

# Disastrous Portal Vein Embolization Turned into a Successful Intervention

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**Abstract** Portal vein embolization (PVE) may be performed before hemihepatectomy to increase the volume of future liver remnant (FLR) and to reduce the risk of postoperative liver insufficiency. We report the case of a 71-year-old patient with hilar cholangiocarcinoma undergoing PVE with access from the right portal vein using a mixture of *n*-butyl-2-cyanoacrylate and ethiodized oil. During the procedure, nontarget embolization of the left portal vein occurred. An aspiration maneuver of the polymerized plug failed; however, the embolus obstructing portal venous flow in the FLR was successfully relocated into the right portal vein while carefully bypassing the plug with a balloon catheter, inflating the balloon, and pulling the plug into the main right portal vein.

**Keywords** Portal vein · Embolization · Cyanoacrylate · Ethiodized oil · Adverse effect · Hepatectomy

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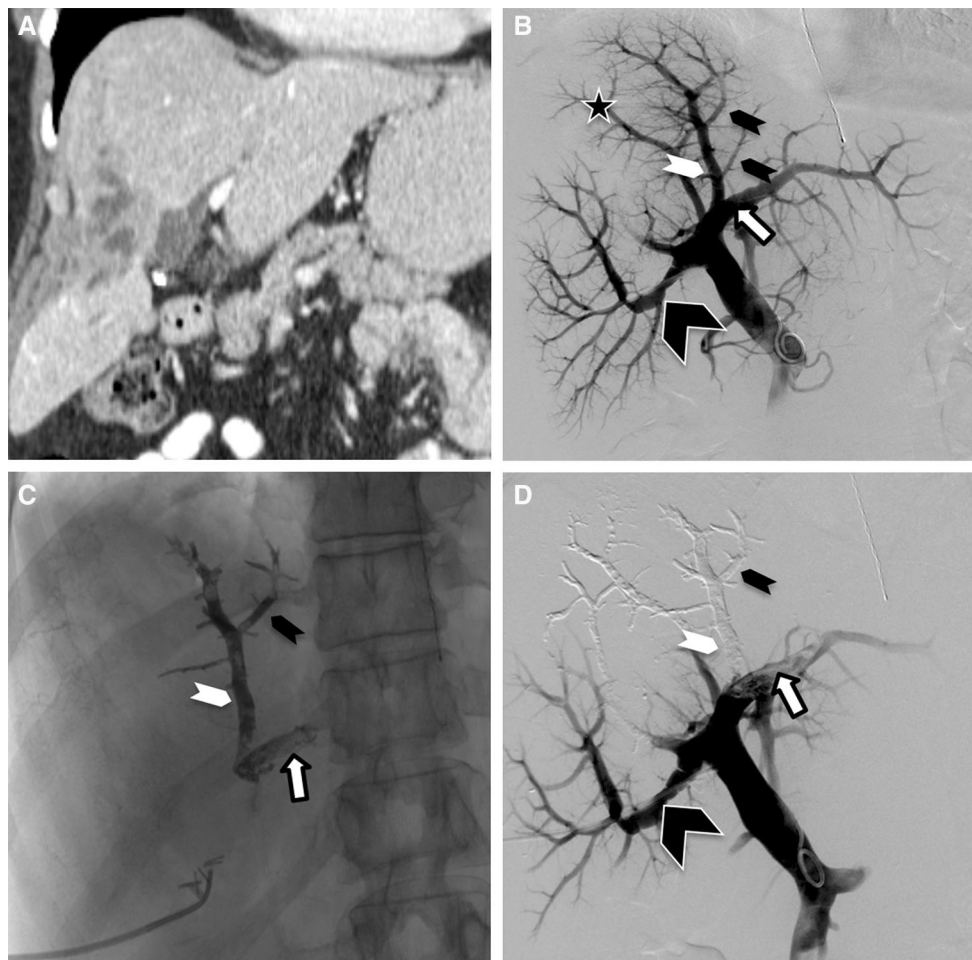
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## Introduction

Primary and secondary liver malignancies are common pathologies with high mortality rates. Major hepatectomy is a potentially curative treatment option but carries a significant risk of mortality that ranges from 0.5 to 4 % overall [1], and increases up to 4–12 % in patients with chronic liver disease [2, 3]. Liver insufficiency due to an insufficient liver remnant volume is the main cause of postoperative mortality. When patients are not eligible for liver resection due to small future liver remnant (FLR), portal vein embolization (PVE) may be applied, allowing a volume increase of the FLR and thus reducing the risk for liver failure [4]. In 1920, Rous and Larimore showed that occlusion of portal branches to a part of the liver leads to a progressive and ultimately complete atrophy of the parenchyma in the region deprived of portal blood and to hypertrophy of the rest of the hepatic tissue, which receives such blood in excess [5]. Since then, varying techniques relying on the same underlying principle of hypo-/hypertrophy after portal vein branch occlusion have been described, including portal vein ligation, transileocolic, and percutaneous transhepatic portal vein embolization.

