

Case Report

## A Case of Acute Superior Mesenteric Artery Embolism with Severe Ischemic Liver Injury Successfully Treated by Endovascular Treatment

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We describe an interesting clinical course of a patient who developed severe ischemic liver injury due to acute embolism of the superior mesenteric artery (SMA) and celiac artery. A 70-year-old man was hospitalized for abdominal pain and diarrhea. Abdominal computed tomography demonstrated a variant common hepatic artery arising from the SMA and multiple thromboembolic occlusions of visceral arteries, including the SMA and celiac artery. Laboratory data showed markedly elevated hepatic enzymes, which increased after admission despite the initiation of systemic anticoagulant and thrombolytic therapy. The patient was successfully treated by endovascular recanalization of the SMA occlusion via transcatheter embolus aspiration, thrombolysis, balloon angioplasty, and stent placement. Severe ischemic liver injury may occur in the setting of synchronous embolism of the SMA and celiac artery, and these phenomena may have a critical impact on the choice of treatment strategies and prognosis. Endovascular treatment appears to an effective treatment option.

**Key words:** superior mesenteric artery, celiac artery, embolism, liver ischemia, endovascular treatment

Acute superior mesenteric artery (SMA) occlusion is a life-threatening condition, with bowel ischemia being the most critical complication. Usually, liver ischemia is not a crucial problem in this condition. Although ischemic liver injury can occur when acute SMA occlusion is accompanied by celiac artery occlusion, this condition is rare with a limited number of published case reports [1-5]. Furthermore, there is only 1 case report that describes endovascular treatment performed for such a condition [5].

The present report describes a unique case of acute thromboembolic occlusions of multiple visceral

arteries, including the SMA, celiac artery, and splenic artery, that developed in a patient who had a variant common hepatic artery arising from the SMA. Ischemic liver injury, rather than bowel ischemia, was the most critical problem that dictated the course of treatment. He was successfully treated by endovascular recanalization of the SMA occlusion.

### Case Report

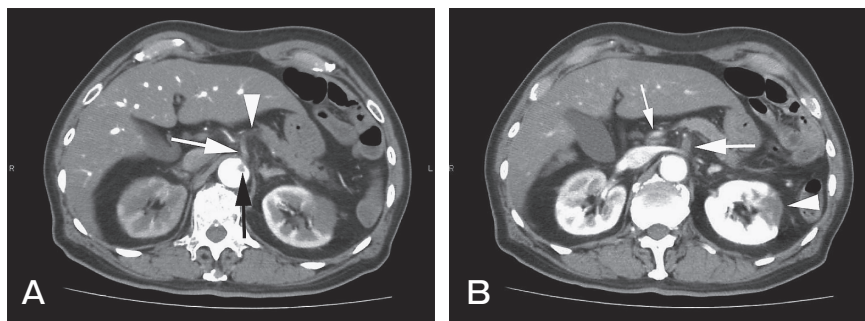
A 70-year-old man was admitted to our hospital because of abdominal pain and diarrhea lasting for 4 days. On physical examination, the abdomen was soft, and there was slight upper abdominal tenderness. Laboratory examination on admission showed an elevated white blood cell count (11,410/ $\mu$ L) and elevated

hepatic enzyme levels: aspartate aminotransaminase (AST), alanine aminotransaminase (ALT), and lactate dehydrogenase (LDH) of 832IU/L, 843IU/L, and 1,212IU/L, respectively. Creatine kinase (CK) was within the normal range. Abdominal dynamic computed tomography (CT) demonstrated a variant common hepatic artery arising from the SMA. The splenic artery and left gastric artery arose from the celiac artery. Thromboembolic occlusion of the SMA, celiac artery, and splenic artery was revealed on the CT (Fig. 1A, B). The thrombus of the SMA was located on the proximal side of the common hepatic artery origin. A large floating thrombus was also observed in the abdominal aorta that was contiguous with the thrombus in the celiac artery and SMA (Fig. 1A). The blood flow in the distal portion of the SMA and the common hepatic artery was barely maintained by collateral flow from the inferior mesenteric artery (IMA), and the hepatic arteries appeared narrow. The portal vein (PV) appeared significantly small, probably because of the reduction in mesenteric and splenic venous return (Fig. 1B). In addition, CT showed partial infarctions of the liver, spleen, and both kidneys (Fig. 1B). Bowel-wall enhancement was normal throughout, except that it was slightly reduced in a portion of the small intestine.

