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CASE REPORT

Recanalization of Splenic Artery Aneurysm After Transcatheter Arterial Embolization Using *N*-Butyl Cyanoacrylate

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Abstract A 65-year-old woman who had been diagnosed as having microscopic polyangiitis developed sudden abdominal pain and entered a state of shock. Abdominal CT showed massive hemoperitoneum, and emergent angiography revealed a ruptured splenic artery aneurysm. After direct catheterization attempts failed due to tortuous vessels and angiospasm, transcatheter arterial embolization using an *n*-butyl cyanoacrylate (NBCA)-lipiodol mixture was successfully performed. Fifty days later, the patient developed sudden abdominal pain again. Repeated angiography demonstrated recanalization of the splenic artery and splenic artery aneurysm. This time, the recanalized aneurysm was embolized using metallic coils with the isolation method. Physicians should keep in mind that recanalization can occur after transcatheter arterial embolization using *N*-butyl cyanoacrylate, which has been used as a permanent embolic agent.

Keywords *N*-Butyl cyanoacrylate · Embolization · Recanalization · Splenic artery · Aneurysm

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Introduction

Splenic artery aneurysms (SAAs) account for 60% of all visceral artery aneurysms [1], which are often seen as complications of gestation, pancreatitis, or angiitis. Once they have ruptured, the prognosis is very poor, with patient mortality amounting to approximately 20%–30% [2]. Hemostasis is achieved by transcatheter arterial embolization or celiotomy. Recently, transcatheter arterial embolization has been performed more often because it is less invasive.

N-Butyl cyanoacrylate (NBCA) is generally accepted as a permanent embolic agent [3] and is used for various conditions. However, some cases of recanalization after embolization for arteriovenous malformation (AVM) have been reported [4, 5]. In our case, recanalization occurred after embolization of ruptured SAA using an NBCA-lipiodol mixture. We herein discuss recanalization after transcatheter arterial embolization with regard to potential contributing factors such as the concentration of NBCA, the mechanism of NBCA solidification, and the conditions of the artery to be embolized.

Case Report

A 65-year-old woman was admitted to the Department of Internal Medicine of our hospital for evaluation of arthralgia and anemia. A definite diagnosis of microscopic polyangiitis was made by renal biopsy. Twenty days after the evaluation, she developed sudden abdominal pain and entered a state of shock. The hemoglobin level fell to 6.9 from 8.9 g/dL. Abdominal computed tomography (CT) depicted abdominal free blood in the left upper quadrant, suggesting active bleeding. Emergent abdominal

