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Case Report

ASSOCIATION OF DUNBAR, MAY-THURNER AND NUTCRACKER COMPRESSION SYNDROMES IN ONE PATIENT

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We report a case of Dunbar syndrome, May–Thurner syndrome and Nutcracker syndrome diagnosed in one patient with clinical presentation and imaging findings on Doppler ultrasonography. Dunbar syndrome or truncus coeliacus *compression syndrome is an under-diagnosed vascular compression syndrome with a lot of controversy around it because of insufficient differentiation from celiac artery stenosis. May–Thurner syndrome or iliac vein compression syndrome is an anatomically variable condition of venous outflow obstruction caused by extrinsic compression by the right common iliac artery as it crosses the iliac vein anteriorly. In Nutcracker syndrome due to compression the outflow from the left renal vein into the inferior vena cava is obstructed. The combination of all these syndromes in one patient was not described before.*

Key words: *Dunbar syndrome, May–Thurner syndrome, Nutcracker syndrome.*

INTRODUCTION

Dunbar syndrome, May–Thurner syndrome (MTS) and Nutcracker syndrome (NCS) are rarely diagnosed, and their association in the same patient has not been described before. Combined NCS and Dunbar syndrome has been reported (Linares *et al.*, 2002). Hartung *et al.* (2005) had a patient with previously stented MTS and Kurklinsky and Shepherd described a case of combined May–Thurner and Nutcracker syndrome (Hartung *et al.,* 2005; Kurklinsky and Shepherd, 2009). There is an anatomical relationship in the aetiology of all these three syndromes. Due to the great variety of symptoms and the often prevailing vegetative complaints the patients experience an odyssey through many medical disciplines, often without getting the correct explanation.

Dunbar syndrome is a consequence of extrinsic celiac artery compression and surrounding neuronal tissues by the median arcuate ligament. As an anatomic finding it was mentioned in 1917 (Stanley *et al*., 2014); the syndrome was first described by Harjola in 1963 and later by Dunbar in 1965 (Desmond and Roberts, 2004; Torres *et al*., 2017). Thus, Dunbar syndrome is also called Harjola's syndrome, coeliac artery compression syndrome, median arcuate ligament syndrome and *truncus coeliacus* compression syndrome etc.

May–Thurner syndrome also called iliac vein compression syndrome, Cockett syndrome, or iliocaval compression syndrome is an anatomically variable condition of venous outflow obstruction caused by extrinsic compression by the right common iliac artery as it crosses the iliac vein anteriorly (Mousa and AbuRahma, 2013; Demir *et al*., 2016; Srisajjakul *et al*., 2017). The Nutcracker phenomenon (NCP), or in other words called left renal vein (LRV) entrapment, was first described in the 19th century by French anatomists. On the other hand, NCS, which was named by De Schepper in 1972, is symptomatic LRV entrapment when patients present with the characteristic clinical signs and symptoms, specifically haematuria, proteinuria, flank pain, pelvic congestion in females, and varicocele in male patients (Ananthan *et al.,* 2017; Boyer *et al.,* 2018).The aim of our study was to report a case of Dunbar syndrome, May–Thurner syndrome and Nutcracker syndrome diagnosed in one patient with a clinical presentation, imaging

findings on Doppler ultrasonography and treatment options. Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

CASE DESCRIPTION

A 33-year-old male man came with complaints of discomfort in epigastrium, heartburn, nausea, and acid regurgitation from 19 year of age. He was diagnosed with gastroesophageal reflux disease and was treated with combined gastric acid reduction treatment with proton pump inhibitors, antacids and H-2-receptor blockers. He had a history of nephrolithiasis with lithotripsy as treatment from the age of 20 years and a history of haemorrhoids. Due to a hiatus hernia and progressive symptoms of gastroesophageal reflux disease, the patient had laparoscopic fundoplication in Moscow in March 2017 (A. F. Chernousov modification). Two months after surgery, all reflux complains came back. Patient was treated with long-term proton pump inhibitors and antacids. Patient went for more investigations to Praxis Prof. Dr. Thomas Scholbach in Leipzig.

