



Strategy for the treatment of spontaneous isolated visceral artery dissection

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ABSTRACT

Objectives: To determine the incidence of rare spontaneous isolated visceral artery dissection (SIVAD), characterize its pathogenesis, and suggest treatment strategies.

Materials and Methods: We reviewed abdominal contrast-enhanced computed-tomography (CE-CT) scans from January 2005 to December 2016 retrospectively in our institution, identified 47 SIVAD patients and classified them into a symptomatic (n = 22) or asymptomatic group (n = 25). Further, we classified the five types based on the CE-CT images. Patient characteristics, incidence, vascular risk factors, complications, symptoms, treatments outcomes, and morphology features on CE-CT images were analyzed.

Results: SIVAD was seen on 0.09% of all abdominal CE-CT scans, and 0.68% of all abdominal CT-CT scans obtained for the evaluation of acute abdominal symptoms. The asymptomatic group had significantly fewer patients with periarterial fat stranding or branch vessel involvement on CE-CT images (p < 0.01). The mean length of the dissection was longer in the symptomatic group (p < 0.05). In the asymptomatic group, dissection-related abdominal symptoms and complications did not develop; followed-up CE-CT scans showed improvement in the dissection lesions in 1 (4.0%) patient, no changes in 22 (88.0%), and complete remodeling in 2 (8.0%). In the symptomatic group, one patient presented with organ ischemia at diagnosis and five patients developed organ ischemia underwent endovascular intervention. In the remaining 16 patients received nonoperative intervention only, followed-up CE-CT scans showed improvement in 13 (86.7%), and complete remodeling in 2 (13.3%).

Conclusions: Symptomatic SIVAD patients should be hospitalized because some of those may experience organ ischemia or aneurysm formation. Endovascular intervention is a feasible treatment for complications of SIVAD.

1. Introduction

Spontaneous isolated visceral artery dissection (SIVAD) is rarely encountered. By 2006, after the first case described by Bauerfeld in 1947 [1], only a few cases were reported in the literature. However, cases of SIVAD have been more frequently reported in recent years [2–16] because of technical advances in multi-detector computed-tomography (MDCT), improved computed-tomography (CT) resolution, and the increasing opportunities of MDCT for investigating abdominal pain [2,10,17,18]. Presumed risk factors include atherosclerotic disease, hypertension, fibromuscular dysplasia, cystic medial necrosis, trauma, pregnancy and connective tissue disorders [3,19]. Treatment options for the management of SIVAD include nonoperative,

endovascular and surgical interventions [5–12,14,3–16,20,21]. However, the optimal treatment strategy remains controversial because the exact etiology and pathophysiological mechanisms of the disease have not been established firmly. In addition, there are no publications examining the incidence of SIVAD in patients existing with acute abdominal pain based on contrast-enhanced computed-tomography (CE-CT) scans. We aimed to determine the incidence, characterize the pathogenesis, and suggest treatment strategies.

Abbreviations: SIVAD, spontaneous isolated visceral artery dissection without aortic dissection; CE-CT, contrast-enhanced computed-tomography; MDCT, multi-detector computed-tomography; CT, computed-tomography; SMA, superior mesenteric artery; CA, celiac artery; SAM, segmental arterial mediolysis

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2. Materials and methods

2.1. Study patients

This retrospective study in a single institution was conducted with approval from the ethics review board of the Nippon Medical School. At our institution, 51,057 abdominal CE-CT scans were performed between January 2005 and December 2016 excluding subsequent abdominal CE-CT scans of the same patients. Furthermore, the indication for 3237 abdominal CE-CT scans was for abdominal symptoms. A Picture Archiving and Communication Systems (PACS) database search for key words (arterial dissection, dissecting aneurysm) in the reports was performed using to extract SIVAD patients. Patients with concomitant aortic dissection, trauma, or iatrogenic causes were excluded [3–6,15,16].

2.2. Definition and measures

SIVAD patients were divided into two groups: asymptomatic (incidental findings) or symptomatic (absence of explanations other than dissection-related). The symptomatic group was divided into non-operative and endovascular groups (no patients had undergone surgery), based on the treatment received. Patient characteristics, incidence, vascular risk factors, medical histories, follow-up duration, length of hospital stay, change of symptoms, treatment strategies and morphology of the dissection in the CE-CT images and morphological changes were analyzed and compared between groups. Further, we classified the five types based on the CE-CT images (Fig. 1): type I (patent true and false lumen revealing entry and re-entry sites); type II (blind pouch of false lumen); type III (partial thrombosis false lumen); type IV (completely thrombosis false lumen); type V (completely thrombosis lumen).

