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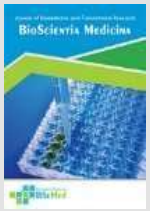

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Management of Thrombocytopenia with Partial Splenic Embolization in Liver Cirrhosis

Septia Harma Putri[1*], Arnelis[1], Saptino Miro[1]

[1]Department of Gastroenterohepatology, Faculty of Medicine, Universitas Andalas/Dr. M. Djamil General Hospital, Padang, Indonesia

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***Corresponding author:**
Septia Harma Putri

E-mail address:
septiaharmaputri@gmail.com

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A B S T R A C T

Thrombocytopenia is a frequent complication in patients with cirrhosis. Thrombocytopenia is generally divided into mild, moderate, and severe thrombocytopenia. Thrombocytopenia in liver cirrhosis not only increases the risk of bleeding during surgery but can also have an impact on patient management, such as liver biopsy, administration of antiviral therapy, and postponement of elective surgery. The pathophysiology of thrombocytopenia in chronic liver disease can be caused by decreased platelet production, sequestration in the spleen, and increased platelet destruction. Partial splenic embolization (PSE) is one option for treating thrombocytopenia in chronic liver disease. PSE is an effective procedure in treating complications associated with hypersplenism and portal hypertension, such as esophageal varices, pancytopenia, portal hypertensive gastropathy and ascites.

1. Introduction

Thrombocytopenia is a frequent complication in patients with cirrhosis. Thrombocytopenia is generally defined by a platelet count below normal, namely mild thrombocytopenia <150 000/µL, moderate thrombocytopenia 50-100,000/µL and severe thrombocytopenia <50 000/µL. As many as 84% of patients with cirrhosis experience thrombocytopenia, and it is an independent variable indicating advanced disease and poor prognosis.[1,2] Thrombocytopenia in liver cirrhosis not only increases the risk of bleeding during surgery but can also have an impact on patient management, such as liver biopsy, administration of antiviral therapy, and postponement of elective surgery. The degree of thrombocytopenia can act as a prognostic marker in liver cirrhosis. This is characterized by severe thrombocytopenia < 50,000/mm^3 associated with significant morbidity. Thrombocytopenia is associated with complications of chronic liver disease such as portal hypertension, esophageal varices, and hepatocellular carcinoma.[1,3]

The pathophysiology of thrombocytopenia in chronic liver disease can be caused by decreased platelet production, sequestration in the spleen, and increased platelet destruction. Hypersplenismus hypothesis, where portal hypertension occurs, causes pooling and sequestration of all blood corpuscular elements, especially platelets in the splenic blood vessels, which experience blockage and congestion and can also reduce the number of platelets. Another mechanism in the form of increased platelet destruction can occur due to immunological

destruction, stress conditions, bacterial translocation, and sepsis.[2,4] Thrombocytopenia is associated with severe liver cell damage, which indicates an increasing degree of fibrosis in the liver. In hepatitis C, thrombocytopenia is most often caused by splenomegaly. Increasing splenomegaly is also closely associated with progressive liver fibrosis. Hepatitis C infection can cause bone marrow suppression. Besides, the use of interferon antivirals in hepatitis C shows a decrease in platelet numbers after therapy. So severe thrombocytopenia is a contraindication for administering interferon antivirals. Excessive alcohol use has a direct toxic impact on megakaryocytes, which reduces platelet counts.[5]

There are several therapies for thrombocytopenia in cirrhotic patients, both non-pharmacological and pharmacological, such as administering platelet transfusions, splenectomy, partial splenic embolization, and administering thrombopoietin agents. Providing platelet transfusions has side effects in daily practice. In addition, there is no established cutoff value for starting platelet transfusions. Platelet transfusions can increase the risk of transmitting infection. Giving platelets can also cause refractory platelets to occur. That is, the expected platelet value cannot be achieved after giving a platelet transfusion.[2] Nonpharmacological measures such as splenectomy can be considered in all patients with chronic liver disease and thrombocytopenia due to portal hypertension. This surgical procedure carries risks such as bleeding and/or thrombosis of the portal vein or splenic vein. Open splenectomy surgery carries a risk of infection and injury to the pancreas. So, open splenectomy is not recommended, while laparoscopic splenectomy is still controversial because it is associated with the risk of patient morbidity.[6]

