

Original Article

Modified morphological CT classification to screen requirement of endovascular or surgery treatment for spontaneous isolated superior mesenteric artery dissection

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Received November 15, 2016; Accepted February 6, 2017; Epub April 15, 2017; Published April 30, 2017

Abstract: This study was aimed to investigate the effects of modified CT classification of spontaneous isolated superior mesenteric artery dissection (SISMAD) for screening requirement of endovascular or surgery treatment once 5-7 days conservative therapy fails. A total of 37 CT angiography diagnosed SISMAD patients were reviewed from Jan. 2008 to Sep. 2015, and classified into symptomatic (n = 22) and asymptomatic (n = 15) groups. Initial and 5-7th days' CT angiography characteristics and clinical data were collected. Then, we performed a modified CT classification to guide treatment algorithm, and a series of follow-up were conducted to assess the outcomes. Symptomatic patients (SPs) showed serious abdominal pain with 68.2% severe complications, whereas asymptomatic patients (ASPs) complained hidden pain or abdominal distension with 13.3% complications. Comparing to ASPs, SPs have a higher incidence of periarterial fatty infiltration (PFI), bowel ischemia (BI), intestinal obstruction, aneurysmal dilatation (AD), thrombosed false lumen, true lumen stenosis, and branch involvement (BIM). Furthermore, 72.7% (16/22) of SPs and 26.7% (4/15) of ASPs presented as Type III-V. During 5-7 days' conservative treatment, no difference ($\chi^2 = 0.004$, $P = 0.947$) was observed in ischemia progress between SPs and ASPs. Only one patient received emergency surgery, and 14 patients and 22 cases underwent endovascular intervention and continued conservative management, respectively. Two patients treated conservatively and one patient with stenting presented a progress during the follow-up. Collectively, both the initial and 5-7 days' CT morphology assessments are beneficial for choosing SISMAD therapeutic strategy.

Keywords: Superior mesenteric artery, artery dissection, computed tomography angiography, therapy, spontaneous isolated superior mesenteric artery dissection

Introduction

Spontaneous isolated superior mesenteric artery dissection (SISMAD) is a rare etiology of acute mesenteric ischemia with few reports in PubMed database [1-4]. Most of the reported cases are from East Asia [5-11]. In recent years, studies of SISMAD have increased due to the improvement of technology and widespread use of multi-slice computed tomography (MS-CT) for acute abdominal pain. Sakamoto et al. [2] firstly classified SISMAD into four types based on the patency of false lumen, and different classifications have been updated on CT angiography (CTA) to demonstrate this vessel disease for guiding a favorable clinical interven-

tion. Referring to the CT classification and clinical features, different therapeutic options to manage SISMAD have been reported, including conservative therapy, endovascular and surgical intervention [12-14]. However, there is no consensus on CT classification for demonstration of superior mesenteric artery (SMA) stenosis and its natural history, mainly because of the existence and likelihood of stenosis of true lumen of SMA [15]. In addition, there is no strong guidance on deciding the optimal time of endovascular treatment (ET) for symptomatic patients [16, 17].

The aim of this retrospective study is to report a modified classification, 5-7th days' transfor-

CT classification to screen treatment for SMA dissection

Table 1. Demographic and baseline clinical characteristics (n = 37)

Characteristic	Symptomatic patients (n = 22)	Atypical symptoms patients (n = 15)	Total (n = 37)
Age (range)	51.6 (22-81)	53.7 (21-70)	52.5 (21-81)
Gender (male %)	90.9% (20/22)	100% (15/15)	94.6% (35/37)
Risks			
Hypertension (%)	54.5% (12/22)	40% (6/15)	48.6% (18/37)
Dyslipidemia	40.9% (9/22)	26.7% (4/15)	35.1% (13/37)
Diabetes	9.1% (2/22)	20.0% (3/15)	13.5% (5/37)
Alcohol	18.2% (4/22)	26.7% (4/15)	21.6% (8/37)
Smoking	59.1% (13/22)	60.0% (9/15)	59.5% (22/37)
Aortic sclerosis	54.5% (12/22)	40.0% (6/15)	48.6% (18/37)
Clinical manifestation			
Abdominal pain	Severe (22)	Mild (8); None (7)	Severe (22); Mild (8); None (7)
Location	Mid abdomen or diffused	Mid upper abdomen	
Model		Dull or discomfort	-
Associated symptoms	Sharp (100%)	13.3% (2/15)	-
Nausea	68.2% (15/22) ^a	0	45.9% (17/37)
Distension	9	2	9
Vomiting	7	0	9
Diarrhea	7	0	7
Bloody stools	5	0	5
Laboratory parameters	3		3
WBC (10 ⁹ /L)		9.6 (6.8-14.7)	
CRP (positive)	12.3 (6.9-19.5) ^b	4	11.2 (6.8-19.5)
D-dimer (positive)	9 ^c	3	13
	9 ^d		12

WBC, white blood cell count; CRP, C-reactive protein (Negative: < 10 mg/L); D-dimer (Negative: < 0.55 mg/L). a, Comparison of incidence of associated symptoms between symptomatic patients and atypical symptoms patients, $P = .007$; b, Comparison of white blood cell count, $P = 0.01$; c, Comparison of positive rate of C-RP, $P = 0.491$; d, Comparison of positive rate of D-dimer, $P = 0.286$.

mation of CT features and clinical data of 37 continually registered patients with SISMAD, and to explore whether this classification can screen out the requirement for ET or surgical treatment.

Materials and methods

Ethics statement

Institutional Review Board of our hospitals has been approved this retrospective study, and the requirement for patients' informed consent has been waived.

Patients

Thirty-seven patients diagnosed with SISMAD using CTA and consecutively registered from January 2008 to September 2015, were selected from the database of MSCT scanners.

