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ORIGINAL ARTICLE

Partial splenic artery embolization in portal hypertension patients with hypersplenism: Two interval-spaced sessions' technique

Amr A. Nassef^{a,*}, Ayman A. Zakaria^a, Mohamed S. Abd ElBary^b

^a The Department of Radiology, Kasr El Ainy Hospital, Faculty of Medicine, Cairo University, Egypt
^b The Department of Tropical Medicine, Kasr El Ainy Hospital, Faculty of Medicine, Cairo University, Egypt

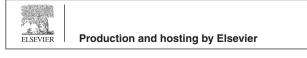
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KEYWORDS Abstract *Purpose:* To evaluate the ability of interval spaced sessions of transcatheter partial splenic artery embolization (PSE) to avoid the potential post procedure major complications, in portal Partial splenic artery embolization (PSE); hypertension patients with hypersplenism. Material and methods: The study included 50 patients (39 male and 11 females). All patients had Hypersplenism; Portal hypertension; liver cirrhosis and portal hypertension with hypersplenism and hyperactive bone marrow. All Thrombocytopenia patients underwent PSE in two sessions separated at least by 1 month interval. Immediate, short and intermediate term follow-up for 1 year were done. Results: We had no post procedure mortality. None of the patients developed septic shock, splenic abscess or needed emergency surgery. Ten of our patients developed subcapsular collections which were treated conservatively. All of our patients showed significant increase in the thrombocyte count after the first session which becomes remarkable after the second session and remained at appropriate levels during the follow up period. Conclusion: PSE using two (interval-spaced) sessions with careful pre- and post procedure medications and care; is really effective non surgical minimally invasive procedure in avoiding the potential post procedure complications while achieving remarkable hematologic response on controlling hypersplenism in cirrhotic patients with portal hypertension. © 2013 Production and hosting by Elsevier B.V. on behalf of Egyptian Society of Radiology and Nuclear Medicine. Open access under CC BY-NC-ND license.

* Corresponding author.

E-mail address: amrnassef@hotmail.com (A.A. Nassef).

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1. Introduction

Portal hypertension has four well-known complications: bleeding esophageal varices, ascites, encephalopathy, and hypersplenism. Hypersplenism is a dangerous complication which may lead to serious bleeding tendency due to thrombocytopenia and recurrent infections due to leucopenia (1). Splenectomy, the

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For the last 20 years, partial splenic embolization has been used to treat patients with hypersplenism effectively; markedly improving the platelet and leucocytic counts, relieving thrombocytopenia and resolving cytopenia (2–4).

However, partial splenic embolization is often associated with considerable high risk of complications which might be severe in many occasions. The real problem is that in severely decompensated patients with late liver disease and remarkable thrombocytopenia there is no chance to surgically interfere with those severe complications such as splenic abscess or total splenic necrosis which may lead to procedure related deaths (2,3,5,6).

Hematologic response correlates with the amount of the infarcted splenic tissue after partial splenic embolization. Almost all of the interventionists attempt to achieve infarction between 50% and 70% of the splenic mass to get good therapeutic hematologic response and alleviate hypersplenism (5,7,8).

The larger the amount of the infarcted splenic tissue, the earlier and greater will be the hematologic response; yet, much more severe the complications will be, as the severity of complications correlates with the amount of the infarcted splenic mass (5,7,8).

A lesser extent of infarction allows reduced sequestration and destruction of the blood elements and preservation of the ante grade flow within the splenic vein reducing the possibility of thrombosis and minimizes the possibility of complications; however it does not ensure the desirable therapeutic response and the long term efficacy (8).

2. Aim of the work

The aim of this study was to achieve the maximum permissible splenic tissue infarction with the least possible complications by performing partial splenic embolization in two separate consecutive sessions.

3. Patients and methods

3.1. Patients

In the period between September 2007 and May 2011, 50 patients (39 men and 11 women), their age ranging from 35 to 58 years (median age of 43 years) with hypersplenism secondary to liver cirrhosis and portal hypertension underwent partial splenic artery embolization (PSE) with the aim to eliminate hypersplenism. The exclusion criteria were patients with hypocellular bone marrow, patients with known collagen vascular or autoimmune disorders, and patients with ischemic heart disease, hypertension, renal failure, or malignant disease.

