Clinical significance of mesenteric arterial collateral circulation in patients with celiac artery compression syndrome

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ABSTRACT

Objective: Although extensive collateral arterial circulation will prevent ischemia in most patients with stenosis of a single mesenteric artery, mesenteric ischemia may occur in these patients, for example, in patients with celiac artery compression syndrome (CACS). Variation in the extent of collateral circulation may explain the difference in clinical symptoms and variability in response to therapy; however, evidence is lacking. The objective of the study was to classify the presence of mesenteric arterial collateral circulation in patients with CACS and to evaluate the relation with clinical improvement after treatment.

Methods: Collateral mesenteric circulation was classified on the basis of angiographic findings. Collaterals were categorized in three groups: no visible collaterals (grade 0), collaterals seen on selective angiography only (grade 1), and collaterals visible on nonselective angiography (grade 2). Surgical release of the celiac artery in patients with suspected CACS was performed by arcuate ligament release. Clinical success after surgical revascularization was defined as an improvement in abdominal pain.

Results: Between 2002 and 2013, there were 135 consecutive patients with suspected CACS who were operated on. In 129 patients, preoperative angiograms allowed classification of collateral circulation. Primary assisted anatomic success was 93% (120/129). In patients with grade 0 collaterals, clinical success was 81% (39 of 48 patients); with grade 1 collaterals, 89% (25 of 28 patients); and with grade 2 collaterals, 52% (23 of 44 patients; $P < .001$).

Conclusions: Patients with CACS and with extensive collateral mesenteric arterial circulation are less likely to benefit from arcuate ligament release than are patients without this type of collateral circulation. The classification of the extent of mesenteric collateral circulation may predict and guide shared decision-making in patients with CACS. (J Vasc Surg 2017;65:1366-74.)

Patients with stenosis or occlusion of a single mesenteric artery seldom develop symptoms of chronic mesenteric ischemia. It is commonly accepted that this is due to the extensive mesenteric arterial collateral circulation. However, patients with mesenteric one-vessel disease may develop symptoms of ischemia. Therefore, it can be challenging to determine if abdominal complaints are related to impaired mesenteric flow due to an arterial stenosis or whether these symptoms have another origin.

A specific group of patients with one-vessel disease is patients with celiac artery compression syndrome (CACS). This nonatherosclerotic, respiration-dependent compression of the celiac artery (CA) may induce ischemia.[1] Several studies have demonstrated successful treatment of CACS with CA release through release of the arcuate ligament.[2-6] However, CA compression is a common finding. Up to 50% of asymptomatic individuals have compressive features of the CA, especially during expiration.[7,8] In only a small number of patients with abdominal complaints is CACS the cause of the symptoms.[1,9] It is assumed that the abundant collateral circulation prevents ischemia in most patients and that these collaterals develop from small embryonic remnants.[10] A lack of collateral mesenteric circulation could then contribute to the development of symptoms in patients with CACS.

We recently studied collateral circulation in mesenteric disease and demonstrated the pathophysiologic effect of single mesenteric vessel stenosis on collateral circulation.[11] Angiography in patients with stenosis in a single

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Author conflict of interest: none.

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mesenteric artery showed collateral circulation in 50% of patients. In half of these patients, the collateral was prominently visible during nonselective visceral angiography. We proposed a grading of collaterals based on angiography findings.

In this study, we investigated the association between the extent of collateral mesenteric circulation and clinical success after arcuate ligament release in patients with CACS.

ARTICLE HIGHLIGHTS

- **Type of Research:** Single-center retrospective cohort study
- **Take Home Message:** In 135 patients with celiac artery compression syndrome, only 52% of the patients with extensive collateral circulation had clinical benefit from ligament release. Benefit was >80% in those with no or small collateral circulation.
- **Recommendation:** The authors suggest that patients with symptomatic celiac artery compression syndrome with extensive collateral flow may not benefit from release of the median arcuate ligament.

METHODS

Study design. All patients who underwent treatment for CACS in the period 2002 to 2013 were prospectively included in an ongoing database and were retrospectively analyzed in the study. Mesenteric collaterals were identified and categorized on preoperative angiography, and the effect on the treatment of single-vessel stenosis was evaluated. Angiography has been the "gold standard" and offers a dynamic picture of the grade of stenosis during respiration. Especially in this category of patients with respiration-dependent stenosis, angiography has been our preference. Additional information of flow direction through collaterals is also an advantage of angiography. According to national regulations, Institutional Review Board approval was not required for this retrospective study. Therefore, no informed consent was obtained from the patients. Patients' data were analyzed anonymously.

