Isolated visceral arteries dissection: Report of three cases and literature review

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Abstract:

Spontaneous, isolated visceral artery dissection represents an infrequent clinical occurrence. Over the past decade, there has been a notable rise in reported cases within the medical literature. There is no optimal therapy for this condition, nevertheless, conservative therapy and close follow-up for uncomplicated cases is accepted. Computed tomography angiography is the diagnostic tool of choice, whereas blood tests usually have no specifical abnormalities. Nowadays, endovascular therapy is therapy of choice for complicated cases, especially where exploration of the abdomen is not necessary. We report three cases of successful conservative therapy and we present a literature review.

Keywords: visceral, dissection, spontaneous, isolated, celiac artery, superior mesenteric artery

INTRODUCTION

Isolated visceral artery dissection represents an infrequent clinical occurrence. Over the past decade, there has been a notable rise in reported cases within the medical literature. This increase is primarily ascribed to the growing utilization of computed tomography angiography (CTA), although it may also be linked to an overall elevation in the incidence of visceral artery dissections. The precise pathophysiology of this condition remains unclear. Common risk factors identified among affected individuals encompass smoking and hypertension, implying that atherosclerosis and heightened shear stress may significantly contribute to the pathophysiological mechanisms. In this context, we present three cases of isolated visceral artery dissection managed conservatively, accompanied by a comprehensive literature review elucidating the characteristics of this uncommon medical phenomenon. Informed consent has been obtained from the patients for publication of the case report and accompanying images. Also, approval was obtained from the local ethics committee.

CASE 1

A 52-year old woman presented to the emergency department complaining of abdominal pain radiating to her back. The pain was characterized as postprandial, commencing four weeks prior and intensifying over the last twenty-four hours. Clinical assessment disclosed a supple abdomen with generalized tenderness. The patient's medical history disclosed inadequately controlled arterial hypertension. Laboratory investigations demonstrated values within the normal range. CTA revealed a celiac artery (CA) dissection with an intimal flap originating approximately 1.2cm from the celiac artery ostium for a length of 1.7cm. (fig.1) Hepatic, splenic and gastric artery were patent with no signs of flow limitation, thrombosis, aneurysm formation or intestinal ischemia. Conservative management was initiated. Subsequently, the pain was effectively alleviated, and the patient has remained asymptomatic for a duration of two years. (fig.2)

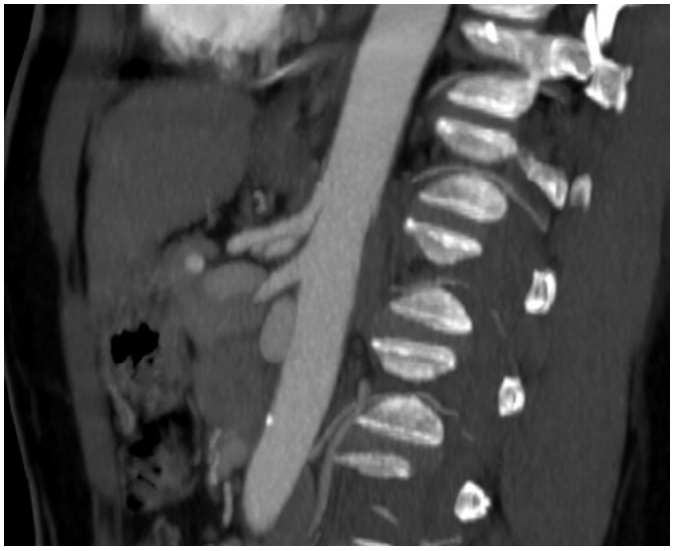

Fig. 1: Celiac artery dissection

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doi: 10.59037/me0nkk18
ISSN 2732-7175 / 2024 Hellenic Society of Vascular and Endovascular Surgery Published by Rotonda Publications

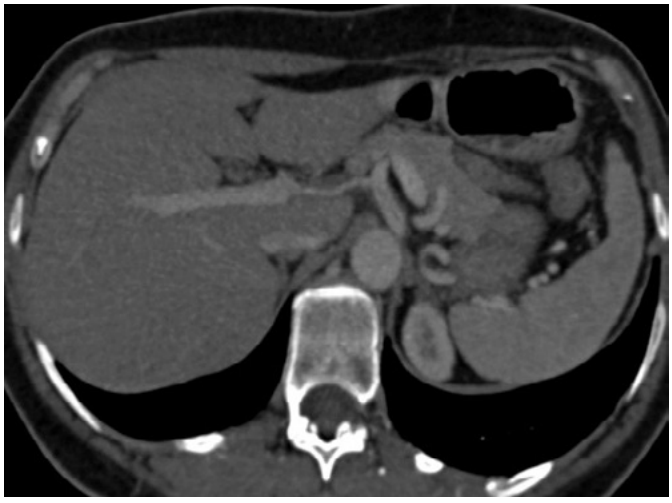

Fig. 2: Stable findings two years after discharge. CA dissection until trifurcation.

