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Case Report

Synchronous Total Occlusion of the Celiac Axis and Superior Mesenteric Artery: An Autopsy Case

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Acute mesenteric ischemia (AMI) is often caused by superior mesenteric artery (SMA) embolization. We report a rare case of synchronous celiac axis and SMA embolization in an elderly woman with initially mild abdominal pain. Ultimately, a second contrast-enhanced computed tomography revealed extensive necrosis from the stomach to the transverse colon together with liver ischemia due to hours of occlusion. Multiorgan failure made palliation the only option, and she died the following evening. Autopsy revealed a fragile atherosclerosis-associated thrombus. Careful examination and repeat diagnostic tests should be performed in patients with mild abdominal symptoms at risk for AMI.

Key words: atherosclerosis, celiac axis, mesenteric ischemia, superior mesenteric artery, thromboembolism

Although acute mesenteric ischemia (AMI) only accounts for 2% of cases of gastrointestinal disease, it is a life-threatening surgical emergency associated with mortality rates as high as 40% to 70% [1]. Usually, most cases of AMI are caused by embolization of the superior mesenteric artery (SMA). In general, embolism occurs when a piece of a blood clot, foreign object, or other bodily substance occludes a blood vessel and obstructs the flow of blood. By contrast, thrombosis is when a thrombus, or blood clot, develops in a blood vessel and reduces the flow of blood through that vessel. Herein, we report a rare case of synchronous embolization of both the celiac axis (CA) and SMA caused by an atherosclerosis-associated thrombus.

Case Report

An 83-year-old Japanese woman who had been previously treated for atrial fibrillation (AF) and hypertension presented to a general practitioner with slight abdominal pain and vomiting. She was admitted to the facility, and intravenous treatment of rehydration therapy was performed. No remarkable findings were observed on laboratory tests or computed tomography (CT) except for atherosclerosis. Vital signs were as follows: body temperature, 35.8°C; blood pressure, 112/98 mmHg; pulse, 58 beats/min. Abdominal pain continued intermittently, always resolving with analgesics such as non-steroidal anti-inflammatory drugs. However, on the following day (18 h from onset), abdominal pain recurred with an increase in intensity. After a bout of hematemesis 2 h later, laboratory tests (Table 1) and CT scan findings revealed intestinal

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Table 1 Changes of laboratory date over time

		Practitioner	Practitioner	Our hospital
Time (hours)		0	20	22
Hematology				
WBC	(/μL)	5,500	22,000	17,000
RBC	($\times 10^6$/μL)	4.46	4.25	3.04
Hb	(g/dL)	14.6	13.9	9.9
PLT	($\times 10^3$/μL)	172	57	41
Coagulation				
PT	(%)	–	–	9.8
PT-INR		–	–	5.0
APTT	(sec)	–	–	92.0
D-dimer	(μg/mL)	–	–	405.1
FDP	(μg/mL)	–	–	870.8
Biochemistry				
T-Bil	(mg/dL)	1.0	2.2	1.7
AST	(U/L)	28	15,158	15,318
ALT	(U/L)	17	6,468	7,106
LDH	(U/L)	316	24,459	18,045
BUN	(mg/dL)	19.1	21.8	23.0
Cre	(mg/dL)	0.87	1.97	2.66
CPK	(U/L)	56	91	336
CRP	(mg/dL)	0.55	0.66	0.43
K	(mEq/L)	4.6	7.2	8.4
Blood gas analysis				
pH		–	–	7.027
Lactate	(mg/dL)	–	–	135.0

The Time shows the progress from presenting the practitioner.

necrosis. The patient became hemodynamically unstable and was immediately intubated and administered catecholamines prior to transfer to our hospital.

On admission to hospital, 22 h later from the onset, the vital signs were as follows: body temperature, 33.3°C; blood pressure, 70/33 mmHg; and pulse rate, 90 beats/min and irregular. Laboratory studies revealed an elevated white blood cell count (17,000/μL) and thrombocytopenia (41,000/μL). Aspartate transaminase, alanine aminotransferase, and lactate dehydrogenase levels were drastically elevated at 15,318 IU/L, 7,106 IU/L, and 18,045 IU/L, respectively. A decrease in prothrombin activity (9.8%) was observed. Arterial blood gas analysis revealed a pH of 7.027 and a lactic acid concentration of 135 mg/dL. Contrast-enhanced CT (CE-CT) showed poor contrast and pneumatosis of the digestive tract, extending from the stomach to the transverse colon and liver, and portal venous gas resulting from the total obstruction of the CA and SMA. The evidence of both sepsis and multiorgan failure due to extensive intestinal necrosis with hypothermia, acidosis, and coagulopathy halted all efforts for curative treatment, and palliative treatment was begun. The patient died the following evening. Figure 1 shows the passage of symptoms and CT imaging from the onset to admission in our hospital.