Participants. We used a standard workup for evaluation of chronic mesenteric ischemia including structured medical history, assessment of vessel anatomy, and tonometry function test.[1] All patients had preoperative duplex ultrasound and visceral angiography before treatment. Celiac compression was diagnosed when an eccentric stenosis of the CA by respiration-dependent compression by the arcuate ligament was present. CACS was suspected when this compression was accompanied by chronic abdominal symptoms not caused by other diseases, an abnormal tonometry function test (gastric exercise tonometry)[1] result, and a significant eccentric compression of the CA. Patients with celiac stenosis <70% needed a positive tonometry test result or typical clinical presentation. The diagnosis was made and treatment advice given in a multidisciplinary working group on mesenteric ischemia, as previously described.[12] Of the 129 patients, 46 patients were also included in a previous study.[11]

Abdominal angiography. Multiplane digital subtraction angiography of the abdominal aorta and its branches enabled multiple oblique projections of the abdominal aorta and of the origin and outflow of the mesenteric arteries. Angiography was performed during maximum expiration and inspiration phases of respiration, as described previously.[13] For nonselective angiography, a pigtail catheter was placed in the aorta 3 to 4 cm above the CA ostium, and a standard bolus of 25 mL of contrast material was given with a pump flow of 15 mL/s. Before 2009, all patients underwent both nonselective and selective angiography. After 2009, selective cannulation was performed in selected cases only if stenosis was doubted on nonselective angiography. Nonselective anteroposterior view angiography was performed during expiration unless duplex ultrasound suggested a stenosis during inspiration. A lateral view was obtained during both expiration and inspiration. Selective angiography was performed during the phase of respiration with maximal grade of stenosis. Each angiogram was reviewed in blinded fashion by three observers, one interventional radiologist (D.G.) and two vascular surgeons (R.H.G., A.P.). When discrepancies occurred between the observers, results were compared and a consensus was reached. The degree of stenosis was assessed as percentage of the normal diameter.[14,15]

Classification of mesenteric collateral circulation. A differentiation was made between normal arterial connections of the three mesenteric vessels and pathologic hypertrophic collaterals. This was based on size and visibility by the three independent observers. We classified only interconnecting vessels as collaterals when they were both clearly visible and hypertrophic. Collateral vascularization was assessed and categorized according to appearance on preoperative angiography as previously described.[11] This categorization is based on visibility of the collateral on nonselective or selective angiography, which differentiates between grade 1 and grade 2 (Figs 1 and 2). Three groups were identified: grade 0, grade 1, and grade 2 collaterals (Table I). Collaterals were further categorized according to their anatomy: gastroduodenal or pancreaticoduodenal artery, arc of Buhler, Riolan artery, and Drummond artery.

Intervention. Release of the median arcuate ligament was performed in a retroperitoneal open (before 2005) or endoscopic procedure (since 2005) as reported.[5] The open CA release was performed by a transperitoneal upper