Partial splenic embolization (PSE) is one option for treating thrombocytopenia in chronic liver disease. Previously, PSE was carried out in patients with thrombocytopenia, in patients with cirrhosis caused by hepatitis C, and in hypersplenism patients who would receive interferon therapy because giving this therapy would cause a more severe decrease in the number of platelets. PSE is an effective procedure in treating complications associated with hypersplenismus and portal hypertension, such as esophageal varices, pancytopenia, portal hypertensive gastropathy and ascites. PSE is also effective in reducing serum ammonia in hepatic encephalopathy. Partial splenic embolization has better advantages than laparoscopic splenectomy because of the lower risk of sepsis and mortality. Partial splenic embolization still carries the risk of complications such as pneumonia, peritonitis, splenic abscess, and portal vein thrombosis.[2,6]

Partial splenic embolization

Partial splenic embolization (PSE) is an embolization procedure that aims to reduce the volume of the functional spleen by injecting embolic material at the end of the splenic artery. In general, PSE is indicated for all complicated conditions caused by hypersplenism and portal hypertension, such as esophageal varices, pancytopenia, portal hypertensive gastropathy, ascites, and encephalopathy. PSE indications can also be used in hematological diseases such as idiopathic thrombocytopenia purpura and hereditary spherocytosis. This procedure is a transcatheter embolization method, and successful embolization of arteries can be seen from devascularization of focal lesions or reduction of blood flow to the target vasculature or the entire organ. Decreased blood flow to the spleen results in ischemic necrosis and may improve hypersplenism. This procedure aimed at hypersplenism has been carried out since 1979 and is more accepted than splenectomy surgery.[7-11] The efficacy of PSE for the management of thrombocytopenia has been proven, but there is no specific literature discussing the consensus on the use of embolic materials. Several embolic materials have been used in PSE including temporary agents such as Gelfoam and permanent agents such as polyvinyl alcohol (PVA) particles, trisacryl gelatin microspheres (embospheres) and others. Gelfoam is absorbed very quickly and is the agent of choice for PSE however its availability is limited. Compared with gelfoam,

permanent agents such as embossed particles and PVA have smaller specific sizes. PVA particles have a diameter of 300-500 μm, and they can reach the splenic sinuses more closely than gel foam, so PSE uses PVA particles more efficiently in treating hypersplenism than gel foam. Apart from that, there is another material in the form of stainless steel mesh, which has lower pain and other side effects but has a higher relapse rate of hypersplenismus.[7,9]

Not all thrombocytopenia is treated. Until now, there has been no specific limit for the number of platelets that must be treated. Patients with thrombocytopenia who have symptoms such as bleeding and petechiae due to thrombocytopenia need immediate evaluation. Meanwhile, patients with thrombocytopenia without symptoms can be monitored every 1 – 4 weeks based on the degree of thrombocytopenia. Partial splenic embolization can be an effective treatment option for complications associated with hypersplenism and portal hypertension, such as pancytopenia, esophageal varices, portal hypertensive gastropathy, and ascites and encephalopathy due to cirrhosis.[5,11] Patients who will undergo PSE procedures must be in stable condition to undergo endovascular procedures. The day before the procedure, the patient must get good hydration. Laboratory tests that need to be carried out before the procedure include a complete blood count, liver function tests including albumin and bilirubin values, coagulation profile, namely prothrombin time, partial thromboplastin time, and international normalized ratio, kidney function tests, and alpha-fetoprotein (AFP). All patients should undergo gastrointestinal endoscopy to look for or treat esophageal varices. An abdominal ultrasound examination should be performed to assess the size of the spleen, liver echo texture, the patency and caliber of the portal and lineal veins, abdominal varicose veins, and the degree of ascites, if any. Assessment of the platelet count to consider administering a platelet transfusion before the procedure if necessary. Platelet transfusions are given in cases with severe thrombocytopenia. Namely, if the platelet count is less than 30,000/mm^3.[7] All patients who will undergo the PSE procedure must receive the pneumococcal, Haemophilus influenza, and meningococcal vaccine before the procedure is carried out. This aims to reduce the risk of patients experiencing complications after the procedure is carried out. Prophylactic antibiotics can be given to prevent complications of sepsis in PSE. Zaitoun et al. (2021) gave third-generation cephalosporin prophylactic antibiotics 1 gram twice a day and an intravenous infusion of metronidazole 500 mg every day. This antibiotic was given 3 days before the procedure and continued 7 days after the procedure, then continued with oral antibiotics, namely ciprofloxacin 500 mg every 12 hours for the following week. Meanwhile, Amir et al. (2019) in their research provided prophylactic antibiotics, namely ceftriaxone 2 grams intravenously every 24 hours and combined with intravenous metronidazole 500 mg every 8 hours.[7,10]

