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LATE DIAGNOSIS OF CHRONIC KIDNEY DISEASE IN A CHILD WITH A HYPOREFLEXIVE BLADDER: A CASE REPORT AND LITERATURE REVIEW

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Abstract.

Chronic kidney disease (CKD) represents a major global health problem because of its high prevalence, significant mortality, and the substantial economic burden associated with renal replacement therapy. Most pediatric cases of CKD are diagnosed at advanced stages, when the condition significantly reduces quality of life and adversely affects disease severity and prognosis. Children with CKD stage 3-5 have a 4.5-fold higher risk of adverse outcomes than those diagnosed at earlier stages. Early identification and mitigation of risk factors therefore remain central to the primary prevention of CKD. Multiple causes of CKD in children have been identified, including bladder hypoactivity (hypotonia, hypo–/areflexia). The present study addresses this particular condition.

Aim. *To analyze a clinical case of late diagnosis of chronic kidney disease in a child with hypotonic neurogenic bladder dysfunction, identify risk factors for disease progression, describe potential complications, and emphasize the importance of early diagnosis.*

Materials and Methods. *A clinical case of stage IV chronic kidney disease in a girl with hypotonic neurogenic bladder dysfunction was analyzed together with a review of relevant literature. The study was performed in accordance with the principles of the Declaration of Helsinki. The study protocol was approved by the Local Ethics Committee; informed consent was obtained from the patient and her mother. A comprehensive bibliographic search was conducted in leading electronic databases: PubMed/MEDLINE, Scopus, and Web of Science.*

Results. *A clinical case of late diagnosis of CKD stage 4 was analyzed in a 16-year–old girl with hypotonic neurogenic bladder and bilateral grade 3-4 ureterohydronephrosis. Diagnostic evaluation was initiated after the patient presentation with menstrual irregularities and severe anemia. Further examination demonstrated marked impairment of renal nitrogen–excretory function: serum creatinine 332.5 μmol/L and the glomerular filtration rate (GFR) was 16.4 mL/min/1.73 m². Ultrasonography revealed bilateral renal enlargement, increased parenchymal echogenicity, loss of corticomedullary differentiation, pronounced dilation of the pelvicalyceal system and ureters, and severe urodynamic impairment: bladder volume before voiding was 1050 mL, with a postvoid residual volume of 420 mL.*

This clinical case demonstrates that hyporeflexive bladder dysfunction may remain asymptomatic and undiagnosed for a prolonged period, leading to recurrent infections, bilateral ureterohydronephrosis, and subsequent CKD progression. Despite the absence of arterial hypertension and growth retardation, the patient developed systemic complications of CKD, including severe anemia, mineral metabolism disorders, acid–base imbalance, and electrolyte disturbances. Factors contributing to delayed diagnosis and disease progression to CKD stage 4 are discussed together with a review of relevant literature.

Conclusions. *Earlier diagnosis in this case would have required regular monitoring of urinalysis with attention to urine specific gravity, follow–up renal ultrasonography after resolution of the acute inflammatory process, pre– and postvoid bladder ultrasonography, and voiding cystourethrography. Continuity of care between inpatient and outpatient settings together with multidisciplinary collaboration among a pediatric nephrologist, pediatric urologist, pediatrician, and general practitioner is essential for the management of patients with bladder dysfunction.*

Keywords: *Children; Chronic Kidney Disease; Kidney Damage; Biomarkers; Glomerular Filtration Rate; Hypotonic Neurogenic Bladder Dysfunction.*

Introduction

Chronic kidney disease (CKD) in the pediatric population is recorded less frequently than in adults: the worldwide prevalence is 15-74.7 cases per million children and 100,000-120,000 cases per million adults [1, 2]. Significant intercountry differences in incidence rates are associated with healthcare resources, availability of registries, access to diagnostic and treatment modalities, etiology, sociodemographic index, and other factors [3, 4, 5]. In European countries, the prevalence of CKD stage 3-5 is 11-12 per million age–matched children; stages 4-5 is 8 per million age–matched children [6]. In low– and middle–income countries, the number of CKD cases may be underestimated because of limited diagnostic infrastructure and the lack of registries [4].

Most studies address advanced stages of CKD, whereas data on early–stage CKD in children are reported considerably less frequently. CKD was diagnosed at an early stage during the initial visit in only 5% of children. Children with CKD stage 3-5 have a 4.5-fold higher risk of adverse outcomes than those diagnosed at earlier stages. Progression of CKD to end-stage renal disease (ESRD) in children with primary glomerular disorders occurs more rapidly than in children with isolated congenital anomalies of the kidney and urinary tract (CAKUT) [3, 7]. About 40-50% of children with glomerular diseases experience a greater than 50% decline in glomerular filtration rate (GFR) and progression to ESRD within 3.8-5 years [8, 9, 10]. Children with CAKUT by contrast more frequently develop acquired disorders, such as urinary tract infections (UTIs) and obstructions, which accelerate CKD progression; the prognosis therefore depends on the presence of complications, and the median time to ESRD is ~8.3 years [10, 11]. According to published data, patients

