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Partial Splenic Embolization Versus Splenectomy for the Management of Hypersplenism in Cirrhotic Patients

Mahmoud A. Amin · Mohamed M. El Gendy · Ibrahim E. Dawoud · Ashraf Shoma · Ahmed M. Negm · Talal A. Amer

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Abstract
Background Hypersplenism occurs in patients with chronic liver disease, and splenectomy is the definitive treatment. However, the operation may be hazardous in patients with poor liver function. In recent years, partial splenic embolization (PSE) has been widely used in patients with hypersplenism and cirrhosis. This study was conducted to assess the safety and efficacy of PSE compared to splenectomy in the management of hypersplenism in cirrhotic patients.
Methods This study comprised 40 patients with hypersplenism secondary to cirrhosis. They were divided into two groups, each including 20 patients. The first group of patients were treated by PSE using polyvinyl alcohol particles to achieve embolization of at least 50% of the distal branches of the splenic artery. Postembolization arteriography and computed tomography were performed to document the extent of devascularization. Patients in the second group were treated by splenectomy with or without devascularization and left gastric ligation according to the presence or absence of esophageal varices.
Results There was marked improvement in platelet and leukocytic counts in both groups, and the counts remained at appropriate levels during the follow-up period. All patients in the first group had problems related to postembolization syndrome that abated by the first week. One patient in the first group died from myocardial infarction. No deaths occurred in the second group. Asymptomatic portal vein thrombosis developed in one patient in the first group that was treated with anticoagulation, and another patient developed splenic abscess treated by splenectomy with a good outcome. In the second group, three patients developed portal vein thrombosis, one of them being readmitted 4 months postoperatively with mesenteric vascular occlusion; that patient underwent a resection anastomosis with good outcome.
Conclusions Partial splenic embolization is an effective therapeutic modality for the treatment of hypersplenism secondary to chronic liver disease. It is a simple, rapid procedure that is easily performed under local anesthesia; and it allows preservation of adequate splenic tissue to safeguard against overwhelming infection.

M. A. Amin (✉) · M. M. El Gendy · I. E. Dawoud · A. Shoma · A. M. Negm
General Surgery Department, Mansoura Faculty of Medicine, Mansoura University Hospital, Mansoura 35516, Egypt
e-mail: dr.mahmoudamin@hotmail.com

T. A. Amer
Radiology Department, Mansoura Faculty of Medicine, Mansoura University Hospital, Mansoura 35516, Egypt

Introduction

Hypersplenism often accompanies chronic liver disease, and surgical splenectomy is the traditional treatment; however, splenectomy is associated with perioperative and postoperative complications [1]. Partial splenic embolization (PSE) has been proposed as an alternative to surgery. The procedure helps occlude the arterial supply of the spleen more peripherally, which results in ischemic necrosis of much of the functional spleen followed by a decrease in splenic size and hypersplenism. It also allows preservation of adequate splenic tissue and thereby avoids the risk of overwhelming postsplenectomy infection [2]. In patients with chronic liver disease, the procedure reduces immunologic induction of thrombocytopenia [3]. However, this therapeutic method occasionally has been associated

with serious complications, such as splenic abscess and septicemia [4]. The study was conducted based on the hypotheses that partial splenic embolization has the advantage of being a nonoperative intervention, that it is safe and effective in the management of secondary hypersplenism, and that it is equivalent or even superior to splenectomy.

This prospective randomized study was conducted to prove the safety and efficiency of partial splenic embolization compared to splenectomy in the management of hypersplenism in patients with chronic liver disease.

Patients and methods

This prospective randomized study was conducted on 40 patients admitted to the Surgical Department, Mansoura University Hospital between November 2002 and April 2005 with a diagnosis of hypersplenism secondary to cirrhosis. Patients were randomly classified into two groups, with each group comprising 20 patients. Group 1 was treated by percutaneous transcatheter partial splenic embolization; group 2 was treated by splenectomy with or without devascularization and left gastric vessel ligation according to the presence or absence of esophageal varices.

On admission, all patients were subjected to a detailed history, thorough clinical examination, laboratory and biochemical investigations (including bone marrow aspiration), abdominal ultrasonography, color-coded duplex scanning of the portal circulation, and upper gastrointestinal endoscopy (UGIT). Abdominal computed tomography (CT) scans, with oral and intravenous contrast, were done on patients belonging to group I before and after embolization. The splenic index was then calculated by multiplying the length, width, and height of the spleen (each expressed in centimeters).

