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Natural course and outcomes of spontaneous isolated celiac artery dissection according to morphological findings on computed tomography angiography

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Abstract

We aimed to identify natural course and optimal management of spontaneous isolated celiac artery dissection (SICAD) according to morphologic classification determined on computed tomography angiography (CTA), and to investigate the association between symptoms and morphological classification of SICAD.

This retrospective observational study included 21 consecutive patients with SICAD from January 2012 to April 2017. Demographic data, clinical features, treatment modalities, follow-up results, and CTA findings including morphologic classification, dissection length, and relative diameter of the true lumen (TLRD) were reviewed. Changes in follow-up CTA were recorded and compared to prior studies to reveal natural course of the disease.

The serial changes of SICAD on follow-up CTA according to morphologic classifications were as follows; type I (5/5, no interval change), type IIa (1/1, no interval change), type IIb (1/1, partial remodeling), type IIIa (1/4, complete remodeling; 1/4, partial remodeling; 1/4, no interval change; 1/4, deterioration), type IIIb (4/6, no interval change; 2/6, partial remodeling), and type IV (2/2, no interval change). Thirteen (61.9%) symptomatic and 8 (38.1%) asymptomatic patients were all treated with conservative management with or without antiplatelet and/or anticoagulation therapies. Symptomatic group (SG) more commonly had type IIb, IIIa, IIIb, and IV than asymptomatic group (AG) (SG; 11 patients, AG; 1 patient, $P = .002$). TLRD in AG was larger than that in SG (SG: $40.5 \pm 24.1\%$, AG: $61.7 \pm 7.0\%$, $P = .045$).

SICAD might be treated by conservative management in stable patients irrespective of the morphologic classification except for with type IV (dissecting aneurysm) and extension of celiac branch who may need an early intervention. Types IIb, IIIa, IIIb, and IV are TLRD are associated with patients' symptoms. Further studies on extended natural course of SICAD with a larger number of subjects are needed to draw a strong conclusion.

Abbreviations: A. fib = atrial fibrillation, AG = asymptomatic group, CHA = common hepatic artery, CT = computed tomography, CTA = computed tomography angiography, DM = diabetes mellitus, F/U = follow-up, HTN = hypertension, LGA= left gastric artery, SA = splenic artery, SG = symptomatic group, SICAD = spontaneous isolated celiac artery dissection, SISMAD = spontaneous isolated superior mesenteric artery dissection, TLRD = relative diameter of the true lumen.

Keywords: abdominal pain, celiac artery, computed tomography angiography, dissection, mesenteric ischemia

1. Introduction

Spontaneous isolated celiac artery dissection (SICAD) without aortic dissection is a very rare disease entity. To the best of our knowledge, around 160 cases of SICAD have been reported as case reports, small case series, or combined SICAD and spontaneous isolated superior mesenteric artery dissection (SISMAD) in the literature.[1–3] Its etiology, natural history, treatment guideline, and prognosis remain unclear.[1–8]

Treatment options for SICAD include nonoperative management with/without antiplatelet or anticoagulation therapies, endovascular stenting, and surgical revascularization.[1,4,6–8] Although there is diversity of treatment strategies, conservative treatment alone without intervention has been accepted as the first-line therapy with reasonable outcomes.[1,4,6–8] In addition, previous study conducted on SISMAD, not SICAD, has recommended that SISMAD stenting should be reserved for more grave patients as long-term patency and safety of stenting in SISMAD are currently disputable.[9] The similarity between SICAD and SISMAD might lead to similar management strategy

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for these 2 different entities. However, conservative treatment for SICAD remains controversial due to the progression into aneurysm or celiac trunk/hepatic artery dilatation even after successful initial conservative management.[3,6,10,11] Given that, previous study suggested that different morphologic types of SICAD may have different fates requiring adapted management similar to SISMAD.[7] Prior studies have recommended to decide for endovascular treatment according to clinical symptoms and morphologic findings based on computed tomography angiography (CTA) because early invasive treatments for SICAD and SISMAD might be beneficial in certain morphologic groups.[5,7] In that regard, specific morphologic types may be liable to progress and cause symptoms. However, in SICAD, the association between morphologic type on CTA and progression or symptoms is largely unknown due to rarity of the disease and relatively scarce attention.

The purpose of this study was to investigate morphologic characteristics of SICAD contributing to patients' symptoms and the natural course of the disease by observing morphologic changes on follow-up CTAs with the review of the relevant literatures on SICAD.

2. Methods

This study was approved by the Institutional Review Board of Ajou University Hospital (IRB number: MED-MDB-17-123). The requirement of informed consent was waived due to its retrospective nature.