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with glomerulopathies initiated dialysis more often than children with CAKUT. The number of children receiving renal replacement therapy increases annually (5.5-6.6 per million age-matched children), as survival among children undergoing dialysis and after transplantation continues to improve [12]. CKD progression in patients with CAKUT is usually slower than in glomerular diseases; patients with CAKUT remain under follow-up until later disease stages [9, 13, 14]. According to the ESPN/ERA–EDTA registry, the five-year survival rate for the overall population of children with ESRD receiving renal replacement therapy in Europe was approximately 94%. Mortality was higher in the following groups of children: those younger than 5 years, those with glomerular diseases, and those with complicated CAKUT [12]. Understanding disease progression through clinical studies provide information that helps anticipate disease risk and identify risk factors for progression [9]. A wide range of causes of CKD in children has now been identified, including urinary bladder hypoactivity (hypo-/atonia, hypo-/areflexia) [15]. The present report focuses specifically on this condition.

Study objective

To analyze a clinical case of late diagnosis of chronic kidney disease in a child with hypotonic neurogenic bladder dysfunction, identify risk factors for disease progression and possible complications, and emphasize the importance of early diagnosis.

Materials and methods

An analysis of the diagnosis of CKD stage 4 in a girl with hypotonic neurogenic bladder dysfunction was conducted. A comprehensive literature search was conducted across PubMed/MEDLINE, Scopus and Web of Science.

The study was performed in accordance with the principles of the Declaration of Helsinki; the study protocol was approved by the Local Ethics Committee; informed consent for participation in the study and publication of clinical data was obtained from the patient and her mother.

Results

A 16-year-old patient was hospitalized in the Department of Urology and Reconstructive Surgery of the Municipal Non–Commercial Enterprise «Regional Medical Center for Family Health» of the DRC (RMCFH). At the time of transfer from the local hospital to the RMCFH, the patient's condition was assessed as moderate because of manifestations of generalized anemic syndrome and CKD. Physical examination revealed marked pallor of the skin and mucous membranes and periorbital darkening. Hemodynamic status was stable, blood pressure (BP) was 116/64 mm Hg, edema was absent, Pasternatsky's sign was negative bilaterally. Urination was managed using a Foley catheter, and urine output was adequate.

The medical history indicates that the patient had recently experienced menstrual irregularities for which she consulted a gynecologist. Complete blood count (CBC) revealed severe anemia (hemoglobin 42 g/L, erythrocyte count 1.8×10^{12}/L), which prompted hospitalization in the intensive care unit at the local hospital. Two erythrocyte transfusions were performed, and further examination and treatment were initiated. Biochemical blood analysis revealed impaired renal nitrogen-excretory function: creatinine 332.5 μmol/L and GFR 16.4 mL/min/1.73 m².

Ultrasonographic findings included bilateral renal enlargement, increased parenchymal echogenicity, impaired corticomedullary differentiation, marked dilation of the pelvicalyceal system and ureters, and severe urodynamic disturbances: bladder volume before voiding was 1050 cm³, whereas postvoid residual urine volume was 420 cm³, which necessitated placement of a Foley catheter.

The identified abnormalities allowed establishment of a preliminary diagnosis already at the initial stage of evaluation: CKD stage 4, hypotonic neurogenic bladder dysfunction, bilateral ureterohydronephrosis, and severe anemia.

For further examination and treatment, the patient was referred to the RMCFH, a tertiary care hospital.

The medical history indicates that the girl was born with a body weight of 4000 g from the first pregnancy, which was complicated by maternal anemia and dysmetabolic nephropathy. The child is an internally displaced person and lived in Kramatorsk until 2022.

At the age of 9 months, the girl developed lacunar tonsillitis of staphylococcal etiology accompanied by allergic dermatitis and dysbiosis. During inpatient treatment, antibacterial therapy was administered. At that time, slight abnormalities were first detected in the general urinalysis (GUA), namely mild proteinuria (0.049 g/L), whereas previous GUA findings had been within normal limits. Other diseases in the patient's history included acute respiratory viral infections occurring 1-2 times per year and acute community-acquired right-sided pneumonia at the age of 10 years. The girl was vaccinated according to age.

Only three episodes of UTI were documented throughout the patient's life: at the ages of 2, 9, and 14 years. As this article aims to analyze the possible causes of late CKD diagnosis, particular attention will be paid to the episodes of UTI in this patient.

The first episode of UTI occurred at the age of 2 years and 2 months, when the child was diagnosed with UTI and pharyngomycosis. The clinical presentation initially corresponded to cystitis and subsequently progressed to acute pyelonephritis; GUA revealed leukocyturia (70-80 per hpf) and proteinuria (0.165 g/L). The girl was hospitalized and received sequential antibiotic therapy, uroantiseptic therapy, and phytotherapy. CBC demonstrated leukocytosis (16.2×10^9/L) and elevated erythrocyte sedimentation rate (ESR). Ultrasonography of the kidneys and bladder revealed no abnormalities at that time; however, a detailed description was absent from the discharge summary. In the Zimnitsky test, only two portions were obtained: first portion – 650 mL, specific gravity 1004; second portion – 195 mL, specific gravity 1007.