An autopsy was performed 2 h following her death with the consent of the bereaved family. Macroscopically, the GI tract from the stomach to the transverse colon, liver, and gallbladder were a dark red color (Fig. 2A). Histologically, the necrotic digestive tract showed severe submucosal edema, emphysematous change, and hemorrhage. The liver showed necrosis with lipid droplet deposition around the central vein. In the abdominal aorta, ulcers and an atherosclerosis-associated thrombus were confirmed upstream of the CA, and the CA and SMA were found to be totally occluded (~2 cm) by the thrombus (Fig. 2B, C). At the entrance

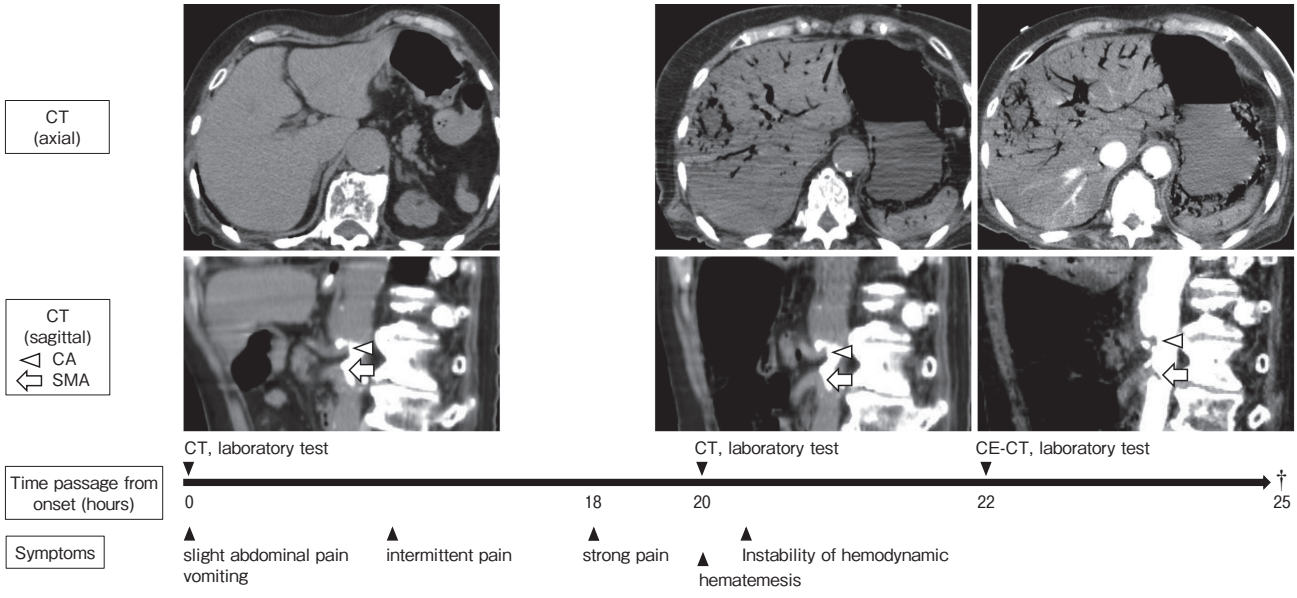

Fig. 1 The timeline of the patient's symptoms and corresponding computed tomography (CT) images. On the initial CT scan, atherosclerosis was observed at the entrance of the celiac axis and the superior mesenteric artery; however, no signs of intestinal necrosis were found. On subsequent CT scans, intestinal necrosis was observed, and blood flow could not be visualized at the CA and the SMA.

of the CA, thromboembolism was detected in addition to local thrombosis (Fig. 2D, E), and there was a trace of a broken thrombus upstream of the atherosclerosed portion (Fig. 2F). There was no thrombus formation in the atria despite the history of AF.