Patients with another artery, such as aortic dissection with splanchnic branches involvement or any other visceral arterial dissection were excluded. All SISMAD patients were described according to our classification scheme (**Table 1**) and were divided into symptomatic group and atypical symptom group. Symptomatic group had a severe onset of abdominal pain, which could be accompanied by nausea, vomiting or diarrhea, while atypical symptom patients often complained hidden pain or abdominal discomfort. All patients underwent dual-phase abdomen CTA prior to admission. Routine non-enhanced CT scan was performed in 18 patients before CTA. However, the abdominal symptoms of these subjects could not be explained by these non-enhanced images. All patients received a re-examination by either CTA or DSA when clinical evaluation showed progression after 5-7 days of conservative treatment.

CT classification to screen treatment for SMA dissection

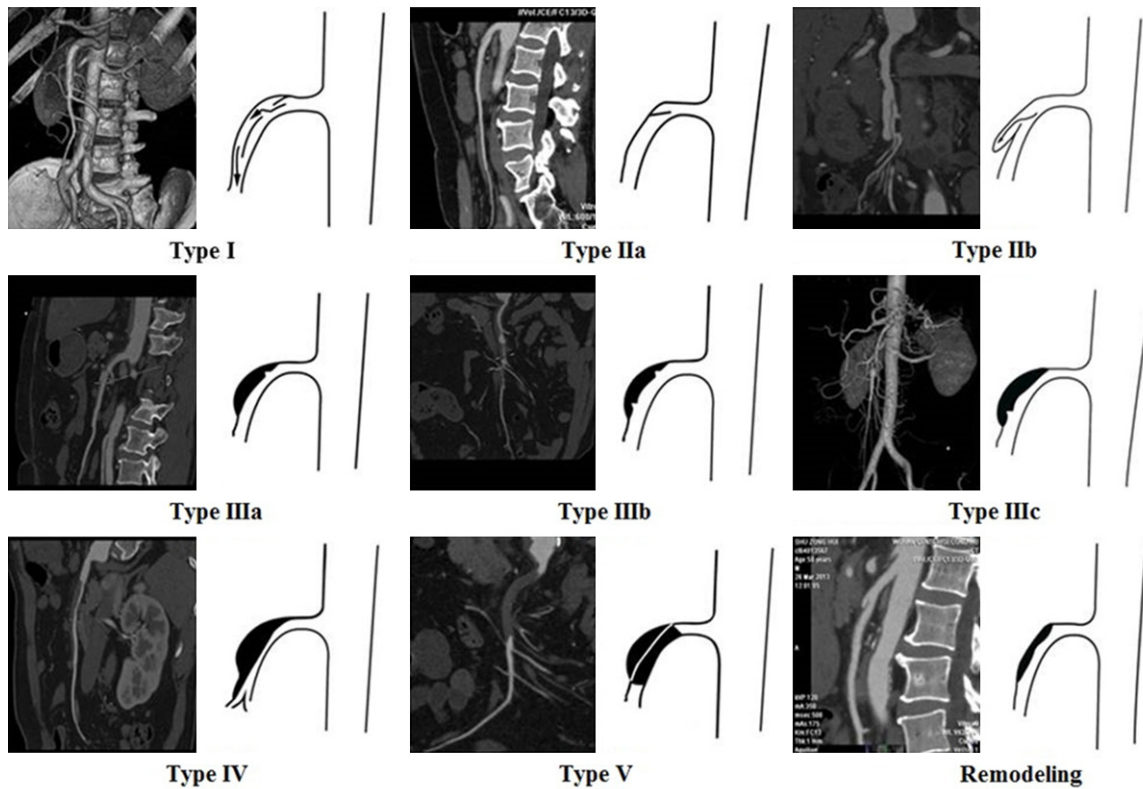


Figure 1. SISMAD classification based on imaging characteristics.

CT protocols and interpretation

All patients underwent contrast-enhanced helical CT (Aquilion 64, Toshiba, Japan or Somatom Definition Flash, Siemens, German). Scanned scope ranged from diaphragm to the pubic symphysis, and the main scan parameters were as follows: detector collimation, 64×0.5 mm or 64×0.6 mm; voltage, 100 kV or 120 kV; pitch, 0.83 or 1.20; effective tube current, 150-165 mAs or 142-250 mAs; filter convolution algorithm, FC43 or B26f. Automatic bolus tracking technique was applied in our standard dual-phase CT examination with administration of 1 ml/kg body weight of non-ionic iodinated contrast media (350 mg/ml) in total at a rate of 4-5 ml/s. Arterial phase dataset was established to determine SISMAD; dual-phase CT was used to identify ischemia or congestion of intestine.

SISMAD was diagnosed by one of the following signs: intimal flap or a crescent-shaped area or both on CTA. CT features were recorded, including periarterial fatty infiltration, intestinal ischemia (edema of bowel wall or mesentery, obstruction), intimal flap, aneurysmal dilatation,

thrombosed false lumen, true lumen stenosis, branch involvement and Rioloan arch. Aneurysmal dilatation was defined as an increase greater than 50% in the diameter relative to the normal diameter of the SMA.

Our CT classification system for SISMAD (**Figure 1**) was based on the previous report proposed by Xiong J *et al.* [18]. We categorized all the patients with various classifications at initial phase and follow-up at 3-6-12 months.

