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A Review on the Celiac Trunk Compression Syndrome: Some Anatomic Clinical-Surgical Aspects

Una Revisión del Síndrome de Compresión del Tronco Celíaco: Algunos Aspectos Anatómicos Clínicos-Quirúrgicos

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SUMMARY: The objective of this study was to review some celiac trunk compression syndrome aspects such as: symptom-posture relationship; absence of symptoms; syndrome-age relationship; angiographic study on anatomy of the celiac trunk stenosis; congenital or acquired origin; invasive diagnostic tests; surgical and postoperative results.

KEYWORDS: Arterial occlusive disease; Celiac artery; Celiac plexus; Diaphragm; Syndrome

Clinical radiologic study of the correlation between abdominal symptoms with celiac trunk compression by diaphragm crura, introduced a new nosological entity into the abdominal vascular pathology field – the celiac trunk compression syndrome (Dunbar *et al.,* 1965). Since then, several cases have been reported with this syndrome (Debray & Leymarios, 1968; Fadhli, 1968; Harjola, 1968; Edwards, 1969; Hivet & Lagadec, 1970; Stanley &Fry, 1971; Joubaud *et al.*, 1977).

Several terms have been proposed for its designation, as compression of the celiac trunk (Dunbar *et al.*; Terpstra, 1966), celiac trunk syndrome (Harjola & Lahtiharju, 1968), celiac compression syndrome (Marable *et al.*, 1968), median arcuate ligament syndrome (Carey *et al*., 1969), compression of the celiac trunk by arcuate ligament of the diaphragm (Hivet & Lagadec), aortic channel syndrome of the diaphragm (Bobbio, 1968).

The term "median arcuate ligament syndrome" came to be often quoted as "celiac trunk compression syndrome", as if to emphasize the hemodynamic aspect of its cause (Szilagyi *et al.*, 1972). Warter *et al*. (1976) chose to denominate it as a phrenoceliac disease, taking into consideration the most complete expression to identify either its etiologic nature or the pathogeny of the lesion.

Symptoms of the celiac trunk compression syndrome. The symptoms of this syndrome are characterized by the triad, postprandial abdominal pain of cramp like nature, disorders in the function of intestinal canalization and weight loss. This symptomatology has similar characteristics to intestinal angina caused by atherosclerotic occlusive lesions of the major splanchnic vessels (Mikkelsen & Zaro, 1959; Dunbar *et al.,* 1965; Bobbio *et al.*, 1967; Curl *et al.,* 1971). However, postprandial discomfort is less severe than in intestinal angina (Marable *et al.*, 1966).

There are some clinical aspects that may differentiate the celiac trunk compression from atherosclerotic celiac-mesenteric ischemia, such as the predominance of pain up to the level of the superior quadrant of the abdomen, and the loss of weight due to intentional reduction in the amount of food and not as a result of malabsorption process (Bobbio *et al.*; Bobbio & Zanella, 1971). Also, differently from diarrhea with blood in the feces and of occasional malabsorption of the atheromatic mesenteric arterial diseases, diarrhea in the celiac trunk syndrome is more related to the irritable bowel than to malabsorption (Gutnick, 1984).

Age range related to the occurrence of syndrome. Several authors have observed that this disorder mainly affects young females, between 20 and 50 years old (Dunbar *et al*.; Marable

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et al., 1966; Lord *et al.*, 1968; Cormier & De La Fontaine, 1970; Warter *et al.,* 1970a; Loffeld *et al.*, 1995). The female-male ratio is 4:1 (Trinidad-Hernandez *et al.*, 2006). Age can eliminate the diagnosis of mesenteric arterial insufficiency of atherosclerotic origin (Marable *et al*., 1966) and the predominant action of the atheroma (Warter *et al*., 1970a). Atheroma plaques are not frequently associated to celiac trunk stenosis by median arcuate ligament (Warter *et al*., 1970a). Nevertheless, it was observed in patients with ages ranging from 60 to 80 years, celiac trunk compression associated to atherosclerosis located in the narrowed segment of this artery (Bobbio & Zanella; Drèze *et al*., 1972; Marable *et al*., 1968; Sautot, 1962).