2.3. Treatment selection

The asymptomatic group was observed without hospitalization. The symptomatic group was hospitalized in all cases, which continued until symptoms disappeared. Patients who presented with organ ischemia based on laboratory data and CT signs, and/or continuous abdominal pain received endovascular intervention. Patients without organ ischemia underwent nonoperative intervention.

2.4. Nonoperative intervention

Nonoperative intervention consisted of fasting with or without medication. Fasting was continued until abdominal pain disappeared. In cases with constriction in the true lumen, antiplatelet (daily oral administration of 75 mg clopidogrel, 200 mg cilostazol, or 100 mg aspirin administered alone or in combination) for 6 months more and/or prostaglandin E1 (daily intravenous administration of 20 mg), and in cases with thrombosis in the true lumen, anticoagulation (intravenous administration of low molecular weight heparin continuously for 24 h) were administered based on the CE-CT findings. Antihypertensives were administered to maintain the systolic blood pressure below 140 mm Hg [3,5–8,11,22,23].

2.5. Endovascular intervention

Angiography of the abdominal aorta was performed to detect collateral perfusion. Selective angiography was conducted to reveal the primary entry and extension of the dissection, perfusion of its main branches, and the distal flow. Bare stenting or coil embolization was performed, depending on the case. Patients who underwent endovascular stent placement were given intravenous low molecular weight heparin continuously for 3 days and were managed with antiplatelet drugs for 6–10 months.

2.6. Statistical analysis

Continuous variables are presented as mean values and SD. CE-CT morphologic features were analyzed using chi-square tests. Comparisons between the symptomatic and asymptomatic groups were performed using Mann-Whitney U tests. A p-value of < 0.05 was considered statistically significant.

3. Results

3.1. Patient characteristics and incidences

Patient characteristics are summarized in Table 1-a. Forty-seven patients (43 males, 4 females; mean, 62.8 ± 12.6 years; range, 35–88 years) were diagnosed with SIVAD. The dissection was located in the superior mesenteric artery (SMA) for 37 patients, and in the celiac artery (CA) for 10 patients. The SIVAD incidence was 0.09% among all abdominal CE-CT scans ($n = 51,057$ scans). Twenty-two of the SIVAD patients had acute abdominal symptoms, resulting in an incidence rate of 0.68% among all abdominal CE-CT scans taken for acute abdominal symptoms ($n = 3237$ scans). The other 25 patients had asymptomatic SIVAD and represented 0.05% among all abdominal CE-CT scans taken for reasons other than acute abdominal symptoms ($n = 47,820$ scans). The vascular risk factors among SIVAD patients were as follows: hypertension ($n = 21$, 44.7%); hyperlipidemia ($n = 11$, 23.4%); diabetes mellitus ($n = 8$, 17.0%); smoking ($n = 29$, 61.7%); Ehlers-Danlos syndrome ($n = 1$, 2.1%); and segmental arterial mediolysis (SAM) ($n = 1$, 2.1%). The medical histories among SIVAD patients were as follows: malignant disease ($n = 23$, 48.9%); myocardial infarction ($n = 4$, 8.5%); aortic aneurysm ($n = 4$, 8.5%); arteriosclerosis obliterans ($n = 3$, 6.4%); cerebral infarction ($n = 2$, 4.3%); atrial fibrillation ($n = 2$, 4.3%); gastrointestinal hemorrhage ($n = 1$, 2.1%); pneumothorax ($n = 1$, 2.1%); and chronic pancreatitis ($n = 1$, 2.1%).

3.2. Morphology of the dissection based on CE-CT images

CE-CT morphologic features are summarized in Table 1-b. The most frequent findings were intimal flap, thrombosed false lumen, and aneurysmal dilatation (≥ 1.5 times the normal diameter of perilesional arteries). In the symptomatic group, intimal flap ($n = 19$, 86.4%), thrombosed false lumen ($n = 11$, 50.0%), and aneurysmal dilatation ($n = 10$, 45.5%) were found. Other findings included branch vessel involvement ($n = 12$, 54.5%) and periarterial fat stranding ($n = 19$, 86.4%). In the asymptomatic group, intimal flap ($n = 23$, 92.0%),

