Case Report

Successful Treatment of Subcapsular Hepatic Hemorrhage Concomitant with Diffuse Arterioportal Shunt by Transcatheter Arterial Embolization

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Abstract

We present a case of subcapsular hepatic hemorrhage with a concomitant diffuse arterioportal shunt successfully treated with transcatheter arterial embolization. An 85-year-old man with duodenal carcinoma developed hemorrhagic shock three days after pancreaticoduodenectomy. Contrast-enhanced computed tomography revealed an extensive subcapsular hepatic hematoma with extravasation. At the same time, diagnostic angiography showed innumerable foci of petechial extravasation from disrupted isolated arteries and the right inferior phrenic artery. In addition, a comorbid diffuse arterioportal shunt in the hematoma area was detected. We performed transcatheter arterial embolization on the peripheral side of the hepatic artery while preserving the proximal portion. Subsequently, the transcatheter arterial embolization for the right inferior phrenic artery was also performed. Complete hemostasis and occlusion of the arterioportal shunt were successful without fulminant liver failure.

Key words: subcapsular hepatic hematoma, arterioportal shunt, transcatheter arterial embolization

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Introduction

Hepatic subcapsular hematoma is rare and associated with blunt trauma, ruptured liver tumor, hemolysis-elevated liver enzymes-low platelets (HELLP) syndrome, abdominal surgery, and endoscopic retrograde cholangiopancreatography [1-6]. Subcapsular hematomas can develop into life-threatening conditions, such as intra-abdominal bleeding and hepatic compartment syndrome, which require immediate treatment [1]. Several previous studies reported the usefulness of transcatheter arterial embolization (TAE) for hemostasis of subcapsular hematoma [1, 4-7]. However, the case of subcapsular hematoma concomitant with a diffuse arterioportal shunt is very rare [6], and treatment strategies for this condition have not been established.

Herein, we report a rare case of subcapsular hematoma concomitant with a diffuse arterioportal shunt successfully treated with TAE.

Case Report

An 85-year-old man with duodenal carcinoma presented to our institute for surgery. His medical history included hypertension and cerebral aneurysm. Preoperative blood tests showed no obvious abnormalities. He also had no coagulation abnormality and did not take anticoagulation drugs. In addition, a preoperative contrast-enhanced computed tomography (CT) revealed no evidence of liver metastasis, liver cirrhosis, or anomaly of blood flow, including an arte-

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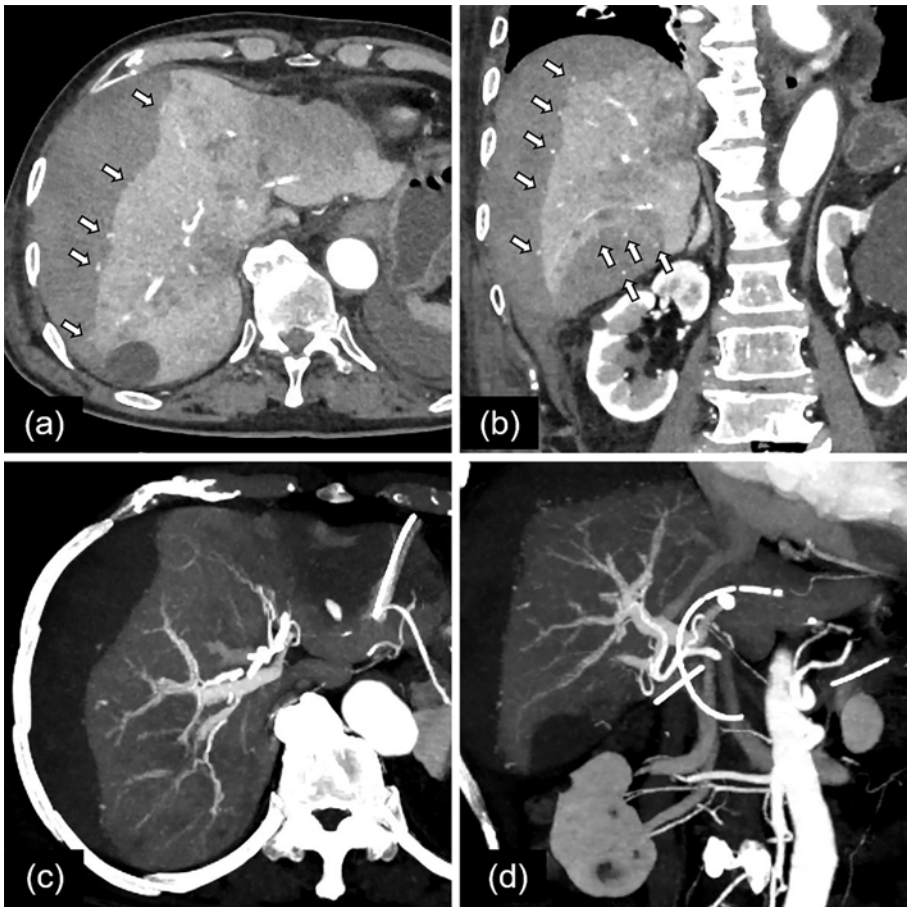