The PSE procedure can be performed by an interventional radiologist using an angiography unit. After local anesthesia is performed on the femoral neck, it is inserted using a 6-F introducer and a 4 or 5-F catheter. Before embolization is performed, an angiogram is performed on the splenic and celiac arteries to assess the anatomy of the splenic artery and collateral routes. A catheter is placed in the pancreatic branch to slowly inject embolic material under fluoroscopy guidance. When the catheter reaches the splenic hilum, the splenic artery is divided into superior and inferior branches. Each branch is divided into 4 - 6 intralien segmental branches. Injections of antibiotics and steroids such as hydrocortisone 500 mg are injected through the splenic artery. A lien can be divided into several small segments, and there are branches between each segment. Embolic material is mixed with 80 mg of gentamicin as prophylaxis, which reduces the risk of splenic abscess formation.[7,9,11] An angiogram is performed after embolization to assess the splenic parenchyma for ablation. Angiography is performed several times during the procedure to ensure that a very large infarction does not occur. Zoitoun et al

(2021) stated that embolization is technically successful and can be declared complete if ablation of around 60-70% of the parenchyma has occurred. Meanwhile, Talwer et al. (2019) stated that the optimal splenic infarct volume is 50-70% of the splenic volume. Infarcts that occur below 50% have a short-term effect in achieving improvement in platelets and leukocytes. After the PSE procedure, the patient is hospitalized and observed for 5-7 days and receives prophylactic antibiotics. The average time required for this procedure is 16 minutes.[7,8]

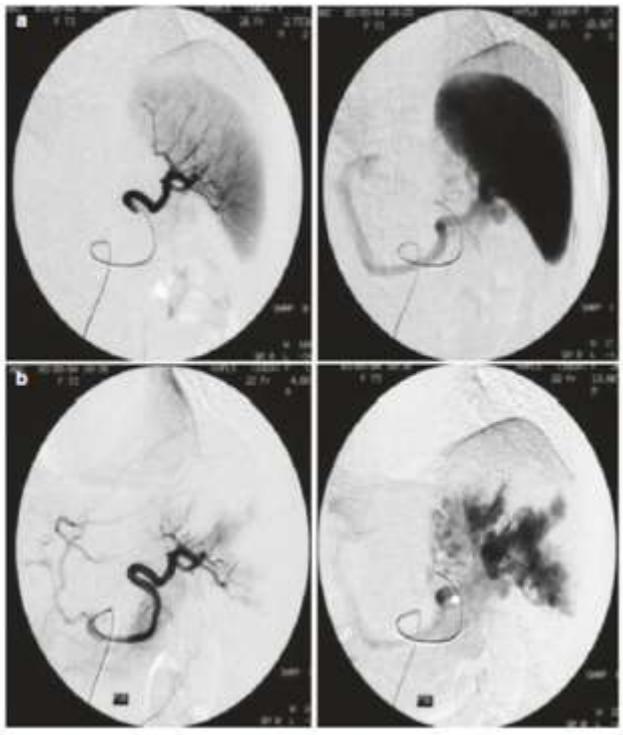

Figure 1. (a) Arteries and veins on splenic angiography before PSE, (b) arteries and veins on splenic angiography after PSE.

