# Aneurysm and stenosis of the celiomesenteric trunk: A rare anomaly

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**A celiomesenteric trunk (CMT) anomaly is extremely rare, occurring in less than 0.5% of people. We describe two cases of CMT anomaly treated surgically for median arcuate ligament compression with stenosis causing intestinal angina and an asymptomatic aneurysm. Disease involving a CMT is extremely uncommon. This comprises the first report of symptomatic median arcuate ligament compression of a CMT. Only four cases of aneurysmal disease are reported so far. Indications and technique of surgical repair of CMT aneurysms are similar to those used for splanchnic artery aneurysms. (J Vasc Surg 2003;37:679-82.)**

Minor variation from the normal developmental anatomy of the visceral arteries, usually aberrant origin of a hepatic artery, occurs in 45% of people.[1] Anomalous origins of the celiac artery (CA) and superior mesenteric artery (SMA) are estimated to occur in 7% of people. A common origin of the CA and SMA from the abdominal aorta is an extremely rare occurrence, reported in 0.5% of the population on the basis of 200 visceral arteriograms and 0.25% on the basis of 500 anatomic dissections.[1,2] Disease arising in such a common celiomesenteric trunk (CMT) has only been reported four times in the literature.[3-6] We describe two cases of CMT, one with stenosis and the other with an aneurysm.

## CASE REPORT

**Case 1.** A 42-year-old woman was referred to our institution for evaluation of recent onset hypertension being treated with two antihypertensive medications. Additional symptoms included postprandial abdominal discomfort, early satiety, nausea, vomiting, and a 30-lb weight loss over 6 months. Physical examination was notable for a thinly built woman (body mass index, 20.3 kg/m$^2$; normal range, 20 to 25 kg/m$^2$) with a blood pressure of 124/84 mm Hg. Systemic examination was unremarkable except for an epigastric bruit.

Laboratory investigation revealed mild proteinuria (36 mg/24 h; normal range, 27 to 93 mg/24 h) and a serum creatinine level of 1.0 mg/dL (normal, 0.6 to 0.9 mg/dL). Serum aldosterone, plasma renin, aldosterone/renin ratio, plasma fractionated catecholamines, and urine metanephrines were all within normal limits, and renal arteries were normal bilaterally on duplex scan examination. Magnetic resonance arteriogram (MRA) revealed normal renal arteries and a common CMT with 60% stenosis. Duplex scan examination confirmed elevated velocities in the CMT, and abdominal computed tomographic scan and upper and lower gastrointestinal endoscopy were normal. Visceral arteriogram showed moderate (60%) CMT stenosis. After selective catheterization of the CMT with a 5F catheter, systolic blood pressure was recorded distal and proximal to the lesion during withdrawal of the catheter. The baseline pressure gradient across the lesion was 18 mm Hg (nonsignificant, <10 mm Hg; Fig 1, *A*).

Operative intervention was performed through a supraumbilical midline abdominal incision. The median arcuate ligament (MAL) was found to form a tight compressive band across the CMT that originated from the aorta at a site caudal to the normal CA origin. An obliterated remnant of an artery in the normal situation of the CA origin contributed to compression of the CMT. MAL release was performed, and the CMT was freed in its entirety. Intraoperative ultrasound scan revealed persistent high flow velocities in the common trunk (300 cm/s; normal peak systolic velocity: CA, <200 cm/s; SMA, <275 cm/s). A polyester patch angioplasty extending from the supraceliac aorta to the bifurcation of the CMT was performed with decrease in flow velocities to 150 cm/s (Fig 1, *B*). The patient recovered from the operation without complications. Ultrasound scan examination performed 3 months after surgery showed patency of the CMT with flow velocities ranging from 120 to 140 cm/s and no change with inspiration and expiration. At this time, the patient was eating better but had not yet regained any weight.

**Case 2.** A 52-year-old woman was referred to us with a diagnosis of SMA aneurysm discovered on MRA. The patient was seen at an outside institution with recent onset hypertension, was found to have an epigastric bruit, and underwent MRA to rule out renal artery stenosis. The patient had no abdominal symptoms, and physical examination was unremarkable except for the epigastric bruit. The patient's condition remained normotensive with no medications. Routine laboratory work-up revealed no abnormality; serum creatinine level was 0.9 mg/dL (normal, 0.6 to 0.9 mg/dL). Visceral arteriogram showed a common CMT with a 2-cm aneurysm arising adjacent to the origin of the SMA (Fig 2, *A*).

