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Median Arcuate Ligament Syndrome: A Single-Center Experience with 23 Patients

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Abstract
Background Median arcuate ligament syndrome (MALS) is a rare entity that occurs when the median arcuate ligament of the diaphragm is low-lying, causing a compression to the underlying celiac trunk. We reviewed the vascular changes associated with MALS in an effort to emphasize the seriousness of this disease and the complications that may result.
Methods This is a retrospective descriptive analysis of 23 consecutive patients diagnosed with MALS between January 1, 2012 and December 31, 2015 at a tertiary medical center. Computed tomographic (CT) scans, medical records, and patient follow-up were reviewed.
Results The number of patients included herein was 23. The median age was 56 years (17–83). Sixteen patients (69.6%) had a significant arterial collateral circulation. Eleven patients (47.8%) were found to have visceral artery aneurysms; 4 patients (36.4%) bled secondary to aneurysm rupture. All ruptured aneurysms were treated with endovascular approach. The severity of the hemodynamic changes appears to be greater with complete occlusion,
Conclusions MALS causes pathological hemodynamic changes within the abdominal vasculature. Follow-up is advised for patients who develop a collateral circulation. Resulting aneurysms should preferably be treated when the size ratio approaches three. Treatment of these aneurysms can be done via an endovascular approach coupled with possible celiac artery decompression to restore physiologic blood flow.

Keywords Visceral aneurysms · Embolization · Median arcuate ligament

Introduction

Median arcuate ligament syndrome (MALS) is a rare challenging entity. It occurs when the median arcuate ligament of the diaphragm, which consists of connective tissue fibers bridging the diaphragmatic crura, is low-lying (Fig. 1). This compresses the underlying celiac trunk compromising blood flow [1]. The diagnosis can be made using thin-section multi-detector computed tomography (CT) scans which delineate the ligament and the compressed vessel [2].

The majority of patients are asymptomatic. However, when symptoms exist they are typically non-specific, such as post-prandial abdominal pain, inadvertent weight loss, nausea, and vomiting [3]. Median arcuate ligament syndrome is often considered a benign anatomical occurrence, with skepticism surrounding its classification as a syndrome and where the need for intervention is controversial despite reports of improvement after surgery [4–6]. In this report, we have presented one of the largest series in the literature of patients diagnosed with MALS. We have described the serious and often overlooked complications that may result from this syndrome, such as visceral artery aneurysm formation and bleeding.

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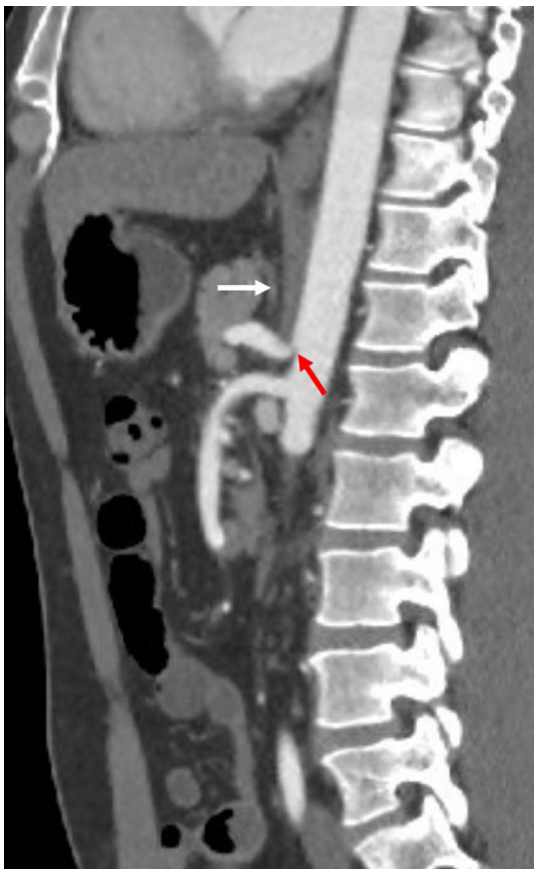

Fig. 1 Sagittal CT scan of the abdomen and pelvis showing the anatomical location of the median arcuate ligament (*white arrow*) with stenosis at the origin of the celiac trunk (*red arrow*)