The collected data were recorded in special preformed sheets for statistical analysis and included the patient's demography, presentation, clinical examination, various hematologic and biochemical investigations, results of UGIT endoscopy, bone marrow aspiration, abdominal ultrasonography, color-oded duplex scanning of the portal circulation, abdominal CT scan, the calculated splenic index, information about a past history of similar attacks, and previous sclerotherapy. The clinical course of the patients, requirements of blood transfusion, complications, and cause of death (if it occurred) were recorded. The Child-Pugh severity grading system was used to assess all patients [5].

Patients with hypocellular or infiltrative bone marrow disease, ischemic heart disease, renal failure, or malignant disease; medically unstable patients; those with a known allergy to contrast medium; and those with advanced liver disease were excluded from the study. Informed consent was obtained from all patients.

Procedure for partial splenic embolization

Under strict aseptic conditions, a percutaneous femoral artery approach was used for superselective catheterization of the splenic artery under local anesthesia using xylocaine 2%. The puncture was done using a Seldinger needle (18 gauge = 0.042 inch) angled parallel to the course of the femoral artery. Once good pulsatile blood returned through the needle, the stylet was removed, and a guidewire (0.032 inch) was gently advanced up to the lower abdominal aorta under fluoroscopic guidance. Then, the needle was removed, and a 5F sheath was inserted along the guidewire and fixed in place. The position of the sheath in the femoral artery was confirmed by saline injection and blood aspiration. A four or five copra head catheter was advanced along the guidewire with selective catheterization of the celiac axis and splenic artery.

A preliminary splenic angiogram was obtained to determine the configuration of splenic artery and the location of its pancreatic branches. The arterial phase showed the tortuosity and branches of the splenic artery. These data were important to predict the difficulty of the procedure. The capillary phase was used to show the size of the spleen. The catheter was then advanced so its tip was located distal to the last major pancreatic branch to minimize the risk of pancreatitis. The injection of one or two of the polar arteries (according to their size) was sufficient to embolize at least 50% of the splenic parenchyma. The guidewire was removed, and the embolic agent was gently injected through the catheter. The injection was done very slowly to avoid its reflux. The embolic agent used in this study was polyvinyl alcohol (PVA) in contour particles (Boston Scientific, Natick, MA, USA) ranging from 250 to 355 μm. During embolization, small amounts of contrast material were periodically injected through the catheter to monitor the flow distribution in the spleen.

After the embolization was complete, splenic angiography was done to confirm a reduction of approximately 50% of the splenic blood flow. In most of our patients, one vial of PVA was sufficient to embolize at least 50% of the parenchyma of the spleen. After that, the catheter was irrigated with saline and removed. The site of puncture was compressed for about 15 minutes. All patients remained in hospital after the procedure until the postembolization syndrome or any other significant complication had disappeared. Supported care included high fluid intake, systemic prophylaxis with intravenous antibiotics, cefoperazone (1 g/12 h) for 5 days, and adapted analgesia using nonsteroidal antiinflammatory drugs (NSAIDs).

In group 2 patients randomized for splenectomy and/or devascularization with left gastric ligation, polyvalent pneumococcal vaccine (Pneumovax) was administered 1 week before the operation, and a prophylactic systemic intravenous antibiotic was given (1 g cefoperazone) with induction of anesthesia. Abdominal ultrasonography was performed on postoperative day 7 (POD 7) to exclude portal vein thrombosis, and sutures were removed on POD 10.

All patients in both groups were followed up after discharge at 2 weeks, 3 months, and 6 months. At each visit, a clinical examination and hematologic and biochemical investigations were done. For group 1 patients, CT scans with intravenous and oral contrast were obtained at each visit to assess the extent of splenic infarction and to determine the reduction of splenic size after embolization. We considered the response at 2 weeks as the initial response and the response at 3 months as the long-term response. The follow-up period ranged from 6 to 38 months (mean 20 months). The obtained data were recorded in the preformed sheet for statistical analysis and included the hospital stay, clinical course, morbidity, and mortality.

Statistical analysis

Data were analyzed using SPSS (Statistical Package for Social Science) version 10 (SPSS, Chicago, IL, USA). Qualitative variables were presented as the number and percent. The chi-squared or Fischer's exact test was used for comparison between groups, as appropriate. Quantitative variables were tested for normality distribution by the Kolmogrov–Smearnov test. These variables are presented as the mean ± standard deviation (SD). For normally distributed variables, the unpaired Student's *t*-test was used for comparison between groups, and the paired Student's *t*-test was used for pre- and postintervention comparison. For nonparametric variables, the Mann–Whitney test was used for group comparison. A value of $p \leq 0.05$ was considered statistically significant.

