

Role of Partial Splenic Artery Embolization (PSE) in the Treatment of Hypersplenism

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Abstract

Background: Hypersplenism mainly manifest by thrombocytopenia with or without obvious bleeding tendency that become an obstacle to undergo any surgical intervention or receive some therapeutic regime. This dilemma can be solved using the evolving partial splenic artery embolization (PSE) techniques.

Aim of Study: The primary aim is to evaluate the role and efficacy of PSE in the treatment of hypersplenism.

Patients and Methods: In the period between March 2016 and August 2018, this prospective case series study was conducted and included 26 patients (11 males and 15 females) with their age ranging from 14 to 72 years old. All patients suffered from hypersplenism secondary to liver cirrhosis and had thrombocytopenia ranging from 20,000/ μ L to 83,000/ μ L, with 17 of them had associated leucopenia ranging from 1,250/ μ L to 3,420/ μ L. 20 patients had hypercellular and 6 patients had normocellular bone marrow. They all underwent one session of PSE by the usage of two sizes of either PVA or Microspheres with the extent of planned embolization volume was set between 30%-70% of initial spleen size.

Results: Up to 1 year follow-up, the platelet counts rose significantly with patients reached their normal values in 1st, 3rd, 6th months and 1st year follow-ups were 80.8%, 71.4%, 91.7% and 83.3% respectively. In all 17 patients who had associated leucopenia, their leucocytic counts rose significantly with patients reached their normal values in 1st, 3rd, 6th months and 1st year follow-ups were 88.2%, 87.5%, 83.3% and 75% respectively. No significant improvement regarding the RBC counts in all patients. Among the 24 patients who had clinical manifestations; 22 (91.7%) of them showed clinical improvement and 2 (8.3%) patients showed less improvement.

Conclusion: PSE with careful care is an effective non-surgical minimally invasive procedure in avoiding the potential post-procedure complications and achieving remarkable hematologic response on controlling hypersplenism.

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Key Words: Partial Splenic Embolization (PSE) – Hypersplenism – Splenomegaly – Thrombocytopenia.

Introduction

HYPERSPLENISM refers to a group of syndromes that involve splenomegaly and peripheral cytopenia of various causes. Hypersplenism can be caused by many diseases which, in turn, affects the prognosis of hypersplenism. The most common hypersplenism is secondary to post-viral hepatitis cirrhotic liver with portal hypertension [1].

The spleen is a major source of antibodies and lymphocytes and augments the phagocytosis of white cells. Hypersplenism promotes the phagocytosis and destruction of blood cells. Splenectomy may eliminate hypersplenism-induced blood cell destruction, but the spleen has an important role in the immune system. Splenectomy thus increases the risk of systemic infection [2].

Operative splenectomy have major complications include portal vein thrombosis and sepsis. Additionally, some cirrhotic patients may be poor surgical candidates, thus necessitating alternative approaches to splenectomy [3].

The surgical cohort had longer procedure times, longer hospitalizations, required transfusions more frequently and reported more post-procedural pain

[4].

PSE is a non-surgical procedure developed to treat hypersplenism as a result of hepatic disease and thus avoid the disadvantages of splenectomy

[5].

PSE is effective in improving platelet count in patients with chronic liver disease and hypersplen-

ism. White blood cell count also showed significant improvement [6].

PSE has a direct effect on the spleen and may cause improved hepatic function. The improvement may be due to an immunologic mechanism or because of decreased splenic venous flow leading to compensatory increase in the flow in hepatic artery and superior mesenteric artery and vein, which may result in more nutritious flow of blood to the liver [7].

Patients and Methods

This prospective case series study was conducted in the period between March 2016 and August 2018 and included 26 patients (11 men and 15 women) with their age ranging from 14 to 72 years old with mean age of 51 years. All patients had hypersplenism secondary to liver cirrhosis due to viral hepatitis with splenomegaly ranging from 18 to 29cm. They all underwent one session of PSE at the Intervention Radiology Department, Nasser Institute Hospital, with the primary aim to eliminate hypersplenism or alternatively to down-grade the severity of the disease. All patients were referred from inpatient or outpatient clinic of Hepato-Gastro-Enterology Department, Nasser Institute Hospital. This study was conducted upon the approval of the ethical committee of our hospital.

Inclusion criteria: Adult patients suffering from hypersplenism 2ry to liver cirrhosis and showing splenomegaly and thrombocytopenia (platelet counts $<100,000/\mu\text{L}$) with or without leucopenia (WBC $<3500/\mu\text{L}$) associated with either hyper or normocellular bone marrow without infiltration by malignant cells and up to class "B" score "8" of Child-Pugh classification.

Exclusion criteria: Patients with any other possible hematological condition leading to thrombocytopenia, malignant disease, infectious disease, ischemic heart disease, heart failure, hepatic encephalopathy, class "C" Child-Pugh classification, prothrombin activity $\leq 48\%$, total bilirubin level $\geq 81.4 \mu\text{mol/L}$ ($=4.76\text{mg/dl}$), moderate/large amount of ascites and hypo-cellular bone marrow.

After written consent, all patients were subjected to a thorough history taking with special stress on bleeding tendency, variceal hemorrhage, repeated infections and previous blood transfusion. Thorough physical examination was done and laboratory investigations including CBC, coagulation profile and liver and renal functions were carried out.

Twenty four out of twenty six patients were complaining from non-spontaneous bleeding tendency, while two patients were not suffering from bleeding tendency but discovered accidentally to have prolonged bleeding time in their coagulation profile. None of the patients suffered from variceal hemorrhage.