CASE 2

A-64-year-old man presented to the emergency department with recurrence of his abdominal pain and indigestion. He had been hospitalized two weeks prior at a different medical facility, where he received treatment involving antibiotics and proton pump inhibitors. The patient's medical background comprised a history of hypertension, coronary artery disease, and diabetes mellitus. Physical examination revealed a diffused, mild abdominal tenderness. Platelets and white blood count deviated from the normal range. X-rays and ultrasonography were normal. CTA revealed stenosis at the celiac artery ostium with post-stenotic dilatation (fig.3) as well as a dissection of superior mesenteric artery (SMA) with a flap originated at 1.2cm from its ostium, with a length of 8.5cm and re-entry point with patent branches of the SMA. (fig.4) The patient was successfully managed conservatively. A CT scan performed three months post the incident exhibited consistent and unchanging results.

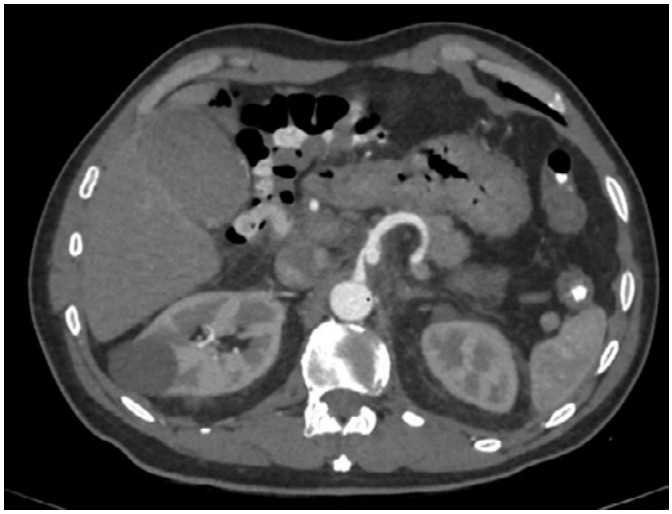

Fig. 3: CA post stenotic dilatation

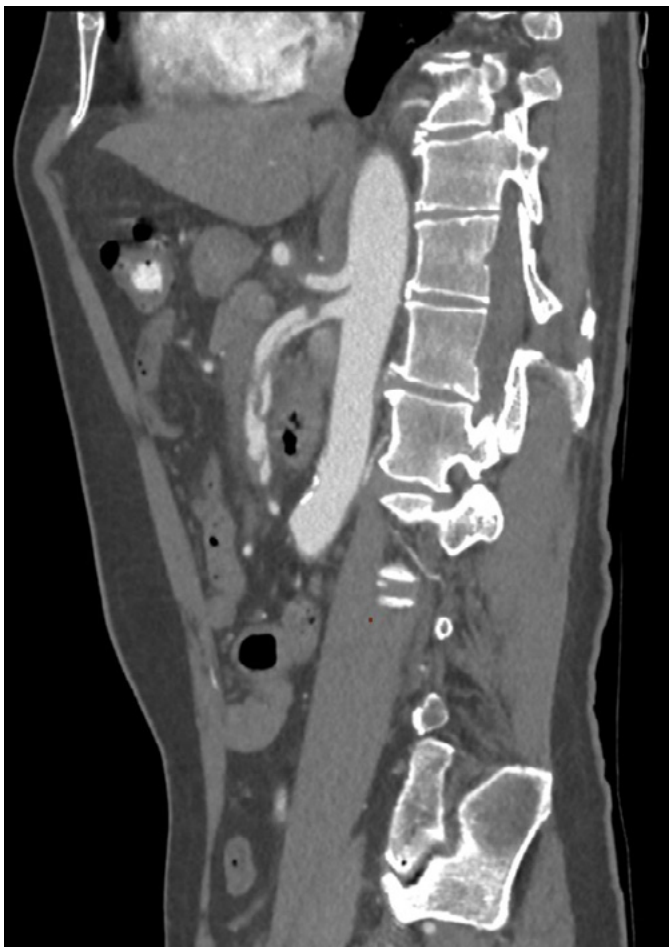

Fig. 4: SMA dissection (length ~8cm), entry and re-entry points

CASE 3

A 51-year-old man presented to the emergency department reporting acute abdominal and left flank pain. The patient's medical history encompassed inadequately controlled hypertension, dyslipidaemia, and a history of tobacco use. CTA revealed dissection of the celiac artery and flow limitation to its branches. The patient was admitted to the hospital and was successfully treated conservatively, including bowel rest, antiplatelet and anticoagulation therapy. Ten days later, he underwent a repeated CTA which revealed patent left gastric artery. (fig.5) He remained asymptomatic, with stable CT scan findings even 7 years after this incident.

All three patients were treated conservatively in the acute phase and the therapy included anticoagulation, antiplatelet, b-blocker medications and bowel rest. Enteral nutrition was incrementally introduced starting from the sixth day of their hospitalization . Upon discharge, they were prescribed antiplatelet agents, and the follow-up period ranged from three months to 7 years.