Results

This prospective randomized study was conducted on 40 patients with a diagnosis of hypersplenism secondary to cirrhosis who were admitted to the General Surgery Department, Mansoura University Hospital over a period of 30 months. Their mean age was 39 years (range 18–55 years), and male sex comprised the majority (Table 1). Patients were divided into two groups. The first group of patients underwent partial splenic embolization, and the second group underwent splenectomy with or without devascularization and left gastric ligation according to the presence or absence of esophageal varices. Preoperative hematologic and biochemical investigations were comparable in the two groups except for the platelet count, which was significantly lower in the PSE group (Table 2).

Table 1 Patients' demographics, clinical presentation, bone marrow biopsy, and gastroesophageal endoscopy ($n = 40$)

Parameter	No.
Patient characteristics	
Age (years), mean ± SD	39.05 ± 8.3
Male sex	29 (72.5%)
Clinical presentation	
Left upper abdominal pain	29 (72.5%)
Hematemesis	20 (50.0%)
Epistaxis	6 (15.0%)
Ecchymosis	3 (7.5%)
Easy fatigability	18 (45.0%)
Pallor	22 (55.0%)
Splenomegaly	40 (100%)
Child-Pugh grading (A/B)	35 (87.5%)/5 (12.5%)
Bone marrow biopsy	
Normocellular	13 (32.5%)
Hypercellular	27 (67.5%)
Gastroesophageal endoscopy	
Esophageal varices	30 (75.0%)
Grades I/II/III	9/14/7

Partial splenic embolization was performed in group 1 patients. Splenic artery angiography was performed prior to embolization. During the arterial phase, the course, shape, and branches of the splenic artery were evaluated (Fig. 1). The capillary phase (Fig. 2) revealed the splenic size and demonstrated an absence of any hypervascular or hypovascular masses. Postembolization angiography was done to demonstrate the nonperfused areas of the spleen due to occlusion of its arterial supply (Figs. 3, 4). Postembolization CT scanning with intravenous and oral contrast was done to evaluate the degree of splenic infarction and to correlate with the angiographic findings (Figs. 5, 6).

Partial splenic embolization was successfully performed in all patients, with marked improvement in the platelet count and leukocytic count at 2 weeks, 3 months, and 6 months after the procedure (Table 3). The increase in platelet and leukocytic counts started immediately after the procedure and reached a peak at 2 weeks, followed by a progressive decrease in cell counts; however, their values remained significantly higher than before PSE.

There was also marked improvement in platelet and leukocytic counts in the splenectomy group, and they had increased significantly more than those in the PSE group at 3 and 6 months (Table 4). There was significant decrease in splenic volume after PSE. The average volume of the

Table 2 Preoperative hematologic and biochemical investigations

Variable	Group 1 (PSE) ($n = 20$)	Group 2 (splenectomy) ($n = 20$)	p
Platelets (1000/mm^3)	39.7 ± 9.7	47.15 ± 10.3	0.023
WBC count (1000/mm^3)	3.3 ± 0.7	2.78 ± 1.1	0.238
Hemoglobin (g/dl)	9.72 ± 1.6	10.52 ± 1.9	0.168
Serum creatinine (mg/dl)	0.9 ± 0.14	0.85 ± 0.22	0.272
ALT (IU/dl)	46.7 ± 14.2	40.0 ± 15.4	0.253
AST (IU/dl)	43.3 ± 17.0	37.8 ± 16.4	0.221
Serum albumin (g/dl)	3.6 ± 0.66	3.7 ± 0.61	0.511
Serum bilirubin (mg/dl)	1.2 ± 0.64	0.95 ± 0.31	0.113
Prothrombin concentration (%)	75.0 ± 13.29	78.8 ± 8.42	0.624
INR	1.3 ± 0.21	1.2 ± 0.13	0.813

PSE partial splenic embolization, *WBC* white blood cells, *ALT* alanine aminotransferase, *AST* aspartate aminotransferase, *INR* International Normalized Ratio (prothrombin)

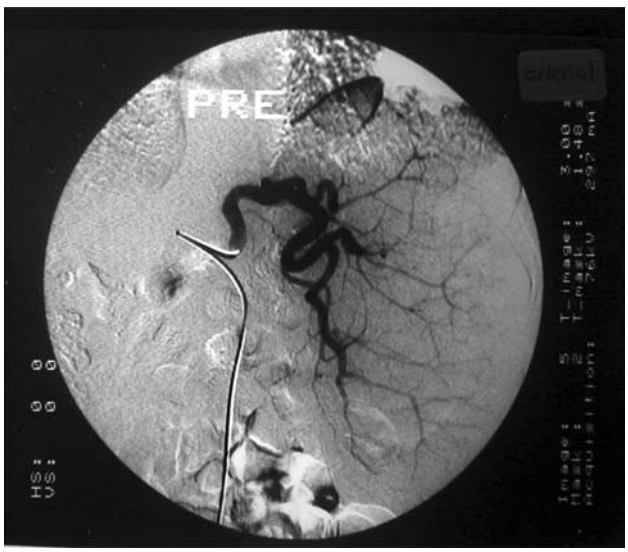

Fig. 1 Arterial phase of splenic artery angiography shows branches and the course of the splenic artery

