AJPMHS GLOBAL RESEARCH NETWORK

**AMERICAN Journal of Pediatric Medicine and Health Sciences**
*Volume 3, Issue 5, 2025 ISSN (E): 2993-2149*

# Clinical Features of Congenital Anomalies of the Kidneys and Urinary Tract in Newborns

**Izzatullaev S.A., Islamov Sh.E., Yuldashev B.A.**
Samarkand State Medical University

**Abstract:** The article is devoted to the analysis of literature data on the clinical features of congenital anomalies of the kidneys and urinary tract in newborns. It is noted that at present, various types of congenital anomalies of the kidneys and urinary tract in children have been identified, but they do not fully reflect the clinical features of the pathologies. It is necessary to conduct morphological studies in this area for a full justification and correct verification of the diagnosis.

**Keywords:** newborns, congenital anomalies of the kidneys and urinary tract, clinical features.

Relevance. The term CAKUT includes combined congenital anomalies of the kidney and urinary tract (Barakat A. J., 1991). CAKUT is a heterogeneous group of combined congenital anomalies of the kidney and urinary tract, which are often diagnosed prenatally and in newborns [9, 13, 27].

In foreign literature, the terminology Congenital anomalies of the kidney and urinary tract (CAKUT) is used [26, 30, 54].

Taking into account modern data, CAKUT is a group of unilateral or bilateral congenital anomalies of the kidneys and urinary tract, characterized by phenotypic and genetic heterogeneity with varying degrees of clinical manifestations and severity [49].

For the first time, A. J. Barakat and J. G. Drougas (1991) systematized combined CAKUT, identifying anomalies of the kidneys, ureters, bladder and urethra:

I. Congenital anomalies of kidney development:

Agenesis/aplasia of the kidney

Dysplasia of the kidney: multicystic kidney, cystic kidney, hypoplasia of the kidney Hydronephrosis

Ectopia of the kidney

Anomalies of the relative position of the kidneys

II. Congenital anomalies of the ureters:

Megaureter

Vesicoureteral reflux (VUR)

Duplicate ureters

Stenosis of the ureteropelvic segment

Stenosis of the ureteral pelvis

III. Congenital anomalies of the urinary bladder:

Bladder exstrophy

Neurogenic bladder dysfunction (NBD)

Bladder diverticulum

IV. Congenital anomalies of the urethra:

Posterior urethral valve

Anterior urethral obstruction.

In 2019, E. Winnicki and H. Copp [54] supplemented the classification of A. J. Barakat and J. G. Drougas (1991) CAKUT:

1) Congenital anomalies of kidney development

Duplexion of the kidney (unilateral and bilateral, complete and incomplete)

Duplexion of the collecting system of the kidney

2) Congenital anomalies of the ureters

Ureterocele

Ectopia of the ureteral orifice.

In pediatric patients, CAKUT is considered as isolated (a combination of congenital anomalies of the kidneys and urinary tract) and syndromic (a combination of congenital anomalies of the kidneys and urinary tract with developmental anomalies of other organs and systems) [26, 27, 34, 50].

N. Ganesan et al. (2023) in a study of 255 patients found CAKUT in 45: isolated (27%), syndromic (51%) and CAKUT with congenital anomalies of other organs and systems (22%) [26].

I. Congenital anomalies of kidney development

Anomalies of the number of kidneys

Bilateral renal agenesis or POTTER syndrome is a congenital malformation incompatible with life. The incidence rate is 1:10,000 newborns [46]. Patients with POTTER syndrome are characterized by "P" - pulmonary hypoplasia - hypoplasia of the lungs, "O" - oligohydramnios - oligohydramnios, "T" - twisted face - facial deformations, "T" - twisted skin - folded "senile" skin, "E" - extremity defects - limb deformations, "R" - renal failure - lack of function of both kidneys in the fetus, most often due to bilateral renal agenesis [1, 2].

In unilateral renal agenesis, one of the kidneys together with the ureter is completely absent. The incidence is 1:2000 – 1:5000 live births. It is more common in boys, and in 59% of cases, agenesis of the left kidney is diagnosed [46]. It can be combined with other defects of the urinary system (absence of half of the urinary triangle, absence of one of the ureteral orifices and/or a blind-ending ureter with a normal localization of its orifice in the bladder) and malformations of other organs and systems (esophageal atresia, heart defects, malformations of the gonads in girls, and testicular hypoplasia in boys). In patients with unilateral renal agenesis, microalbuminuria was detected in 21%, renal arterial hypertension in 16%, and CKD C3-5 in 10% of cases [54].

