CASE REPORT

Severe thrombocytopenia due to hypersplenism treated with partial splenic embolisation

Mohammad Hanafiah,^{1,2} Azura Mohamed Mukhari Shahizon,² Soo Fin Low,² Man Harun Shahrina³

¹Department of Radiology, MARA University of Technology, Sungai Buloh, Selangor, Malaysia
²Department of Radiology, Universiti Kebangsaan Malaysia Medical Centre, Cheras, Kuala Lumpur, Malaysia
³Department of Radiology, Pantai Hospital, Bangsar, Kuala Lumpur, Malaysia

Correspondence to
 Dr Mohammad Hanafiah,
 mhanafiah8804@gmail.com

SUMMARY

A 35-year-old woman with background of liver cirrhosis and portal hypertension secondary to chronic hepatitis C presented with complication of hypersplenism and thrombocytopenia. She developed severe menorrhagia requiring multiple blood transfusions. In addition, her interferon therapy was withheld owing to the underlying thrombocytopenia. Partial splenic embolisation was performed, which improved her platelet counts. Subsequently, the menorrhagia was resolved and her interferon therapy was restarted.

BACKGROUND

Thrombocytopenia in chronic liver disease secondary to hepatitis virus is most commonly attributed to hypersplenism secondary to cirrhosis and portal hypertension.¹ It also can be due to autoantibodies against platelets or myelosuppressive action of the hepatitis virus. Partial splenic embolisation (PSE) is an effective method to treat thrombocytopenia in patients with cirrhosis.² In view of its known potential complications, PSE is normally reserved for specific clinical setting after calculating the risks and benefits. This case is an example of PSE as a feasible, but often an overlooked, method to treat severe thrombocytopenia secondary to hypersplenism.

CASE PRESENTATION

A 35-year-old woman with a history of chronic hepatitis C genotype 1 and child's B liver cirrhosis presented with recurrent episodes of menorrhagia for 2 months. The patient's menorrhagia was severe and debilitating. There was no symptom of lethargy. She was on interferon therapy for her chronic hepatitis.

On examination she was pale. There was no evidence of koilonychia or angular cheilitis and no external stigmata of chronic liver disease. The patient's abdomen was soft with splenomegaly.

She was haemodynamically stable (HR 85 bpm, blood pressure 135/70).

INVESTIGATIONS

The patient's blood showed evidence of pancytopenia. The platelet, haemoglobin and white cell levels were $35.0 \times 10^9/L$, 6.0 g/dL and $3.1 \times 10^9/L$, respectively. International normalised ratio was raised measuring 1.4. The renal and liver profiles were within normal limits. The peripheral blood smear revealed microcytic and hypochromic red blood cells, suggestive of underlying chronic blood

loss. The platelet count progressively decreased to the lowest level of $9.0 \times 10^9/L$. In view of the pancytopenia, bone marrow aspirate trephine was performed showing normocellularity consistent with peripheral destruction.

The ultrasound study revealed coarse liver echotexture in keeping with cirrhosis associated with sign of portal hypertension (splenomegaly with splenic hilar varices).

Pipelle investigation of the uterus was negative for malignancy and consistent with dysfunctional bleeding. There was no sign of any bleeding elsewhere. Both colonoscopy and oesophagogastroduodenoscopy (OGDS) were performed. The colonoscopy result was normal while the OGDS showed only small erosion at the stomach antrum with very mild gastropathy in the fundus, unlikely to be the culprit of her anaemia.

It was concluded that the pancytopenia was caused by hypersplenism secondary to cirrhosis and portal hypertension. The anaemia was further worsened by severe menorrhagia.

TREATMENT

She was started on provera 10 mg twice daily. However, despite the medication, her menorrhagia continued to become a problem as the platelet level was very low. She also developed anaemia from chronic blood loss requiring multiple blood transfusions to keep the haemoglobin at a safe level. In addition, her interferon therapy was stopped as it was contraindicated in severe thrombocytopenia.

In view of limited treatment option coupled with debilitating recurrent menorrhagia secondary to thrombocytopenia, she was referred for PSE. The platelet level pre-procedure was $11 \times 10^9/L$. She was explained about the procedure and the risk involved with it, that is, pain, bleeding, postembolisation syndrome, venous thrombosis and splenic abscess. Written consent was taken. Four units of platelets and 1.5 g of cefuroxime were given prior to the procedure. Three bags of packed blood cells were transfused a week before, a day before and during the procedure.

PSE was finally performed under a strict aseptic technique. The catheterisation of the splenic artery was achieved through right femoral artery approach. Lower pole and mid pole branches of the splenic artery were selectively cannulated and injected with polyvinyl alcohol 335–500 μm via a 5Fr Yashiro catheter. Postembolisation angiogram revealed an approximate total of 50% of spleen was embolised (figure 1). No immediate

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Figure 1 Digital subtraction angiogram of the splenic artery. (A) Image shows selective lower pole splenic arteriogram. The thick white arrow indicates the lower pole branch of the splenic artery. The curved white arrow shows a splenunculus receiving its arterial supply from the lower pole branch. (B) Image shows selective middle and upper pole splenic arteriogram. The thin white arrows indicate upper pole branches of the splenic artery. The white arrow head indicates the middle segment branch of the splenic artery. (C) Capillary phase of the spleen pre-embolisation. (D) Capillary phase of spleen postembolisation showing 50% residual parenchyma.

complication was encountered during the procedure and the patient's vital signs remained stable throughout.

OUTCOME AND FOLLOW-UP

The platelet count tripled within a day and continued to rise above $110 \times 10^9/L$. The patient's white cell count also raised from less than $3.0 \times 10^9/L$ to an average of $13.0 \times 10^9/L$ and her haemoglobin stabilised at level of 11.0–13.0 g/dL.