Patients were referred from the inpatient or outpatient clinic of tropical medicine department, Kasr Al Ainy hospital. The Child-Pugh classifications of these patients were Child A in 26 patients, Child B in 14 patients and Child C in 10 patients. All patients were selected to have thrombocytopenia,

platelet counts of 30–60,000/mm³ (mean 42800/mm³), with or without leucopenia; leucocytic count 1500–3800/mm³ (mean 2360/mm³). Bone marrow aspiration was done to all patients and proved hyper cellular.

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 to evaluate the state of the liver, spleen, ascites, or manifestations of liver cell failure. Laboratory investigations including complete blood picture (CBC), complete liver function, coagulation profile and serum creatinine were carried out.

3.2. Pre-procedure preparation

PSE, like splenectomy, can theoretically increase the risk of infection by encapsulated organisms including *Streptococcus pneumoniae*, haemophilus influenza type B, and *Neisseria meningitides*. Consideration should be given to vaccines such as 23-valent pneumococcal polysaccharide vaccine (PPV-23). It was given 2 weeks before PSE. All patients should be given proper information to reduce infectious complications.

The day before examination the patients were admitted to hospital, all of their laboratory data were revised and they started antibiotics as follows: cephalosporin 1 g IV every 12 h and metronidazole 500 mg every 8 h.

Six units of fresh frozen plasma were given in every patient in the night before examination.

A baseline platelet count was obtained in the morning of the examination.

3.3. Technique

All of the patients underwent two sessions of PSE separated by at least 1 month interval. The common femoral artery was punctured and catheterized with placement of 5F introducer sheath, and then the splenic artery was selectively catheterized by 5F Cobra catheter (Imager- Boston Scientific, USA). Diagnostic angiograms were obtained to define the arterial anatomical distribution of the intra-splenic branches and demonstrate the splenic parenchymal blush (splenogram). The Cobra catheter is advanced distally as much as we can near the splenic hilum. If this was not feasible by the cobra catheter owing to the marked tortuosity of the hypertrophied splenic artery a coaxial microcatheter (Renegade HI-FLO microcatheter, Boston Scientific, USA) was advanced through the mother cobra catheter to reach the splenic hilum or a desired intra-splenic branch. Embolization was then done by administration of 355-500 µm poly vinyl alcohol particles (Contour- PVA, Target therapeutics, Boston Scientific USA).

In the first session, administration of the particles was continued and control angiograms were obtained till splenograms demonstrate defective parenchymal patches (defective splenogram) between 30% and 40% (Figs. 1b, 3a and 5b) of the initial splenogram (Figs. 1a and 5a) denoting the rate of splenic infarction, thus avoiding major devitalization. At this point the examination was stopped the sheath was removed and manual vascular compression was applied for at least 30 min.

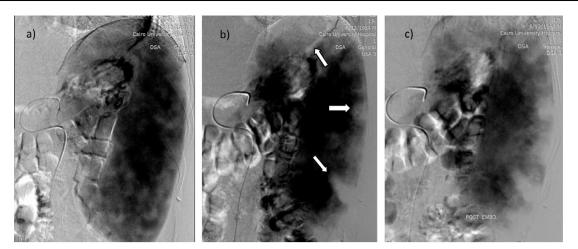


Fig. 1 Selective splenic angiography delayed phase showing splenic parenchymal blush (splenogram): (a) Before embolization showing homogeneous complete blush, (b) After completion of the 1st session showing defective splenogram with multiple parenchymal filling defects (arrows) counting for overall 30–40% splenic infarction, (c) After the 2nd session further splenic parenchymal blush defects are noted with evident decrease of its density giving an overall 70–80% splenic infarction.

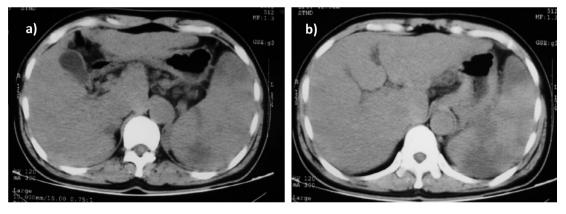


Fig. 2 Unenhanced CT scan for the upper abdomen showing splenic infarctions: (a) 1 week after the 1st session showing hypodense splenic focal defects representing infarctions, (b) 1 week after the 2nd session showing further extensive splenic infarctions with reduced size of the spleen notably its anterior pole from previous infarction.

