—Original—

Management of Asymptomatic Spontaneous Isolated Superior Mesenteric Artery Dissection and Morphology Features and Variations on Abdominal Contrast-Enhanced Computed Tomography: A Single-Center Experience

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Background: Spontaneous isolated visceral artery dissection (SIVAD) is rare. Recently, appropriate treatment strategies for symptomatic SIVAD have been proposed. We aimed to determine the management of asymptomatic spontaneous isolated superior mesenteric artery dissection (ASISMAD), which is relatively frequently encountered in SIVAD.

Methods: We retrospectively reviewed abdominal contrast-enhanced computed-tomography (CE-CT) scans from January 2015 to December 2020 in our institution and identified 24 patients with ASISMAD. Patient characteristics, vascular risk factors, complications, morphology features on CE-CT images, changes in abdominal CE-CT, and treatments outcomes were analyzed.

Results: All patients were male. The mean age of the patients was 66.0 ± 8.9 (standard deviation) years, and the follow-up period was 24.8 ± 28.7 months. The CE-CT images revealed that 1 patient had periarterial fat stranding, 15 patients had aneurysmal dilatation, and 7 patients had branch vessel involvement. The mean length of the dissection was 19.9 ± 13.5 mm. The mean distance from the orifice of the superior mesenteric artery to the dissection origin point was 14.9 ± 8.8 mm. The mean branching angle was 54.8° ± 19.7°. None of the patients had dissection-related abdominal symptoms or complications. Follow-up CE-CT scans showed progression of the dissection in 2 (8.3%), improvement in 2 (8.3%), stable dissection in 17 (70.9%), and complete remodeling in 3 (12.5%).

Conclusions: Patients with ASISMAD do not require hospitalization because the pathology does not usually progress to visceral ischemia. Nevertheless, follow-up CE-CT is required because of progression of the dissection in rare cases. (J Nippon Med Sch 2024; 91: 465–471)

Key words: superior mesenteric artery, arterial dissection, contrast-enhanced computed tomography, Yamaguchi classification

Introduction

Spontaneous isolated visceral artery dissection (SIVAD), which was first described by Bauersfeld[1] in 1947, is a rare condition, and only a few cases have been reported in the literature until 2006. Yamaguchi et al.[2] reported that SIVAD was observed in 0.68% of all abdominal contrast-enhanced computed tomography (CE-CT) scans taken for acute abdominal symptoms and in 0.05% of all abdominal CE-CT scans taken for other reasons. SIVAD cases are being reported more frequently in recent years[3–17] owing to technical advances in multidetector CT, improved CT resolution, and the increasing opportunities for investigating abdominal pain[3,11,18,19]. Presumed risk factors include atherosclerotic disease, hypertension, fibromuscular dysplasia, cystic medial necrosis, trauma, pregnancy, and connective tissue disorders[4,20]. Treatment options for SIVAD include non-operative, endovascular, and surgical interventions[6–13,15–17,21,22]. The optimal treatment strategy remains controversial, as its etiology and pathophysiological mechanisms are not firmly established. Recently, more

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https://doi.org/10.1272/jnms.JNMS.2024_91-511
Journal Website (https://www.nms.ac.jp/sh/jnms/)

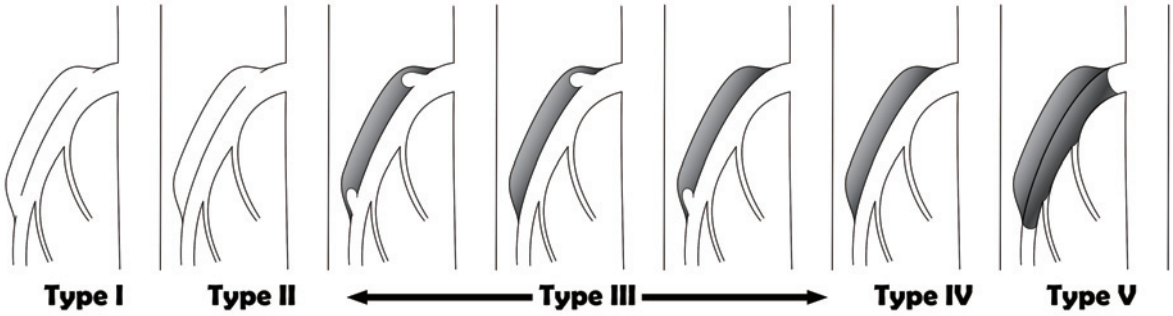


Fig. 1 Yamaguchi classification: type I, patent true and false lumens revealing entry and re-entry sites; type II, blind pouch of the false lumen; type III, partial thrombosis of the false lumen; type IV, complete thrombosis of the false lumen; type V, complete occlusion of the true and false lumens.