Higher syndrome incidence in ectomorphic females can be explained by closer relationship between celiac trunk and the arcuate ligament. In these females the origin of both structures is frequently higher (Lindner & Kemprud, 1971). Angiographic studies in 17 patients, ten of them asymptomatic, with occlusion or severe stenosis of the celiac trunk, trying to correlate the abdominal angina to the unintentional discovery of the lesion, revealed that most of the symptomatic patients are under the age of 40 years and suffered from arcuate ligament syndrome. The asymptomatic ones are older and affected by various degrees of atherosclerosis (Cornell, 1971).

The occurrence of this syndrome in mid-age patients is not understandable since mediane arcuate ligament anomaly seems to be congenital. However, a feasible answer is that the effects of the compression may become manifest clinically later in life when the hemodynamic compensation mechanisms due to compression begin to fail (Warter *et al.*, 1970a; Bobbio & Zanella).

Manifestation of this syndrome during the childhood or adolescence has seldom been described (Foertsch *et al*., 2007).

Relationship between body posture and pain. Clinical reports demonstrate that some body position postures have been adopted by patients suffering from this syndrome in order to relieve pain. The relief came adopting a knee-chest position (Dunbar *et al.*; Marable *et al.,* 1966; Fadhli; Harjola, 1968; Houdard *et al*., 1970; Leger *et al*., 1970; Drèze *et al*.; Joubaud *et al*.; Abate *et al*., 1980; Lawson & Ochsner, 1984; Mulder *et al.,* 1971; Tseng *et al*., 2007), by abdominal pressure (Lawson & Ochsner), by resting (Edwards *et al*., 1970), by lateral decubitus (Joubaud *et al*.) and in recumbency position (Leger *et al*., 1970). It was suggested that the knee-chest position would relieve the pressure on the celiac trunk by gravitating the viscera and diaphragm forward (Fadhli). It was also observed that pain worsens under physical effort (Fadhli; Edwards; Hived & Lagadec; Joubaud *et al*.; Williams *et al.*, 1985), hours in a standing position (Joubaud *et al*.; Dreze *et al*.), upright posture, fatigue, emotional disorder, hunger (Willians *et al*.; Warter et al., 1976), recumbency (Edwards *et al*.; Plate et al., 1981) sitting position and decubitus (Van De Berg *et al.*, 1972), during running (Baldasarre *et al*., 2007) and physical activity (Mensink *et al*., 2006; Foertsch *et al*.). These results indicated that, under physical effort, the arcuate ligament contraction against aorta causes discomfort (Hivet & Lagadec). Thus, the postural influence becomes an eventual sign of celiac trunk compression syndrome (Edwards).

Angiographic studies of the celiac trunk stenosis anatomy. The celiac trunk is originated in the anterior face of the aorta and draws a concavous superior and anterior curve (Hivet & Lagadec). The curve in "S" is lost with the compression by arcuate ligament (Olivier *et al.*, 1970).

Some anomalous aspects of the celiac trunk direction and caliber obtained by lateral or profile aortography allowed the diagnosis of stenosis of this vessel by the median arcuate ligament, these signals being the precise evidence of this lesion. One of them is the vertical direction of the proximal segment of the celiac trunk, in its first centimeters, becoming parallel and compressed against the anterior wall of the aorta by the inferior margin of the arcuate ligament, that considerably compresses its lumen and provides to this vessel a notch in its anterior face shaped like an "ax blow" or "flute peak" (Hivet & Lagadec; Huguet *et al*., 1972; Joubaud *et al*.; Gutnick). Another aspect is the reduction or suppression of the 90° angle between the posterior face of the celiac trunk and the subjacent aorta, to visualizing the stenosed segment flattened against the aorta. The vertical course of the first centimeters of the celiac trunk leads to the disappearance of the parallelism with normal superior mesenteric artery (SMA), which acquires a closer proximity to that vessel (Bobbio *et al*.; Furnemont, 1974). Besides these aspects, poststenotic dilatation after the narrowed portion of the celiac trunk is frequent (Warter *et al*., 1970b; Tongio *et al*., 1971; Huguet *et al*.; Joubaud *et al*.). Also, distally to the "notch", the celiac trunk assumes a horizontal or oblique ascendant deviation (Olivier *et al.*; Hivet & Lagadec; Warter *et al*., 1976) as an "elephant's trunk" (Bobbio *et al*.) or a "hook" (Tongio *et al*.; Karaban *et al.*, 2007).