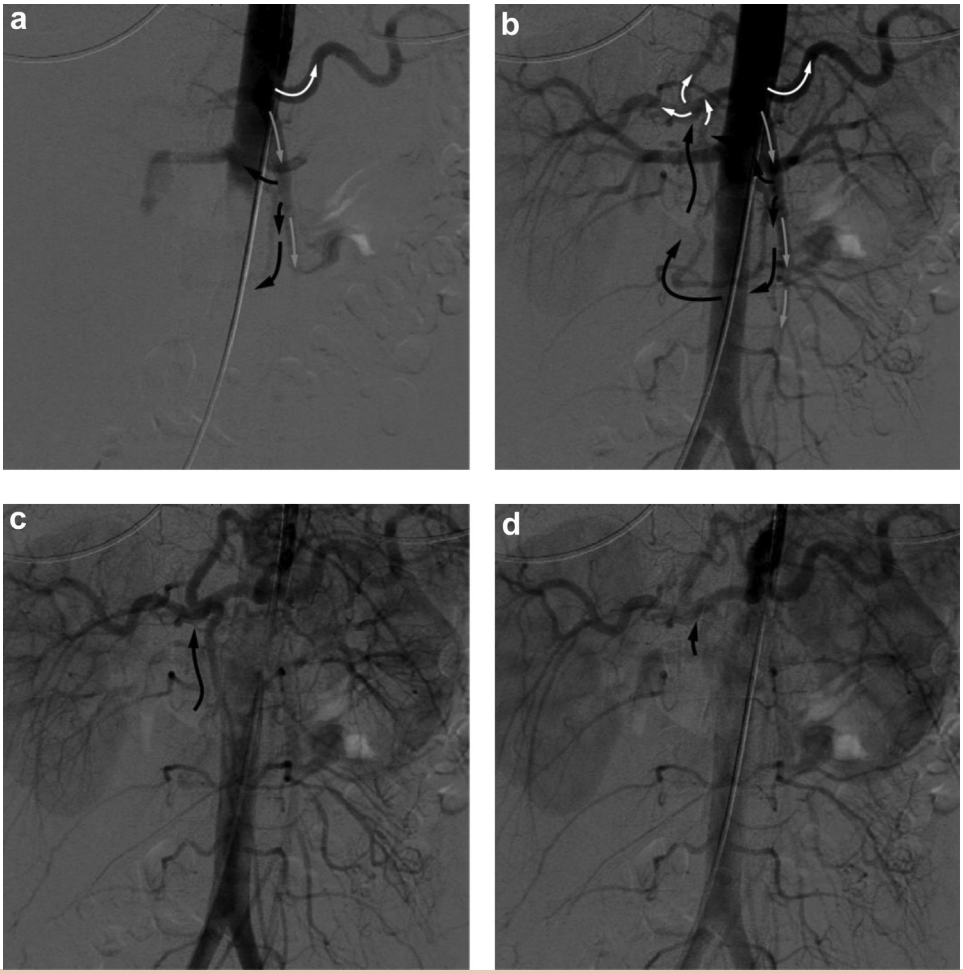

Fig 1. Nonselective angiography **(a-d)** in a patient with celiac artery (CA) compression. Direction of collateral flow is visible through the pancreaticoduodenal arcade (*black arrow*) from the superior mesenteric artery (SMA; *gray arrow*) to the CA (*white arrow*) with delayed filling. Collaterals prominently visible during nonselective angiography are defined as grade 2.

midline laparotomy or a left subcostal incision with retroperitoneal approach. In case of persisting intraluminal stenosis after surgical release, additional endovascular percutaneous transluminal angioplasty was performed.

Outcomes and assessment. All patients were observed postoperatively for assessment of symptoms and duplex ultrasound examination after 1 year, 3 years, and 5 years. Postoperative angiography was performed routinely from 2004 until 2009 and when indicated after 2009.

Anatomic success was defined as <50% residual stenosis during inspiration and expiration on postoperative duplex ultrasound or <30% on angiography. Clinical response to treatment was categorized in five groups (A-E): A, complete disappearance; B, sustained reduction; C, temporary reduction; D, no change; and E, worsening of complaints. Clinical success was defined as sustained reduction or complete disappearance of abdominal symptoms after treatment (group A/B) in combination with primary assisted anatomic success.

Statistical analysis. Analysis was performed on the basis of intention to treat. The χ^2 test was used for categorical variables, such as success of CA release and type of intervention or presence of grade 2 collateral. Data were expressed as mean. Independent samples Student *t*-test was used for comparison of duplex ultrasound parameters between groups. The difference in outcome between the three collateral groups was done by χ^2 test for trend (linear-by-linear association) with Holm-Bonferroni correction. The results after CA release for patients with and without type 2 collaterals were presented in a Kaplan-Meier curve. For statistical analysis, the date of intervention was used if intervention was not successful. Statistical significance was assumed for $P < .05$. All data were analyzed using SPSS 21 (IBM Corp, Armonk, NY).

RESULTS

In the 12-year inclusion period, 135 consecutive patients were operated on for CACS. In six patients, the angiogram was no longer available for revision, and they