The second episode was recorded at the age of 9 years, when the girl was diagnosed with acute pyelonephritis and acute vulvovaginitis. Complaints included fever and abdominal pain. Urinalysis revealed massive leukocyturia (entire HPF) and slight proteinuria (0.99 g/L). In the Zimnitsky test, specific gravity values again indicated hyposthenuria (1005-1007), and the ratio of daytime

and nighttime urine volume was 790 mL to 610 mL. Ultrasonography of the kidneys revealed thickening and stratification of the left renal pelvic wall with preserved corticomedullary differentiation. GUA before discharge showed specific gravity of 1010, whereas the remaining parameters were within normal limits. After inpatient treatment, recommendations regarding outpatient follow-up ultrasonography of the kidneys and GUA were not followed.

The third episode of UTI occurred at 14 years, probably in the form of acute cystitis, which was treated remotely by the family physician by telephone. The patient presented with dysuria, for which an antibacterial agent of the nitrofuran group was prescribed. No examination was conducted either during or after this episode of UTI.

The child's outpatient record contained CBCs performed at the ages of 7 and 10 years; all parameters were within the age-specific reference range.

Upon admission to the RMCFH, detailed examination continued. CBC revealed moderate hypochromic microcytic anemia (Hb 85 g/L, erythrocytes 3.56×10^{12}/L). CBC dynamics are presented in Table 1.

Hyposthenuria was recorded in all GUAs; microhematuria and a moderate increase in squamous epithelial cells were also detected (Table 2).

Biochemical blood analysis demonstrated a significant impairment of renal nitrogen-excretory function: creatinine – 342.41 μmol/L and urea – 20.8 mmol/L. Estimated GFR was 15.1 mL/min/1.73 m², corresponding to CKD stage 4 (Table 3).

Table 1

Dynamics of complete blood count indicators in the child

Indicators/date	24.09.25	8.10.25
Hemoglobin, g/L	85	102
Erythrocytes, 10^{12}/L	3.56	4.06
Color index	0.71	0.75
Hematocrit, %	27.55	32.32
Platelets, 10^9/L	209	306
Leukocytes, 10^9/L	5.3	6.0
Segmented neutrophils, %	51.8	53.2
Lymphocytes, %	36.0	32.9
Basophils, %	0.4	0.5
Eosinophils, %	6.1	3.9
Monocytes, %	5.7	9.5
MCV, fl	77.38	79.66
MCHC, g/dL	31.03	31.51
MCH, pg	24.01	25.1

Table 2

Dynamics of general urinalysis indicators in the child

Indicators/date	25.09.25	29.09.25
Quantity (mL)	50.0	60.0
Color	Yellow	Light–yellow
Transparency	Transparent	Transparent
Specific gravity (relative density), g/L	1.004	1.005
Reaction (pH)	6.0	7.5
Protein (g/L)	Absent	0.071
Glucose (mmol/L)	Absent	Absent
Ketone bodies	Absent	Absent
Erythrocytes (unchanged)	3-4 per hpf	Absent
Leukocytes	2-3 per hpf	18-25 per hpf
Squamous epithelial cells	7-8 per hpf	1-4-6 per hpf
Casts	Not detected	Not detected
Bacteria	Not detected	Not detected
Mucus	Not detected	Not detected

Table 3

Dynamics of biochemical blood indicators and GFR of the child

Indicator/Date	24.09.25	26.09.25	28.09.25	10.11.25
Creatinine, μmol/l	342.41	273.67	398.12	261.15
Urea, mmol/L	20.8	16.2	24.1	13.0
Urea nitrogen, mmol/L	9.9	10.0	–	6.9
Potassium, mmol/L	5.4	4.5	5.7	4.6
Sodium, mmol/L	144.0	145.1	148.2	139.1
Chlorine, mmol/L	113	112	109	106
GFR, ml/min/1.73 m²	15.10	20.05	13.03	29.08

Electrolyte imbalance was noted, including hyperkalemia, hypernatremia, and hyperchloremia. The parathyroid hormone level was significantly elevated (501 pmol/L), indicating secondary hyperparathyroidism. Acid-base balance analysis revealed metabolic acidosis upon admission (BE – 9.5 mmol/L).

Instrumental examinations (ultrasonography and computed tomography (CT)) revealed signs of chronic cystitis, bilateral bladder-dependent ureterohydronephrosis, absence of corticomedullary differentiation, bladder wall thickening up to 5 mm, dilation of the calyces up to 7 mm, pelvic wall thickening up to 2.5 mm, thinning of the renal cortex to 4-5 mm, multiple hypodense areas up to 10 mm within the parenchymal, and enlargement of retroperitoneal lymph nodes up to 16 mm. Dynamic kidney ultrasonography findings are presented in Table 4.

