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MINIREVIEWS

Renal venous hypertension

Makhmud Muslimovich Aliev, Rustam Zafarjanovich Yuldashev, Gulnora Srajitdinovna Adilova, Avazjon Abdunomonovich Dekhqonboev

Makhmud Muslimovich Aliev, Rustam Zafarjanovich Yuldashev, Gulnora Srajitdinovna Adilova, Avazjon Abdunomonovich Dekhqonboev, Department of Pediatric Surgery, Republican Specialized Scientific Practical Medical Centre of Pediatrics, Tashkent 100179, Uzbekistan

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Correspondence to: Rustam Zafarjanovich Yuldashev, MD, Department of Pediatric Surgery, Republican Specialized Scientific Practical Medical Centre of Pediatrics, District Olmazar, str. Chimbay № 2, Tashkent 100179, Uzbekistan. paediatric.surgery@yahoo.com
Telephone: +998-90-9575082
Fax: +998-371-2293873

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Abstract

Renal venous hypertension usually seen in young, otherwise healthy individuals and can lead to significant overall morbidity. Aside from clinical findings and physical examination, diagnosis can be made with ultrasound, computed tomography, or magnetic resonance conventional venography. Symptoms and haemodynamic significance of the compression determine the ideal treatment method. This review of the literature discusses normal and pathological developmental aspects of renocaval venous segment and related circulatory disorders, summarizes congenital and acquired changes in left renal vein and their impact on development of renal venous hypertension. Also will be discussed surgical tactics of portosystemic shunting and their potential effects on renal hemodynamics.

Key words: Renal venous hypertension; Nutcracker syndrome; Kidney; Portal hypertension; Splenorenal shunts

Core tip: Renal venous hypertension characterized by the presence of left renal vein dilatation, varicocele and hematuria. Being a rare cause of hematuria its etiology is diverse but of precise characteristics. Diagnosis is not easy and treatment requires ruling out its precise etiology and considering the intensity of the compression phenomenon because of interventionist attitudes have important implications and are not risk free.

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INTRODUCTION

Renal venous hypertension (RVH) - venous insufficiency caused by inadequate drainage of blood through the renal vein[1]. There are two main reasons in the development of the RVH: Structural abnormalities of

renocaval segment; acquired changes of the left renal vein.

Structural anomalies of renocaval segment and their clinical importance in the development of the RVH

Development of renocaval segment (inferior vena cava, renal, gonadal, adrenal and lumbar veins) - is a complex process in which there is consecutive regression and shifting of three venous structures, such as posterior cardinal, supracardinal and subcardinal veins[2-5]. Inferior vena cava (IVC) and its branches formed from different embryological structures; their segments formed from all three systems mentioned above. The formation of these veins can be impaired at any stage of development[2,3].

Among the developmental abnormalities of the IVC clinical significance in development of the RVH matters - left-sided IVC, with this type of abnormality abdominal aorta compresses IVC at the site of their contact, thus will cause congestion in left renal vein (LRV) and recurrent left sided hematuria[6,7].

Typical abnormality of the right renal vein is anomaly of their quantities, which is due to the fact, that the right renal vein embryogenesis does not undergo significant transformations. Essentially, the significance of these abnormalities in development of RVH negligible[2,3,8].

Clinically significant abnormalities often observed in the LRV, which related to its development. For example, retention of both limbs of the left portion of circumaortic venous ring leads to the formation of the circumaortic LRV, which occurs in 1%-17% of cases according to different authors[2,3,5,8-13]. In this type of anomaly there are pre-aortic and retroaortic limbs. In this situation, the pre-aortic limb usually receives the adrenal, gonadal, and phrenic veins; the retro-aortic limb receives the lumbar and the hemiazygous veins. The retroaortic limb passes obliquely and downward to reach the inferior vena cava at a lower level[8-13]. The clinical significance of this anomaly is that the impeded outflow from the retroaortic limb leads to congestive venous hypertension and increased blood flow in pre-aortic limb[8,9,11-13]. Knowledge of this anomaly is important for the angiographer performing renal and/or adrenal venography. In addition, it is of surgical importance when a left renal transplant and/or splenorenal shunt are considered. As for splenorenal shunt operations, opinions are contradictory. For example, some authors[14] recommend to perform splenorenal shunt, without the risk of RVH, while according to other researchers[15,16] the connection of splenic vein to retroaortic limb leads not only to inadequate drainage portal system with recurrent bleedings, but also the risk of development of RVH.

Another type of abnormality is retroaortic LRV (single or multiple). The retroaortic type of LRV occurs in approximately 2-6.6/cent[17-22]. When the ventral limb atrophies a retroaortic renal vein occurs. In this transformation, there are anatomical prerequisites for disorders of venous hemodynamics - the emergence of congestive venous hypertension, clinically manifested by proteinuria or hematuria and the development of the secondary varicocele[1,5,11,19,23]. Performing splenorenal shunt in this type of LRV is not advisable, since drainage of a large amount of blood from the portal system leads to RVH one hand and recurrent bleeding from gastroesophageal varices on the others[15].