2.1. Study population and data collection

This study retrospectively reviewed 21 patients who were diagnosed with SICAD at a tertiary teaching hospital in South Korea between January 2012 and April 2017. Data of patients with *International Classification of Diseases, tenth Revision* codes 443.29 (dissection of other artery), 557.0 (acute vascular insufficiency of intestine), 557.1 (chronic vascular insufficiency of intestine), or 557.9 (unspecified vascular insufficiency of intestine), and CT deciphered reports were collected from the database of our hospital. Diagnosis of SICAD based on spiral CTA findings was made when there was an intimal flap in the celiac artery and/or thrombosis of false lumen. Patients with concomitant aortic dissection, recent abdominal trauma, or operation-related SICAD were excluded from this study.

Demographic information, presenting complaints, comorbidities, and initial management strategies for each patient were obtained retrospectively. The clinical course of each patient was also reviewed. Interventions including medication changes, endovascular interventions, and open surgical repair were documented. Treatment strategy was determined according to patient's symptoms and signs, as well as initial CTA findings by the treating specialists. Treating physician in charge of the patient during hospitalization determined the time of CT follow-up and treatment strategy during follow-up period according to symptoms or follow-up CT findings. Enrolled patients were contacted by phone to investigate the latest situations for long-term follow-up. They were asked about symptoms of abdominal or back pain at rest, pain after eating, weight loss, or changes in eating habit since the diagnosis of celiac artery dissection, any current anticoagulant use, and treatment that might have been received at another institution. Similar data points were extracted from medical record of patients who had been seen in the clinic within the last year.

2.2. CT measurements and classification

CTAs were retrospectively reviewed to obtain imaging characteristics including morphologic classification of the dissection, distance from the aortic orifice to dissection point, length of the dissection, relative diameter of the true lumen (TLRD), and the presence of aneurysmal dilatation. TLRD was defined as relative ratio of the diameter of the true lumen compared to that of the adjacent uninvolved celiac axis. In case of dissection extending to celiac branches including common hepatic artery (CHA), splenic artery (SA), or left gastric artery (LGA), the length of the dissection was measured from the celiac artery continuously to the involved branching artery.

A modified morphologic classification for SISMAD proposed by Li et al[5] (Fig. 1) was used, including type I (patent false lumen with entry and re-entry), type II (nonthrombosed "cul-de-sac" type of false lumen without re-entry site) which was further subdivided into IIa (TLRD >30%) and IIb (TLRD ≤30%), type III (thrombosed false lumen with/without an ulcer-like projection) which was subdivided into IIIa (TLRD >30%) and IIIb (TLRD ≤30%), and type IV (dissecting aneurysm). Subtypes of type II and III dissections were determined after measuring TLRD from CTAs. Dissecting aneurysm was defined as more than 50% of increase in diameter relative to that of the uninvolved celiac artery.[12]

2.3. Determination of outcomes and definition

Patients were divided into symptomatic group (SG) and asymptomatic group (AG) according to the presence or absence of symptoms at the time of the diagnosis for SICAD.

Follow-up CTAs were reviewed to classify the status of SICAD into 1 of the following 4 categories: (1) no interval change, (2)

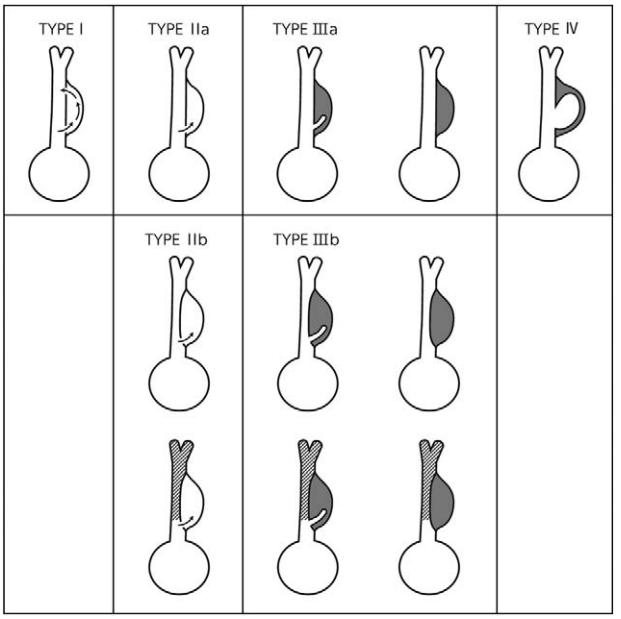

Figure 1. Morphologic classification of spontaneous isolated celiac artery dissection. Type I (patent false lumen with entry and re-entry), type II (nonthrombosed "cul-de-sac" type of false lumen without re-entry site) which was further subdivided into IIa (TLRD >30%), and IIb (TLRD ≤30%), type III (thrombosed false lumen with/without an ulcer-like projection) which was subdivided into IIIa (TLRD >30%), and IIIb (TLRD ≤30%), type IV (dissecting aneurysm). TLRD = relative diameter of the true lumen.