Two types of stenosis were observed by the lateral angiography in stenosis or occlusion cases, in the origin or proximal third of the celiac trunk , accordingly to the lumen involvement: the eccentric and the concentric stenosis. In the eccentric type, the lumen is inferiorly and/or superiorly asymmetrically compromised, and in the concentric type,

circumferentially (Bron & Redman, 1969). The compression of the celiac trunk by the crural fibers of the diaphragm and/or celiac ganglion produces an eccentric type stenosis in the superior part of the celiac trunk, differentiating from atheromatous stenosis, which is generally of a concentric type (Deutsch, 1968; Bron & Redman and Brandt & Boley, 1978). Sometimes the presence of a periarterial neurofibrous envelope of the celiac trunk reveals itself as a concentric stenosis (Harjola & Lahtiharju; Tongio *et al.*).

Szilagyi *et al.* defines the terms compression and stenosis. So, stenosis generally assumed to be due to athereosclerotic changes in the intimae and appeared as a concentric diminution of the circumference of the artery, and compression appeared as an indentation in the downward curvature of this artery appearently brought about by the pressure of a sharp edge.

The compression of the celiac trunk by the median arcuate ligament acquires characteristics of stenosis that occurs slightly distant from the ostium, smooth and mostly short (Broussin *et al*., 1970).

Proposals were made in order to classify the different morphological and radiological characteristics of the celiac trunk stenosis, considering its more or less accentuated degrees (Warter *et al.*, 1970b; Colapinto *et al*., 1972; Di Marino *et al.*, 1972).

Warter *et al*. (1970b), in a study using lateral angiography of 48 individuals aged between 12 and 72 years (the majority with less than 50 years), with celiac trunk stenosis and, comparing the cases that have been published in literature, classified this stenosis into five types. According to the proposed classification, diaphragmatic stenosis of the celiac trunk is the most frequent, affecting younger individuals and figures in type I with three subtypes. Type I is characterized by an initial narrowing of the celiac trunk and a poststenotic dilatation with well established etiology in the literature. In IA and IC subtype, the narrowed and long part of the celiac trunk seems compressed against the aorta while in IB subtype, we noticed an empty gap between the stenosed segment and the aorta. Type II confers to the celiac trunk a fusiform aspect with or without poststenotic dilatation and can also identify in this type the atheromatous isolated stenosis (Dunbar *et al*.) and the fibromuscular hyperplasia of the celiac trunk (Ripley & Levin, 1966). In type III, it is possible to verify the presence of superior and lower notches in the celiac trunk, with no poststenotic dilatation, including the compression by the celiac plexus hypertrophyated ganglions (Snyder *et al.*, 1967) and by the periarterial fibrous invelope like a ring (Harjola, 1963). In type IV, the caliber of the celiac trunk is reduced and includes the Reuter & Olin's atheroma (1965). Type V is characterized by complete obliteration of the celiac trunk, as in the congenital atresias (Morettin *et al*., 1965; Chavez *et al*., 1966), in the pancreatitis (Frumusan *et al.*, 1968), and in the Debray & Leymarios´s retroperitoneal fibrosis.

If pathognomonic type IA is the most frequent and is found mainly in adults, it seems that in young people or in the slim woman the narrowed segment is longer (type IB) (Dreze *et al*.).

A similar study obtained by Colapinto *et al.* on 74 patients aged between 16 and 85 years, average of 50.9 years, classified four types of stenosis of the celiac trunk according to its prominent morphologic aspect. It was associated to Type I, a celiac trunk compression by the arcuate ligament or ganglia tissue and to the other II, III and IV types, the stenosis of atherosclerotic nature. Forty-seven cases (63%) belonged to type I, and 30 of these with stenosis greater than 50%. Type I occurred in all ages and both sexes.

Therefore, the majority of the angiographic images corresponding to the extrinsic celiac trunk stenosis fits in type I (Di Marino *et al*.).

The celiac trunk compression by median arcuate ligament occurs more distally (Marable *et al.*, 1968).

The caliber of the portion of the celiac trunk that precedes a high grade stenosis, due to compression by median arcuate ligament, is slightly reduced (Hivet & Lagadec; Joubaud *et al*.).

The angle between the celiac trunk at its origin and the aorta in normal individuals decreases with age (Lindner & Kemprud).