Table 4

Dynamics of ultrasound indicators in the child

Indicators/date	15.09.25	23.09.25
Right kidney (size, mm)	131 × 74 × 60	108 × 42 × 57
Left kidney (size, mm)	126 × 86 × 60	102 × 68 × 52
Parenchyma (mm)	Right: 3-10, left: 3-9	Right: 7-17, left: 5-10
Parenchymal echogenicity	Increased	Increased
Cortico–medullary differentiation	Impaired	Absent
Pyelocaliceal system (PCS) – expansion, mm	Right pelvis 52 x 44 mm, calyces 26 x 40 mm. Left pelvis 55 × 50 mm, calyces 26 mm	Right pelvis 27 x 35 mm, calyces 18 mm. Left pelvis 49 x 39 mm, calyces 19 x 22 mm
PCS – contour	Uneven, thickened	Uneven, thickened
Ureter (mm)	Right: 13, left: 12	Right: 7-8, left: 10
Bladder volume (cm³)	1050	370
Bladder wall (mm)	4 mm	5 mm
Echogenicity of bladder contents	Thick fine–dispersed suspension	Reduced
Residual urine volume (cm³)	420	Absent

Magnetic resonance imaging (MRI) of the lumbar spine revealed signs of lumbar osteochondrosis (Pfirrmann II), L3–L4 disc protrusion with compression of the right lateral nerve root and moderate foraminal stenosis (Kang 2), and L4–L5 disc prolapse with right-sided predominance accompanied by moderate compression of the dural sac and right lateral nerve root. The identified spinal abnormalities however did not confirm an association with the patient's bladder hyporeflexia.

Cystoscopic examination revealed hyperemia and enhanced vascular pattern of the entire bladder mucosa, vascular injection, irregularity and trabecularity of the bladder wall, and isolated pseudodiverticula. In the area of the bladder neck and Lieutaud's triangle, isolated petechiae and white deposits were observed. Both ureteral orifices were normally positioned, closed, and oval in shape. These findings allowed confirmation of the diagnosis of neurogenic bladder, chronic cystitis, and urinary bladder leukoplakia. Metaplastic changes (leukoplakia) of the bladder mucosa are shown in Figure 1, whereas multiple diverticular bladder changes are presented in Figure 2.

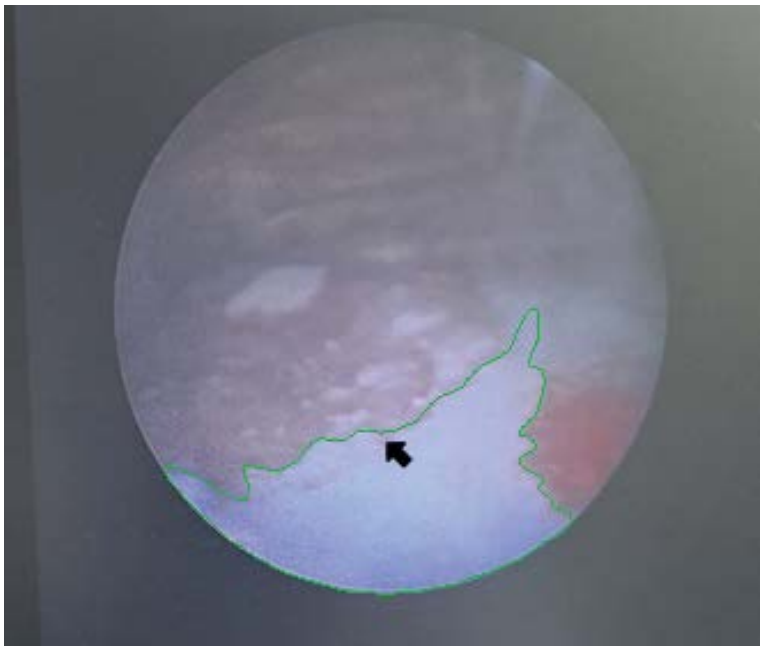

Fig. 1. Cystoscopy 1

Note: the arrow indicates leukoplakia.