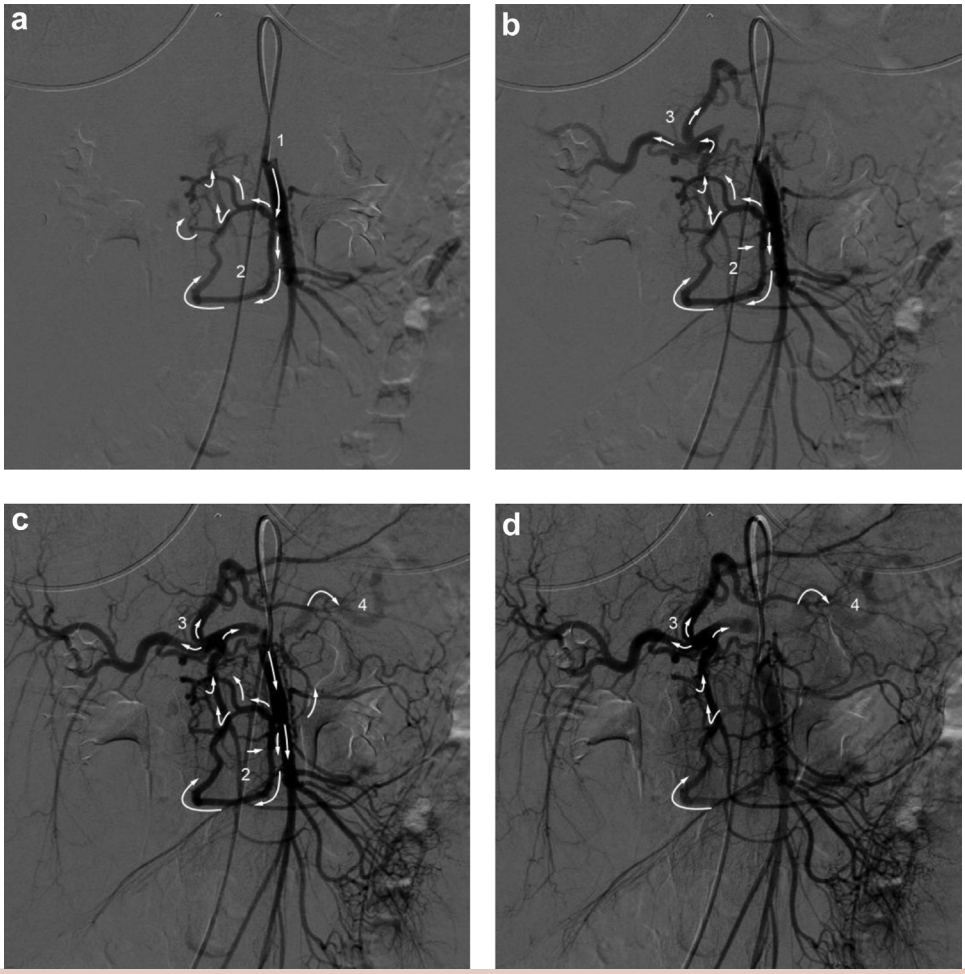

Fig 2. Selective angiography **(a-d)** of the superior mesenteric artery (SMA; *1*) in the same patient as in Fig 1. Collateral flow and direction of flow are shown. Selective catheterization of the SMA results in filling of the celiac artery (CA) and its branches, hepatic and left gastric artery (*3*), and splenic artery (*4*) through the pancreaticoduodenal arcade (*2*). If collaterals are found only during selective angiography, they are defined as grade 1.

Table I. Grade and type of collaterals

Grade 0 collaterals: no visible collaterals or partial filling of the recipient artery on selective angiography
Grade 1 collaterals: at least one collateral clearly visible on selective but not on nonselective angiography
Grade 2 collaterals: at least one collateral clearly visible on nonselective angiography
Gastroduodenal or pancreaticoduodenal artery
Arc of Buhler
Riolan artery and Drummond artery

were therefore excluded. The mean age of the 129 included patients was 35 years (range, 15-76), and 106 (82%) of them were female. Mean follow-up time was 5.5 (range, 1-12) years.

Preoperative angiographic findings. Preoperative nonselective angiography was available in 129 patients and selective angiography was performed in 103 patients (80%); 111 patients had a CA stenosis of >70%. The remaining 18 patients had a 50% to 70% stenosis. The CA stenosis existed predominantly during expiration in 115 (89%) patients and predominantly during inspiration in 7 (5%) patients; in 7 (5%) patients, a fixed eccentric noncalcified stenosis was found. None of the 129 patients had a superior mesenteric artery (SMA) or inferior mesenteric artery (IMA) stenosis.

Table II. Patients treated for celiac artery compression syndrome (CACS), presence and type of collaterals, and clinical improvement (group A/B)

Collateral type	Grade			Total
	0	1	2	
Gastroduodenalis	—	23 (79)	40 (83)	63 (82)
Buhler	—	4 (14)	2 (4)	6 (8)
Riolan/Drummond	—	1 (3)	3 (6)	4 (5)
Gastroduodenal and Riolan/Drummond	—	1 (3)	3 (6)	4 (5)
Total	52 (40)	29 (22)	48 (37)	129
No clinical improvement	10 (19)	3 (10)	24 (50)	37 (29)
Clinical improvement	42 (81)	26 (90)	24 (50)	92 (71)

Values are reported as number (%).

A grade 0 collateral was found in 29 patients; a grade 1 collateral, in 52 patients; and a grade 2 collateral, in 48 patients. Collateral circulation was through the gastroduodenal artery in 70 of the 77 patients with a grade 1 or grade 2 collateral, through the arc of Buhler in 9 patients, and through Riolan or Drummond artery in 5 patients (Table II). A combination of two types of collaterals was observed in seven patients.

Anatomic outcome. An open retroperitoneal approach was performed primarily in 19 patients. A retroperitoneal endoscopic approach was performed in 110 patients as previously described.[5] Two cases were converted to an open procedure, one for bleeding from the left renal artery and one because adhesions prohibited retroperitoneal access. In three other patients, a persisting stenosis after surgical release necessitated an additional open release of the arcuate ligament.