Management options

The decision of treatment was made on the basis of symptoms, signs and morphologic characteristics from the CT findings. Patients with the following characteristics were indicated for endovascular or surgical intervention: (1) signs of bowel infarction; (2) signs of bowel ischemia after internal medical treatment for 5-7 days; (3) severe compression or occlusion of the true lumen, signs of arterial rupture ascribed to aneurysmal dilatation of the SMA, and deficiency of potential collateral bypass such as Rioloan arch from the inferior mesen-

CT classification to screen treatment for SMA dissection

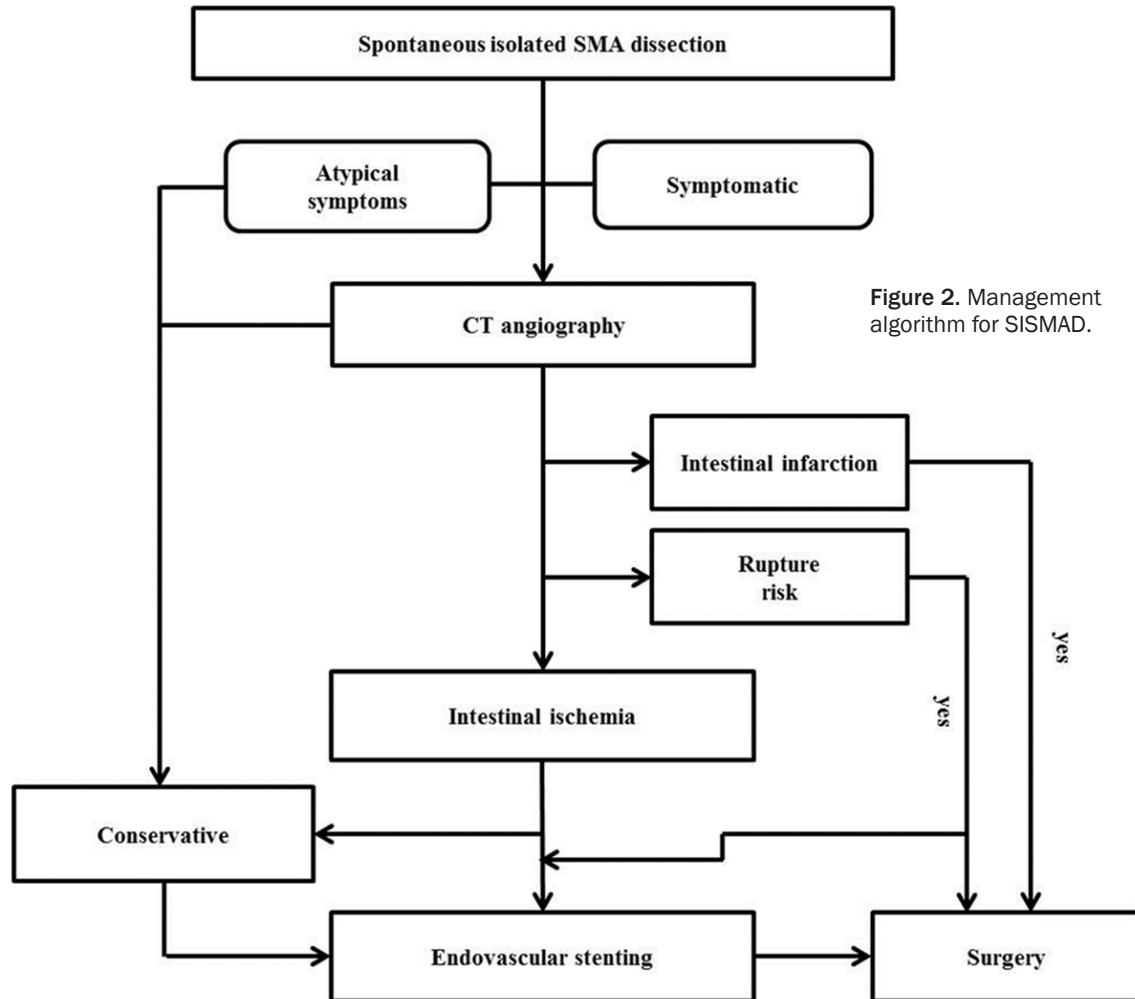


Figure 2. Management algorithm for SISMAD.

teric artery. Conservative management included bowel rest, parenteral nutrition support, hypertension control, antiplatelet, anticoagulant and thrombolytic treatment. ET included balloon dilatation, self-expandable stent implantation in the true lumen and coil embolization of the pseudoaneurysm in the dissected SMA. Surgical intervention consisted of SMA thrombectomy and infarcted bowel resection. Patients undergoing endovascular stent placement received antiplatelet therapy or drugs for 6 months postoperatively. Treatment of SISMAD from an institution was performed independently by a therapeutic team referring to algorithm for SISMAD (Figure 2).

Follow-up

All patients were reviewed or followed up by telephone at 3 months, 6 months and 12 months after discharge. The data were obtain-

ed by CTA or duplex. Clinical symptoms and follow-up findings on CTA or duplex were recorded. Three scales (improvement, stationary state, and progression) were accessed by both a gastroenterologist and a radiologist. Treatment for progressive patients was allowed by the present algorithm.

Statistical analysis

Kruskal-Wallis test or Fisher's exact test was used to compare clinical characteristics, each CT morphologic feature and outcomes of follow-up between symptomatic group and atypical symptom group. Mann-Whitney test was used for continuous variable comparison between these two groups. Statistical analysis was conducted using commercially available software (SPSS, version 19.0, SPSS, Armonk, NY). *P* values of less than 0.05 indicated a statistical significance.