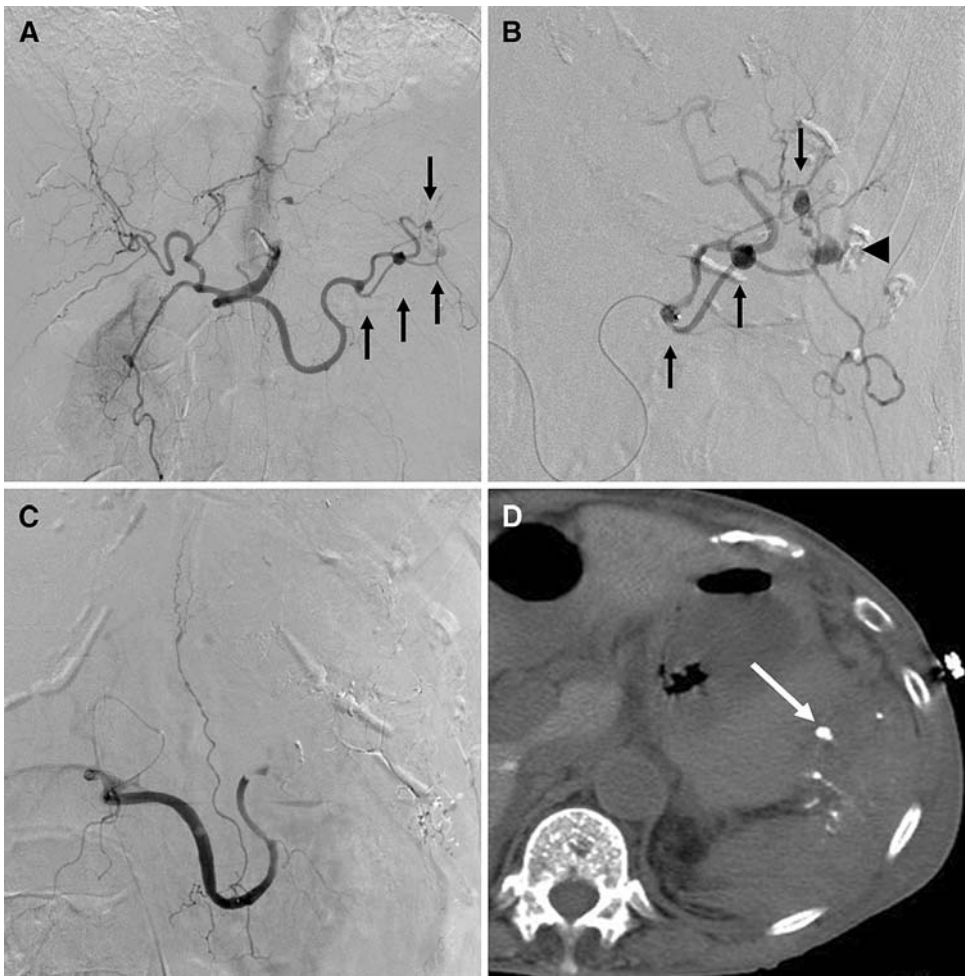

Fig. 1 **A** Celiac arteriogram revealed four splenic artery aneurysms (SAAs; *arrows*). Note the tortuosity of the splenic artery. **B** Selective splenic arteriogram shows SAAs (*arrows*) and extravasation of contrast medium from the distal SAA (*arrowhead*). Splenic artery branches were narrow due to hypovolemic shock. Both the aneurysm and the main trunk of the splenic artery were embolized using NBCA. **C** Splenic arteriogram after embolization shows occlusion of the splenic artery and no visualization of the aneurysms. **D** Unenhanced CT of the abdomen shows accumulation of lipiodol within the ruptured aneurysm (*arrow*)

angiography was performed to detect the bleeding point and to achieve hemostasis.

The celiac arteriogram showed considerable narrowing of branch arteries, which was thought to be due to hypovolemic shock (Fig. 1A). A splenic artery arteriogram revealed four aneurysms of the inferior branch of the splenic artery: a massive extravasation of contrast medium was illustrated from one of the aneurysms, which was located in the secondly distal part of the splenic artery, suggesting active bleeding from the ruptured SAA (Fig. 1B). Attempts to catheterize in the distal side of the ruptured aneurysm with the microcatheter were unsuccessful because of the angiospasm and tortuous anatomy of the splenic artery. Proximal embolization using coils was expected to fail to stop the bleeding because of the likely growth of collateral arteries such as short gastric arteries. Hence, we planned to embolize by injection of a liquid NBCA-lipiodol mixture from the proximal side. NBCA was mixed with lipiodol at a ratio of 1:9, considering the flow velocity and overall length of the target vessel including the four aneurysms. A total of 0.4 ml of the NBCA-lipiodol mixture was injected from the proximal side. Postembolization splenic arteriography showed occlusion of the splenic artery and no extravasation of contrast medium (Fig. 1C).

After this treatment the patient recovered from the shock. Follow-up CT showed a deposit of lipiodol within the ruptured aneurysm (Fig. 1D), reduction of abdominal hematoma, and splenic infarction, with an infarction rate of 60%. No complication such as pancreatitis occurred.

Fifty days after the initial embolization, the patient again developed abdominal pain and abdominal distention. Abdominal CT showed a washout of lipiodol within the aneurysm and a new large hematoma near the chronic hematoma, suggesting rupture of the SAA (Fig. 2A).