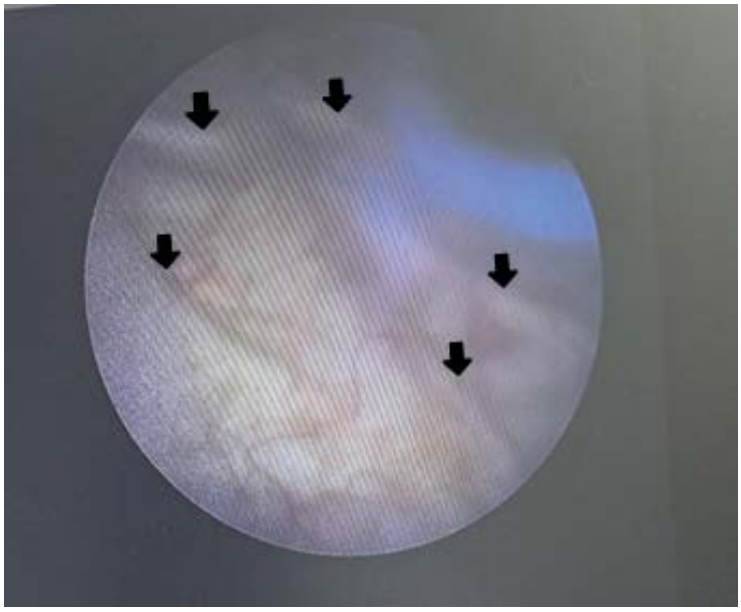

Fig. 2. Cystoscopy 2

Note: the arrow indicates trabecularity.

Voiding cystography was uninformative and did not allow either confirmation or exclusion of vesicoureteral reflux. Spontaneous voiding did not occur either during the first filling of the urinary bladder up to 370 mL or during the second filling up to 550 mL of contrast medium.

Dopplerography of the renal vessels revealed decreased blood flow velocity and increased resistance indices (Table 5).

At the RMCFH, based on the medical history, objective examination, laboratory findings, and instrumental studies, a final diagnosis was established: Chronic kidney disease, stage 4. Hypotonic neurogenic bladder dysfunction. Chronic cystitis. Bilateral grade 3-4 ureterohydronephrosis. Chronic pyelonephritis. Severe anemia. Dysmenorrhea.

Table 5

Dopplerography Indicators of the Child's Renal Vessels

Renal Arteries Level	Right Kidney		Right Kidney	
	Vps, cm/s	RI	Vps, cm/s	RI
Ostia (Main)	50	0.72	47	0.75
Segmental	34	0.72	30	0.73
Interlobar	18	0.72	17	0.72
Main (Trunk)	Not visualized		Not visualized	

Treatment strategy included bladder catheterization using a Foley catheter for normalization of urodynamics for 2 weeks. The next stage involved percutaneous cystostomy. The patient was subsequently trained in intermittent bladder catheterization and switched to this method of bladder emptying. The patient and her mother were informed about appendicovesicostomy (Mitrofanoff procedure) as an alternative method of bladder evacuation that may provide better social adaptation for patients with this condition.

Medical therapy included correction of electrolyte disturbances and acid–base imbalance, and administration of iron supplements, erythropoietin, and agents for reduction of nitrogenous waste levels. Preventive and supportive measures, including prevention of urinary tract infections, regulation of bowel movements, and management of menstrual cycle disturbances, were also implemented.

Following treatment, positive dynamics were observed: hemoglobin increased to 107 g/L, nitrogenous waste product levels decreased (creatinine decreased to 261.15 μmol/L), and GFR increased to 29.08 mL/ min/1.73 m²; metabolic acidosis and electrolyte disturbances resolved. At discharge, mild anemia and impaired renal nitrogen-excretory function persisted, corresponding to CKD stage 4.

Follow-up examination after one month demonstrated further positive changes: hemoglobin 102 g/L, creatinine 241.8 μmol/L, and GFR 34.0 mL/min/1.73 m². One month later, the following values were recorded: hemoglobin 137 g/L, creatinine 242.8 μmol/L, and GFR 34.2 mL/ min/1.73 m².

Discussion

CKD is a significant medical issue because of its high prevalence in the population, elevated mortality rates, and the substantial economic burden associated with renal replacement therapy. Correction and mitigation of risk factors therefore remain the primary focus of CKD prevention [6, 4]. Modifiable conditions, including blood pressure control, correction of metabolic acidosis and anemia, management of uric acid levels and growth failure, and improved adherence to therapy, have demonstrated important clinical value in delaying CKD progression [9].

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frequent manifestation of CKD is severe anemia, which served as the basis for CKD diagnosis in this clinical case. In a study by Bishnu Kumar Thapa et al., 110 (63%) of 174 children with CKD presented with anemia, with prevalence rates of 44%, 43%, 74%, 64%, and 92% in stages 1, 2, 3, 4, and 5, respectively [17]. Anemia severity correlates with CKD stage, as impaired renal erythropoietic function leads to loss of the normal physiological response to hypoxia, decreased endogenous erythropoietin synthesis, and dysregulation of iron metabolism, which overall affects erythrocyte synthesis. Inflammatory reactions accompanying CKD additionally reduce erythropoiesis efficiency [18, 19]. In the patient described in this study, the lowest hemoglobin level (42 g/L) was recorded during gynecological evaluation for menstrual disorders. Following blood transfusion, the hemoglobin level increased to 85 g/L and subsequently normalized to 137 g/L during further treatment. Analysis of this case suggests that the anemia had a combined etiology, including menstrual disorders and reduced renal erythropoietic function associated with CKD.

