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# The smaller superior mesenteric vein sign in acute superior mesenteric artery ischemia

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**Abstract:** An 87-year-old man had undergone a medical examination and was prescribed medicine to treat epigastralgia at a facility near our hospital one day before his arrival at our hospital. When his son checked on him the next day, he was unconscious. He was in a state of coma with shock and was therefore transported to our hospital by ambulance. Whole body computed tomography, which was performed to detect the focus of unstable circulation, revealed both pleural effusion (right dominant), ascites, small bowel distention with fluid level, intraluminal gas (pneumatosis cystoides intestinalis), and showed that the diameter of the superior mesenteric vein (SMV) was smaller than that of the superior mesenteric artery (SMA) (the smaller SMV sign), suggesting SMA ischemia and intestinal necrosis. As he had severe multiple organ failure due to septic shock, the surgeon decided not to perform surgical resection. The smaller SMV sign suggests intestinal hypo-perfusion and a decrease in blood flow in the SMA. For diagnosing acute superior mesenteric artery ischemia, the detection of smaller SMV on plain CT is useful for patients with severe condition. **Keywords:** acute superior mesenteric artery ischemia; smaller SMV sign; plain CT

#### INTRODUCTION

Acute superior mesenteric artery (SMA) occlusion is rare, and accounts for less than 1 of every 1000 hospitalizations. The associated mortality rate is reported to be up to 60–80%, and a delay in diagnosis can result in life-threatening consequences [1-3]. The smaller superior mesenteric vein (SMV) sign is a computed tomography (CT) parameter that is used to detect acute SMA occlusion [4, 5]. Although it is potentially beneficial for the early diagnosis of acute SMA occlusion, few reports have documented this sign. We herein report a case that demonstrates the importance of the smaller superior mesenteric vein sign in the diagnosis of acute superior mesenteric artery ischemia.

### **CASE REPORT**

An 87-year-old man had undergone a medical examination and was prescribed medicine to treat epigastralgia at a facility near our hospital one day before his arrival at our hospital. When his son checked on him the next day, he was unconscious. He was in a state of coma with shock and was therefore transported to our hospital by ambulance. He had a history of prostatic hypertrophy. Upon arrival, his vital signs were as follows: Glasgow Coma Scale, E1V1M1; blood pressure, 50/26 mmHg; pulse rate, 30 beats per minute; and his peripheral oxygen saturation on 10 liters of oxygen per minute with a bag-bulb mask, was not measurable. He underwent tracheal intubation. A

physiological examination revealed abdominal distension. Chest roentgenography revealed cloudiness in the right lung field. Electrocardiography showed junctional rhythm. An arterial gas analysis (FiO<sub>2</sub> 1.0) revealed the following: pH, 6.90; PCO<sub>2</sub>, 49 mmHg; PO<sub>2</sub>, 60 mmHg, HCO<sub>3</sub>, 9 mmol/l and base excess, -24 mmol/l. Whole body computed tomography, which was performed to detect the focus of unstable circulation, revealed both pleural effusion (right dominant), ascites, small bowel distention with fluid level, intraluminal gas (pneumatosis cystoides intestinalis), and showed that the diameter of the SMV was smaller than that of the SMA (the smaller SMV sign), suggesting SMA ischemia and intestinal necrosis. The results of the biochemical analyses of the blood on arrival were as follows: white blood cells, 8,300/µl; hemoglobin, 13.9 g/dl; platelets,  $9.5 \times 10^4$ /µl; aspartate aminotransferase, 157 IU/L; alanine aminotransferase, 79 IU/L; glucose, 23 mg/dl; blood urea nitrogen, 50.6 mg/dl; creatinine, 2.85 mg/dl; sodium, 145 mEq/L; potassium, 6.5 mEq/L; chloride, 115 mEq/L; creatine phosphokinase, 3010 IU/L; prothrombin time 19.6 (12.1) s; activated partial thromboplastin time, 40.7 (27.3) s; Fibrinogen, 233 mg/dl and fibrin degradation product, 96.9 µg/ml. As he had severe multiple organ failure due to septic shock, the surgeon decided not to perform surgical resection. No autopsy was performed as we the patient's family did not give their consent.

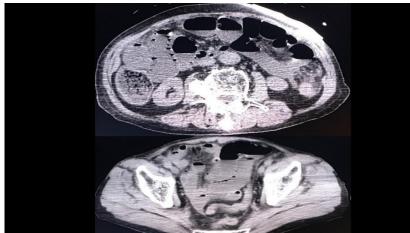
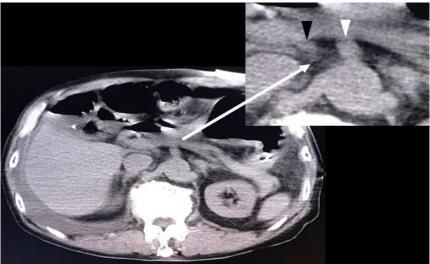


Fig 1: Abdominal CT on arrival

CT reveals ascites, small bowel distention with fluid level and intraluminal gas (pneumatosis cystoides intestinalis).



### Fig 2: Abdominal CT on arrival

CT revealed that the diameter of the SMV (black triangle) was smaller than that of the SMA (white triangle), suggesting SMA ischemia and intestinal necrosis.

#### DISCUSSION

The smaller SMV sign can be detected on plain CT images, without the use of a contrast agent: thus, the use of a contrast agent can be avoided. The smaller SMV sign suggests intestinal hypo-perfusion and a decrease in blood flow in the SMA, which are affected by low blood pressure, hormones, heart failure, arterial narrowing, catecholamine, and inflammation of the intestinal tract [5]. For diagnosing acute abdomen, the detection of smaller SMV on plain CT does not require the use of a contrast agent; thus smaller SMV can be quickly, safely, and reliably detected. Unfortunately, this patient died. However, had SMA ischemia been detected earlier based on the CT findings, including the detection of the smaller SMV sign, the patient might have received appropriate treatment at an earlier stage, leading to a favorable outcome.

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