

Recanalization after Successful Occlusion by Transcatheter Arterial Embolization with N-Butyl Cyanoacrylate for Traumatic Splenic Artery Injury

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ABSTRACT

A 70-year-old male with advanced pancreatic cancer went into shock after sustaining a traumatic abdominal injury. Computed tomography (CT) showed a hematoma with extravasation around the pancreas and hemorrhagic ascites. After direct catheterization failed due to angiospasm, the ruptured splenic artery was successfully occluded by transcatheter arterial embolization (TAE) using an N-butyl cyanoacrylate (NBCA)-lipiodol mixture and the patient recovered from shock without complications. A follow-up CT obtained 20 days later showed a recurrent splenic artery pseudoaneurysm without extravasation. A repeat angiogram demonstrated recanalization of the splenic artery and pseudoaneurysm via antegrade. We embolized the recanalized pseudoaneurysm using metallic coils for isolation. Our experience indicates that adequate concentration and volume of the NBCA-lipiodol mixture should be considered depending on the vascular spasm in a patient with hypovolemic shock.

Key words: *N-butyl cyanoacrylate, Recanalization, Splenic artery injury*

In interventional procedures, isolation of the bleeding point by proximal and distal embolization is necessary for splenic artery injury because proximal embolization alone often fails to stop bleeding due to the development of collateral arteries⁵⁾. Although metallic coils are usually used for isolation, their introduction is difficult when the site distal to the bleeding point cannot be accessed by the microcatheter¹⁾. In such cases, the injection of N-butyl cyanoacrylate (NBCA) from the proximal side is a good alternative.

NBCA is a widely-used permanent embolic agent that immediately polymerizes in the blood and induces complete vessel occlusion¹⁾. The reported success rates of TAE with NBCA for splanchnic arterial bleeding range from 75 to 100%^{1,5,7)}. Although post-embolization recanalization has been documented in patients with intracranial arteriovenous malformations, the reported incidence of recanalization after embolization with NBCA of splanchnic arteries is very

low²⁾. We report a case of recanalization after embolization with NBCA for a traumatic splenic artery injury.

CASE REPORT

A 70-year-old male with advanced pancreas cancer (Fig. 1) sustained an injury to his upper abdomen by being pushed against a wall by farm equipment. He had received systemic chemotherapy as an outpatient for over a month. On arrival at our hospital his blood pressure was 103/62 mmHg and hemoglobin (Hb) was 8.7 g/dl. Contrast-enhanced computed tomography (CT) showed a hematoma with extravasation around the pancreas. Immediately after CT examination, his systolic blood pressure and his hemoglobin level fell to 70 mmHg and 6.2 g/dl despite a blood transfusion. He was immediately transferred to the angiography room.

A celiac angiogram using a 4-F catheter (Selecon

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Fig. 1. Contrast-enhanced CT obtained before the injury showed advanced cancer of the pancreatic body (arrowheads) and encasement at the proximal portion of the splenic artery (arrow).

catheter, Terumo Clinical Supply, Kakamigahara, Japan) showed a pseudoaneurysm with massive extravasation at the proximal portion of the splenic artery, which was diffusely narrowed due to hypovolemic shock (Fig. 2A); the common hepatic artery was not visualized. A superior mesenteric arteriogram demonstrated the hepatic artery via a gastroduodenal-pancreatic arcade. As vessel spasm due to hypovolemic shock prevented the advancement of a 2.0-F microcatheter (Masters, Asahi Intec Co. Ltd., Nagoya, Japan) beyond the bleeding point, we attempted to embolize the splenic artery by injecting an NBCA (Histoacryl; Aesculap, Tuttlingen, Germany)-lipiodol (André Guerbet, Aulnay-sous-Bois, France) mixture from the proximal side. We placed the microcatheter at the origin of the splenic artery and slowly injected a total of 0.3 ml of the 1:2 ratio NBCA-lipiodol mixture after flushing it with a 50% glucose solution. Although the mixture accumulated in the celiac trunk, the proximal portion of the splenic artery and the pseudoaneurysm, sufficient accumulation was not observed at the distal portion of the pseudoaneurysm (Fig. 2B). However, we finalized the procedure because no extravasation on celiac and superior mesenteric angiograms was found (Fig. 2B).

