Journal of Liver
ISSN: 2167-0889

Nojiri et al., J Liver 2013, 2:3
DOI: 10.4172/2167-0889.1000128

Case Report Open Access

A Case of Rapidly Developed Splenomegaly after Percutaneous Transhepatic Obliteration (PTO)

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Abstract

A 70-year-old woman underwent Balloon-Occluded Retrograde Transvenous Obliteration (BRTO) for bleeding gastric varices, which was unsuccessful. Then, she underwent Percutaneous Transhepatic Obliteration (PTO) to block the main blood supply route. On the 5th postoperative day, the white blood cell and platelet counts suddenly fell markedly, and CT showed that the splenomegaly had worsened rapidly after PTO. Since blood flow to the gastric varices was still present, Partial Splenic Embolization (PSE) was performed in an attempt to reduce portal venous blood flow. However, rebleeding from the gastric varices occurred, and so embolization with Histoacryl was performed. Although portal hypertension after PTO is a relatively common event, we report this case because no case has been reported in which massive splenomegaly with leukocytopenia developed within a short period of time.

Keywords: PTO; Splenomegaly; Leukopenia

Abbreviations: PTO: Percutaneous Transhepatic Obliteration; PSE: Partial Splenic Embolization; BRTO: Balloon-Occluded Retrograde Transvenous Obliteration

Introduction

Esophageal and gastric varices associated with portal hypertension are sometimes accompanied by massive bleeding, and are important factors influencing the prognosis. Direct surgery, Percutaneous Transhepatic Obliteration (PTO), and Balloon-Occluded Retrograde Transvenous Obliteration (BRTO), including medical Endoscopic Injection Sclerotherapy (EIS) and Endoscopic Variceal Ligation (EVL), are becoming the mainstay of treatment for these varices. However, the frequent recurrence of varices and their development at multiple sites have posed a problem. Such varices are usually associated with splenomegaly due to chronic portal hypertension, but generally speaking splenomegaly worsens progressively with time, rarely developing rapidly. Herein, we report a patient who rapidly developed splenomegaly after the treatment of bleeding from gastric varices (PTO), and later developed rebleeding.

Case Report

Clinical course

A 70-year-old woman, who had been receiving outpatient treatment for hypertension and non-B, non-C cirrhosis at a hospital, was admitted there because of hematemesis. Since bleeding from esophageal varices was suspected, EVL was performed, and the patient was followed-up. However, the next day, hematemesis recurred with bleeding noted from the gastric varices. After temporary hemostasis was achieved by clipping, she was referred to our hospital.

On admission, her consciousness was clear. Her blood pressure was 123/77 mmHg, and temperature 36.5°C. No superficial lymph nodes were palpable, and her bulbar conjunctivae were not icteric, but slightly anemic. Auscultation revealed no abnormal heart or lung sounds. The abdomen was flat and soft. The liver was palpable one finger-breadth below the right costal margin. No leg edema was present. Neurological examination showed no asterixis.

Results

Laboratory tests on admission revealed anemia (hemoglobin 9.2 g/dl), a reduction in coagulation activity (prothrombin time 37.9%), and liver dysfunction (aspartate amino transferase AST 659 U/l, alanine aminotransferase 555 U/l, lactate dehydrogenase 1789 U/l), which raised a suspicion of shock liver due to bleeding. The cause of the cirrhosis was not known (Table 1).

Endoscopy at the previous hospital was showed (Figure 1). The location, form, and color of the varices were Lm, F1, Cb, and Lg-f (Figure 1a). The esophageal varices had been treated by EVL (Figure 1b). The next day bleeding recurred from the gastric varices (Figure 1c) and was treated by clipping (Figure 1d).

Abdominal CT revealed gastric varices located mainly at the gastric fornix (Figure 2a). Since the varices were contiguous with the left renal vein through a gastrorenal shunt (Figures 2b and 2c) we performed BRTO.