Table 1

Demographics, clinical characteristics, outcomes, and initial computed tomography morphology.

	Total (n = 21)	Asymptomatic group (n = 8)	Symptomatic group (n = 13)	*P* value
Age, years	56.5 ± 12.7 (35–82)	61.9 ± 11.1 (44–75)	53.2 ± 12.9 (35–82)	.157
Male gender	19 (90.5%)	8 (100%)	11 (84.6%)	.371
Risk factor				
Smoking	9; 42.9%	3	6	.528
Hypertension	8; 38.1%	6	2	.011
Hyperlipidemia	4; 19.0%	2	2	.498
Diabetics	2; 9.5%	2	0	.133
Connective disorder	0	0	0	–
Vasculitis	0	0	0	–
Cancer	3; 14.3%	2	1	.316
Treatment				.123
Conservative therapy only	8; 38.1%	5	3	
Conservative therapy with antiplatelet (aspirin)	7; 33.3%	3	4	
Conservative therapy with anticoagulation (warfarin)	4; 19.0%	0	4	
Conservative therapy with antiplatelet and anticoagulation	2; 9.5%	0	2	
Endovascular therapy	0	0	0	
Open surgery	0	0	0	
Bleeding complication	0	0	0	–
Death	2 (9.5%)	2	0	.133
Follow-up, weeks	42.2 ± 47.4 (3–180)	34.0 ± 50.8 (3–141)	47.3 ± 46.5 (6–180)	.088

partial remodeling (true lumen diameter was increased or dissection length was shortened), (3) complete remodeling (dissection and thrombus disappeared), and (4) deterioration of SICAD on CT (true lumen diameter was decreased, dissection length was longer than previous CT, or aneurysm developed). Status of SICAD was recorded and categorized based on each follow-up CTA to identify serial changes.

The treatment strategy was categorized as follows: (1) conservative management without antiplatelet agent or anticoagulation, (2) conservative management with antiplatelet agent, (3) conservative management with anticoagulation, (4) conservative management with both antiplatelet agent and anticoagulation, (5) endovascular treatment, and (6) surgery. Conservative treatment comprised strict blood pressure control defined as maintaining normal range of systolic blood pressure, bowel rest, intravenous fluid administration, nutritional support, and close observation. Anticoagulation was generally continued by the treating physician.

2.4. Statistical analysis

Data of 21 patients were analyzed using SPSS 23 statistics software (SPSS Inc., Chicago, IL). Continuous data are expressed as means and standard deviations (range minimum to maximum). Categorical data are treated as absolute values. Chi-square tests or Fisher's exact tests were performed to compare categorical data between 2 groups. Difference in continuous variables between 2 groups was analyzed using Mann–Whitney *U* test or independent *t*-test.

To evaluate the CTA morphologic classification associated with the symptoms, linear by linear analysis was conducted to observe the tendency according to significance the CTA morphologic classification. *P* value $\leq .05$ was defined as significance.

To investigate natural course of SICAD, morphologic changes were observed, including changes in CT morphologic classifications, TLRD, distance from the aortic orifice to dissection point, and length of the dissection in serial CT scans for patients who underwent CTA follow-up.

3. Results

3.1. Patient characteristics and clinical features

Demographics, risk factors of interest, clinical characteristics, and outcomes between SG and AG are summarized in Table 1. Clinical characteristics of all 21 patients are summarized in Table 2. The mean age of these 21 patients was 56.5 ± 12.7 years (range, 35–82 years). Of these 21 patients, 19 (90.5%) were males and 2 (9.5%) were females. Comorbidities included smoking (n = 9, 42.9%), hypertension (n = 8, 38.1%), hyperlipidemia (n = 4, 19.0%), cancer (n = 3, 14.3%), diabetes (n = 2, 9.5%), atrial fibrillation (n = 1, 4.8%), congestive heart failure (n = 1, 4.8%), and end-stage renal disease (n = 1, 4.8%). The duration of follow-up was 42.2 ± 47.4 weeks (range, 3–180 weeks). There were 13 (61.9%) symptomatic patients and 8 (38.1%) asymptomatic SICAD patients. Of the 13 symptomatic SICAD patients, 12 (92.3%) had abdominal pain while the remaining 1 patient had chest pain without abdominal pain. Locations of abdominal pain included epigastric area (n = 5, 41.7%), diffuse area (n = 3, 25.0%), periumbilical area (n = 2, 16.7%), lower abdominal area (n = 1, 8.3%), and left abdominal area (n = 1, 8.3%). The mean interval from the onset of symptoms to admission was 26.8 ± 30.4 hours (range, 1–72 hours) in 12 patients. The remaining 1 patient visited the hospital for chronic abdominal pain 1 month later. Endovascular or surgical treatment was not conducted in our series. All 21 patients received conservative treatment, including 8 (38.1%) who did not receive antiplatelet or anticoagulation therapy, 7 (33.3%) who received antiplatelet therapy, 4 (19.0%) who received anticoagulation therapy, and 2 (9.5%) who received both antiplatelet and anticoagulation therapies. There was no anticoagulation therapy-related mortality or morbidity. Two (9.5%) of 21 patients died of cancer not related SICAD.