appropriate treatment strategies have been proposed for symptomatic SIVAD than for asymptomatic SIVAD[2,23]. Therefore, we aimed to determine the management of asymptomatic spontaneous isolated superior mesenteric artery dissection (ASISMAD), which is relatively frequent in SIVAD, and to investigate its morphological features and variations in abdominal CE-CT images.

Materials and Methods

This retrospective study was approved by the Ethics Committee of Nippon Medical School Tama Nagayama Hospital (no. 493), and the requirement for informed consent was waived.

We investigated 20,419 abdominal CE-CT scan reports performed at our institution between January 2015 and December 2020. CE-CT was performed using a 64-multidetector-row CT system (Lightspeed VCT; GE Healthcare, Milwaukee, WI, USA) or a 64-multidetector-row CT system (Scenaria; Hitachi, Kashiwa, Japan). CT technical parameters included the following: 512 × 512 matrix, 5-mm slice, 150-600 mA (standard deviation [SD], 10 [GE Healthcare]; SD, 8-10 [Hitachi]), and 120 kV. A picture archiving and communication systems database search for keywords ("superior mesenteric artery," "arterial dissection," "dissecting aneurysm") was performed to extract reports of patients with SIVAD. Patients with concomitant symptomatic (absence of explanations other than dissection-related ones) SIVAD, aortic dissection, trauma, or iatrogenic causes were excluded[4–7,16,17].

Reports of asymptomatic patients with SIVAD (incidental findings) were screened for ASISMAD. Patient characteristics, vascular risk factors, medical histories, follow-up durations, changes in symptoms, and treatment courses were analyzed using the patient's charts. The morphological features on CE-CT images were intimal flap, thrombosed false lumen, and aneurysmal dilatation (≥1.5 times the normal diameter of perilesional arteries), including branch vessel involvement and periarterial fat stranding. We classified each dissection according to the Yamaguchi classification, which has five types based on findings on CE-CT images (**Fig. 1**): type I, patent true and false lumens revealing entry and re-entry sites; type II, blind pouch of the false lumen; type III, partial thrombosis of the false lumen; type IV, complete thrombosis of the false lumen; type V, complete occlusion of the true and false lumens. We then tracked the morphological changes in CE-CT images.

In cases with constriction of the true lumen, reports of patients on antiplatelet therapy (daily oral administration of 75 mg clopidogrel, 200 mg cilostazol, or 100 mg aspirin, alone or in combination) for 6 months and/or prostaglandin E1 (daily oral administration of 120 μg) were examined. Patients who were taking antihypertensives to maintain the systolic blood pressure below 140 mm Hg[3,5–8,11,22,23] were also examined.

SPSS version 24.0 (IBM Corp., Armonk, NY, USA) was used for the statistical analyses, and mean values were used in each evaluation. The Kruskal-Wallis test was to compare among the Yamaguchi types. Differences were considered significant at $P < 0.05$.

Results

Twenty-four patients (all men) (mean ages ± SD, 66.0 ± 8.9 years; range, 45-88 years) were diagnosed with ASISMAD. Patient characteristics are summarized in **Table 1**. As the cases were incidental findings without acute abdominal symptoms, hospitalization and invasive treatments were delayed. The vascular risk factors were as follows: hypertension (n = 12, 50.0%), hyperlipidemia (n = 6, 25.0%), diabetes mellitus (n = 5, 20.8%), smoking (n = 14, 58.3%), Ehlers-Danlos syndrome (n = 0, 0.0%), and segmental arterial mediolysis (n = 0, 0.0%). Their medical

history included malignant disease (n = 18, 75.0%), aortic aneurysm (n = 2, 8.3%), arteriosclerosis obliterans (n = 2, 8.3%), myocardial infarction that underwent endovascular therapy (n = 1, 4.2%), cerebral infarction (n = 1, 4.2%), atrial fibrillation (n = 1, 4.2%), gastrointestinal hemorrhage (n = 1, 4.2%), and chronic pancreatitis (n = 1, 4.2%).