Nineteen patients needed variable amount of blood components transfusions and five patients were complaining from repeated infections.

According to the Child-Pugh classification, there were 19 patients with class "A" and 7 patients with class "B". None of the patients were in class "C".

According to their CBC analysis; 8 patients had monocytopenia, 8 patients had bicytopenia and 10 patients had pancytopenia. All patients had thrombocytopenia with their PLTs ranging from $20,000/\mu\text{L}$ up to $83,000/\mu\text{L}$, with or without leucopenia. 17 patients had leucopenia ranging from $1,250/\mu\text{L}$ up to $3,420/\mu\text{L}$.

Twenty patients had hypercellular bone marrow, while 6 patients had normo-cellular bone marrow. None of the cases had hypo-cellular bone marrow or bone marrow infiltration.

PSE procedure:

1- Pre-procedure preparation: The day before examination, all patients were admitted to Nasser Institute Hospital in the Hepato-Gastro-Enterology Department. All their previous investigations were revised and they started antibiotics as follows: Rocephin 2gm IV every 24hrs and Metronidazole 500mg IV every 8hrs.

A baseline CBC count, coagulation profile, liver and renal functions were obtained earlier before the examination.

Six units of fresh frozen plasma were given in five patients suffering from low coagulation profile at the early morning of the examination day.

2- Technique: Using the Seldinger's technique, the common femoral artery was punctured under local anaesthesia with placement of 5F introducer sheath, selective catheterization and angiograms of the celiac trunk followed by splenic artery were obtained using 5F Cobra catheter (Optitorque-Terumo, Tokyo, Japan) Fig. (1). An alternative reversed curve "Simons" curve II guiding catheter (Optitorque-Terumo, Tokyo, Japan) was used in some cases if the celiac trunk was elongated or difficult to access.

The Cobra catheter was advanced distally as much as possible near the splenic hilum and diagnostic angiograms were obtained to define the

arterial anatomical distribution and demonstrate the pre-PSE baseline splenic parenchymal blush (splenogram) Figs. (2,3).



Fig. (1): A 41 female hypersplenic patient. The celiac angiogram showing the common celiac trunk arterial branches (Trifurcation).



Fig. (2): Splenic angiogram following distal advancement of the Cobra catheter.



Fig. (3): Delayed splenic angiogram showing splenic parenchyma (Splenogram).

If the Cobra catheter was not feasible to be advanced distally near the splenic hilum owing to marked splenic artery tortuosity, a co-axial microcatheter (Renegade HI-FLO microcatheter, Boston Scientific, USA) was advanced through Cobra catheter to reach the splenic hilum or into desirable intra-splenic branches Fig. (4).

The selective technique was preferentially performed by reaching into one or more of the subdivision arterial branches according to the desirable targeted splenic infarction ratio. This technique was done to avoid non-targeted embolization, it also had better delineation of the accurate infarcted splenic ratio during the procedure. Five cases were embolized non-selectively due to presence of technical problems.

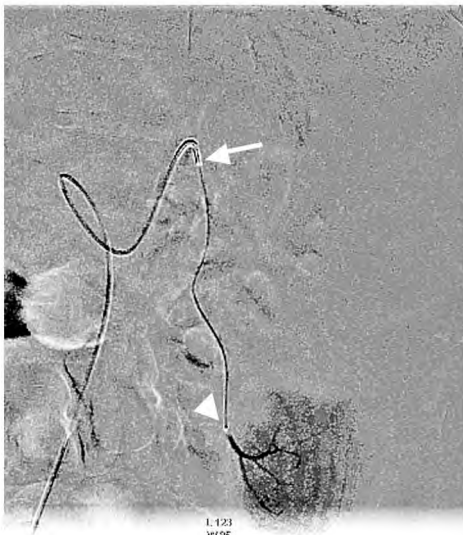


Fig. (4): Super-selective catheterization and angiography of the inferior polar splenic artery showing inferior polar splenic parenchymal blush. The super-selective catheterization was performed using a co-axial Renegade HI-FLO microcatheter passing into the Cobra catheter.

Note: The distal ends of both Cobra catheter (arrow) and Renegade micro-catheter (arrowhead) in this image.

After the guiding catheter or microcatheter was advanced as much as possible into the targeted vascular supply, embolization was performed by injecting two sizes of same embolic material of either Poly-Vinyl Alcohol (PVA) or Embosphere particles soaked within the antibiotic solution. Small size was administered first either PVA 300-500 μ m (Cook, Bloomington, IN, USA)/355-500 μ m (Contour-PVA, Target therapeutics, Boston Scientific USA), or Embosphere 300-500 μ m (Merit Medical Systems Inc., Paris, France). Followed by administration of large size of either PVA 500-700 μ m/500-710 μ m, or Embosphere 500-700 μ m.

Smaller size was injected first to embolize the splenic vascular bed that constitute the functional zone of the spleen causing hypersplenism, then embolizing their vascular supply by the larger one.

The extent of embolization volume was set between 30%-70% of the initial splenogram and was performed progressively under fluoroscopy to avoid reflux and non-target embolization. Administration of the particles was continued followed

by serial control angiograms were obtained till the splenogram demonstrated parenchymal defects between 30%-70% Fig. (5).

At this point the embolization was stopped, the guiding catheter and micro-catheter (if any) as well as the femoral sheath were removed. Manual vascular compression was applied at the puncture site for at least 30 minutes.