Figure 1. Contrast-enhanced computed tomography (CT) at the onset of hepatic subcapsular hemorr hage. a), b) Multiplanar reconstruction image of late arterial phase. The arrows indicate microaneurysms originating from isolated arteries and capsular plexus. c), d) Partial maximum intensity projection images of early arterial phase with the focus on blood vessels. Note that the portal veins are visualized along the hepatic arteries.

rioportal shunt. The patient underwent pancreaticoduodenectomy with no complications, and the postoperative course was good. However, three days after the surgery, he developed sudden onset of severe abdominal pain with hemorrhagic shock. Laboratory tests revealed elevated liver enzymes (alanine aminotransferase 148 IU/L, aspartate aminotransferase 181 IU/L), mildly elevated serum bilirubin (total bilirubin 1.17 mg/dl, direct bilirubin 0.48 mg/dl), and a drop of hemoglobin from 15.9 g/dL to 10.7 g/dL. Contrast-enhanced CT showed an extensive subcapsular hepatic hematoma in the right hepatic lobe with multiple extravasations and microaneurysms thought to originate from the isolated artery and capsular plexus (**Fig. 1a and b**). In addition, the portal vein was adjacent to the subcapsular hematoma in the arterial phase. However, the main trunk of the portal vein was difficult to see, indicating a diffuse arterioportal shunt (**Fig. 1c and d**). After discussing the treatment options with the surgeon, we decided to perform TAE because reoperation in the early postoperative period is a high-risk procedure for this patient. The patient and the patient's family provided informed consent.

We inserted an 4-F sheath into the right femoral artery and performed digital subtraction angiography (DSA) of the celiac artery using a 4.2-F shepherd hook catheter (Goodman, Nagoya, Japan), which revealed innumerable foci of petechial extravasation and microaneurysms from disrupted isolated arteries and capsular plexus (**Fig. 2a**). Additionally, there was a reversal portal vein flow in the area corresponding to the subcapsular hematoma in the parenchymal phase, which was consistent with the diagnosis of a diffuse arterioportal shunt (**Fig. 2b**). Moreover, the hepatic portal vein flow from the splenic artery was not visualized in the late phase. A similar finding was observed in the selective DSA of the anterior and posterior segmental arteries of the right hepatic artery using a coaxial system with a 1.7-F microcatheter (Prograte λ, Terumo, Tokyo, Japan). Based on these findings, a nonselective embolization of the right hepatic artery was needed, but it might have led to extensive hepatic infarction. Therefore, to preserve the hepatic artery as much as possible, we performed a TAE of the peripheral side of the hepatic arteries while preserving the proximal side as much as possible. We selected the subsegmental arteries of the right hepatic artery (A5, 6, 7, and 8, respectively) and embolized each artery with approximately 1 mm of gelatin