## Case Report

We report a case of a 71-year-old female undergoing clinical and imaging workup for elevated liver enzymes and high bilirubin levels. On computed tomography (CT), a large tumor in liver segments V and VIII leading to segmental cholestasis was noted (Fig. 1A). No visible tumor in the left liver lobe, no infiltration of the main portal vein, and no extrahepatic metastasis at time of diagnosis was observed. Diagnostic laparoscopy and liver biopsy



**Fig. 1** **A** CT image (*coronal plane*) showing an inhomogenous liver lesion in segment V/VIII with contrast uptake in the venous phase causing segmental cholestasis. No tumor visible in left liver lobe. **B** Direct portogram in anteroposterior (AP) projection (*cranial angulation 30°*), showing normal hepatopetal flow and no evidence of portal hypertension. As a variant of portal vein branching pattern, the right anterior portal vein arises from the left portal vein (*white arrow*). Portal vein branches of segment IV (*small black arrowhead*) arising from segment VIII branches (*small white arrowhead*). Segment V

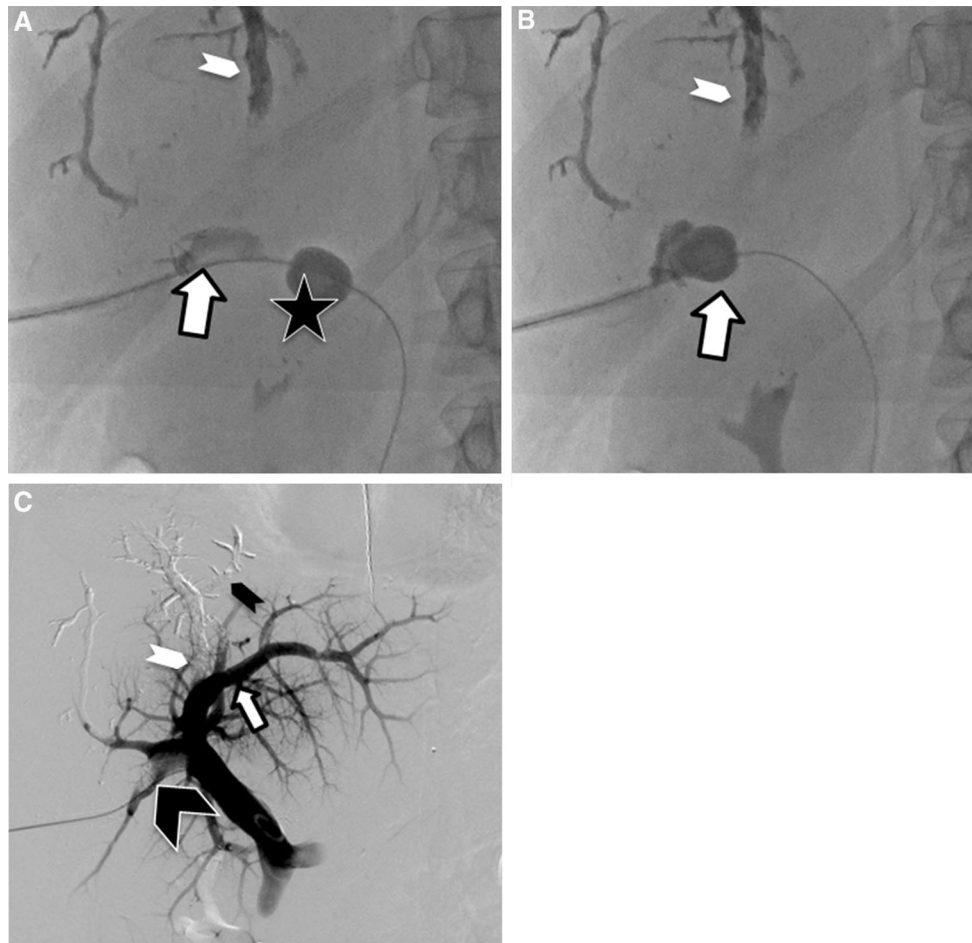
branches (*asterisk*) and the right posterior branch (*large black arrowhead*). **C** Radiopaque embolization material in portal vein branches of segment VIII and IV (*small white and black arrowhead*). Dislodged glue embolus in the proximal part of the left portal vein (*white arrow*). **D** Control portogram showing complete obstruction of flow in the anterior branches of the right portal vein (*small white and black arrowhead*) and patent flow in the right posterior branch (*large black arrowhead*). Subtotal obstruction of the left portal vein with a floating glue embolus in the central part (*white arrow*)