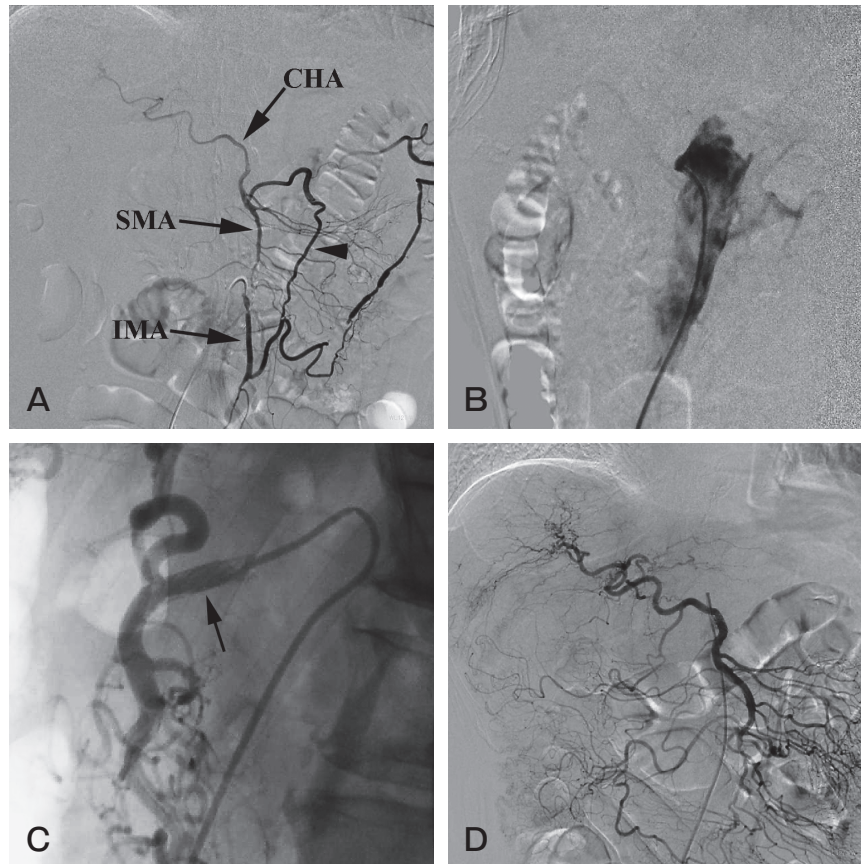
Because the patient had a history of atrial fibrillation, the presence of multiple cardiogenic emboli in the visceral arteries was suspected. Although immediate endovascular treatment was considered an option, we were concerned that catheter intervention might cause migration of the thrombi leading to peripheral embolization.

Therefore, systemic anticoagulant and thrombolytic therapy by transvenous administration of heparin and urokinase (UK) was selected as the initial treatment. Continuous infusion of 600IU per hour of heparin and 20,000IU per hour of UK was initiated, followed by blood examination approximately 6h after admission. The examination revealed markedly elevated hepatic enzyme levels: AST, ALT, and LDH were 3,919IU/L, 3,789IU/L, and 6,929IU/L, respectively. However, CK remained within the normal range, and no other signs of bowel necrosis were observed. We suspected that severe ischemic liver injury would be fatal if conservative therapy was continued; therefore, we elected to perform emergent endovascular treatment.

We inserted a 6-French (Fr) sheath from the right femoral artery. IMA angiography using a 4-Fr shepherd hook catheter showed collateral blood flow from the IMA to the SMA, a common hepatic artery arising from the SMA, and scarcely delineated peripheral branches of the hepatic artery (Fig. 2A). Total obstruction of the proximal SMA was observed on SMA angiography using a 6-Fr cobra-shaped guiding catheter (Brite Tip, Cordis, Miami Lakes, FL, USA) (Fig. 2B). A 0.035-inch guide wire was advanced past the embolus deep into the SMA, and then a 4-Fr cobra-shaped catheter was inserted through the 6-Fr guiding catheter. These catheters were advanced over the wire into the embolus. Embolus aspiration was performed using the 6-Fr and 4-Fr catheters. These catheters were extracted during aspiration and re-introduced for further aspiration;



**Fig. 1** Dynamic computed tomography images on admission. **A**, An axial early-phase image of a slice at the level of the origin of the celiac artery. The white arrow and arrowhead indicate thromboembolic occlusion of the celiac and splenic arteries, respectively. The black arrow indicates the floating thrombus in the abdominal aorta; **B**, An axial portal-phase image of a slice at the level of the origin of the superior mesenteric artery (SMA). The large arrow indicates thromboembolic occlusion of the proximal SMA. The portal vein appears small (small arrow). The arrowhead indicates partial infarctions of the kidney.