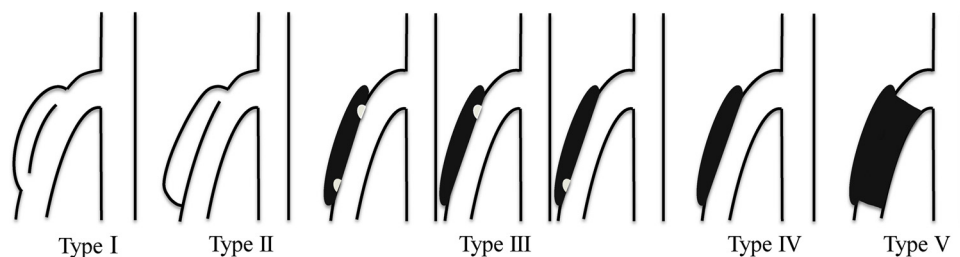


Fig. 1. Drawings illustrate the five types classification based on the abdominal contrast-enhanced computed-tomography for spontaneous isolated visceral artery dissection. Type I: patent true and false lumen revealing entry and re-entry sites; type II: blind pouch of false lumen; type III: partial thrombosis false lumen; type IV: completely thrombosis false lumen; type V: completely thrombosis lumen.

Table 1
Patient characteristics and morphology of the dissection on CE-CT images.

	All SIVAD = 47	Symptomatic SIVAD = 22	Asymptomatic SIVAD = 25
Sex (male : female)	n = 43 : 4	n = 19 : 3	n = 24 : 1
Age (mean ± SD)	62.8 ± 12.6 years old	58.3 ± 14.5 years old	66.7 ± 9.4 years old
Incidence (%)	0.09% (All abdominal CE-CT = 51,057 scans)	0.68% (Abdominal CE-CT for acute abdominal symptoms = 3,237 scans)	0.05% (Abdominal CE-CT for non-acute abdominal symptoms = 47,820 scans)
SMA : CA	n = 37 : 10	n = 17 : 5	n = 20 : 5
Vascular risk factors (%)			
Hypertension:	n = 41 (87.2%)	n = 19 (86.4%)	n = 22 (88.0%)
Hyperlipidemia:	n = 21 (44.7%)	n = 7 (31.8%)	n = 14 (56.0%)
Diabetes mellitus:	n = 11 (23.4%)	n = 6 (27.3%)	n = 5 (20.0%)
Smoking:	n = 8 (17.0%)	n = 3 (13.6%)	n = 5 (20.0%)
Ehlers-Danlos syndrome:	n = 29 (61.7%)	n = 14 (63.6%)	n = 15 (60.0%)
SAM:	n = 1 (2.1%)	n = 1 (4.6%)	n = 0 (0.0%)
	n = 1 (2.1%)	n = 1 (4.6%)	n = 0 (0.0%)
Medical histories (%)			
Malignant disease:	n = 23 (48.9%)	n = 4 (18.2%)	n = 18 (72.0%)
Myocardial infarction:	n = 4 (8.5%)	n = 2 (9.1%)	n = 2 (8.0%)
Aortic aneurysm:	n = 4 (8.5%)	n = 0 (0.0%)	n = 4 (16.0%)
Arteriosclerosis obliterans:	n = 3 (6.4%)	n = 1 (4.5%)	n = 2 (8.0%)
Cerebral infarction:	n = 2 (4.3%)	n = 1 (4.5%)	n = 1 (4.0%)
Atrial fibrillation:	n = 2 (4.3%)	n = 1 (4.5%)	n = 1 (4.0%)
Gastrointestinal hemorrhage:	n = 1 (2.1%)	n = 1 (4.5%)	n = 0 (0.0%)
Pneumothorax:	n = 1* (2.1%)	n = 1* (4.5%)	n = 0 (0.0%)
Chronic pancreatitis:	n = 1 (2.1%)	n = 0 (0.0%)	n = 1 (4.0%)
	All SIVAD = 47 (SMA : CA = 37 : 10)	Symptomatic SIVAD = 22 (17 : 5)	Asymptomatic SIVAD = 25 (20 : 5)
CE-CT findings (%)			
Intimal flap:	n = 42 (89.4%)	n = 19 (86.4%)	n = 23 (92.0%)
Thrombosed false lumen:	n = 19 (40.4%)	n = 11 (50.0%)	n = 8 (32.0%)
Aneurysmal dilatation:	n = 18 (38.3%)	n = 10 (45.5%)	n = 8 (32.0%)
Branch vessel involvement:	n = 14 (29.8%)	n = 12 (54.5%)	n = 2 (8.0%)
Periarterial fat stranding:	n = 19 (40.4%)	n = 19 (86.4%)	n = 0 (0.0%)
Distance from the orifice to the intimal flap (mean ± SD mm)			
SMA:	20.2 ± 13.8 mm	20.9 ± 14.7 mm	9.1 ± 12.5 mm
CA:	9.3 ± 3.0 mm	9.4 ± 2.8 mm	9.2 ± 3.9 mm
Length of the dissection (mean ± SD mm)			
SMA:	37.2 ± 20.1 mm	46.6 ± 22.8 mm	27.9 ± 11.5 mm
CA:	19.3 ± 6.3 mm	23.9 ± 4.3 mm	13.6 ± 1.1 mm
Our CE-CT classifications (SMA : CA)			
Type I:	n = 9 (24.3%) : 1 (10.0%)	n = 2 (11.8%) : 0 (0.0%)	n = 7 (35.0%) : 1 (20.0%)
Type II:	n = 11 (29.8%) : 7 (70.0%)	n = 5 (29.4%) : 4 (80.0%)	n = 6 (30.0%) : 3 (60.0%)
Type III:	n = 12 (32.4%) : 2 (20.0%)	n = 7 (41.2%) : 1 (20.0%)	n = 5 (25.0%) : 1 (20.0%)
Type IV:	n = 4 (10.8%) : 0 (0.0%)	n = 2 (11.8%) : 0 (0.0%)	n = 2 (10.0%) : 0 (0.0%)
Type V:	n = 1 (2.7%) : 0 (0.0%)	n = 1 (5.8%) : 0 (0.0%)	n = 0 (0.0%) : 0 (0.0%)