The patient subsequently recovered from hypovolemic shock with no further complications. A plain CT scan obtained 7 days after the procedure showed a cast of the NBCA-lipiodol mixture at the celiac and proximal splenic artery. A contrast enhanced CT confirmed the occlusion of the pseudoaneurysm and the patency of the distal splenic artery. The CT scan also showed hematoma and pancreatic fluid leakage around the pancreas. We treated our patient by placing a percutaneous drainage for them. He was trans-

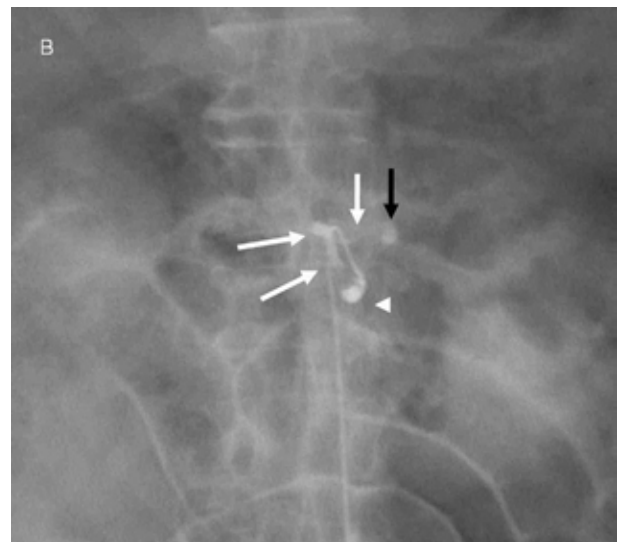


Fig. 2

- A** Celiac angiogram (right-anterior oblique view) obtained before embolization showed a pseudoaneurysm (black arrowheads) with extravasation (white arrowheads) of contrast medium at the proximal portion of the splenic artery. The common hepatic artery cannot be seen. The small white-, large white-, and black arrows indicate the celiac trunk, the main trunk of the splenic artery, and the left gastric artery, respectively.
- B** Post-embolization celiac angiogram showed obliteration of the celiac trunk and disappearance of the pseudoaneurysm and extravasation. Accumulations of the NBCA-lipiodol mixture were seen at the celiac trunk just proximal to the splenic artery (white arrows) and within the pseudoaneurysm (black arrow). They are not visualized in the distal portion of the pseudoaneurysm. The white arrowhead indicates contrast material injected through the 4-F catheter.

ferred out of the intensive care unit 11 days later.

A follow-up CT scan obtained 20 days post-embolization showed a recurrence of a 10 mm sized pseudoaneurysm at the splenic artery. The status of the NBCA-lipiodol mixture at the celiac trunk and proximal splenic artery was as seen on the CT images (Fig. 3), leading us to suspect that reverse flow in the splenic artery through a collateral pathway had resulted in the revascularization of the pseudoaneurysm. We performed another arteriogram which showed recanalization of the celiac and splenic arteries and a recurrent pseudoaneurysm without extravasation (Fig. 4). The diameter of the splenic artery was larger than that of the initial embolization (Fig. 2A) and the hepatopetal blood flow was observed via the celiac trunk. On a superior mesenteric angiogram the pseudoaneurysm was not visualized through the collateral vessels. These findings suggested that blood flow into the pseudoaneurysm was completely ante-grade via the celiac trunk. Using a microcatheter, we succeeded in isolating the pseudoaneurysm by placing several 0.016-inch and 0.018-inch microcoils (Interlocking Detachable Coil; Boston Scientific/Target Therapeutics, Fremont, CA; Tornado Coil; Cook, Bloomington, IN, and C-stopper; Piolax, Yokohama, Japan) at the proximal and distal sites of the pseudoaneurysm. The patient was discharged home 8 days after the second embolization.