All patients had postoperative duplex ultrasound examination, and postoperative angiography was performed in 80 patients. Primary anatomic success was 77% (99/129; Fig 3). After additional percutaneous endovascular angioplasty treatment in 25 patients and open reintervention in 3 patients, primary assisted anatomic success was 93% (120/129). These data are also presented in a Kaplan-Meier survival curve (Figs 4 and 5). No significant difference was found for patients with and without type 2 collaterals.

Clinical outcome. On follow-up, pain improved in 92 patients (group A/B, 71%), whereas in 16 patients (group C, 12%), a temporary response was seen. In 21 patients (group D, 16%), pain did not change. No patient had worsening of complaints (Table II). No difference in success was observed in relation to age, sex, degree of stenosis, and type of operation. Clinical success in patients with >70% stenosis was 71% compared with 82% in patients with <70% stenosis ($P = .326$). Clinical success was 79% for open release and 71% for endoscopic release ($P = .493$).

The presence of extensive collaterals on preoperative mesenteric angiography was related to postoperative clinical success. Clinical success rate was 81% in patients with grade 0 collaterals (39 of 48 patients) and 89% in patients with grade 1 collaterals (25 of 28 patients). In patients with grade 2 collaterals, the clinical success rate was 52% (23 of 44 patients; $P < .001$). In Fig 6, clinical success after CA release is presented in a Kaplan-Meier survival curve, divided into patients with type 2 collaterals (clinical success of 52%) and without type 2 collaterals (clinical success of 84%; $P < .001$).

DISCUSSION

This study shows that patients with CACS and with extensive mesenteric collateral circulation are less likely to respond to CA release. These extensive collaterals are likely to have a compensating function preventing ischemia in the CA outflow region. This assumption is in line with the increased flow in the unaffected SMA that regularly accompanied these collaterals.[11] Furthermore, collaterals are seen in many asymptomatic patients, but seldom in the absence of mesenteric disease.[10,16] The higher percentage of grade 2 collaterals found in two-vessel mesenteric disease compared with one-vessel disease and rare occurrence in patients without mesenteric disease also support this assumption.[11]

The pathophysiologic mechanism of the syndrome of CA compression by the median arcuate ligament is not fully understood and is still under debate. An ischemic pathophysiologic mechanism of CACS is supported by a previous publication from our group.[1] The main arguments against the ischemic existence of symptomatic CA compression are the high frequency of asymptomatic isolated CA compression and the idea that mesenteric collateral circulation prevents ischemia in case of one-vessel disease.[7,8] Other theories, such as intramesenteric steal or involvement of the ganglion nerve, have also been suggested. However, our release is single-sided and left the other half of the plexus ganglion unharmed.[5] The formation of collaterals demonstrated in this study does not really fit in a neurogenic hypothesis. The pathophysiologic mechanism of mesenteric collateral circulation is not fully

