower-pole splenic infarction, as a satisfactory result of the partial splenic embolization (Figure 5).

DISCUSSION

Portal hypertension is a clinical diagnosis identified through history and physical exams. The two most common clinical manifestations of pediatric portal hypertension that may prompt referral are upper GI bleeding and splenomegaly (5).

Splenomegaly is often first discovered during routine physical exams. Children with enlarged spleens sometimes complain of abdominal pain in the left hemiabdomen and are often first referred to a hematologist to rule out any possible hematologic processes, especially if leukopenia is present (6). Once hematologic causes are ruled out, referral to a pediatric gastroenterologist or hepatologist is appropriate (6). Additionally, the enlarged spleen traps platelets and other blood cells and prevents them from circulating in the bloodstream; this causes thrombocytopenia and low white blood cell. This theory is the same as our patient’s conditions, who had low platelet and low white blood cell count (6).

Our patient, a 6-year-old girl, came with a chief complaint of bloody vomiting along with coughs for the past 3 days before coming to the hospital. The vomit was dark red, and there was red-to-black clot, occurring 3 to 4 times each day with a volume of equal to 2 glasses each time. She already had the same complaint of bloody vomiting 6 months before. This case presents the symptoms being the same as those of variceal bleeding. This patient complained of fever as well, in line with the theory stating that variceal bleeding is often followed by infection with fever.

Initial treatment for pediatric portal hypertension focuses on preventing and controlling the variceal formation and upper GI bleeding by utilizing medical therapy (e.g., nonselective β-blockers), sclerotherapy, and band ligation of varices (3). Endoscopic sclerotherapy and endoscopic variceal ligation are effective in 80–90% of patients in controlling acute bleeding from esophageal varices and preventing rebleeding. Endotherapy is more effective with less rebleeding rates when combined with vasoactive drugs (5). Surgery is primarily indicated in patients with variceal bleeding who fail to respond to endoscopic management.

Various types of surgical procedures are as follows (5):

1. Shunt/Bypass procedures: Non-physiological shunts bypass the portal blood either totally or partially into the systemic circulation. Total and partial shunts are also known as non-selective and selective shunts, respectively, as the latter selectively decompresses the gastroplenic zone (4). Physiological shunts, like mesenteric-left PV bypass or the Rex shunt, maintain the hepatic portal blood flow, while bypassing the level of obstruction. It decompresses the splanchnic bed from the superior mesenteric vein to the left branch of PV via an autologous graft (usually internal jugular vein) (3).

2. Ablative procedures: Patients with unsuccessful shunts—those without any shuntable veins or those in emergency situations with refractory variceal bleed—undergo esophagogastric devascularization alone or in combination with splenectomy (7). These techniques have become obsolete because of significant rebleeding rates and mortality.
In 8–12% of cases, endotherapy may fail to control acute variceal bleeding. In emergency scenarios, surgical ablative procedures, transjugular intrahepatic portosystemic shunt (TIPS), or balloon-occluded retrograde transvenous obliteration (BRTO) can be done—decisions of which remain individualized (8). TIPS is the gold standard for endoscopy and pharmacological therapy for refractory upper GI bleeding or for patients who are at high risk of secondary treatment failure, by induced acidity in gastric mucosa (2).

In some patients, TIPS implantation is not possible due to co-morbidities, vascular disorders, or other limitations. Splenectomy may be a promising alternative in these settings (4). However, it is an invasive procedure with several disadvantages, as patients with portal hypertension induce upper GI bleeding frequently in conditions unfit for surgery. Partial splenic embolization is a technique used for the mitigation of portal hypertension to reduce the risk of upper GI bleeding (2). Splenic embolization is a transcatheter embolization method, and successful embolization of the selected arteries results in the devascularization of a focal lesion or in intentional reduction or cessation of blood flow into a target vascular bed or to the entire organ (7). Several studies indicate that partial embolization of the spleen with or without variceal ligation significantly reduces variceal rebleeding. Ohмотo et al. described 52 cirrhotic patients with upper GI bleeding and compared bleeding rates after splenic embolization in combination with variceal ligation to variceal ligation alone, showing a significant reduction of re-bleeding in follow-up from 39% to 12% (2). Several studies (2,3) reviewed the results involving 50 patients with portal hypertension from five different studies. Bleeding episodes per year decreased from 2.4 (mean) to 0.48 after partial splenic embolization. In addition, Uflacker et al. reported improvements in hepatic encephalopathy, lasting up to 2 years after partial splenic embolization. Partial splenic embolization also increases blood counts (3). The platelet count starts rising as early as 12–24 hours after partial splenic embolization and peaks in 1–2 weeks. The count usually stabilizes in about 2 months at approximately double the value before embolization and then slowly decreases over the next several years. The RBC count increases significantly by 6 months after embolization and stays elevated for years (4). Finally, Shimizu et al. described the successful treatment of a critically-ill patient with refractory bleeding from portal hypertensive not eligible for TIPS placement (2).

Many materials have been developed for embolization use, including stainless steel coils, gelatin sponges, silk sutures, and autologous blood clot and polyvinyl alcohol (PVA) (2). Embolization pain caused by PVA is earlier and more severe than by other materials; it is because the infarction area of PVA embolization is located more closely to the periphery of the spleen, and the exudation which resulted from infarction should result in swelling even fluid accumulation under the adventitia of the spleen (1). However, as a permanent embolic material, PVA provides an embolization with less recanalization rate and

Figure 4. Treatment Diagram (4,7)
lower incidence of fever than other materials do, which may be related to the fewer chances of fever and infection of PVA-induced dry infraction of the functional areas of the spleen (7).

The techniques of PSE reported vary greatly. Basically, they can be classified into two categories, including non-selective and selective catheterization methods:

1. **Low-Pressure Flow Control Protocol.** According to our experience, the low-pressure flow control procedure is a non-selective procedure in which the embolization materials are injected into a primary branch of the splenic artery by a catheter and float downstream (3,9). The procedure is quite simple; unfortunately, it is difficult to determine the volume of the embolized organ and the unintended occlusion of the pancreatic artery that may happen. The results of the main coil and sub-selective embolization are similar, and typical complications do not appear to have an impact on the outcome.

2. **Embolization of the Lower or Upper Polar Splenic Artery.** The catheter tip is inserted into the lower polar splenic artery's primary branch. Later, the embolization would be accomplished with permanently embolic materials after an estimation of the volume to be embolized is made by an observation by injecting a contrast medium. Some PSE consequences, such as atelectasis or pneumonia, as well as symptoms produced by a diaphragm or pleural irritation, may be reduced with this strategy. Furthermore, selecting selective catheterization may help to minimize inadvertent embolization of the pancreatic artery. The lower and upper polar arteries can be embolized in conjunction with achieving parenchymal tissue peripheral infarction (2,4).

**CONCLUSION**

Splenectomy is a technique that can be used with other treatments for portal hypertension. In our case, partial splenic embolization can decrease the incidence of variceal bleeding and can be helpful as an alternative or adjunctive procedure. Although, abscess formation may develop in response to splenic infarction.

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**REFERENCES**