RESULTS

The patient was diagnosed using colour Doppler ultrasonography with Dunbar syndrome, May–Thurner syndrome and Nutcracker syndromes. The patient presented the most frequent complaints of compression of the celiac trunk and the adjacent celiac ganglion, such as abdominal pain, tachycardia (also manifesting as postural tachycardia syndrome), heartburn, nausea, and reduced tolerance to physical activities. Figure 1 gives the colour Doppler of the compressed and displaced coeliac trunk in mid-position of the diaphragm presented in the patient. The following image shows the acute-angled displacement of the *truncus coaliacus* caudally by the axcess *ligamentum arcuatum*.

Changes of the compression of the left common iliac vein between the right common iliac artery anteriorly at the level of the crossover position and the fifth lumbar vertebral body posteriorly are seen in the patient's colour Doppler image where flow acceleration is shown (Fig. 2). The sacral bone and its promontory compresses the left common iliac vein from behind. We can observe a low flow velocity of 25 cm/s before the compression site (Fig. 3).

Episodically, the patient appeared with such symptoms like abdominal pain and haemorrhoidal bleeding, which are common for Nutcracker phenomena. The diagnosis of it is made straightforward by means of functional colour Doppler ultrasound. Figure 4 shows a colour Doppler sonography of the patient, where a representation of the renal vein (red colour) is incarcerated between the aorta and the arteria mesenterica.

The operative procedure of Dunbar syndrome is based on the visualisation and division of the arcuate ligament with decompression of the celiac nervous plexus as well as the

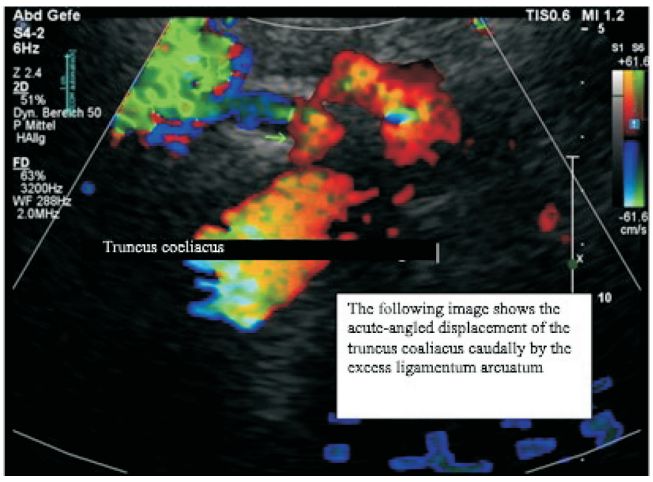

Fig. 1. Dunbar syndrome colour Doppler sonography of presented patient.

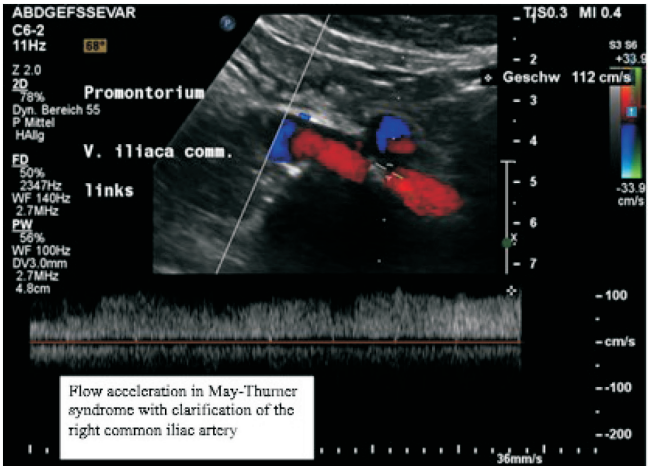

Fig. 2. May–Thurner syndrome colour Doppler sonography of presented patient.