An abdominal transperitoneal exposure was performed through a bilateral subcostal incision, and the CMT aneurysm was exposed through the gastrohepatic omentum. The aneurysm was saccular, arising from the SMA just beyond its origin. The left

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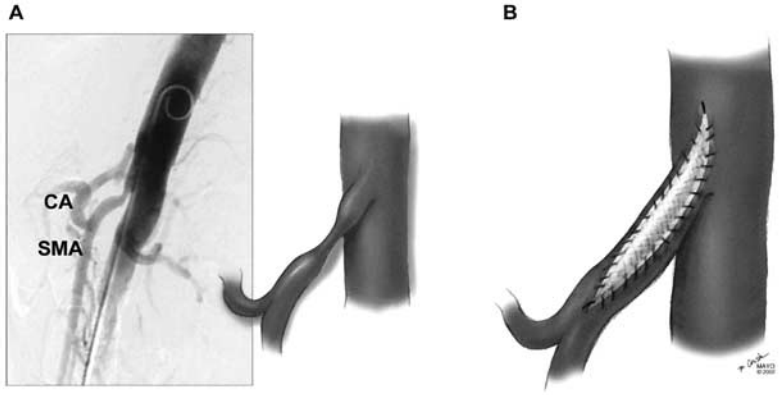


**Fig 1. A,** Lateral aortogram shows common CMT with 60% stenosis. Pressure gradient of 18 mm Hg was found across lesion at rest. **B,** Operative repair performed with MAL release followed by polyester patch angioplasty.

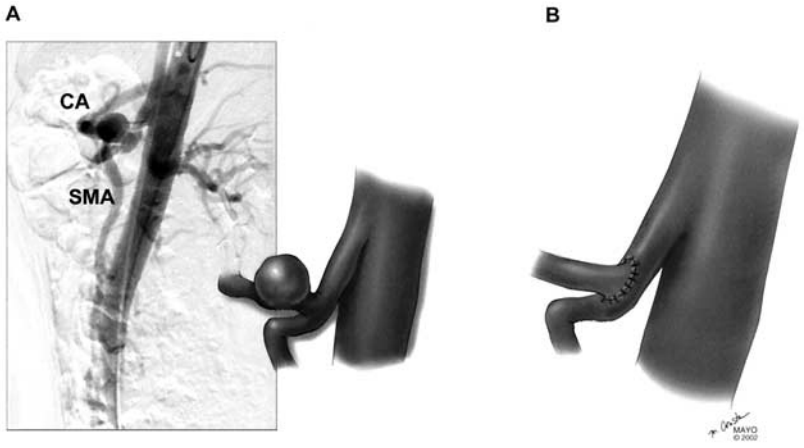


**Fig 2. A,** Visceral arteriogram shows common CMT with 2-cm saccular aneurysm arising adjacent to origin of SMA. **B,** Operative repair performed with aneurysmectomy and reimplantation of celiac trunk onto SMA.

gastric, splenic, and hepatic arteries arose from a short celiac trunk off the side of the aneurysm. The aneurysm was completely resected, and the CA stump was anastomosed primarily end-to-side to the defect in the SMA (Fig 2, *B*). Pathologic examination revealed a true aneurysm with no evidence of vasculitis or atherosclerosis. The patient recovered from the procedure uneventfully. Computed tomographic examination 8 months after surgery showed patency of the CMT reconstruction.

## DISCUSSION

The splanchnic arteries arise at an early stage of fetal development (fourth week) as paired ventral segmental or vitelline arteries from the paired dorsal aortae to supply the yolk sac.[7] They are soon reduced in number by fusion concomitant with fusion of the dorsal aortae and by disappearance of several stems from their origins. The roots persisting form single midline vessels in the dorsal mesentery. Progressive preaortic longitudinal anastomoses form with successive recruitment of more caudal stems and dissolution of cephalic roots, enabling caudal migration along with migration of the gut (Fig 3). Eventually, four ventral roots remain (Fig 3, *C*); roots 1 to 3 coalesce to form the celiac trunk and the fourth root develops into the SMA. The ventral longitudinal anastomosis between roots 3 and 4 is normally interrupted to result in anatomic separation of the origins of the CA and SMA.[7] A common CMT would result from failure of disruption of this anastomosis.

This report comprises the first case of MAL compression of a CMT. In spite of reported symptomatic relief in more than two thirds of patients after MAL release, the true existence of MAL compression as a cause of intestinal angina has been questioned by many.[8] In the face of a rich collateral network between the CA and SMA, symptoms attributable to stenosis of the CA alone are difficult to explain, and the diagnosis of MAL compression is always subject to skepticism. In addition, radiologic evidence of CA compression is not infrequently documented in asymp-