The angiographic aspect of the intrinsic and extrinsic stenosis of the celiac trunk ranges from a minimum "notch" to complete occlusion (Ducellier *et al.*, 1974). It was observed that stenosis of the celiac trunk by the arcuate ligament presents different percentages of stenosis of luminal diameter, considered to be mild (less than 50%), moderate (50 to 75%), or severe (greater than 75%) (Colapinto et al.). In some cases, the percentage of the stenotic area is greater than 50% (Koikkalainen & Köhler, 1971; Levin & Baltaxe, 1972; Szilagyi *et al*.; Ducellier *et al*.; Ghosn *et al.*, 1982; Thevenet *et al*., 1985; Mensink *et al*.; Trinidad - Hernandez *et al*.), in others less than 50% (Szilagyi *et al*.), in others still 50% (Lord & Tracy, 1980; Loffeld *et al*.) or total (Rubush, 1970; Cornell; Ghosn *et al*.; Loffeld *et al*.; Lawson & Ochsner; Jaik *et al*., 2007).

Noninvasive diagnostic tests. The grade of stenosis, the systolic and diastolic velocities of flow in the celiac trunk and branches and narrowing, can be evaluated by the use of noninvasive tests: doppler ultrasonography (Trinidad-Hernandez *et al*.; Farma & Hoffman, 2007; Foertsch *et al*.; Jaik *et al*.; Tseng *et al.*), digital subtraction angiography (Desmond & Robert, 2004; Mensink *et al*.; Foertsch *et al.*; Tseng *et al*.), spiral computed tomography (CT) angiography (Baldassarre *et al*.), multislice CT (Karaban *et al.*), multidetector CT angiography (Farma & Hoffman; Ilica *et al.,* 2007; Tseng *et al*.), magnetic resonance angiography (Dordoni *et al*., 2002; Foertsch *et al.*; Gloviczki & Duncan, 2007).

The modality doppler ultrasonography (Dordoni *et al.*; Trinidad-Hernandez *et al.*; Farma & Hoffman; Foertsch *et al.*; Ilica *et al.*), the magnetic resonance angiography (Foertsch *et al.*) has been also used postoperatively to confirm adequacy of velocity systolic and diastolic of flow in the syndrome.

Duplex imaging showed that preoperative velocity systolic of the celiac trunk was 363 cm/s (Jaik *et al*.) and 450 cm/s (Trinidad- Hernandez *et al*.) and the postoperative was 145 cm/s (Jaik *et al*.) and 182 cm/s (Trinidad-Hernandez *et al*.). Ilica *et al.* using the multidetector tomography observed that preoperative systolic velocity of the celiac trunk was greater than 200 cm/s.

Recently, it was introduced the gastric exercise tonometry able to identify patients with suspected celiac trunk compression syndrome (Mensink *et al.*).

Jaik *et al*. were the first to treat this syndrome successfully using the da Vince TM (Institute Surgical, Sunnyvale, CA, USA) via robotic-assisted laparoscopy (telemanipulation), an approach minimally invasive surgery for division of the arcuate ligament.

The congenital origin of the syndrome. The young age of the patients favors of the congenital nature of this syndrome (Warter *et al.*, 1970a; Warter *et al*., 1976). The familiar observations of mother and daughter (Van de Berg *et al.*; Dondival & Drèze, 1972), two sisters (Warter *et al.*, 1974), father and his sons (Warter *et al*., 1976) and two twin brothers (Bech *et al*., 1994), all of them with celiac trunk compression syndrome reinforces this conviction.

All these malformations due to a probable pleiotropic genetic factor can be grouped, or accept that these dysmorphic syndromes (myotonic dystrophy, Marfan syndrome) may occasionally include, through its different manifestations, a celiac trunk stenosis. In this case the celiac trunk compression by the arcuate ligament will have a very precise genetic etiology (Dondinval & Drèze).

The confirmation of an identical formation of mother and daughter does not fit in this frame and genetic studies that have been undertaken lead to believe in a congenital malformation due to an embryologic development problem, probably due to an autosomal dominant gene (Drèze *et al*.).

Theory of the syndrome acquired origin. The control of karyotypes did not reveal anomalies in any case (Warter *et al.,* 1976).