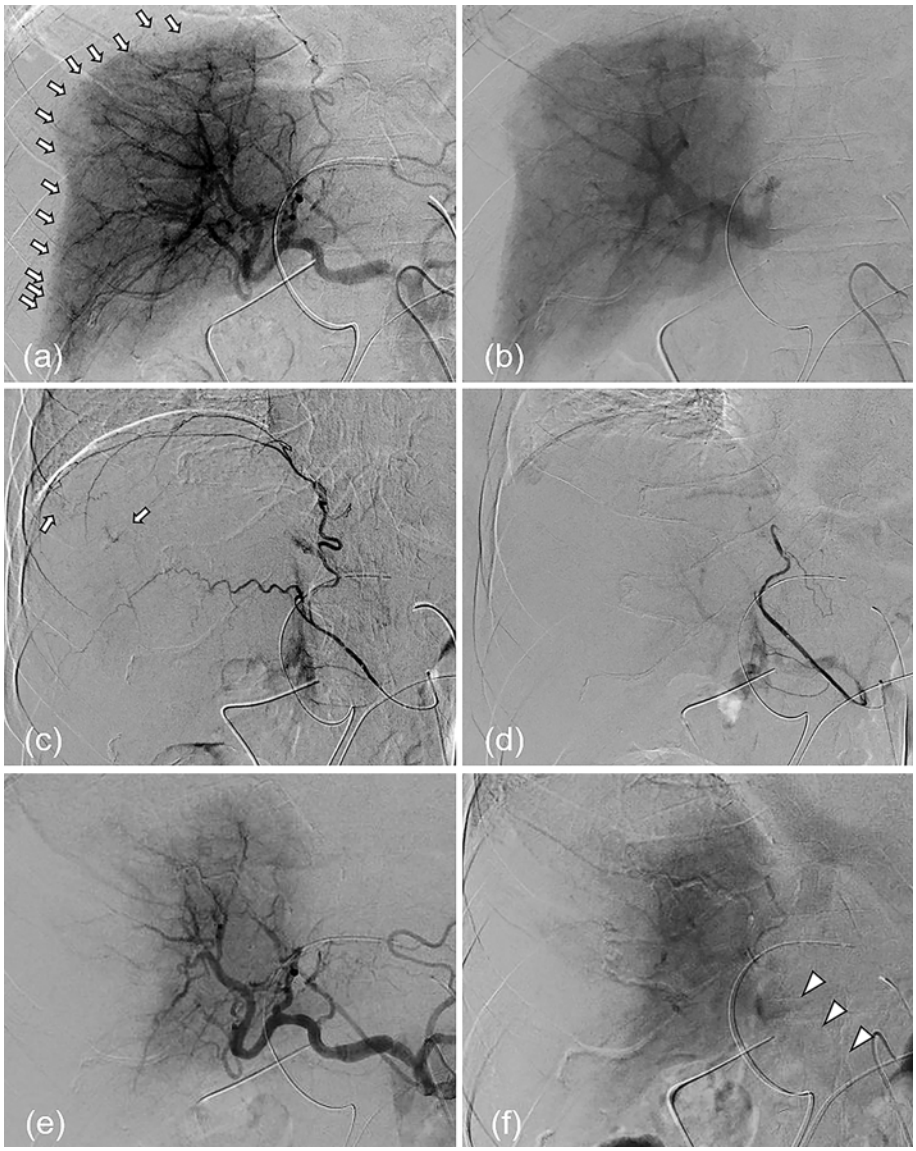

Figure 2. Angiographic findings during transcatheter arterial embolization (TAE). a), b) Digital subtraction angiography (DSA) of the celiac artery. Multiple microaneurysms are observed in the subcapsular hematoma area (arrows in a). b) In the parenchymal phase, reversal of portal vein flow is observed (b). c), d) DSA of the right inferior phrenic artery. Multiple extravasations are observed in the area of subcapsular hematoma (arrows in c). DSA after selective embolization of anterior and posterior branches (d). e), f) DSA of the celiac artery after TAE. Note the disappearance of microaneurysms, whereas the visualization of the proximal portion of hepatic arteries is relatively preserved (e). In the parenchymal phase, the hepatopetal portal vein flow from the splenic vein is observed (arrowheads in f).

sponge particles (Seresucue, Astellas Pharma Inc., Tokyo, Japan) until the extravasation and microaneurysms disappeared. We confirmed the preservation of a proximal portion of the target arteries during TAE with frequent angiography. Subsequently, selective DSA of the right inferior phrenic artery was performed using a coaxial system with a 2.4-F steerable microcatheter (Leonis Mova, Sumitomo Bakelite Co., LTD., Tokyo, Japan), which revealed extravasation and microaneurysms in the hematoma (**Fig. 2c**). TAE using approximately 1 mm gelatin sponge particles was performed, and hemostasis was achieved (**Fig. 2d**). The DSA of the celiac artery after TAE showed that the reversal of portal vein flow, as well as the extravasation and microaneurysms, disappeared (**Fig. 2e**). Additionally, the hepatopetal portal vein flow from the splenic vein was observed (**Fig. 2f**). At that time, DSA of the superior mesenteric artery and right renal capsular artery was not performed because large amounts of contrast media were used in contrast-enhanced CT and TAE, and hepatopetal portal vein flow was maintained in the DSA of the celiac artery after TAE. The next day, diagnostic angiography was performed to confirm the portal blood flow and hemostasis. The hepatopetal portal vein flow was clearly

