The Celiac ganglion (artery) compression syndrome (CGCS)

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Acknowledgement
I thank Dr. Annegret Klimas for insightful discussions about CGCS.

A preface

This article is solicited by the journal's editor and it is a joy for me (as well as an opportunity to present a personal view of the subject) to use this opportunity to express my gratitude to the Norwegian Ultrasound Society who invited me to give some lectures at its annual congress in Trondheim in 2011.

We report here our experience with sonographic diagnosis in more than 1000 cases with actually 53 operated on. It was in Trondheim that some Norwegian colleagues expressed their interest in my talk dealing with abdominal vascular compression syndromes and now inspired this contribution to this journal. But the links to Norway and the Nordic countries in general go deeper - especially with respect to this topic. It is not only that the Finnish surgeon Harjola described for the first time – and successfully operated on – this vascular compression syndrome but I draw a lot from the Norwegian Doppler pioneers as Liv Hatle, Bjørn Angelsen and Sturla Eik-Nes, all from Trondheim, which were my remote teachers by means of their books and publications when we tried to catch up with the western technique and technology while we were shut away behind the "iron curtain" living in the GDR. It was barely inconceivable to give back something someday. Now we have got so many friends and colleagues just in Norway – in part due to many German doctors living here in part due to the openness of mind in many Norwegian colleagues with whom I had the privilege to make friends with. This still moves me and the whole story reminds me to another deep experience related not only to Doppler and blood flow: πάντα ῥεῖ - everything flows …and thus changes.

A clinical approach to CGCS

The clinical apprehension of CGCS has changed from its first description [1] – and still does.

Historical notes

Harjola and Lahtiharju [2] coined the term "celiac axis syndrome" for a symptom complex consisting of postprandial epigastric pain, commencing 20 to 60 minutes after a meal, in addition of epigastric fullness and vomiting or diarrhea in some of the reported 13 patients (10 women and 3 men, 32–63 years old).

In 3 of them, an epigastric bruit was audible prompting the suspicion of an arterial stenosis which was confirmed by preoperative angiography. The other 10 patients underwent surgery for a variety of other reasons, and in all cases, celiac artery compression was found. Harjola's description was refined later on leading to the often repeated so called characteristic (clinical) triad. The constituents of this triad but changed: postprandial pain, vomiting, and weight loss [3], abdominal pain, an epigastric bruit, and angiographic evidence of celiac compression[4], epigastric pain, postprandial pain and weight loss of more than 5 kg [5], postprandial abdominal pain, weight loss, and vomiting [6]. Nevertheless, not rarely a shortcut constellation of three well defined components has become a mantra to guide vascular surgeons. Others extended the scope of symptoms to include an abdominal bruit (primarily mentioned in Harjola's pioneering paper) [7-9]. Some also describe the change of this bruit's volume with breathing – i.e. diaphragms position and a decrease in inspiration [9]

The coexistence of a variety of symptoms was seen by some authors as clearly disqualifying for the diagnosis of celiac artery compression ("…Patients likely to benefit [from an operation –authors insertion] are those with epigastric pain related to food or hunger who do not have a galaxy of other unrelated symptoms".)[10]

Diverging pathophysiological concepts and therapeutic conclusions

Not rarely the terms compression and stenosis were used interchangeably or even in a joint manner [11, 12] what may have further contributed to blur the apprehension of the exact nature of this syndrome [7, 13, 14].