S. G. Woud et al. (2023) presented the results of a 20-year cohort study of 944 patients with a single kidney, 75% of which were due to agenesis of the contralateral kidney and 25% due to nephrectomy of the contralateral kidney for various reasons. Patients with a combination of unilateral renal agenesis with other VAPM or malformations of other organs and systems were not included in this study. The authors found that 6% of patients with unilateral renal agenesis

were diagnosed with a significant decrease in SCF by the age of 15 years and 30% by the age of 30 years [53].

In renal aplasia, the kidney is macroscopically absent, but histologically, embryonic tissues are identified in the mesenchyme with a rudimentary ureter. Aplasia is divided into unilateral and bilateral. Unilateral aplasia does not affect life expectancy. In contrast to hypoplasia, in renal aplasia there is a rudimentary organ rudiment without a pelvis and vascular pedicle [46]. Frequency of occurrence: 1:1000 newborns [24].

Kidney doubling is the division of the kidney into two segments: the main (lower) and additional (upper), while the renal parenchyma is one, its increase in longitudinal size is noted. The frequency of occurrence is 1:150 newborns. Kidney doubling is divided into unilateral and bilateral, complete and incomplete [6]. With complete doubling, both renal pelvises and both ureters open into the bladder with two orifices. The main orifice opens in the corner of the Lieto triangle. The accessory orifice may be ectopic in the bladder neck or extravesical: in girls, distal to the internal urethral opening (into the urethra, genitals, and derivatives of the urogenital sinus), in boys, always above the external urethral sphincter (into the seminal vesicle, seminal tubercle). In incomplete doubling, the two ureters join at different levels: from the renal hilum to the intramural part of the ureter. Doubling of the kidney often leads to the development of vesicoureteral and uretero-ureteral reflux [24, 46].

Multicystic renal dysplasia (non-functioning) is a severe form of renal dysplasia in which the parenchyma is absent and completely replaced by a conglomerate of multiple, tightly adjacent thin-walled cysts of varying sizes that have no blood flow and do not communicate with each other; the renal pelvis and ureter are abnormally formed or absent [1, 2, 3, 29, 40]. The incidence of unilateral multicystic renal dysplasia is 1:3000–5000, and bilateral – 1:10,000 [33,41,43].

Anomalies of kidney size

In Russian literature, kidney dysplasia includes rudimentary and dwarf kidneys. The incidence of kidney dysplasia is 1:4,000 – 1:4,500 live-born newborns [38].

A rudimentary kidney is an organ whose development has stopped at an early stage of the embryonic period.

The dwarf kidney is significantly reduced in size (up to 2–5 cm) and the number of glomeruli in its parenchyma is sharply reduced, while the interstitial fibrous tissue is excessively developed. The number of renal vessels and their caliber are also significantly reduced, the ureter is sometimes obliterated. This form of renal dysplasia is often complicated by nephrogenic arterial hypertension [11].

Renal hypoplasia refers to congenital anomalies of size and is a decrease in the size of the kidney by more than 2 sigmoid deviations from the norm. Unilateral and bilateral renal hypoplasia are distinguished. A hypoplastic kidney has a smooth outer contour, a reduced ultrasound-metric volume of the kidney compared to the age norm, uniformly thinned parenchyma with normal echogenicity, fewer lobes and pyramids, and less than 5 calyces are visualized [12]. Renal hypoplasia is a common cause of parenchymal arterial hypertension, progression of CKD to stage C5 [32].

Anomalies of kidney location

Dystopia (ectopia) of the kidney is characterized by abnormal location of the kidney. The incidence rate is 1:900 live births. According to the location, dystopia is divided into thoracic, lumbar, iliac, pelvic and crossed [38].

Thoracic dystopia of the kidney is often combined with congenital diaphragmatic hernia and, when initially detected, differential diagnosis with mediastinal tumors is required.

Lumbar dystopia of the kidney remains in the lumbar region, but is located lower than usual. It is often increased in length [38].

With iliac dystopia, the kidney is visualized at the entrance to the large pelvis. Lumbar and iliac dystopia in children often manifests itself as abdominal pain syndrome and dysuric phenomena [24].

With pelvic dystopia, the kidney is visualized behind the bladder in men and behind the uterus in women. Pelvic dystopia accounts for 40% of all ectopic kidneys; when initially detected, differential diagnostics with tumors of the pelvic organs is required [38].