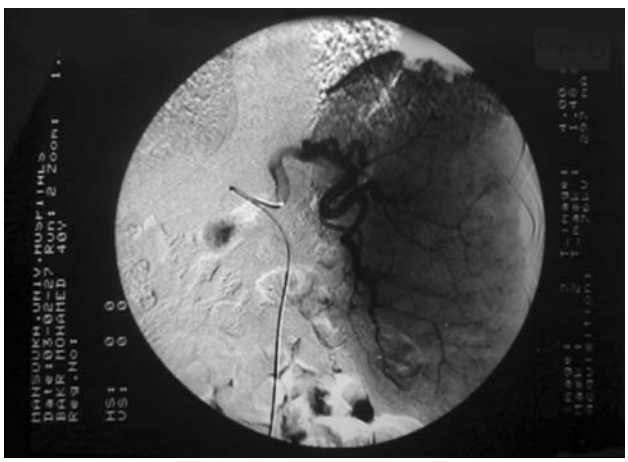

Fig. 2 Capillary phase of splenic artery angiography demonstrates the size of the spleen and an absence of focal lesions

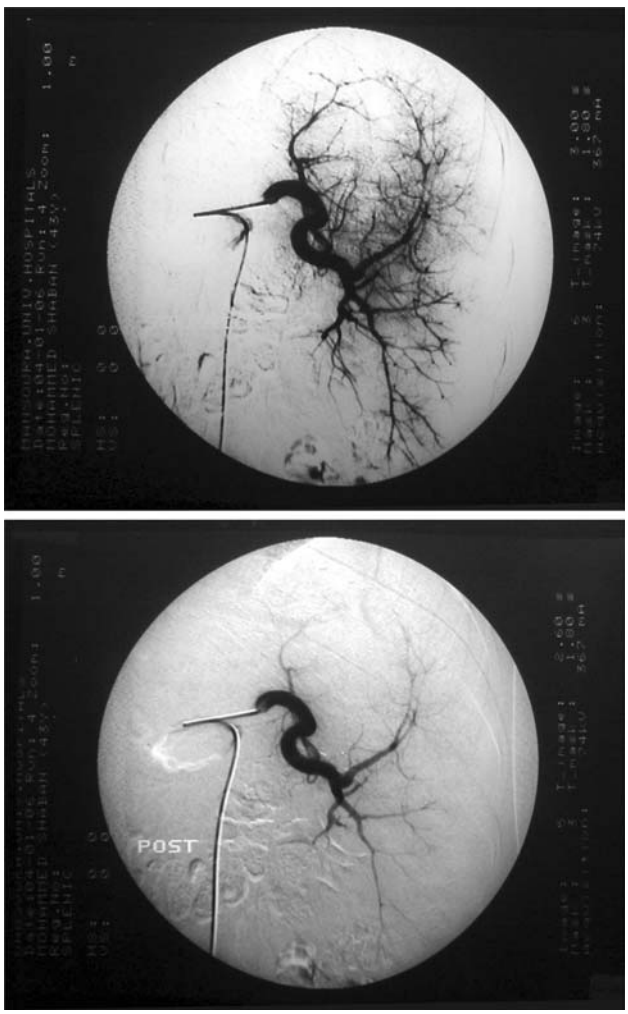

Fig. 3 Splenic artery angiography before (**a**) and after (**b**) embolization

spleen in group 1 patients before embolization was 1885 ± 155 cm^3 (range 933–2120 cm^3). The average volume of the spleen 6 months after PSE was

