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Celiac Trunk Compression Syndrome. A Review

Una Revisión del Síndrome de Compresión del Tronco Celíaco

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SUMMARY: The purpose of the present review is to report the anatomic and the clinical-surgical aspects involved in the celiac trunk compression syndrome by the median arcuate ligament of the diaphragm, reviewing the major findings of the syndrome in the anatomic field during dissection of cadavers, followed by clinical-surgical findings of stenosis of the celiac trunk, the relationship of this stenosis with the patient's symptoms and healing after decompression of that artery; invasive and non-invasive methods used to diagnose compression; the stenotic effect of physiologic mechanisms of the median arcuate ligament, aorta and celiac trunk displacement during respiration; anatomy of the aortic channel and celiac plexus; the median arcuate ligament and the celiac plexus as constrict agents; skeletopy of the celiac trunk, the median arcuate ligament and predisposition to syndrome; association of the syndrome with morphological and metabolic aspects.

KEY WORDS: Celiac artery; Ligaments; Celiac plexus; Syndrome; Diaphragm; Arterial occlusive disease.

A Relationship of the celiak trunk with diaphragm crura. The first investigations on acknowledge of the celiac trunk compression by diaphragm crura happened in the anatomic field during cadaver dissection (Rio Branco, 1912; Lipshutz, 1917; George, 1934; Michels, 1955). Dissections of 62 cadavers revealed that sometimes the origin of the celiac trunk is overlapped by the diaphragm (Lipshutz). In his study, George also reported eight cases of overlap by the diaphragm in 38 dissections of cadavers. Additionally, it was observed in these cases that the constrictive action of the diaphragm reduces the caliber of the celiac trunk at its origin (George, and Michels).

Discover of the celiac trunk compression syndrome. Later, this stenoic anatomical anomaly of the celiac trunk was angiographically individualized in a series with 15 patients and correlated etiologically with some clinical manifestations, such as postprandial abdominal pain and weight loss. Additionally, the clinical evaluation showed the symptoms healing in 12 individuals after surgical release of this vessel compression by sectioning the median arcuate ligament (Dunbar *et al.*, 1965).

The surgical success of Dunbar *et al.* in the medical area, led many authors to confirm and attribute this arterial compression to the diaphragm (Marable *et al*., 1966; Stoney & Wylie, 1966; Terpstra, 1966; Lord *et al.*, 1968; Marable *et al.*, 1968; Taheri, 1968; Hivet & Lagadec, 1970; Ciscato *et al.*, 1976; Warter *et al.*, 1976).

Invasive and non-invasive diagnostic methods. The compression of the celiac trunk by the median arcuate ligament is an entity angiographically well characterized (Marable *et al.,* 1966; Warter *et al.*, 1973a) since its first observations by the routine practice (Dunbar *et al.*).

The stenosis visualization by the celiac trunk compression syndrome can also be performed according to non-invasive methods as the echo-Doppler (Tridico *et al.*, 1988); color pulsed Doppler (De Pauw *et al*., 1992; Sproat *et al*., 1993) and computerized tomography (Patten *et al.*, 1991; Sponza *et al.,* 1933; Loffeld *et al*., 1995); digital subtraction angiography (Desmond & Roberts, 2004); magnetic resonance angiography (Dordoni *et al.,* 2002; Alehan & Dogan, 2004); three-dimensional computerized tomography (Horton *et al*., 2005); as well as the visualization of the adequacy flow in the aorta and celiac trunk by means of Doppler ultrasound scanning (Roayaie *et al*., 2000; Dordoni *et al.* and Alehan & Dogan).

Sponza *et al.* published a study in which visibility of the celiac trunk, its arteries and the superior mesenteric artery as well as its anatomic variations and vascular anomalies were analyzed in 100 patients by means of computerized

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tomography. The results were in accordance with the angiographic data reported in the literature.