In crossed dystopia, one kidney is displaced to the opposite side, so that both kidneys are located on the same side. It is usually asymptomatic. In 50% of cases, crossed dystopia of the kidney is combined with anomalies in the development of the reproductive system (cryptorchidism or absence of the vas deferens in men and vaginal atresia or uterine defects in women) [46].

Anomalies in the relative position of the kidneys include adhesions of the kidneys, which make up 15-20% of all renal anomalies. Symmetrical (horseshoe-shaped, biscuit-shaped) and asymmetric adhesions (S-, I- and L-shaped kidneys) are distinguished [51].

Horseshoe kidney develops when kidneys of the same poles fuse, most often the lower (90%) and very rarely the upper poles. It accounts for 90% of all anomalies of the relative position of the kidneys and 10–15% of all congenital anomalies of the kidneys. The isthmus of the horseshoe kidney can be located in the midline or laterally, which leads to an asymmetric horseshoe kidney, 70% of which remains dominant. The isthmus in 80% of cases consists of renal parenchyma (functioning), in 20% of cases of fibrous cord (non-functioning) and is located anterior to the aorta and inferior vena cava, mainly at the level of the L3–L4 vertebrae. The incidence is 1:400–1:500 newborns, more often in boys [37, 39].

According to the literature, 50% of children with horseshoe kidneys are diagnosed with VUR and recurrent urinary tract infections, 5% with developmental anomalies of the genitals, and 3% with VACTERL association [36, 44].

A horseshoe kidney is a symmetrical fusion of the kidneys along their medial surface in the embryonic period, even before the completion of rotation with a violation of subsequent upward movement and the formation of an atypical bed.

S-shaped kidney is characterized by asymmetric fusion of the kidneys by the upper pole of one kidney with the lower pole of the other, while the kidney resembles the Latin letter "S". The incidence rate is 1:4,000 newborns [54].

L-shaped kidney is an asymmetric fusion of the kidneys, in which the long axes of the kidneys are perpendicular to each other at the level of the spine or sacrum, the cavity system is atypical and does not have a specific characteristic shape. The incidence rate is 1:4,000 newborns.

I-shaped kidney is an asymmetric fusion of the kidneys by the lower pole of one kidney and the upper pole of the other, while the longitudinal axes of the kidneys coincide [46].

II. Congenital anomalies of ureter development

Megaureter (neuromuscular dysplasia of the ureter) is a collective term that includes various types of ureter dilation. Megaureter is formed as a result of dilation of the collecting system of the kidney and ureter in VUR III – V. It occurs 1.5 times more often in women than in men [24].

Hydronephrosis is a persistent, progressive expansion of the renal cystic system, leading to impaired urine outflow. Hydronephrosis with ureteral dilation is considered ureterohydronephrosis. A. V. Papayan and E. N. Stolova (1999) developed a classification of organic and functional obstructions and obstructive nephropathies in childhood [14,18].

Vesicoureteral reflux (VUR) is divided into passive or active retrograde reflux of urine from the bladder into the upper urinary tract, leading to damage to the tissues of the kidney and ureter, constant infection of the urinary system, the development of reflux nephropathy, nephrogenic arterial hypertension, and CKD [15, 17, 20, 42].

VUR is considered as primary and secondary. Primary reflux is understood as an isolated developmental anomaly characterized by the presence of various types of dysplasia of the vesicoureteral junction. Secondary reflux is considered to be its manifestation in other developmental anomalies or disorders of the urinary tract, with dysfunction of the vesicoureteral junction. VUR is divided into passive, occurring at rest, active - during urination and passive-active (mixed) [16, 31, 52].

The degree of VUR I–V is determined according to the international radiological classification of R.L. Lebowitz, H. Olbing, K.V. Parkkulainen et al. (1985) by the degree of urine regurgitation and changes in the ureters and renal cavity systems [10, 19].

VUR in childhood is often characterized by an erased or asymptomatic form [8]. According to I.M. Umalatova and G.M. Letifov (2018), the incidence of VUR is 1–3% of the population [20].

H. Miyakita et al. (2020) provided the results of a meta-analysis of 770 children and reported that the incidence of VUR in the structure of kidney and urinary system diseases is 0.4–1.8%. According to their data, VUR is diagnosed during examination of a patient with frequent UTIs (88%), with dysfunctions of the bladder and intestines (4%), with no clinical manifestations, but with a burdened heredity for VUR (7.4%) and examination of a newborn with suspected hydronephrosis based on the results of antenatal ultrasound (1%) [36].

Ureteral duplication is classified as complete and incomplete. In complete duplication, both ureters go separately to the bladder. In incomplete duplication, the ureters merge with each other, not reaching the bladder at different levels. Ureteral duplication is often unilateral, combined with kidney duplication, with each ureter originating from a separate pelvis [46].