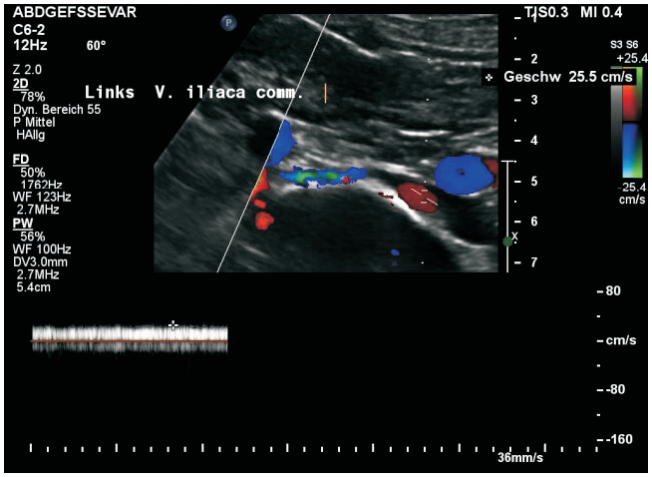

Fig. 3. Patient's presented compression of the left common iliac vein while crossing the promontory, low flow velocity before the compression site.

celiac artery. May–Thurner syndrome is an incidental finding in most cases as well as for our patient. Treatment of MTS is based on the clinical presentation. Treatment is initiated only in symptomatic patients. Treatment for NCS varies based on the age of the patient and scope of symptoms. Only symptomatic NCS with severe symptoms should be

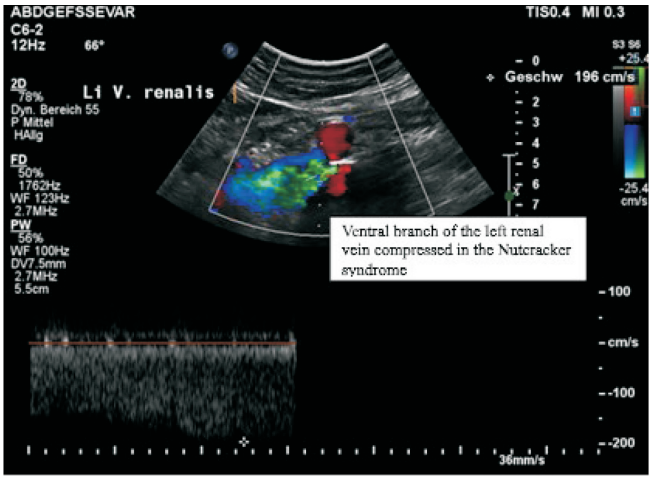

Fig. 4. Nutcracker syndrome colour Doppler sonography of presented patient.

treated with endovascular or open surgical techniques. The patient is planned to be treated in Germany and with the operative procedure of Dunbar syndrome.

DISCUSSION

Dunbar syndrome. Dunbar syndrome is frequently overlooked, it is not so rare vascular compression syndrome with a lot of controversy around it because of asymptomatic persons with evidence of celiac artery compression and unsuccessful operations if the neural tissue is spared (Stanley *et al*., 2014). The female to male ratio is 9 : 1 and the classic patient is a female aged between 18 and 30 years (Torres *et al*., 2017).

The median arcuate ligament connects the left and right diaphragm crus (Tracci, 2015), and the mean level at which the median arcuate ligament crosses the aorta is at the junction of the middle and lower third of the first lumbar vertebra (Stanley *et al*., 2014). At the same time, the mean celiac artery's origin is between the lower third of the 12^{th} thoracic vertebra and middle third of the first lumbar vertebra, leading to potential compression of artery at the aortic hiatus (Stanley *et al*., 2014). But because of the celiac artery's variable caudal descending during embryonic development, its origin is very inconsistent. The most commonly encountered origin of the superior mesenteric artery (SMA) is also at the lower third of the first lumbar vertebra, and its close proximity to the celiac artery and aortic hiatus explains its occasional narrowing in patients with the Dunbar syndrome (Stanley *et al*., 2014).

Minor indentations along the superior border of the celiac artery by the ligament affect approximately 40% of the adult population (Stanley *et al*., 2014), less common (in 15% of cases) severe narrowing occurs resulting in celiac artery occlusion and extensive gastroduodenal and pancreaticoduodenal collateral vessels development between the superior mesenteric artery and celiac artery circulations.