Fig. (5): The final control angiogram of the splenic artery showing a large defective parenchymal blush involving the lower half of the spleen consistent with the infarcted parenchymal volume that been estimated about 60% of the total splenic volume.

Note: The preservation of the short gastric arteries arising from the inferior polar splenic artery branches (arrows).

3- Post -procedure care:

All patients were hospitalized for at least the next 5 days after PSE where they continued on the same doses of the antibiotics for about one week; also all patients were given anti-inflammatory and anti-edema; oral Alphintern 2 tab every 8hrs as well as analgesics and antipyretics; oral Paracetamol every 6hrs for 5 to 7 days and the fever chart monitor was followed-up.

One patient needed stronger analgesia; Pethidine 1.5mg/kg body weight was given IV every 6h in the 2nd and 3rd days post-PSE beside the Paracetamol.

The patients were discharged after their hospital stay on the same doses and frequencies of the oral antibiotics till the end of the antibiotic regime.

4- Follow-up:

CBC and coagulation profile were obtained at different times post-PSE. CBC was done 1 day, 1 and 2 weeks after the procedure before patient's discharge.

Then all patients were followed-up after 1, 3 and 6 months from the procedure day respectively, and subsequently follow-up was done once per year or when indicated clinically. A follow-up CBC was revised and compared to the last count and was recorded in the patient's follow-up chart. Coagulation profile were done only once at least one month after the PSE session.

Follow-up U/S was performed shortly after examination. Post-contrast abdominal CT scan was performed in only two cases, one case showed signs of disease recurrence to evaluate the extent of splenic infarction ratio and the other case showed the development of splenic abscess to confirm the diagnosis and to evaluate the response after therapy.

Statistical analysis:

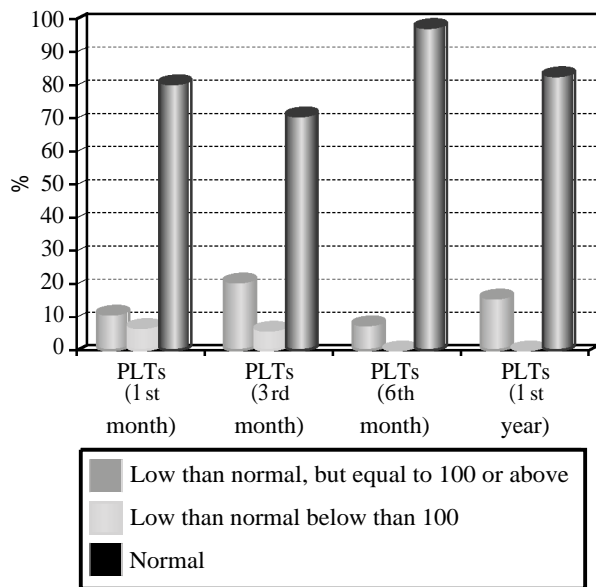
Data were coded and entered using the statistical package SPSS (Statistical Package for the Social Sciences) version 25. Data was summarized using mean, standard deviation, median, minimum and maximum in quantitative data and using frequency (count) and relative frequency (percentage) for categorical data. For comparison of serial measurements within each patient the non-parametric Friedman test and Wilcoxon signed rank test were used. *p*-values less than 0.05 were considered as statistically significant [8].

Results

Follow-up CBC samples were scheduled in the 1st, 3rd and 6th months as well as 1st year following the PSE sessions.

The platelet counts rose significantly and immediately in the next day following PSE and kept rising in the 1st week till reaching the peak in the 2nd week then gradual decline in count starting from 1st month with patients reached their normal values in the 1st, 3rd, 6th months and one year follow-ups were 80.8%, 71.4%, 91.7% and 83.3% respectively, and patients had low platelet counts equal to or above 100,000 were 11.5%, 21.4%, 8.3% and 16.7% respectively, while patients had low platelet counts below 100,000 were 7.7%, 7.1 %, 0% and 0% respectively [see (Table 1) and Column (1)].

The *p*-values of the 1st, 3rd, 6th months and 1st year follow-ups showed significant values of <0.001, 0.083, 0.002 and 0.036 respectively, as compared to the baseline values [see (Table 2) and Curve (1)].



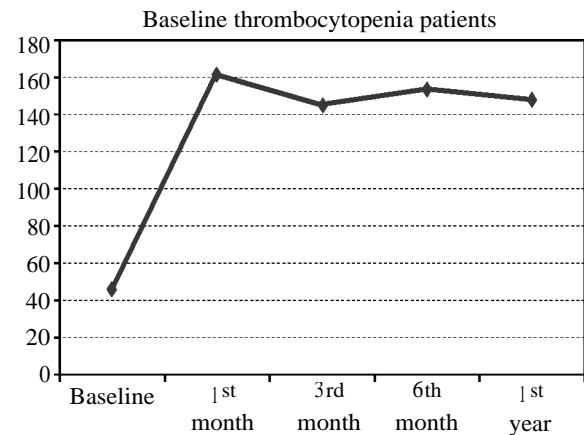
Column (1): Showing post-PSE platelet counts results.

Table (1): Showing post-PSE platelet counts results.