Table 1 Patient characteristics

Gender (male:female)	n = 24:0
Age (mean ± SD)	66.0 ± 8.9 years old
Risk factors (%)	
Hypertension	n = 12 (50.0%)
Hyperlipidemia	n = 6 (25.0%)
Diabetes mellitus	n = 5 (20.8%)
Smoking	n = 14 (58.3%)
Medical histories (%)	
Malignant disease	n = 18 (85.0%)
Aortic aneurysm	n = 2 (8.3%)
Arteriosclerosis obliterans	n = 2 (8.3%)
Myocardial infarction	n = 1 (4.2%)
Cerebral infarction	n = 1 (4.2%)
Atrial fibrillation	n = 1 (4.2%)
Gastrointestinal hemorrhage	n = 1 (4.2%)
Chronic pancreatitis	n = 1 (4.2%)

The morphological features observed in CE-CT images are summarized in **Table 2**. Intimal flap (n = 22, 91.7%), thrombosed false lumen (n = 5, 20.8%), and aneurysmal dilatation (n = 15, 62.5%) were found. Other findings included branch vessel involvement (n = 7, 29.2%) and periarterial fat stranding (n = 1, 4.2%). The mean distance from the orifice of the superior mesenteric artery (SMA) to the dissection origin point was 14.9 ± 8.8 mm (range, 3.0-33.0 mm). The mean length of the dissecting SMA was 19.9 ± 13.5 mm (range, 7.0-43.7 mm). The mean branching angle from the aorta was 54.8° ± 19.7° (range, 26.4°-89.3°). The Yamaguchi classifications based on CE-CT images were as follows: type I (n = 10, 41.7%), type II (n = 9, 37.5%), type III (n = 3, 12.5%), type IV (n = 2, 8.3%), and type V (n = 0, 0.0%).

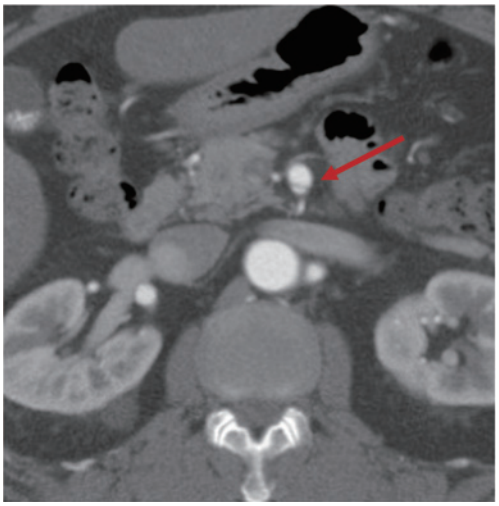
Ambulatory follow-up included assessment of abdomi-

Table 2 Morphology of the dissection on contrast-enhanced computed tomography images

No	Intimal flap	Thrombosed false lumen	Aneurysmal dilatation	Branch vessel involvement	Periarterial fat stranding	Distance from the orifice of the SMA to the dissection	Length of the dissecting SMA	Branching angle from the aorta	Yamaguchi classification
1	+	−	+	−	−	6	8	71.5	I
2	+	+	+	−	+	3	12	36.5	III
3	−	+	+	−	−	4	11	40.8	IV
4	−	+	−	−	−	19	7	89.3	IV
5	+	−	+	−	−	9	9	78.8	I
6	+	+	+	−	−	4	15	47.8	III
7	+	−	+	−	−	20	10	80	I
8	+	−	−	−	−	10	9	82.3	II
9	+	−	+	−	−	18	14	28.8	II
10	+	−	−	−	−	8.2	7.8	27.4	I
11	+	−	+	−	−	9.3	14	26.4	I
12	+	−	+	−	−	4.2	19	51	I
13	+	−	+	+	−	29	25	61.7	I
14	+	−	+	+	−	18	33	60.1	II
15	+	−	+	−	−	28	19	35	I
16	+	+	−	−	−	33	19	30.2	III
17	+	−	+	+	−	12	43.7	36.2	II
18	+	−	+	+	−	13.3	68	61.6	I
19	+	−	−	−	−	17.8	19.7	77.3	II
20	+	−	−	+	−	28.5	27.5	51.7	I
21	+	−	−	−	−	8	23.5	42.8	II
22	+	−	−	−	−	19	17.1	69	II
23	+	−	−	+	−	13.5	23.9	57	II
24	+	−	+	+	−	21.98	22.62	71.9	II
	n = 22	n = 5	n = 15	n = 7	n = 1	14.9 ± 8.8 (SD) mm	19.9 ± 13.5 mm	54.8 ± 19.7°	