3.4. Post-procedure care

The patients were hospitalized for the procedure day and the next 5 days where they continued on the same dose of IV antibiotics, also all of them were given analgesics and antipyretics like oral paracetamol every 6 h and fever chart monitor was followed up. Fifteen patients needed stronger analgesia. pethidine 1.5 mg/kg body weight was given IV every 6 h in the second and third days after the procedure beside the paracetamol.

The patients were discharged after their hospital stay on oral 3rd generation cephalosporin or quinolones 500 mg/12 h for 1 week.

In the second session the same steps were done but administration of PVA particles was continued till 70–80% defective splenograms were obtained (Figs. 1c, 3b and 5c). The same post procedure care applied.

3.5. Follow up

Post procedure follow up US was performed on the day of the examination and repeated 1 week later before discharge and then every 1 month in the first 3 months and every 3 months during the 1 year follow up.

Platelet count was done 1 week after the procedure before discharge of the patients and 1 week later.

All of our patients were followed every 1 month in the first 3 months and every 3 months during the 1 year follow-up period in outpatient clinic, a follow up platelet count was done and revised.

Unenhanced CT scan of the upper abdomen was done after 1 week from the procedure before discharge in all patients to evaluate the extent of devitalization and infarctions as well as to detect any complications (Figs. 2, 4and 6). In 10 patients with subcapsular collections follow up CT after 1, 2 and 3 months were done if needed to evaluate the progress of the condition.

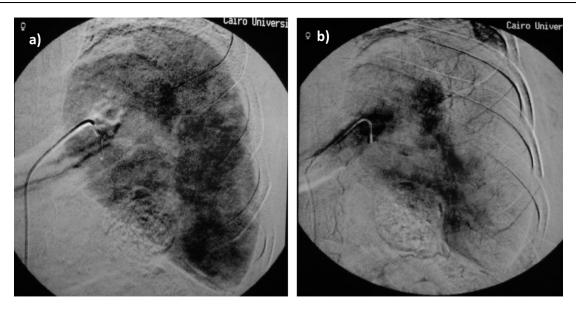


Fig. 3 Selective splenic angiography delayed phase showing splenic parenchymal blush (splenogram): (a) After completion of the 1st session showing defective splenogram with multiple parenchymal filling defects (arrows) counting for overall 30–40% splenic infarction. (b) After the 2nd session showing remarkable splenic infarction with evident decrease and loss of the splenic parenchymal blush as well as large defects giving an 80% splenic infarction.

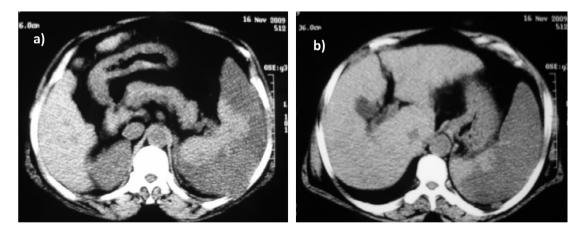


Fig. 4 (a) and (b) Unenhanced CT scan 1 week after the 2nd session showing extensive splenic infarction counting for about 80%.

4. Results

Moderate fever up to 38.5 °C, lasting 2 to 5 days was noticed almost in all patients. All of our patients complained of moderate pain in the left hypochondrial which was easily controlled with light analgesics. Thirty percent of our patients had severe pain that warranted medications other than analgesics in the second and third days after the procedure.

Mild pleural effusions with or without minimal basal atelectases were noted in 20% of our patients They were noted on the follow up CT scan and were not followed by any clinical signs of chest infection.

Ten of our patients developed subcapsular collections (Fig. 7) which were treated conservatively and needed no interventions (Fig. 8).

None of our patients developed or gave signs of splenic abscess formation.

We had no procedure related mortality in the immediate post procedure period or during the 1 year follow up period.

The platelet counts rose significantly after one week from the first session and it was still rising in the second week. By the end of one month 40% of the patients reached the normal values.

After 1 week from the second session all of our patients (100%) reached the normal platelet count values. The platelet count at 6 months and one year follow-up was within normal values in all of our patients with slight decline in three patients yet still within the normal values.

Leucocytic counts also rose significantly after 2 weeks of PSE compared to counts before PSE. At follow up, leucocytic counts were within the normal range and were significantly higher than before PSE for all patients despite the decline noted after 1 month and 1 year follow up counts.