The gastric varices themselves could not be visualized during BRTO, and multiple collateral venous channels were identified (Figure 3a); therefore, this procedure was discontinued, and PTO was performed (Figure 3b). Angiography of the splenic-portal venous system led to a suspicion of blood flow from both the posterior and left gastric veins to the gastric varices. Angiography of the posterior gastric vein after left gastric vein embolization showed visualization of gastric varices draining through the left inferior phrenic vein and a gastrorenal shunt (Figure 3c); therefore, the varices were embolized. After embolization, blood flow from the left and posterior gastric veins decreased, and no visualization of the varices was noted. Although the short gastric vein appeared to join the gastric varices, imaging after a change in body position excluded its involvement in the varices; therefore, the short gastric vein was not treated (Figure 3d). Her post-PTO course was

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Received August 26, 2013; **Accepted** September 25, 2013; **Published** October 04, 2013

Citation: Nojiri S, Fujiwara K, Shinkai N, Endo M, Joh T (2013) A Case of Rapidly Developed Splenomegaly after Percutaneous Transhepatic Obliteration (PTO). J Liver 2: 128. doi:10.4172/2167-0889.1000128

Hematology

WBC	6600 /mm^3
RBC	2.88×10^6 /mm^3
Hb	9.2 g/dl
Ht	27.2 %
Plt.	6.8×10^4 /mm^3

Coagulation test

Fib	167 mg/dl
PT	37.9 %
HPT	41.7 %

Biochemistry

BUN	68 mg/dl
Cre	1.1 mg/dl
TP	3.6 g/dl
Alb	2.3 g/dl
T.Bil	1.1 mg/dl
D.Bil	0.3 mg/dl
ALP	227 U/l
γ-GTP	45 U/l
AST	659 U/l
ALT	555 U/l
LDH	1789 U/l
T.Chol	163 mg/dl
NH_3	140 μg/dl
Glucose	148 mg/dl

IgG	1192 mg/dl
IgA	250 mg/dl
IgM	106 mg/dl

HBs antigen	negative
HBc antibody	negative
HCV antibody	negative

ANA	negative
AMA	negative

Abbreviations: WBC: White Blood Cells; RBC: Red Blood Cells; Hb: Hemoglobin; Ht: Hematocrit; plt: Plate: PT: Prothrombin Time; HPT: Hepaplastin Time; BUN: Blood Urea Nitrogen; Cre: Creatinine; γGTP: γ-Glutam; LDH: Lactate Dehydrogenase; T.Chol: Total Cholesterol; NH_3: Ammonia; T-Bil: Total Bilirubin; AST: Asparta transferase; ALT: Alanine Aminotransferase; ALP: Alkaline Phosphatase; TP: Total Protein; Alb: Albumin; A: Antibody; AMA: Anti-Mitochondria Antibody

Table 1: Laboratory test on admission.

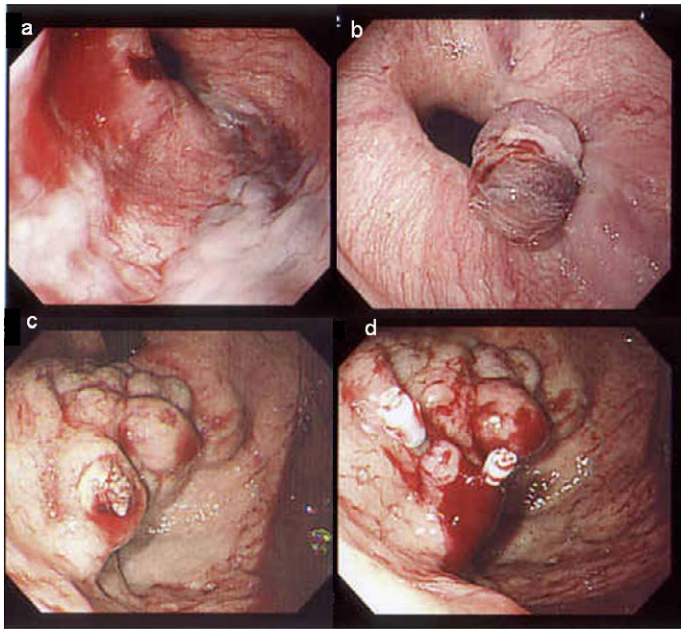

Figure 1: a: Endoscopy revealed esophageal varices (Lm, F1, Cb). b: Esophageal varices were treated by EVL. c: Bleeding from the gastric varices. d: Temporary hematostasis was achieved by clipping.

uneventful until the 5th postoperative day, when the white blood cell count fell rapidly to 1,000/microliter (Figure 7).