Emergency angiography was immediately performed. The splenic artery aneurysm showed recanalization of the splenic artery from the main trunk to the peripheral branches and reperfusion and enlargement of the previously embolized aneurysm (Fig. 2B). This time, the vital signs were stable and there was no spasm of visceral arteries. We successfully catheterized to the distal part of the splenic artery communicating to the short gastric artery located on the distal side of the aneurysm, and microcoils were used to

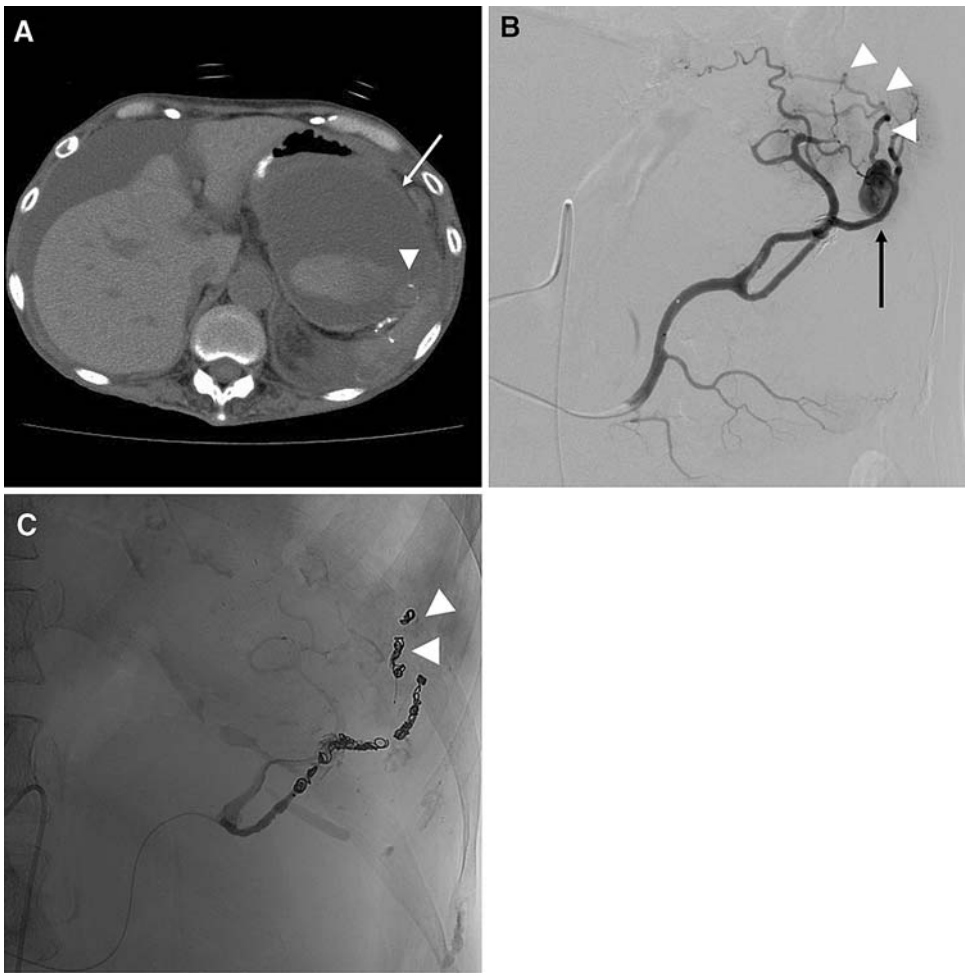

Fig. 2 **A** Unenhanced abdominal CT 50 days after the initial embolization shows enlargement of the treated hematoma in the left upper quadrant (*arrow*) and washout of lipiodol within the ruptured aneurysm (*arrowhead*). **B** Splenic arteriogram shows recanalization of the splenic artery, from the main trunk to the peripheral branches, and revisualizes the aneurysm which was previously ruptured and embolized (*arrow*). Note the enlarged short gastric artery distal to the ruptured aneurysm (*arrowheads*). **C** The recanalized splenic artery aneurysm was embolized again from the distal to the proximal side of the aneurysm using microcoils. The short gastric artery distal to the ruptured aneurysm was also embolized (*arrowheads*)

embolize the splenic artery from the distal to the proximal side of the lipiodol-accumulated aneurysm, including proximal aneurysms (Fig. 2C). Since this second embolization, there has been no recurrence of bleeding.