CT classification to screen treatment for SMA dissection

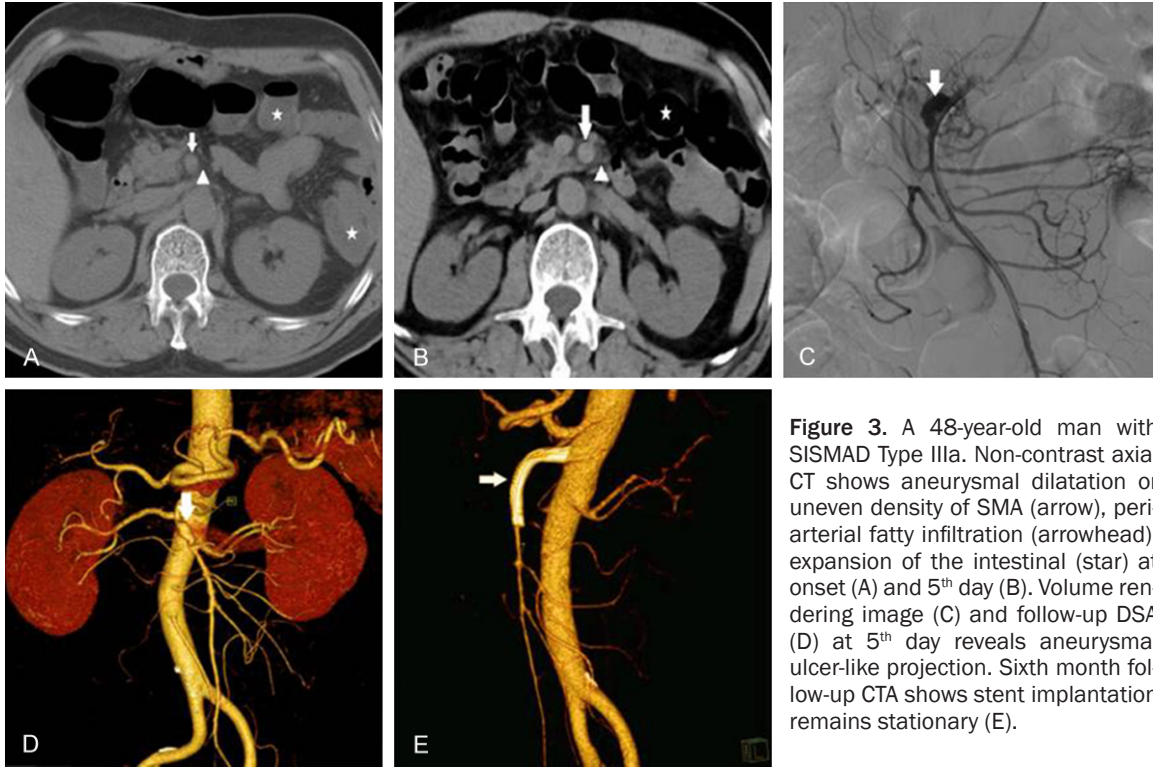


Figure 3. A 48-year-old man with SISMA Type IIIa. Non-contrast axial CT shows aneurysmal dilatation or uneven density of SMA (arrow), periaortic fatty infiltration (arrowhead), expansion of the intestinal (star) at onset (A) and 5th day (B). Volume rendering image (C) and follow-up DSA (D) at 5th day reveals aneurysmal ulcer-like projection. Sixth month follow-up CTA shows stent implantation remains stationary (E).

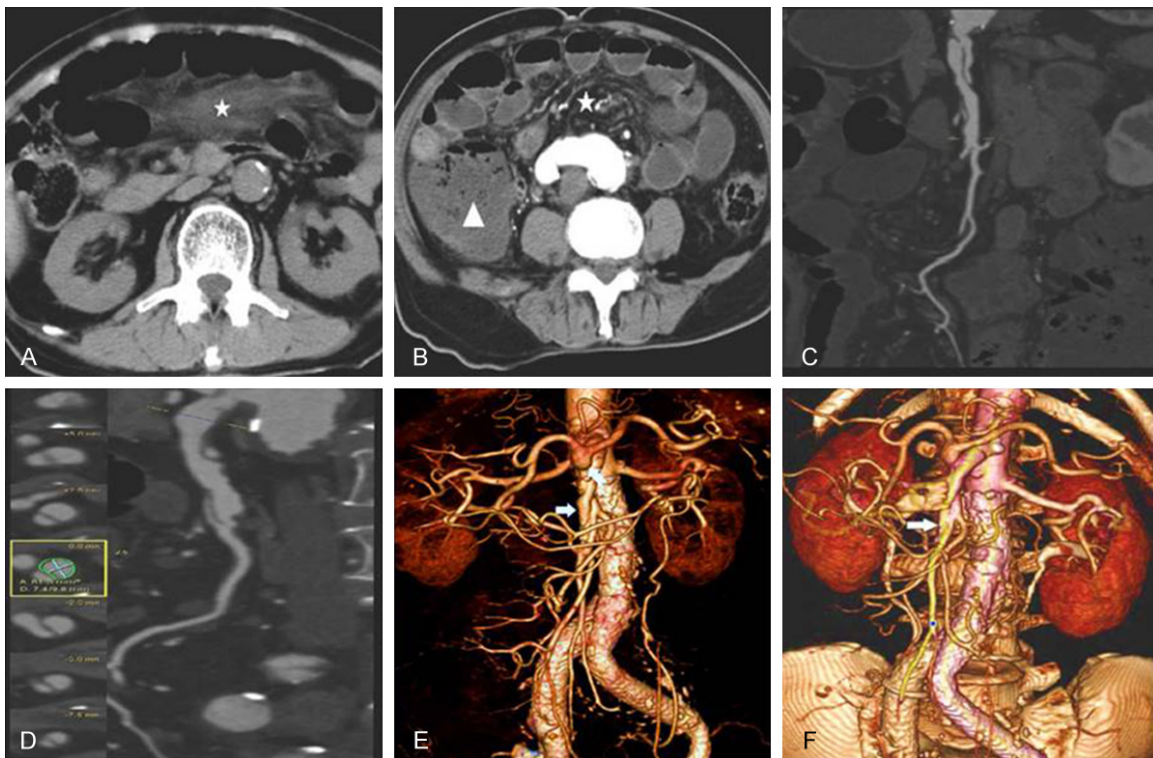


Figure 4. A 59-year-old man with SISMA Type I. Non-contrast axial image shows improvement of edema of mesentery (star) and progression of intestinal obstruction (triangle) from onset (A) to 6th day (B) during conservatively treatment. Curved planar reformations at 1st day (C) and 7th day (D) indicate the dissection with aneurysmal dilatation of false lumen (arrow) under a stable condition. Volume rendering images by initial CTA (E) shows that celiac artery stems from aneurysmal trunk (dovetail arrow) and remains stationary after 3 months conservative management (F).