3.2. Morphologic classification and other measurements on CTA at diagnosis of SICAD

Initial CT morphologic classification and findings between SG and AG are summarized in Table 3. Based on CT classifications

Table 2

Initial characteristics in 21 patients with spontaneous isolated celiac artery dissection.

Patient no.	Age	Gender	Symptoms	Comorbidity	Associated aterial branch	Initial CT classification	Length of dissection, cm	True lumen residual diameter, %	Treatment	Follow-up periods, weeks
1	44	Male	Epigastric pain	HTN, dyslipidemia, smoking	SA, CHA, LGA	Type IIIa	12.9	50.7	Conservative care Anticoagulation	43
2	48	Male	Epigastric pain	Smoking	SA, CHA	Type IIIb	15.2	29.4	Conservative care Anticoagulation	30
3	62	Male	Abdominal pain	Dyslipidemia	–	Type IV	1.5	n/a	Conservative care Antiplatelet	180
4	39	Male	Chest pain	–	–	Type IIIa	2.7	36.2	Observation	30
5	65	Male	–	HTN, DM, A. fib, smoking	–	Type IIa	3.3	62.5	Conservative care	5
6	70	Male	–	HTN, smoking	–	Type IIa	2.1	73.0	Conservative care	3
7	47	Male	–	–	–	Type IIIa	2.5	56.7	Conservative care Antiplatelet	17
8	75	Male	–	HTN, DM, dyslipidemia	–	Type IIa	1.8	59.0	Conservative care	9
9	48	Male	Abdominal pain	–	SA, CHA	Type IIIa	5.9	68.8	Conservative care	25
10	57	Male	Abdominal pain	HTN	–	Type IIIa	3.3	64.6	Conservative care Antiplatelet	55
11	52	Female	Abdominal pain	–	SA	Type IIIb	12	28.6	Conservative care Antiplatelet	30
12	35	Male	Epigastric pain	Smoking	SA, CHA	Type IIIb	4.3	0	Conservative care Anticoagulation Antiplatelet	72
13	66	Male	–	–	–	Type I	1.5	58.0	Conservative care Antiplatelet	10
14	44	Male	–	HTN, dyslipidemia, smoking	–	Type I	1.3	71.4	Observation	83
15	48	Male	Epigastric pain	Smoking	SA	Type IIIb	7	27.9	Conservative care Anticoagulation Antiplatelet	6
16	69	Male	–	HTN	–	Type I	2.3	53.3	Conservative care Antiplatelet	4
17	72	Male	Abdominal pain	Smoking	–	Type IIIb	2	0	Conservative care	90
18	82	Female	Abdominal pain	–	–	Type IIa	2	55.0	Conservative care Anticoagulation Antiplatelet	16
19	54	Male	Abdominal pain	Smoking	–	Type I	2.8	66.2	Conservative care Anticoagulation	28
20	50	Male	Epigastric pain	–	–	Type IIb	2.3	58.3	Conservative care Anticoagulation	10
21	59	Male	–	HTN	–	Type I	4.4	60	Conservative care	141

A. fib = atrial fibrillation, CHA = common hepatic artery, CT = computed tomography, DM = diabetes mellitus, HTN = hypertension, LGA = left gastric artery, SA = splenic artery.

for SICAD, 5 patients had type I (AG, n = 4; SG, n = 1), 4 had type IIa (AG, n = 3; SG, n = 1), 1 had type IIb (AG, n = 0; SG, n = 1), 5 had type IIIa (AG, n = 1; SG, n = 4), 5 had type IIIb (AG, n = 0; SG, n = 5), and 1 had type IV (AG, n = 0; SG, n = 1). Three symptomatic patients and 1 asymptomatic patient at the time of diagnosis had concomitant spontaneous SISMAD. However, these 4 patients showed no symptoms during the follow-up period. The mean ± standard deviation for TLRD, distance from

Table 3

Initial computed tomography morphologic classification and findings.