SMA, superior mesenteric artery

(a)



(b)

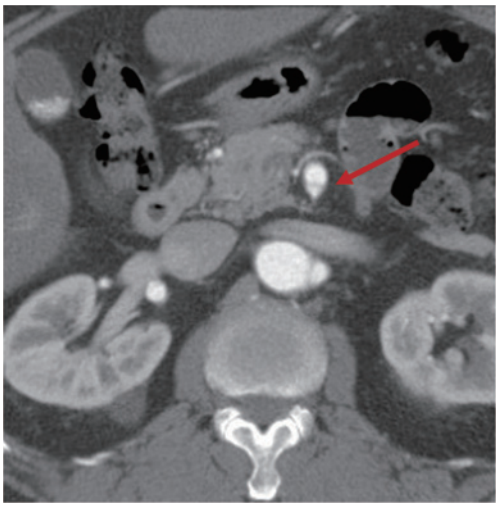


Fig. 2 Type II superior mesenteric artery dissection (arrow) incidentally found preoperatively in a 45-year-old man with colorectal cancer. (a) Contrast-enhanced computed tomography at diagnosis revealed a blind pouch of the false lumen, and the false lumen showed aneurysmal dilatation. (b) Six months after the diagnosis, the blind pouch of false lumen has dilated further.

nal symptoms and morphological changes in CE-CT images. The mean follow-up period was 24.8 ± 28.7 months (range, 2-71 months). No abdominal symptoms, organ ischemia, or dissection-related complications were observed. Follow-up CE-CT scans showed slow progression of the dissection in 2 patients (8.3%, types I and II) (**Fig. 2**), improvement in 2 patients (8.3%, type III), stable dissection in 17 patients (70.9%, types I and II), and complete remodeling in 3 patients (12.5%, types III and IV) (**Fig. 3**). The average complete remodeling time was 8.3 ± 1.5 months (range, 6-10 months). The periarterial fat stranding disappeared completely. Of the 8 patients with constriction in the true lumen, antiplatelet and/or prostaglandin E were administered to 5 patients. Antihypertensives were administered to all 12 patients with hypertension. None of the patients died of ASISMAD, excluding 4 patients with malignancy.

Discussion

Spontaneous isolated artery dissection has been reported frequently in the carotid and renal arteries but rarely in the visceral arteries, such as the celiac or hepatic artery[2–17,22,24]. Recent studies have advocated treatment strategies for symptomatic SIVAD[2,23]. Therefore, we determined the management of ASISMAD, which is relatively frequent in SIVAD, and investigated its morphological changes on abdominal CE-CT. This retrospective study examined a large patient population from a single institution and their clinical follow-up and treatment strategies.

Previous studies have documented that 91.3% of patients with SIVAD were men with an average age of 50.0 years[9]. This finding is nearly identical to that of the present study. Yamaguchi et al.[2] reported that SIVAD was observed in 0.09% of all abdominal CE-CT scans, and Foord and Lewis[20]. reported a 0.06% incidence postmortem. These findings suggest that SIVAD is rare, and consensus regarding its pathology and optimal therapy is lacking. Its pathogenesis is unknown, except for Ehlers-Danlos syndrome and segmental arterial mediolysis. Some investigators have associated SIVAD with hypertension, atherosclerosis, cystic medial necrosis, fibromuscular dysplasia, and connective tissue disease[2,4,20]. Hypertension may be a predisposing factor; however, no data support its role in causing intimal tears. Hypertension and smoking were common and carried higher risk than other suspected risk factors; 83.3% of patients reported at least one of these factors. In cases where the celiac artery is stenotic or occluded by arteriosclerosis and the median arcuate ligament, a compensatory increase of flow in the SMA may lead to increased shear stress[3,11], which may be a possible mechanism for dissection. However, celiac artery stenosis of the median arcuate ligament was found in only one case and arteriosclerosis in two cases. Dissection occurred despite the absence of obvious arterial diseases in 87.5% of patients. Anatomical weakness can be considered a cause of this. Anatomically, 10.0-30.0 mm from the orifice of the SMA between the fixed retropancreatic portion and mobile portion is a weak point[12]. The mean distance from the orifice of the SMA to the origin of the dissection (14.8 ± 9.9 mm) is concordant with this