SIVAD, spontaneous isolated visceral artery dissection; CE-CT, contrast-enhanced computed-tomography; SMA, superior mesenteric artery; CA, celiac artery; SAM, segmental arterial mediolysis. *Ehlers-Danlos syndrome.

thrombosed false lumen (n = 8, 32.0%), and aneurysmal dilatation (n = 8, 32.0%) were found. The asymptomatic group had significantly fewer patients with periarterial fat stranding (n = 0, 0.0%) or branch vessel involvement (n = 2, 8.0%) (p < 0.01). The mean distance from the orifice of the SMA to the intimal flap in the symptomatic group (mean, 20.9 ± 14.7 mm; range, 0–50.1 mm) was not significantly different from that in the asymptomatic group (9.1 ± 12.5 mm, 0–44.8 mm) (p > 0.05). The distance for the CA in the symptomatic group (9.4 ± 2.8 mm, 7–13.3 mm) was not significantly different from that in the asymptomatic group (9.2 ± 3.9 mm, 6–13.5 mm) (p > 0.05). The mean length of the dissecting SMA was longer in the symptomatic group (46.6 ± 22.8 mm, 15.3–101.2 mm) than in the asymptomatic group (27.9 ± 11.5 mm, 12.2–40.7 mm) (p < 0.05). The mean length of the dissecting CA was 23.9 ± 4.3 mm

(18.3–29.6 mm) in the symptomatic group and 13.6 ± 1.1 mm (12.3–15.0 mm) in the asymptomatic group (p < 0.05). No significant differences in the morphology of the dissection in the CE-CT images were found between the nonoperative and endovascular groups (p > 0.05). Our classification based on the CE-CT images were below: type I (SMA = 2, CA = 0 in symptomatic group; SMA = 7, CA = 1 in asymptomatic group); type II (SMA = 5, CA = 4 in symptomatic; SMA = 6, CA = 3 in asymptomatic); type III (SMA = 7, CA = 1 in symptomatic; SMA = 5, CA = 1 in asymptomatic); type IV (SMA = 2, CA = 0 in symptomatic; SMA = 2, CA = 0 in asymptomatic); type V (SMA = 1, CA = 0 in symptomatic; SMA = 0, CA = 0 in asymptomatic).

Table 2
Treatment categories and the course of treatment.

	All SIVAD = 47	Symptomatic SIVAD = 22	Asymptomatic SIVAD = 25
Hospitalization			
No:	n = 25	n = 0	n = 25
Yes (mean ± SD days):	n = 22	n = 22 (12.8 ± 7.1 days)	n = 0
Conversion symptoms		Organ ischemia = 1 Developed organ ischemia or/and persistent symptoms = 5 Disappeared symptom = 16	None = 25
Treatment contents			
Untreated:	n = 25	n = 0	n = 25
Fasting only:	n = 6	n = 6	n = 0
Fasting and drugs*:	n = 10	n = 10	n = 0
Endovascular:	n = 6	n = 6	n = 0
Surgery:	n = 0	n = 0	n = 0

