Celiac Axis Compression Syndrome
A Critical Review

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The celiac axis compression syndrome (CACS) has been a subject of controversy since the description by Harjola in 1963 of a patient with abdominal pain associated with external compression of the celiac axis (1). Two basic questions concerning the syndrome remain unresolved: (1) does the entity really exist as a cause of abdominal pain, and (2) if so, is the pain a result of gastrointestinal ischemia?

In this review we have critically evaluated the available clinical and experimental data to determine the bases for the partisan opinions held on both sides of the questions. A MEDLARS search was initiated for articles published between 1960 and 1976 with the term "celiac axis" in their title. Of 327 publications listed, 95 concerned isolated celiac axis stenosis or occlusion. Of these, 61 were in major journals and written in English, French, German, Italian, or contained English summaries; these were retrieved and reviewed. Of these 61 articles, 38 provided the basis for a clinical review. Twenty-eight of these were reports of clinical series, 7 of radiological series and 3 were experimental studies. Single case reports and articles which were grossly incomplete in their description of patient material were not included.

Despite the volume of publications devoted to the subject, it does not appear to us that there is conclusive clinical or experimental data which either establish or exclude compression of the celiac axis as a cause of abdominal pain. Moreover, there is even less evidence that gastrointestinal ischemia is the cause of the pain described in association with this anatomic abnormality.

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HISTORICAL REVIEW

Harjola in 1963, described a single patient with postprandial pain and an epigastric bruit in whom radiographic examination of the stomach was normal. The angiogram showed a narrowing of the celiac axis which at surgery was shown to result from constriction of the artery by a fibrotic celiac ganglion. The artery was released, the murmur disappeared, and the patient remained free of his postprandial pain. Harjola thought this an example of retroperitoneal fibrosis. Two years later, Rob reported several similar cases but implicated an anatomic variation of the celiac ganglion as the cause of the arterial compression (2). Also in 1965, abdominal pain associated with diaphragmatic compression of the celiac axis was first reported by Dunbar and associates (3). Although earlier reports of the anatomic relationships of the celiac axis and the diaphragm were made by Lipshutz in 1917 (4), Adachi in 1928 (5), and George in 1934 (6), it was in 1955 that Michels observed many cases in which the caliber of the celiac trunk was diminished at its origin by a constrictive action of the crura of the diaphragm (7). By the mid-1960s therefore the concept of a painful abdominal syndrome associated with external compression of the celiac axis was established in the medical literature.

ANATOMY

The celiac axis arises from the anterior aspect of the abdominal aorta at a level that most frequently is between the lower third of the 12th thoracic vertebra and the middle third of the first lumbar vertebra (8). This level is inconstant as a result of the vessel's variable caudal descent during embryonic development. Two important anatomic structures that are intimately related to the celiac axis are the

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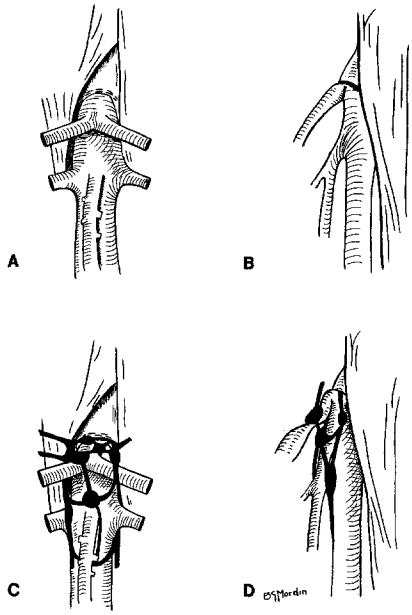

Fig 1. Anatomy of celiac axis compression syndrome. (A) Normal anatomic position of median arcuate ligament cephalad to the origin of the CA. (B) With abnormally high origin of the CA or low position of ligament, the proximal portion of the artery is compressed. (C) Normal anatomic position of the celiac ganglion. (D) Compression of proximal portion of CA by ganglion.

median arcuate ligament of the diaphragm and the celiac ganglion (Figure 1).