	Count	Column N %
<i>PLTs (1st month):</i>		
• Low than normal, but equal to 100 or above	3	11.5%
• Low than normal below than 100	2	7.7%
• Normal	21	80.8%
<i>PLTs (3rd month):</i>		
• Low than normal, but equal to 100 or above	3	21.4%
• Low than normal below than 100	1	7.1%
• Normal	10	71.4%
<i>PLTs (6th month):</i>		
• Low than normal, but equal to 100 or above	1	8.3%
• Low than normal below than 100	0	0.0%
• Normal	11	91.7%
<i>PLTs (1st year):</i>		
• Low than normal, but equal to 100 or above	1	16.7%
• Low than normal below than 100	0	0.0%
• Normal	5	83.3%

Table (2): Showing post-PSE platelet counts results regarding to mean, standard deviation, median, minimum, maximum and *p*-values.

	Mean	Standard deviation	Median	Minimum	Maximum	<i>p</i> -value
• PLTs (baseline)	44.97	16.14	42.00	20.00	83.00	–
• PLTs (1st month)	160.92	45.23	162.50	46.00	245.00	<0.001
• PLTs (3rd month)	144.07	20.22	145.00	95.00	174.00	0.083
• PLTs (6th month)	152.83	20.88	149.50	105.00	196.00	0.002
• PLTs (1st year)	147.17	8.23	145.50	136.00	158.00	0.036



Curve (1): Showing post-PSE platelet counts follow-up.

In all 17 patients who had leucopenia, their leucocytic counts rose significantly with patients reached their normal values in 1st, 3rd, 6th months and 1st year follow-ups were 88.2%, 87.5%, 83.3% and 75% respectively [see (Table 3)].

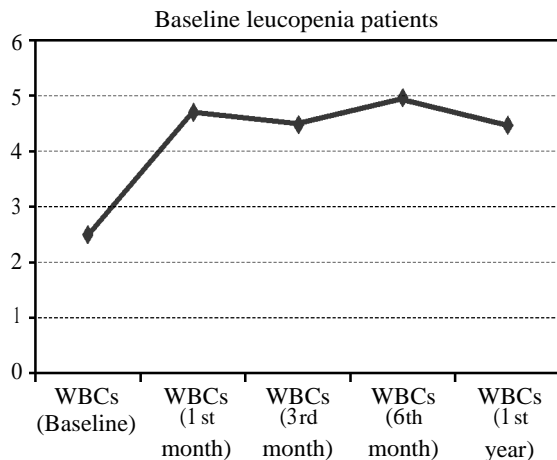
Table (3): Showing post-PSE white blood cell counts results.

	Count	Column N %
<i>WBCs (baseline):</i>		
Low	17	65.4%
Normal	9	34.6%
<i>WBCs (1st month):</i>		
Low	2	11.8%
Normal	15	88.2%
<i>WBCs (3rd month):</i>		
Low	1	12.5%
Normal	7	87.5%
<i>WBCs (6th month):</i>		
Low	1	16.7%
Normal	5	83.3%
<i>WBCs (1st year):</i>		
Low	1	25%
Normal	3	75%

The *p*-values of the 1st, 3rd, 6th months and one year follow-ups showed significant values of <0.001 in all follow-up periods [see (Table 4) and Curve (2)].

Table (4): Showing post-PSE white blood cell counts results regarding to mean, standard deviation, median, minimum, maximum and *p*-values.

	Mean	Standard deviation	Median	Minimum	Maximum	<i>p</i> -value
• WBCs (baseline)	2.48	0.55	2.50	1.25	3.42	
• WBCs (1st month)	4.66	0.95	4.30	3.10	6.30	<0.001
• WBCs (3rd month)	4.44	0.87	4.45	2.73	5.40	<0.001
• WBCs (6th month)	4.90	0.74	5.05	3.70	5.68	<0.001
• WBC (1st year)	4.43	0.52	4.50	3.80	4.90	<0.001



Curve (2): Showing post-PSE WBCs counts follow-up.

Apart from two (7.7%) patients who had developed disease relapse, yet showed mild WBCs and PLTs counts improvement up to one month follow-up.

The RBCs results showed no significant improvement noted in all patients.

Among the 24 patients who had clinical manifestations; 22 (91.7%) of them showed clinical improvement, and 2 (8.3%) patients showed less improvement [see (Table 5) and Chart pie (1)].

On the other hand regarding the coagulation profile results; all (100%) patients showed variable degrees of improvement in the post-PSE results in comparison to the pre-PSE baseline values [see (Table 5) and Chart pie (2)].

While regarding the liver function tests results that were recorded in 19 patients only, 15 (78.9%) of them showed improvement, 3 (15.8%) of them showed deterioration and 1 (5.3%) had same results with no significant change comparable to pre-PSE results [see (Table 5), Chart pie (3) and Column (2)].

Table (5): Showing post-PSE clinical status as well as comparison between pre and post-PSE results of liver functions and coagulation profile.

		Count	%
<i>Clinically (post-PSE):</i>	Less	2	8.3%
	Improved	22	91.7%
<i>Pre liver functions:</i>	Normal	4	20.0%
	Abnormal	16	80.0%
<i>Post liver functions:</i>	Same	1	5.3%
	Improved	15	78.9%
	Deteriorated	3	15.8%
<i>Pre coagulation profile:</i>	Normal	3	15.0%
	Low	17	85.0%
<i>Post coagulation profile:</i>	Improved	20	100.0%

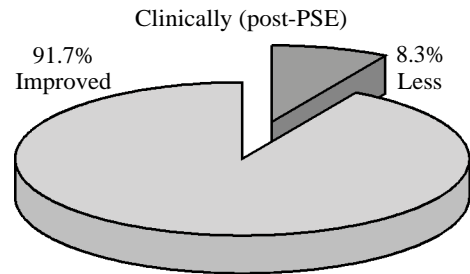


Chart Pie (1): Showing post-PSE clinical status.