Proteinuria is an independent risk factor for CKD progression [2], and in combination with arterial hypertension (AH) or reduced GFR, it doubles the risk of rapid progression to stage 5 CKD compared with AH without proteinuria [20]. The combination of these two factors therefore has a synergistic effect. The maximum proteinuria recorded in the patient was 0.071 g/L. Although the absence of arterial hypertension and significant proteinuria suggests a more favorable CKD prognosis, the existing anemia, also regarded as an aggravating factor, may nevertheless accelerate GFR decline to 5-10 mL/min/year.

Impaired regulation of calcium, phosphorus, parathyroid hormone, and vitamin D in children with CKD leads to disorders of bone mineralization [21]. In our patient, the parathyroid hormone level was 501 pmol/L (reference range: 10-65 pmol/L), whereas assessment of vitamin D, calcium, and phosphorus levels was not possible. As a result, even in the absence of AH and growth retardation, the patient developed systemic complications of CKD, including severe anemia, mineral metabolism disorders, and acid–base and electrolyte imbalance.

Bladder dysfunction in children is clinically significant and should be excluded in patients with inflammatory diseases of the urinary system.

The causes of hypotonic neurogenic bladder dysfunction (NBD) are proposed for consideration. The causes of hypotonic neurogenic bladder dysfunction (NBD) are proposed for consideration. Hypotonic NBD may result from direct central nervous system trauma, particularly spinal cord injury, which represents one of the primary causes of its development and accounts for 3-5% of all spinal cord lesions. Other causes include diseases affecting the central or peripheral nervous system, specifically neurological disorders involving the lumbar region, sacral micturition center, or peripheral nerves. Detrusor areflexia/hypoactivity following spinal cord injury was identified in 18% of patients [22, 23]. It should be emphasized that the hypotonic type reflects the level of injury: the detrusor acquires hypotonic features following damage to the sacral parasympathetic pathways or peripheral nerves. No definitive evidence of trauma that could have caused hypotonic NBD was identified in this patient.

Among congenital causes of NBD, spinal dysraphism (SD) accounts for 93% of cases (more than 85% open and up to 8% occult), cerebral palsy accounts for 3%, whereas sacral agenesis, imperforate anus, and spinal cord lesions each account for 1%. Myelomeningocele is the most common form of open SD and occurs more frequently (in 30-50% of cases) at the lumbosacral level. In 90-98% of patients with myelomeningocele, bladder areflexia or NBD is also be present [22, 24, 25].

Other less frequent causes include brain or spinal cord tumors, pelvic surgery, sacrococcygeal teratoma, and posterior urethral valves [26, 27]. Causes more characteristic of adults but also observed in children include acute transverse myelitis and neuromyelitis optica. In acute transverse myelitis, detrusor atony confirmed by urodynamic data was identified in 12% of patients aged 10-69 years. In patients aged 11-64 years diagnosed with neuromyelitis optica, an acontractile detrusor was identified in 47.6% of cases [28]. In this clinical case, none of the aforementioned causes were identified.

The pathogenesis of chronic kidney disease development in the presence of bladder hyporeflexia is of particular clinical interest. Impaired bladder innervation disrupts its functional phases, reducing the control and coordination of micturition. During the filling phase, impairment of bladder filling sensation is accompanied by a decrease in detrusor tone, which facilitates excessive bladder distension, while intravesical pressure remains low. Changes may occur in the afferent fibers, urothelium, or suburothelial intercellular structures involved in sensing bladder distension. The voiding phase is disrupted as detrusor contraction is insufficient, leading to a large residual urine volume [22, 24], whereas disturbances may also occur in the effector or central components of nervous regulation. Effector denervation is associated with impaired contractility, resulting in detrusor underactivity [29]. In sacral/infrasacral spinal cord injury, lower motor neurons are damaged and the micturition reflex arc is interrupted. The pontine micturition center remains intact, leading to detrusor areflexia and flaccidity of the external urethral sphincter, resulting in overflow incontinence [30].

Excessive residual urine volume observed in this case predisposes to urinary tract infections and chronic bladder overdistension, ultimately resulting in loss of bladder wall elasticity, vesicoureteral reflux formation, and hydronephrosis. In cases of elevated intravesical pressure, the risk of renal scarring and long-term renal damage associated with vesicoureteral reflux increases. At low pressure, renal function deteriorates, which is associated with replacement of parenchyma by connective tissue, partly due to recurrent urinary tract infections, eventually leading to chronic kidney disease formation [15, 25].

A scheme for the pathogenesis of chronic kidney disease development in the presence of bladder hyporeflexia is proposed (Figure 3).