Discussion

NBCA is a liquid embolic agent that causes rapid polymerization after injection and embolizes vessels. It has various uses: in transcatheter arterial embolization of aneurysms and AVMs of visceral, spinal, and cerebral arteries, in percutaneous embolization of superficial arteriovenous malformations, and in endoscopic embolization of esophagogastric varices.

When we use metallic coils to embolize a visceral aneurysm, we have to deliver the catheter very close to the releasing site of the coils. If a catheter cannot be delivered to such a location, it is impossible to use a microcoil. NBCA offers the advantage of being able to reach the aneurysm through the bloodstream even if it is difficult for us to deliver a catheter near the diseased segment or beyond the diseased segment because of the extreme tortuosity or angiospasm of parent arteries. Hence, we can embolize both the aneurysm and the parent artery quickly by injection of a NBCA-lipiodol mixture from proximal to the diseased segment. On the other hand, for sufficient embolization, it is necessary to carefully determine the dose, concentration, and speed of injection of the NBCA-lipiodol mixture by considering both the speed of blood flow and caliber of the parent artery and the volume of embolized area. Generally speaking, the more lipiodol used to dilute the NBCA, the longer the duration of polymerization. In this sense, NBCA administration requires some experience with its use because of the various difficulties of flow control, the potential for rapid polymerization, and tissue-adhesive complications.

Reactions after embolization with NBCA are considered to be a combination of tissue reactions and polymerization of NBCA. The former occurs by infiltration of neutrophils and macrophages, which does not depend on the concentration of NBCA with respect to the severity of vascular injury and the subsequent tissue reaction to it [6]. The latter occurs by physical occlusion of the vessels by the

polymerized NBCA itself; the degree of polymerization can differ depending on the concentration of the NBCA-lipiodol mixture. Recanalization may occur when a low concentration of NBCA-lipiodol mixture is used, as in this case (NBCA:lipiodol, 1:9) or when the infused NBCA-lipiodol mixture is watered down by high blood flow.

Results of investigations suggesting the possibility of recanalization after embolization with NBCA have been reported [7]. However, Sadato et al. demonstrated that recanalization was not seen after embolization of renal arteries, which are end arteries, because collateral vessels rarely grow after embolization of this kind of artery [8]. Recanalization after embolization is a possibility when the embolic materials are washed away by newly formed collateral flow [7]. Also, in clinical settings, cases of recanalization of an AVM, which was previously embolized even with permanent embolic materials, have been reported [4, 5]. It is considered that unless the entire nidus is packed with permanent embolic materials, the residual unpacked portion of the AVM may recruit new feeding vessels and be recirculated from them [8]. In this way, reperfusion within the lesions due to the development of collateral vessels may participate in recanalization after NBCA-lipiodol embolization.

In this case, the angiospasm of the splenic artery had improved when the aneurysm recurred. In our case, the instability of the hemodynamics might have affected the permanence of the embolization. Previous basic studies of NBCA embolization using models did not examine the changes of hemodynamics in various settings, especially that of massive bleeding [8]. When patients return to hemostasis after hemorrhagic shock, twitched vessels might return to normal condition, then the embolic materials can be washed away, leading to the recanalization of aneurysm. The results of this case demonstrate that reperfusion by improved systemic circulatory hemodynamics may participate in the recanalization of the embolized aneurysm.

The causes of recanalization in our case were considered to be as follows: (i) a weak thrombogenic effect due to the use of a low concentration of NBCA-lipiodol mixture; (ii) improvement of angiospasm due to release from hemorrhagic shock; and (iii) retrograde reperfusion via the collateral vessels.

In summary, we report a case of ruptured SAA which was recanalized after embolization using NBCA. Although NBCA is believed to be a permanent embolic material, it may only have a temporary embolic effect in some cases, as recanalization might occur due to variables such as the concentration of the NBCA-lipiodol mixture and the instability of patient hemodynamics. The interventional radiologist should fully recognize that recanalization after NBCA embolization can occur due to various causes. In using an NBCA-lipiodol mixture to embolize a ruptured visceral aneurysm, it is critical both to carefully select the optimal variables for embolization according to the case particulars and to provide careful follow-up after treatment.

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