CONSSERVATIVE MANAGEMENT OF SPONTANEOUS ISOLATED SUPERIOR MESENTERIC ARTERY DISSECTION: A CASE REPORT

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1 | INTRODUCTION

Spontaneous isolated superior mesenteric artery (SMA) dissection (SISMAD) is a rare but potentially fatal disease requiring emergency treatment for acute abdominal pain. SISMAD was first reported by Bauersfeld in 1947, and since the widespread use of computed tomography (CT), there have been increasing numbers of reports of SISMAD. However, the causes of this disease have not been elucidated, treatment strategies have not been established, and it is often difficult to determine the treatment method, even after diagnosis. Rapid diagnosis and treatment of SMA dissection lead to a reduced incidence and mortality of intestinal infarction. This case report highlights the use of conservative therapy based on a rapid diagnostic imaging evaluation provided for a SISMAD patient.

2 | CASE PRESENTATION

A 47-year-old Japanese man was referred to our hospital because of acute epigastric pain. His medical and family histories were unremarkable, he was not receiving any medical drugs, and he did not smoke tobacco or drink alcohol. The patient's normal systolic blood pressure was approximately 130 mmHg, and he had no history of abdominal trauma. He had been vaccinated against Coronavirus disease 2019 (COVID-19) with the Pfizer formulation 5 h prior to the onset of symptoms. On admission, his temperature was 37.9°C, with blood pressure of 173/104 mmHg and a regular heart rate of 98/min. Physical examination revealed deep tenderness in the lower abdomen. Laboratory data indicated leukocytosis (white blood cell count 18,460/μl) with a slightly elevated
creatine kinase isoenzyme level (446 U/L). Other data, including C-reactive protein, blood coagulation factor, and lactic acid levels, were within normal ranges (Table 1). A CT scan of the abdomen with contrast revealed an area, no contrast effect was observed in the pseudo-cavity with findings accompanied by true lumen stenosis. In the proximal SMA, beginning just after the bifurcation of the middle colic artery (MCA) and extending into the ileocolic artery for approximately 5 cm with no aneurysm formation, aortic dissection, or stenosis at the origin of the celiac artery (Figure 1A–C). From the above results, it was considered to be an SISMAD. Blood flow in the small intestine was maintained, probably due to the collateral circulation from the MCA (Figure 2), and no intestinal ischemia or ascites was observed. CT angiography was also considered for diagnosis but was not performed because the contrast-enhanced CT clearly showed no intestinal ischemia.

Although tenderness in the abdomen and inflammatory findings were observed by blood analysis, conservative treatment was selected first based on the imaging findings. The conservative treatment consisted of anticoagulation using heparin, bowel rest, and the control of pain and hypertension. He was monitored in the intensive care unit and his symptoms improved with the conservative measures. A follow-up CT after 7 days showed no exacerbation of SISMAD and the true lumen appeared slightly more open than in the initial CT images (Figure 3A–C). Blood pressure and blood examination confirmed improvements in inflammatory findings before resuming his diet (Figure 4). No obvious thrombotic findings were found on blood examination including fibrin degradation products (FDP) and CT; therefore, the antithrombotic drug was discontinued 7 days after admission. He had no problems during his subsequent hospitalization and was discharged 14 days after admission after confirmation by CT that there was no exacerbation. He returned to our hospital 2 weeks later—his symptoms were resolved, CT showed no exacerbation of SISMAD, and collateral circulation from the MCA showed persistent patency. He was followed up without the use of antithrombotic drugs, and no recurrence was observed 2 months after the onset. He has not received a second dose of coronavirus vaccine.