Stenosis of the ureteropelvic junction is a common cause of hydronephrosis diagnosed by ultrasound during pregnancy. In children, in 60% of cases, obstruction is localized on the left, in boys it occurs twice as often as in girls. In 10% of cases, obstruction is bilateral. Due to high intrarenal pressure, stenosis of the ureteropelvic junction leads to hydronephrosis and significant deterioration of kidney function [35].

Stenosis of the ureteral pelvis is a narrowing of the ureter at its border with the renal pelvis, which prevents the outflow of urine from the pelvis; functional and organic stenosis of the ureteral pelvis are distinguished.

Ureteroceles are formed by intravesical hernia-like protrusion of all layers of the intramural section of the ureter. A distinction is made between ectopic (always accompanies kidney doubling and affects its accessory segment) and orthotopic (rarely reaches large sizes and is almost never encountered) ureteroceles. Depending on the level of ectopia of the orifice of the accessory ureter and the degree of its stenosis, three stages of ectopic ureterocele are distinguished:

Stage I is accompanied by ureterohydronephrosis of the upper half of the duplicated kidney; Stage II leads to total ureterohydronephrosis on the affected side; Stage III is accompanied by bilateral ureterohydronephrosis, and sometimes there are prolapses of ectopic ureterocele from the external opening of the urethra in girls.

Ectopia of the ureteral orifice is characterized by an abnormal location of the ureteral orifice in the posterior part of the urethra, vaginal vault, vulva, and rarely in the rectum. The main clinical manifestation is urinary incontinence. The kidney and ureter with an ectopic orifice are subject to frequent infection [46].

III. Congenital anomalies of the urinary bladder

Bladder exstrophy is the absence of the anterior wall of the urinary bladder and the presenting part of the anterior abdominal wall. The pubis is absent, the urinary bladder is turned outward with the posterior wall. The incidence is 1:40,000 newborns, three times more often in boys. Often combined with a split urethra and external genitalia [46].

A bladder diverticulum is a sac-like protrusion of the bladder wall. True and false diverticula are distinguished:

A true diverticulum consists of all layers of the bladder wall.

A false diverticulum is a protrusion of the mucous membrane between the muscle bundles of the detrusor, which occurs when there is difficulty emptying the bladder. A bladder diverticulum is most often located on the posterolateral walls and in the area of the ureteral orifices [46].

Neurogenic bladder dysfunction (NBD) is a variety of disorders of the reservoir and evacuation functions of the bladder, developing as a result of damage to the mechanisms regulating urination of various origins and at different levels (cortical, spinal centers, peripheral innervation). There are two types of neurogenic bladder dysfunction [46]:

Hyperreflexive type - pollakiuria, imperative urges, imperative urinary incontinence, increased urge to urinate, nocturnal enuresis repeatedly during the night, children wake up after wetting themselves; pathological positions to relieve imperative urges (squeezing the head of the penis with hands, bringing the thighs together). Urinary incontinence occurs as a result of a sharp increase in intra-abdominal pressure (when coughing, laughing, physical exertion).

Hyporeflexive type - rare urination, weakening or absence of the urge to urinate, impaired urine stream, large volume of urine per urination, possible urinary incontinence of the ischuria paradoxa type (paradoxical urinary incontinence) - urine is excreted drop by drop.

IV. Congenital anomalies of the urethra

The posterior urethral valve is a hyperplastic fold of the mucous membrane located in the posterior urethra. The incidence is 9.34 per 100,000 newborn boys [21].

Depending on the location, there are three types of posterior urethral valves:

Type I below the seminal tubercle, often cup-shaped;

Type II from the seminal tubercle to the neck of the bladder, often multiple and funnel-shaped;

Type III transverse diaphragm above or below the seminal tubercle.

Posterior urethral valves lead to infravesicular obstruction in boys. The posterior urethral valve is considered one of the causes of secondary diabetes insipidus in children.

In children with a posterior urethral valve, in the vast majority of cases, a combination with grade III–V vesicular reflux and hydronephrosis is diagnosed [21].

Urethral stenosis is a narrowing of the urethral lumen with impaired urine outflow. Frequency of occurrence Urethral stenosis is divided into stenosis of the prostatic part (more common in boys) and distal part (more common in girls) of the urethra. Clinical manifestations: urinary incontinence, frequent urination, dribbling of urine [24].