Coagulation studies, serum bilirubin, and serum albumin values were not altered significantly by PSE. Serum alanine

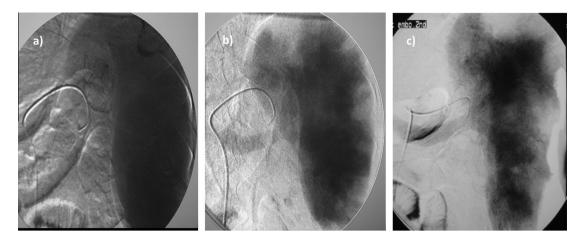


Fig. 5 Selective splenic angiography delayed phase showing splenic parenchymal blush (splenogram): (a) Before embolization showing homogeneous complete blush. (b) After completion of the 1st session showing defective splenogram with multiple parenchymal filling defects (arrows)) counting for overall 30–40% splenic infarction. (c) After the 2nd session further splenic parenchymal blush defects are noted with evident decrease of its density giving an overall 70–80% splenic infarction.

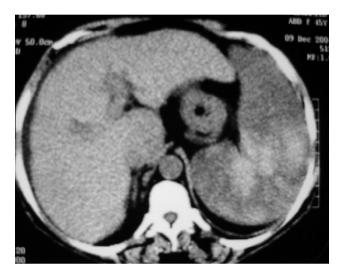


Fig. 6 Unenhanced CT scan 1 week after the 2nd session showing extensive splenic infarction counting for about 80%.

aminotransferase, and aspartate aminotransferase levels were elevated immediately after PSE, and returned to normal levels after 2 months (Table 1).

5. Discussion

Partial splenic embolization has been used in the last three decades to treat patients with hypersplenism effectively; markedly improving the platelet and leucocytic count and relieving thrombocytopenia (2,3,8).

Being minimally invasive, it is presumably precluding the risk of surgery and splenectomy complications; however, partial splenic embolization is often associated with considerable high risk of complications in most series which might be severe in many occasions (3,5,6). The real problem is that in severely

decompensated patients with late liver disease and remarkable thrombocytopenia there is no chance to surgically interfere with those severe complications such as splenic abscess or total splenic necrosis which may lead to procedure related mortality.

Hematologic response and the severity of complications correlate with the amount of the infarcted splenic tissue after partial splenic embolization (8). This was clearly demonstrated in Zhu K et al., study in which they were classifying their patients into three groups according to the splenic infarction rate after partial splenic artery embolization as: group (A) more than 70%, group (B) 50-70%, and group C less than 50% with no significant differences among the sex, age, splenic volume, Child-Pugh class, varices and peripheral blood count before PSE. After PSE they reported significant differences in the leucocytic and platelet counts among the three groups; where in group A and B the counts remained significantly higher from 2 weeks up to 5 years and even much higher in group A, while in group C the improvement only lasted for 6 months (5). Concurrently, they reported severe complications in half (50%) of patients in group A population, 8.8% in group B, while in group C they reported no (0%) severe complication. Based on this and on the general acceptance by most of the interventionists that infarction between 50% and 70% of the splenic mass is needed to get good therapeutic hematologic response and alleviate hypersplenism (7-9); we performed partial splenic embolization for all of our patients in this study in two separate consecutive sessions spaced by at least 1 month interval. We aimed to achieve between 30% and 40% splenic tissue infarction of the splenic mass after the first session to reach 70-80% after completion of the second session of the procedure. Our aim was to achieve such large amount of splenic tissue infarction to get a desirable good hematologic response with the least possible complications. By using this technique we succeeded to have no severe complications in our series; we had no short or intermediate related procedure related mortality, we had no severe complications, splenic abscess formation or septic shock. Twenty percent (20%) of our patients developed subcapsular collections

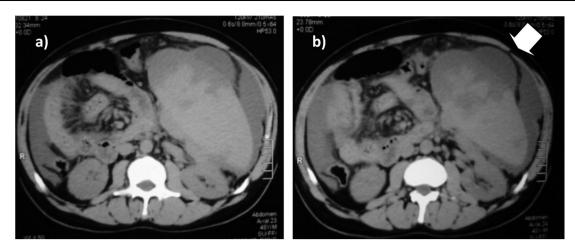


Fig. 7 Unenhanced CT scan for the upper abdomen 1 week after the 2nd session of PSE showing splenic infarctions with subcapsular collection along the anterolateral surface of the spleen. Also note mild ascites seen in the perisplenic space and right paracolic gutter which was newly developed in this patient.