It is possible to suppose that the arterial narrowing is an acquired process, although the diaphragmatic abnormalities appear to be congenital (Carey *et al*.). Some authors conjecture the acquired nature and believe it to be a sclerosis process (inflammatory or degenerative) that causes a cicatricial retraction of the cruciform fibers of the diaphragm with consequent descent of the inferior margin of the aortic hiatus, reducing the space delimitated by it. Supporting this hypothesis there is also the frequent association of sclerosis process of an adipose-cellular connective tissue, in individuals older than 50 years, occupying the retroperitoneal space around the aorta, involving up to the nervous structure of the celiac plexus and acquire an intimate anatomic-topographic relationship with the celiac trunk and with its branches. It is considered that this acquired factor, but with pathogenic bases, appears in a specific moment of life and alters the normal anatomic conformation, determining the arterial compression and affecting the flux of that area, in the anatomic conditions of the compressed vessel and in its poststenotic dilatation.The extended friction of the fibersclerotic structure over the celiac trunk could cause alterations in the muscular-elastic tonicity of the vessel and later will induce to form the athereosclerotic lesion of this vessel or the aorta (Bobbio *et al*.).

Coexistence of celiac trunk compression and SMA. The uncommon occurrence of reported cases on simultaneous compression of the celiac trunk and SMA by the arcuate ligament can be explained by the particular disposition of these trunks in the aorta. Its origins are close and at times, the initial segments of both are stuck together (Watson & Sadikali, 1977; Langeron *et al*., 1980).

Lower SMA disposition explains the smaller stenosis grade of this artery in relation to the celiac trunk that can be totally occluded (Langeron *et al*.). In some cases, a total obstruction of the celiac trunk with only one concave impression in SMA was observed angiographically (Loffeld *et al*.), a slight narrowing of the SMA, the celiac trunk

compressed by left crus of the diaphragm and an adenopathy and fibrous reaction around the celiac trunk and SMA (Cornell), and a moderate SMA stenosis without any significant hemodynamic implication (Edwards; Lord *et al*.). Also, a total occlusion of the celiac trunk with SMA stenosis of approximately 90 to 95% was demonstrated (Broussin *et al*; Langeron *et al*.; Lawson & Ochsner; Houssin *et al*., 1979), stenosis 50% of the SMA (Mulder et al.; Tison *et al*., 1989; Bech *et al*.), and a total SMA occlusion with the left crus being more involved than the arcuate ligament in the compression of the SMA, which has its origin on the left side of the aorta (Watson & Sadikali; Bacourt *et al*., 1984). A thick transversal fibrous formation tightly compressing the SMA (Langeron *et al*.) and the compression of a common celiac-mesenteric trunk by an abnormal crus of the diaphragm was observed. Arteriography revealed that the left crus performed a closer compression in the SMA than in the celiac trunk, both arteries originating on the left side of the aorta and very close to each other (Gautier *et al*., 1965).

Arteriographic diagnosis is more difficult in occlusive lesions. Therefore, in the case of the occluded celiac trunk, the SMA stenosis is the best diagnostic argument and in the occlusion of both arteries, the young age and the atheroma's absence orientate the diagnosis (Bacourt *et al*.).

Parallel to the activity of the compression by the arcuate ligament, the celiac- mesenteric nervous plexus contributed to the compression (Bacourt *et al*.). Evidence obtained with surgical intervention demonstrates a thick nervous tissue corresponding to the celiac-mesenteric ganglia surrounding the SMA (Houssin *et al*.).

Studies realized by several authors demonstrated the recovery of beats of arterial pulse with re-expansion of the celiac trunk and SMA and the disappearance of the bruit after liberation of tight fibrous formation of transversal disposition (Langeron *et al*.; Lawson & Ochsner; Bacourt *et al*.).

Rare angiographic experiments revealed the aorta compression or indentation on its anterior face, simultaneously to the celiac trunk compression by the arcuate ligament (Bacourt *et al*.; Ilica *et al*.). Fadhli was the first author to demonstrate this simultaneous compression, causing symptoms of intermittent claudicating of the lower extremities and abdominal angina, due to obstruction of the aorta and celiac trunk, respectively. The compression was caused by very tight ring of the aortic hiatus, encompassing the aorta and celiac trunk just proximal to its trifurcation. The postoperative patient was free of symptoms and gained weight.

Celiac trunk and branches compression. In patients with arcuate ligament compression syndrome, it was demonstrated that the diaphragmatic orifice crosses the celiac trunk in front of aorta, this vessel being involved by the abnormally tight diaphragmatic arcade close to the left gastric artery (Olivier *et al*.).