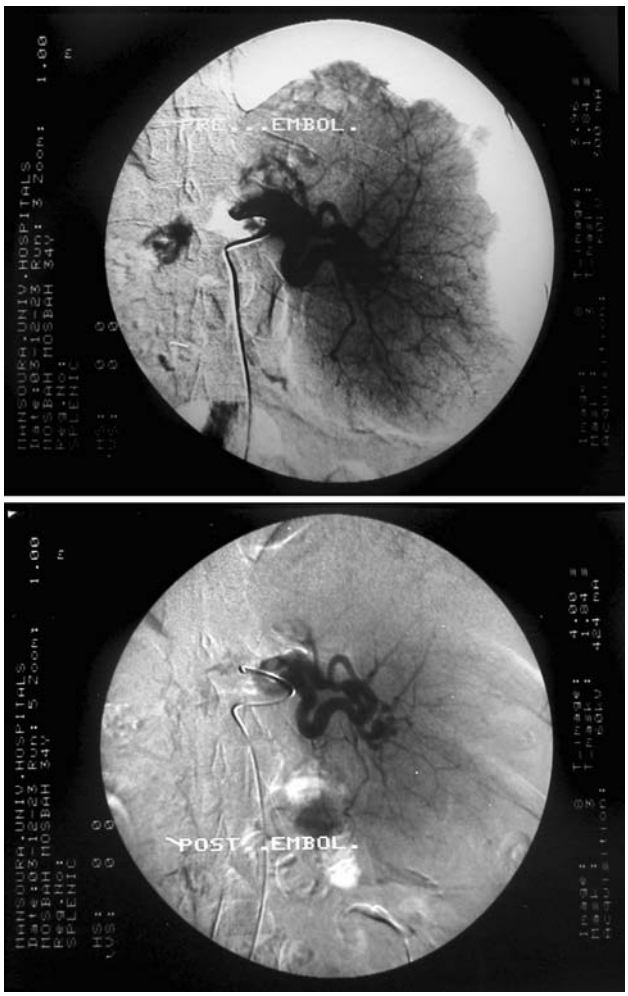

Fig. 4 Splenic artery angiography before (**a**) and after (**b**) embolization

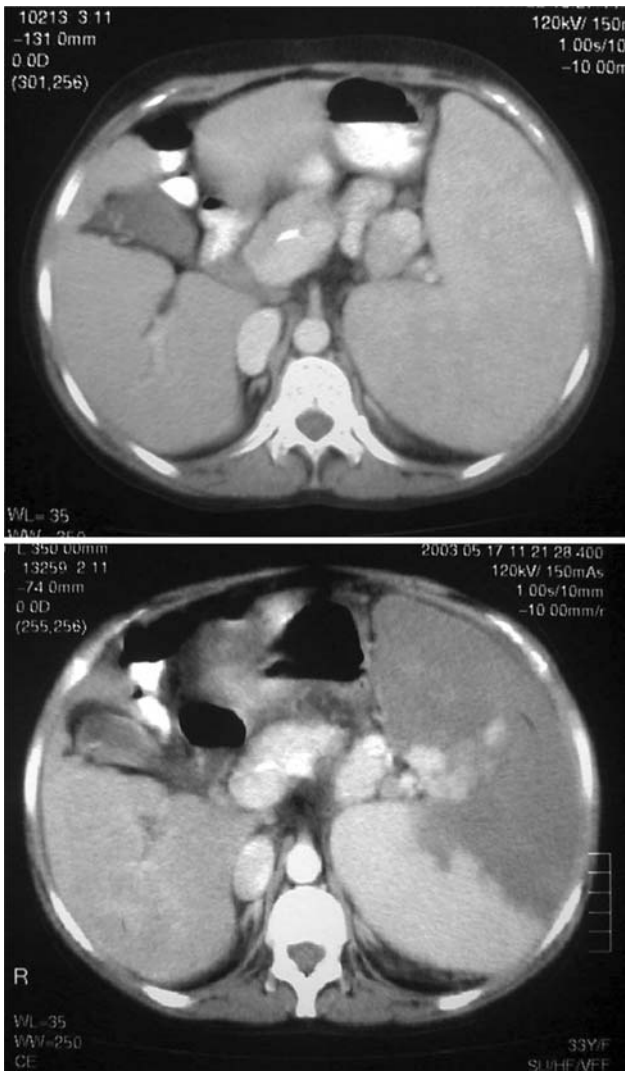

Fig. 5 Computed tomography (CT) scan of the abdomen with intravenous and oral contrast before embolization (**a**) and 2 weeks after partial splenic embolization (PSE). The infarcted area of the spleen is the low-density region

1412 ± 174 cm^3 (range 718–1825 cm^3). Almost all of patients in the first group had problems related to postembolization syndrome. Pain was mild (7 patients) and relieved by the third day, moderate (11 patients) and relieved by simple analgesics (NSAIDs) and abated completely by the fifth day, and severe (2 patients) and was relieved by the seventh day. Fever was mild (<38°C) in 14 patients, moderate (<39°C) in 3 patients, and high (≥39°C) in 2 patients. One of the two with a high fever proved to have a splenic abscess, which was treated by splenectomy with a good outcome; and the other patient proved to have portal vein thrombosis treated conservatively with a good outcome. Nausea developed in six patients but disappeared by the second day. Vomiting occurred in three patients; it was treated by antiemetic, and it abated completely by the third day. Blood transfusion was not required in the first group. In contrast, 11 patients in the second group required blood transfusion (1–4 units).