Materials and Methods

Patient Population

A keyword search between January 1, 2012 and December 31, 2015 with the keywords "Median Arcuate Ligament Syndrome or MALS" was performed on our PACS system to identify the patients with potential MALS. This was done by the PACS technician at the department (supervised by the main investigator of the study). Fifty-five Patients were identified as having Possible MALS. The CT images were reviewed to confirm or refute the diagnosis. Findings required for diagnosis were encroachment of the diaphragmatic ligament on the celiac axis with more than 50% stenosis, which was calculated using the North American Symptomatic Carotid Endarterectomy Trial (NASCET) method [7], exaggerated stenosis during inspiration by CT scan [2], and with classical hooked appearance on sagittal reconstructed images (Fig. 2). However, because of the retrospective nature of the study, additional expiratory scans were not performed. Patients who were known to have or suspected of having congenital

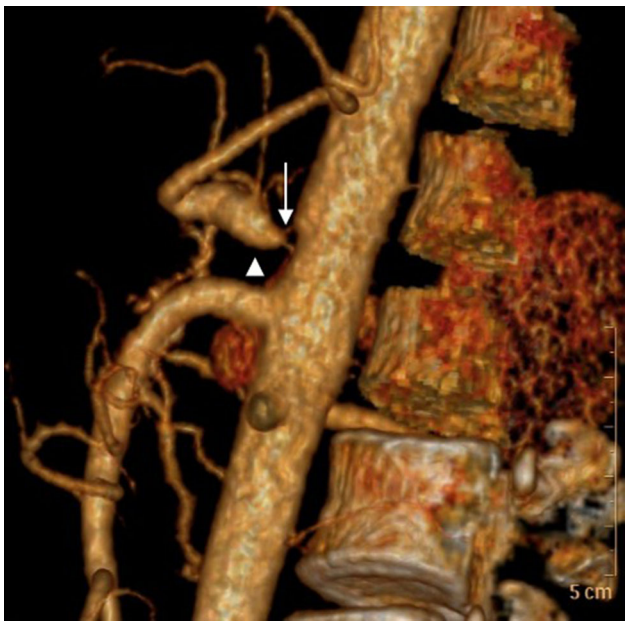

Fig. 2 3D sagittal reconstructed image of a patient diagnosed with median arcuate ligament syndrome, showing the severe stenosis at the origin of the celiac trunk (*arrow*) and the resultant post-stenotic dilation (*arrowhead*)

vascular malformations were excluded from the study. We identified 23 consecutive patients with median arcuate ligament syndrome.

Variables

Medical charts of patients were reviewed by a resident (LN) and a senior interventional radiologist (AH); review of acquisition of diagnostic CT scans was done by a radiographer and confirmed by a resident (LN) and two senior interventional radiologists (AH and AK); and if available, digital subtracted angiographic studies and endovascular treatment procedures were reviewed and performed by three senior interventional radiologists (AH, AK and MH). The resident and three interventional radiologists are all co-authors of the study.

The following variables were recorded and captured: patient demographics; Celiac artery stenosis vs occlusion, and post-stenotic dilation of the celiac artery (defined as a dilation greater than 8 mm) [8, 9]; Superior mesenteric artery diameter, and presence, size, and location of visceral artery aneurysms, diameter of feeding artery of the aneurysm, and aneurysm-to-feeding artery ratio; bleeding at any time; presence of collaterals, notably in the pancreaticoduodenal arcade (PDA). All vascular measurements were corrected to the centerline on two planes and maximum size on the third plane was measured and rounded to the highest 0.5 mm on reformatted enhanced CT images (Table 1). CT protocol; 256-slice scanner, the