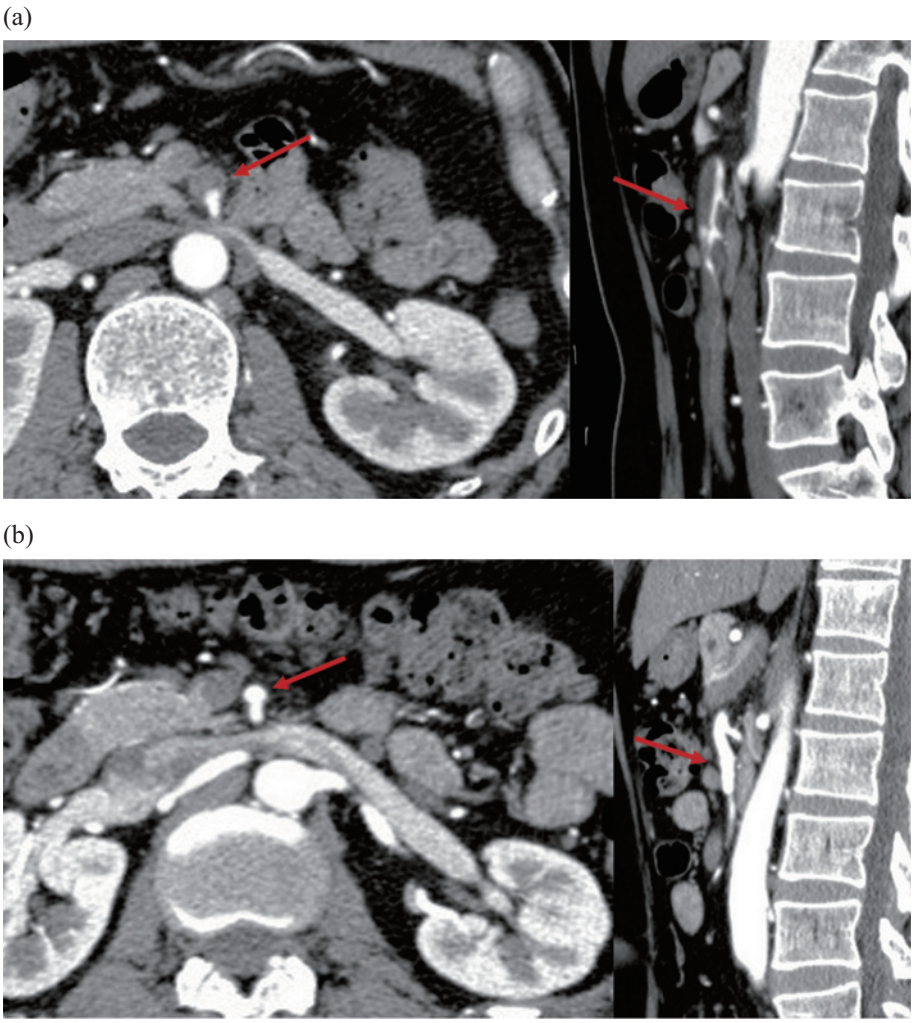


Fig. 3 Type III superior mesenteric artery dissection (arrow) incidentally found post-surgery in a 59-year-old man with colorectal cancer. (a) Contrast-enhanced computed tomography at diagnosis revealed a partially thrombosed false lumen and the true lumen narrowed and retracted by the false lumen. (b) Six months after diagnosis, the partially thrombosed false lumen and dissection were completely remodeled.

weak point. These results suggest a direct involvement of anatomical weakness and indirect involvement of increased shear stress and pressure in the pathogenesis. Most patients with ASISMAD had a history of malignancy. The malignancy associated cytokines released and the treatments for the malignancy (surgery, radiotherapy, and chemotherapy) may play a role in the development of SIVAD. However, no statistically significant difference in the mean length of the dissecting SMA ($P > 0.05$) or in the mean branching angle from the aorta ($P > 0.05$) was found between each classification.