CT classification to screen treatment for SMA dissection

Table 2. Baseline CT findings and classification (*n* = 37)

CT findings	Symptomatic patients (<i>n</i> = 22)	Atypical symptoms patients (<i>n</i> = 15)	<i>P</i> value
Initial CT features			
Periarterial fatty infiltration	12 (54.5%)	1 (6.7%)	.035
Bowel ischemia	11 (50.0%)	4 (26.7%)	.190
Bowel wall/mesentery edema	7 (31.8%)	3 (20.0%)	-
Intestinal obstruction	4 (18.2%)	1 (6.7%)	-
Intimal flap	19 (86.3%)	7 (46.7%)	.025
Aneurysmal dilatation	10 (45.5%)	4 (26.7%)	.090
Thrombosed false lumen	15 (68.2%)	4 (26.7%)	.020
True lumen stenosis	11 (50%)	2 (13.3%)	.035
Branch involvement	5 (22.7%)	0	.001
Riolan arch	3 (13.6%)	1 (6.7%)	.627
SISMAD type^a			
I	1 (4.5%)	6 (40.0%)	-
II			-
IIa	1 (4.5%)	1 (6.7%)	-
IIb	4 (18.2%)	4 (26.7%)	-
III			-
IIIa	10 (45.5%)	3 (20.0%)	-
IIIb	2 (9.1%)	0	-
IIIc	2 (9.1%)	1 (6.7%)	-
IV	1 (4.5%)	0	-
V	1 (4.5%)	0	-

a, difference of number of type III, IV and V between symptomatic patients and atypical symptoms patients.

Table 3. Outcomes of 5-7 days conservative treatment

	Type I-II		Type III-V		Total
	SP	ASP	SP	ASP	
Improvement	5	7	7	1	20
Stationary	1	4	5	3	13
Progression	0	0	3	0	3
Total	6	11	15	4	36

SP: Symptomatic patients; ASP: Atypical symptom patients.

Results

Patient characteristics

The demographic and baseline clinical characteristics of all 37 patients with SISMAD were shown in **Table 1**. The duration of abdominal pain before admission to our hospitals ranged from 2 hours to 46 days (median 7.2 days). One patient with type V SISMAD received infarcted bowel resection and thrombectomy with emer-

gency abdominal operation, while the rest of patients allowed observational management referring to our algorithm for SISMAD. Fourteen patients underwent ET (**Figure 3**), while the other twenty two patients were treated conservatively (**Figure 4**).

CT features

Signs of CT scan and the classification were shown in **Table 2**. 72.7% (16/22) symptomatic patients and 26.7% (4/15) asymptomatic cases presented as type III-V. On initial CT scan, 7 of 18 patients showed aneurysmal dilatation or uneven density of SMA on the onset, which were identified as dissection later by non-enhanced CT scan (**Figure 3A** and **3B**). Two cases with type IIa SISMAD only showed a linear intimal flap (**Figure 1**), 20 cases had partial (**Figure 3C** and **3D**)

or complete thrombosis (**Figure 1**) of a false lumen and 15 patients (Type I = 7 cases; IIb = 8 cases) without thrombus (**Figure 1**). Entry, re-entry or both were found in 13, 3 and 2 patients, respectively (**Figure 1**). Among 37 patients, the mean diameter of the true lumen of SMA at the region of maximal stenosis between the original and ileocolic branch was 2.9 mm (range 0-6.1 mm) in symptomatic cases and 3.3 mm (range 0-5.7 mm) in asymptomatic group, and mean percent of compression of true lumen was 62.2% in symptomatic cases and 30% in atypical symptom group, respectively. Eighteen patients were found ulcer-like projection and were hence classified into type III, and 88.9% (16/18) of the ulcer-like projection occurred at the maximum curve of SMA. Simultaneous dissections of the SMA and celiac artery were found in 2 patients. Six patients showed significant stenosis as a result of celiac artery atherosclerotic plaque and compression of median arcuate ligament. A case was found celiac artery stemming from aneurysmal trunk of SMA (**Figure 4E**).

CT classification to screen treatment for SMA dissection

Table 4. Treatment outcomes by follow-up at 3 months, 6 months, 12 months

Outcome ^a	Conservative (n = 17)	Endovascular (n = 11)	Surgery (n = 1)
3 months			
Improvement	13 (59.1%)	10 (71.4%)	1 (100%)
Stationary	3 (13.6%)	0	0
Progression	1 (4.5%)	1 (14.3%) ^b	0
6 months			
Improvement	4 (15.2%)	6 (42.9%)	0
Stationary	11 (50.0%)	4 (28.6%)	1 (100%)
Progression	1 (4.5%)	0	0
12 months			
Improvement	5 (22.7%)	1 (7.1%)	0
Stationary	10 (45.5%)	9 (64.3%)	1 (100%)
Progression	0	0	0

Data are numbers of patients with percentages in parentheses. a, 8 patients (5 cases in conservative treatment and 3 cases with endovascular intervention) were excluded from assessment the outcomes of original treatment; b, patients underwent by the subsequent therapy due to the progression of SISMAD were excluded too.