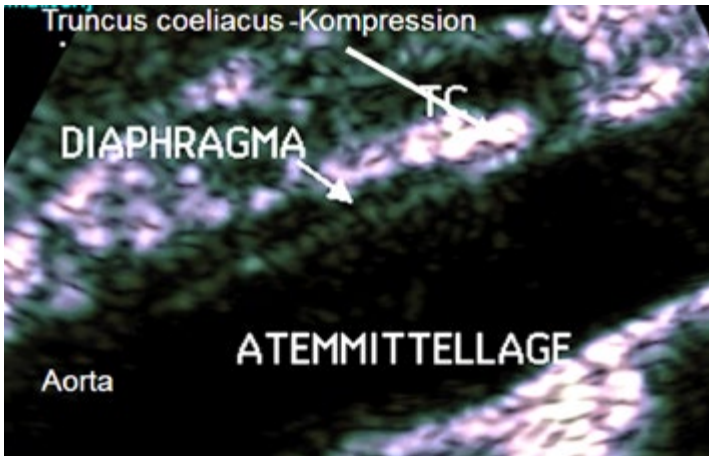

Figure 1. B-mode image of the longitudinal section of the abdominal aorta showing clearly the compression of the celiac trunk (TC) by the inferior portion of the diaphragm – the arcuate ligament (arrow).

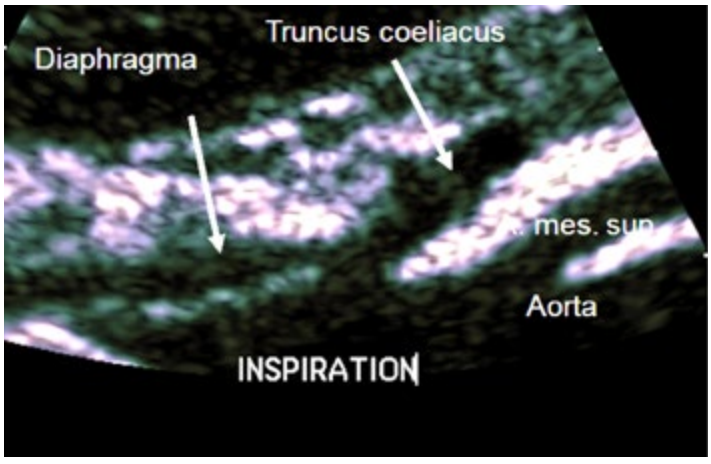

Figure 2. B-mode image of the longitudinal section of the abdominal aorta during inspiration demonstrating the release of the celiac trunk (right arrow) by the retracting diaphragm (left arrow).