The median arcuate ligament is a highly variable structure both in location and in composition, ranging from a well-defined anatomic entity to an amorphous mass of connective tissue. The level at which it crosses the aorta varies with the age, sex, and habitus of the patient and is generally highest in the young female who is ectomorphic (9). However, it is usually sufficiently close to the celiac axis so that compression of the artery may occur during expiration and be relieved during inspiration (10) (Figure 2). This is so because, during inspiration, the celiac axis and aorta move caudad and the median arcuate ligament moves ventrally (11). Thus, most epigastric bruits in patients with the CACS are accentuated during expiration and diminished during inspiration (10, 12).

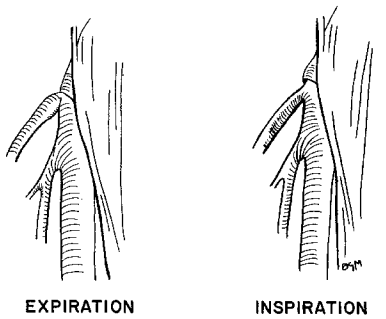

Fig 2. The effect of respiration on compressed celiac axis. With inspiration, the aorta and CA move downward in relation to the median arcuate ligament which moves ventrally. This relieves the compression of the CA.

The celiac ganglion consists of left and right divisions, the fibers of which interconnect around the celiac axis and surround it 3–5 mm ventral to its point of origin from the aorta. In approximately 50% of the patients, it joins with fibrous and fatty tissue to form a thick and potentially compressing shield anterior and inferior to the median arcuate ligament (9) (Figure 1c and d).

CLINICAL FEATURES

In our review of the 28 clinical series (3, 12–38) which include 330 patients with celiac axis compression, one of the major difficulties in determining the validity of the syndrome was the different criteria used by various authors to define it. Abdominal pain was the sole consistent symptom and even an adequate description of the character of the pain was found to be lacking in the majority of reports. Thus in only 30% of reports was any mention made of the relation of the pain to eating, but in these the pain was characteristically postprandial. Descriptions of the pain typical of intestinal angina, ie, pain beginning 30–45 min after a heavy meal and lasting approximately 1–3 hr was notably absent. The location of the pain, when described, was most often epigastric although occasionally was in the right or left upper quadrants. Rarely, flexing of the knees or assuming a recumbent position were mentioned as maneuvers to relieve the pain. Diarrhea or loss of weight were noted in only 50% of the cases, and the average loss, when described, was 20 pounds. Upper-gastrointestinal symptoms including nausea and vomiting were mentioned in approximately 25% of the patients.

An abdominal bruit, the only physical sign considered to be an essential feature of the CACS by some authors, was frequently not mentioned and in rare instances was specifically stated to be absent. Even when an abdominal bruit is present, however, its diagnostic significance must be questioned, since epigastric bruits occur in 6.5 (12)–15.9% (39) of healthy adults. It has recently been suggested that

while the presence of the bruit is nonspecific, its phonoarteriographic characteristics may be more diagnostic (40). Thus, bruits that are loud, long (mean maximum duration of 0.50 sec), begin in early or mid-systole and extend into early diastole are usually heard with celiac axis stenosis. Conventional radiologic opacification studies of the stomach and small bowel, when reported, were usually normal.

Results of absorption tests were included infrequently in the reported diagnostic studies and evidence of malabsorption was extremely rare. Only one case of CACS and intestinal malabsorption was found in the literature in which sufficient pre- and postoperative data were presented to associate celiac axis compression with the malabsorption (41); even in this case the mechanism of the malabsorption syndrome was not elucidated. Jejunal biopsies were infrequently performed and were most often normal (29). When abnormal, no specific features were noted to aid in the diagnosis of the CACS.

There have been several reports of an association of celiac axis stenosis and pancreatitis, but documentation of improvement after decompression of the artery is uncommon (26, 42, 43). However, in one recent study (37) 3 of 12 patients with CACS but no evidence of pancreatitis had abnormal pancreatic function tests which improved after operative decompression. Of two other patients stated to have pancreatitis and an occluded celiac axis, one became asymptomatic upon decompression whereas the other did not. In the latter patient arteriography demonstrated the celiac axis to still be occluded.