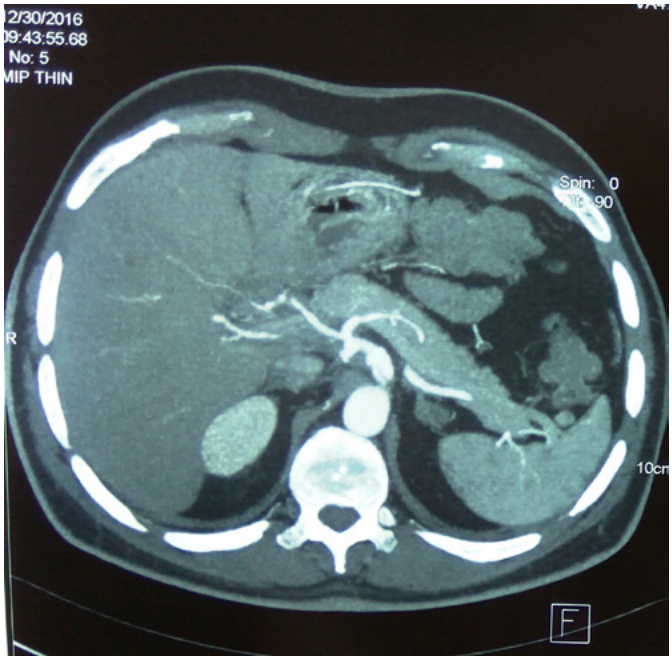

Fig. 5: Ct scan before discharge, CA dissection, patent left gastric artery

DISCUSSION

Spontaneous dissection of visceral arteries was traditionally considered an exceedingly rare condition, given an estimated incidence of approximately 0.08% whereas synchronous dissection of more than one visceral artery is even more extraordinary.[1-7] A comprehensive review of published articles within the PubMed database (2012-2022) was conducted to investigate isolated visceral artery dissection. The search yielded information on more than 600 cases. The augmented identification of spontaneous visceral artery dissection is attributed either to a genuine increase in the prevalence of such cases or to the enhanced detectability, facilitated by contemporary imaging modalities.[8] The demographic profile of affected individuals typically comprises middle-aged, Asian men. Upon presentation, these patients commonly exhibit a history of smoking and hypertensive urgency.[2,4,9-12] The superior mesenteric artery is most often affected, with the celiac artery following as the next most commonly affected vessel.[10,13,14]

Bax et al, in a very interesting article, discuss on pathophysiology of arterial dissections.[15] Various factors including hypertension, age, gender, connective tissue diseases, atherosclerosis, arterial cystic necrosis, trauma and smoking are considered to be associated with the disease. An intriguing pathophysiologic mechanism was suggested by Wu et al, proposing that SMA is more susceptible to shear stress in the transition zone from fixed retropancreatic to relatively mobile segment in the mesenteric root, analogous to what is seen at the ligamentum arteriosum in thoracic aortic dissection.[16] Celiac artery dissection should include investigation for median arcuate ligament syndrome.[17-19] An additional finding identified in this study, is that pancreatic enzymes released during pancreatitis can erode adjacent arterial wall and thus, the presence of acute pancreatitis might have triggered arterial dissection.[6,20]