	Total (n = 21)	Asymptomatic group (n = 8)	Symptomatic group (n = 13)	*P* value
Initial CT classification				.002
Type I	5 (23.8%)	4	1	
Type IIa	4 (19.0%)	3	1	
Type IIb	1 (4.8%)	0	1	
Type IIIa	5 (23.8%)	1	4	
Type IIIb	5 (23.8%)	0	5	
Type IV	1 (4.8%)	0	1	
True lumen residual diameter, %	49.0 ± 21.7 (0–73.0)	61.7 ± 7.0 (53.3–73.0)	40.5 ± 24.1 (0–68.8)	.045
Distance from the aorta to dissection point, cm	0.6 ± 0.6 (0–1.8)	0.8 ± 0.4 (0–1.3)	0.5 ± 0.7 (0–1.8)	.223
Dissection diameter, cm	4.4 ± 4.0 (1.3–15.2)	2.4 ± 1.0 (1.3–4.4)	5.7 ± 4.7 (2.0–15.2)	.064

CT = computed tomography.

Table 4

Computed tomography morphological classification and change during follow-up period total in 19 among 12 patients.

Patient No	Age	Previous CT classification	F/U CT classification	CT change	Previous length of dissection, cm	F/U length of dissection, cm	Previous true lumen residual diameter, %	F/U true lumen residual diameter, %	Previous distance from the aorta to dissection point, cm	F/U distance from the aorta to dissection point, cm	The time when CT F/U, week
1	44	Type IIIa	Type IIb	Deterioration	12.9	12.9	50.7	29.0	0	0	4
1	44	Type IIb	Type IIa	Partial remodeling	12.9	2.6	29.0	43.2	0	0	10
1	44	Type IIa	Type IIa	No interval change	2.6	2.6	43.2	82.9	0	0	19
2	48	Type IIIb	Type IIIb	No interval change	15.2	15.2	29.4	29.4	0	0	3
2	48	Type IIIb	Tyepe IIa	Partial remodeling	15.2	2.8	29.4	46.7	0	0.9	10
3	62	Type IV	Type IV	No interval change	1.5	1.5	–	–	1.7	1.7	8
3	62	Type IV	Type IV	No interval change	1.5	1.5	–	–	1.7	1.7	70
4	39	Type IIIa	Type IIa	Partial remodeling	2.7	1.2	36.2	60.3	0	1.3	30
9	48	Type IIIa	Complete remodeling	Complete remodeling	5.9	–	68.8	100.0	0	–	12
10	57	Type IIIa	Type IIIa	No interval change	3.3	2.6	64.6	71.6	0.5	0.5	4
12	35	Type IIIb	Type IIIa	Partial remodeling	4.3	3.2	0	66.7	1.5	1.2	44
14	44	Type I	Type I	No interval change	1.3	1.3	71.4	71.4	1.2	1.2	77
15	48	Type IIIb	Type IIIb	No interval change	7.0	7.0	27.9	27.9	1.8	1.8	0.5
17	72	Type IIIb	Type IIIb	No interval change	2.0	2.0	0	0	0	0	85
17	72	Type IIIb	Type IIIb	No interval change	2.0	2.0	0	0	0	0	88
19	54	Type I	Type I	No interval change	2.8	2.8	66.2	66.2	1.1	1.1	2
19	54	Type I	Type I	No interval change	2.8	2.8	66.2	66.2	1.1	1.1	18
21	59	Type I	Type I	No interval change	4.4	4.4	60.0	60.0	0.7	0.7	93
21	59	Type I	Type I	No interval change	4.4	4.4	60.0	60.0	0.7	0.7	51

CT = computed tomography, F/U = follow-up.

the aorta to dissection point, and dissection length were 49.0 ± 21.7% (range, 0–73.0%), 0.6 ± 0.6 cm (range, 0–1.8 cm), and 4.4 ± 4.0 cm (range, 1.3–15.2 cm), respectively. There was no significant difference in distance from the aorta to dissection point and dissection length between SG and AG ($P = .223$, $P = .064$, respectively). The CT classification between AG and SG showed significantly different proportion of distribution ($P = .002$). The tendency to have symptoms increased with type IIb, type IIIa, type IIIb, type IV in CT classification: type I (n = 5, SG; n = 1, AG; n = 4), type IIa (n = 4, SG; n = 1, AG; n = 3), type IIb (n = 1, SG; n = 1, AG; n = 0), type IIIa (n = 5, SG; n = 4, AG; n = 1), type IIIb (n = 5, SG; n = 5, AG; n = 0), type IV (n = 1, SG; n = 1, AG; n = 0). TLRD in NG tended to be larger than that in SG (SG; 40.5 ± 24.1%, AG; 61.7 ± 7.0%, $P = .045$). In our series, 6 patients had SICAD extending dissection to celiac branches. Patient number 1 had dissections extending to SA, LGA, and CHA. Patients number 2, 9, and 12 had dissections extending to SA and CHA. Patients number 11 and 15 had dissections extending to SA only. Among these patients, patients number 1 and 2 had splenic infarction. However, these patients were successfully treated by conservative management (Table 2).