ANGIOGRAPHY

Some radiologists believe that compression of the celiac axis is best demonstrated by lateral aortography (44), while others prefer a selective injection of the artery (45). If a selective injection is employed, care must be taken not to advance the catheter past the point of arterial narrowing. Rapid sequence filming is important because the origin of celiac axis is usually seen clearly only during the initial 0.5 seconds of the film sequence. However, even if the stenosis is not seen, its presence can be inferred from collateral flow patterns and subsequently confirmed by lateral aortography (45). Compression of the celiac axis by the crural fibers of the diaphragm, the celiac ganglion, or both produces a smooth asymmetric narrowing of the superior aspect of the artery and possible inferior displacement of it towards the superior mesenteric artery (Figure 3). These findings vary with respiration (10, 11). During inspiration the celiac axis and the aorta may move cauded an average of 3 mm and 6 mm, respectively, while the median arcuate ligament moves ventrally an average of 8 mm. This divergent movement of the celiac axis and median arcuate ligament reduces the compression of the artery and explains the diminution of the epigastric bruit during inspiration in the CACS (Figure 2).

CELIAC AXIS NARROWING ON ANGIOGRAPHY—SIGNIFICANT OR NOT?

The clinical significance of angiographically demonstrated celiac axis narrowing remains controversial. Bron and Rodman (46) found the celiac axis to be the most commonly occluded or stenosed splanchnic vessel in their review of 713 flush aortograms but reported the incidence of stenosis or occlusion of that vessel to be only 12.5%. In a later study of 50 patients with no gastrointestinal symptoms who underwent abdominal angiography, 12 had celiac axis narrowing of 50% or greater, usually with the characteristic configuration associated with compression by the median arcuate ligament or celiac ganglion (47). In an analysis of the arteriograms of 185 patients evaluated for unexplained abdominal or back pain or weight loss, Meaney and Kistner (48) found 50 patients with occlusive disease of the celiac axis, 15 of whom had the characteristic angiographic appearance of celiac axis compression. However, the clinical symptoms of patients without occlusive disease were essentially the same as in those with diseases compromising 50% or more of the arterial lumen.

More recently Szilagyi et al (32) reviewed 157 angiograms from three goups of patients: (1) those in whom intestinal angina was suspected, (2) those with gastrointestinal diseases not primarily characterized by pain, and (3) those with miscellaneous diagnostic problems not involving the alimentary tract. The incidence of narrowing was similar in all three groups (44%, 52.8%, 50%), but the more severe degrees of narrowing occurred most frequently in the miscellaneous group.

The surprisingly high frequency of celiac axis narrowing among asymptomatic individuals and the lack of close correlation between the severity of symptoms and the degree of narrowing in symptomatic patients indicate the need for considerable