Abdominal CT scans showed that the splenomegaly had worsened rapidly after PTO, and also revealed hepatomegaly and edema in the transverse and ascending colon (Figure 4). Although the gastric varices were partially thrombosed, there was residual blood flow. Therefore, Partial Splenic Embolization (PSE) was performed in an attempt to reduce portal blood pressure (Figure 5a: before, 5b: after). PSE embolized the superior polar artery, part of the inferior polar artery, and short gastric artery. Splenic arteriography confirmed that PSE had produced a good embolic effect mainly on the superior splenic pole. However, on the venous phase of splenic arteriography, the short gastric vein was more dilated than before PTO, and blood flow to the gastric varices was also suspected (Figure 5c). Post-PSE CT showed that the extent of splenic embolization was high, at about 60%, but blood flow to the gastric varices persisted (Figure 5d). Thereafter, in consideration of the need for additional treatment for the gastric varices, the patient was followed-up until the 6th day after PSE, when she developed hematemesis (Figure 6); therefore, EIS with Histoacryl was performed. The sclerosing agent exerted a strong embolic effect on the portal system from the site apparently joining the gastrorenal shunt to the site of coil embolization of the posterior gastric vein. Figure 7 show the course of treatment. The first hospital day was defined as the first day of her visit to our hospital. BRTO was performed on the first hospital day, and PTO was performed on the 6th hospital day, after which the white blood cell count fell. PSE was performed on the 13th hospital day, followed 6 days later by EIS. Her subsequent course was uneventful, and Gastrointestinal Fiberscopy (GIF) 7 days later showed

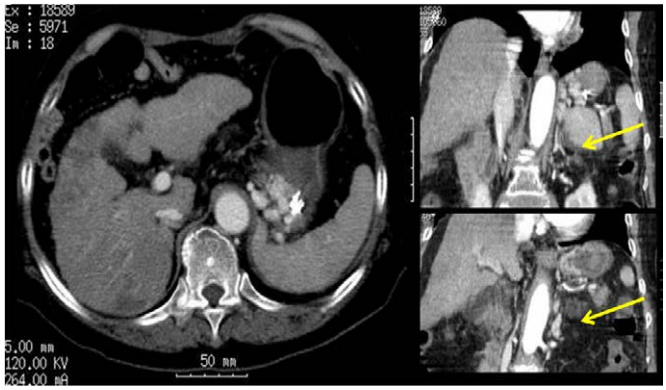

Figure 2: a: Enhanced CT revealed gastric varices. b, c: Gastric varices were contiguous with the left renal vein through a gastric renal shunt.

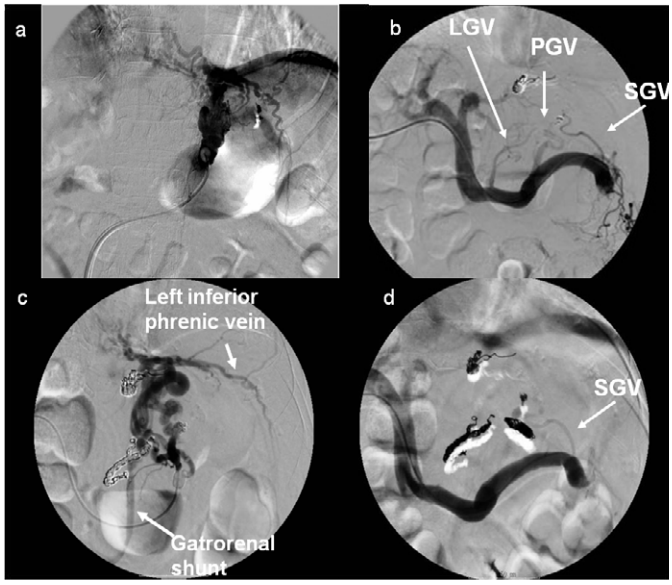

Figure 3: a: During BRTO multiple collateral veins were identified and gastric varices were not visualized clearly. b: PTO was performed. Left Gastric Vein (LGV), Post Gastric Vein (PGV), and Short Gastric Vein (SGV) were identified. c: After LGV embolization gastric varices were identified through PGV. d: After LGV and PGV embolization there are no gastric varices shown by portography.

that the size of the gastric varices had markedly decreased, indicating an excellent effect of EIS (Figure 8a). CT also showed the complete accumulation of the sclerosing agent in the varices, with complete blood flow block (Figure 8b).