There are several splenic embolization techniques. the first is splenic artery trunk embolization, namely placing a stainless steel mesh or balloon on the main branch of the splenic artery, which causes a decrease in the portal vein and prevents bleeding from esophageal varices, which has the same effect as ligation. This technique also reduces the risk of surgery by increasing the platelet count before splenectomy. This procedure does not cause an extensive infarction, but hypersplenism may relapse after the development of collateral circulation.[9] The second technique is non-selective PSE, namely PSE procedure with a low-pressure flow control protocol. The material is injected into the main splenic artery branch using a catheter and follows the blood flow until the material stops and no longer follows the blood flow. This procedure is more difficult to evaluate. The third technique is embolization of the superior and inferior splenic arteries. This procedure is a selective method. Namely, the tip of the catheter is placed in the main branch of the inferior splenic artery. This method has fewer complications.[9] After the PSE procedure is carried out, the patient is monitored for the total number of days of treatment and assessed whether there are any complications that occur after the procedure. Evaluation of the therapeutic effect can be seen from several indicators, including platelet count, leukocyte count, erythrocyte count, hemodynamic changes, changes in spleen size, and improvement of the gastric mucosa. An increase in platelet count can

be found 12 to 24 hours after the PSE procedure, while it reaches its peak 1 to 2 weeks after the procedure. The platelet count should stabilize within 2 months, around 2 times higher than the platelet count before PSE was performed. There is a positive correlation between platelet count and splenic infarct volume.[9,11] Wu et al. (2017) found a drastic increase in platelets after the 7th day of the PSE procedure, with an average increase in platelets before the procedure of 35,077/mm^3, increasing to 134,833/mm^3. The average platelet value during the maintenance period is around 80,000/mm^3, which can be maintained for 1 year after the PSE procedure. The same thing was also stated by Ishikawa et al. (2019); in their research, there was a significant increase in platelets after the PSE procedure. The average platelet count before the procedure was 48,000/mm^3 and increased to an average of 134,000/mm^3 after 1 month of the PSE procedure.[11-16] Kogure et al. (2017) showed that platelet values increased from 57,000/mm^3 to 153,000/mm^3 in 7 days after the patient underwent PSE. Then, there was a decrease in platelets after 1 year of treatment to around 81,000/mm^3 and remained at the maintenance value. Asar et al. (2019) compared the effectiveness of PSE compared with percutaneous microwave ablation, concluding that PSE had the same effective impact as percutaneous microwave ablation in treating hypersplenism in liver cirrhosis patients. There was an increase in hemoglobin, leukocytes, and platelets from an average of 9.47 g/dL, 2,600/mm^3, 42,750/mm^3 to 9.95 g/dL, 6,880/mm^3 and 313,500/mm^3 in patients who underwent PSE procedures.[17-19]

The number of leukocytes normally increases after splenectomy on the first day and peaks on the third day after surgery. Splenectomy and PSE are both effective for increasing leukocyte and platelet counts. Although splenectomy is able to increase higher leukocyte and platelet counts, PSE is a simple and minimally invasive procedure and can be an effective option for patients who are at risk for surgery or for whom splenectomy is contraindicated. In research by Taniai et al. (2019), the number of leukocytes increased by 51% in the first month and 30% in 6 months. The number of erythrocytes increased was found at 3 months after PSE, increased significantly at 6 months after the procedure and could even increase up to 7.5 years.[9,11,20] Research by Zaitoun et al. in 2021 on 179 patients divided into 3 groups underwent PSE for the management of hypersplenism. Each group was divided into different embolic materials, namely consisting of groups that used Gelfoam, Embosphere and Countour SE. There were no significant differences between age, gender, Child-Pugh classification, the presence of esophageal varices and the presence of Hepatocellular Carcinoma in the three groups. The technical success rate reached 100%.[7] Dawoud et al. (2018) compared the use of embolic materials between Gelfoam and Microsphere. His research showed that the efficacy was almost the same between the two embolic materials, and there was no significant difference in laboratory results after the procedure was carried out. However, the use of these two embolic materials differs in the incidence of complications. The use of microspheres appears to cause fewer serious complications compared to the use of gel foam.[18] Loffroy et al. (2019) used another material to treat hypersplenism in patients requiring chemotherapy, namely the use of cyanoacrylate glue, showing safety and effectiveness for the management of thrombocytopenia associated with hypersplenism. Its use can increase platelets. Pang et al. (2018) assessed the use of a balloon for partial splenic embolization, showing satisfactory results with a significant increase in platelets and leukocytes after embolization using a balloon.[21-23] Laboratory monitoring showed a significant increase in leukocyte and platelet results after the PSE procedure in all three groups from the second week to one year after the procedure. This increase is also in accordance with the research results of Talwer et al. (2019), which stated that the PSE procedure was generally effective in increasing the number of platelets and leukocytes. The increase in leukocytes and platelets was found to be highest in the group that received Embosphere and PVA materials compared to the group that received