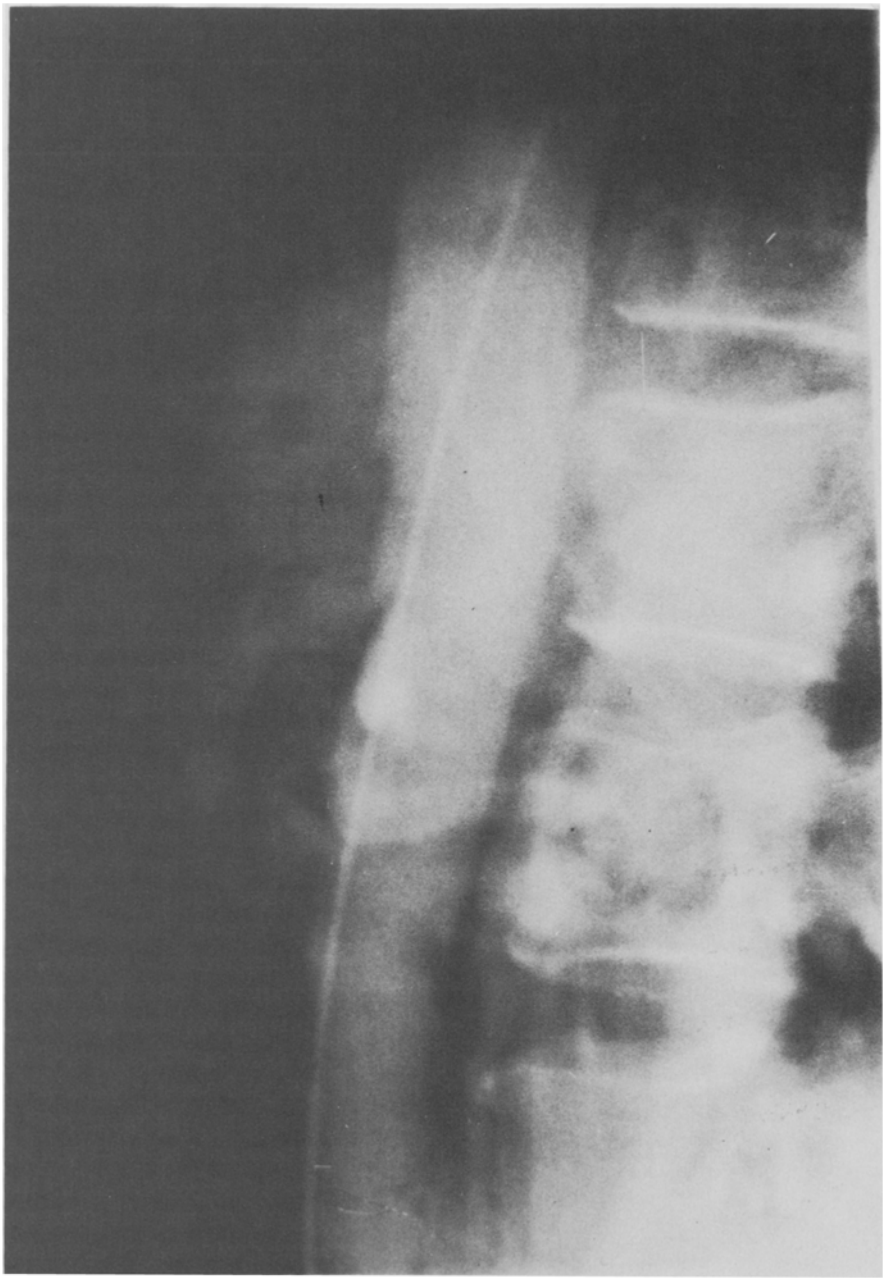

Fig 3. A lateral aortogram demonstrating the smooth asymmetric narrowing of superior aspect of the celiac axis.

caution in attributing a patient's symptoms to the arterial stenosis.

PATHOPHYSIOLOGY OF PAIN—IS IT ISCHEMIC?

Since the anatomic lesion forming the basis for the syndrome is narrowing of the major artery to the upper abdominal viscera, the pain has most frequently been attributed to ischemia (16, 19). This concept has persisted in spite of voluminous clinical and experimental evidence that isolated compromise of the celiac axis is almost always compensated for by collateral circulation from both the superior and inferior mesenteric arteries. A clear demonstration of the immediate availability of ade-

quate collaterals was supplied by Appleby (49) who, as therapy for gastric carcinoma, excised the celiac axis together with the origins of the left gastric, splenic, and hepatic arteries with no ill effects in 13 patients. Similarly, in experiments in dogs, 50–100% constriction of the CA produced no gross or microscopic changes in the stomach, duodenum, liver, or pancreas, despite the creation of significant pressure gradients between the celiac axis and the aorta (50). Extensive experience gained since the advent of angiography has additionally demonstrated that gradual atherosclerotic occlusion of two or even three of the major mesenteric arteries is common in asymptomatic patients and that narrowing of at least two of three major abdominal arteries must be present before abdominal angina occurs (51). Hence, there is strong evidence that chronic and acute interruption of the celiac axis are well tolerated if normal collaterals are present. Ischemia due simply to diminution in celiac flow, therefore, is an unlikely explanation for the pain of the CACS.