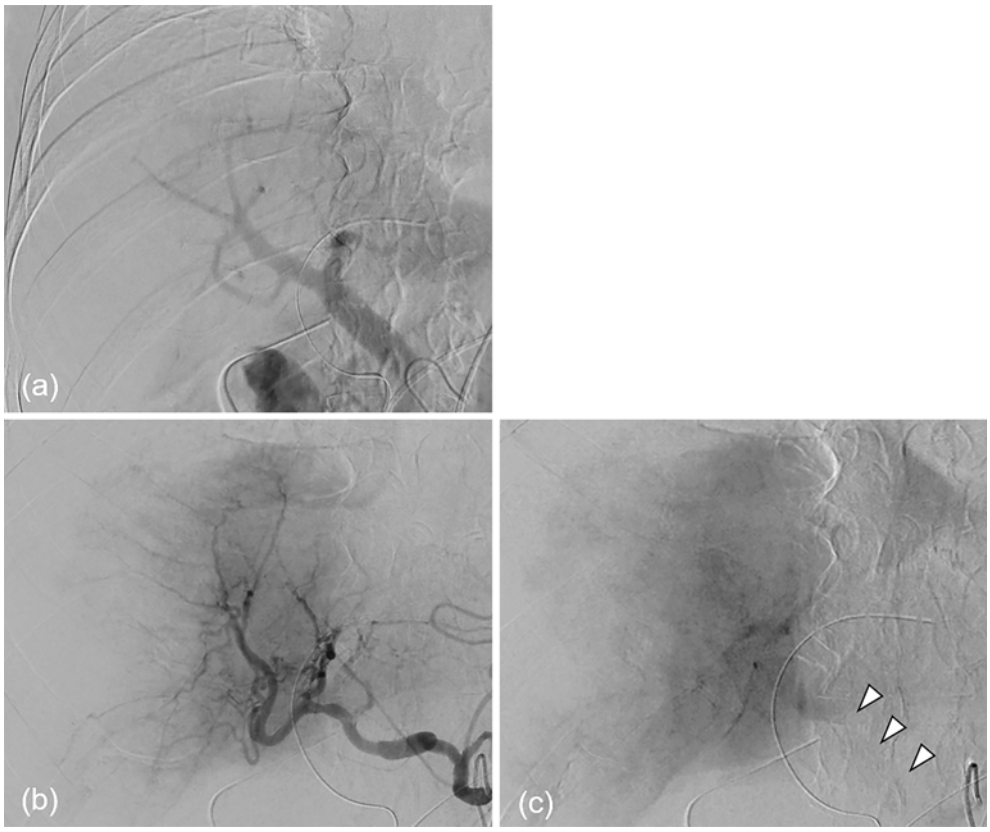

Figure 3. DSA findings performed at 1 day after TAE. a) DSA of the superior mesenteric artery. The hepatopetal portal vein flow is clearly visualized. b), c) DSA of the celiac artery. No evidence of recurrence of microaneurysms and reversal of portal vein flow. Arrowheads indicate hepatopetal portal vein flow from the splenic vein (arrowheads in c).

visualized on DSA of the superior mesenteric artery (**Fig. 3 a**). In addition, DSA of the celiac artery revealed no evidence of extravasation, aneurysms, or reversal of portal vein flow (**Fig. 3b and c**). After TAE, liver enzymes temporarily increased but gradually improved and recovered to almost normal levels after 8 days (**Fig. 4**). A CT scan performed 8 days after TAE showed a mild hepatic infarction adjacent to the subcapsular hematoma (**Fig. 5**). The follow-up CT revealed a gradual reduction of the subcapsular hematoma. However, the patient died approximately 4 months after TAE due to sepsis secondary to aspiration pneumonia.

Discussion

The major clinical relevance of this case report is that we successfully treated a hepatic subcapsular hemorrhage with a concomitant diffuse arterioportal shunt by TAE.