Gelfoam. No significant differences were found in the values of leukocytes and platelets in the embossed and PVA groups.[6,8]

Splenic volume was assessed radiologically using CT examination after the PSE procedure was performed. Zaitoun et al. (2021) stated that there were no significant differences between the three groups of embolic materials, but it was found that the volume of the spleen that experienced infarction was smaller in the group that received Gelfoam. There was no difference in splenic size before and 1 month after the PSE procedure. The size of the spleen decreased after 1 year in the three groups studied by Zoitoun. The size of the spleen decreased significantly in the group that received embossment and PVA. This is most likely caused by the embosphere and PVA material being smaller than the Gelfoam material so that it has better performance in achieving distal embolization of the splenic artery branch. In addition, the Gelfoam material is temporary, so recanalization of the splenic artery can occur after the PSE procedure. There were no significant differences in portal vein diameter before and after PSE procedures in the three groups.[7] Radiographic examination after the PSE procedure, namely an abdominal CT scan, was carried out on all patients after 1 month of the PSE procedure. The radiologist measured the volume of the spleen and residual spleen visible in the venous phase. Meanwhile, an abdominal ultrasound examination is carried out when the patient is allowed to be outpatient to assess complications from the procedure that has been carried out. Follow-up examinations were carried out after 3 months, 6 months and 1 year to assess the size of the spleen and the caliber of the portal vein as indicators of portal hypertension.[6]

PSE procedure also has the benefit of reducing the incidence of recurrent esophageal varices. Ishikawa et al. (2019) stated that there was a significant reduction in the incidence of recurrent esophageal varices in patients who underwent endoscopic treatment combined with PSE compared to esophageal varices treated using endoscopic treatment alone. Kogure et al. (2017), in a case report of a patient with recurrent variceal bleeding who underwent PSE, showed that there were no signs of recurrent varicose veins in the 2 years after the PSE was performed. There was a decrease in the size of the spleen within 5 years after PSE and up to 13 years after PSE, and even until the patient died with hepatocellular carcinoma, the patient did not show any recurrent variceal bleeding or esophageal varices. Buechter et al (2017) also stated that PSE is a meaningful alternative treatment in patients with recurrent portal hypertension which causes recurrent upper gastrointestinal bleeding. This can be seen in the research. There were no patients who experienced recurrent bleeding or required blood transfusions during a total follow-up of 159 months after the PSE procedure.[14,17,23-26] Hemodynamic changes can occur in patients after PSE. Patients with Child's C cirrhosis have lower portal flow velocities compared to those with Child's A cirrhosis. Decreased blood flow and increased congestion in the portal and splenic veins are associated with impaired liver function in cirrhotic patients. PSE is able to create a hyperdynamic condition in the splenic area, and can reduce venous blood flow to the spleen and reduce portal vein pressure, and reduce the ratio of spleen to liver size. Changes in splenic size of around 50-80% occur due to devascularization due to embolization. A splenic volume of 30-40% after embolization results in reduced morbidity. Splenic necrosis of more than 70% is associated with splenic abscess and carries a risk of death.[9,13] The PSE procedure also has a good effect on liver function in the long term. Tajiri reported that cholinesterase activity and serum albumin increased 6 months after PSE and could last for several years. PSE also has a positive impact for cirrhosis patients in increasing liver protein synthesis. Jiao said that serum albumin levels increased significantly and bilirubin, INR and globulin values decreased sharply after 6 months of PSE treatment. Child's Pugh grading also appeared to have improved significantly in the PSE group compared to the preoperative condition. Ishikawa et al (2019) stated that in their research, there was a change in the Child's Pugh stage 1 month after the PSE procedure was carried out.[14]