Patten *et al.* showed in five individuals submitted to a computerized tomography to evaluate several anomalies, occurrence of high-grade stenosis of the celiac trunk by the median arcuate ligament. The surgery and/or angiography confirmed the diagnosis (Patten *et al.* and Loffeld *et al.*).

Recently, the laparoscopy, a less invasive surgical maneuver became equally effective in the celiac trunk decompression (Kokotsakis *et al.*, 2000; Roayaie *et al.*; Dordoni *et al.*; Desmond & Roberts and Carbonell *et al.*, 2005).

Displacement of the median arcuate ligament and the celiac trunk during respiration. Arteriographic experiments showed the mechanisms of the median arcuate ligament, celiac trunk and aorta displacement during respiration in individuals with compression of the celiac trunk syndrome. According to the lateral incidence arteriography, it could be observed that during inspiration, the median arcuate ligament caudally moves an average of 8mm and ventrally 4.4mm. Concomitantly, the celiac trunk and the aorta caudally move an average of 3 to 6mm respectively, resulting in a distance between the orifice of celiac trunk and the median arcuate ligament (Reuter *et al.*, 1971; Stanley & Fry, 1971; Reuter & Bernstein , 1973).

The "notch" in the emergency of the celiac trunk, caused by the median arcuate ligament compression is more accentuated during expiration and in a lesser degree or inexistent in inspiration (Curl et al., 1971; Williams *et al.*, 1985 and Loffeld *et al.*).

Stenosis of the celiac trunk by the median arcuate ligament. Several studies showed that the constrictor agent of the celiac trunk compression is the median arcuate ligament (Dunbar *et al.*; Gautier *et al.*, 1965; Stoney & Wylie; Deutsch, 1968; Lord *et al.*; Edwards *et al.*, 1970; Hivet & Lagadec; Rubush, 1970; Bobbio & Zanella, 1971; Stanley & Fry; Auché *et al.*,1972; Drèze *et al.*, 1972; Van De Berg *et al.*, 1972; Watt, 1972; Conti *et al.*, 1973; Meves & Beger, 1973; Warter *et al.*, 1973a, 1973c; Ducellier *et al.*, 1974; Ciscato *et al.;* Ferrandiz *et al.*, 1976; Lynch, 1976; Datta & Vichery, 1979; Abate *et al.*, 1980; Lord & Tracy, 1980; Mongelli *et al.*, 1980; Pailler et al., 1982; Gutnick, 1984; Reilly *et al.*, 1985; Aburahma, 1995; De Cecchis *et al.*, 1996).

Stenosis of the celiac trunk by the median arcuate ligament and celiac plexus. Marable *et al.* (1966) described the celiac trunk compression by the median arcuate ligament and by the celiac plexus as a new clinical entity. Thus, the plexotomy alone does not seem to cause a symptom relief. Bobbio *et al.* (1967) observed intrasurgically that if the section of the fibrotic ganglion of the celiac plexus is performed, the pulsations of the celiac trunk and its branches do not return to normal, being necessary decompression of the celiac trunk by sectioning arciform fibers of the diaphragm, which also exert compression.

When the median arcuate ligament is sectioned during surgery, some nervous fibers need to be sectioned leading to an effective result (Joubaud *et al.*, 1977). It is possible to figure out that relief of symptoms after division of the median arcuate ligament in patients with persistent stenosis could be due to increase in blood flow after periarterial sympathectomy and denervation of the celiac ganglion inevitably performed during sectioning of the median arcuate ligament (Carey *et al.*, 1969).

After Marable *et al.* (1966), several authors reported the celiac plexus compression by both agents - the median arcuate ligament and the celiac axis (Rob, 1966; Stoney & Wylie; Bobbio *et al.*; Harjola & Lahtiharju, 1968; Tahery (1968); Carey *et al.*; Cormier & De La Fontaine, 1970; Olivier *et al.,* 1970; Balmes *et al.*, 1971; Lindner & Kemprud, 1971; Stanley & Fry; Tongio *et al.*, 1971; Drèze *et al.*; Conti *et al.*; Beger *et al.*, 1975; Joubaud *et al.*; Watson & Sadikali, 1977; Guibert *et al.*, 1980; Daskalakis, 1982; Ghosn *et al.*, 1982; Matesanz *et al.*, 1982; Thevenet *et al.*, 1985; Bacourt *et al.*, 1984 and Roayaie *et al.*).