Table 1 Patient characteristics

Patient number	Gender	Age (years)	Celiac	SMA[b]	SMA diameter	IMA[b]	Collaterals	Aneurysm location	Aneurysm size (mm)	Parent artery diameter (mm)	Ratio aneurysm-to-artery	Bleed	Treatment	Complications
1	M	50	Stenosed	Patent	8	Patent	–	–	–	–	–	–	–	–
2	F	59	Stenosed	Patent	6	Patent	PDA	PDA (×3)[a]	7,8,15	4	1.75, 2, 3.75	No	–	–
3	F	80	Occluded	Patent	8	Occluded	PDA	Celiac artery	15	7	2.14	No	–	–
4	F	69	Stenosed	Patent	5.5	Patent		–	–	–	–	–		–
5	M	17	Stenosed	Patent	7	Patent	–	–	–	–	–	–	–	–
6	M	79	Stenosed	Patent	8	Occluded	PDA	Celiac bifurcation	15	8	1.87	No	–	–
7	M	36	Stenosed	Patent	9	Patent	PDA	–	–	–	–	–	–	–
8	M	68	Stenosed	Stenosed	7	Patent		–	–	–	–	–	–	–
9	M	19	Stenosed	Patent	9	Patent	PDA	–	–	–	–	–	–	–
10	M	83	Stenosed	Patent	7.5	Patent	PDA	Splenic	17	7	2.43	No	–	–
11	F	54	Stenosed	Patent	8.5	Patent	GDA	–	–	–	–	–	–	–
12	M	69	Stenosed	Stenosed	7.5	Patent	PDA	Splenic	40	4	10	Yes	Coil embolization	Deceased due to complications of bleeding
13	F	56	Stenosed	Patent	7	Patent	PDA	PDA	5	3	1.67	No	–	
14	M	77	Occluded	Patent	9	Patent	PDA	PDA	37	4	9.25	Yes	Coil embolization	None
15	M	40	Occluded	Patent	8	Patent	PDA	–	–	–	–	–	–	
16	M	77	Stenosed	Patent	10.5	Patent	–	–	–	–	–	–	–	
17	F	34	Stenosed	Patent	6	Patent	–	–	–	–	–	–	–	
18	M	51	Stenosed	Patent	9.5	Patent	–	–	–	–	–	–	–	
19	M	36	Stenosed	Patent	8.5	Patent	PDA	–	–	–	–	–	–	
20	M	30	Stenosed	Patent	9	Patent	PDA	PDA	15	5	3	Yes	Coil embolization	Biliary ischemia
21	M	23	Stenosed	Patent	7	Patent	PDA	PDA	10	3	3.33	Yes	Coil embolization	None
22	M	64	Occluded	Patent	8.5	Patent	PDA	PDA	32	5.5	5.82	No	–	–
23	F	59	Stenosed	Patent	7	Patent	PDA	Splenic	21	6	3.5	No	–	–

PDA pancreaticoduodenal arcade, *GDA* gastroduodenal artery

[a] Indicates that patient# 2 had three distinct aneurysms in his pancreaticoduodenal arcade, with all three sizes and their respective ratios displayed in the table

[b] We have observed that despite having a collateral circulation and patency of the SMA and IMA in most patients, there is a high number of patients complaining of post-prandial abdominal pain. *Please refer to text for further discussion*

scans were done on inspiration from xiphoid process to the groins. The arterial and venous phases were done at 20 and 60 s post contrast injection, respectively.

The Interventional Procedures were reviewed, and the following variables were noted: nature of embolizing agents (coils, particles or liquid agents), vessels embolized, accessing vessels (SMA or retrograde access vs celiac artery or antegrade access), celiac artery recanalization or stenting.

Follow-up for bleeding, re-hospitalization, and delayed complications was obtained for fifteen patients; three patients passed away (two in hospital and one in Hospice), and five patients were lost to follow-up.

Ethical Approval

In accordance with our institution guidelines (which follow the United States code of Federal Regulations for the Protection of Human Subjects), the Institutional Review Board (IRB) approval was obtained at our university medical center.

Results

See Table 1 for a Summary of our Results

Twenty-three patients were diagnosed with median arcuate ligament syndrome. The median age was 56 years (range 17–83 years). There were 16 (69.6%) males and 7 (30.4%) females, with a male-to-female ratio of 16:7 (2.3). This is in contrast to what has historically been reported in the literature, where the majority of patients are women in their twenties and thirties [2, 6]. Nineteen (82.6%) patients had a variable degree of stenosis (all exceeding 50% stenosis), while the remaining 4 (17.4%) patients had a completely occluded celiac. The presenting symptoms as well as the number of patients with each symptom are outlined in Table 2.