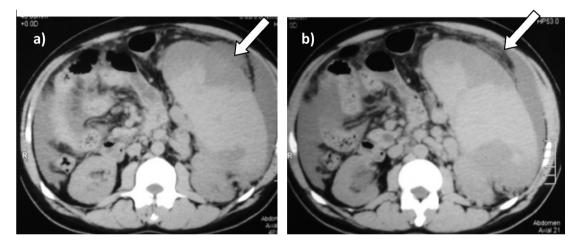


Fig. 8 Follow up unenhanced CT scan for the upper abdomen 1 month after the 1st session of PSE showing decreased size of the subcapsular collection. The ascites is still seen.

Table 1 Clinical picture of patients before and after PSE.					
	Before procedure	After one week from 2nd session	After one month from 2nd session	After one year from 2nd session	P value
Child-Pugh grading (A/B/C)	26/14/10	26/14/10	24/16/10	22/15/13	
AST (IU/L)	47.3 ± 31.17	52.3 ± 25.66	61.7 ± 16.09	65.7 ± 15.7	> 0.05
ALT (IU/L)	61.6 ± 51.8	72.3 ± 41.2	76.4 ± 17.5	64.5 ± 13.9	> 0.05
Serum albumin (g/dL)	3.4 ± 0.5	3.3 ± 0.4	3.3 ± 0.5	2.9 ± 0.3	> 0.05
Total bilirubin (mg/dL)	1.2 ± 1.0	1.1 ± 0.5	1.2 ± 0.5	1.3 ± 0.5	> 0.05
Prothrombin time (%)	67.0 ± 18.1	63.4 ± 13.1	62.2 ± 12.1	55.4 ± 12.2	> 0.05
WBC/µL	3.1 (+0.7)	10.5 (+1.6)	7.6 (+1.7)	6.3 (+1.1)	$= 0.002^{*}$
Platelets/µL	48.762 (+16.5)	293.470 (+33.3)	251.230 (+23.5)	235.330 (+22.7)	< 0001**

* Paired T test is used to measure significant difference between before procedure and one week follow up.

** Paired T test is used to measure significant difference between before procedure and 1 week and between 1 month and 1 year F.up.

(Fig. 7) which were treated conservatively and needed no interventions and subsided later during the follow up period (Fig. 8).

On targeting our goal of minimizing the potential post technique complication we planned a strict careful pre and post procedure medications and care including 1 week hospital stay and IV antibiotics in each step to avoid potential infection and to reduce the risk of the serious splenic abscess.

Nevertheless, we encountered moderate complications as we had two patients who showed a notable increase in the amount of ascites and another one who newly developed ascites after the procedure (Fig. 7), both (6%) are accepted as moderate complications and responded to conservative treatment by oral diuretics. The afore mentioned incidence of ascites is almost the same in N'Kontchou et al. who reported (6.25%) 2 out of 32patients who had ascites after PSE in their study (3); but much less than Abdella et al. who reported 50% of their patients to have either increase in the amount or newly developed ascites (6).

Also we had mild complications in similar rates like most of other studies; almost all of our patients developed moderate fever up to 38.5 °C and left hypochondrial pain which usually lasted for 2 to 5 days and easily controlled with antipyretics and light analgesics. Thirty percent of the patients complained of severe pain in the left hypochondrial which needed medications other than paracetamol.

Mild pleural effusions with or without basal atelectases were noted in 20% of the patients, they were detected in the basal chest cuts obtained during the follow up abdominal CT scans. None of them was complicated by chest infection.

On evaluating the hematologic response, the platelet count rose significantly after one week from the first session and it was still rising in the second week. By the end of one month 40% of the patients reached the normal values while the reminder 60% dose not reach the normal values. After 1 week from the second session all of our patients (100%) reached the normal values and it was still rising two weeks after the treatment. The platelet count at 6 months and one year follow-up was within normal values in all of our patients with slight decline in three patients yet still within the normal values.

6. Conclusion

Partial splenic artery embolization PSE using two intervalspaced sessions with careful pre- and post procedure medications and care; is a really effective non surgical minimally invasive procedure in avoiding the potential post procedure complications while achieving remarkable hematologic response on controlling hypersplenism in cirrhotic patients with portal hypertension.

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