Postoperative complications were comparable in the two groups (Table 5); however, pain was significantly reduced in the first group. Also, the operating time and hospital stay were significantly reduced in the first group. One death occurred in the first group during the first postembolization day due to myocardial infarction. In the second group, one of the three patients who developed portal vein thrombosis experienced an acute abdomen 4 months postoperatively and underwent operative intervention with resection anastomosis of 1.5 m of small bowel due to mesenteric vascular occlusion, with a good outcome.

Discussion

This prospective randomized study was conducted on 40 patients with hypersplenism secondary to chronic liver disease. They were divided into two groups. The first group

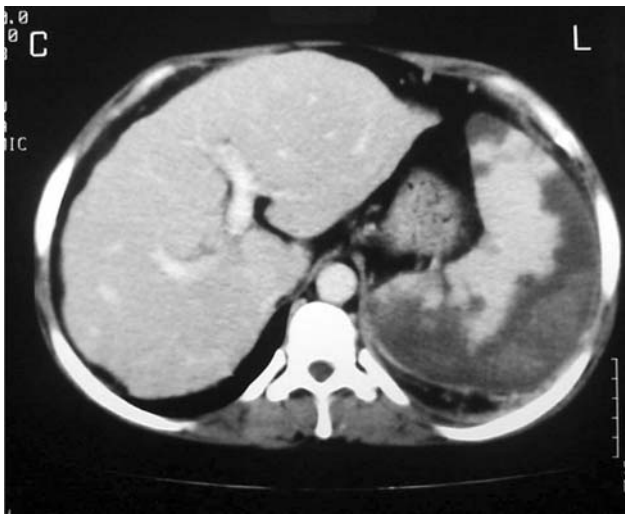

Fig. 6 CT scan of the abdomen with intravenous and oral contrast after PSE shows the areas of splenic infarction

included 20 patients who underwent PSE, and the second group included 20 patients who underwent splenectomy with or without devascularization and left gastric ligation.

Partial splenic embolization was done under local anesthesia using PVA in contour particles ranging from 250 to 355 μm under completely aseptic measures with intravenous antibiotic before and after the procedure. Kumpe and coworkers [6] used general anesthesia during embolization. For every embolization procedure, inadvertent passage of the embolic material to the vessels of the nontarget organs is the most dangerous complication. During splenic embolization, this complication may occur in pancreatic branches of the splenic artery. To avoid this potentially life-threatening complication, good knowledge of the vascular anatomy of the splenic artery and its branches is required. Of particular importance is the location of the last pancreatic branch of the splenic artery, as the tip of the catheter should be distal to the origin of this vessel to achieve safe embolization. Sindel and colleagues [7], in their study to measure the average distance between the origin of the last pancreatic branch and the splenic hilum, concluded that to avoid the risk of pancreatitis embolic materials should be delivered through a catheter whose tip is located in the distal 3.07 cm of the splenic artery. PSE under antibiotic protection has been suggested to avoid the occurrence of splenic abscess [8].

The extent of embolization seems to be critical for long-term efficacy of PSE. Embolization of less than 50% of the splenic mass was almost always associated with a relapse of hypersplenism [4]. In contrast, relapse did not occur among patients who had ≥50% of their spleen embolized. Romano and coworkers [9] aimed to embolize and infarct a higher percentage of splenic parenchyma (75–80%). All patients tolerated the procedure. N'Kontchou and colleagues [10] found that the volume of splenic necrosis was more than 70% in the two patients who died in their series, suggesting that overembolization could result in a severe outcome. Two other patients with similar necrosis also had significant clinical complications (i.e., ascites or portal thrombosis). In our study, we aimed at reducing the splenic blood flow by at least 50%. The extent of embolization was assessed by evaluating the peripheral amputation of segmental branches on digital subtraction angiography. In most of our patients, one vial of the embolizing material was sufficient to embolize at least 50% of the splenic parenchyma. N'Kontchou and coworkers [10] strictly advised that PSE should be limited to 50% of the splenic volume.

Postembolization syndrome is the most common side effect of PSE [11]. It consists of left abdominal pain, fever, malaise, and gastrointestinal symptoms in the absence of a positive blood culture or other evidence of infection. It is a self-limiting, benign phenomenon that usually indicates extensive tissue necrosis and/or local intravascular thrombosis. It is unavoidable [12]. Mild to moderate left-sided abdominal pain occurred in most of our patients and was relieved by NSAIDs. It was severe in two patients, who required morphine administration. N'Kontchou and coworkers [10] reported that abdominal pain occurred in 25 patients (78%) and was severe enough to require morphine administration in 24 patients.