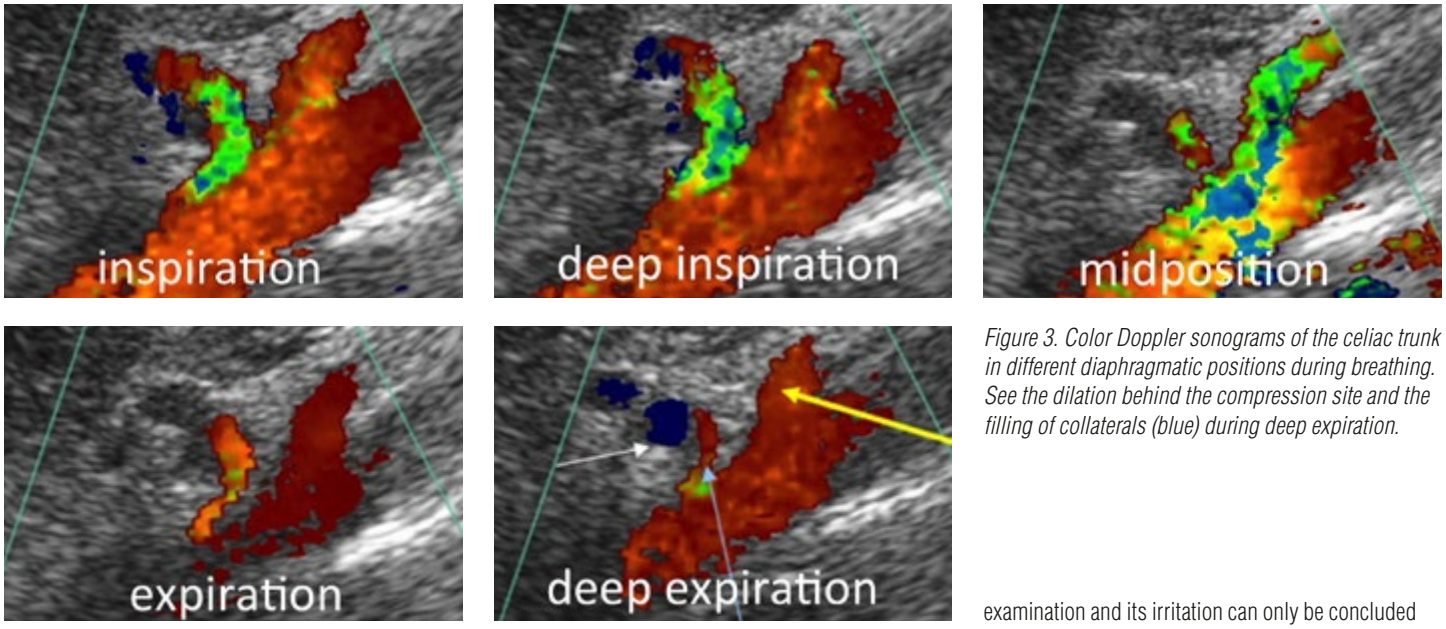

Figure 3. Color Doppler sonograms of the celiac trunk in different diaphragmatic positions during breathing. See the dilation behind the compression site and the filling of collaterals (blue) during deep expiration.

The origin of pain was suspected in diminished blood flow thus causing ischemia downstream the stenosis (i.e. in the stomach, liver and spleen). Some questioned this causal connection and favored the neuropathic origin from the compressed neural tissue of the celiac ganglion [1, 2, 15, 16].

The longstanding uncertainties of the true origin of the prevailing pain [17, 18] has much contributed to the diversification of operation techniques [19-24], recommendations [25] and mixed results [13, 26, 27]. This is the main reason for disbelief of the very existence of the syndrome at all [28] and the decades of debate to find the best operation technique with the longest lasting results [29]. The main objection against the ischemia theory came from the frequent observation, that in severe cases of compression collaterals arising from the superior mesenteric artery bridged the narrowing [30]. For some authors it seemed thus highly unlikely for ischemia to play a major role in the disease [31] or they even declined the entire concept [32, 33]. Others stressed that patency of the celiac artery (CA) in postoperative follow up was linked to persistence of pain relief [29]. Other reasons brought forward against the clinical relevance of celiac artery compression were the discouraging results many observed after successfully restoring the vessel's patency [26] or implementing venous bypasses. Rarely attempts were made to stent the compression site aiming at restoring the original lumen in order to ease the blood flow. The many recommendations often simply ignored the fundamental difference between an arterial stenosis and arterial compression from outside [34]. Often the argument was raised, that the many severe stenoses of the celiac trunk found at autopsies [35] which were completely asymptomatic during lifetime and thus ruled out a causal link of celiac compression and complaints[36]. The results were mixed and so the debate persists to date [37, 38].

Need to distinguish stenosis from compression of the celiac artery

Therefore, it seems worthwhile to highlight the differences between both entities (Scholbach's reply to [12]). The main and fundamental difference is the cause of lumen reduction of the celiac artery. In celiac ganglion compression syndrome (synonyms are Dunbar-syndrome, celiac artery compression syndrome, celiac trunk compression syndrome, median arcuate ligament syndrome [MALS]) the celiac artery and the straddling celiac ganglion are both compressed by the arching arcuate ligament. So, the compression is one sided, guillotine-like and often results in a sharp incision of the celiac artery from above, which is easily visible in color Doppler and very often, but substantially less clear in B-mode images of the longitudinal section of the abdominal aorta (figure 1). This compression is variable with breathing – because while the diaphragm is moving the position of the arcuate ligament is changing thus causing changing degrees of squeezing the vessel (figure 2). The celiac ganglion is not visible with the conventional imaging techniques. Thus it evades the direct examination and its irritation can only be concluded from the compression of the underlying celiac artery. This fact contributes much to concepts which focus on the vessel's diameter and forget about the ganglion. This is the reason why the author proposes a new name for this condition: celiac ganglion compression syndrome.