Clinical presentation most often includes acute abdominal or flank pain. However, it could be present in a more indolent fashion, with an insidious onset, lasting days or weeks before the pain becomes more severe.[5] Yamaguchi et al reported a case where a patient presented with acalculous cholecystitis as a result of hepatomesenteric trunk dissection.[21] The pain can be postprandially or irrelevant with food intake, and the character may be sharp or dull. Some patients remained totally asymptomatic, and they were diagnosed incidentally or they were misdiagnosed.[5,14,19,22] Abdominal pain may be implying bowel ischemia, perforation and peritonitis or aneurysmal formation with imminent rupture of the artery. Nevertheless, the abdominal pain could alternatively be explained as an inflammatory response triggered by the dissection, thereby provoking pain through stimulation of the visceral nerve plexus. Current literature supports that the degree of pain is positively correlated with the length of the dissected blood vessel.[23] An author suggests that there were no significant differences in medical history or medications between symptomatic and asymptomatic patients. However, patients with abdominal symptoms tended to be younger and were more frequently hospitalized.[24] Nevertheless, in another study, it was noted that asymptomatic patients were younger (53.9±11.4 vs 58.7±11.2, p = 0.032) and that no significant differences were presented between the artery which was involved (CA vs SMA) in patients with or without symptoms, however there was a trend towards SMA involvement in symptomatic patients (23 (46%) vs 7 (26%), p = 0.085).[25]

The literature documents that many patients were misdiagnosed. Radiologic modalities are determinant of an accurate diagnosis.[26] CT angiography is the preferred diagnostic tool, as it is a rapid, non-invasive, and high resolution examination, which contributes to the visualization of the vessels and of complications from abdominal organs such as necrosis and perforation.[5,13] Regarding laboratory blood tests, coagulation markers, such as fibrin degradation products, are known to increase in acute aortic dissection. However, these markers were not markedly elevated in this condition, even in symptomatic patients, possibly because the amount of thrombus in SIVAD is smaller, due to the size of the vessel.[24] According to the same study, there were significant differences between symptomatic and asymptomatic patients in white blood cell count and creatine kinase levels, but not in FDPs or d-dimer levels.[24]

There are no specific guidelines regarding the ideal treatment of visceral dissection. However, a reasonable algorithm is the following: Surgery (open/endo/hybrid) is recommended if the patient in the acute phase presents with rupture, signs of end-organ ischemia, enlargement of the artery (>2cm) or blood flow limitation, in correlation with pain not responding to medication. If none of these conditions exists, the physician can choose conservative therapy (medication, bowel rest). Our strategy includes bowel rest for 5 to 6 days, staged nutrition, anti-hypertensive medication, antiplatelet therapy, low molecular weight heparin at a prophylactic dose, repeated

laboratory blood tests including arterial blood gases, and CT angiography on the day of the admission, before discharge, at 3 and at 12 months after patients' discharge. After the first year, patients are followed up with duplex scan in turn with CT scan to avoid exposure to radiation. Although there is an ongoing debate regarding the efficacy of antithrombotic therapy (anticoagulation and antiplatelet medication), studies revealed that they do not demonstrate any advantages in terms of clinical or morphological outcomes.[7,27] Endovascular therapy includes bare metal stent, coil assisting bare stent therapy, coil embolization[3,6,18,28,29], whereas open surgical therapy is preferred in cases where exploration of the abdomen is mandatory. Patch angioplasty or bypass is still an option, but endovascular therapy is the preferred method because of its high technical success and low complication rate.[30] Nowadays, hybrid approach is almost always available, however there is still no case report announced in Pubmed database.

Regarding prognosis, dissection can progress in various ways. It may exhibit a self-limited course with symptom resolution, progress to involve distal branches, develop into aneurysmal dilatation, or, in more severe cases, culminate in rupture.[19] Current literature supports that approximately 20% of patients who were treated conservatively, developed aneurysmal dilatation requiring intervention during the follow-up period.[31,32] In a review by Wang it is reported that 8% of the symptomatic celiac artery dissection patients and 12% of the symptomatic superior mesenteric artery dissection patients who were managed conservatively, required secondary intervention during follow-up, whereas none of the asymptomatic patients needed further intervention.[33] Superior mesenteric artery seems to fail to achieve complete remodeling, and therefore, it is correlated with more complications.[33] Moreover, comparing visceral artery dissection with renal artery dissection it can be supported that the latter has worst prognosis, since it is correlated with increased complications and mortality.[34,35] Patients who have visceral artery dissection with otherwise normal appearing arteries carry a higher risk of major adverse arterial events compared with those with fibromuscular dysplasia, primarily because of recurrent dissections.[36,37]

