Paralytic Ileus due to Superior Mesenteric Venous Thrombosis after Transarterial Injection for Hepatocellular Carcinoma

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Abstract

A 69-year-old man was admitted to hospital with abdominal pain. In the four years prior to his presentation, he had undergone repeated transarterial chemoembolizations and injections for hepatocellular carcinoma. He underwent his 8th transcatheter arterial therapy one month prior to admission. Abdominal X-rays and contrast-enhanced computed tomography showed large amounts of small intestinal gas and venous thrombosis from the portal vein to the superior mesenteric vein, respectively. The thrombosis was reduced after anticoagulation therapy (heparin, antithrombin III, danaparoid sodium and warfarin). This is the first case report of paralytic ileus due to superior mesenteric venous thrombosis after transcatheter arterial therapy for hepatocellular carcinoma with an arterioportal shunt.

Key words: superior mesenteric venous thrombosis, transarterial chemoembolization, hepatocellular carcinoma, arterioportal shunt


Introduction

Hepatocellular carcinoma (HCC) often recurs despite the provision of curative treatments such as liver resection, liver transplantation and radiofrequency ablation. Transarterial chemoembolization (TACE) is a therapeutic option for the treatment of unresectable HCC and has improved the prognosis for HCC patients (1, 2). On the other hand, various adverse effects, which are collectively referred to as postembolization syndrome, have been associated with TACE (3, 4). Although arterial therapies such as TACE are in wide use and ileus is one of the most common adverse effects after TACE, few cases of ileus after arterial therapy have been described in the literature.

Arterioportal (A-P) shunting is often observed in patients with HCC. It makes TACE difficult to perform because of the risk of liver failure. Transcatheter arterial therapy without embolization may thus be performed in HCC patients with an A-P shunt in order to maintain liver function after therapy. We herein report a rare case of paralytic ileus due to mesenteric venous thrombosis after transcatheter arterial injection and discuss the cause.

Case Report

A 69-year-old man, who had been diagnosed with HCC due to hepatitis C virus-related liver cirrhosis 4 years previously (Fig. 1), had been undergoing repeated treatment with transcatheter arterial therapy. He underwent his 8th transcatheter arterial therapy (Lipiodol transcatheter arterial injection; Lip-TAI) 1 month prior to his admission. Lip-TAI was performed after the presence of an A-P shunt was confirmed on arteriography at the 6th transcatheter arterial therapy. With the 8th transcatheter arterial therapy, an arterial catheter was inserted into the right femoral artery using the Seldinger method and advanced to the hepatic artery. The feeding artery for the HCC was selected and a solution containing 30 mg of cisplatin (IA call, Nihon Kayaku Pharmacy, Tokyo, Japan) and 3.0 mL of iodized oil (Lipiodol,
Figure 1. Contrast-enhanced CT at the time of the initial HCC diagnosis. The HCC (arrow) is located near the umbilical portion. A) Early phase. B) Equilibrium phase.

Figure 2. CT during arterial portography at the 8th transcatheter arterial therapy. No obvious thrombosis is evident in the main portal tract.

Terumo, Tokyo, Japan) was injected through a 2.3-F microcatheter. No obvious portal vein thrombosis (PVT) was detected during the therapy (Fig. 2). Moreover, there was no thrombosis within the extrahepatic portal venous system including superior mesenteric vein.