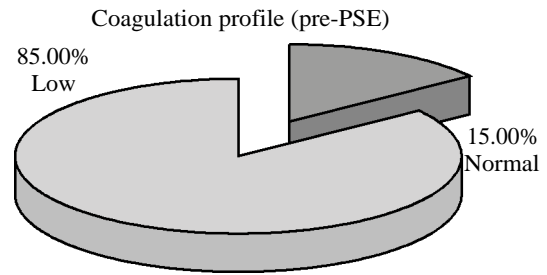


Chart Pie (2): Showing pre-PSE coagulation profile.

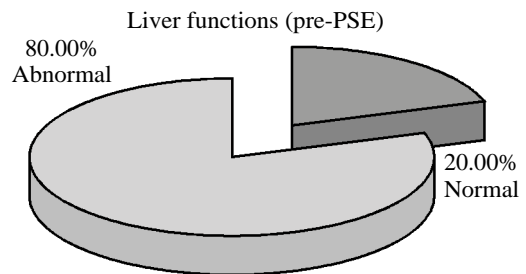
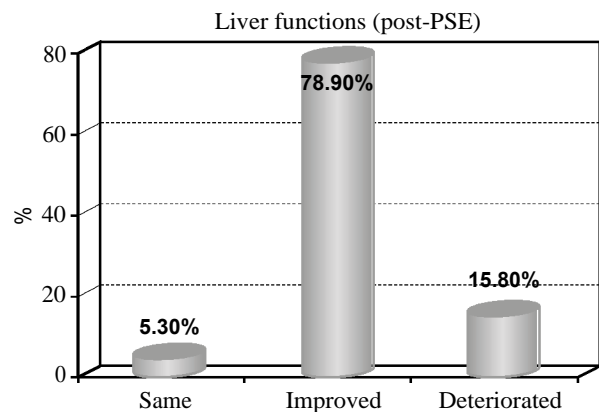


Chart Pie (3): Showing pre-PSE liver functions values.



Column (2): Showing post-PSE liver functions results.

In these 26 patients, the extent of the planned embolization ratio was set between 30-70% of the initial splenic size (mean splenic infarction ratio =48.7%). 12 (46.2%) patients showed splenic infarction ratios between 30-50%, while 14 (53.8%) patients showed splenic infarction ratios between 50-70%. 21 (80.8%) patients were done using selective embolization technique and 5 (19.2%) patients were done using non-selective technique.

The embolic materials used in this study were either Embosphere or PVA particles. 10 (38.5%) patients were injected with Embospheres and 16 (61.5%) patients were injected with PVA particles.

The incidence of overall complications was about 27% (7/26). From those complications, two of them were considered major complications according to the Society of Interventional Radiology, with the incidence was about 7.7% (2/26), including one case developed marked ascites and another case developed splenic abscess, they warranted additional medical care and prolonged hospitalization more than 48 hours.

All (100%) patients experienced one or more symptoms of Post-Embolization Syndrome (PES). Twenty five (96.2%) patients complained of mild to moderate left hypochondrial pain which was easily controlled by light analgesics and one (3.8%) patient had severe pain that warranted medications other than analgesics. Almost all cases 25 (96.2%) patients had mild to moderate fever up to 38.5°C for 2 to 5 days and 1 patient showed no fever.

Eight (30.7%) patients developed free abdominal ascites; one (3.8%) patient developed mild to moderate pleural effusions with underlying minimal to mild basal lung atelectasis and three (11.5%) patients developed sub-capsular splenic collection. They were treated conservatively and needed no intervention, apart from one patient that developed marked abdominal ascites and warranted further medical attention with prolonged hospitalization more than 48 hours.

Although strict aseptic condition was performed and about 55% estimated splenic infarction ratio was done, one (3.8%) patient had developed splenic abscess about three weeks post-PSE presented by recurrent fever exceeding 38.5°C for more than 5 days associated with left hypochondrial pain non-responding to analgesics. The splenic abscess was resolved after instant drainage tube application and intensive IV antibiotic regime administration Figs. (6-8).

Two (7.7%) patients developed disease relapse after one month following PSE and initially showed mild increase in WBCs and PLTs values in the 1st and 2nd week post-PSE. At the 1st month follow-up, their labs showed progressive platelet decrement.

During this study, there was one (3.8%) patient died about 3 months after the PSE procedure and not directly related to the procedure.



Fig. (6): A 54 years old female patient two weeks post-PSE, the post-contrast abdominal CT axial cuts revealed splenic infarction with sub-capsular collection.

Note: That the splenic artery is seen arising directly from the abdominal aorta.

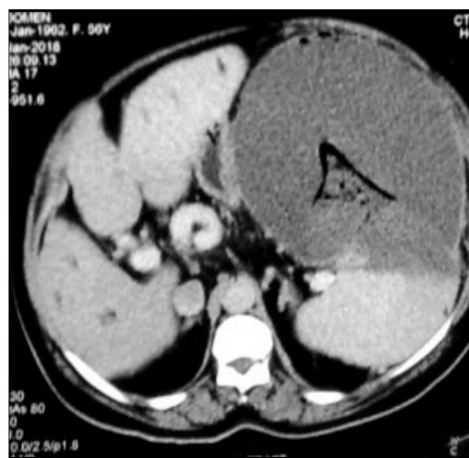


Fig. (7): In the follow-up period one month post-PSE, the axial cuts post-contrast CT scan of the abdomen revealed huge splenic collection showing multiple air loculi seen within; representing splenic abscess with gas forming organism.