A more complex concept of an ischemic origin of the pain implicates a "steal phenomenon" between the celiac and mesenteric circulations. According to one theory, the collateral flow from the superior mesenteric artery to the celiac bed deprives the small intestine of an adequate blood supply. After eating, this deficiency is accentuated by the increased demand for blood by the small intestine producing "intestinal angina." This is supported by the provocative *d*-xylose tests performed preoperatively by Stanley and Fry (28) in 3 patients with the CACS. Postoperatively 2 patients were reevaluated and no longer showed the impaired absorption of *d*-xylose following a meat meal. Although a reduced *d*-xylose absorption was also demonstrated experimentally (52) following celiac axis occlusion, corroboration of their observations and elimination of other possible factors affecting absorption, eg, altered motility, are required before the significance of this test is established. Conversely, another theory is that increased blood flow to the small bowel after eating decreases the collateral flow to the celiac bed producing ischemic pain in the viscera supplied by the celiac axis (22). Both theories are predicated on the "borrowing–lending" concept of distribution of blood flow, namely that increasing blood flow to one segment of the vascular bed is achieved only at the expense of flow to another (53). However, during extensive animal studies of the mesenteric circulation (54), we have observed that increasing blood flow to one segment of the splanchnic bed is associated with increased rather than decreased flow to the rest of the bed. Furthermore, if one major vessel is partially occluded, collateral blood flow develops promptly, and increasing flow to either the bed of the occluded or unoccluded vessel is associated with increased flow in both beds. Only when both the superior mesenteric artery and the celiac axis are partially occluded so that splanchnic arterial inflow is fixed and limited, does increasing flow to one segment produce a decrease in flow to others. These observations indicate that at least under acute conditions the occurrence of a vascular steal within the splanchnic circulation is unlikely unless flow through both the celiac and mesenteric arteries is compromised. As this is not the case in most patients thought to have the CACS, the concept that the abdominal pain is the result of a form of mesenteric or celiac steal syndrome seems unlikely.

Stanley and Fry (28) also have attempted to explain the pain on the basis of diminished pulsatile flow despite a normal volume flow in the distal mesenteric branches. They attribute this phenomenon to "excessive flow velocity of inordinately large volumes of blood coursing through the proximal superior mesenteric artery prior to its egress through collateral channels to the celiac arterial bed." While this is an interesting theory, there is no experimental data to support it.

ALTERNATIVES TO ISCHEMIA

A widely accepted alternative to the ischemic explanation for the pain in the CACS is that the pain arises in the celiac ganglion itself, possibly from pressure or throbbing by the compressed artery (17). Some support for this theory is provided by several authors who obtained better results when the ganglion was excised than when only the ligament was incised. However, celiac ganglionectomy and periarterial sympathectomy also result in an increase in celiac axis blood flow (22).

Lastly, a less-accepted explanation for the pain is that compression of the celiac axis produces multiple small emboli to the upper abdominal organs (55).

THERAPY

The most difficult aspect of the treatment of CACS is the selection of patients for operative relief

of the compressed artery. Results of surgical procedures have varied from series to series, and no specific criteria can be well correlated with a successful outcome. In view of the continuing lack of objective evidence that stenosis of the celiac axis produces any pathologic changes in the viscera supplied by that artery, only patients fulfilling strict criteria should be operated upon. These criteria are abdominal pain, preferably related to eating, significant weight loss, an abdominal bruit, and angiographic demonstration of the typical narrowed celiac axis. It is interesting that in a large medical center with special interests in vascular surgery and vascular disorders of the gastrointestinal tract, we have not encountered a single patient with these findings during the past ten years.

The operative approach to CACS varies with the surgeon's beliefs concerning the cause of the pain. Those who believe it is ischemic emphasize the necessity for reestablishing celiac axis blood flow either by division of the median arcuate ligament, with or without ganglіectomy, or by arterial reconstruction or bypass. Those who believe that the pain is neurogenic emphasize the division or resection of the celiac ganglion. Few surgical reports describe a plan of operative management that is individualized for each patient based upon intraoperative angiography or measurements or pressure gradients (3, 12, 15, 18, 19, 22, 31, 36).