The patient was referred to our hospital with abdominal pain 26 days after the 8th arteriotherapy (Lip-TAI). On admission, his consciousness was clear and his body temperature was 36.1°C. The conjunctivae were not jaundiced, and his heart and respiratory sounds were normal. No abnormalities of the chest and skin were identified. The abdomen was soft and distended, showing tenderness in the periumbilical region without signs of peritoneal irritation or audible bowel sounds. A laboratory examination on admission revealed an inflammatory status [white blood cells, 11,000/μL; C-reactive protein (CRP), 4.54 mg/dL] (Table). Abdominal X-ray and computed tomography (CT) showed the noticeable accumulation of small intestinal gas and PVT from the main portal vein to the superior mesenteric or splenic vein, respectively (Fig. 3, 4). Paralytic ileus due to superior mesenteric vein thrombosis was diagnosed based on these findings. Because there was no apparent intestinal tract necrosis on CT, anticoagulation therapy composed of heparin, antithrombin III, and danaparoid sodium was initiated immediately after the insertion of the ileus tube. After the treatment resulted in the reduction of both small intestinal gas and PVT, heparin was changed to warfarin. The clinical findings steadily improved and the patient was discharged 21 days
The prevalence of A-P shunting in patients with HCC is approximately 30-60% (10, 11). A-P shunting attenuates liver function due to the reduction of effective hepatic blood flow in addition to the induction of portal hypertension or hepatic coma (12-14). Moreover, TACE is sometimes difficult to perform in HCC patients with an A-P shunt due to the risk of liver failure or weakness associated with the anticancer effects of therapy (15, 16). We performed Lip-TAI without gelatin particles in the present case after the 6th therapy to avoid the deterioration of liver function; however, paralytic ileus developed unexpectedly after treatment. The current case illustrates that repeated Lip-TAI without embolization could therefore also be a risk factor for ileus in HCC patients with an A-P shunt, similar to TACE. Some studies have reported the efficacy of the embolization of A-P shunts (17-19). Embolization might therefore be worth considering in similar cases to avoid not only liver failure, but also PVT. Of course, the indications for embolization should be carefully considered, because severe hepatic failure has been described after A-P shunt embolization (19).

Although the development of PVT in patients with HCC generally only represents thrombosis due to blood coagulation and tumor embolus (or both), many reports have investigated tumor embolus from a treatment perspective (20-24). In the present case, the clinical course suggested that the PVT was mainly composed of coagulated blood, rather than tumor embolus. Moreover, the thrombosis extended continuously from the left portal vein to the superior mesenteric vein. Superior mesenteric venous thrombosis (SMVT) could thus have been the direct cause of the patient’s paralytic ileus.

Many cases of SMVT are secondary (25). Liver cirrhosis and portal hypertension, both of which were present in this case, represent major causes of secondary SMVT or PVT (26-29). Liver cirrhosis is thought to easily result in PVT due to the congestion of portal blood flow. The development of PVT is associated with the baseline severity of liver disease in patients with cirrhosis (29). Moreover, TACE...
could have greatly affected the formation of PVT and SMVT in the present case, because the presence of an A-P shunt raises the possibility of iodized oil outflow to the portal vein during Lip-TAI. To the best of our knowledge, this is the first case of paralytic ileus due to SMVT after transcatheter arterial therapy for HCC in a patient with an A-P

**Figure 4.** Enhanced CT in hospital. Thrombosis is observed to have spread from the main portal vein to the superior mesenteric vein or splenic vein on admission (A: arrows). The HCC has also spread (dashed circle). The patient’s thrombosis almost completely disappeared after anticoagulant therapy (B: arrows).

**Figure 5.** The clinical course.
The symptoms of SMVT, which include abdominal pain, fullness, nausea, vomiting, diarrhea and melena, are non-specific. Moreover, the progression of the symptoms is gradual in comparison to superior mesenteric arterial thrombosis (30, 31). Thus, it is not rare for SMVT to be first diagnosed in an advanced state with findings such as intestinal necrosis, which warrant surgical intervention (25, 32). Anticoagulant therapy is effective in patients who receive an early diagnosis (33). Reports have described the efficacy of danaparoid sodium in addition to traditional urokinase and tissue plasminogen activator in patients with SMVT or PVT (34-36). The route of anticoagulant therapy is usually through a peripheral vein, but a small number of reports have described using a route through the portal vein via a transcervical or percutaneous transhepatic approach (37, 38). We should take the different treatment strategies into account for patients with SMVT.

In conclusion, TACE for HCC patients carries a risk of shunt. We should be aware of the complications that may be associated with A-P shunting and consider transcatheter arterial therapy for HCC patients with an A-P shunt in order to avoid PVT or SMVT. Immediate therapy is required in cases where PVT is detected, thus careful attention should be paid to patients after TACE.

The authors state that they have no Conflict of Interest (COI).

References


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