3.3. Morphologic changes on follow-up CT scans

Nineteen follow-up CTAs were available for 12 patients. Changes in CT morphologic classification, TLRD, distance from the aortic orifice to dissection point, and length of dissection in serial CT scans are summarized in Table 4. For asymptomatic SICAD, there was no follow-up CT because there was no symptom during the follow-up period. As 1 patient with symptoms at the time of diagnosis (patient number 11) was free of symptoms during the follow-up period, CTA was not conducted during the follow-up period.

Partial remodeling was found in 4 (21.1%) of 19 follow-up CTAs. In details, remodeling from type IIb to IIa, type IIIb to IIa or IIIa, and type IIIa to IIa was observed. Complete remodeling occurred in 1 type IIIa (5.3%) case of the 19 follow-up CTAs (Fig. 2). No interval change was observed in 13 (68.4%) of 19 cases (type I, n = 5; type IIa, n = 1; type IIIa, n = 1; type IIIb, n = 4; type IV, n = 2). Deterioration was noted in 1 (5.3%) of 19 cases. Even in this patient (patient number 1) progression was observed only during the 1st follow-up showing narrowing of true lumen from type IIIa to IIb, which was partially remodeled to type IIa on the 2nd follow-up (Fig. 3). On the 3rd follow-up CT, the dissection was stable without change (type IIa). The patient was asymptomatic during the entire follow-up period.

Serial changes of SICAD according to CT classifications were as follows. Type I had no interval change (5/5). Type IIa had no interval change (1/1). Type IIb had partial remodeling (1/1). Type IIIa had complete remodeling (1/4), partial remodeling (1/4), no interval change (1/4), or deterioration (1/4). Type IIIb had no interval change (4/6) or partial remodeling (2/6). Type IV had no interval change (2/2).

4. Discussion

In this study, all patients with SICAD were successfully treated by conservative management with or without antiplatelet or anticoagulation therapy. They were free from harmful events. In addition, the natural course of SICAD according to CT morphologic classification was favorable during a mean follow-up period of 42.2 ± 47.4 weeks (range, 3–180 weeks) without invasive treatment. Patients with types IIb, IIIa, IIIb, and IV in CTA classification and smaller TLRD are likely to be symptomatic, though these results have limited transferability to general population due to small sample size.

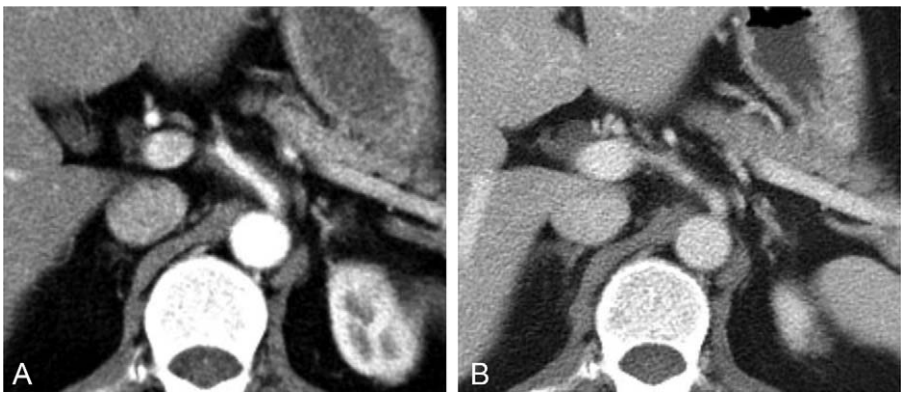

Figure 2. A symptomatic 48-year-old male patient initially presented with (A) type IIIa celiac artery dissection (thrombosed false lumen with relative diameter of the true lumen > 30%) showing (B) complete remodeling on the 12-week follow-up computed tomography scan.

SICAD without associated aortic dissection is rare, but the frequency of reporting asymptomatic dissection has increased due to technical advances in multidetector CTA with improved spatial/temporal resolution, and increased popularity of CTAs.[1,3,4,6–8,13,14] Because of its rarity, incidence or natural history of SICAD has not been completely characterized yet.[1,3,4,6–8,13,14] Previous studies have revealed that SICAD mainly occurs in male patients in their fifth decade of life (male ratio ranging from 82.6% to 100%; age ranging from 40 to 61 years) and abdominal pain being reported as the most common symptoms ranging from 82.6% to 100%.[1,6–8,10,15–18] Results of the current study revealed that demographic characteristics of SICAD patients were similar to those reported in previous studies. In our series, SICAD was more common in men (90.5%; 19/21), smokers (42.9%; 9/21), and hypertensive subjects (38.1%; 8/23). No connective tissue disorder or vasculitis was diagnosed in any patients enrolled for this study. The most common presenting symptom was abdominal pain (92.3%; 12/13).