Sonographic diagnosis

Color Doppler sonography is the method of choice to rule in and rule out the diagnosis of CGCS. B-mode sonography in many cases can clearly delineate all relevant structures (figure 1) but is of limited value in adipose patients and in cases with a slightly laterally directed CA. Color Doppler, often at a first glance, can show if there are turbulences and depicts the course of the CA quite clearly. Also aneurysms with a typically whirling flow patterns and post-compression dilation are easily recognized. Moreover, collateral arteries from the superior mesenteric artery to distal portions of the CA can be shown – nearly impossible with B-mode ultrasound. The compression is clearly visible in a midposition of the diaphragm (figure 3) and diminished in inspiration. Expiration leads to the strongest compression (figure 4). Often, this position cannot be held for a long time and many patients do not expire deep enough to develop a pronounced increase of flow velocity and a maximum reduction of

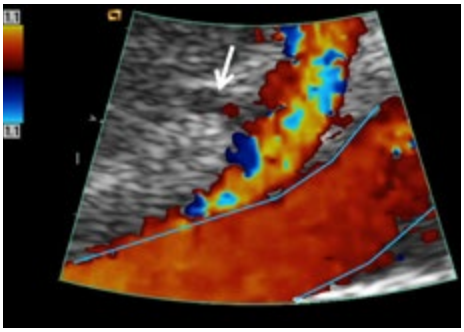

Figure 4a. Deep expiration in CGCS: Only the superior mesentric artery is still visible. The course of the aorta points to the strong diaphragmatic compression of the celiac trunk bending the underlying vessels (i.e. superior mesentric artery and aorta). No flow in the celiac trunk (arrow)

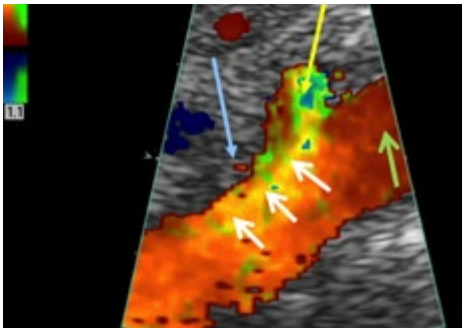

Figure 4b. Deep expiration in CGCS: Only the superior mesentric artery is still visible. The coloration of the ventral part of the aorta (white arrows) makes evident that here the superior mesentric artery is pressed into the aortic surface. No flow in the celiac trunk (blue arrow), Slower flow in the aorta itself (green arrow)

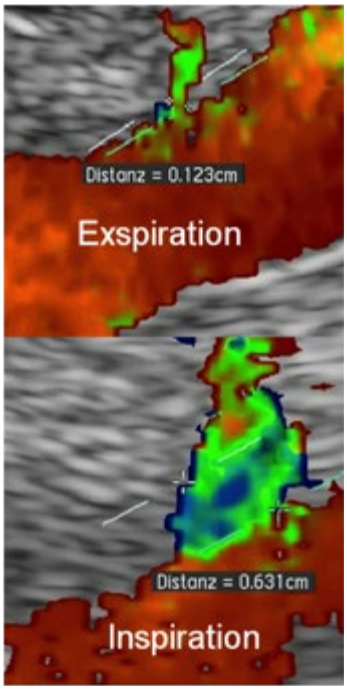

Figure 5. Change of the diameter of the compresssed celiac trunk from expiration to inspiration.

the artery's lumen. But a clear change of diameter and flow velocities can always be demonstrated with deep inspiration (figure 5). This maneuver is more easily accomplished by many children –compared to expiration. Another confusing fact is that in many cases a complete compression of the artery occurs with deep expiration (figure 4). In fact, not rarely, a total compression can also be seen already in mid-breathing. If the lumen is near total obstruction the velocities decline instead to rise further. The experienced sonographer surely won't be mocked by this well-known phenomenon which is also observed in closing ventricular septum defects and similar situations. The energy dispersion by the strong turbulences in relation to the decreasing mass of blood which is transported via the increasing narrowing causes an exponentially increasing drop of flow velocities.