The celiac artery's narrowing can be caused by two factors: one is anatomical abnormalities such as abnormally low-lying median arcuate or unusually high celiac artery's origin. The other, more important, is the lordotic bowing of the lumbar spine, which drags down the aortic hiatus thus pinching the celiac artery. The relative position of the celiac axis and MAL varies with respiration, the celiac indentation typically is less apparent during inspiration, and compression of the celiac axis typically increases during expiration. Severe compression occurs in approximately 1% of patients and persists during inspiration (Baskan *et al.*, 2015). This compression causes a scarring and inflammation as well as excessive proliferation of constricting neural and fibrous tissue, which can entrap and narrow the celiac artery origin additionally (Stanley *et al*., 2014). In some patients, abnormal blood flow within the celiac artery leads to secondary intimal fibroplasia and a further narrowing of the already compressed vessel (Stanley *et al*., 2014).

One of the hypothesis suggests that compression of the celiac artery causes purely intestinal ischaemia (Torres *et al*., 2017), but it has low objective support because the splanchnic circulation, which is supplied by the celiac artery and superior and inferior mesenteric artery, has a large network of collateral vessels; to cause intestine ischaemia in case of Dunbar syndrome a haemodynamically significant superior mesenteric stenosis would be necessary. Usually these patients become symptomatic, if the arterial blood flow is reduced by 60–75% (Xian-Ming *et al*., 2018). Still, it is conceivable that in addition to celiac artery compression, a concomitant narrowing within the SMA itself could contribute to the symptoms encountered in some patients. It is also possible that the collaterals from the SMA to the celiac artery bed are inadequate in supplying blood to the foregut circulation.

Abdominal discomfort is most often periumbilical and epigastric (Stanley *et al*., 2014). Association with vegetative symptoms such as nausea, vomiting, dizziness, tachycardia, diarrhoea, weight loss and sweating is very common due to the irritation of the celiac nervous plexus (Torres *et al*., 2017; Scholbach, 2006). An upper abdominal bruit that varies with respiration that is more pronounced with fixed inspiration and hyperventilation during physical exercises is a common finding. Others clinical symptoms are bloating and reduced appetite (Torres *et al*., 2017). A minority of patients have gastroparesis (Tracci, 2015), and retrosternal pain is rarely reported (Karavelioglu, 2015). The pain is caused by the mechanical irritation of the celiac plexus nerve fibres. A study by Scholbach in 2006 showed the results of fifty-nine patients who had criteria of Dunbar syndrome: 71% of patients had abdominal pain, 29% nausea, thoracic pain 22%, heartburn 17%, weight loss 15%, vomiting 15%, systolic murmur 15%, postprandial accentuation of symptoms 15%, diarrhoea 14%, respiratory discomfort 14% and syncope 12% (Scholbach, 2006).

The non-specific nature of these clinical symptoms mimics digestive diseases in a small subset of patients, thus delay-

ing diagnosis and resulting in anxiety, depression and other psychiatric symptoms (Xian-Ming, 2018).

Dunbar syndrome is diagnosed by colour Doppler ultrasound (Scholbach, 2006). Some other diseases may cause similar symptoms like esophagitis, pancreatitis, cholelithiasis, and food intolerance. The diagnosis of Dunbar syndrome may be made by selective angiography, magnetic resonance angiography, spiral computed tomographic angiography, and colour Doppler ultrasound, which is today the method of choice. Respiratory manoeuvres are necessary for complete diagnosis. The compression is respiratory-dependent, it is stronger in expiration and resolves during deep inspiration. A significant celiac artery compression is suspected when the peak systolic velocity exceeds the aortic one by a factor of two. But more important is the irritation of the surrounding nervous tissue, which is highlighted by the deformation of the celiac artery. The typical hook-like downward displacement followed by a dilatation of the celiac artery is a typical finding (Torres *et al*., 2017; Stanley *et al.,* 2014).

The operative procedure is based on the visualisation and division of the arcuate ligament with decompression of the celiac nervous plexus as well as the celiac artery.

Early results of median arcuate ligament transection have documented resolution or improvement of symptoms in greater than 80% of cases. Late outcomes of operative therapy are salutary in 50% to 75% of patients and are reported to be greater in series of carefully selected patients (Stanley *et al*., 2014).