Fever always accompanied pain in our study. It was mild in most of them, and exceeded 39°C in only two patients. Kumpe and colleagues [6] reported the occurrence of fever in 9 of their 10 patients. They attributed fever to

Table 3 Hematological changes pre-PSE and post-PSE

Variable	Pre-PSE	Post-PSE		
		2 Weeks	3 Months	6 Months
Platelet count (1000/mm^3)	39.7 ± 9.7	211.53 ± 36.21*	185.26 ± 13.5*	146.53 ± 50.0*
WBC count (1000/dl)	3.30 ± 0.7	12.61 ± 2.63*	6.82 ± 1.89*	6.36 ± 2.29*
Hemoglobin (g/dl)	9.72 ± 1.6	9.78 ± 1.18**	10.2 ± 0.92***	11.3 ± 1.17*

Results are the mean ± SD

* $p = 0.001$; ** $p = 0.941$; *** $p = 0.213$

Table 4 Preoperative and postoperative platelet count, leukocytic count, and hemoglobin concentration in the two groups

Variable	Group 1 ($n = 20$)	Group 2 ($n = 20$)	p
Platelets (1000/ml)			
Preoperative	39.7 ± 9.7	47.15 ± 10.3	0.023
Postoperative			
2 Weeks	211.526 ± 36.21	240.7 ± 52.02	0.378
3 Months	185.263 ± 13.5	259.85 ± 49.68	0.009
6 Months	146.526 ± 50.0	322.35 ± 51.06	0.001
Leukocytic count (1000/ml)			
Preoperative	3.3 ± 0.7	2.78 ± 1.1	0.238
Postoperative			
2 Weeks	12.61 ± 2.63	7.745 ± 1.96	0.001
3 Months	6.821 ± 1.89	8.525 ± 1.69	0.005
6 Months	6.363 ± 2.29	8.48 ± 1.51	0.002
Hemoglobin (gm/dl)			
Preoperative	9.72 ± 1.6	10.5 ± 1.9	0.168
Postoperative			
2 Weeks	9.78 ± 1.18	10.5 ± 1.1	0.062
3 Months	10.2 ± 0.92	11.2 ± 0.8	0.092
6 Months	11.3 ± 1.17	11.6 ± 0.8	0.071

Values are means ± SD

infarction of splenic tissues, and they noted that the degree of fever was related to the extent of infarction in the absence of infection.

The efficacy of PSE observed in our study confirms the results of previously published studies in patients with hypersplenism [4, 10–13]. In particular, there was marked improvement in the platelet count and leukocytic count in all patients in the initial and long-term responses; there was also an insignificant initial increase in hemoglobin concentration that was followed by a significant increase at 6 months. A progressive decrease in the platelet and leukocytic counts was observed, but the values remained significantly higher than before PSE (platelets > 146,000/mm^3). Romano and coworkers [9] observed a significant, progressive increase in platelet counts in all patients; the platelet count increased, on average, by 120,000/mm^3 (range 95,000–145,000/mm^3). N'Kontchou and colleagues [10] also found that both platelet and leukocytic counts had markedly improved at POD 7 after PSE. A progressive decrease in cell counts was also observed during the follow-up period, but the values remained significantly higher than before PSE at 1 and 6 months (> 80,000/mm^3).

Partial splenic embolization increases platelet counts via two mechanisms: by (1) reducing the splenic size, thereby reducing the trapping of thrombocytes in the embolized spleen; and (2) decreasing levels of platelet-associated immunoglobulin G (IgG). Reduction of platelet-associated IgG leads to decreased immunologic induction of thrombocytopenia [1]. The increase in leukocytes after PSE can be attributed to the activation of body defense mechanisms against infarcted splenic tissues [14], which can explain the significantly greater degree of increase of leukocytes after PSE than after splenectomy in our study. Sakai and colleagues [4] recorded improved leukocytic and platelet counts after PSE in 16 of 17 patients in their study, and the improvement persisted for at least 1 year.

In our study, the volume of the spleen was reduced 6 months after PSE by about 25% as measured by follow up CT scans with oral and intravenous contrast. Romano and coworkers [9] found that the reduction in the volume of the spleen ranged from 59% to 78% at 18 months after PSE as measured by contrast-enhanced spiral CT. Partial splenic embolization causes permanent ablation of sufficient splenic parenchyma to allow subsequent improvement of the

Table 5 Operating time, hospital stay, blood transfusion, morbidity, and mortality in the two groups