Eleven out of 23 (47.8%) patients were found to have visceral artery aneurysms. One of these 11 patients had 3 aneurysms in the pancreaticoduodenal arcade (PDA) for a total of 13 measured aneurysms among all 11 patients. The median size of the 13 aneurysms was 15 mm (range 5–40 mm). They were located in the following arterial branches: the PDA ($n = 8$) (Fig. 3), the splenic artery ($n = 3$), and the celiac axis ($n = 2$). The average aneurysm-to-artery ratio—which is the ratio of the size of the aneurysm to the diameter of its parent artery—for all patients was 3.89 (range 1.67–10). Four patients (36.4% of those with aneurysms, 18.2% of total population) presented with bleeding from aneurysmal rupture, as confirmed by enhanced CT of the abdomen and/or DSA (Fig. 4). The median size of the bleeding aneurysms was 26 mm (range 10–40 mm), while the average ratio of bleeding aneurysm-to-artery was 6.40 (range 3–10).

Sixteen (69.6%) patients had a dilated collateral circulation either in the PDA or gastroduodenal artery (GDA). Collateral circulation was present in all patients with a complete occlusion of the celiac and in all patients with aneurysms. The median diameter of the SMA was 8 mm (range 5.50–10.50 mm).

The 4 patients who bled underwent selective angiography (Fig. 5) of the celiac trunk and SMA which showed retrograde filling of the celiac and its branches from the SMA. They were treated by trapping the aneurysm with coils. None of the other 19 patients underwent invasive angiography. None of the 23 patients underwent celiac recanalization/stenting.

Table 2 Presenting symptoms of patients with MALS with the number of patients reporting each symptom

Symptoms	Number of patients
Weight loss	7/23
Nausea	11/23
Diarrhea	3/23
Vomiting	10/23
Anorexia	8/23
Post-prandial abdominal pain	16/23

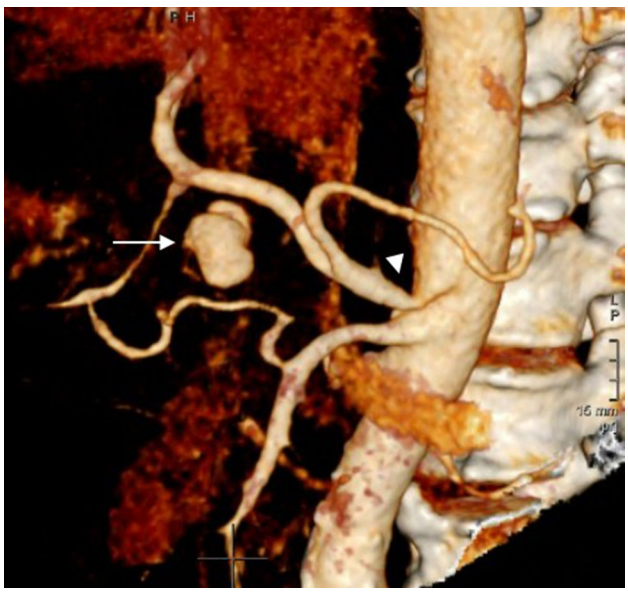

Fig. 3 3D reconstructed image of a patient with median arcuate ligament syndrome (*arrowhead*) and a prominent pancreaticoduodenal artery collateral circulation with aneurysm formation (*arrow*)

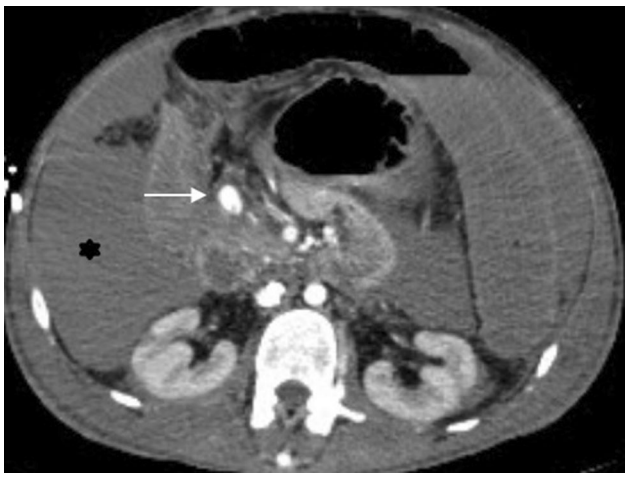

Fig. 4 Axial CT angiography image showing acute blood in the abdomen (*star*) and extravasation of contrast into the abdomen from a ruptured pancreaticoduodenal artery aneurysm (*arrow*)