Discussion

AMI is a life-threatening surgical emergency and accounts for 2% of patients with gastrointestinal diseases and up to 10% of that in patients aged >70 years and causes acute abdominal pain [2]. Risk factors for AMI include heart failure, AF, coronary heart disease, arterial hypertension, and peripheral arterial occlusion [3]. Among patients with thrombosis of the SMA, 33% were found to have thrombosis or severe atherosclerotic changes of the CA. Furthermore, synchronous embolism of the CA was present in 10 out of 83 SMA-embolized patients [4]. There are a few reports of synchronous occlusion of the CA and SMA [5, 6]. In our case, results of the autopsy confirmed atherosclerotic changes upstream of the CA, along with minor thrombosis of the CA and SMA. A huge fragile thrombus, formed on the atherosclerotic plaque, was peeled off and trapped synchronously into the CA and SMA, causing extensive and fatal abdominal organ necrosis within just a few hours.

Clinically, the initial stage of AMI, which is characterized by a sudden onset of cramp-like abdominal pain, is followed by a deceptive pain-free interval after approximately 3 to 6 h, caused by a decline in intramural pain receptors as a result of sustained underperfusion of the intestinal wall. Subsequently, the mucosal barriers collapse with bacterial translocation and gangrene of the intestinal wall, with peritonitis resulting from bacterial infiltration, ileus, sepsis, and multiorgan failure [2]. In cases of CA occlusion alone, most patients remain asymptomatic owing to the gradual progression, as well as the vast collateral circulation from the SMA [6]. In our case, the patient's initial abdominal pain, which resolved with analgesics on her first admission, was possibly the initial sign that suggested AMI. Based on her initial symptoms, laboratory tests, and CT scan results, the necrotic change of the digestive tract had not started yet, although the thrombus might have been at the entrance of the CA and SMA. The CA and SMA were synchronously embolized, and extensive necrosis, including that of the stomach, occurred before collateral circulation could develop.

The prognosis of AMI is poor unless diagnosis and treatment are performed promptly. The mortality rate