May–Thurner syndrome. May–Thurner syndrome was first described by McMurrich in 1908 as isolated left lower extremity swelling caused by left iliac vein compression (Mousa and AbuRahma, 2013). But the syndrome was fully understood only in 1957. After the evaluation of 430 cadavers, May and Thurner found that intraluminal venous spurs existed in about 22% of this cohort (Mousa and AbuRahma, 2013). MTS is believed to be rare and the exact incidence and prevalence of MTS is unknown. It has been estimated to occur in 2–3% of patients who present with some sort of lower extremity venous disorder. But in autopsy studies MTS is diagnosed in 22–32% (Mousa and AbuRahma, 2013; Srisajjakul *et al*., 2017)

MTS is defined as extrinsic compression of the left common iliac vein between the right common iliac artery anteriorly at the level of the crossover position and the fifth lumbar vertebral body posteriorly (Mousa and AbuRahma, 2013; Srisajjakul *et al*., 2017). The left common iliac vein is more likely prone to compression, given the more longitudinally transverse course it takes, compared to the more vertically oriented right common iliac artery (Zhou *et al.,* 2015). This causes partial or complete impedance to the left iliac vein outflow with subsequent possible obstruction and congestion as well as ipsilateral deep vein thrombosis (DVT) of the pelvis and left leg. Because compression usually occurs against lower lumbar vertebrae, MTS should be suspected in patients with stronger lordosis, scoliosis and dilated perimedullary veins (Mousa and AbuRahma, 2013).

It has been found that, as the result of this vascular compression and chronic arterial pulsation, the iliac vein develops a vascular thickening by extensive proliferation of intima that causes stenosis in some patients (Demir *et al*., 2016; Zhou *et al.,* 2015).

The latest research indicates that MTS may be present in 66% of the general population without any symptoms (Srisajjakul *et al*., 2017). If symptoms occur, it usually presents in the second or third decade of life and is more common in women. Patients may present with acute or chronic pain and swelling of the left lower extremity, venous stasis and skin discoloration. Other presentations of MTS include: acute DVT, venous claudication, chronic venous insufficiency (CVI), recurrent superficial venous thrombophlebitis, and, in rare instances, pulmonary emboli, iliac vein rupture or *phlegmasia cerulea dolens* (Mousa and AbuRahma, 2013; Demir *et al*., 2016; Srisajjakul *et al*., 2017).

Diagnosis of MTS can be made with a combination of the clinical presentation and objective testing. Colour Doppler ultrasound as well as cross-axial imaging, including computed tomographic scans with venous phase or magnetic resonance venography, are sensitive tools for estimating the degree of stenosis and other anatomic variations that may need to be evaluated (Mousa and AbuRahma, 2013). The important diagnostic CT or MRI findings are extrinsic compression of the left common iliac vein at the crossover point, presence of venous collaterals crossing the pelvis to join the contralateral vein, and venous thrombosis (if present). The left common iliac vein compression is best seen in the axial plane (Srisajjakul *et al*., 2017). Of course, colour Doppler ultrasound should be preferred, since it delivers haemodynamical information, not yielded by the other imaging techniques.

Ascending venography has been traditionally performed for definitive diagnosis but often fails the diagnosis. This venography illustrates the venous compression and allows haemodynamic assessment by pressure gradient measurement (Srisajjakul *et al*., 2017).

The treatment is aimed to reduce symptoms and minimise risk of complications (Demir *et al*., 2016). Treatment includes staged thrombolysis with or without prophylactic retrievable inferior vena cava filter placement, followed by angioplasty stenting of the left iliac vein in MTS patients with extensive DVT (Mousa and AbuRahma, 2013). Surgical repair of MTS was done previously; however, with advancement of technology less invasive endovascular treatment has gained popularity (Srisajjakul *et al*., 2017).

Nutcracker phenomen. The most common mechanism of LRV compression is lordotic lifting up of the abdominal aorta, thus compressing the left renal vein from behind. Other possible mechanisms are abnormal posterior ptosis of the left kidney with secondary stretching of the LRV over

the aorta and an abnormally high course of the LRV. Compression is aggravated by the standing position because of the increase of lordosis and may be underestimated in imaging examinations performed in the decubitus position (CT, MR angiography). The most frequent form of NCP is anterior NCP when compression of the LRV is by the superior mesenteric artery (SMA) and the aorta. A second variant is the posterior NS, in which the LRV is compressed between the aorta and the vertebral body.