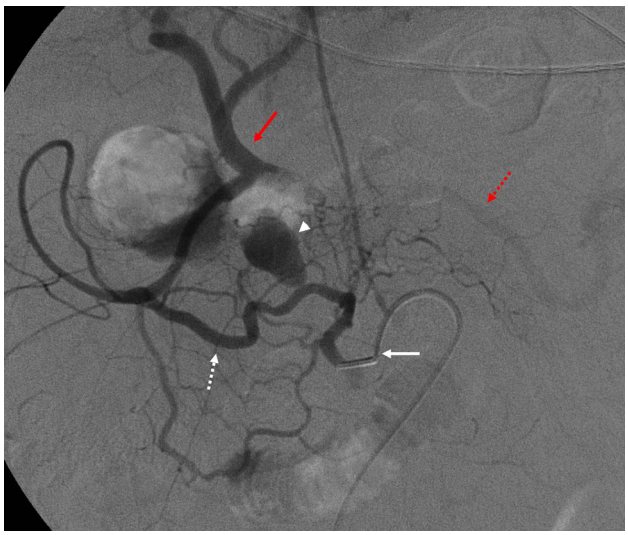

Fig. 5 Digital subtracted angiogram of a patient with a pancreaticoduodenal artery (PDA) aneurysm. The tip of the catheter is seen via the superior mesenteric artery selectively catheterizing the PDA (*white arrow*). Note is made of the aneurysm (*arrowhead*) with flow of contrast through the PDA collaterals (*dashed white arrow*) and retrograde filling of the hepatic artery (*red arrow*) and the splenic artery which appears faint (*dashed red arrow*)

Patient Outcome and Follow-up

Five patients (21.7%) were lost to follow-up. One patient died from complications of GI bleeding. Two others died from complications of illnesses unrelated to MALS, for a total of 3 (13.0%) deceased. Follow-up was available for 15 patients (65.3%). The median time to follow-up was 5 patient-months (range 1–42), with a total follow-up time of 180 patient-months. Of the untreated patients with and without aneurysms ($n = 12$), none had bled, no new aneurysms developed, and there was no change in the size of the existing aneurysms. Of the treated patients who survived ($n = 3$), two made a full recovery and suffered no complications or recurrent bleeding. One patient developed biliary strictures secondary to coiling of the gastroduodenal artery aneurysm. He then underwent percutaneous transhepatic cholangiography (PTC) with biliary drain insertion followed by a surgical hepatico-jejunostomy. There were no other complications following these two procedures.

Discussion

Throughout the literature, MALS is considered a rare benign entity as most patients with compression of the celiac artery by the MAL are asymptomatic [10]. In fact, it is estimated that roughly 10–24% of the general population have some degree of compression of the celiac artery as an anatomic variant [11, 12]. We have noticed that most of our patients have symptoms of post-prandial abdominal pain, which would seem surprising given that the majority have also developed a collateral circulation which should prevent visceral ischemia. However, we have found that our results are consistent with other studies in the literature which have reported such abdominal pain as a symptom in nearly all patients with MALS included in those studies [4, 13, 14]. It is also noteworthy that the mechanism of pain has been a long-held subject of debate with questionable pathophysiology; it is believed that it is also partly due to neurogenic pain brought on by the compression and overstimulation of the celiac ganglia. Another additional theory is that collaterals are instead a major source of pain whereby they generate a form of vascular steal syndrome where they shunt blood away from a patent SMA to the smaller branches distal to the stenosis in the celiac artery [5, 10].

More importantly, our data suggest that MALS is not a benign entity and it is associated with visceral artery aneurysm formation and bleeding.

Visceral artery aneurysms (VAA) in general are uncommon, with a total prevalence of 0.1–2%, although incidence has been increasing over the past decade due to incidental findings on imaging [15]. In an autopsy study done by Katz-Summercorn et Bridger, 33% of cadavers were found to have compression of the celiac artery by the MAL, and yet there was no mention of aneurysmal formation throughout the study [16]. Despite that, in other observations, it has been reported that MALS increases significantly the risk of aneurysm formation namely PDA aneurysms [17, 18]. This has been reproduced in our series where nearly half of our patients (47.8%) were found to have aneurysms.