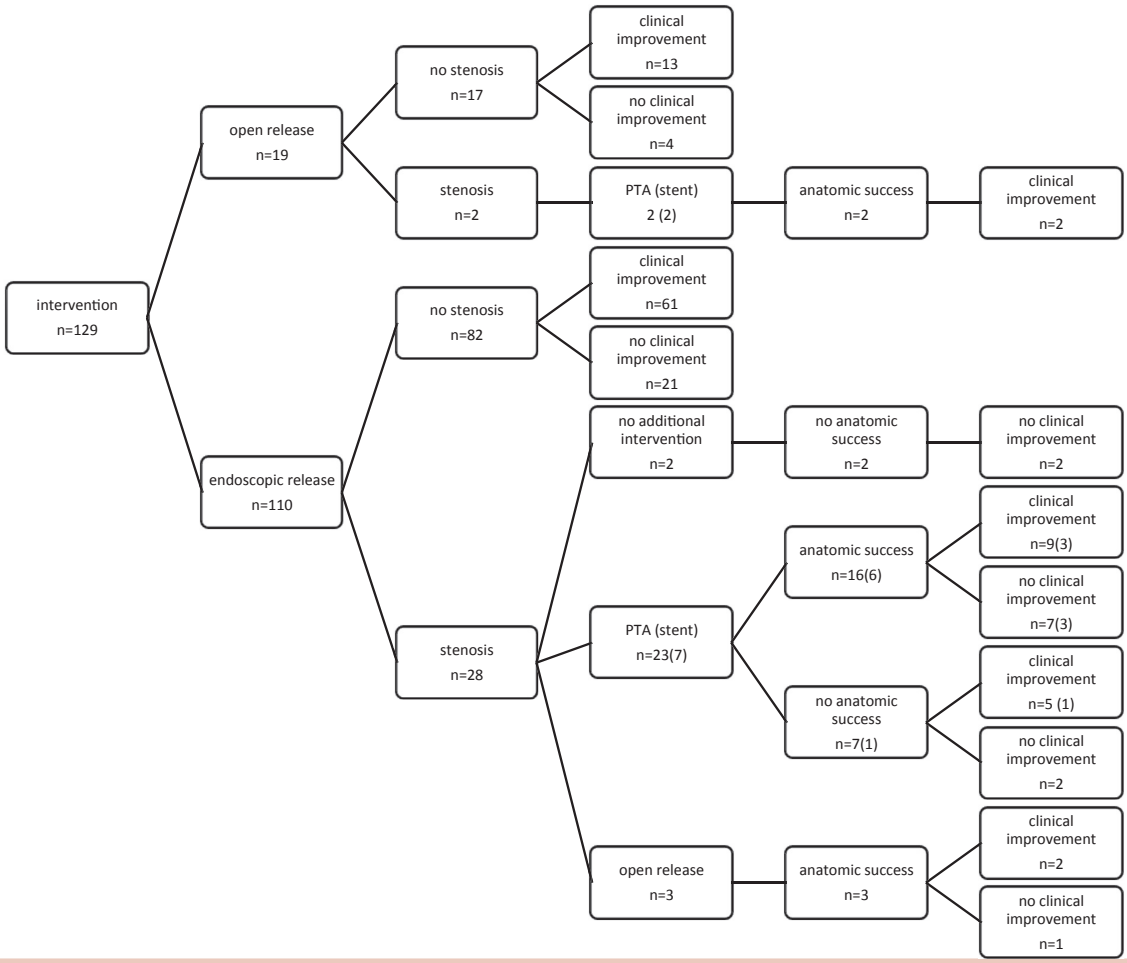

Fig 3. Patients treated for celiac artery compression syndrome (CACS), type of treatment, additional intervention, and anatomic and clinical improvement (group A/B). *PTA*, Percutaneous transluminal angioplasty.

understood, and most human studies on collaterals are descriptions of anatomy.[17] The classic anatomic study of the blood vessels to the upper abdomen was published in 1955 by Michels, who described the upper abdominal blood supply and anatomy by analysis of 400 dissections and 300 angiograms.[16,18] Collateral pathways between the CA and the SMA include the pancreaticoduodenal arcades, the dorsal pancreatic arteries, and occasionally the arc of Buhler.[10] Nebesar et al described 300 arteriograms of the CA and SMA.[16] Hypertrophied pancreaticoduodenal arcade collaterals were found predominantly in patients with stenosis in the CA or SMA and are found in many asymptomatic patients. However, as already mentioned by Nebesar, such hypertrophied collaterals are rarely seen without an evident stenosis. They also discussed the importance of technique of angiography to identify collaterals and the difference between selective and nonselective angiography. Well-established collaterals are clearly defined by angiography; however, potential collaterals (before enlargement) may be more difficult to demonstrate. This is in concordance with our findings, which led us to the distinction of different types of collaterals (grades 1 and 2). In this study, a possible role in preventing ischemia was found only for grade 2 collaterals, clearly present on nonselective angiography.

Collateral circulation is potentially available to connect all mesenteric arteries, similar to the circle of Willis of the brain.[17] This is demonstrated during endovascular thoracic aortic aneurysm repair with intentional covering of the CA. In the majority of these patients, intentional covering of the CA can be performed after ensuring sufficient collateral circulation.[19] It emphasizes the importance and potency of collaterals in the mesenteric circulation in acute occlusion of one mesenteric artery. The most important collateral vessels observed during balloon occlusion, as a pretest for endovascular covering, were the pancreaticoduodenal arcades and the dorsal pancreatic artery.[19]

It is not always clear if these connecting arteries are present. If present, are they functional? If they are not

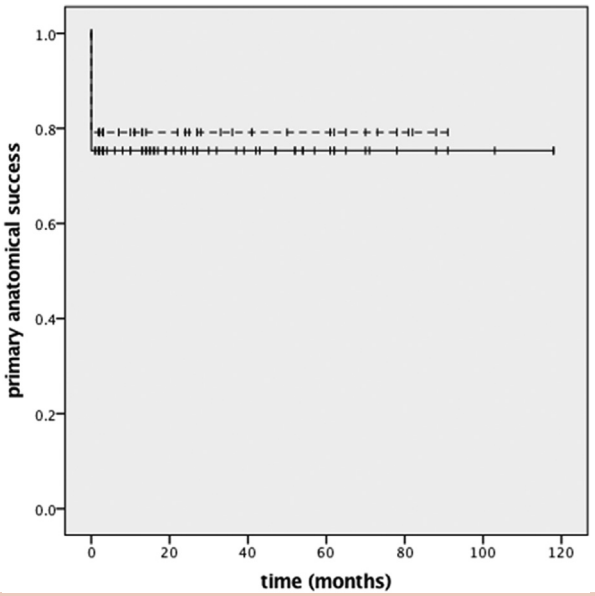

Fig 4. Primary anatomic success after celiac artery (CA) release. Kaplan-Meier survival curve of treated patients, divided into patients with (*dashed line*) and without (*solid line*) type 2 collaterals, with no significant differences between the curves ($P = .617$).