*anticoagulants ,antiplatelet, prostaglandin and/or antihypertensive

3.3. Clinical management of patients

The treatment categories and course of treatment are summarized in Table 2. To prevent intestinal ischemia, all the 22 symptomatic patients were hospitalized and instructed to fast. Sixteen symptomatic patients underwent nonoperative intervention only and except one patient who died of sepsis due to aspiration pneumonia, all could resume eating and were discharged after the disappearance of symptoms. A bare-stent was implanted in one patient (type V) presenting with organ ischemia at diagnosis (Fig. 2) and five patients (n = 1 in type I; n = 3 in type II; n = 1 in type III) who developed organ ischemia after hospitalization (progress of the dissection in CE-CT images and progressive/persistent abdominal symptoms). For bare-stent placement, navigating bare-stent

(S.M.A.R.T. CONTROL®, Cordis Corporation, New Jersey, USA) via a 6-French guiding-catheter (Destination®, Terumo Clinical Supply Co. Ltd., Tokyo, Japan) was deployed over a 0.035-inch guide-wire (Amplatz Super Stiff™, Boston Scientific Corporation, Boston, USA) into the true lumen. The remaining one patient underwent coil embolization. Because the diameter of entry and re-entry was large, we speculated that it is difficult to reduce the blood flow in the false lumen even if the bare-stent is implanted. For coil embolization, a detachable coil (Interlock™ Fibered IDC™, Boston Scientific Corporation, Massachusetts, USA) was placed in the false lumen of the SMA via the bilateral femoral approach under micro-balloon (Attendant®, Terumo Clinical Supply Co. Ltd.) occlusion at the point of re-entry and entry through the true lumen to prevent coil migration [12,20]. All patients undergoing endovascular