In conclusion, given the heightened frequency of case series and reports in the literature over the past decade, one may infer a probable contemporary escalation in the prevalence of isolated visceral artery dissection. This condition poses challenges in the emergency department where its symptoms, encompassing abdominal and back pain, are commonplace, and clinical presentation may mimic other gastrointestinal or musculoskeletal disorders, potentially leading to misdiagnosis. While conservative management during the acute phase is often feasible, further comprehensive data on mid and long-term outcomes and management are needed.

ACKNOWLEDGMENTS

None.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

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author	year	country	patients	age	gender	symptomatic/ asymptomatic	vessel	risk factors	therapy	in hospital regimen	home prescription	fup
Aciu et al	2022	USA	1	57	male		CA extended to splenic (Segmental splenic infarction)	hypertension	conservative	antihypertensive + dual antiplatelet	antihypertensive + antiplatelet	6 months
Yamaguchi et al	2022	Japan	1	48	male		hepatomesenteric trunk (acalculous cholecystitis)	none	conservative	n/a	n/a	n/a
Ialani et al	2021	India	1	60	male		CA, SMA	hypertension	conservative	hep 5000u three times daily	anticoagulation	3 months
He et al	2021	China	27	24-77	24 male, 3 women		16 sma, 6 abdominal, 3 splenic, 2 renal	n/a	n/a	n/a	n/a	n/a
Yamaguchi et al	2021	Japan	1	80	male		SMA	hypertension	false lumen coiling	n/a	n/a	
Gao et al	2021	China	28	43-68	23 male, 5 women		CA, SMA	smoking, hypertension, drinking, diabetes, cancer, gastrointestinal, cerebrovascular disease	Bare metal stents	anticoagulation + antiplatelet	anticoagulation + antiplatelet	3-77months
Mkangala et al	2020	Chine	62	52.55 ±7.22	83.9% male		SMA	hypertension 41.9% (n = 26, atherosclerosis 41.9% (n = 26), smoking 25.8 (n = 16)	14 concervative, 48 endovarscular	hep 1mg/kg twice daily and aspirin 100mg	dual antiplatelet in endovascular group	28.76 ±12.87
Yokoyama et al	2020	Japan	1	41	male		SMA, renal	hypertension	conservative, endovascular	anticoagulation + antiplatelet	antiplatelet	6 months
Shiraki et al	2020	Japan	36 (2010-2016)	mean age 54.6	29male/7female		26 SMA, 9 CA, 2 splenic, 1 common hepatic, 1 gastroduodenal, 1 left gastric	hypertension, smoking	94.4% conservatively, 5.6% endovascularly	anticoagulation + antiplatelet	n/a	n/a
Hoglund et al	2020	USA	1	54	male		CA extended to common hepatic, proper hepatic, right and left hepatic arteries, as well as the proximal gastroduodenal artery	hypertension	conservative	anticoagulation	apixaban	3 months
Moran et al	2020	USA	1	middle aged	male		CA, 2 renals	variant of transforming growth factor beta receptor 1 gene (TGFBR1).	Bare metal stents	anticoagulation	n/a	5 months
Komem et al	2019	Israel	1	51	female	symptomatic	CA, splenic, IMA, right renal , both external iliac, right internal carotid artery	COL3A1 subtype mutation (c.3199A>T, Ser1067Cys variant), smoking, migraines	endovascular (Stenting)	anticoagulation	dual antiplatelet	12 months
Jeong et al	2018	Korea	23+40 (2010-2016)	48 (25-82)	91% male	symptomatic	23 renals (results compared with 40 SMA)	Diabetes mellitus 13%, Hypertension 17%, Smoking 70%	35% endovascular (stent, coil, stent+coil)	iv heparin 78%	warfarin for 6 months	median fup 20 months (range 0-63)
Yamaguchi et al	2018	Japan	47 (2005-2016)	mean, 62.8 ± 12.6 years; range, 35-88 years	43 male, 4female	22 pts symptomatic, 25 asymptomatic	SMA 37, 10 CA	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%)	conservatively, 1 pt endo, 5 pts endo during fup	antiplatelet or anticoagulation, prostaglandin E1	antiplatelet 6-10 months	mean follow-up 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