A follow-up CT scan obtained 1 month after the second embolization showed no recanalization. The patient suffered no rebleeding until the time of his death 3 months later due to advanced pancreatic cancer.

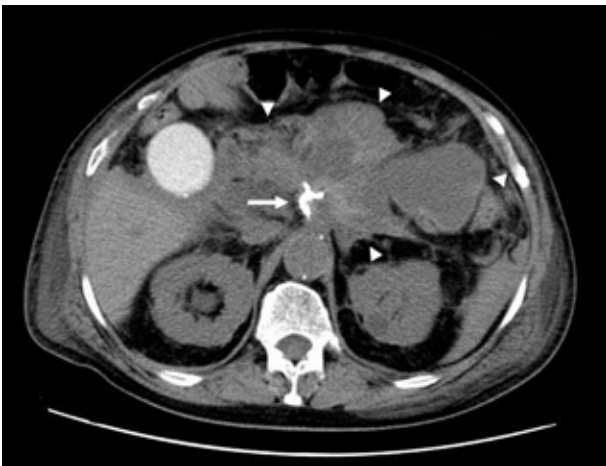


Fig. 3. Plain CT obtained 7 days after the initial embolization showed the persistence of an accumulation of the NBCA-lipiodol mixture at the celiac and proximal splenic artery (arrow). Arrowheads indicate hematoma around the pancreas.



Fig. 4. Celiac angiogram (right-anterior oblique view) obtained 20 days after the initial embolization showed recanalization from the celiac trunk to the splenic artery and a recurrent pseudoaneurysm (arrow) of the splenic artery. The caliber of the main trunk of the splenic artery was enlarged compared to the initial celiac angiogram.

DISCUSSION

NBCA is a permanent liquid embolic agent. The mechanisms of vascular occlusion by NBCA are by a physical blockage of the cast and by an induction of thrombus formation. The cast is produced by the polymerization in the blood. The polymerization time depends on the NBCA concentration. The lower concentration is used by mixing with more lipiodol, the longer time is required⁶. NBCA induces vessel thrombosis. Thrombus formation is induced by an inflammatory reaction in the vascular structures³. The severity of thrombosis does not depend on the NBCA concentration.

In our patient 2 factors contributed to the recanalization of the splenic artery; one was a vessel spasm due to hypovolemic shock, and the other was the smaller occlusion range due to the inadequate concentration of the NBCA-lipiodol mixture. Hypovolemic shock tends to decrease the vessel caliber⁴. We performed splenic artery embolization while the patient was in hypovolemic shock. He recovered immediately and the diameter of the splenic artery increased. Consequently, the size of the NBCA-lipiodol mixture cast and the diameter of the splenic artery were dissociated, resulting in recanalization of the splenic artery. On the first embolization, we used a 1:2 ratio NBCA-lipiodol mixture, but this high concentration led to a too rapid polymerization resulting in a

shorter range of vascular obstruction with a smaller volume of the mixture. Consequently, the effect of NBCA embolization was weak. Based on our experience, we suggest that to prevent recanalization after embolization for splenic artery injury, the vessel from the proximal to the peripheral site of the pseudoaneurysm should be completely filled with the NBCA-lipiodol mixture, thereby eliciting a higher severity of thrombosis and more complete physical occlusion. We should use a lower concentration of NBCA in hypovolemic shock patients undergoing embolization. This might facilitate the delivery of the NBCA-lipiodol mixture downstream under conditions of slower blood flow and vessel spasms. In our case, a ratio of 1:4 or 1:5 may have been appropriate for permanent occlusion, judging by our more recent experiences using NBCA.

We reported a case of traumatic rupture of the splenic artery that was recanalized after embolization with NBCA in a patient with hypovolemic shock. To prevent recanalization, interventional radiologists have to determine carefully the appropriate concentration and volume of the NBCA-lipiodol mixture based on the speed of the blood flow and the caliber and range of the parent artery to be embolized.

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