Periarterial fat stranding may predict symptom severity. Periarterial fat stranding reflects inflammation that stimulates the visceral nerve plexus and causes symptoms. It is speculated that the patient with periarterial fat stranding did not present with symptoms because of diabetes mellitus.

The classification by Sakamoto et al.[3] did not include a complete thrombosis lumen, and that by Yun et al.[7] did not include partial thrombosis of the false lumen. Therefore, we used the Yamaguchi classification[2]. We found a tendency for the following morphological changes in this classification: progression of dissection in types I and II, improvement in type III, stable dissection in types I and II, and complete remodeling in types III and IV. No complete predictors of disease progression were identified. However, types I and II had the possibility of progression of dissection, albeit rare. Types III and IV were more likely to remodel and demonstrate complete resolution. In our study, type V dissection could not be confirmed and would probably cause acute abdominal symptoms.

The optimal treatment has not been established and

may involve observation with or without drugs, surgery, and endovascular intervention, depending on clinical features. Endovascular interventions are preferred for patients with persistent symptoms and those who develop ischemia or necrosis, as the outcomes are comparable to those with surgical interventions[2,3,5–9,11–16,22,25,26]. These results suggest that patients with ASISMAD do not require hospitalization because there are no fatal complications. To prevent thromboembolic complications, some studies have suggested the administration of anticoagulant or antiplatelet for 3-6 months with a target international normalized ratio of 2.0-3.0, as slowing the progression of a false lumen by blood pressure control may decrease hemodynamic turbulence[4,6–9,24,27]. This argument for anticoagulation is based on reports published before 1970, in which non-operative intervention without anticoagulation led to necrosis. However, Yun et al.[7] reported that the absence of anticoagulation or antiplatelet therapy does not affect clinical outcomes. Thus, there is no consensus regarding drug use. In general, anticoagulants, antiplatelets, or prostaglandins are prescribed for narrowing of the true lumen, whereas antihypertensives are prescribed for hypertension. However, it is impossible to completely prevent the progression of dissection using these drugs. We observed slow progression in 2 patients taking antihypertensives (8.3%), which is similar to the 5.2%[5] overall failure rate in spontaneous SMA dissection. These 2 slow-progressing patients were classified as types I and II, respectively. Progression was suspected to be approximately several millimeters in 1 year, as observed in CE-CT images, and follow-up is still underway. However, in a small number of cases, it was speculated that spontaneous SMA dissection without thrombosis (types I and II) may be at risk of progression.

This study has some limitations. First, we were unable to obtain a prior history of acute abdominal pain from the patients with ASISMAD. Second, no pathological evidence was obtained. Third, this retrospective study was confined to a particular ethnic group. Treatment outcomes should be further examined in prospective studies.

In conclusion, patients with ASISMAD do not require hospitalization because the condition does not usually progress to visceral ischemia or necrosis. Dissection in most patients with ASISMAD tends to remain stable or improve. However, follow-up CE-CT is required on account of the slow progression of dissection, albeit rare, especially in type I and II dissections.

Conflict of Interest: The authors declare that they have no conflict of interest.