Stenosis of the celiac trunk by the celiac plexus. Harjola (1963) was the first to describe the extrinsic compression of the celiac trunk by fibrosis of the celiac ganglion. The angiogram showed stenosis of the celiac trunk and in the surgery a constriction of the artery by the fibrosed celiac ganglion was observed. The artery was released and the epigastric bruit disappeared and the patient remained with no symptoms. The author confirmed a retroperitoneal fibrosis and, according to them, in spite of the pain being caused by fibrosis, it could not explain its appearance after meals. Based on this clinical report, many authors agreed with this defined clinical entity, considering the celiac plexus as the etiologic agent in the celiac trunk compression syndrome, which is formed by an abundant neurofibrous tissue able to constrict this vessel (Drapanas & Bron, 1966; Debray *et al.*, 1967a, 1967b; Snyder *et al.*, 1967; Harjola, 1968; Harjola & Lahtiharju; Jamieson, 1970; Leger *et al.*, 1970; Olivier *et al.*; Di Marino *et al.,* 1972; Mc Sherry, 1977; Van Gossun *et al.*, 1984; Jamieson, 1986).

Evidence of celiac ganglion compression was based on macroscopic observation that the ganglion abnormally dense and fibrosed constrict the celiac trunk as a ligature or in an annular manner, from a distance of 3 to 4 mm (Harjola & Lahtiharju).

Anatomy of the celiac plexus. The terms celiac, splanchnic and solar plexuses are equivalent for the same nervous structure. The etymology of these terms emphasizes the adjacent celiac trunk, the accessory splanchnic nerves and its location (Thompson *et al.,* 1977).

The major part of the sympathetic system in the abdomen is formed by an autonomous nerve plexus extending front and sides all along the entire length of the abdominal aorta. Its superior part is designated celiac plexus (Ward *et al.,* 1979). This is the largest of the three plexuses of the autonomous nervous system (cardiac, celiac, hipogastric); it supplies the abdominal organs and is situated at the T12-L1 level (Thompson *et al.*). It is located in front of the diaphragm crura, medially to the adrenal glands and upon the abdominal aorta surrounding the origin of the celiac trunk and the superior mesenteric artery, composed of dense strands of splanchnic nerve fibers (Snyder *et al*.).

The right and left semilunar celiac ganglions emerge from the celiac plexus and are found upon the diaphragm crura, in the origin of the celiac trunk and lateral to the aorta. Its fibers are interconnected around the celiac trunk and its branches, accompany these up to the respective superior abdominal organs (stomach, liver, gallbladder,pancreas, adrenal glands and kidneys (Lindner & Kemprud; Lowe, 1975; Joubaud *et al*. and Ward *et al*.). Both ganglions are immersed into an extensive network of nervous fibers and in one mass of connective tissue (Snyder *et al*. and Ward *et al*.).

Anatomy of the diaphragm crura. The aortic hiatus is a foramen in which the aorta passes from the thorax to the abdomen. It is delimited medially and laterally by tendons of the right and left crura of the diaphragm and its internal extensions consist of a triangle whose inferior margin forms the median arcuate ligament rounding the acute angle formed by the interconnection of the two crura (Bobbio *et al*.; Hivet & Lagadec and Furnemont, 1974). This leaning position of these two crura and their relationship with the pre-vertebral musculature forms the real "aortic channel" (Bobbio *et al*.).

Regarding the anatomic aspect, the median arcuate ligament is connective fibrous or thick fibromuscular, arciform structure, transversally crossing the aorta at the T12 vertebra level and attaching right in front of this vessel to these tendinous portions of both crura to form the anterior margin of the aortic hiatus (Reuter & Bernstein; Joubaud *et al*.; Langeron *et al*., 1980 and Patten *et al*.). This fibrous ligament is infiltrated by nervous formations of the celiac plexus (Guibert *et al*.).