confirmed a hilar cholangiocarcinoma involving the confluence and extending into the right hepatic duct (Bismuth classification type IIIa). Due to a small FLR, the patient was referred to a right portal vein embolization including segment IV prior to extended right hepatectomy. Blood tests obtained preinterventionally were normal except a slightly elevated  $\gamma$ -glutamyltransferase (GGT) and alkaline phosphatase (AP).

The procedure was performed using general anesthesia due to patient preference. A distal branch of the right portal vein in segment VI was punctured with a 21-gauge (G) Chiba needle (Ecojekt Hospital Service, Rome, Italy) via an right lateral intercostal approach under fluoroscopic-guidance (DSA, Axiom, Siemens Healthcare, Erlangen, Germany). A

curved hydrophilic guide wire (length 180 cm, diameter: 0.035 in.; Terumo Medical, Tokyo, Japan) was inserted, and a 5-French (Fr) sheath (Terumo) was placed to secure the pathway to the biliary tree. A 4-Fr pigtail catheter (Cook, Bjaeverskov, Denmark) was advanced over the wire into the main stem of the portal vein. Subsequent injection of contrast media (Iopamiro 300, Bracco, Switzerland) showed normal portogram with hepatopetal flow and no evidence of portal hypertension (Fig. 1B).

For embolization, a 4F-Cobra catheter (Cook) was engaged in the anterior branch of the right anterior portal vein and a 2.7-Fr microcatheter (Progreat 130 cm, Terumo) was further advanced into the branches to segment VIII and IV. The microcatheter was flushed with a 5 %-glucose



**Fig. 2** **A** Dislodged glue material partially obstructing the left portal vein (white arrow) was crossed with a curved tip hydrophilic guidewire. A balloon catheter was advanced over the wire (asterisk) and inflated distally to the embolus. Embolized portal vein branches in segment IV and VIII (white arrowhead). **B** Relocation of dislodged

glue material into the anterior branch of the right portal vein by slightly pulling the inflated balloon. **C** Final portogram showing occlusion of right posterior portal vein (large black arrowhead), segment VIII (small white arrowhead) and segment IV branches (small black arrowhead). Patent left portal vein (white arrow)

solution. Then, a mixture of 2-*N*-butyl-cyanoacrylate (NBCA, Histoacryl<sup>®</sup>, B. Braun Dexon, Spangenberg, Germany) and iodized oil (Lipiodol<sup>®</sup> ultrafluid, Laboratoire Guerbet, Roissy, France) at a 1:6 ratio was injected slowly into the vessel lumen. During embolization of segment IV branches, dislodging and migration of glue into the left portal vein occurred (Fig. 1C and D). Introduction of a 7-Fr sheath (Terumo) for aspiration with a 7-Fr aspiration catheter and syringe (VacLok<sup>®</sup> 60 ml, Merit Medical, UT) failed. Control portogram showed the floating glue embolus in the left main portal vein almost fully obstructing the flow into the distant left portal vein and successfully embolized portal branches of segment VIII and IV.

Using a curved tip hydrophilic guidewire (standard guidewire, 180 cm, 0.035 in; Terumo) the glue embolus in the left portal vein was carefully crossed and a balloon catheter (LeMaitre<sup>®</sup>, 80 cm, 5 Fr, 12 mm, Sulzbach, Germany) was subsequently advanced over the guidewire. The

balloon was then inflated distally to the glue embolus within the left main portal vein and pulled back slowly into the right portal vein, hereby relocating the previously dislodged embolus. There was subsequent, uneventful embolization of the remaining posterior branches of the right portal vein. Final portogram (Fig. 2C) showed occlusion of right portal vein, including segment IV branches and patent left portal vein. The percutaneous access to the right portal vein was plugged with a small amount of Histoacryl on removal of the vascular sheath. Six weeks after PVE, the patient underwent successful right extended hemihepatectomy and bilioenteric anastomosis.