References

1. Bauersfeld SR. Dissecting aneurysm of the aorta; a presentation of 15 cases and a review of the recent literature. Ann Intern Med. 1947;26:873–89.
2. Yamaguchi H, Murata S, Onozawa S, Sugihara F, Hayashi H, Kumita SI. Strategy for the treatment of spontaneous isolated visceral artery dissection. Eur J Radiol Open. 2018;6:9–15.
3. Sakamoto I, Ogawa Y, Sueyoshi E, Fukui K, Murakami T, Uetani M. Imaging appearances and management of isolated spontaneous dissection of the superior mesenteric artery. Eur J Radiol. 2007;64:103–10.
4. D'Ambrosio N, Friedman B, Siegel D, Katz D, Newatia A, Hines J. Spontaneous isolated dissection of the celiac artery: CT findings in adults. AJR Am J Roentgenol. 2007; 188:W506–11.
5. Takayama T, Miyata T, Shirakawa M, Nagawa H. Isolated spontaneous dissection of the splanchnic arteries. J Vasc Surg. 2008;48:329–33.
6. Gobble RM, Brill ER, Rockman CB, et al. Endovascular treatment of spontaneous dissections of the superior mesenteric artery. J Vasc Surg. 2009;50:1326–32.
7. Yun WS, Kim YW, Park KB, et al. Clinical and angiographic follow-up of spontaneous isolated superior mesenteric artery dissection. Eur J Vasc Endovasc Surg. 2009;37: 572–7.
8. Min SI, Yoon KC, Min SK, et al. Current strategy for the treatment of symptomatic spontaneous isolated dissection of superior mesenteric artery. J Vasc Surg. 2011;54:461–6.
9. Park YJ, Park KB, Kim DI, Do YS, Kim DK, Kim YW. Natural history of spontaneous isolated superior mesenteric artery dissection derived from follow-up after conservative treatment. J Vasc Surg. 2011;54:1727–33.
10. Jibiki M, Inoue Y, Kudo T. Conservative treatment for isolated superior mesenteric artery dissection. Surg Today. 2013;43:260–3.
11. Jung SC, Lee W, Park EA, Jae HJ, Chung JW, Park JH. Spontaneous dissection of the splanchnic arteries: CT findings, treatment, and outcome. AJR Am J Roentgenol. 2013;200:219–25.
12. Pang P, Jiang Z, Huang M, Zhou B, Zhu K, Shan H. Value of endovascular stent placement for symptomatic spontaneous isolated superior mesenteric artery dissection. Eur J Radiol. 2013;82:490–6.
13. Perini P, Baque J, Chau Y, Sedat J, Batt M. Percutaneous embolization of symptomatic dissecting aneurysms of the celiac artery. Acta Radiol. 2014;55:1076–81.
14. Park UJ, Kim HT, Cho WH, Kim YH, Miyata T. Clinical course and angiographic changes of spontaneous isolated superior mesenteric artery dissection after conservative treatment. Surg Today. 2014;44:2092–7.
15. Satokawa H, Takase S, Seto Y, et al. Management strategy of isolated spontaneous dissection of the superior mesenteric artery. Ann Vasc Dis. 2014;7:232–8.
16. Li DL, He YY, Alkalei AM, et al. Management strategy for spontaneous isolated dissection of the superior mesenteric artery based on morphologic classification. J Vasc Surg. 2014;59:165–72.
17. Alcantara S, Yang CK, Sasson J, et al. The evidence for nonoperative management of visceral artery dissections: a single-center experience. Ann Vasc Surg. 2015;29:103–8.
18. Furukawa H, Moriyama N. Spontaneous dissection of the superior mesenteric artery diagnosed on multidetector

helical CT. J Comput Assist Tomogr. 2002;26:143–4.
19. Luan JY, Li X. Computed tomography imaging features and classification of isolated dissection of the superior mesenteric artery. Eur J Vasc Endovasc Surg. 2013;46:232–5.
20. Foord AG, Lewis RD. Primary dissecting aneurysms of peripheral and pulmonary arteries: dissecting hemorrhage of media. Arch Pathol. 1959;68:553–77.
21. Takeda H, Matsunaga N, Sakamoto I, Obata S, Nakamura S, Hayashi K. Spontaneous dissection of the celiac and hepatic arteries treated by transcatheter embolization. AJR Am J Roentgenol. 1995;165:1288–9.
22. Wagenhauser MU, Sagban TA, Witte M, Duran M, Schelzig H, Oberhuber A. Isolated dissection of the superior mesenteric artery treated using open emergency surgery. World J Emerg Surg. 2014;9:47.
23. Sosogi S, Sato R, Wada R, et al. Clinical course of conservative management for isolated superior mesenteric arterial dissection. Eur J Radiol Open. 2019;6:192–7.
24. Fenoglio L, Allione A, Scalabrino E, et al. Spontaneous dissection of the celiac artery: a pitfall in the diagnosis of acute abdominal pain. Presentation of two cases. Dig Dis Sci. 2004;49:1223–7.
25. Froment P, Alerci M, Vandoni RE, Bogen M, Gertsch P, Galeazzi G. Stenting of a spontaneous dissection of the superior mesenteric artery: a new therapeutic approach? Cardiovasc Intervent Radiol. 2004;27:529–32.
26. Takach TJ, Madjarov JM, Holleman JH, Robicsek F, Roush TS. Spontaneous splanchnic dissection: application and timing of therapeutic options. J Vasc Surg. 2009;50:557–63.
27. Schievink WI. Spontaneous dissection of the carotid and vertebral arteries. N Engl J Med. 2001;344:898–906.

(Received, January 15, 2024)
(Accepted, July 12, 2024)

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