Obstruction of the kidneys and urinary tract is a violation of the outflow of urine due to various causes. In foreign literature, the term urinary tract obstruction is used and the level of obstruction is indicated (pyelourethral, ureteral, vesicoureteral, infravesical) [45, 48].

Violation of urodynamics in obstruction, present for a long time and disrupting the outflow of urine from the renal pelvis, leads to damage to the renal parenchyma and the development of obstructive nephropathy. The main clinical manifestations of obstructive nephropathy are urinary tract infections and tubulointerstitial nephritis [5].

Obstructive uropathy (obstructive urophathy) is characterized by structural and functional changes in the urinary tract due to impaired urine outflow due to obstruction. Pyelectasis is often diagnosed, caused in 70% of cases by the immaturity of the fetal urinary system. In children with CKD C5, congenital obstructive uropathy is diagnosed in more than 40% of cases [7].

Obstructive uropathy can be acute (urolithiasis) and chronic (CAKUT). Acute obstructive uropathy is mostly reversible. According to localization, obstruction of the upper urinary tract (obstruction of the renal pelvis, upper ureters and ureteropelvic junction) and lower urinary tract (obstruction of the lower ureters, bladder and urethra) are distinguished. In case of lower urinary tract obstruction, the process is most often bilateral, whereas in case of upper urinary tract obstruction, the pathological process is most often unilateral. Lower urinary tract obstruction is often associated with VUR [25].

Congenital obstructive uropathy, according to O. L. Chugunova and M. V. Shumikhina (2021), leads to the progression of CKD to stage C5 in 41% of cases [23].

In the structure of kidney diseases in children, CAKUT is diagnosed in 5.4 - 32.5% of cases. The frequency of congenital anomalies of the kidneys and urinary tract, according to the literature, averages 1.6 per 1000 live births. In 30% of cases, CAKUT is combined with congenital anomalies in the development of other organs and systems, most often with malformations of the musculoskeletal system, digestive system (for example, anal atresia), heart defects and anomalies of the central nervous system (for example, spinabifida). CAKUT is one of the causes of CKD C5 in children receiving renal replacement therapy [4].

M. Richter-Rodier et al. (2012) and C. Policiano et al. (2015) showed that routine ultrasound examination in the second trimester of pregnancy can make a preliminary diagnosis of CAKUT in the fetus in 73–88% of cases. Maternal factors associated with the development of CAKUT in the child include diabetes, older age, white race, various kidney diseases, and recurrent genitourinary infections [34]. L. Jadresić et al. (2021) conducted a meta-analysis and showed that maternal obesity before pregnancy is a risk factor for CAKUT [47]. According to Chien-Ning Hsu and You-Lin Tain (2021), folate and vitamin A deficiency in a pregnant woman, as well as a low-carbohydrate diet during pregnancy, can lead to the formation of CAKUT [28].

Yu. Yu. Chebotareva et al. (2021) proved that adolescent girls with CAKUT combined with developmental anomalies of the uterus and vagina require in-depth examination for a timely assessment of their reproductive health [22].

H. Miyakita et al. (2020) indicate that VUR in the structure of CAKUT occurs in 46% of cases. In children with an adverse heredity (VUR in the mother in the anamnesis), VUR is diagnosed in the first year of life in 40% of cases [36].

**Conclusion.** Currently, various types of congenital anomalies of the kidneys and urinary tract in newborns have been identified, but they do not fully reflect the clinical features of pathologies. It is necessary to conduct morphological studies in this area for a full justification and correct verification of the diagnosis.

**References.**


1. Andreeva, E. F. Multicystic dysplasia of the kidney in children: terminology, classification, diagnosis and treatment. Textbook for pediatricians, nephrologists, clinical residents / E. F. Andreeva, N. D. Savenkova; edited by prof. N. D. Savenkova. - St. Petersburg: SPbSPMU, 2023. - 31 p.
2. Andreeva, E. F. Renal oligohydramnios and Potter sequence in cystic kidney diseases / E. F. Andreeva, N. D. Savenkova // Russian Bulletin of Perinatology and Pediatrics. - 2021. - Vol. 66. - № 1. - P. 47 - 51.
3. Andreeva, E. F. Epidemiology, terminology and classification, diagnosis and treatment of hereditary cystic kidney diseases in children / E. F. Andreeva, N. D. Savenkova; edited by N. D. Savenkova. - St. Petersburg: SPbSPMU, 2021. - 32 p.
4. Bayko, S. V. Chronic kidney disease in children: definition, classification and diagnosis / S. V. Bayko // Nephrology and dialysis. - 2020. - Vol. 22. - № 1. - P. 53-70.