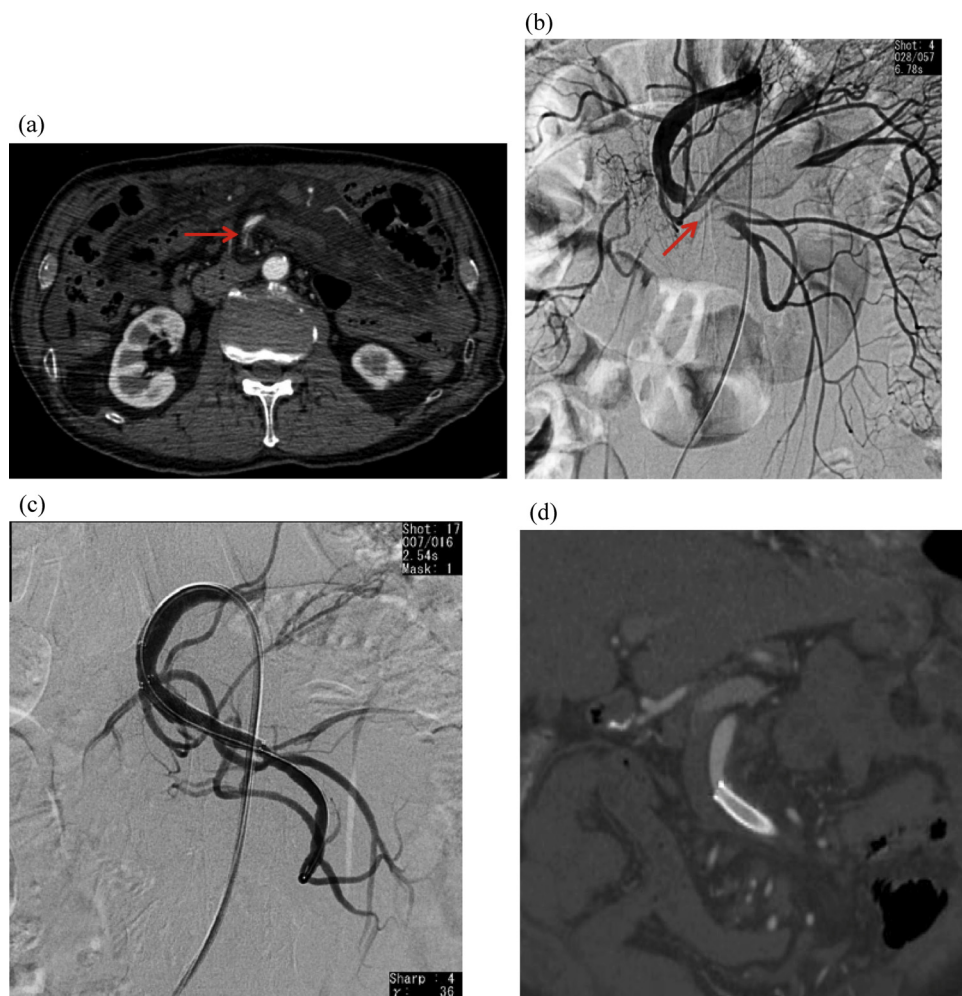


Fig. 2. Case 1 – A 73-year-old man with dissection of the superior mesenteric artery (SMA) had symptoms of severe abdominal pain and vomiting. The intestinal tract was ischemic at diagnosis. (a) Axial contrast-enhanced computed-tomography (CE-CT) scans through the upper abdomen showed that the false lumen was thrombosed and the true lumen was retracted by the false lumen (arrow). The intestinal tract exhibited edema. (b) Digital subtraction angiography showed an occluded region ~3 cm in length above the SMA (arrow). (c) Bare-stent (φ6 × 40 mm S.M.A.R.T. CONTROL®) placement in the narrowing true lumen is shown. Blood flow in the true lumen of the stent and peripheral artery was maintained. (d) Eighteen months after bare-stent placement, coronal CE-CT scans revealed the patency of the stent.

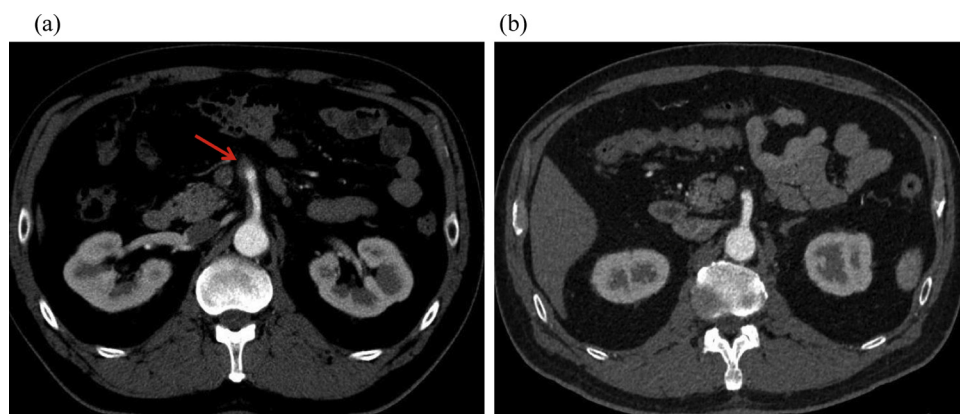


Fig. 3. Case 2 – In a 39-year-old man with dissection of the superior mesenteric artery, abdominal symptoms disappeared after non-operative intervention only. (a) At diagnosis, the false lumen was thrombosed and the true lumen was narrowed and retracted by the false lumen (arrow). (b) Four months after discharge, the thrombosed false lumen and dissection were completely remodeled.

intervention were administered anticoagulants and/or antiplatelet drugs, could resume eating, and were discharged after the disappearance of symptoms. The average hospital stay for symptomatic patients was 12.8 ± 7.1 days. All asymptomatic patients were untreated.

3.4. Ambulatory follow-up

Ambulatory follow-up included assessment of abdominal symptoms and changes in the CE-CT images. The mean follow-up period was 925.1 ± 383.0 days for the symptomatic group ($n = 21$: 1 patient died of sepsis during hospitalization) and 710.4 ± 737.6 days for the asymptomatic group. Abdominal symptoms, organ ischemia, and dissection-related complications were absent in both the symptomatic group after discharge and the asymptomatic group. In the endovascular group, CE-CT scans revealed patent stents and complete remodeling of the dissections in all patients. In patients who underwent stenting, the mean time for complete remodeling was 5.8 ± 4.0 months. In the nonoperative group, CE-CT scans showed improvement in the dissection lesions in 13 (86.7%) patients, and complete remodeling (Fig. 3) in 2 (13.3%, type IV). Periarterial fat stranding and branch vessel involvement disappeared completely in the symptomatic patients. In the asymptomatic group, CE-CT showed improvement in the dissection lesions in 1 (4.0%) patient, no change in 22 (88.0%), and complete remodeling in 2 (8.0%, type IV). No patients showed dissection progression or dissection of other arteries in both groups. None of patients died of SIVAD.

4. Discussion

SIVAD has been reported frequently in carotid and renal arteries, but rarely in the visceral arteries such as the celiac or hepatic artery [2–16,20,22]. This study examined a larger patient population, clinical follow-up, and treatment strategies from a single institution. This is the first report of SIVAD incidence based on CE-CT scans among living cases.