**Fig. 2** **A**, Pre-treatment inferior mesenteric artery (IMA) angiography. The SMA, IMA, and common hepatic artery (CHA) are indicated in the figure. The arrowhead indicates the collateral vessel from the IMA to the SMA; **B**, Pre-treatment SMA angiography (lateral view). Total obstruction of the proximal SMA is delineated; **C**, SMA angiography (lateral view) 1 day after stent placement. The stents were placed in the proximal SMA, covering the entire length of the stenotic portion (arrow). Recanalization of the SMA was demonstrated; **D**, SMA angiography performed on the 3rd hospital day. Blood flow of the SMA and the common hepatic artery is markedly improved, and peripheral branches of the hepatic artery are articulately delineated.

this process was repeated several times. SMA angiography after aspiration showed recanalization of the proximal SMA, but severe stenosis due to the residual thrombus persisted. Next, pharmacomechanical thrombolysis was performed using a 4-Fr pulse-spray catheter (Fountain Infusion System, Merit Medical, South Jordan, UT, USA). The thrombolytic solution was prepared by dissolving 120,000 IU of UK in 20 mL of saline. Pulse injections were applied by manual injection of 0.5-mL aliquots approximately every 30 sec. After thrombolysis, the patency of the stenotic portion of the proximal SMA had slightly improved, but further intervention appeared necessary. Subsequently, percutaneous transluminal angioplasty (PTA) was performed using a balloon

catheter (4 mm in diameter; Sterling, Boston Scientific, Natick, MA, USA). Unfortunately, no further improvement in stenosis was observed on SMA angiography after PTA. We therefore decided to perform stent placement. Two stents (18 mm and 15 mm long, both 5 mm in diameter; PALMAZ Genesis, Cordis, Miami Lakes, FL, USA) were placed covering the entire length of the stenotic portion of the proximal SMA (Fig. 2C). Thereafter, a 5-Fr shepherd hook catheter was placed in the proximal SMA, and continuous infusion of UK was initiated at a rate of 10,000 IU per hour.

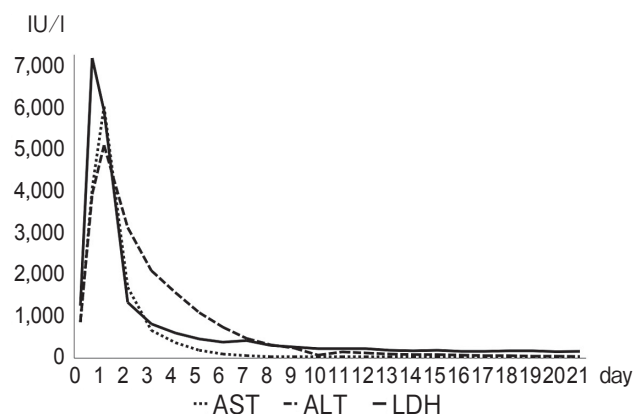
Transarterial infusion of UK was continued for 3 days with concurrent systemic anticoagulant therapy with heparin. Follow-up angiography performed on the

third hospital day showed marked improvement in SMA and hepatic arterial blood flow (Fig. 2D). CT on the same day revealed a small amount of residual thrombus at the origin of the SMA. The CT also revealed disappearance of the floating thrombus in the abdominal aorta and reduction of thrombus volume in the celiac and splenic arteries. For prevention of stent thrombosis, aspirin (100mg/d) therapy was started the day after continuous UK infusion was completed. Transvenous anticoagulant therapy was continued for 10 days and was then switched to oral warfarin anticoagulant therapy. Hepatic enzyme levels steadily declined and had normalized by 2 weeks after admission (Fig. 3). CT performed on the 18th hospital day showed disappearance of the residual thrombus in the

SMA, celiac artery, and splenic artery. The patient fully recovered and was discharged, remaining asymptomatic at the most recent visit, approximately 1 year from the onset.

## Discussion

Despite progress in diagnostic and treatment modalities, a recent study showed that the mortality rate of acute SMA occlusion remains high, at approximately 60% [6]. Because bowel ischemia is generally the most critical complication, rapid revascularization is needed before bowel ischemia progresses to irreversible bowel necrosis. The clinical course of the present case is interesting in the sense that ischemic liver injury, rather than bowel ischemia, was the most critical problem that dictated the course of treatment. To the best of our knowledge, there are only 5 published case reports in English literature that describe acute SMA occlusion with ischemic liver injury (Table 1). All previously reported cases presented with synchronous occlusion of the SMA and celiac artery. Among them, only 1 case underwent endovascular treatment. Therefore, the present case represents the second reported case describing endovascular treatment for ischemic liver injury in association with acute SMA occlusion. Furthermore, there have been no case reports of acute SMA occlusion in a patient with anatomical variation of the hepatic artery, as was the case in our patient. Because of this anatomical variation of the hepatic artery, *i.e.*, a common hepatic artery arising from the SMA, the SMA embolism induced hepatic arterial hypoperfusion as well as mesenteric blood flow reduc-



**Fig. 3** Fluctuation of hepatic enzyme levels. The levels of aspartate aminotransaminase (AST), alanine aminotransaminase (ALT), and lactate dehydrogenase (LDH) were elevated exponentially on the day of admission; they declined steadily after catheter intervention, and normalized by 2 weeks after admission.