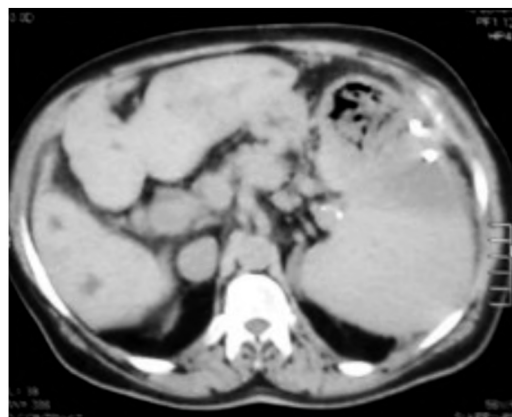


Fig. (8): The next follow-up after three months post-PSE, the axial non-contrast CT abdominal examination showed the previously applied drainage tube within the previously seen huge splenic collection that seen remarkably regressed in the current image.

Discussion

The primary aim of this study was to evaluate the role and efficacy of PSE in the treatment of hypersplenism by setting the extent of embolization between 30%-70% of initial spleen size with the demonstration of the PSE-related complications.

The platelet counts rose immediately in the next day in almost all patients and kept rising in the first week compared to their baseline values. They increased significantly in their values in the second week "Peak", followed by mild count decrement starting from the first month follow-up till the end of the follow-up periods through which alternative mild counts increment and decrement were noted compared to their last platelet counts with most of patients reached normal values. This decline in the platelet counts that started after one month following PSE might be attributed to regeneration of the residual splenic parenchyma.

These results are in keeping with:

Tajiri et al., 2002 [9] who reported that the platelet count rose 12-24 hours after PSE reaching the peak value in 1 or 2 weeks, the platelet count usually stabilized in about 2 months at approximately double the value before embolization and then slowly decreased over the next several years. Similar results were reported by Nio et al., 2003 [10] and Yoshida et al., 2008 [11].

These results also matched with results of Kimura et al., 2003 [12] who found that the peak platelet response occurred between 3 days and 1 month after PSE.

Zhu et al., 2009 [13] who revealed that PSE had significantly improved the platelet count in 2 weeks and 6 months.

Nassef et al., 2013 [14] who mentioned that the reason of the late decrease in platelets could be attributed to splenic regeneration after PSE.

Hadduck and McWilliams, 2014 [15] who reported that the platelet count rose 12-24 hours after PSE reaching the peak value in 1 or 2 weeks. These results were in accordance with Ahuja et al., 2015 [16].

Hussein et al., 2017 [17] who mentioned that PSE had significantly improved the platelet count in 2 weeks and 6 months. The mean platelet counts were lower after 6 months than after 2 weeks yet still above normal values.

Lee et al., 2017 [18] who reported that the platelet counts increased and peaked at approxi-

mately 3 days and 2 weeks respectively after embolization.

Wu et al., 2017 [19] who found that the platelet count dramatically increased soon at day 7 after PSE. A progressive decline in platelet count was observed from 1 to 3 months after PSE, and it was then constantly maintained thereafter up to 1 year. However; the platelet count could be maintained in a higher level than pre-PSE stage after 3 months of PSE without clinical symptoms. Also they reported that those patients who were followed for at least 12 months after PSE had a sustained platelet count $>85,000/\mu\text{L}$ at the twelfth month. Thus the platelet count could be maintained over an acceptable level of $80,000/\mu\text{L}$ at 1 year after PSE in most of the cases.

Dawoud et al., 2018 [20] who reported that there was a significant increase in the platelet count which reached its peak within the first month after the procedure and decreased gradually in its level within three months after the procedure.

Also regarding the leucocytic counts that rose significantly in the next day and started to subside in the first week or maximum by the end of the second week following PSE compared to their baseline counts. This elevation in the leucocytic counts might be attributed to normal reactive response of body defensive mechanism towards the infarcted splenic parenchyma. Afterwards the rest of the following leucocytic counts till the end of the patients' follow-up periods showed mild slow counts decrement with fluctuating values with most of the patients reached their normal values.

These results are in keeping with:

N'Kontchou et al., 2005 [21] who revealed that a transient elevation of WBC count was a normal physiological response following PSE, which could be found at day 1 post-PSE and might reach their peak values at day 3 post-PSE.

Sundaresan et al., 2005 [22] who mentioned that the mean WBCs counts were lower after 6 months than after 2 weeks yet still above normal values. This descend could be justified by the initial leucocytosis that occurred in some cases.

McCormick, 2007 [23] who explained that the increase in leukocytes post-PSE could be attributed to the activation of body defense mechanisms against infarcted splenic tissues.

Zhu et al., 2009 [13] who revealed that PSE had significantly improved the leucocytic count in 2 weeks and 6 months.

Hadduck and McWilliams, 2014 [15] who reported that the leucocytic counts increased significantly "Jumping" within two weeks following PSE.

Hussein et al., 2017 [17] who revealed that PSE had significantly improved the leucocytic count in 2 weeks and 6 months. The mean WBCs counts were lower after 6 months than after 2 weeks yet still above normal values. This descend could be justified by the initial leucocytosis that occurred in some cases.