In most cases the lumen changes are perfectly mirrored by appropriate changes of peak systolic velocity (figure 6). In diaphragmatic midposition the flow velocity exceeds 200 cm/s, often more than 300 cm/s. In inspiration, the velocity drops significantly and rises to maximum values in deep expiration, if the vessel is not pinched to an extent, where flow volume is severely reduced or nearly peters out at all. Then a reduction of flow velocity and a total obstruction with blocked flow an be found. Flow velocities above 2 m/s are one criterion for CGCS, useful especially in children and adolescents. These young patients often show rather fast aortic flow velocities above 1,5 m/s. In elderly patients, where aortic flow velocities above 150cm/s are rarely found, the most relevant criterion is a significant acceleration of CA flow compared to the aortic flow. Here a twofold increase of peak flow velocity is often requested to make the diagnosis. Most important in elderly patients is the need to demonstrate the change of flow velocities with breathing. Otherwise an atheromatous stenosis is

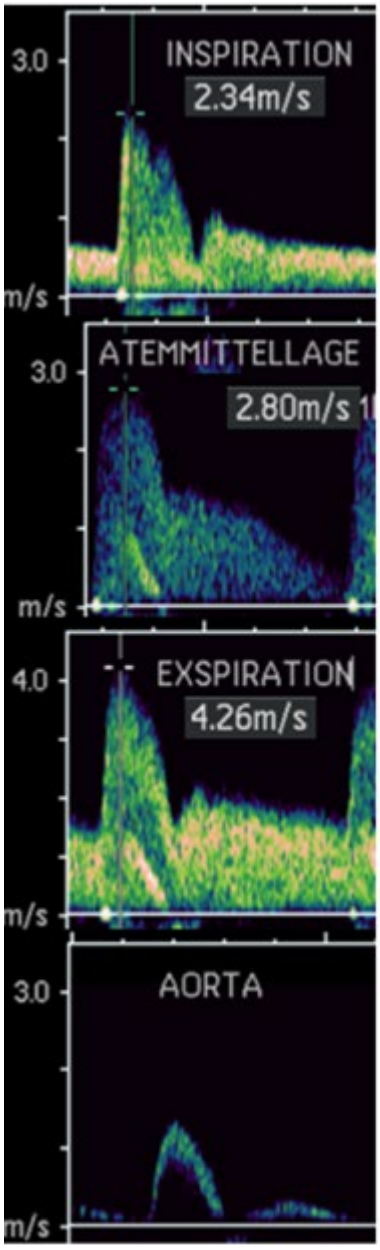

Figure 6. Comparison of angle-corrected flow velocities in the celiac trunk in CGCS. Typically flow velocities decrease from expiration via midposition to inspiration. Aortic flow is substantially slower (less than 50%).

the likely diagnosis if accelerated and turbulent flow is found at the origin of the CA.

In summary, three sonographic constituents must be sought to make the diagnosis of the celiac ganglion compression syndrome:

1. The narrowing of the vessel by compression from above with an elevated flow velocity (>200 cm/s or doubling compared to the aortic flow velocity) and
2. The decrease of compression with inspiration accompanied by the drop of the increased flow velocity.
3. In expiration contradictory changes occur compa red to those from midposition to inspiration.

MRI Imaging

MR-Angiography is helpful to provide a preoperative overview and to reassure the surgeon about the diagnosis and its severity. Images in in- and expiration

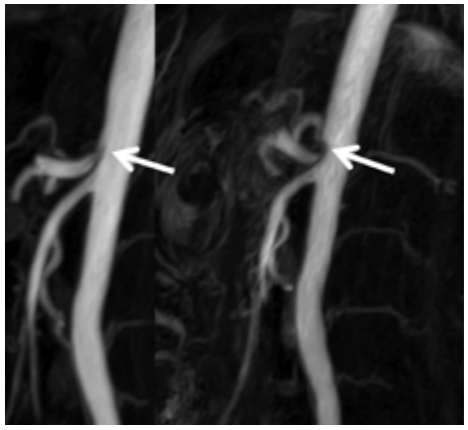

Figure 7. MR-Angiography in CGCS. Clearly visible increase of celiac trunk compression (arrows) from inspiration (left) to expiration (right)

are necessary to demonstrate the characteristic change of compression degree, most pronounced in expiration (figure 7). In very severe compression the CA is sometimes pressed into the aortic lumen for the first centimeter of its course. Then by coalescence of both lumina one might miss the diagnosis since the postcompression segment of the CA seems to be standing erect often rectangularily to the aorta. Only deep inspiration can then disclose the sharp downward course of the CA and helps to discriminate the posterior wall of the CA from the anterior wall of the abdominal aorta. Figure... shows the typical wedge or guillotine-like compression of the CA from above by the straddling arcuate ligament.