Previous studies have documented that 91.3% of SIVAD occur in men with an average age of 50.0 [8]. This is nearly identical to the findings in our study. The 0.09% incidence is similar to 0.06% reported in a postmortem [19]. This result suggests that SIVAD is rare; a consensus regarding the pathology and optimal therapy is lacking. The pathogenesis was unknown with the exception of Ehlers-Danlos syndrome and SAM. Some investigators have associated SIVAD with hypertension, atherosclerosis, cystic medial necrosis, fibromuscular dysplasia, and connective tissue disease [3,19]. Hypertension may be a predisposing factor; however, no data supporting its role in causing intimal tear. Hypertension and smoking were common and relatively higher than other suspected risk factors; 87.2% patients were reporting at least one of these factors. In cases where the CA is stenotic or

occluded by arteriosclerosis and median arcuate ligament, the compensatory increase in flow in the SMA may lead to increased shear stress [2,10]. This may be a possible mechanism for the dissection. However, the CA stenosis by the median arcuate ligament was found in only four cases and arteriosclerosis in only one. Despite the absence of obvious arterial diseases in 85.1% patients, dissection had occurred. The following can be considered as a cause of this. Anatomically, 10.0–30.0 mm from the orifice of the SMA between the fixed retropancreatic portion and the mobile portion is a weak point [11]. The mean distance from the orifice of the SMA to the intimal flap is concordant with this weak point. All of the CAs ran to the caudal and then upside-down to the cranial side. Dissections were located at the portion of the inversion to the cranial side from the caudal side. Turbulent flow can also lead to weakening the arterial wall by increased shear stress. These results suggest that increased shear stress and anatomical weakness are directly involved in pathogenesis. The majority of asymptomatic SIVAD patients had malignancy history and may be affected by some kind of malignancy. The malignancy itself and cytokines released from itself as well as treatments for the malignancy (surgery, radiotherapy, and chemotherapy) may probably play a role in the development of SIVAD.

The periarterial fat stranding, length of the dissection and branch vessel involvement might predict the severity of symptoms. The periarterial fat stranding reflects inflammation, which stimulates the visceral nerve plexus causing symptoms. It is speculated that longer dissection is, the more visceral nerve plexus are stimulated and the more branch vessels are involved.

The classification of Sakamoto et al. [2] did not include completely thrombosis lumen, and Yun et al. [6] did not include partial thrombosis false lumen. So, we classified five types combining their classifications and also applied to the CA. Our classification was not significantly different between the symptomatic and asymptomatic group, and also between the SMA and CA. However, important signs were found in the symptomatic SIVAD: progress of dissection in type I; rapid expansion of blind pouch in type II; progress of partial flow in false lumen in type III; and confirmation of ischemia in type V. These changes were associated with the symptoms of ongoing and unresolving bowel ischemia. As a resulting, endovascular intervention was performed in cases appearing these signs. In type IV, complete remodeling had occurred regardless of presents of symptoms.

The optimal treatment has not been established, may involve non-operative and surgical or endovascular intervention, depending on clinical features. Endovascular intervention with good outcomes has become popular for patients with persistent symptoms and developing ischemia or necrosis because of comparable outcomes with surgical intervention [2,4–8,10–15,21,24,25]. We could perform life-saving treatment with endovascular interventions. Therefore, endovascular has become the first choice at our institution due to minimal invasiveness.