Dawoud et al., 2018 [20] who reported that the WBC counts showed significant increase after one month following PSE. Follow-up after three months showed slight decrease in the level in comparison to the first month follow-up but still with significant increase in comparison to pre-PSE.

On the other hand these results are on contrary to Tajiri et al., 2002 [9] who reported that the white blood cell counts did not show significant changes after PSE during short-or long-term follow-up.

Apart from two patients whom developed relapse after one month following their PSE sessions, however, they also showed mild WBCs and PLTs counts improvement measuring up to double their baseline values.

The RBCs results showed no significant improvement noted in all patients.

These results matched with Zhu et al., 2009 [13] who reported that there was no significant change in the hemoglobin level or RBCs counts after PSE. These results were also in accordance with Abdella et al., 2010 [24]; Cai et al., 2016 [25] and Hussein et al., 2017 [17].

However, these results are in contrast with Tajiri et al., 2002 [9] and Yoshida et al., 2008 [11] studies that mentioned that the RBCs counts increased significantly by 6 months after PSE and remained elevated for up to many years.

All the patients showed variable degrees of improvement in their coagulation profile and most (78.9%) of patients showed minimal to mild improvement of their liver function tests at least one month after PSE in comparison to the pre-PSE baseline values. The forementioned improvements might be attributed to the decrease in the portal venous pressure and subsequently improving the hepatic function.

These results are consensus with:

Tajiri et al., 2002 [9] who found that serum albumin concentration increased after embolization.

Abdella et al., 2010 [24] who reported that there was improvement in prothrombin time occurred three months after PSE.

Lee et al., 2017 [18] who demonstrated that PSE appeared to be beneficial in cirrhotic patients by decreasing portal venous pressure and in turn improving hepatic function and reducing hepatic encephalopathy.

Dawoud et al., 2018 [20] who reported that there was deterioration in the liver functions in the form of elevation of prothrombin time and INR with decreased level of albumin and increased amount of ascites in the first month follow-up, then this condition showed improvement in the next follow-up visit.

On the contrary to:

Nassef et al., 2013 [14] who mentioned that the coagulation studies, serum bilirubin and serum albumin values were not altered significantly by PSE.

Hussein et al., 2017 [17] who reported that PSE had no significant change on liver function tests after 6 months.

Wu et al., 2017 [19] who mentioned that no significant change was observed in serum bilirubin level and prothrombin activity.

The incidence of overall complications was about 27% (7/26). From those complications, two of them were considered major complications according to the Society of Interventional Radiology, with the incidence was about 7.7% (2/26), including one case developed marked ascites and another case developed splenic abscess, they warranted additional medical care and prolonged hospitalization more than 48 hours.

Similar results were published by Hussein et al., 2017 [17] who reported that the incidence of the complications was 26.7% which was comparable to other studies as Zhu et al., 2009 [13] that involved 25% of their cases. Hussein et al., 2017 [17] also considered the incidence of the major complications was 13.3%.

All patients experienced one or more symptoms of post-embolization syndrome. Twenty five (96.2%) patients complained of mild to moderate left hypochondrial pain which was easily controlled

by light analgesics, one (3.8%) patient had severe pain that warranted medications other than analgesics. Almost all cases 25 (96.2%) patients had mild to moderate fever up to 38.5°C for 2 to 5 days and 1 patient showed no fever.

These results are matching with:

Nassef et al., 2013 [14] who revealed that all patients complained of moderate pain in the left hypochondrium which was easily controlled with light analgesics. Some patients had severe pain that warranted medications other than analgesics. They also revealed that moderate fever up to 38.5°C lasting 2 to 5 days was noticed in almost all patients.

Hussein et al., 2017 [17] who reported that the post-embolization syndrome had occurred in 27 (90%) patients and this was in keeping with most of the previous studies as 95% with Zhu et al., 2009 [13] and 100% with Amin et al., 2010 [4]. The post-embolization syndrome was considered as a side effect rather than being a complication being a sequale of the intended infarction.

Hussein et al., 2017 [17] also demonstrated that the mean duration of post embolization syndrome was 7.4 ± 4.16 days that was close to that of Zhu et al., 2009 [13]. Wu et al., 2017 [19] study also revealed that the PES developed within 1 week after PSE that would be resolved rapidly in almost all patients after conservative treatment. Dawoud et al., 2018 [20] found that the post-embolization syndrome developed after the procedure and within the first 24 hours.

On the other hand: Nassef et al., 2013 [14] reported that 15 (30%) of their patients complained of severe pain in the left hypochondrium which needed medications other than Paracetamol.

Wu et al., 2017 [19] revealed that 9 (69.2%) of 13 patients had experienced of PES in different degrees after PSE. Five (38.5%) patients were febrile and 8 (61.5%) patients complained of abdominal pain in whom medications were necessary to relief the symptom.

Dawoud et al., 2018 [20] showed that post-embolization syndrome occurred after PSE procedure and there was fever and pain, fever occurred in 4 (13.3%) patients and abdominal pain was in two (6.6%) patients.

In the current study, eight (30.7%) patients developed free abdominal ascites. Our results are less than those of Abdella et al., 2010 [24] who reported that 50% of their patients had either an increase in the amount or newly developed ascites.

On the other hand, these results are more than those of:

Nassef et al., 2013 [14] who revealed that two patients showed a notable increase in the amount of ascites and another one who newly developed ascites after the procedure, both (6%) are accepted as moderate complications and responded to conservative treatment by oral diuretics. The forementioned incidence of ascites was almost the same in N'Kontchou et al., 2005 [21] study that reported 2 out of 32 (6.25%) patients who had ascites after PSE.