5. Readiness for transition to adult healthcare in adolescents with chronic kidney disease: a descriptive review of international literature / E. N. Kulakova, T. L. Nastausheva, A. P. Savchenko, T. G. Zvyagina // Issues of Practical Pediatrics. - 2021. - Vol. 16. - № 3. - P. 62-71.
6. Dlin, V. V. Personalized therapy in pediatric nephrology; problems and prospects / V. V. Dlin, S. L. Morozov // Russian Bulletin of Perinatology and Pediatrics. - 2021. - Vol. 66. - № 2. - P. 6-12.
7. Dumova, S. V. Obstructive uropathy, vesicoureteral reflux, reflux nephropathy // Pediatric nephrology. Edited by: Shumilova P. V., Petrosyan E. K., Chugunova O. L. / S. V. Dumova, S. S. Paunova. – Moscow: MED press-inform, 2021. – P. 449 – 473.
8. Zaikova, N. M. Diagnosis of reflux nephropathy in young children / N. M. Zaikova, V. V. Dlin // Pediatrician practice. – 2017. – № 2. – pp. 54 – 59.
9. Zaykova, N. M. Patterns of formation and prediction of the course of reflux nephropathy in children in justification of early renoprotective therapy (clinical and experimental study): author's abstract. dis. doc. med. sciences: 14.01.08 / Zaykova Natalya Mikhailovna - M., 2020. - 48 p.
10. Zorin, I. V. Structural and functional state of the kidneys in children with reflux nephropathy at different stages of tubulointerstitial damage / I. V. Zorin, A. A. Vyalkova // Attending physician. - 2016. - № 9. - Access mode: https://www.lvrach.ru/2016/09/15436548.
11. Ignatova, M.S. Hereditary diseases of the urinary system in children: A guide for doctors / M. S. Ignatova, V. V. Dlin, P. V. Novikov. - M .: Overlay, 2014. - 348 p.
12. Kapustin, S. V. Ultrasound examination in urology and nephrology / S. V. Kapustin, R. Owen, S. I. Pimanov. - M .: Smart doctor, 2017. -176 p.
13. Lysova, E. V. CAKUT syndrome in the etiological structure of chronic kidney disease in children and adolescents / E. V. Lysova, N. D. Savenkova // Nephrology. - 2017. - Vol. 21. - № 3. - P. 69 - 74.
14. Markers of collagen formation and sclerosis in the diagnosis of reflux nephropathy progression in children / N. M. Zaykova, V. V. Dlin, L. A. Sinitsyna, A. V. Eremeeva [et al.] // Nephrology. - 2018. - Vol. 22. - № 3. - P. 33 - 42.
15. Prevention of the development of chronic kidney disease in children with vesicoureteral reflux / I. V. Zorin, A. A. Vyalkova, E. V. Gunkova, L. V. Kutsenko // Attending physician. - 2019. - № 1. - P. 40 - 42.
16. Vesicoureteral reflux and reflux nephropathy // Urinary tract infections in children. Edited by: Dlin V. V., Osmanov I. M., Chugunova O. L. / S. V. Dumova, O. L. Chugunova, V. V. Dlin, N. M. Zaykova. T. V. Lepayeva // M.: Overley, 2017 - P. 244 - 268.
17. Vesicoureteral reflux as a manifestation of CAKUT syndrome in children: the problem of late diagnosis / T. P. Makarova, N. V. Samoilova, Yu. S. Melnikova, L. V. Poladova [et al.] // Nephrology. - 2021. - Vol. 25. - № 3. - P. 84 - 90.
18. Reflux nephropathy in children: pathogenesis and prognosis. Part 1 / O. L. Morozova, D. Yu. Lakomova, N. B. Zakharova, L. D. Maltseva [et al.] / Urology. - 2021. - № 3. - P. 150 - 154.
19. The role of angiotensin-converting enzyme gene polymorphism in children with vesicoureteral reflux in the progression of reflux nephropathy / N. M. Zaykova, D. Dan-Dumitru, V. Sacare, N. Ushurelu [et al.] // Russian Bulletin of Perinatology and Pediatrics. - 2015. - Vol. 60. - № 4. - P. 203 - 204.
20. Umalatova, M. I. Vesicoureteral reflux in children: clinical presentation, diagnostics and approaches to therapy / M. I. Umalatova, G. M. Letifov, B. M. Makhachev // Modern

science: current problems of theory and practice. Series: Natural and technical sciences. – 2018. – T. 6. – P. 230 – 233.