During the first 5-7 days' conservative treatment, the intestinal ischemia-related symptoms of 20 patients (16 remarkable cases, 4 mild cases) relieved with a reduction of mesenteric edema and improvement of bowel obstruction (**Figure 4A** and **4B**), but 16 patients were observed persistent abdominal pain. A repeat CTA (13 patients) and DSA (3 patients) were underwent, which not only showed a progression of thrombosis in 3 patients, improvement of true lumen size in 1 patient, and stationary state of 12 patients, but also revealed that the false lumen got blood supply from collateral vessels in 3 patients. No statistical difference ($\chi^2 = 0.004$, $P = 0.947$) in ischemia progress between symptomatic patients and atypical symptom patients was shown in **Table 3**.

Follow-up

During hospitalization, 3 cases were of Type II progressing to Type IIIa, 2 cases were of Type IIIb, and 2 cases were of Type IIIc transforming to Type IV. The rest of patients remained unchanged in CT classification. Eight patients (5 cases with conservative treatment and 3 cases with endovascular intervention) missing one or more follow-up records were excluded. Treatment outcomes by follow-up at 3 months, 6 months, 12 months were shown in **Table 4**. Among 29 patients, 2 cases with

conservative treatment had a progression at 3 or 6 months; 2 patients treated conservatively showed a mild remodeling of true lumen (**Figure 4E** and **4F**). All patients either kept improvement or remained stationary state (**Figure 3E** and **3F**), except one who underwent endovascular stenting and was found thrombosis filled in the stent and the distal segment of SMA during 3 months' follow-up. Interestingly, an increase from 6 to 9 cases showed that SMA were supplied by the inferior [19] via Riolan arch.

Discussion

With an increasing use of CT scan, SISMAD is gradually reported as a rare but important cause of acute superior mesenteric ischemia. A review revealed that 86% of 278 SISMAD patients were first diagnosed by contrast-enhanced CT and the rate increased to 95% recently [20]. Most of these cases initially presented with acute abdominal pain, but some of the clinical manifestations of atypical cases were rarely concerned. Thus, the incidence of SISMAD might be underestimated without identification by CT or DSA.

The pathogenesis of this vascular disease is still poorly understood. Regarding the causes of SISMAD, cigarette and hypertension have been pointed out as common risk factors [21-23] and these were respectively noted in 59.5% and 48.6% of our patients, with 77.8% of these 18 patients losing hypertension control. In our study, we found 48.6% of our patients presented non-calcified arteriosclerosis in the ascending aorta, but only 4 patients had atherosclerotic plaque in the proximal segment of SMA, which showed that atherosclerotic plaque increased the risk of SISMAD occurrence. Another important factor is that the anterior wall of the SMA at maximum curvature bears turbulent shear stress, which may be explored the hemodynamic mechanism of the occurrence and development of dissection [24]. In addition, several other risk factors, including cystic medial necrosis, fibro muscular dysplasia, heredity, blunt or iatrogenic *injuries* and connective tissue disorders have been involved [25-28]. Thus, it seems complicated in mechanisms, both arterial intima damage and hemodynamic abnormalities in the proximal

CT classification to screen treatment for SMA dissection

segment of SMA may be major causes of SISMAD [24, 29].

By comparing symptomatic group and atypical group, there was a statistical difference in thrombosis in false lumen, stenosis of true lumen and branch involvement. As a result, the sum of Type III, IV and V of symptomatic group was higher than that of atypical group. Similarly, white blood cells and CRP increased in symptomatic group, but D-dimer in these two groups had no statistical difference accordingly. Interestingly, few patients with Type I-II dissection complained with severe abdominal pain but the symptom relieved within 24 hours. Generally, no difference in bowel ischemia was found between symptomatic group and atypical group and no correlation between pain severity and dissection type had been identified [20]. We hypothesize that acute intestinal ischemia is the main cause of abdominal pain, and sudden reduction of blood supply causes acute mesenteric ischemia.

An intimal flap, a true and false lumen are reliable signs for dissection of SMA on CT angiography [2]. However, 7 patients (4 symptomatic; 3 atypical symptom) in the present study did not display an intimal flap. There was not enough difference in the density between teared intimal flap and thrombosed false lumen. False lumen was filled with low density thrombus in 40%-71.4% patients with SISMAD according to their various CT classifications [2, 29, 30]. A previous study had described increased high attenuation of fat segmentally around the SMA on CT scans of patients with SMA dissection, which similarly showed that in patients suffered acute embolism of superior mesenteric artery. However, it was unclear what exact pathologic process caused the phenomenon, because various diseases were known to be able to cause the phenomenons, including inflammation (e.g. mesenteric panniculitis), tumor and tumor-like lesions (e.g. retroperitoneal fibrosis, lymphoma, other malignancies), trauma, mesenteric edema or congestion (e.g. cirrhosis, hypoalbuminemia), and vascular lesions (e.g. SMA dissection). The pathologic process in vascular lesions is difficult to be clarified because of poor pathologic evidence [31, 32]. In 12 of our patients with fatty infiltration (mainly in symptomatic patients), the white blood cells and CRP were increased. Thus, we

considered that the periarterial fatty infiltration could be one part of secondary inflammation related to artery dissection.

The first CT-guided classification scheme for SISMAD was reported by Sakamoto *et al.* [2]. Many studies have shown the benefits of CT images for classification of SISMAD types [33]. We adopted CT classification of SISMAD and divided type II into IIa and IIb to explain a patient who had a localized intimal tearing without false lumen. In addition to this classification, other patients could be described by the classifications of Xiong J [18].