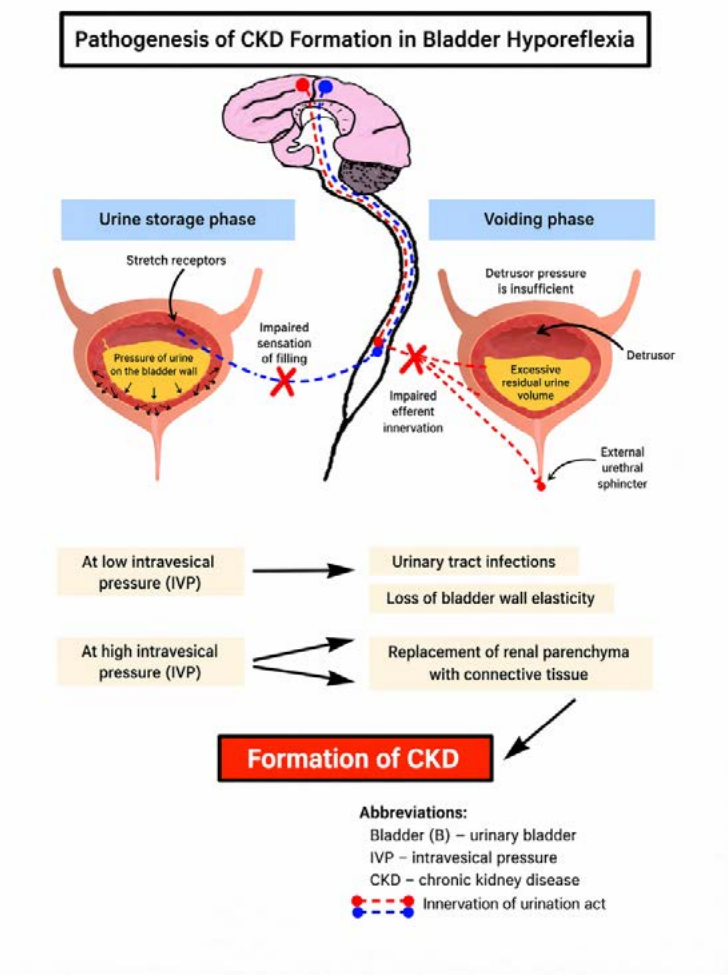

Figure 3. Scheme of the pathogenesis of CKD formation in the presence of bladder hyporeflexia.

A careful review of the medical history data allows the conclusion that one of the reasons for the late diagnosis of urodynamic disorders and progression to CKD stage 4 was insufficient interaction between inpatient and outpatient stages of medical care. After the second episode of UTI at the age of 9 years, ultrasonography performed during hospitalization revealed thickening and stratification of the left renal pelvic wall; however, the recommended follow-up kidney ultrasonography at the outpatient stage was not performed. Furthermore, bladder ultrasonography before and after voiding with assessment of bladder filling volume and postvoid residual urine volume was not performed either during hospitalization or at the outpatient stage, despite its critical importance for the diagnosis of bladder dysfunction. According to the 2025 EAU recommendations, ultrasonography should be performed in children before and after voiding to assess bladder changes depending on bladder filling and to measure postvoid residual urine volume, which may represent the primary diagnostic sign of voiding dysfunction [16].

Particular emphasis should be placed on the necessity of excluding vesicoureteral reflux in the child using voiding cystography; the examination itself was not performed in a timely manner. According to the 2025 EAU guidelines, this diagnostic method is not recommended for all children after the first episode of UTI but is required in children with recurrent febrile UTI or ultrasonographic abnormalities [16]. In the clinical case presented in this article, the patient had indications for voiding cystourethrography after the second episode of UTI at the age of 9 years.

Persistent hyposthenuria was not taken into consideration; consistently low urine specific gravity values indicated impaired renal concentrating capacity. Regular monitoring of general urinalysis was not performed.

The patient's GFR of 34.2 mL/min/1.73 m² does not currently require initiation of renal replacement (dialysis) therapy; however, the need for such therapy may arise in the future. To improve the patient's quality of life and social adaptation, surgical methods aimed at optimizing urinary bladder emptying, specifically appendicovesicostomy (Mitrofanoff procedure), may be recommended as an alternative method of urine evacuation.

The patient requires further follow-up by a nephrologist, regular monitoring of urodynamics, blood pressure, cardiovascular status (including echocardiography), mineral metabolism parameters, and prevention of UTIs to slow CKD progression.

Conclusions

The vast majority of chronic kidney disease cases in children are diagnosed at advanced stages, when the condition significantly reduces quality of life and adversely affects disease severity and prognosis.

The presented clinical case demonstrates that hyporeflexive bladder dysfunction may persist for a prolonged period without pronounced clinical

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manifestations, remaining undiagnosed and leading to severe complications such as hydronephrosis and CKD.

For earlier diagnosis in this case, the following were critically important: regular monitoring of general urinalysis with particular attention to urine specific gravity, follow-up renal ultrasonography after resolution of the inflammatory process, bladder ultrasonography before and after voiding, and voiding cystography.

In the management of patients with bladder dysfunction, continuity between inpatient and outpatient stages of medical care is essential, as is multidisciplinary collaboration among the pediatric nephrologist, pediatric urologist, pediatrician, and family physician.

Prospects for further research. Further investigation of disease course in the patient during long-term follow-up appears promising. Retrospective and prospective analyses of a series of similar clinical observations are planned to identify common patterns of disease course and optimize strategies for early diagnosis of chronic kidney disease and nephroprotection.