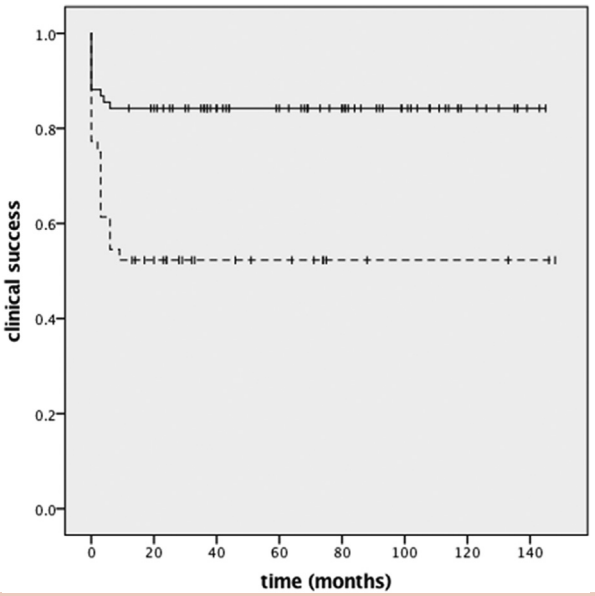

Fig 6. Clinical success after celiac artery (CA) release. Kaplan-Meier survival curve of treated patients, divided into patients with (*dashed line*) and without (*solid line*) type 2 collaterals, with significant differences between the curves ($P < .001$).

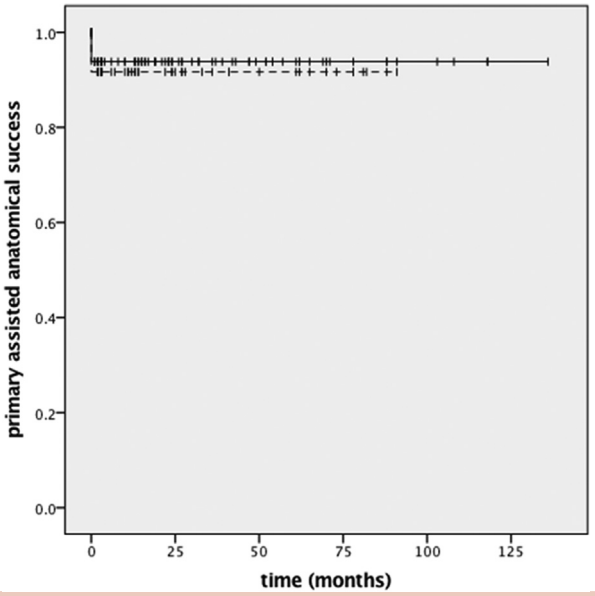

Fig 5. Primary assisted anatomic success after celiac artery (CA) release. Kaplan-Meier survival curve of treated patients, divided into patients with (*dashed line*) and without (*solid line*) type 2 collaterals, with no significant differences between the curves ($P = .643$).

present, how do they develop?[20] The mechanism behind the development of collaterals remains largely unknown. Why do some patients develop grade 2 collaterals and consequently have no symptoms of ischemia and others do not? From animal studies, we know that the extent of the pial collateral circulation exhibits surprisingly wide differences because of genetic variation. These differences also closely predict the severity of ischemic injury.[21] Environmental factors may also play a role. Exercises have been shown to improve myocardial perfusion in patients with coronary artery disease and peripheral arterial disease.[22] However, the evidence of exercise-induced increases in collateral blood flow is limited and inconsistent.[23]

A normal compensatory response to arterial occlusion is the flow-mediated enlargement of pre-existing vessels, or collaterals, that bypass the site of occlusion and supply blood to distal ischemic tissues.[24] The current understanding of collateral network development involves a complex remodeling of pre-existing conduit vessels that is probably driven by inflammatory processes.[25] Controversy still exists about the primary stimuli for collateral growth. The stimulus can be chemical (ischemia) or physical, and if it is physical, it can be pressure or flow related.[26,27]

Unthank et al studied mesenteric collateral circulation and blood flow in the rat.[28-31] Their results suggest that the microvasculature distal to the occlusion is able to increase flow by dilation and collateral adaptations, resulting in lower total resistance of the collateral-dependent region.[28] Mesenteric collateral enlargement is correlated with increases in arterial blood flow and shear stress, determined by blood flow and vessel radius, and can occur independent of tissue ischemia.[29,30] When shear

stress is reduced after enlargement of collaterals, they are evolving to become more like control arteries.[31] We can only speculate if and how the observed higher flow in the normal SMA in patients with grade 2 collaterals observed in this study and our previous study[11] in non-CACS patients is related to this.