Regarding treatment, natural course, initial CTA, and follow-up CTA findings for SICAD based on literature review of articles related to SICAD, 164 patients have been reported.[1,4,6–8,10,14–23] Of these 164 patients, 17 (10.4%) have received endovascular (n = 11) or surgical treatment (n = 6) from the beginning, while the remaining 147 (89.6%) received conservative treatment.[1,4,6–8,10,14–23] Sixteen (10.9%) of these 147 patients treated by conservative management had treatment failure.[1,6,7,15,18–20,23] Therefore, 10.4% (17/164) patients with SICAD needed invasive treatment as their initial treatment, and conservative management failed in 10.9% (16/147) patients.[1,4,6–8,10,14–23] CTA findings for 9 patients among 17 patients who initially underwent invasive treatment were described as follows; aneurysmal dilation (n = 5),[7] extension of celiac branch (n = 2),[23] huge aneurysm (n = 1),[21] and celiac trunk rupture (n = 1),[20] largely correspond to type IV dissection in morphologic classification of current study.[7,19–21,23] Initial CTA findings for 4 patients among 16 patients who failed conservative management were described as follows; celiac artery dilation (n = 2),[18,23] and extending dissection to celiac branches (n = 1),[6] remarking half of the cases corresponds to type IV dissection.[1,6,18,23] Five of these 16 patients had worsening symptoms and deteriorating CTA findings at follow-up, though their initial CTA findings were not described in the literature.[1,6,7,15,18,23] Eighteen among 164 patients showed type IV dissection on initial CTA.[7,15,21] Among them, 7 patients were successfully treated by conservative management and the other 11 patients were managed by invasive treatment.[7,15,21] Additionally in 1 prior study, extension of dissection to hepatic or splenic arteries and aneurysmal dilatation have been reported in 44% (10/23) and 39% (9/23) of patients, respectively, ultimately resulting in 33% (4/12) of patients requiring endovascular salvage for aggravating symptom.[7] Regarding SICAD extending dissection to celiac branches, it was difficult to identify the total number of SICAD with dissection of its branches in previous studies because some previous studies did not mention the extension of dissection

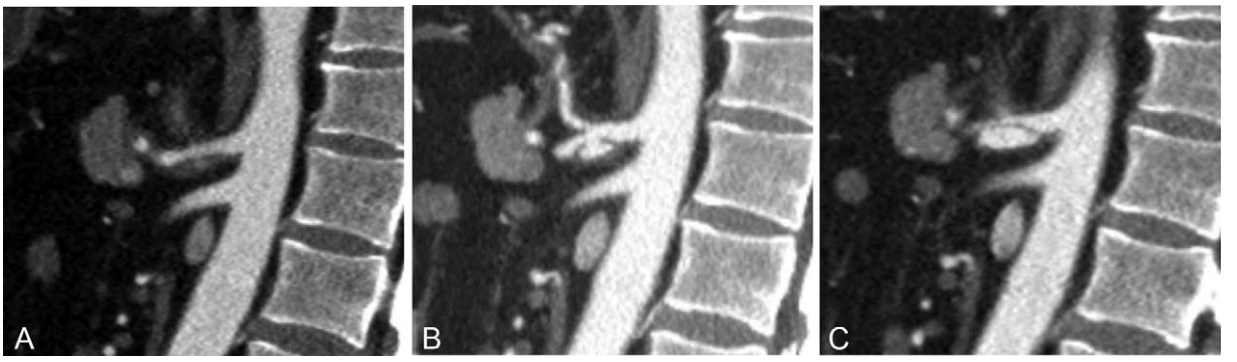

Figure 3. A symptomatic 44-year-old male patient initially presented with (A) type IIIa celiac artery dissection (thrombosed false lumen with relative diameter of the true lumen > 30%) deteriorated to (B) type IIb (nonthrombosed "cul-de-sac" type of false lumen with relative diameter of the true lumen < 30%) after 4 weeks. (C) On 10-week follow-up, the dissection partially remodeled to type IIa (nonthrombosed "cul-de-sac" type of false lumen with relative diameter of the true lumen > 30%).

to celiac branches. In a review of previous studies that mentioned whether the branch was involved, a total of 33 patients were identified as SICAD with extending dissection to celiac branches.[6–8,10,15,21–23] Among these patients, 21 (63.6%) were treated successfully by conservative management.[6–8,10,15,21–23] The other 12 (36.4%) patients were managed by invasive treatment such as endovascular treatment (n = 9)[6,7] and open surgery (n = 3)[21,23] due to malperfusion of internal organ except 2 cases that were treated by coli embolization (n = 1)[6] or open surgery (n = 1)[21], although there was no ischemic symptom. The reason was that 1 patient had SICAD with an aneurysm of SA[6] while the other patient had SICAD with a large dissecting aneurysm of the proper hepatic artery.[21] In our series, 6 patients had SICAD extending dissection to celiac branches. However, these patients were successfully treated by conservative management with anticoagulation therapies. They had no ischemic symptoms after conservative management. Overall, 131 out of 147 patients (89.1%) were successfully treated and showed symptomatic improvement with conservative therapy alone. Although these success rates for conservative management seem acceptable, details of the literatures emphasize that type IV dissection (dissecting aneurysm) and dissection that had extended to celiac branches are candidates for invasive treatment that may ultimately fail in conservative management alone. Given these, careful observation and consideration of early intervention is warranted for patients with extended dissection to celiac branches and dissecting aneurysm (type IV). In our series, the success rate with conservative management (100%) was higher than that (89.1%) obtained from the review of literatures. It may be attributed to the characteristics of subjects. Although 1 patient with dissecting aneurysm (type IV) and 6 patients with dissection involving the branch vessels in our series were included, these patients had no ischemic symptoms after conservative management. Further study is needed to evaluate the factors for predicting the progression of SICAD.