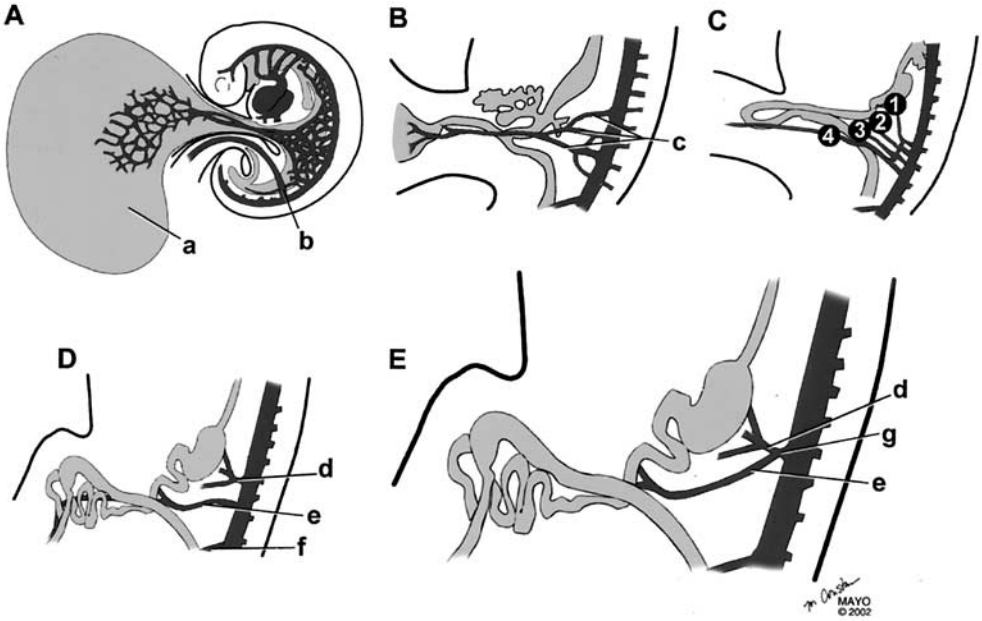


**Fig 3.** Embryologic development of CA and SMA. Week 4 to 9 of intrauterine life. **A,** Multiple ventral longitudinal anastomoses form between segmental arteries. **B,** Caudal migration of splanchnic vessels by recruitment of caudal roots and loss of cephalad roots. **C,** Four roots remain to supply foregut and midgut. **D,** Interruption of longitudinal anastomosis between roots 3 and 4 to separate origins of CA and SMA with resultant adult configuration. **E,** Failure of dissolution of anastomosis between roots 3 and 4 results in formation of common CMT. *a,* yolk sac; *b,* umbilical artery; *c,* ventral segmental arteries; *d,* celiac axis; *e,* SMA; *f,* inferior mesenteric artery; *g,* common CMT.

Reported cases of operative repair of CMT lesions

| *Author* | *Year* | *CMT lesion* | *Age (y)/gender* | *Repair* | *Histology* |
|---|---|---|---|---|---|
| Stanley et al[3] | 1970 | Aneurysm | 48/Male | Aneurysmectomy +? | ? |
| Bailey et al[4] | 1991 | Aneurysm (saccular) | 46/Female | Aneurysmectomy + polyester patch | Medial degeneration and atherosclerosis |
| Detroux et al[5] | 1998 | Aneurysm (saccular) | 51/Male | Aneurysmectomy + suture of neck | Atherosclerosis |
| Matsumoto et al[6] | 1999 | Aneurysm (fusiform) | 53/Male | Aneurysmectomy + polytetrafluoroethylene interposition graft | Atherosclerosis |

tomatic patients. However, it is somewhat easier to explain the symptoms of postprandial pain and weight loss in our case on the basis of the anatomic lesion resulting from compression and stenosis of the CMT with compromise of both CA and SMA circulatory beds. In fact, occasional cases of MAL compression resulting in occlusion of the CA and stenosis of the SMA have been reported in the literature.[9] A solitary report in identical twins highlights local anatomic development as a causative factor, which was undoubtedly a key contributor in our case.[10] Early relief from postprandial pain in our patient is encouraging; however, whether the patient starts regaining weight remains to be seen. Further follow-up is necessary to make definite conclusions regarding the significance of the CMT lesion in causing the symptoms.

Celiac artery and SMA aneurysms account for less than 10% of all splanchnic artery aneurysms. Mycotic aneurysms and postdissection aneurysmal dilatation account for more than half of all SMA aneurysms.[3] Atherosclerosis is seen in only 20% to 27% of aneurysms affecting the CA and SMA and is more likely a secondary phenomenon.[11] Medial degeneration is a frequent cause of celiac aneurysms, with 17% reported to be developmental in origin. Congenital defects in the muscle and elastic layers of the arterial wall at branch points have been hypothesized to originate from two different mechanisms: a vestigial origin from persistence of an embryonic vessel and a mesenchymatous origin from inclusion of defective mesenchymatous tissue during differentiation.[12]

Surgical treatment of only four aneurysms involving a CMT is reported in the literature (Table). All occurred in relatively young patients and were situated at the bifurcation of the CMT into the CA and SMA. Pathologic results revealed early atherosclerosis or medial degeneration. A

congenital cause could be postulated in the background of the CMT amomaly in view of the fact that all five reported patients were a decade younger than the mean age of presentation of CA and mesenteric artery aneurysms, that all aneurysms occurred at the bifurcation of the CMT, and that an absence of infection, arteritis, or advanced atherosclerosis on pathologic examination was seen.

## SUMMARY

Disease involving the rarely encountered CMT anomaly is extremely uncommon. This is the first reported case of symptomatic MAL compression of a CMT. Stenotic lesions of a CMT potentially endanger both celiac and superior mesenteric vascular beds. Aneurysmal degeneration of the anomalous CMT may be a consequence of abnormal embryologic development. Operative intervention should be performed on the basis of the same size criteria used for splanchnic artery aneurysm repair. The technique of repair undertaken depends on the location, size, and extent of the aneurysm.

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