Higher flow in mesenteric vessels in coexistence with a stenosed vessel has also been demonstrated in an animal study of Boley et al.[32,33] They demonstrated that occlusion of the SMA led to increased blood flow in both the CA and IMA. A small human study showed that SMA occlusion in five patients (three isolated cases of SMA occlusion and two with CA and SMA occlusions) was associated with an increase in IMA blood flow in all.[34] The higher flow in the SMA in coexistence with collateral circulation also found in this study (data not shown) is likely to be linked to the respiration-dependent stenosis of the CA in CACS. As the SMA is nonstenosed, a lower resistance in the distribution area of the artery is likely to cause this higher flow. This lower resistance may be facilitated by collateral circulation. Interestingly, the flow (peak systolic velocity) in the nonstenosed mesenteric artery was higher in grade 2 collaterals compared with grade 1. One could speculate that grade 1 collaterals could evolve to grade 2 under given circumstances. On the other hand, as most patients have a longer history of symptoms before presentation, it seems unlikely that these patients will develop adequate (grade 2) collateral circulation. Whether arteriolar collateral vessels are pre-existent or form de novo is still a matter of debate in the literature.[27]

This is the first study to document the relation between extensive mesenteric collateral circulation and clinical symptoms. The observed primary anatomic success rate of 77% in this study is somewhat low. This may be due to either a selection process or the remaining CA damage after years of compression. This is supported by the fact that additional percutaneous transluminal angioplasty is successful in most cases (primary assisted patency of 93%), which would not be the case if release was incomplete (with remaining compression by the ligament).

A limitation of using angiography for grading collaterals is that the position of the catheter and the force of injection are operator dependent, which could affect outcome. This may play a role in the selective angiogram (grade 1 collaterals). For the nonselective angiogram (grade 2 collaterals), the variability is limited because all angiograms were strictly performed according to standard protocol mentioned in the Methods section. Follow-up was done by postoperative mesenteric duplex ultrasound, and not all patients had postoperative angiography. This could result in an underestimation of persisting complaints because of technical failure (insufficient release). However, in a subset of 71 patients with anatomic success proven by postoperative angiography, the effect of presence of grade 2 collaterals was even stronger, with only 21% positive responders after CA release compared with 79% in patients without ($P < .001$). Not all patients had selective angiography. Patients without selective angiography and with no collaterals visible on nonselective angiography were categorized as no collateral. This might underestimate the number of patients with a grade 1 collateral. Because this did not influence the number of grade 2 collaterals, and the outcomes for grade 0 and grade 1 were similar, the effect on that finding was probably negligible. The presence of these collaterals essentially does not exclude ischemia but makes the chance of positive effect of treatment smaller. We did not quantitate actual blood flow, which could further elucidate why only half of these patients benefit from intervention. Collateral blood flow could be different in responders and nonresponders with grade 2 collaterals. Also, the metabolic demand may differ, but we are unaware of any method to measure it. In any case, it seems that in approximately 50% of patients with grade 2 collaterals, these do prevent them from ischemic complaints, whereas in the other 50%, these are either insufficient or complaints preoperatively had a nonischemic cause.

Clinical success in patients without grade 2 collaterals was 84%. Presence of grade 2 collaterals reduced the success rate of intervention to around 50%. Still, in the other patients, these were apparently insufficient, and they benefited from intervention. Therefore, it would not be a definite contraindication to intervention. The physician should share this information with the patient before mutual agreement to intervene.

CONCLUSIONS

CACS patients with collaterals clearly visible on nonselective angiography (grade 2) are less likely to benefit from revascularization than are patients without this type of collateral. Whether the presence of grade 2 collaterals is a contraindication to treatment should be confirmed in prospective series. Our study certainly points to better appreciation of these large collaterals.

AUTHOR CONTRIBUTIONS

Conception and design: AP, JK, CZ, RG
Analysis and interpretation: AP, JK, JP, RG
Data collection: AP, JK, DG, RG
Writing the article: AP, JK, DG, CZ, RG
Critical revision of the article: AP, JK, DG, JP, CZ, RG
Final approval of the article: AP, JK, DG, JP, CZ, RG
Statistical analysis: AP, JP
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Overall responsibility: AP

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APPENDIX.

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