Hepatic artery stenosis by fibrous ring (Debray *et al*., 1967; Leger *et al*., 1967), stenosis of the celiac trunk and hepatic artery (Bessot *et al*., 1970), stenosis of the celiac trunk and their splenic and gastric branches (Van de Berg *et al*.; Trinidad-Hernandez *et al*.) were observed arteriographically in patients with epigastric pain, loss of weight and digestive problems.

Absence of symptoms in syndrome. The anatomic abnormality of the celiac trunk by compression of the median arcuate ligament can occur in asymptomatic individuals (Sutton, 1967) and frequently (Stoney & Wylie, 1966). Patten *et al*. (1991) state that this syndrome is an uncommon angiographic and surgical finding, rarely symptomatic.

In asymptomatic patients with isolated compression of the celiac trunk by the arcuate ligament and/or celiac ganglia (Rob, 1965; Drapanas & Bron, 1966; Sutton; Charrette *et al*., 1971; Colapinto *et al*.; Levin & Baltaxe; Guibert *et al*., 1980; Meaney & Kistner, 1967; Stoney & Wylie), and with compression SMA and of the celiac trunk (Houssin *et al*.; Langeron *et al*.) the stenosis of these vessels was incidentally found through angiography performed for other affections.

Levin & Baltaxe analyzed 50 patients with celiac stenosis due to arcuate ligament compression and/or celiac plexus with complete absence of abdominal symptoms or referring to the gastrointestinal tract, submitted to lateral aortogram by other clinical causes. Twelve were observed with stenosis at 50% or more.

Angiography after an abdominal trauma incidentally revealed the double celiac trunk and AMS compression (Langeron *et al*.).

Typical angiographic findings in asymptomatic individuals do not invalidate the arcuate ligament syndrome, since it is well known that the atherosclerotic occlusion of both celiac trunk and SMA can also be asymptomatic (Colapinto *et al*.).

Asymptomatic character in cases of the celiac trunk compression by the arcuate ligament led to the hypothesis that the collateral vessels from the SMA supply the terminal branches of the celiac trunk without compromising the

blood flow (Stoney & Wylie; Sutton). Similarly, in the double celiac trunk and SMA compression, the absence of symptoms seems to depend on the existence of a prominent Riolan´s arcade (Houssin *et al*.). It is clear that in the asymptomatic individual the arcuate ligament section is performed to prevent a decompensation of vascularization in the visceral splanchnic area up to now supplied by the inferior mesenteric artery through the Riolan´s arcade (Langeron *et al*.).

Based on angiographic findings demonstrating both asymptomatically and symptomatically individuals with compression syndrome presented developed collateral vessels, it was supposed that the symptoms are not caused by the lack of collateral vessels (Cornell). Levin & Baltaxe finds a collateral circulation in the frontal aortogram or in the superior mesenteric arteriogram in only 3 of the 12 asymptomatic patients with celiac trunk compression syndrome.

Surgical Considerations. With the arciform fibers section of the diaphragm aortic hiatus, the retraction of the muscular fibers creates an ogive or an arcade (Bobbio *et al*.) allowing the exposure of the initial celiac trunk segment and its origin in the aorta (Bobbio *et al*.; Curl *et al*. and Furnemont). The compressed segment has a "diabolo" aspect (Furnemont). After the arcuate ligament section it can be verified that the anomaly is promptly corrected, with the normal expansion of the celiac trunk and reduction of pressure gradient between the celiac trunk and the aorta. (Houdard *et al*.; Marable *et al*., 1966; Fortner & Watson, 1981; Tridico *et al*., 1988; Joubaud *et al*. and Charrette *et al*.). The weak pulse in the hepatic, gastroduodenal arteries and absence of pulse in left gastric is recovered after the ligament division (Furnemont, 1974).

The surgical correction is usually an incision or excision of the arcuate ligament (Szilagyi *et al*.). The encouraging results observed in the literature, the simple and harmless intervention granting zero mortality and morbidity is sufficient to believe in the efficacy of the arcuate ligament division and to recommend it (Cormier & De La Fontaine; Houdard *et al*.; Stanley & Fry; Kieny & Cinqualbre, 1976; Guibert *et al*.; Ghosn *et al*. and Tridico *et al*.).