Vein compression induces obstruction with LRV, venous hypertension and the development of varicose veins and collateral pathways, which cause the symptoms. Beinart and associates showed that the renocaval gradient is less than 1 mm Hg in control patients and Nishimura and colleagues regarded pressure gradients of 3 mm Hg or higher as indicative of renal hypertension (Yves and Hartung, 2014).

Prevalence of NCS is high in our experience, but despite characteristic symptoms the diagnosis of NCS is frequently overlooked.

Another symptom commonly noted with NCS is orthostatic proteinuria, hypothesised to be caused by increased pressure in the LRV, mild subclinical immune injury, and changes in renal haemodynamic on standing, altering the release of angiotensin II and norepinephrine (Yves and Hartung, 2014).

NCS is easily diagnosed with colour Doppler Ultrasound and should be searched for in all patients with abdominal pain.

Doppler ultrasonography should be used as the initial diagnostic test in patients with suspected NCS. Doppler ultrasonography can assess the anterio-posterior diameter and peak velocity of the LRV in two places: at the level of the renal hilum; and at the point where the LRV crosses between the aorta and the SMA (Kuo-Kang *et al.*, 2013; Herman *et al*., 2018).

Furthermore, findings such as an LRV hilar diameter to aortomesenteric diameter ratio of 4.9, the "beak sign," and an SMA branching angle of # degrees from the aortic origin can be also useful for the diagnosis of NCS (Velasquez *et al.,* 2018).

Selective LRV phlebography can show compression of the LRV with dye stasis, varicose veins, and collateral pathways. In the case of left gonadic vein reflux, pelvic views must be obtained.

In cases of haematuria, urologic evaluations should also be performed (urine microscopy, cystoscopy, flexible ureterorenoscopy, sometimes renal biopsy).

Treatment for NCS varies based on the age of the patient and scope of symptoms. Only symptomatic nutcracker syndrome with severe symptoms should be treated with endovascular or open surgical techniques. Children should be treated conservatively. Indications for conservative treatment are mild haematuria or with mild and tolerable symptoms. Surgery is considered in cases of persistent or anaemising haematuria, lumbar or incapacitating pelvic pain, or the presence of severe congestive pelvic symptoms, autonomic dysfunction, impairment of renal function including persistent orthostatic proteinuria, varicocele formation; and for ineffective conservative measures after some months. Before initiating treatment, differential diagnoses should be excluded (Yves and Hartung, 2014; Ananthan *et al.,* 2017).

CONCLUSION

Dunbar syndrome, May–Thurner syndrome and Nutcracker syndrome are rarely diagnosed, and their association in the same patient has not been described before. There is an anatomical relationship in the etiology of all these three syndromes.

CONFLICTS OF INTEREST

The authors declare that there is no conflict of interest regarding the publication of this article.

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SAISTĪBA STARP KOMPRESIJAS SINDROMIEM *DUNBAR, MAY–THURNER* UN *NUTCRACKER*, KAS DIAGNOSTICĒTI VIENAM PACIENTAM

Mūsu ziņojumā apskatīts klīniskais gadījums par vienam pacientam diagnosticētu kompresijas sindromu kopumu (*Dunbar* sindroms, *May–Thurner* sindroms un *Nutcracker* sindroms) ar klīnisko izklāstu un radioloģisku atradi pēc Doplera ultrasonogrāfijas metodes. *Dunbar* sindroms jeb trijzaru stumbra kompresijas sindroms ir reti diagnosticēts asinsvadu kompresijas sindroms, par kuru notiek daudz diskusiju, jo nav pietiekamas atšķirības no trijzaru stumbra stenozes. *May–Thurner* sindroms jeb kreisās kopējās iegurņa vēnas saspiešanas sindroms ir anatomiski mainīgs venozās plūsmas aizsprostojuma stāvoklis, ko izraisa ārējā saspiešana ar labo iegurņa kopējo artēriju, kad tā šķērso iegurņa vēnu priekšpusē. *Nutcracker* sindroma gadījumā kompresijas dēļ tiek aizkavēta plūsma no kreisās nieru vēnas uz apakšējo dobo vēnu. Pēc literatūras datiem visu šo sindromu kombinācija vienam pacientam iepriekš nebija aprakstīta.