In terms of therapeutic strategy, conservative management is usually recommended as first-line treatment unless such condition as bowel necrosis and/or high risk of rupture of artery occurs, which indicates a surgical or endovascular procedure. There is no consensus on the duration of antiplatelet, anticoagulant and thrombolytic treatment. Long term anticoagulation therapy was advised because it was necessary to prevent thrombosis of a true lumen [2]. However, a previous study including 28 SISMAD patients showed that there was no statistical difference between the two groups with or without anticoagulation treatment [29]. In the present study, symptomatic patients were received both antiplatelet and anticoagulation treatment during hospitalization for 5-7 days as first-line treatment, which showed favorable efficacy in 55.6% (20/36) patients whose abdominal pain were gradually alleviated. In addition, 5-7 days of conservative treatment will not increase the risk of aggravation of intestinal ischemia in symptomatic patients. However, we cannot conclude that combination antiplatelet with anticoagulant therapy is superior to use them separately.

In general, endovascular treatment is preferred to SMA dissection patients with failed conservative management rather than surgery [13], because of low invasiveness and rapid improvement of ischemia that shortens the hospital stay. Min *et al.* [33] state that ET should be attempted when intestinal ischemia is suggested with either stenosis more than 80% or dilating to 2.0 cm or more of the true lumen. We expect that the indications for endovascular interventional procedures will be widened with the development of techniques and instruments. Referring to our management algorithm

CT classification to screen treatment for SMA dissection

for SISMA, endovascular intervention will be applied for a patient whose condition is inappropriate for conservative management. Although anti-platelet agents were regularly prescribed for all of our patients received ET after discharge, one patient who involved the branches of SMA was found secondary thrombosis at 24-day follow-up after an 8 cm stent implantation. If dissection extends to the branches of SMA, the repaired vessel may be restored or remodeled. But in some cases, the false lumen cannot be eliminated immediately. Thrombosis may occur again at the distal segment of the stent. Therefore, it is necessary to study a larger number of patients and make a longer-time follow-up carefully until dissection is completely resolved. Several factors including stenosis of SMA, collateral circulation, enhancement of intestinal wall and clinical manifestations should be integrally considered when intestinal ischemia is confirmed.

Some researchers argued that open surgical treatment should be first applied in the case of bowel infarction and rupture of SMA dissection. Gobble *et al.* [34] consider the treatment should be done if symptoms persist over 24 hours. Nevertheless, dissected SMA rupture in the acute phase is rare. Nomura *et al.* reported one patient was observed rupture of false aneurysm after 3 years' follow-up [35]. In our study, no aneurysm developed over middle-term follow-up in 37 patients, and a longer periodic observation might be continued until dissection of SMA is completely cured.

The major limitations of this study are its retrospective nature and the small number of surgical treatment groups, hampering analysis of the correlation between radiologic findings and clinical features, including treatment and outcomes. In addition, the identification of bowel infarction and rupture of SMA dissection are based on investigators' experience in clinical manifestation and CT features. The follow-up duration was relatively short to determine the development of aneurysmal dilatation.

In conclusion, our new classification on CTA for SISMA can satisfactorily demonstrate artery dissection and related intestinal ischemia and secondary inflammation. Within these categories type III-V should be screened out for requirement of ET or surgery once conservative management for 5-7 days fails. In view of the

risk of re-embolism, a long-time careful follow-up is necessary irrespective of what therapies patients receive.

Disclosure of conflict of interest

None.

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References

- [1] Vignati PV, Welch JP, Ellison L, Cohen JL. Acute mesenteric ischemia caused by isolated superior mesenteric artery dissection. *J Vasc Surg* 1992; 16: 109-112.
- [2] 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: 103-110.
- [3] Mousa AY, Coyle BW, Affuso J, Haser PB, Vogel TR, Graham AM. Nonoperative management of isolated celiac and superior mesenteric artery dissection: case report and review of the literature. *Vascular* 2009; 17: 359-364.
- [4] Lee WH, Lim CH, Kim SW. Spontaneous isolated superior mesenteric artery dissection mimicking superior mesenteric artery syndrome. *Kor J Gastroenterol* 2013; 62: 310-312.
- [5] Okada M, Ishiguchi T, Itoh H. Management of spontaneous dissection of the superior mesenteric artery. *Internal Med* 2004; 43: 451-452.
- [6] Matsushima K. Spontaneous isolated dissection of the superior mesenteric artery. *J Am Coll Surg* 2006; 203: 970-971.
- [7] Wu B, Zhang J, Wang L, Li X, Yang D, Duan ZQ, Xin SJ. Isolated superior mesenteric artery dissection: case for non-operative management. *Natl Med J Chin* 2008; 88: 25-27.
- [8] Kim HK, Jung HK, Cho J, Lee JM, Huh S. Clinical and radiologic course of symptomatic spontaneous isolated dissection of the superior mesenteric artery treated with conservative management. *J Vasc Surg* 2014; 59: 465-472.
- [9] Luan JY, Li X. CT classification and endovascular management of isolated dissection of the superior mesenteric artery with anatomical variations. *Eur J Vasc Endovasc Surg* 2014; 47: 209.
- [10] Akuzawa N, Seki H, Oku Y, Totsuka M, Hatori T, Imai K, Kitahara Y, Aoki J, Tashiro M, Kuraba-