Wu et al., 2017 [19] who reported that transient ascites occurred in two (15.4%) patients. In both cases, ascites was resolved after diuretic therapy.

Dawoud et al., 2018 [20] who observed four (13.3%) patients with increased amount of ascites.

Inspite the usage of selective embolization technique and sparring the upper splenic pole, one (3.8%) patient developed mild to moderate pleural effusions following PSE in the present study.

These results are less than those of Nassef et al., 2013 [14] who reported that mild pleural effusions were noted in ten (20%) of their patients and Dawoud et al., 2018 [20] who also noticed that pleural effusion appeared in two (6.6%) of their patients.

On the other hand, Wu et al., 2017 [19] study mentioned that there were no cases who developed pleural effusion in their study, and further suggested that pleural effusion might be preventable by the preservation of splenic upper pole.

In the present study, three (11.5%) patients developed sub-capsular splenic collection. These results are much less than those of Nassef et al., 2013 [14] who reported that ten (20%) of their patients developed sub-capsular collections.

Although strict aseptic condition was performed and about 55% estimated splenic infarction ratio was done, one (3.8%) patient developed splenic abscess about three weeks post-PSE presented by recurrent fever exceeding 38.5°C for more than 5 days associated with left hypochondrial pain non-responding to analgesics. It might be due to missing of one day antibiotic regime post-PSE during hospital stay. It was managed by intense IV antibiotic regime and U/S guided percutaneous drainage tube application using Pigtail catheter.

These results are in accordance with Dawoud et al., 2018 [20] who reported that one case showed

abscess formation after PSE. The patient showed clinical manifestations of fever and left hypochondrial severe pain three weeks after the procedure. The condition was treated by strong IV antibiotics and percutaneous U/S guided drainage using Pigtail catheter. They also demonstrated that the incidence of splenic abscess could be attributed to excessive embolic volume or damaged immune function of the spleen.

On the contrary, Wu et al., 2017 [19] revealed that all their patients underwent 80% PSE without any septic complications developed in the long-term follow-up period. They further reported that it might be contributed to the tight protocol of prophylactic antibiotic usage and the unique technique of splenic embolization using sterile technique and material for embolization.

During this study, there was one (3.8%) patient died about 3 months after the PSE procedure from hepatic encephalopathy and liver cell failure and not directly related to the procedure.

These results are less than those of Wu et al., 2017 [19] who reported that four (30.8%) of their patients died during the study period. None of the deceased cases were directly related to the procedure.

These results are partially in keeping with Abdella et al., 2010 [24] who mentioned that one of the cases died in their study as a complication from sepsis and hepatic encephalopathy.

On the other hand, other studies showed results where the mortalities are procedure-related, such as Hussein et al., 2017 [17] who reported that the mortality rate related to PSE was 3.3% in their study. Similarly Dawoud et al., 2018 [20] also reported that one (3.3%) case died within few days following PSE.

Limitations:

There are few limitations in this case series as follows: (1) This is not a randomized control trial, (2) The number of patients is relatively limited to twenty six patients only, (3) The wide range of planned splenic infarction ratio set between 30% to 70% and (4) The usage of two different embolic material types either PVA or Embosphere instead of using one embolic material type for all patients in this case series. All these factors limit the precise evaluation of PSE outcome and the proper risk assessment of PSE-related complications.

Conclusion:

PSE with careful pre-and post-procedure medications and care is an effective non-surgical minimally invasive procedure in avoiding the potential post-procedure complications and achieving remarkable hematologic response on controlling hypersplenism.

In spite PSE has a great potential in the treatment of hypersplenic state, yet, further conducted studies are highly recommended for each of the multiple factors each individually. These studies will help to understand the outcome and standardize a better protocol for best patient's selection (including the inclusion and exclusion criteria) in order to reach the best beneficial therapeutic outcomes with less complications at the same time.

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دور الإنصمام الجزئي لشريان الطحال في علاج فرط نشاط الطحال

أجريت هذه الدراسة في الفترة ما بين شهر مارس ٢٠١٦ وشهر أغسطس ٢٠١٨ وذلك لفحص مدى فعالية الإنصمام الجزئي لشريان الطحال في علاج حالات فرط نشاط الطحال وتأثيره الإيجابي على عدد كرات الدم بالأخص الصفائح الدموية وكرات الدم البيضاء. إشتراك في هذه الدراسة ٢٦ مريض (أعمارهم بين ١٤ و٧٤ سنة) يعانون من فرط نشاط الطحال الناتج عن التليف الكبدى بسبب الإلتهاب الكبدى الفيروسي. جميع المرضى لديهم نقص في عدد الصفائح الدموية منهم ١٧ مريضاً لديهم أيضاً نقص في عدد كرات الدم البيضاء. تم عمل جلسة واحدة لغلق شريان الطحال جزئياً لجميع المرضى يتراوح الغلق الجزئي بين ٣٠٪ و٧٠٪ من إجمالي حجم الطحال بإستخدام حجمين من المواد المجلطة سواء مادة PVA أو الحبيبات الجيلاتينية. ومن نتائج الدراسة تم إستنتاج أن الإنصمام الجزئي لشريان الطحال له أثر إيجابي في تحسين عدد الصفائح الدموية وكرات الدم البيضاء في حالات فرط نشاط الطحال.