The etiology of hepatic subcapsular hematoma reportedly included ruptured liver tumor, trauma, HELLP syndrome, and iatrogenic causes. In addition, it reportedly occurs after surgeries and endoscopic procedures, including endoscopic retrograde cholangiopancreatography, open or laparoscopic cholecystostomy, and laparoscopic adrenalectomy [1-6, 8]. The authors in previous case reports speculated that excessive traction of the gallbladder and excessive manipulation of the liver during surgery were the causes of the hematoma [3]. In this case, cholecystectomy was performed in association with pancreaticoduodenectomy, but it is unclear whether it was involved in developing a subcapsular hematoma.

Cases of subcapsular hematoma concomitant with diffuse arterioportal shunts are rare. There is only one case of HELLP syndrome with subcapsular hematoma comorbid with an arterioportal shunt [6]. The precise mechanism of a diffuse arterioportal shunt in the liver parenchyma adjacent to the hematoma is unknown. We speculate that rapid hemodynamic changes in capillary arteries and narrowing of sinusoids due to compression by the hematoma may be involved in the pathogenesis. Ahn et al. proposed that hemodynamic changes in transvasal communications, transsinusoidal communications, and transplexal communications are the mechanisms by which arterioportal shunts occur in cirrhotic and non-cirrhotic livers [9]. Previous studies using animal models of liver cirrhosis have demonstrated that progressive hypertrophy of the peribiliary capillary plexus (transplexal communications) and widening of the vasa vasorum of the portal vein (transvasal communications) at the peripheral subcapsular portion of the liver could accentuate the gross appearance of the arterioportal shunt [9]. Additionally, in liver cirrhosis, capillarization of sinusoids and obliteration of peripheral hepatic venules could lead to retrograde filling of small portal vein branches (transsinusoidal communications)[9]. Although there is no sufficient *in vivo* histological

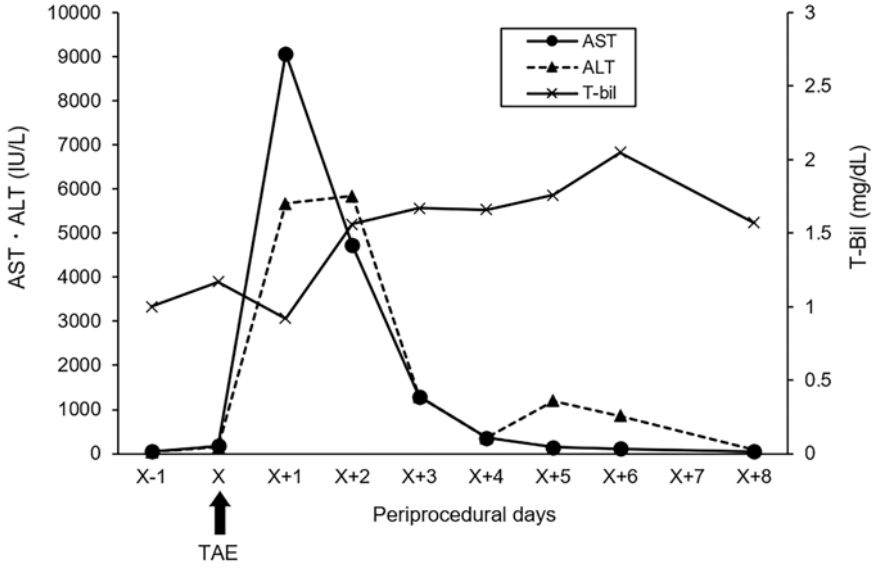

Figure 4. Changes in liver enzymes in the periprocedural period. TAE: transcatheter arterial embolization, AST: aspartate aminotransferase, ALT: alanine aminotransferase, T-Bil: total bilirubin.