### 3 | DISCUSSION

Spontaneous isolated superior mesenteric artery dissection is considered a rare condition, and clinical presentations can range from an asymptomatic incidental finding to severe or fatal abdominal pain from bowel ischemia. Several causal factors of SISMAD have been postulated, including congenital connective tissue disorders, connective tissue disorders, and aortic dissection. In the present case, the patient had no history of connective tissue disorders, and the aortic dissection was ruled out by CT scan. The dissection occurred spontaneously in the SMA, and the true lumen appeared slightly more open than in the initial CT images. Blood pressure and blood examination confirmed improvements in inflammatory findings before resuming his diet. No obvious thrombotic findings were found on blood examination including fibrin degradation products (FDP) and CT; therefore, the antithrombotic drug was discontinued 7 days after admission. He had no problems during his subsequent hospitalization and was discharged 14 days after admission after confirmation by CT that there was no exacerbation. He returned to our hospital 2 weeks later—his symptoms were resolved, CT showed no exacerbation of SISMAD, and collateral circulation from the MCA showed persistent patency. He was followed up without the use of antithrombotic drugs, and no recurrence was observed 2 months after the onset. He has not received a second dose of coronavirus vaccine.

### Table 1 Laboratory test results upon hospital admission

| Variables         | Laboratory tests results |
|-------------------|--------------------------|
| WBC (×10^3/µl)    | 18.46                    |
| RBC (×10^6/µl)    | 5.01                     |
| Hemoglobin (g/dl) | 16.0                     |
| Hematocrit (%)    | 47.0                     |
| Platelet (×10^4/µl)| 26.5                     |
| Total protein (g/dl) | 7.1                 |
| Albumin (g/dl)    | 4.5                      |
| CRP (mg/dl)       | 0.64                     |
| BUN (mg/dl)       | 16.0                     |
| Creatinine (mg/dl)| 0.72                     |
| Na (mEq/L)        | 138                      |
| K (mEq/L)         | 4.2                      |
| Cl (mEq/L)        | 101                      |
| AST (IU/L)        | 27.0                     |
| ALT (IU/L)        | 24.0                     |
| ALP (IU/L)        | 199                      |
| LDH (IU/L)        | 192.0                    |
| Total bilirubin (mg/dl) | 1.8             |
| Direct bilirubin (mg/dl) | 0.3       |
| Amylase (IU/L)    | 107.0                    |
| CK (IU/L)         | 446.0                    |
| T-chol (mg/dl)    | 223.0                    |
| TG (mg/dl)        | 88.0                     |
| HDL-C (mg/dl)     | 47.0                     |
| LDL-C (mg/dl)     | 184.0                    |
| BS (mg/dl)        | 121.0                    |
| HbA1c (%)         | 5.9                      |
| PT (INR)          | 1.09                     |
| PT (%)            | 84.2                     |
| APTT (sec)        | 30.4                     |
| Fibrinogen (mg/dl)| 314.0                    |
| FDP (µg/ml)       | <2.5                     |
| FDP-DD (ng/ml)    | <0.5                     |
| PH                | 7.431                    |
| HCO3 (mmol/L)     | 25.1                     |
| Base Excess (mmol/L) | 1.5             |
| Lactate (mg/dl)   | 7.0                      |

Abbreviations: ALP, alkaline phosphatase; ALT, Alanine aminotransferase; APTT, activated partial thromboplastin time; AST, aspartate aminotransferase; BS, blood sugar; BUN, blood urea nitrogen; CK, Creatinine Kinase; CRP, C-reactive protein; FDP, fibrin degradation product; FDP-DD, fibrin degradation product D-dimer; HbA1c, Hemoglobin A1c; HCO3: bicarbonate; HDL-C, high-density lipoprotein cholesterol; LDLH, lactate dehydrogenase; LDL-C, low-density lipoprotein cholesterol; PH, power of hydrogen; PT, prothrombin time; PT-INR, prothrombin time-international normalized ratio; RBC, red blood cell; T-chol, total cholesterol; TG, triglycerides; WBC, white blood cell.
Fibromuscular dysplasia, trauma, and hypertension; however, the pathogenic mechanisms involved have yet to be fully elucidated.5