A similar thing was also stated by Assal et al. (2017), who compared the PSE procedure with microwave ablation, showing a significant changing effect on bilirubin, albumin, creatinine, and prothrombin time in the group that received the PSE procedure, while in the microwave ablation group, there was no change in the indicators. the. Ishikawa et al. (2020) stated that patients who underwent PSE after retrograde transvenous obliteration using a balloon showed a decrease in hepatic venous pressure, improvement in Child's Pugh scores, and blood ammonia levels after all therapy was carried out.[11,20,27] Long-term monitoring is carried out for up to 1 year after the procedure to assess responses such as recurrent variceal bleeding, bleeding at other sites such as gingiva or epistaxis, and the ability to respond to the planned intervention. Laboratory examination after the PSE procedure is in the form of a complete blood picture to assess the number of erythrocytes, leukocytes, and platelets, which are assessed every 2 weeks, 1 month, 6 months, and 1 year after the PSE procedure.[7] PSE procedures are contraindicated in hypersplenism with a terminal disease, pyemia, or other serious infections, which result in a high risk of splenic abscess after the procedure. In patients with a prothrombin time below 70% of normal control, repeat therapy is required if necessary before PSE is carried out. Patients who are allergic to contrast agents are also contraindicated to this procedure. Patients with a history of asthma and kidney disorders should be given an adapted contrast agent.[9,11] Complications of the procedure in the form of minor complications can include pain, fever, vomiting, and major complications can include ascites, pleural effusion, splenic abscess, bacterial peritonitis, variceal bleeding, and portal vein thrombosis. So, in post-procedure monitoring, you must check for symptoms and signs that may appear related to these complications. Pain can be classified as a major complication if there is pain that requires therapy for more than 24 hours. The following are some complications of PSE procedures.[7]

Post-embolization syndrome is a frequent occurrence, reported to occur in up to 30% of cases and can usually be treated without leaving residual symptoms. This syndrome is usually characterized by intermittent fever every day below 39 degrees Celsius, abdominal pain, nausea, vomiting, feeling full in the stomach, and decreased appetite. The most common symptoms are fever 94% and abdominal pain 82%. Intermittent fever can occur due to the release of pyrogens by inflammatory cells in the infarct area. However, a fever that persists for more than 7 days and is around 39 degrees Celsius indicates an infection. Abdominal pain is caused by edema in the area of infarction and should be managed with effective pain medication within 3 to 10 days. The size of the embolization particle is the main factor influencing the incidence of post-embolization syndrome.[9] Hegazy et al. (2019) stated that the most common complication was post-embolic syndrome (93.3%). This was also found by Morsy et al. (2018) in their research, and post-embolization syndrome occurred in all patients (100%). This complication can be managed conservatively using antipyretics and antibiotics. Patients are allowed to be outpatient when these symptoms have reduced. Taniai et al. (2019) found that the most frequent complications were left abdominal pain (77.6%), fever (94.8), ascites (5.2%), left pleural effusion (3.4%), and abscess. spleen (1.7%). Ascites can be resolved within 1 month with the use of diuretics and supportive liver treatment.[11,21,23] Complications of PSE procedures on the lungs can occur, including pneumonia, atelectasis, and pleural effusion. This complication usually occurs on the left side of the body and after embolization has been performed. This is associated with respiratory limitations due to pain in the left upper quadrant, pleural reaction, and inadequate drainage of inflammatory effusion. Mild and moderate pleural effusions can be absorbed after effective antibiotics and pain therapy, whereas thoracentesis needs to be performed if there is a severe pleural effusion.[9] Serious complications of PSE are peritonitis and splenic abscess, which are associated with large embolic volumes, impaired immune function, poor aseptic processes, and retroinfection by intestinal anaerobic

bacteria. Adequate antibiotic therapy needs to be given, and in some cases, surgical intervention is needed. Complications of splenic abscess after PSE are influenced by several things, namely impaired immune function of the spleen, cirrhotic patients who are more susceptible than portal hypertension caused by other causes, or massive infarction with bacterial contamination.[9,28-30]