Acquired changes of the LRV

Human body has anatomical preconditions, which may cause significant haemodynamic alterations that may lead to clinical symptoms and significant associated morbidity. The clinical manifestations of this predisposition is nutcracker syndrome[24]. In view of the insufficiency of symptoms during the first decade of life, specified condition practically have not described in pediatric patients, in most cases classified as associated finding. The nutcracker syndrome refers to compression of the LRV between the superior mesenteric artery and abdominal aorta. Obstruction of LRV occasionally causes clinically significant venous hypertension resulting in unexplained left flank, gross haematuria, with formation of periureteric and gonadal varices and varicocele in relatively young and previously healthy patients[25-29]. Other possible symptoms include pelvic congestion, chronic pediatric fatigue syndrome and orthostatic proteinuria[30-38].

Other rare acquired causes of RVH includes renal vein thrombosis, organic renal vein stenosis and arteriovenous fistula[39-41].

Well known that LRV mostly used in performing various types portosystemic shunts for portal hypertension. Issues related to presence of RVH in patients who underwent portosystemic shunting recent years draw increasing attention of researchers[15,16,42,43]. The data about the state of the left kidney after portosystemic shunting operation are very controversial. For example, some authors argue that performing end-to-end splenorenal shunt provides the venous drainage from the portal system to IVC without renal dysfunction[44-48]. But according to other data[14-16,42,43], impeded outflow of the left renal vein leads to not only venous hypertensive nephropathy, but can be cause of insufficiency of created anastomosis and therefore unsatisfactory results of surgical treatment. Furthermore, impeded outflow of LRV results in venous hypertension with the formation of intra- and extrarenal collaterals and/or the development of gonadal vein reflux resulting retrograde flow and has been implicated in the development of varicocele or ovaricocele[49]. According to experimentally induced extrahepatic portal hypertension[50-54] shunting end renal vein to side splenic vein (renosplenic) after ligation of the LRV lateral to the adrenolumbar tributary, leads to haemorrhagic necrosis of the left kidney. Thus, the ureteric, lumbar and pericapsular collaterals cannot adequately drain the left kidney. Ligation of the LRV on the medial side of the adrenolumbar tributary maintained a patent left renal vein in all cases[50,52,53].

Practical experience has shown that performing splenorenal anastomosis with ligation of the LRV proximal to the confluence of the adrenal vein - in one third of cases causes decreasing of renal function (according to the excretory urography), renal infarction, hematuria and proteinuria[52,53].

In addition, in the pathogenesis of the RVH renal arterial blood flow is essential[55]. High pressure in the renal artery in systemic arterial hypertension increases tone of sympathetic-adrenal system, which causes vasoconstriction in the cortex and increases medullary blood flow. Autoregulation mechanisms lead to increasing pressure in the renal venous system, which are the anatomical and functional characteristics of the vascular bed of the kidney. The diversity of intrarenal arteriovenous shunts, venous network ensures acceptance of a large amount of blood in the face of increasing its arterial delivery - this is the pathogenesis of RVH in systemic arterial hypertension[55]. On the other hand, congenital or acquired arteriovenous fistula leads to the restructuring of angioarchitectonics of kidneys and in this case pressure in renal veins increases due to shunting of blood through the abnormal arteriovenous communications. The blood from the arterial bed drains to venous rout bringing extraordinary pressure to the veins. Thus, developed the renal venous hypertension[55,56].

Diagnostics of RVH

In the evaluation of renal hemodynamics, intravascular pressure indicators are most important. Retrograde left renal venography and measurement of the pressure gradient between the left renal vein and the IVC are procedures of choice for diagnosing RVH. Normally, this gradient is determined in a horizontal position from a healthy child was equal to 0.13 ± 0.02 kPa, with individual variations 0.33 ± 0.05 kPa[57,58]. A number of studies indicated that the anomalies of the LRV (usually circumaortic and retroaortic LRV), the pressure gradient increases significantly (up to 0.86 kPa). However, these techniques are invasive and use of such invasive examinations is generally deemed imprudent in children, and non-invasive imaging studies are preferable. Recently progressive development of non-invasive imaging techniques led to that Doppler ultrasound (US) has become the method of choice in the diagnosis of RVH. During the last decade, increased the number of publications describing different ultrasound descriptions of renocaval segment anomalies[1,27-29,34-36,57-60]. Also in details described intrarenal arteriovenous shunts[61-63]. Recent publications dedicated in most cases for nutcracker syndrome[27-29,34-36,57-60]. Kim *et al*[58] suggested that a ratio of the AP diameter, and peak velocity (PV) between the hilar and aortomesenteric portions of the LRV of greater than 5.0 could be used as the cut-off level for the diagnosis of nutcracker syndrome with a sensitivity of 80% and a specificity of 94%. However, it has not yet been confirmed whether these criteria can be applied to children with clinically suspected nutcracker syndrome. In addition, detection of collateral veins around the left renal vein at color Doppler US is a reliable criterion for the diagnosis of nutcracker phenomenon[27]. However, the LRV flow patterns and collateral vein formations associated with nutcracker phenomenon depend on the degree and stage of the phenomenon[58]. In patients with early nutcracker phenomenon, LRV distention and high pressure gradients exist before collateral veins develop. Moreover, in patients with collateral veins, the presence of a distended left renal vein and hypertension of the left renal vein indicate that the nutcracker phenomenon is noncompensatory[58].