21. Surgical treatment of children with posterior urethral valve / I. B. Osipov, D. A. Lebedev, S. A. Sarychev, A. I. Osipov [et al.] // Issues of practical pediatrics. - 2021. - Vol. 16. - № 4. - P. 16 - 27.
22. Chebotareva, Yu. Yu. Combined urogenital developmental anomalies and the formation of reproductive health in girls / Yu. Yu. Chebotareva, G. M. Letifov, M. A. Rodina // Nephrology. - 2021. - Vol. 25.-№ 6.-P. 99-105.
23. Chugunova, O. L. Congenital and hereditary kidney diseases in children // Children's nephrology. Edited by: Shumilova P. V., Petrosyan E. K., Chugunova O. L. / O. L. Chugunova, A. I. Safina, M. V. Shumikhina. – M: MEDpress-inform, 2021. – P. 171 – 190.
24. Shumilov, P. V. Pediatric nephrology / Edited by P. V. Shumilov, E. K. Petrosyan, O. L. Chugunova. – MEDpress-inform – M.: 2021. – 616 p.
25. Yuryeva, E. A. Guide to clinical and laboratory diagnostics of kidney diseases in children. / E. A. Yuryeva, V. V. Dlin. Edited by E. A. Yuryeva, V. V. Dlin. - M: Overlay, 2020. - 244 p.
26. A Spectrum of Congenital Anomalies of the Kidney and Urinary Tract (CAKUT) - Diagnostic Utility of Perinatal Autopsy / N. Ganesan, U. Gurusamy, Ch. Venkataswamy, K. Sankar // Indian Journal of Pediatrics. – 2023. – V. 90. – №. 2. – P. 139 – 145.
27. Chevalier, R. L. CAKUT: A Pediatric and Evolutionary Perspective on the Leading Cause of CKD in Childhood / R. L. Chevalier // Pediatric Reports. – 2023. – V. 15. – №. 1. – P. 143 – 153.
28. Chien-Ning, H. The First Thousand Days: Kidney Health and Beyond / H. Chien Ning, T. You-Lin // Healthcare. – 2021. – V. 9. – №. 10. – P. 1332.
29. Classifying and evaluating fetuses with multicystic dysplastic kidney in etiologic studies / M. Cai, C. Guo, X. Wang, M. Lin [et al.] // Exp Biol Med (Maywood). – 2023. – V. 248. – №. 10. – P. 858 – 865.
30. COL4A1 mutations as a potential novel cause of autosomal dominant CAKUT in humans / T. M. Kitzler, R. Schneider, S. Kohl, C. M. Kolvenbach [et al.] // Human Genetics. – 2019. – V. 138. – №. 10. – P. 1105 – 1115
31. Dimercaptosuccinic acid scan challenges in childhood urinary tract infection, vesicoureteral reflux and renal scarring investigation and management / S. Roupakias, X. Sinopidis, G. Tsikopoulos, I. Spyridakis [et al.] // Minerva urologica e nefrologica. – 2017. – V. 69. – № 2. – P. 144 – 152.
32. Gabriele, M. M. Management of Hypertension in CAKUT: Protective Factor for CKD / M. M. Gabriele, P. C. K. Nogueira // Frontiers in Pediatrics. – 2019. – V. 7. – №. 1. – P. 222.
33. Gene Reviews / M. P. Adam, J. Feldman, G. M. Mirzaa, R. A. Pagon [et al.] // Seattle (WA): University of Washington, Seattle. – 2020. – Bookshelf ID: NBK559434.
34. Genetic, environmental, and epigenetic factors involved in CAKUT / N. Nicolaou, K. Renkema, E. Bongers, R. H. Giles [et al] // Nature Reviews Nephrology. – 2015. – V. 11. – P. 720 – 731.
35. Groothoff, J.W. Long-Term Outcome of Kidney Failure in Children. In: Pediatric Kidney Disease. Eds.: F. Schaefer, L. A. Greenbaum / J. W Groothoff // Springer Cham. – 2023. – P. 1937 – 1962.
36. Guidelines for the Medical Management of Pediatric Vesicoureteral Reflux / H. Miyakita, Y. Hayashi, T. Mitsui, M. Okawada [et al.] // International journal of urology. – 2020. – V. 27. – № 6. – P. 480 – 490.

37. Horseshoe kidney splitting technique for transplantation / P. D. Pablos-Rodríguez, J. F. Suárez, L. R. Canals, P. Sanz-Serra [et al.] // Urology case reports. – 2021. – V. 37. – P. 1064.