Need to correlate and subordinate ultrasound findings under clinical aspects

The diagnosis but cannot be made on sonographic grounds alone. Always a characteristic pattern of clinical symptoms needs to be present. They are mandatory, and emerge from the altered celiac ganglion, which is touched and compressed too. Many individual variations of the ganglion's development, extension and position [39] may be responsible for the inconstant correlation of clinical symptoms to the clearly visible compression of the celiac artery [40].

The invisibility of the ganglion has for long times veiled its pathophysiologic contribution to the fancy clinical spectrum of the disease. Many symptoms can only be explained by referring to its irritation. Pain is the most impressive and outstanding feature but by far not the most frequent one.

Puzzling diversity of mainly vegetative symptoms may mock (not only) the unaware

Many patients complain of a variety of vegetative symptoms that emanate from the ganglion. These are dizziness, vertigo, anorexia, (near)-fainting, diarrheal bouts, gastric fullness soon after starting to eat, nausea, shortness of breath [32]. In animal experiments even eclamptic seizures could be produced by irritating the CG [41]. Difficulties in breathing are often confused with exertional asthma, since many patients complain about shortness of breath in sport activities. The correct diagnosis can be suspected if the patient

is asked to describe clearly if the problem consists of blocked deep inspiration (which is provoked by physical activity – thus pointing to CGCS) or if the expiration is not fully possible (bronchial asthma). My hypothesis is that wide amplitudes of diaphragmatic movements are suppressed by reflexes to diminish the unpleasant sensations caused by the celiac ganglion compression which are substantially aggravated by deep breathing and expiration.

The typical patient is a young girl or woman with slender constitution and deep hollow back. The lordosis can be estimated by the attempt to push the hand between the patient's back and the examination couch. Many afflicted patients have no contact to the bed with their central parts of the lumbar spine while lying supine.

Often they suffer more from vegetative symptoms than from the pain. Pain is not regularly aggravated by eating as often claimed or sought for. It has its focus in the epigastric angle but sometimes radiates to the left thorax or the sternum.

Often another pain component is clearly discernible by its typical location from the celiac pain. This a dull but also activity-dependent pain whose maximum lies about 5 cm more caudally and merely radiates to the left flank, down towards the left paramedian region. Here it has a typical second maximum which often can be clearly demonstrated by one-finger palpation of the region of the left ovary. This is caused, according to my experience, in many cases by the congestion of the left renal vein. This disease is the "older sister" of the celiac ganglion compression syndrome, starting earlier in life and is evoked by the same anatomical constellation: a certain female constitution with a tall physique and a loose connective tissue. The compression of the left renal vein, the so called nutcracker phenomenon, causes large amounts of left renal blood volumes to circumvent the compression site, the aortic-mesenteric arterial pinch, via retroperitoneal collaterals. As a consequence pelvic congestion occurs, easily detected by left ovarian (figure 8) and uterus varices in color Doppler sonography (figure 9).

Preponderance of female sufferers – personal hypothesis

Girls and women make up more than 80% of the sufferers of both disorders. I can offer the following explanation:

With puberty the female pelvis extends more laterally than in boys. The widening of the pelvis [42, 43] pushes both femurs away from each other. Both psoas muscles form a triangle whose basis is the virtual line between both trochantera minora and whose peak is the insertion of both psoas muscles at the lumbar spine. If the basis of this triangle widens and its sides do not elongate to the same extent then only one postural response is possible - the peak is forced to come down. Because the spine is not compressible, only one way out remains – it has to bow in order to reduce the vertical distance to the base of this triangle. This – from my point of view – is the reason for the obvious female body shaping in puberty. A wide pelvis combined with a strong lordosis (compared to males) [44-46] is the dominant female feature. In fact, gender is the only variable that determines the extent of lordosis [46-48], albeit Korean authors could not find gender differences of lumbar lordosis in their local study group [49]. Pregnancies, and height were significantly and positively correlated and weight significantly and negatively correlated to the lordosis degree [50].