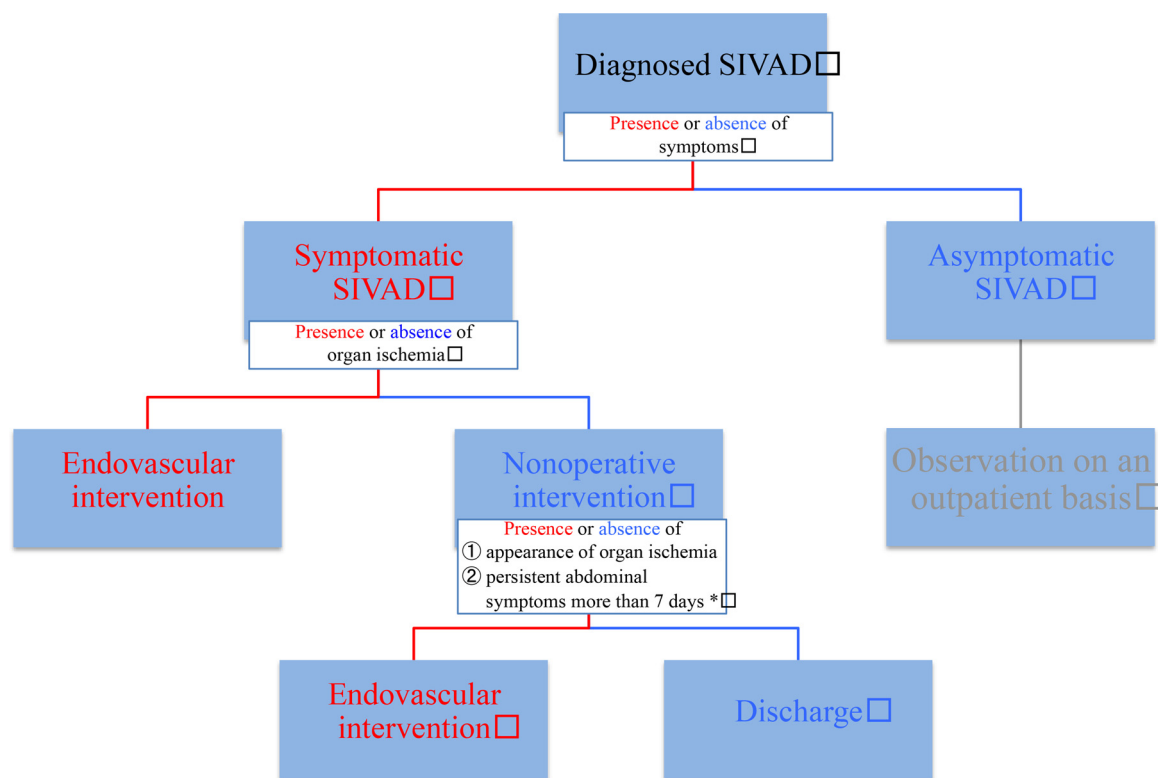


Fig. 4. Treatment strategy – A proposed strategy for treatment based on our results is shown. SIVAD, spontaneous isolated visceral artery dissection * Surgical or endovascular intervention recommended.

The hospitalization and drug treatments have been widely discussed. The results suggest that asymptomatic SIVAD patients do not require hospitalization since there was no progression of the pathology to visceral ischemia, whereas symptomatic should be hospitalized because of the possible progression of the problem to visceral necrosis. To prevent thromboembolic complications, some authors have suggested 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 [3,5–8,22,23]. This argument for anticoagulation is based on reports published before 1970 in which nonoperative intervention without anticoagulation led to necrosis. However, no evidence exists supporting the anticoagulation. Anticoagulation has usually been prescribed to prevent the distal thrombosis in spontaneous carotid artery dissection. Yun et al. [6] reported that the absence of anticoagulation or antiplatelet made no difference in the clinical outcomes. Thus, no consensus regarding drugs exists. In general, anticoagulant, antiplatelet, or prostaglandin are prescribed for a narrowing true lumen, while antihypertensives are for hypertension. However, it is impossible to completely prevent ischemia using these drugs. We observed 5 ischemia development patients and obtained the 23.8% nonoperative failure among symptomatic. This rate is similar to the 38.5% [24] and 33% [25] overall failure in spontaneous SMA dissections. In contrast, Takayama et al. [4] reported the 5.2% nonoperative failure. Given variable results, failure rates analysis is required. On the other hand, 76.2% symptomatic patients were managed successfully with nonoperative, and symptoms subsided within 7 days at the most (mean, 4.8 days). In contrast, all the symptomatic patients with nonoperative intervention failures had persisting symptoms for ≥ 7 days. Our proposed treatment strategy based on current results is summarized in Fig. 4.

Some limitations are that we were unable to obtain a prior history of acute abdominal pain from all of the asymptomatic patients, the pathological evidence was not obtained, and this retrospective study was

confined to a certain ethnic group. Treatments outcomes must be further examined in prospective studies.

5. Conclusion

Asymptomatic patients may be managed with observation on an outpatient basis because none of developed organ ischemia and progressed dissection. Persistent symptoms not relieved by nonoperative intervention (≥ 7 days) may be progression to ischemia, necrosis or aneurysm formation, necessitate the evaluation of CE-CT images based on the presence of signs in our classification: progress of dissection in type I; rapid expansion of blind pouch in type II; progress of partial flow in false lumen in type III. Therefore, symptomatic patients should be hospitalized. Endovascular intervention is a safe and feasible treatment for complications of SIVAD.

Conflicts of interest

The all authors have no potential conflicts of interest related with this article.

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