Administration of contrast agents, redistribution of blood flow to organs, and renal hypoperfusion are associated with damage to renal function after PSE procedures. The pathogenesis of contrast nephropathy is closely related to cellular hypoxia and intrarenal hemodynamic disturbances. Contrast media decreases medullary blood flow and oxygen tension, leading to hypoperfusion and hypoxia. In many cases, damage to kidney function is reversible and can be prevented with the use of several agents, such as adenosine receptor antagonists.[9] Evaluation of liver function damage after PSE can be carried out using a useful method before the procedure, Child-Pugh grading. Although necrosis occurs in areas of embolism, decreased portal venous flow, portal venous thrombosis and administration of contrast agents are associated with liver damage after PSE.[9] Decreased portal venous flow and rapid increase in platelet count after embolization can lead to a hypercoagulable condition of portal venous flow. This is related to complications that can occur after the PSE procedure is carried out, namely portal vein thrombosis. This risk can be overcome by administering heparin. Hegazy et al. (2019) found two cases (13.3%) of patients who experienced complications in the form of portal vein thrombosis. Ogawa et al. (2021) stated the importance of assessing the risk of portal vein thrombosis before the procedure. Risk factors that influence this include the size of the splenic vein before the procedure, the percentage of splenic infarction after the procedure, and the volume of the splenic infarction. The larger the diameter of the splenic vein, the higher the risk of thrombosis; likewise, the higher the percentage of spleens that experience infarction, the higher the risk of thrombus formation.[9,15,21]

In Zoitoun et al's study, patients who received Gelfoam had a longer hospital stay after the procedure to overcome procedural complications. Some minimal complications such as post embolization syndrome, namely pain, fever and vomiting, appeared more frequently but there was no significant difference between the groups that received Gelfoam, Embosphere or Countour SE. 4 The group that received Gelfoam needed analgesics for more than 24 hours by 20%, Embosphere 31% and group PVA 32.3%. The incidence of fever in the Gelfoam group was 24.6%. 7 Major complications were found in 3 patients who received Gelfoam, namely bacterial peritonitis within 10 days after the PSE procedure was carried out, and two of them suffered from splenic abscesses which required antibiotic therapy and abscess drainage using a pigtail catheter. Meanwhile, 1 patient each from the three groups experienced a splenic abscess requiring intensive antibiotic therapy and pigtail catheter drainage. Complications in the form of hematemesis were found in 5 patients, and it was necessary to take action in the form of endoscopic intervention. Portal vein thrombosis was found in 5 patients during follow-up using ultrasound and CT within the first month after the procedure. Patients with ascites and/or pleural effusion that causes abdominal discomfort and/or shortness of breath undergo thoracentesis and peritoneosynthesis.[7] Long-term monitoring carried out in Zoitoun et al's study showed that there was recurrent hematemesis in 10 of 36 patients (27.7%) who received Gelfoam, 5 patients of 30 (16.6) patients who received Embosphere, and 5 patients of 32 patients (15.6%) who received PVA. Talwer et al (2019) in their study stated that several patients who received the PSE procedure experienced post-procedure complications such as fever, nausea and abdominal pain. Minor complications occurred more often than major complications, namely around 73.4%. Major complications that often occur are 0.6% gastrointestinal bleeding, 1.3% splenic abscess, 1.3% bacterial peritonistis, 8m1% ascites, and 9.4% pleural effusion. Mortality is very rare, mortality is usually caused by sepsis caused by splenic abscess or

bacterial peritonitis.[7,8] The complications that occur are related to the extent of the infarction that occurs in the spleen that has undergone embolization. Complications increase with infarctions that exceed 70% of the splenic volume. Complications are also related to underlying liver disease. Talwer et al. stated that the complication rate increased with the higher Child-Pugh class of the patient.[8]

2. Conclusion

Partial splenic embolization is one of the procedures of choice for treating thrombocytopenia in chronic liver disease. Partial splenic embolization can treat thrombocytopenia caused by hypersplenism. The splenic parenchymal target expected to experience infarction after embolization is 60 – 70%. Partial splenic embolization has lower risks compared to splenectomy. Partial splenic embolization is not only useful for increasing platelets after the procedure but also provides other benefits, namely increasing erythrocytes and leukocytes, improving gastric mucosa, and reducing the incidence of recurrent esophageal varices.

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