In one series reported by Lord et al (18) such an approach was employed in 8 of their 12 patients whose median arcuate ligaments were divided without a change in gradient between the aorta and celiac axis (average 33 cm water) and with persisting abnormal caliber of the celiac axis. These patients then underwent an arterial reconstruction subsequent to which all gradients were abolished; wide patency of the reconstructed segment was demonstrated in seven of the eight patients, all of whom were symptom-free at follow-up an average of 26 months later.

Excellent immediate results following operation have been reported by most authors, but critical analysis of large series and recent long-term evaluations have tempered much of the early enthusiasm for these procedures. Contradictory results have been attributed to patient selection, poor follow-up, and varying operative techniques. Surgical series are also difficult to compare because of the paucity of reports of intraoperative arterial pressure gradients and the lack of adequate postoperative evaluation.

However, in the largest study of the long-term results of patients treated for the CACS, Evans made two pertinent observations (35). First, although 83% of 47 patients were asymptomatic six months after a decompression procedure, only 41% remained asymptomatic 3–11 years later. Furthermore, no correlation existed between the presenting symptoms and the results of surgery, and no clinical patterns emerged to identify those patients that might benefit from surgery. Moreover, of 12 patients treated nonoperatively, nine remained free of pain at the time of his report. In most surgical series in which gradients were measured, a good correlation existed between anastomotic patency, abolition or reduction of the pressure gradient between the celiac axis and the aorta, and a symptom-free state. This is not a universal experience, however, as shown by Edwards et al (12), who measured blood flow in the hepatic and splenic arteries intraoperatively in four patients. While cautioning the applicability of these data obtained under conditions of mechanical inflation of the lungs to those while the patient is upright and breathing naturally, they observed that that patient with the greatest improvement in hepatic artery flow following celiac axis decompression showed no improvement of symptoms. In most series reviewed, some patients are reported in whom symptoms resolved or dramatically improved despite the postoperative angiographic demonstration of a defect in the celiac axis or the persistence of a gradient between the celiac axis and the aorta. Conversely many patients are reported in whom symptoms persist in the absence of any objective data demonstrating an anatomic or functional reduction in celiac axis blood flow. In a recently reported surgical series of 6 patients with findings typical of the CACS, a uniform negative correlation was found between surgical correction of the celiac axis stenosis and relief of symptoms (38). No patient was benefited more than temporarily by the surgery, despite angiographic or hemodynamic proof of operative correction. Additionally, angiographic evaluation in all patients demonstrated that despite high-grade stenosis of the celiac axis, there was no radiographically discernible reduction in contrast flow to the celiac axis because of well-developed collaterals from the superior mesenteric artery.

THE FUTURE OF THE CACS

The controversy concerning CACS is a continuing one. At this time we agree with Watson and

Sadikali (37) that there are a small number of patients "with distressing abdominal pain not explained by customary diagnoses and not helped by customary management" who are relieved by some aspect of the operations performed for celiac axis compression. We do not view the entity as a well-defined one with a well-understood pathophysiologic basis and especially do not support the concept that ischemia plays a pathogenetic role in the syndrome. It is our belief that the pain is most likely neurogenic in origin, resulting from pressure or pulsations from the conpressed artery. The relationship of the pain to meals can be explained by the increased splanchnic blood flow and dilatation of the artery which accompanies the ingestion of food. Thus in the surgical approach to the few patients requiring operation, the median arcuate ligament should be incised and the neural tissue and ganglia adjacent to the artery should be excised. This should be followed by a measurement of the aortic–celiac axis pressure gradient and a celiac axis angiogram. If a gradient is present or the angiogram reveals a defect in the celiac axis, a procedure to reestablish normal blood blow is performed—either arterial reconstruction or bypass. If only patients fulfilling the criteria previously described and operated upon, unnecessary procedures should be kept to a minimum. Detailed preoperative and postoperative angiographic, hemodynamic, and metabolic evaluations should ultimately provide the answers to this puzzling entity.

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