If changing lifestyle and body shape is to be blamed for stronger lumbar lordosis in young people due to weaker back muscles and taller statures and thus might increase the susceptibility to CA compression remains an open question due to conflicting reports [50, 51].

If the girl is tall, the flexion of the lumbar spine will be stronger as well as if the connective tissue is less firm. During childhood and adolescence lumbar lordosis, and compensatory thoracic kyphosis, increase in both sexes [52, 53] from 5 to 22 years of age [54-56]. In 5-6 years old children the sex differences of lumbar lordosis are still minimal (0,4°) with the extension ability being slightly stronger in girls than in boys (2,5°). 10 years later, in 15-16 years old adolescents of the same cohort, the lordosis in girls is 4,7° stronger and extension capability of the lumbar spine is 5,0° greater than in boys [57]. Craniofacial morphology and head posture also were correlated to spine curvature with lumbar lordosis correlated to mandible length and prognathism in children [58].

So morphometric data of the vertebral column support the clinical observation that tall, slender adolescent girls with lax ligaments and weak rump muscles are at the highest risk to develop lordosis related vascular compression syndromes, celiac artery (ganglion) compression syndrome, nutcracker syndrome [59-62] and May-Thurner- Syndrome [63, 64].

It is in fact the lordosis which is the reason for the pronounced compression of the left renal vein and also for the descent of the arcuate ligament.
This hypothesis needs to be confirmed, refuted or expanded by measurements of all mentioned components throughout childhood, adolescence and adult life in both sexes.

Our treatment strategy and operation technique

Once the diagnosis is confirmed by color Doppler sonographic examination showing the characteristic pattern of respiratory-dependent changes of celiac artery compression and often synchronously changing shape and course of this vessel we start an observation phase to objectify the type, strength and day-time pattern of symptoms. We use a questionnaire and a pain report which includes also vegetative symptoms and free space for patient remarks (an inexhaustible source of learning) as well as a column for treatment measures. Pain and other symptoms are classified on a 0 to 10 scale according to the patient's internal scale from no to unbearable symptoms. After 4-6 weeks we repeat the ultrasound exam and validate the questionnaire. If the sonographic diagnosis is steadfast an important co-diagnosis needs to be ruled in or out: the nutcracker phenomenon (NKP) of the left renal vein. Its symptoms are similar but nevertheless clearly distinguishable for the experiences investigator. NKP has a pain maximum about 3 cm below the epicenter of the celiac compression pain, also in the midline. In contrast to the latter the NKP also causes lumbar pain, headache and a distinct pain on compression of the left ovary (For details see [65]). The differentiation has therapeutic implications since NKP-complaints can be often completely resolved with a 6 week low dose aspirin treatment [65].

Of course, already the primary sonography includes all abdominal viscera to rule out other causes of pain. Gastroscopy, in some cases colonoscopy, laboratory exams (including hydrogen exhalation tests to rule out fructose and lactose mal-absorption/-digestion), and symptom-focused additional diagnostic tests are added. If other causes are excluded a preoperative MR-angiography of the abdominal vessels is carried out in in- and expiration. The celiac axis is recon-

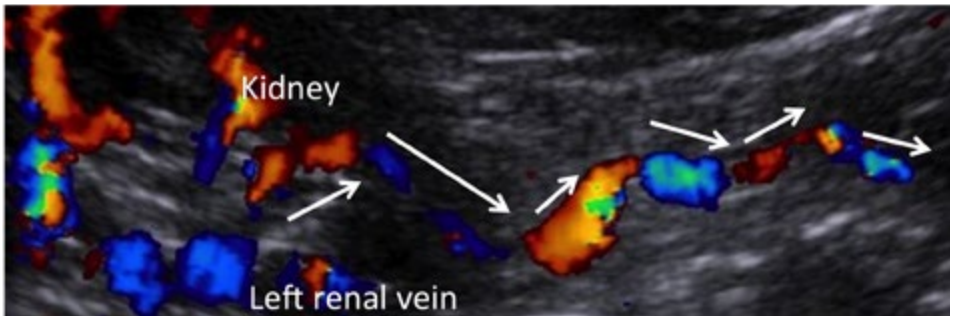

Figure 8. Collateralization of the left renal vein: here via the left ovarian vein. Meandering course with inversed flow towards the left ovary.