CT classification to screen treatment for SMA dissection

- yashi M. Three cases of spontaneous isolated dissection of the superior mesenteric artery. *J Emerg Med* 2015; 48: e111-116.
- [11] Luan JY, Guan X, Li X, Wang CM, Li TR, Zhang L, Han JT. Isolated superior mesenteric artery dissection in China. *J Vasc Surg* 2016; 63: 530-536.
- [12] Zhang X, Sun Y, Chen Z, Li X. Therapeutic regimen options for isolated superior mesenteric artery dissection. *Vasc Endovasc Surg* 2012; 46: 277-282.
- [13] Satokawa H, Takase S, Seto Y, Yokoyama H, Gotoh M, Kogure M, Midorikawa H, Saito T, Maehara K. Management strategy of isolated spontaneous dissection of the superior mesenteric artery. *Ann Vasc Dis* 2014; 7: 232-238.
- [14] Suzuki K, Shimohira M, Hashizume T, Shibamoto Y. Stent placement for acute superior mesenteric artery occlusion associated with type B aortic dissection. *Case Rep Vasc Med* 2015; 2015: 485141.
- [15] 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: 2092-2097.
- [16] Rong JJ, Qian AM, Sang HF, Meng QY, Zhao TJ, Li XQ. Immediate and middle term outcome of symptomatic spontaneous isolated dissection of the superior mesenteric artery. *Abdom Imag* 2015; 40: 151-158.
- [17] Dong Z, Ning J, Fu W, Guo D, Xu X, Chen B, Jiang J, Wang Y. Failures and lessons in the endovascular treatment of symptomatic isolated dissection of the superior mesenteric artery. *Ann Vasc Surg* 2016; 31: 152-162.
- [18] Xiong J, Wu Z, Guo W, Liu X, Wang L, Zhang H, Jia X, Ma X. The value of a new image classification system for planning treatment and prognosis of spontaneous isolated superior mesenteric artery dissection. *Vascular* 2015; 23: 504-512.
- [19] Mertens J, Daenens K, Fourneau I, Marakbi A, Nevelsteen A. Fibromuscular dysplasia of the superior mesenteric artery—case report and review of the literature. *Acta Chir Belg* 2005; 105: 523-527.
- [20] 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: 232-235.
- [21] Sheldon PJ, Esther JB, Sheldon EL, Sparks SR, Brophy DP, Oglevie SB. Spontaneous dissection of the superior mesenteric artery. *Cardiovasc Interv Radiol* 2001; 24: 329-331.
- [22] Tameo MN, Dougherty MJ, Calligaro KD. Spontaneous dissection with rupture of the superior mesenteric artery from segmental arterial mediolysis. *J Vasc Surg* 2011; 53: 1107-1112.
- [23] Park YJ, Park KB, Kim DI, Do YS, Kim DK and Kim YW. Natural history of spontaneous isolated superior mesenteric artery dissection derived from follow-up after conservative treatment. *J Vasc Surg* 2011; 54: 1727-1733.
- [24] Park YJ, Park CW, Park KB, Roh YN, Kim DI, Kim YW. Inference from clinical and fluid dynamic studies about underlying cause of spontaneous isolated superior mesenteric artery dissection. *J Vasc Surg* 2011; 53: 80-86.
- [25] Pessaux P, Regenet N, Arnaud JP. Resection of the retroportal pancreatic lamina during a cephalic pancreaticoduodenectomy: first dissection of the superior mesenteric artery. *Ann Chir* 2003; 28: 633-636.
- [26] Jang ES, Jeong SH, Kim JW, Lee SH, Yoon CJ, Kang SG. A case of acute ischemic duodenal ulcer associated with superior mesenteric artery dissection after transarterial chemoembolization for hepatocellular carcinoma. *Cardiovasc Interv Radiol* 2009; 32: 367-370.
- [27] Srinivasan KG, Srividya S, Ushanandhini P, Ramprabananth S. Spontaneous isolated superior mesenteric artery dissection—Report of two cases. *J Radiol Case Rep* 2009; 3: 6-13.
- [28] Jia Z, Zhang X, Wang W, Tian F, Jiang G, Li M. Spontaneous isolated superior mesenteric artery dissection: genetic heterogeneity of chromosome locus 5q13-14 in 2 male familial cases. *Ann Vasc Surg* 2015; 29: 1019, e1011-1015.
- [29] Yun WS, Kim YW, Park KB, Cho SK, Do YS, Lee KB, Kim DI, Kim DK. Clinical and angiographic follow-up of spontaneous isolated superior mesenteric artery dissection. *Eur J Vasc Endovasc Surg* 2009; 37: 572-577.
- [30] Luan JY and Li X. CT. Classification and endovascular management of isolated dissection of the superior mesenteric artery with anatomical variations. *Eur J Vasc Endovasc* 2014; 47: 209-209.
- [31] Okino Y, Kiyosue H, Mori H, Komatsu E, Matsumoto S, Yamada Y, Suzuki K, Tomonari K. Root of the small-bowel mesentery: correlative anatomy and CT features of pathologic conditions. *Radiographics* 2001; 21: 1475-1490.
- [32] Suzuki S, Furui S, Kohtake H, Sakamoto T, Yamasaki M, Furukawa A, Murata K and Takei R. Isolated dissection of the superior mesenteric artery: CT findings in six cases. *Abdom Imag* 2004; 29: 153-157.
- [33] Min SI, Yoon KC, Min SK, Ahn SH, Jae HJ, Chung JW, Ha J, Kim SJ. Current strategy for the treatment of symptomatic spontaneous

CT classification to screen treatment for SMA dissection

- isolated dissection of superior mesenteric artery. *J Vasc Surg* 2011; 54: 461-466.
- [34] Gobble RM, Brill ER, Rockman CB, Hecht EM, Lamparello PJ, Jacobowitz GR, Maldonado TS. Endovascular treatment of spontaneous dissections of the superior mesenteric artery. *J Vasc Surg* 2009; 50: 1326-1332.
- [35] Nomura Y, Yamaguchi M, Kitagawa A, Okada T, Okita Y, Sugimoto K. Hybrid management of ruptured isolated superior mesenteric artery dissecting aneurysm. *J Vasc Surg* 2011; 54: 1808-1811.