Dunbar *et al*. and Gautier *et al*. were the first authors to observe the cure of the painful abdominal syndrome due to the arcuate ligament sectioning. Harjola was the first to observe the celiac trunk stenosis due to celiac ganglia fibrosis and cure of the symptoms by the resection of the same. After several studies reported the success of the surgical treatment by sectioning the arcuate ligament and/or the celiac plexus (Terpstra; Marable *et al*., 1966; Stoney & Wylie; Marable *et al*., 1968; Harjola; Harjola & Lahtiharju; Snyder *et al*.; Taheri, 1968; Jamieson & Grieg, 1970).

It is possible that the complete celiac trunk hemodynamic liberation depends from the arcuate ligament section as well as from resection of the periarterial neurofibrous tissue (Balmes *et al*., 1971; Harjola & Lahtiharju; Hivet & Lagadec; Lindner & Kemprud; Conti *et al*., 1973; Van De Berg *et al*.; Watson & Sadikali; Daskalakis, 1982 and Ghosn *et al*.). In some patients, a certain degree of periarterial fibrosclerosis can be intra-surgically observed (Drèze *et al*.). In some cases an extensive celiac plexus denervation and a periarterial sympathectomy have been performed (Cormier & De La Fontaine; Watson & Sadikali).

Tahery verified the celiac trunk compression release after a division of the right crus of the diaphragm associated to ganglionectomy.

In the compression cases of the celiac trunk by the arcuate ligament (Dreze *et al*.; Joubaud *et al*.) and associated SMA (Houssin *et al*.) when the very dense fibronervous envelope formed by nervous and lymphatic ramifications involving both arteries and celiac trunk branches was sectioned, an arciform bridge, considered to be the diaphragmatic arcuate ligament was observed crossing the celiac trunk. A dense tissue of approximately 5 mm thick continuous with the arcuate ligament was divided to release the artery (Lord & Tracy).

Some authors believe that despite the celiac plexus section be an inevitable surgical act during celiac decompression, this limited plexotomy is not responsible for cure of the symptom (Marable *et al*., 1968).

Williams *et al*. during the decompression of the celiac artery by dividing the arcuate ligament, tried not to damage the celiac ganglia and to divide only the intercalating fibers of the celiac plexus as they crossed the celiac trunk. Considering that they managed to cure the symptoms by simply dividing the arcuate ligament, they concluded that the limited periarterial sympathectomy alone would not produce lasting relief of the symptoms.

Also, the recurrence of pain after arcuate ligament section denied this belief (Stanley & Fry).

It is possible to suppose that in cases of cure of the symptoms, a more extensive resection in the area of the celiac trunk origin, consequently involving the celiac ganglion division could be the cause of symptom cure (Edwards *et al*.).

It is appropriate to be restricted only to the sectioning of the nervous structure that narrows the celiac trunk and its branches, respecting the other elements (Bobbio *et al*.).

It was arteriographically demonstrated stenosis of the celiac trunk due to fibrosis of ganglion, and the total cure of the symptoms and disappearance of the abdominal bruit after sectioning nervous fibers (Rob, 1966; Snyder *et al*.).

After surgery and biopsy it becomes difficult to decide whether the responsible component for the celiac trunk compression is the arcuate ligament or the celiac ganglion fibrosis (Edwards *et al*.; Colapinto *et al*.). However, several cases do not need the extrinsic stenosis surgical treatment of the celiac trunk by arcuate ligament since it does not cause any problem (Joubaud *et al*.).

The arterial blood flow and pressure of the aorta and its branches should be recorded before and after releasing the compression of the celiac trunk (Daskalakis). Specific pressure gradients are measured intra-surgically (Dunbar *et al*.; Stoney & Wylie; Terpstra; Lord *et al*.; Carey *et al*.; Edwards *et al*.). These pressure gradients present individual variations and can be weak (Dunbar *et al*.; Balmes *et al*.) or absent (Carey *et al*.). Some authors reported gradients between 5 and 30 mmHg (Dunbar *et al*.), others of approximately 30 mmHg (Lord *et al*.; Lord & Tracy) or between 20 and 60 mmHg (Stoney & Wylie), between the aorta and the celiac trunk. If the prestenotic segment of the celiac trunk is masked by the muscular fibers of the diaphragm pillars, it becomes difficult to measure the pressure gradient of this vessel (Houdard *et al*.).