Most of the prior literature on SICAD did not specified the CT morphologic types or CT finding at initial and follow-up. There was no description on how each type changed in 131 patients with successful conservative management either. Nonetheless, the majority of the patients successfully managed with conservative treatment showed no interval change or regression of false lumen on follow-up CT.[1,4,6,8,10,14–22] In our study, follow-up CT showed no change or improvement in 94.7% of cases (partial remodeling, 21.1%; complete remodeling, 5.3%; no interval change, 68.4%) in terms of morphologic types. No recurrence or complication occurred at the last follow-up. From our experience and literatures review, conservative management might be chosen as the first-line treatment for SICAD regardless of CT morphologic classification except type IV and extension of celiac branch. It might be better to decide invasive treatment after symptomatic observation following conservative treatment. However, these concepts might not be generalizable due to its retrospective nature and small sample size of this study.

To investigate the association between the CTA classification and the symptoms, we used our classification system to reassign initial morphologic types of 147 patients who received conservative management as their first treatment from the literatures: type I (n = 6, symptomatic; n = 4,[14,16,22] unknown; n = 2[7]), type II (n = 13, symptomatic; n = 4,[17] asymptomatic; n = 2,[17] unknown; n = 7[7,8]), type III (n = 46, symptomatic; n = 38,[14–18] unknown; n = 8[7,22]), and type IV (n = 8, symptomatic; n = 8). Follow up CTA showed transformation into type I (n = 9),[7,14,18,22] type II (n = 16),[17,22] type III (n = 15)[7,14,17,18,22] and complete remodeling (n = 18).[15] Although statistical analysis was not conducted, symptoms associated with SICAD might often occur in types III and IV. Likewise in our series, patients with types IIb, IIIa, IIIb, and IV in CTA classification were frequently symptomatic. Thus, we suggest to distinguish type IIb and higher from types I and IIa, and to use a shorter follow-up period for the former to monitor for new symptoms that may need prompt interventions.

In our study, TLRD in NG tended to be larger than those in AG, respectively. To our best knowledge, the association between TLRD and symptoms has not been highlighted in SICAD. However, the previous study of SISMAD has reported that TLRD values are larger in observation groups than those in endovascular or surgically managed groups in SISMAD patients.[5] Smaller TLRD is likely to cause symptoms by reducing visceral perfusion. In light of current and prior studies, a watchful observation is warranted for patients with small TLRD who are having symptoms or at the risk of developing symptoms. Additional studies are needed to determine the relationship between TLRD and symptoms in SICAD.

This current study has several limitations. First, due to its retrospective design, not all patients were treated with a standardized protocol. Accordingly, part of asymptomatic patients might not have been followed up to confirm the degeneration of aneurysm. Differences such as TLRD and clinical features between groups might be due to selection bias. Second, the number of enrolled patients was rather small and the follow-up period was relatively short, although our follow-up period was longer than that in most previous studies. Additional studies are needed to establish clinical features and optimal therapeutic management for this disease. Third, the statistic result should not be generalized due to its small simple size. Through several journal reviews, however, we figured that patients with type III and type IV dissection were more often symptomatic. Integrating the results of this study and conclusions of journal reviews, there seems a relationship between symptom and CT classification types III and IV. Additional studies are needed to determine the relationship between CT findings and symptoms in SICAD. Forth, key elements including length of dissection, distance from the aorta to the dissection point, and TLRD were measured primarily on CT scans. Such measurements might differ from actual values. However, CT scans are currently widely available in most centers. They are likely to be performed as the choice of examination for patients with suspected dissection. Therefore, it is believed that these measurements are reflective of everyday clinical practice.

5. Conclusions

Our results suggest that initial conservative treatment might be adequate for patients with SICAD regardless of CT morphologic classification except for those with type IV (dissecting aneurysm) and extension of celiac branch. Types IIb, IIIa, IIIb, and IV along the TLRD are related with patients' symptoms. However, further evaluation of natural course with long-term follow-up and relation between TLRD and symptoms is needed.

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