38. Kher, K. K. Clinical Pediatric Nephrology. Third Edition / Eds.: K. K. Kher, H. W. Schnaper, L. A. Greenbaum // CRC Press Taylor & Francis Group. – 2017. – P. 1093.

39. Kirkpatrick, J. J. Horseshoe Kidney / J. J. Kirkpatrick, S. W. Leslie // Treasure Island (FL). – Stat Pearls Publishing, 2023. –Bookshelf ID: NBK431105.

40. Kopač, M. Associated Anomalies and Complications of Multicystic Dysplastic Kidney / M. Kopač, R. Kordič // Pediatric reports. – 2022. – V. 14. – №. 3. – P. 375 – 379.

41. Mashat, S. D. Outcome of Multi-Cystic Dysplastic Kidneys in Children / S. D. Mashat, S. M. El-Desoky, J. Abdulaziz Kari // Pediatr. – 2015. – Режим доступа: https://pubmed.ncbi.nlm.nih.gov/26495094/.

42. Mattoo, T.K. Primary Vesicoureteral Reflux and Renal Scarring / T.K. Mattoo, D. Mohammad // Pediatric clinics of North America. – 2022. – V. 69. – №. 6. – P. 1115 – 1129.

43. Multicystic dysplastic kidney disease: an in-utero diagnosis / R. Chaubal, S. C. Pokhriyal, A. Deshmukh, U. Gupta [et al.] // Cureus [Электронный ресурс]. – 2023. – Режим доступа: https://pubmed.ncbi.nlm.nih.gov/37214047/.

44. Nonfamilial VACTERL-H Syndrome in a Dizygotic Twin: Prenatal Ultrasound and Postnatal 3D CT Findings / S. Y. Hong, S. J. Kim, Mi-Hye Park, K. A. Lee // Medicina. – 2023. – V. 59. – №. 8. – P. 1387.

45. Obstructive Uropathies // Pediatric Kidney Disease. Eds.: F. Schaefer, L. A. Greenbaum / B. D. Chiodini, K. Ismaili, D. A. Diamond, M. P. Kurtz // Springer Cham. – 2023. – P. 1211 – 1220.

46. Pediatric Nephrology. Eighth Edition // Eds.: F. Emma, S. L. Goldstein, A. Bagga, C. M. Bates, R. Shroff // Springer Nature Switzerland. – AG.: 2022. – 2134 p.

47. Pre-pregnancy obesity and risk of congenital abnormalities of the kidney and urinary tract (CAKUT)-systematic review, meta-analysis and ecological study. / L. Jadresić, H. Au, C. Woodhouse, D. Nitsch // Pediatric Nephrology. – 2021 – V. 36. – №. 1. – P. 119 – 132.

48. Reed, J. N. Reflux and Obstructive Nephropathy // Tubulointerstitial Nephritis. Eds.: M. G. Atta, M. A. Perazella / J. N. Reed, O. Nicoara, B. A. McMahon // Springer Cham. – 2022. – p. 199 – 205.

49. Rosenblum, N. D. Disorders of Kidney Formation // Pediatric Kidney Disease. Eds.: F. Schaefer, L. A. Greenbaum / N. D. Rosenblum, I. R. Gupta // Springer Cham. – 2023. – P. 257 – 285.

50. Severe congenital anomalies of the kidney and urinary tract: epidemiology can inform ethical decision-making / P. Danziger, D. R. Berman, K. Luckritz, K. Arbour [et al.] // Journal of Perinatology. – 2016. – V 36. – P. 954 – 959.

51. Transplanting a horseshoe kidney: A case report and review of surgical strategies / F. Vasconcelos Ordones, H. Yamamoto, P. Ivo Pajolli, R. Ribeiro Meduna [et al.] // Trauma and reconstruction. – 2018. – V. 21. – P. 119 – 121.

52. Tullus, K. Vesicoureteric reflux in children / K. Tullus // Lancet. – 2015. – V. 385 – № 9965. – P. 371 – 379

53. Uncovering risk factors for kidney injury in children with a solitary functioning kidney / S. G. Woud, N. Roeleveld, R. Westland, K. Y. Renkema [et al.] // Kidney International. – 2023. – V. 103. – №. 1. – P. 156 – 165.

54. 54. Winnicki, E. Congenital Anomalies of the Kidney and Urinary Tract in Adolescents // Adolescents with chronic kidney disease. From Diagnosis to End Stage Disease. Eds.: M. N. Haddad, E. Winnicki, S. Nguyen. – Springer Nature Switzerland. – AG.: 2019. – P. 81- 92.