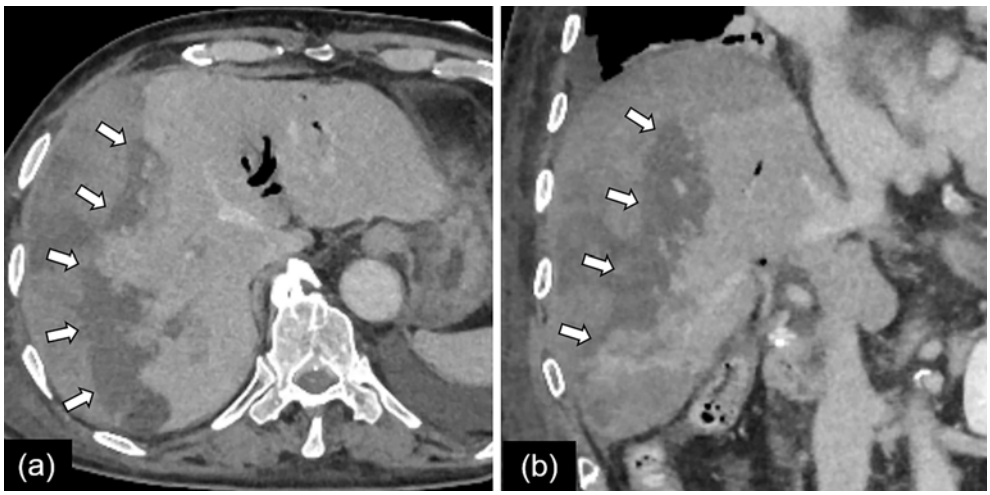

Figure 5. Contrast-enhanced CT image acquired 8 days after TAE. a) Axial image; b) Coronal image. Arrows indicate the border between the subcapsular hematoma and liver parenchyma. Note the area of the liver parenchyma with poor contrast enhancement just below the capsule; this finding is consistent with the hepatic infarction.

proof, in cases of subcapsular hematoma, hemodynamic changes similar to liver cirrhosis may have occurred in the liver parenchyma adjacent to the hematoma, possibly causing an arterioportal shunt.

There is no established consensus for the standard treatment of subcapsular hepatic hematomas. Traditionally, surgical hemostasis has been performed; however, TAE has become the first treatment choice in recent years because it is effective and less invasive. However, in a diffuse arterioportal shunt concomitant with subcapsular hematoma, as in our case, TAE should be performed with discretion. In patients with reduced portal vein flow, excessive embolization of hepatic arteries could lead to acute liver failure due to hepatic infarction. In such cases, post-embolization mortality is known to be high [1]. However, the subcapsular hematoma in our case was in a wide area of the right hepatic lobe, making selective embolization difficult. Therefore, we performed a TAE with the preservation of the proximal portion of the hepatic artery as much as possible, resulting in hemostasis and occlusion of the arterioportal shunt without fulminant hepatic failure. Additionally, collateral circulation should be considered when performing TAE for subcapsular hematoma. The isolated arteries and capsular plexus are generally known to be the arteries responsible for subcapsular hematomas, but these arteries usually communicate with extrahepatic arteries, such as the inferior phrenic artery and intercostal artery [7]. Therefore, if contrast-enhanced CT and DSA indicate extrahepatic blood flow, TAE should be performed for these vessels. Regarding the detection of extravasation from the isolated artery, the usefulness of DSA

using carbon dioxide gas has been reported [5]. In our case, embolization by TAE in the right inferior phrenic artery was performed together with TAE in the hepatic artery, resulting in a good clinical outcome without rebleeding.

Percutaneous hematoma drainage is reportedly another treatment option for subcapsular hematomas. Terayama et al. reported that hematoma drainage was needed in cases of hepatic compartment syndrome to preserve liver function [5]. Lee et al. reported a case of hepatic compartment syndrome due to subcapsular hematoma in which liver function improved rapidly by performing percutaneous hematoma drainage in addition to TAE [1]. In our case, we did not perform hematoma drainage early because of concerns about rebleeding and the relatively rapid improvement of liver function after TAE. However, it may be better to perform rapid hematoma drainage in patients with severe liver failure.

In conclusion, TAE with preservation of the proximal portion of the hepatic artery could be a useful non-invasive treatment for hepatic subcapsular hemorrhage, even in cases with diffuse arterioportal shunts.

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Conflict of Interest: None

Author Contribution: Emiko Chiba: Methodology, Data curation, Writing- Original draft preparation.

Kohei Hamamoto: Conceptualization, Data curation, Writing- Reviewing and Editing.

Maya Oishi: Writing- Reviewing and Editing.

Hironao Yuzawa: Writing- Reviewing and Editing.

Noriko Oyama-Manabe: Writing- Reviewing and Editing.

Hiroshi Shinmoto: Writing- Reviewing and Editing.

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