**Table 1** Reported cases of acute SMA occlusion with ischemic liver injury

Author	Age/Sex	Etiology	Occluded Artery	Treatment	Outcome
Semb et al. [1]	61/M	Thrombus	SMA and Celiac artery	Surgical thrombectomy and necrotic bowel resection	Died
Gillet et al. [2]	55/F	Not described	SMA and Celiac artery	Surgical thrombectomy and necrotic bowel resection	Recovered
Young et al. [3]	62/F	Not described	SMA, IMA, and Celiac artery	Aortomesenteric bypass and necrotic bowel resection	Recovered
Rajkovic et al. [4]	70/F	Embolus	SMA and Celiac artery	Only exploratory laparotomy	Died
San Norberto et al. [5]	67/F	Embolus	SMA and Celiac artery	Endovascular recanalization of SMA and celiac artery	Recovered

SMA, superior mesenteric artery; IMA, inferior mesenteric artery.

tion and subsequent portal hypoperfusion. We assume that concurrent celiac and splenic artery embolism was also responsible for the development of severe liver ischemia because of the reduction in splenic venous flow that resulted in portal hypoperfusion.

Because the common hepatic artery arose from the SMA in the present case, we were able to successfully restore mesenteric and hepatic arterial flow concurrently via SMA recanalization without catheter intervention for the celiac and splenic artery emboli. In the case of synchronous thromboembolism to the SMA and celiac artery in a patient with a normal hepatic artery arising from the celiac trunk, intervention for both the SMA and the celiac artery occlusion may be desirable, as in the above-mentioned case report [5]. However, the SMA should perhaps be given priority for recanalization, given that SMA recanalization may contribute to restoration of both mesenteric and hepatic blood flow due to collateral circulation via the pancreatic arcade.

Although laparotomy and surgical embolectomy are still major treatment procedures for acute SMA embolism, endovascular recanalization of the SMA can be an alternative option in patients with no signs of bowel necrosis at the time of diagnosis. Several studies have shown that endovascular treatment has been used with increased frequency for acute mesenteric ischemia in the last decade [7, 8]. The proportion of patients who undergo endovascular procedures for the treatment of acute mesenteric ischemia has been reported as 12–36%, varying by institution [6–8]. In the present case, we performed a combination of trans-catheter embolus aspiration, thrombolysis, balloon angioplasty, and stent placement. Recent studies have shown favorable outcomes following trans-catheter embolus aspiration and thrombolysis [9, 10]. Although PTA has been described as a useful method to treat acute embolic SMA occlusion, it is also associated with a risk of peripheral embolization and potential further worsening of bowel ischemia [11]. The efficacy of the stent for acute SMA occlusion is indicated by several studies, but the causes of occlusion in the majority of reported cases were arteriosclerotic thrombosis or dissection of the SMA, rather than cardiogenic embolism [10, 12]. There are only a few reported cases of stent placement for cardiogenic SMA embolism [5, 12, 13]. Thus, the indication for stent placement in such cases remains contro-

versial. We first attempted to revascularize the SMA by embolus aspiration and thrombolysis, but the SMA and common hepatic artery flow remained insufficient after completion of these strategies. Therefore, we performed PTA and stent placement to secure hepatic inflow and avoid irreversible liver failure. However, uncertainty regarding the long-term outcomes following stent placement for acute SMA occlusion remains problematic. Regarding chronic mesenteric ischemia, a recent study showed that the primary 1- and 2-year patency rates of stents placed in mesenteric arteries were 86% and 60%, respectively; the primary-assisted patency rates were 88% and 79%, respectively [14]. The long-time patency of SMA stents in acute settings remains unknown because of the lack of data. Therefore, careful follow-up to check for in-stent stenosis is necessary, and more cases are required before definitive determination of the long-term utility of this technique can be completed.

In conclusion, acute embolism of multiple visceral arteries is a rare but critical condition. Severe ischemic liver injury may occur in the setting of synchronous embolism of the SMA and celiac artery, and these phenomena may have a critical impact on the choice of treatment strategies and prognosis. Endovascular treatment appears to have been an effective treatment option in this rare clinical scenario.

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