Regardless of the incidence angle, the resistances in the renal artery can be evaluated by measuring the resistive index and pulsatility index if the vessel is identified by colour Doppler. Increasing these rates in some cases may be indirect evidence of the venous outflow disturbances from LRV[15,16].

Recently, non-invasive methods such as computed tomography (CT) and magnetic resonance imaging (MRI) have been used in the diagnosis of nutcracker syndrome[10,12,41,64-66]. Performing of the study for our opinion, more appropriate to carry out not only for diagnostic purposes of RVH but also to assess the topographic anatomy course of renocaval segment and their relative position to the vessels of the v. porta and abdominal aorta in the planning of vascular surgery in the retroperitoneal space.

The clinical manifestations of RVH

The clinical presentation of RVH include the development of collateral blood flow and symptoms of renal function disorders[1,5,28,29,67]. The increased venous pressure within the renal circulation promotes the development of collaterals of the renal pelvis, and this plexus of abnormal hypertensive veins causes microhematuria or gross hematuria, orthostatic proteinuria[6,19,30-38]. Other possible symptoms include left flank pain, left-sided varicocele, pelvic congestion, chronic pediatric fatigue syndrome, and gastrointestinal symptoms[1,43,67].

Performing various types of splenorenal shunts using abnormally developed LRV due to portal hypertension can become a reason of unsatisfactory results with recurrent bleeding from gastroesophageal varices[14-16,42,43,68]. In addition, shunting the large amounts of blood from portal vein and its tributaries to abnormally developed LRV manifests as clinical signs of renal venous hypertension[14,16,69].

Different therapeutic methodologies have been used in treatment of RVH. In general, moderate manifestations may be controlled with conservative methods[70]. Nearly all surgical approaches aim to relieve the LRV outflow obstruction[70-85]. Surgical modalities including autotransplantation of the left kidney, LRV bypass with graft interposition and reanastomosis to the IVC anteriorly has been performed with satisfying results[73-75]. Renal autotransplantation may offer maximal efficiency in terms of normalizing renal venous

circulation. In more severe cases with hematuria, significant stenosis of LRV, varicocele, left flank pain and pressure gradient more than 1.33 kPa preferable intervention on LRV. Lot of evidence of the efficacy of endovascular interventions - methods of stenting and balloon angioplasty[76-85]. Initially performed *via* a transperitoneal approach, an external stent can be wrapped around the renal vein to prevent its compression by the mesoaortic clamp. The procedure has now also been performed by laparoscopic surgery. External and internal stenting procedures by either minimally invasive or endovascular approaches are promising treatment options. However, the risk of erosion of adjacent structures and dislodgment of the stent has not been defined yet.

However, surgical treatment methods have certain disadvantages. Thus, venous vascular suture can be considered as a potential source of thrombosis[72,83]. Postoperative complications may even lead to nephrectomy[84]. Even traditionally performed safe operations intravascular stents placement - can have few complications[79-82].

CONCLUSION

There are reasonable basis for research on the status of renocaval segment for modern pediatric surgeons, urologists, specialists concerned in portal hypertension, liver kidney transplant surgeons. The presence of RVH should be considered on the basis of a thorough clinical examination in patients with hematuria, left flank pain, varicocele, and symptoms of pelvic venous congestion. Dilatation of LRV and its tributaries, anomalies, additional communications observed on ultrasonography, computed tomography CT, or MRI should alert the physician to consider the diagnosis. If the symptoms merit, in particular if cystoscopy demonstrates left ureteral hematuria, selective left renal venography with pullback determination of renocaval pressure gradient is the diagnostic test of choice and should be performed in all patients. At the same time, complexity of revealing the causes of RVH with above mentioned methods, it is feasible to study the role of arterial blood, not only because of their lack of data, but also well-known factors associated with abnormal blood supply, and it is widely performed operations of decompression of the portal system through the LRV. Despite numerous studies, reasonableness of performing various types of splenorenal shunts in portal hypertension with prerequisites for RVH remains debatable. Finally, it is not enough studied phenomenon of nutcracker syndrome after surgical and congenital splenorenal shunts.

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