## Discussion

As published in the meta-analysis by van Lienden et al. of 1,790 patients undergoing PVE, minor complications are

very frequent, including fever (37 %), transient elevation of transaminase (35 %), abdominal pain (23 %), and nausea (2 %) [6]. However, major complications leading to nonresectability are very rare, occurring in only 0.4 % of procedures and include severe cholangitis, large abscesses, sepsis, and portal venous, or mesentericoportal venous thrombosis [6, 7].

Embolization of nontarget vessel occurred in 0.6 % of procedures [6] and may preclude the patient from hemihepatectomy if the main portal vein or major branches of the FLR are involved. With 32.5 % the most common embolic agent used for PVE is NBCA, a glue-like substance that immediately polymerizes when it comes in contact with ionic fluids, such as blood or with vascular endothelium. To allow safe application through a microcatheter, NBCA is usually diluted with Lipiodol, an oily radiopaque substance that delays the polymerization process. A NBCA to Lipiodol ratio of 1:1, 1:2, 1:3, and 1:4 leads to a polymerization time of 3.2, 4.7, 7.5, and 11.8 s, respectively [8].

We assume the dislodged glue embolus partially obstructing flow in the left portal vein in our patient (Fig. 1C and D) was not adherent to the vessel wall but rather floating within the lumen and linked to glue material previously applied in the right anterior portal vein. When injecting further glue, reversed flow may have facilitated dislodging glue back into the left portal vein. The higher NBCA to Lipiodol dilution ratio might have had a favorable effect. The reason for diluting NBCA in a rather high ratio was to achieve good embolization of small caliber peripheral branches of the portal vein at the beginning of the procedure. As published by Takasawa et al. with progressive dilution of NBCA, the embolic material is recognized in smaller diameter arteries, and embolization of a larger vascular bed may be accomplished [9]. In general, we adjust the NBCA to Lipiodol ratio from the peripheral (1:6) to more central veins (1:4 or 1:2).

We hypothesize that it would not have been possible to relocate a glue embolus fully obstructing the vessel lumen due to a firm attachment to the vessel wall or if the embolus had migrated into more peripheral branches of the left portal vein.

In conclusion, migration of embolizing substance during PVE to branches of the FLR that need to be preserved is a

rare complication but has potentially far-reaching consequences for the individual patient, leading to nonresectability. NBCA is the most frequent embolic agent used for PVE, which requires great skill and experience from the interventionalist. Relocation of dislodged glue embolus partially obstructing the portal venous lumen may be possible with a balloon catheter.

**Conflict of Interest** Tomas Dobrocky, Joachim Kettenbach, Ruben Lopez-Benitez, and Levent Kara declare that there is no conflict of interest. This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

**Statement of Informed Consent** Informed consent was obtained from all individual participants included in the study.

**Statement of Human and Animal Rights** For this type of study formal consent is not required.

## References

1. Nordlinger B, Peschard F, Malafosse R (2003) Resection of liver metastases from colorectal cancer—how can we improve results? *Colorectal Dis* 5:515–517
2. Fong Y, Fortner J, Sun RL et al (1999) Clinical score for predicting recurrence after hepatic resection for metastatic colorectal cancer: analysis of 1001 consecutive cases. *Ann Surg* 230:309–318 discussion 318–321
3. Farges O, Belghiti J, Kianmanesh R et al (2003) Portal vein embolization before right hepatectomy: prospective clinical trial. *Ann Surg* 237:208–217
4. Denys A, Bize P, Demartines N et al (2010) Quality improvement for portal vein embolization. *Cardiovasc Intervent Radiol* 33:452–456
5. Rous P, Larimore LD (1920) Relation of the portal blood to liver maintenance: a demonstration of liver atrophy conditional on compensation. *J Exp Med* 31:609–632
6. Van Lienden KP, van den Esschert JW, de Graaf W et al (2013) Portal vein embolization before liver resection: a systematic review. *Cardiovasc Intervent Radiol* 36:25–34
7. Abulkhir A, Limongelli P, Healey AJ et al (2008) Preoperative portal vein embolization for major liver resection. 247:49–57
8. Stoesslein F, Ditscherlein G, Romaniuk PA (1982) Experimental studies on new liquid embolization mixtures (histoacryl-lipiodol, histoacryl-panthopaque). *Cardiovasc Intervent Radiol* 5:264–267
9. Takasawa C, Seiji K, Matsunaga K et al (2012) Properties of N-butyl cyanoacrylate-iodized oil mixtures for arterial embolization: in vitro and in vivo experiments. *J Vasc Interv Radiol* 23:1215–1221.e1