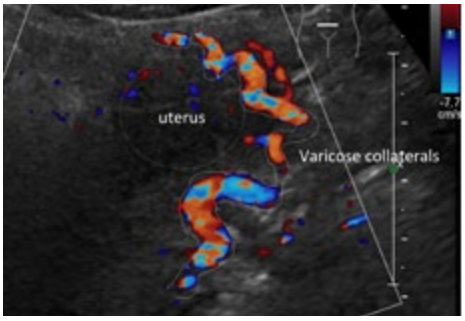

Figure 9. Varicose veins on the left side of the uterus – not rarely seen in patients with ample congestion of the midline organs in left renal vein compression.

structed in a sagittal view and its shape and course is evaluated.

If the clinical symptoms and diagnostic tests are conclusive and the suffering needs to be settled the surgeon decides to operate.

The surgical technique should be a laparoscopic one. Its advantages are earlier relief of symptoms postoperatively (immediate symptom resolution on postoperative day 1), shorter hospital stays, and a higher success rate [66], in one series of 16 adult cases a complete resolution of symptoms at a mean follow-up of 28.3 months in all of them [67]. Earlier reports used an open approach but recently laparoscopic technique has evolved to the preferred method [6, 23, 24, 66, 68-75].

Open surgery is withheld for complications and has the known general drawbacks. Moreover, the visualization of the spider-web-thin fibrotic strands of the celiac ganglion is greatly enhanced by the laparoscopic optics. These fibers, in general, are adjacent to the aortic wall so intimately that they might go unnoticed by the surgeons' naked eye. From my point of view it is but crucial to displace all parts of the celiac ganglion from the compression site, to excise all scarred remnants of the ganglion and to cut out deliberately parts of the diaphragm and its arcuate ligament. These measures should ensure the complete resolution of the celiac artery compression. Finally the vessel stretches straight and pulsates or whirs evidently.

Anesthesia combines epidural and general technique. Immediately after the operation most of the patients describe a completely liberated sensual experience which is described as being freed from a constriction, ability to breathe freely in an unprecedented manner, and, after washout of the anesthetics a complete loss of the former pain and vegetative symptoms.

With follow up ultrasound exams the operation result is documented. In some cases a residual gradient can still be found. Despite this, a complete relief from all symptoms can be stated regularly. The exact description of the pressure gradient in all respiratory phases must be documented. More important than the residual gradient is to state, that there is no variation of this gradient still existent. Especially in adults, operated in an open technique, recurrent pain can occur when the celiac ganglion is not reduced meticulously and sufficiently. Then it is crucial to know the immediate postoperative ultrasound finding. An increase of the pressure gradient might point to recurring compression, now due to scar plates or sprouting neurinomas. In some cases no pressure gradient can be found or no increasing gradient is demonstrated. Then the entire consideration if a recompression of the celiac ganglion warrants a re-operation hinges on the characteristic clinical presentation. Typically vegetative symptoms recur and point to their origin in the celiac ganglion. Intraoperatively, thick fibrotic plates or bulging buds of nervous tissue need to be removed. Sometimes a synthetic mesh can help to prevent keloid development or at least scar compression of the ganglion.

Summary

The CGCS is not as rare as always suspected. Its prevalence is about 2 %. It is easily diagnosed by color Doppler ultrasound. Clinical symptoms are mainly vegetative ones and abdominal pain. Females, mostly slender and tall individuals, comprise 80% of the cases. Laparoscopic resection of the compressing diaphragm (arcuate ligament and sometimes muscular tissue above it) and resection of the damaged parts of the celiac ganglion lead to prompt and lasting relief of all symptoms ■

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