Leger *et al.* described the median arcuate ligament as a fascia fulfilling the space resembling a triangle, formed by the two crura interconnection of the diaphragm, which in some cases may cover the origin of the celiac trunk. This of both crura (Ruiz Liard *et al.*, 1964). Also Lindner & Kemprud identified the median arcuate ligament as a structure variable in size, shape and position usually presented a round fibrous of 1 to 3 mm in width formed by condensing the fibrous margin of the two crura of diaphragm decussating to form the ventral border of the aortic hiatus easily observed by naked eye. Also, there was evidence that this connective margin is occasionally inexistent or difficult to be identified, which seems to produce little or no compression on the celiac trunk.

Di Marino *et al.* concluded that the median arcuate ligament is a thin fibrous connective formation recovering the deep face of the diaphragm, adhering to the tendinous-muscular contour of the aortic hiatus and that sometimes overlapping the celiac trunk and is not able to play the role of stenosis. However inferior border of the diaphragmatic aortic ring, mainly the fibrous border, may provoke compression of the celiac trunk.

Anomalous display of the celiac trunk and/or median arcuate ligament. Several authors believe that the anatomic substrate of the celiac trunk compression syndrome is represented by the more caudal anomalous display of the median arcuate ligament, varying from the intervertebral disk T12-L1 towards the inferior third of the L2 vertebra (Curl *et al*.; Stanley & Fry; Warter *et al*., 1976) sometimes reaching the level of the L3 and L4 vertebras (Rubush *et al*.). The anomalous insertion of the diaphragm crura, the hypertrophy or lowering its fibrous arcade, hardly constricts the proximal segment of the celiac trunk when it crosses the aortic hiatus (Marable *et al.*, 1966; Stoney & Wylie; Harjola & Lahtiharju; Marable *et al.*, 1968; Olivier *et al*.; Rubush; Bobbio & Zanella; Curl *et al*.; Conti *et al.*; Kieny & Dietz, 1973; Furnemont; Loffeld *et al.*). Other authors observed that the celiac trunk might have an intrathoracic origin (Fadhli, 1968; Warter *et al*., 1970b; Gutnick). Due to these situations, this celiac trunk compression syndrome was denominated celiac-phrenic disorder (Warter *et al.*, 1976)

PETRELLA, S. & PRATES, J. C. Una revisión del síndrome de compresión del tronco celíaco. ***Int. J. Morphol., 24(3)***:429-436, 2006.

RESUMEN: La presente revisión tiene por objetivo conocer tanto los aspectos anatómicos como clínicos y quirúrgicos relacionados con el síndrome de compresión del tronco celíaco causado por el ligamento arqueado mediano del diafragma. Se revisan los principales descubrimeinos del síndrome, tanto en el plano anatómico durante la disecación de cadáveres, como en la clínica-quirúrgica de la estenosis del tronco celíaco. Además, se revisa la relación de esta estenosis con los síntomas del paciente y cura después de la descompresión del tronco celíaco. Por otra parte, se explican los métodos no invasivos e invasivos utilizados en la descompresión; el efecto estenótico de los mecanismos fisiológicos del desplazamiento del ligamento arqueado mediano, aorta y tronco celíaco durante la respiración; anatomía del canal aórtico y plexo celíaco; el ligamento arqueado mediano y el plexo celíaco como agentes constrictores; la esquelotopía del tronco celíaco y del ligamento arqueado mediano y la predisposición para el síndrome. Finalmente, se hace una asociación del síndrome del tronco celíaco con anomalías morfológicas y metabólicas.

PALABRAS CLAVE: Arteria celíaca; Ligamentos; Plexo celíaco; Síndrome; Diafragma; Enfermedad Arterial Oclusiva.

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