Variable	Group 1 ($n = 20$)	Group 2 ($n = 20$)	p
Operating time (min), mean ± SD	21.33 ± 4.16	85.2 ± 29.7	0.001
Hospital stay (days), mean ± SD	2.05 ± 0.40	4.3 ± 1.05	0.001
Blood transfusion	0	11.0 (55%)	0.001
High fever (>39°C)	2 (10%)	4 (20%)	0.376
Severe pain	2 (10%)	20 (100%)	0.001
Portal vein thrombosis	1 (5%)	3 (15%)	0.292
Mesenteric vascular occlusion	–	1 (5%)	0.311
Recurrent hematemesis	–	1 (5%)	0.311
Ascites	2 (10%)	2 (10%)	1.0
Left pleural effusion	2 (10%)	–	0.147
Atelectasis	–	1 (5%)	0.311
Splenic abscess	1 (5%)	–	0.311
Wound hematoma	–	1 (5%)	0.311
Mortality	1 (5%)	–	0.311

hematologic status [2]. In adults, splenic regeneration after PSE is quite rare. It is possible that children have a greater capacity for splenic regeneration than adults. If clinical symptoms recur, the repeatability of PSE offers an advantage [6]. Kimura and colleagues [15] found that repeat PSE was effective in patients with chronic idiopathic thrombocytopenic purpura who responded to the initial PSE but later relapsed.

In our study, improvement in platelets, leukocytes, and hemoglobin occurred in all patients who underwent splenectomy. These results are similar to that of Sunderson and associates [16], who found improvement in the three cell lines in all patients. Letoquart and coworkers [17] observed a positive response in 94% of patients.

Severe complications, such as splenic abscess, rupture of the spleen, pneumonia, pleural effusion, and septicemia, have been previously reported after PSE [18, 19]. In our study, PSE-related complications occurred in four patients. Portal vein thrombosis occurred in one patient and was detected by imaging on POD 7; it eventually resolved after anticoagulation. Romano and associates [9] had no cases of portal vein thrombosis, and N'kontchou and colleagues [10] reported 2 in a series of 32 patients.

Portal vein thrombosis occurred in three patients after splenectomy in the present series. One developed a mesenteric vascular occlusion 4 months after the operation and was managed by resection anastomosis with a good outcome. Ezzat and associates [20] reported an incidence of 20% portal vein thrombosis after splenectomy, and Abou El Hoda and colleagues [21] reported an incidence of 30%. Removal of the splenic venous contribution to portal flow with a subsequent reduction of blood flow in the portal vein [22] and the presence of thrombocytosis could explain the high incidence of portal vein thrombosis after splenectomy [23]. On the other hand, Sunderson and colleagues [16] observed no thromboses in any patient despite their high platelet counts.

Splenic abscess is one of the most dangerous complications that may follow PSE. In this study, splenic abscess occurred in one patient. It was discovered by CT scan as a large area of cystic necrosis of the spleen 1 month after the procedure and was accompanied by fever, ascites, and left pleural effusion. The patient was treated by splenectomy with a good outcome. The most suitable therapeutic approach for splenic abscess is still under debate. As suggested, percutaneous puncture and drainage of the abscess should be performed early to obtain bacterial identification and to evacuate most of the purulent collection [24]. This treatment can result in a favorable outcome [4]. N'Kontchou and colleagues [10] reported two patients with a splenic abscess treated by percutaneous puncture and drainage with initial improvement, followed by their death from septic shock as a result of recurrent infection due to poor penetration of antibiotics into these cystic lesions. These authors suggested early splenectomy if the condition failed to resolve after percutaneous drainage.

In our study, two patients developed ascites after PSE. One of them was associated with splenic abscess and left pleural effusion. N'Kontchou and colleagues [10] found transient ascites in two patients that resolved with medical treatment. The other patient with left pleural effusion was due to irritation of the undersurface of the diaphragm by the infarcted spleen. This occurred only in the patient with an upper pole infarction. Romano and associates [9] reported the same complication, whereas Gerlock and colleagues [25] found that no patients developed pleural effusion.

There was one death after PSE. It occurred on the first postembolization day and was due to myocardial infarction. Sakai and coworkers [4] reported one death in a series of 17 patients, and it was due to acute-on-chronic liver disease. Xu and associates [26] reported one death because of pulmonary embolism, and N'Kontchou and associates [10] reported two deaths due to a splenic abscess and septic shock, respectively. No deaths occurred in our group 2 patients. Ezzat and colleagues [20] reported an incidence of 3.1% hospital mortality.

Conclusions

Partial splenic embolization (PSE) can be an effective therapeutic alternative to splenectomy for management of hypersplenism secondary to cirrhosis. It is a simple, rapid procedure, easily performed under local anesthesia, incurs less morbidity, and there is no need for blood transfusion. Moreover, the splenic vein is preserved for further shunt operation if required, and a portion of functional splenic tissue is left in place to safeguard against overwhelming infection. This study has some limitations: The number of patients was small, and they belonged to Child class A or B only. To avoid severe, lethal complications, especially splenic abscess, it is suggested that the splenic necrosis be kept at 50% and that when a splenic abscess is diagnosed it should be treated with early splenectomy.

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