The "thrill" is clearly palpable, with variable intensity and, at times, does not even exist (Houdard *et al*.; Tseng *et al*.). A diminished pulse pressure within the celiac arterial bed and a "thrill" of this artery suggest compression (Stanley & Fry). It was verified in patients with SMA and trunk celiac compression, weak pulse and strong "thrill" in the SMA and absent pulse in the celiac trunk (Houssin *et al*.).

Frequently, after the division of the arcuate ligament, it is intrasurgically detected a minimum residual stenosis in the compressed area of the celiac trunk, distal bruit, "thrill" less intense of this vessel and gradient pressure. The postoperative arteriography showed, over time, regression and disappearance of this discrete "notch" and returning to the normal caliber of the stenosed trunk (Edwards *et al*.; Charrette *et al*.; Guibert *et al*.; Leger *et al*., 1970; Foertsch *et al*.).

If after the division of the diaphragm construction fibers and/or the periarterial nervous conjunctive involving it, the strangled celiac trunk does not recover its normal caliber and the pressure gradient crossing the stenosed segment remains significant, a revascularization of the segment with sequels is needed by resection of this segment, followed by anastomosis or reconstruction by autograft, bypass, transplant or endarterectomy. The histological results of these high grade stenosis with alterations and fragilities of the vessel walls, is frequently related to fibrosis or intimae hyperplasia, at times with associated atheromatosis lesions (Stoney & Wylie; Lord *et al*.; Hivet & Lagadec; Olivier *et al*.; Lindner & Kemprud; Houssin *et al*.; Daskalakis; Thevenet *et al*.; Edwards; Edwards *et al*.). The moderate changes in the intimae by fibrosis and hyperplasia are likely reversible in young patients and do not need reconstruction (Charrette *et al*.). The arterial pressure of the hepatic or left gastric artery, before and after release of the compressive band of the celiac trunk, is a more sensitive and reliable test in order to evaluate the immediate results. If there is no significant increase, arterial reconstruction can be promptly undertaken (Daskalakis).

The arterial reconstruction abolished pressure gradient and postsurgical arteriograms confirm the patency of the reconstructed segment (Lord *et al*.).

The permanence of abdominal bruit after arterial compression surgical dilatation, suggests as cause the persistence of a poststenotic dilatation, which can be sufficient to cause turbulences (Watson & Sadikali). These signals can disappear after months or not disappear completely (Conti *et al*.).

Control arteriography. Postoperative control aortogram confirms anatomic correction of the celiac trunk deformity with recovery of its normal caliber, and revascularization through the collaterals (Bacourt *et al*.; Lawson & Ochsner; Hivet & Lagadec; Houdard *et al*.; Snyder *et al*.; Tridico *et al*.). The return of the normal angiographic patterns represents the most definitive proof that the improvement in the clinical picture of ischaemia depends on the restoration of normal anatomical relationships at the level of the aortic canal of the diaphragm (Bobbio & Zanella).

Presurgical duration of the symptoms. Presurgical duration of the symptoms is variable. It was verified that in a patient the abdominal pain originated in his childhood (Conti *et al*.), in another, 65 years old, abdominal pain lasted 30 years (Loffeld *et al*.).

Postsurgical results and follow-ups. Although the percentage of the favorable surgical results is not known, in different series described in literature, they appear compatible to approximately 86% (Warter *et al*., 1976). The

case selection in the literature of patients treated between 1971 and 1987, showed 82% of surgical success (Tridico *et al*.).

Literature shows results on a long term of 1 to 3 years (Lord *et al*.; Ghosn *et al*.; Williams *et al*.; Bacourt et al.; Mensink *et al*.), and 7 years (Foertsch *et al*.)

PETRELLA, S. & PRATES, J. C. Una revisión del síndrome de compresión del tronco celíaco: Algunos aspectos anatómicos clínicos-quirúrgicos. ***Int. J. Morphol., 26(2)***:293-304, 2008.

RESUMEN: El objetivo de este estudio fue hacer una revisión del síndrome de compresión del tronco celíaco, en cuanto a aspectos tales como: relación síntoma-postura; ausencia de síntomas; relación síndrome-edad; estudio angiográfico sobre la anatomía de la estenosis del tronco celíaco; origen congénito o adquirido; tests diagnósticos no invasivos; resultados quirúrgicos y post-quirúrgicos.

PALABRAS CLAVE: Arteria celíaca; Diafragma; Enfermedad arterial oclusiva; Ligamento; Plexo celíaco; Síndrome.

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