Although SISMAD is a rare disease and requires early diagnosis, it is possible to diagnose this disease only with contrast-enhanced CT, and catheter-based arteriography for diagnostic purposes may not be essential.5 In particular, multidetector CT enables the rapid and accurate evaluation of blood flow in the true lumen, patency, or thrombosis of the false lumen, and enlargement or reduction of the false lumen.6 Moreover, when checking CT images, several points such as the diameter and length of the site of arterial dissection, the patency of the false and true lumens, the entrance and reentry site of arterial dissection, the major collaterals of SMA, the mesenteric artery hematomas, signs of intestinal ischemia, and the presence of other simultaneous arterial dissections are important diagnostic points for treatment decision making.

Treatment approaches for SISMAD patients include open surgery, endovascular surgery, interventional radiology, and conservative management; however, clear guidelines for the treatment of SISMAD have not been established.5–8 Conservative medications often include antiplatelet and anticoagulant medications that reduce the blood flow due to stenosis of the true lumen, which prevents thrombus formation in small peripheral arteries, and antihypertensive therapy reduces hemodynamic stress in the blood vessel wall, preventing the progression of dissociation.5,8,10 However, this treatment has many

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**FIGURE 1** Initial imaging findings. Initial axial and sagittal imaging by enhanced abdominal computed tomography (CT) shows spontaneous isolated superior mesenteric artery (SMA) dissection (SISMAD) (A, B, red arrow) with extension into the ileocolic artery (C, red arrow)

**FIGURE 2** Three-dimensional CT angiogram. The arterial arcade from MCA (yellow arrows) and SISMAD (red arrow) are shown
problems including the risk of dilation of the dissection cavity due to antiplatelet therapy, which is related to the type of drug and the administration period. Several studies have reported no difference in the clinical course of SISMAD patients with and without adjuvant antithrombotic therapy. Therefore, further cases need to be accumulated for the evaluation of the usefulness of antithrombotic therapy.

Our patient was symptomatic and had high levels of inflammation, but fortunately the collateral circulation from the MCA was well developed and the blood flow in the small intestine was maintained; therefore, conservative treatment was selected. Anticoagulation using heparin was performed initially but discontinued after 1 week because there was no consistent increase in FDP and the symptoms had improved. Furthermore, CT findings 1 week after discontinuation showed no intestinal ischemia or obvious thrombotic findings and the antithrombotic treatment was left discontinued thereafter.
Coronavirus vaccination is urgently required because of the COVID-19 pandemic. However, the Japanese media have reported the sudden death of young people without underlying disease after coronavirus vaccination, although a causal relationship has not been demonstrated. Previous reports indicated that SISMAD commonly occurs in middle-aged men, as seen in our case. Although this has never been proven to be a side effect of the vaccine and is likely to be just a coincidence, our case has no underlying disease and SISMAD developed 5 h after coronavirus vaccination. Further investigation is needed on the side effects of the coronavirus vaccine, but we report as case experience that the possible side effects after coronavirus vaccination cannot be completely ruled out.

In conclusion, although the etiology and treatment information for SISMAD are rare and lacking, this case report could be accurately diagnosed by contrast-enhanced CT. Furthermore, it is a valuable case in which the treatment of SISMAD was successfully selected from conservative management based on the imaging findings. This case report provides a useful guide for the clinical diagnosis and treatment of this disease.

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CONFLICT OF INTEREST
The authors declare that they have no competing interests.

AUTHOR CONTRIBUTIONS
T Miyata and HT performed a central role in treating this patient. T Miyata drafted the manuscript. YS, TN, RK, HN, AH, YF, SM, DK, YT, NN, TM, HF and NU also managed the patient. All authors have approved the manuscript.

ETHICAL APPROVAL
The study design was approved by the ethics committee of the Kanazawa Medical University.

CONSENT
Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

DATA AVAILABILITY STATEMENT
Not applicable.

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