Discussion

The hemodynamics of intraperitoneal varices associated with portal hypertension due to cirrhosis is complex [1]. The treatment of varices is often followed by the development of portal hypertension [2]. In general, among the post-PTO complications, rebleeding is the biggest problem [3-5]. Reportedly, the development of collateral circulation most commonly involves the short gastric vein. Studies have also reported the worsening of varices even after BRTO and the development of rectal varices immediately after EIS [6]. However, our search of the literature revealed no case in which splenomegaly worsened within a short period of time after treatment for varices. Proposed causes of splenomegaly associated with cirrhosis include splenic congestion due to portal hypertension [7] and an increase in splenic blood storage due to increased splenic arterial blood flow [8]. The presumed mechanisms of the arterial blood flow increase involve the action of vasodilators and increase in blood flow from the celiac artery to other organs due to increased hepatic arterial resistance [9-11].

In this study, portal hypertension developed after BRTO and PTO alone, which was unlikely to change arterial blood flow, suggesting the development of portal hypertension due to decreased blood flow drained from the portal vein. Some degree of portal hypertension is seen after BRTO and PTO [12]. According to a study by Ishikawa et al., portal angiography, performed again after PTO, showed that collateral blood vessels other than embolized veins became dilated and tortuous, and new vascular channels formed within 2 days in all their patients.

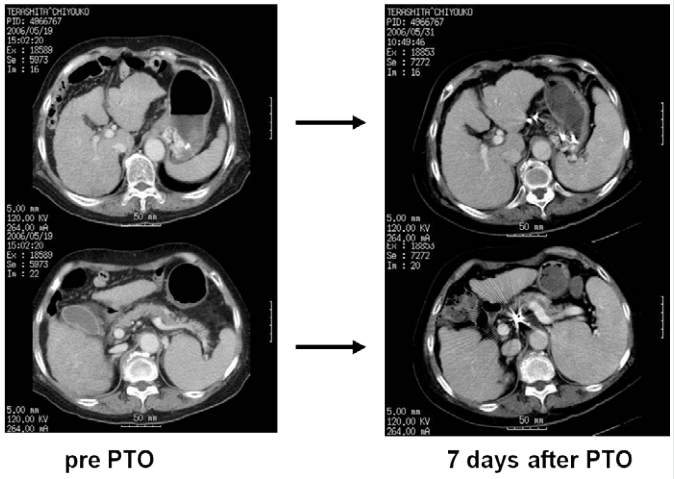

Figure 4: After PTO the splenomegly had worsened within few days and residual blood flow was shown in the gastric varices.

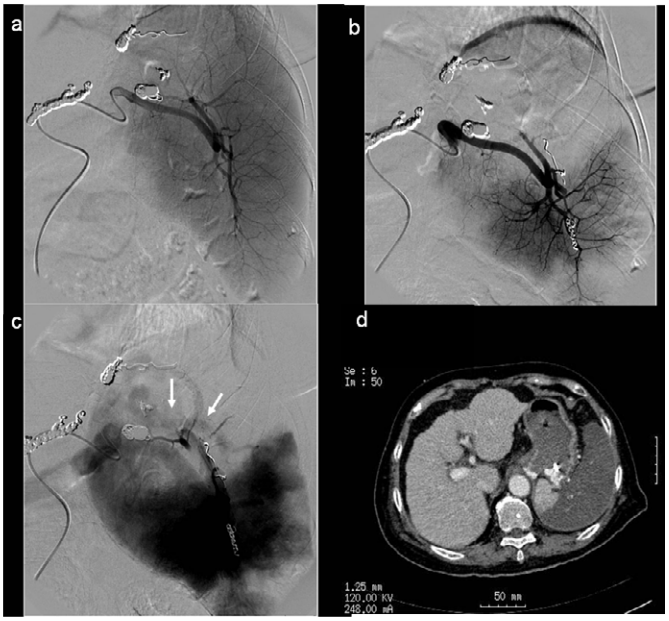

Figure 5: Splenic arteriography before PSE (a) and post PSE (b). However even after embolization the short gastric vein was more dilated than before PTO and blood flow to the gastric varices was also suspected (c, d).

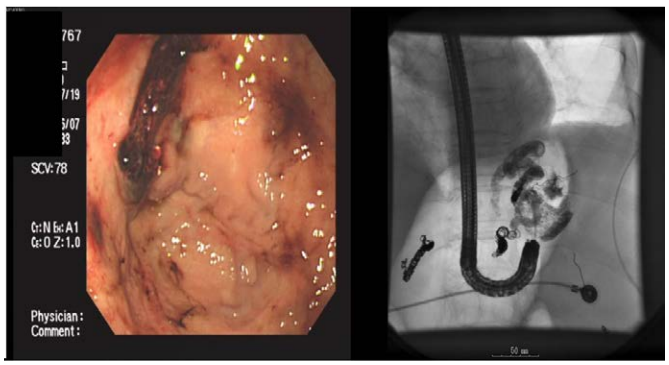

Figure 6: Bleeding from the gastric varices and EIS with Histoacryl was performed.