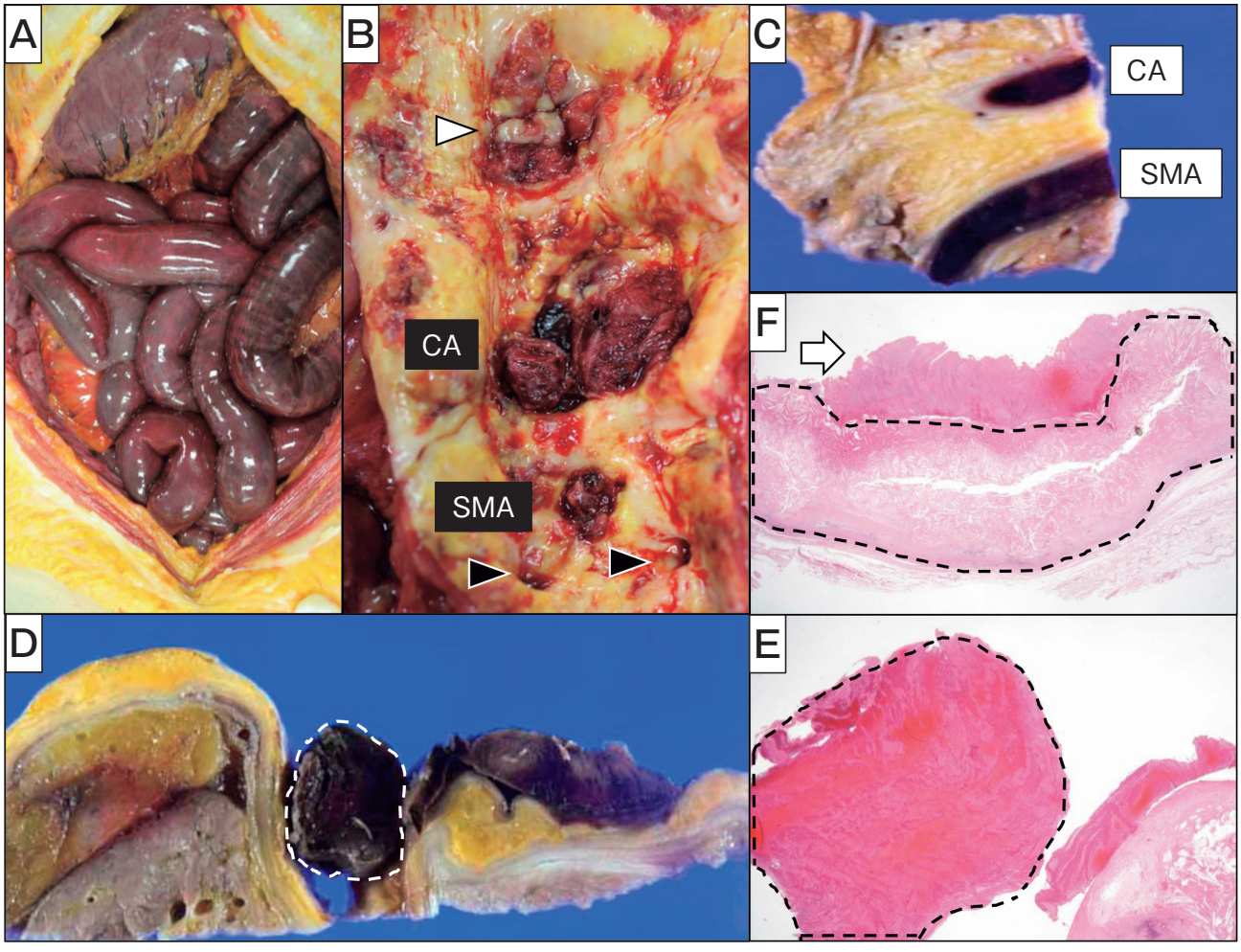

Fig. 2 Autopsy findings. (A) Extensive necrosis from the stomach to the transverse colon. (B) An ulcer and a thrombus (white arrowhead) caused by atherosclerosis, upstream of the celiac axis (CA). The CA and the superior mesenteric artery (SMA) were totally occluded by the thrombus; however, the renal arteries (black arrow heads) were intact. (C) In the sagittal section, approximately 2 cm of the CA and SMA were occluded by the thrombus. (D, E) Macroscopic and histological section of the CA. There was a covering thrombus (white and black dotted line, respectively) that was peeled off upstream. (F) There was a trace of a broken thrombus (white arrow) on the atheroma (black dotted line).

rises from 0% to 10% in those with swift treatment to 50% to 60% in those with treatment delays of 6 to 12 h and 80% to 100% in those with treatment delays of more than 24 h after symptom onset [7]. AMI results from arterial embolism in 50% of patients, arterial thrombosis in 20%, nonocclusive mesenteric ischemia in 20%, and venous thrombosis in 10% [8]. If AMI is suspected, biphasic CE-CT is the diagnostic tool of choice. The sensitivity and specificity of CE-CT are 93% and 100%, respectively [9]. Pneumatosis intestinalis, portal venous gas, bowel dilation, and massive ascites are the findings noted with respect to intestinal necrosis. Diagnosis and treatment within 6 to 12 h from the onset are life-saving; in our case, repeat laboratory tests within 6 h and early stage CE-CT should have been performed before hematemesis prompted such measures. Calcification and stenosis in the CA and SMA could have been revealed in more detail if CE-CT had been performed, and minor changes could have been detected earlier with more frequent laboratory tests. Furthermore, our patient had previously been treated for AF, and atherosclerosis had already been observed in the CA and SMA by CT. Thus, the possibility of AMI could have been suspected based on the symptoms of

mild, intermittent abdominal pain and vomiting.

Endovascular intervention can be performed for synchronous occlusion of the CA and SMA, with low rates of complication and mortality as long as the viability of the digestive tract is maintained [5]. More specifically, endovascular intervention includes the possibility of angiographically directed catheter-aspiration embolectomy and/or catheter lysis with recombinant tissue plasminogen activator, urokinase, or prostaglandin E1 [10]. Surgery is the only option for patients with intestinal infarction, acute peritonitis, or failure of endovascular intervention. It is difficult to recognize transmural bowel necrosis; therefore, even if the necrotic intestine could be resected at initial surgery, a second-look surgery should be considered to ensure resection of any nonviable intestine.

As mentioned above, severe atherosclerotic changes or embolism in the CA may coexist with occlusion in the SMA. Even in cases with mild abdominal symptoms, conducting repeat laboratory tests within 6 h and a CE-CT should be considered to exclude AMI in any patient with a high risk of AMI.

In conclusion, we investigated a rare case of synchronous total occlusion of the CA and SMA confirmed on autopsy. Such AMI cases have high mortality, thus, a high suspicion for the possibility should be present and repeat diagnostic tests ordered to allow for timely intervention.

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