Morgan et al	2018	USA	77 (2006-2016)	56 years (range 26 - 86)	80% male	64% symptomatic, 36% asymptomatic	CA, SMA, 18% combined CA+SMA	13% connective tissue disorder	4 pts open surgical repair,1 pt stenting, the rest conservatively	anticoagulation 52%, 30% antiplatelet, 13% observation	3-5 months anticoagulation, indefinite antiplatelet	mean fup 21 months
Tanaka et al	2018	Japan	39 (2007-2016)	median age 52	94.9% male		CA, SMA	hypertension (48.7%), smoking (79.5%)	32pts conservative, 7pts open or endo	anticoagulation or antiplatelet	n/a	median fup 11 months
Heo et al	2017	Korea	116 (2001-2016)	mean age, 54.7±10.8 years	92% male	76% symptomatic	SMA	hypertension, smoking, connective tissue disorder, cancer	83pts conservatively, 6 open surgical repair or endovascularly	anticoagulation or antiplatelet or none	n/a	53±39 months (range:1-173 months)
Otsuka et al	2017	Japan	32 (2005-2015)	mean age 54 years (30-85)	28male, 4 female	symptomatic	10 CA, 15 SMA, 5 CA+SMA	13 pts hypertension	29 pts conservatively, 1pt open surgical repair, 3pts embolization	six pts took heparin 10,000 unit/day	no antiplatelet/ anticoagulation	3.8 ±2.6 (1-10) years
Melnychuk et al	2017	USA	1	37	male		CA extented to hepatic artery	hypertension, smoking, possible fibromuscular dysplasia	conservative	heparin infusion	antiplatelet	2 months
Binh Le et al	2016	Korea	1	55	male		CA, SMA, both renals	hypertension	conservative (CA,renal) + endovascular (stenting SMA, 1 renal)	heparin infusion	anticoagulation + antiplatelet	84 months
Nonami et al	2016	Japan	56 (2004-2015)	54 years (range, 32-86 years)	89.3% male		40 SMA, 16 CA	hypertension, smoking, DM, ischemic disease, other visceral dissection, dyslipidemia	conservative, 3 endo, 2 open	12.5% anticoagulation, 0% antiplatelet		median fup 22.5 months (1-112)
Su et al	2016	Australia	1	43	female		hepatic artery	fibromuscular dysplasia, hypertension	conservative	dual antiplatelet	dual antiplatelet	four months
Ichiba et al	2016	Japan	1	51	female		left gastric	hypertension	pseudoaneurysm coil embolization	none	none	6 months
Sun et al	2016	China	23 (2009-2014)	median age 50 (30-82)	83%male, 17% female		CA	48% hypertension, 48% smokers	15 endo (stenting or embolization), 8 conservatively	n/a	n/a	mean fup 21 months (6-70 months)
Hee Ko et al	2015	USA	23 (2005-2014)	58.4 (31-80)	78%male	78% symptomatic	CA, SMA	65.2% hypertension, Hyperlipidemia 56.5%, Smoking 21.7%, Diabetes 8.7%	observation in 4, anticoagulation in 13, endovascular stenting in 6 patients	13 pts anticoagulation or antiplatelet	antiplatetel those treated	23.8 months
Alcantara et al	2015	USA	10 (2009-2013)	mean age 61.5 ± 10.3 (range 41-77 years)	90% male	6pts asymptomatic, 4 symptomatic	CA,SMA	hypertension, smoking,	observation without anticoagulation in 8 pts (80%), anticoagulation in 1 pt (10%), endovascular stenting in 1 pt (10%)	antiplatelet	antiplatelet	fup 14.7 ± 11.6 (1-31 months)
Galastri et al	2015	Brazil	10 (2009-2014)	44.8 (35-55)	90%male	symptomatic	CA	hypertension 40%, polyarteritis nodosa 30%	conservative 9pts , 1 pt stent + coil embolization	anticoagulation	anticoagulation (+antiplatelet in 5 pts)	mean fup 19 months (2-59)
Black et al	2014	UK	1	51	male	symptomatic	CA	none significant	conservative	n/a	n/a	n/a
Perini et al	2013	Brazil	4 (2010-2011)	mean age, 51 years; range, 43-65 years	male	symptomatic	CA	n/a	coil embolization, plug	n/a	n/a	median, 4.5 months; range, 3-24 months