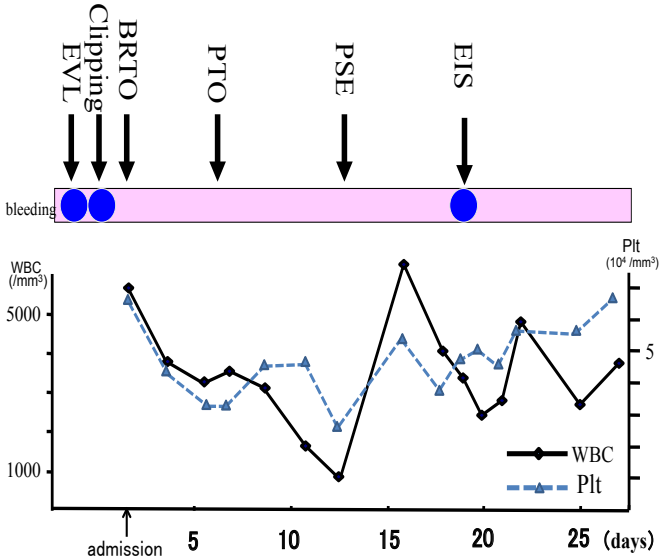

Figure 7: The course of treatment was shown.

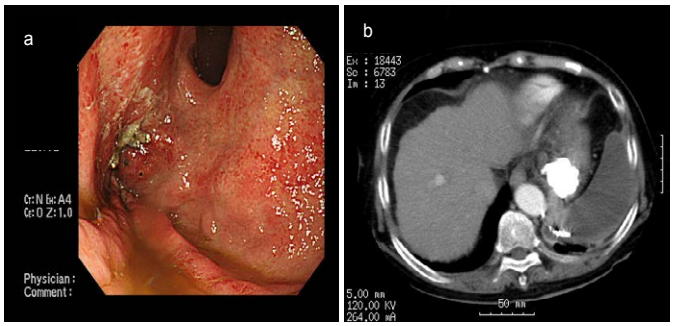

Figure 8: a: Endoscopy was performed 7 days later from EIS showed that the varices had markedly decreased. b: CT showed that blood flow were blocked completely.

The portal pressure rapidly rises, and then falls as more collateral blood vessels develop [13]. The recanalization of embolized veins depends on the procedure, embolization material used, and patient's condition. Lunderquist et al. reported that recanalization occurred in 13 of 16 patients who underwent PTO using Gelfoam. However, in fact, rebleeding from recanalized veins occurs less often, and it should be considered that rebleeding through collateral vessels may occur despite complete embolization [12,14]. In this patient, the gastric varices were mainly supplied by the posterior gastric vein, but they may have also been supplied by the left and short gastric veins. These varices drained into the left inferior phrenic vein and renal vein through the gastrorenal shunt. When BRTO was performed, multiple venous drainage routes were obliterated for the retention of embolization material in the target varices. In addition, PTO with coils resulted in complete embolization, and on re-angiography, the short gastric vein was found to be dilated, but no other significant collateral vessels were identified. These findings suggest that the obliteration of multiple venous drainage routes during BRTO resulted in a rapid rise in the portal pressure, which, coupled with the complete blockage of the blood supply routes during PTO and poor development of collateral vessels, led to rapid splenic enlargement. It is also possible that the failure to treat the short gastric vein during PTO resulted in both splenomegaly and short gastric vein dilatation, leading to rebleeding from the gastric varices.

Bleeding is a typical complication of PTO [15,16]. It is rare, although theoretically possible, to encounter such a rapidly progressive splenomegaly, as seen in this patient, in daily clinical practice because of the development of collateral vessels. Adequate care is needed when searching for blood supply routes during PTO. To prevent rebleeding from gastric varices, it is necessary not to leave blood supply routes untreated. If rapid leukopenia and thrombopenia occur, as in this case, rapidly progressive portal hypertension with hypersplenism should be suspected, and re-examination and re-treatment should be performed immediately.

Conclusion

We encountered a case of gastric varices with splenomegaly developing rapidly after PTO. The hemodynamics of isolated gastric varices appears to be complex, and the portal pressure rises rapidly after BRTO and PTO in some patients, requiring careful follow-up.

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