Contrast-enhanced CT scan was performed at 2 months post-procedure and showed expected area of necrosis of the spleen (figure 2).

The increase in platelet count was sustained for a year and her interferon therapy was restarted. She no longer had menorrhagia while still on provera 5 mg twice daily. No observable complication related to PSE within the period of 2-year-follow-up.

DISCUSSION

Thrombocytopenia in chronic liver disease is a fairly frequent phenomenon. The prevalence ranges between 15 and 70%, being greater among patients with advanced cirrhosis.¹ Among other aetiological mechanisms, thrombocytopenia is most commonly attributed to portal hypertension with hypersplenism.¹ Hypersplenism represents the increased pooling and destruction of the corpuscular elements of the blood by the enlarged spleen.

Owing to current lack of effective pharmacological treatment options able to increase platelet count, splenectomy and transcatheter arterial PSE continue to be applied in specific settings. PSE is more favourable than splenectomy as it is generally a gentler approach and is associated with lower mortality rate.³ Preservation of some functional splenic parenchyma is also thought to provide protection from overwhelming bacteria sepsis.³ Splenic embolisation for hypersplenism was first performed in the 1970s and the subsequent studies have demonstrated its consistent efficacy in treating thrombocytopenia of

Figure 2 Selected axial contrast-enhanced CT taken 2-month post partial splenic embolisation demonstrating infarcted splenic parenchyma as homogenous non-enhancing hypodense area (arrows).

patient with chronic liver disease.² The present study is an example where severe thrombocytopenia secondary to hypersplenism needs to be treated. The thrombocytopenia has caused severe menorrhagia and was a contraindication to the patient's treatment of interferon therapy.

Apart from to treat hypersplenism secondary to hepatic disease, other indications of PSE include chronic idiopathic thrombocytopenic purpura, hereditary spherocytosis and splenic trauma in haemodynamically unstable patient.⁴ The use of splenic artery embolisation for treatment of splenic artery aneurysms and spontaneous rupture of splenic haemangioma have also been documented.²

PSE is not a procedure without any risk. The potential complications include post-embolisation syndrome, splenic abscess, splenic rupture, refractory ascites, distal pancreatitis and gastrointestinal bleeding.^{5–6} Pulmonary disorders, such as pneumonia, atelectasis and pleural effusions, are the other potential complications, which usually develop in the left lung and are associated with embolisation of the upper pole of the spleen.⁴ Post-embolisation syndrome is the by far the most frequent side effect and is regarded as minor,^{6–7} with fever, malaise, discomfort and leucocytosis and generally lasts not more than 3–5 days. Some patients may need major analgesic administration and long hospital stay. More severe complications like portal/splenic vein thrombosis or splenic abscess resulting in death may occur.⁷ Hence, careful selection of patient is crucial to prevent these serious complications.

When embolisation is planned, visualisation of the pancreatic arteries is important to avoid unintended embolisation as the splenic artery has many branches that supply the pancreatic body and tail. Contraindications of PSE include terminal end stage of the underlying disease as well as presence of on going

infection. Severe infection is associated with formation of splenic abscess postprocedure.² Our patient did not develop any significant complication during and after the PSE procedure.

The increase in platelet count observed following PSE is associated with reduction of platelet pool in the diminished volume of infarcted spleen. In addition, increased availability of thrombopoietin owing to decreased intrasplenic degradation also contributes to more platelet production.⁸ The extent of embolisation is important to determine the success of the procedure. Embolisation of less than 50% is thought to yield high chances of relapse of hypersplenism whereas infarction of more than 70% is associated with a high rate of complication, favouring bacterial contamination.⁹

Learning points

- ▶ Partial splenic embolisation (PSE) is a useful treatment in patients with thrombocytopenia secondary to hypersplenism.
- ▶ PSE is more favourable than splenectomy as it is generally a gentler approach and is associated with a lower mortality rate.
- ▶ Careful selection of the patient with thorough calculation of the risks and benefits are crucial to prevent complication.

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REFERENCES

- 1 Peck-Radosavljevic M. Hypersplenism. *Eur J Gastroenterol Hepatol* 2001;13:317–23.
- 2 Yoshida H, Mamada Y, Taniai N, et al. Partial splenic embolization. *Hepatol Res* 2008;38:225–33.
- 3 Pandey R, Garg R, Darlong V, et al. Role of splenic artery partial embolization in a patient with portal hypertension and pancytopenia undergoing hysterectomy under anesthesia. *AANA J* 2012;80:96–8.
- 4 Toprak SK. Partial splenic embolization versus splenectomy for the management of autoimmune haemolytic anemia: a response. *Turk J Hematol* 2011;28:250–1.
- 5 He XH, Gu JJ, Li WT, et al. Comparison of total splenic artery embolization and partial splenic embolization for hypersplenism. *World J Gastroenterol* 2012;18:3138–44.
- 6 Herrador Benito J, Zunzarren MG, Pozancos de Simón T, et al. Urgent splenectomy after partial splenic embolization in liver-transplanted patient: a case report. *Case Rep Transplant* 2012;2012:959635.
- 7 Sakai T, Shiraki K, Inoue H, et al. Complications of partial splenic embolization in cirrhotic patients. *Dig Dis Sci* 2002;47:388–91.
- 8 Giannini EG, Savarino V. Thrombocytopenia in liver disease. *Curr Opin Hematol* 2008;15:473–80.
- 9 Zhu K, Meng X, Qian J, et al. Partial splenic embolization for hypersplenism in cirrhosis: a long-term outcome in 62 patients. *Dig Liver Dis* 2009;41:411–16.

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