Overall, it is estimated that 15–22% of VAAs rupture [19], with a mortality rate that could reach 100% depending on their location and associated comorbidities of the affected patient [20]. In our series, 4 patients bled, which equates to more than a third (36.4%) of the cases with aneurysms and nearly one out of 6 of all patients with MALS. In addition, one of those 4 patients died from bleeding.

Timing the treatment of VAA is controversial as some authors believe that aneurysms larger than 2 cm in diameter, those that show expansion on follow-up imaging, and that are symptomatic should be treated [21, 22]. Others have refuted that notion and suggested that VAAs, especially PDA aneurysms, should be treated regardless of their size [17, 23]. In our series, two of our four patients who bled had aneurysms less than 2 cm in diameter, but all of our patients who bled had aneurysm-to-artery ratio of at least 3 (range 3–10), which suggests that in medium-size vessel aneurysms, it is not absolute size (like in abdominal aortic aneurysms) but the relative size of the aneurysm to the originating vessel which increases the risk of rupture. This is in agreement with what has been published in the literature about the risk of bleeding and intracranial aneurysms, where authors found that the size ratio of the aneurysm-to-parent artery is an independent risk factor for rupture [24–26].

The first-line treatment of ruptured visceral aneurysms in otherwise stable patients is endovascular coiling and/or stent placement. Other well-established therapeutic options for MALS-associated aneurysms without rupture include open to laparoscopic surgical decompression of the ligament with or without surgical reconstruction or stenting, as well as more recently described techniques such as robot-assisted release and retroperitoneal endoscopic release [6, 27, 28]. In terms of endovascular approach, some encourage both embolization of the aneurysm coupled with recanalization of the celiac trunk [1, 29]. Others suggest that coiling alone is enough, with no recurrence of bleeding on long-term follow-up [17, 18, 30]. All our patients who bled were treated with coil embolization alone. Even though clinically significant ischemic complications of endovascular embolization are generally rare [31], one of our cases was complicated by biliary ischemia and stricture. We believe that this occurred because of ischemia to the biliary tree after embolizing the major collateral blood supply to the liver via the hepatic artery, and we believe that in such cases, recanalization of the celiac trunk via an endovascular or a surgical approach, or a combination of both, should be performed to avoid such complications.

The pathogenesis of visceral aneurysms in MALS is important to consider; when the celiac trunk is compressed, blood is shunted through its collaterals with the SMA, namely the pancreaticoduodenal arcade; this increase in blood flow causes dilation of these collaterals and a predisposition to aneurysm formation [29]. Our results support the above theory and have led us to make several additional observations that may be pertinent to MALS patients. First patients who had aneurysms all had significant collateral formation. Second, the risk of aneurysmal rupture seems to depend more on the aneurysm-to-artery ratio rather than on the absolute aneurysm size.

There are several limitations to our study. First of all, the small sample population limits our ability to draw a final, definitive conclusion, especially in the matter of aneurysms and their treatments. Nonetheless, our small population is in fact a reflection of the syndrome's overall low prevalence. Second, this is a retrospective study and the rate of symptomatic MALS with aneurysmal formation might be exaggerated and concomitantly the rate of the MALS without aneurysmal formation might be underestimated. Third, it has limitations inherent to all retrospective studies. Finally, consistent long-term follow-up of our identified patients was not possible for this study, and we acknowledge the need for medium- and long-term follow-up in future reviews.

Conclusions

In our study, we have observed that complete occlusion and collateral formation predispose to aneurysm formation. This and formation of a celiac-SMA collateral circulation appear to be important precursors to aneurysm development. Rupture of the aneurysm, however, appears more dependent on its size in relation to the diameter of its parent artery: (aneurysm-to-artery ratio more than 3). We recommend close follow-up of patients diagnosed with MALS who develop collaterals without aneurysms. Once aneurysms are diagnosed, treatment should be considered when the aneurysm-to-artery size ratio approaches three. Treatment of these aneurysms can be done via various approaches including endovascular treatment. This may consist of coil embolization of the aneurysm coupled with celiac artery decompression and recanalization as suggested by some authors to prevent ischemic complications in cases where major collateral circulation cannot be otherwise preserved [23].

Compliance with Ethical Standards

Conflicts of interest All authors declare that they have no conflicts of interest.

Ethical Approval For this type of study, formal consent is not required.

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