Author contributions. L. I. Vakulenko– development of the study concept and design, reviewing and editing the manuscript with intellectual contribution, final approval of the article; responsibility for conducting the work and its integrity; O. H. Babak – preparation of the literature review, analysis of results, manuscript writing; D. E. Ochyhava – data collection, interpretation of results, manuscript preparation; A. V. Riznyk – manuscript preparation; responsibility for conducting the work and its integrity; V. V. Postovoi – data collection, interpretation of results, manuscript writing; responsibility for conducting the work and its integrity; A. V. Obertynskyi – data selection for further analysis; participation in preparation the literature review.

Conflict of interest. The authors declare that no real or potential conflicts of interest exist in relation to the present study.

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ПІЗНЯ ДІАГНОСТИКА ХРОНІЧНОЇ ХВОРОБИ НИРОК У ДИТИНИ З ГІПОРЕФЛЕКТОРНИМ СЕЧОВИМ МІХУРОМ: АНАЛІЗ КЛІНІЧНОГО ВИПАДКУ ТА ОГЛЯД ЛІТЕРАТУРИ

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Резюме.

Хронічна хвороба нирок (ХХН) є вагомою медичною проблемою через її високу поширеність у популяції, значний рівень смертності та великі економічні витрати, пов'язані з проведенням замісної ниркової терапії. Переважна більшість діагностики випадків ХХН у дітей відбувається на пізніх стадіях, що значно знижує якість життя, впливає на тяжкість перебігу та прогноз захворювання. Діти з 3-5 стадіями ХХН мають в 4,5 рази вищий ризик несприятливих наслідків порівняно із тими, у кого ХХН діагностовано на ранніх стадіях. У зв'язку з цим виявлення та зменшення впливу факторів ризику є ключовим напрямом первинної профілактики ХХН. На сьогодні виокремлено цілу низку причин ХХН у дітей, серед яких, зокрема, розглядають гіпоактивність (гіпо/атонічність, гіпо/арефлексію) сечового міхура. У нашій роботі ми зосередили увагу саме на цій проблемі.

Мета дослідження. Проаналізувати клінічний випадок пізньої діагностики хронічної хвороби нирок у дитини з нейрогенною дисфункцією сечового міхура за гіпотонічним типом, виділити фактори ризику прогресування захворювання, можливі ускладнення та підкреслити важливість ранньої діагностики.

Матеріали та методи дослідження. Проведено аналіз встановлення діагнозу хронічної хвороби нирок IV стадії дівчинці на тлі нейрогенної дисфункції сечового міхура за гіпотонічним типом. Дослідження виконано за принципами Гельсінської декларації. Протокол дослідження ухвалено Локальним етичним комітетом. На проведення дослідження отримано інформаційну згоду дівчинки та її мами. Здійснено комплексний бібліографічний пошук у провідних електронних ресурсах: PubMed/ MEDLINE, Scopus та Web of Science.

Результати дослідження. Проведено аналіз клінічного випадку пізньої діагностики хронічної хвороби нирок 4 стадії у дівчинки 16 років на фоні нейрогенного сечового міхура за гіпотонічним типом, двобічного уретерогідронефрозу 3-4 ступеню. Діагностичний пошук почався зі звернення пацієнтки з приводу порушення менструального циклу та виявлення тяжкої анемії. У подальшому виявлено значне порушення азотовидільної функції нирок – креатинін – 332,5 мкмоль/л, ШКФ – 16,4 мл/хв/1,73 м2. Діагностичною знахідкою були виявлені на УЗД збільшення розмірів обох нирок, підвищення ехогенності парен-

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хіми, порушення кортико-медулярної диференціації, значне розширення чашково-мискового комплексу і сечоводу, критичні порушення уродинаміки: об'єм сечового міхура до мікції становив 1050 см³, а об'єм залишкової сечі – 420 см³. Представлений клінічний випадок демонструє, що дисфункція сечового міхура за гіпорефлекторним типом може тривалий час перебігати без вираженої симптоматики та залишатися недіагностованою, спричиняючи рецидивуючі інфекції, формування двобічного уретерогідронефрозу і, як наслідок, – прогресування ХХН. Навіть за відсутності артеріальної гіпертензії та затримки фізичного розвитку у пацієнтки сформувалися системні ускладнення ХХН, зокрема, тяжка анемія, порушення мінерального обміну, кислотно-основного стану та рівня електролітів. Виділені причини пізньої діагностики порушень уродинаміки та прогресування ХХН до 4 стадії у дівчинки, проведений аналіз інформаційних джерел.

Висновки. Для більш ранньої діагностики в даному випадку був важливим динамічний контроль загального аналізу сечі з урахуванням показника щільності, контроль УЗД нирок після ліквідації активності запального процесу, виконання УЗД сечового міхура до та після мікції, проведення мікційної цистограми. У веденні пацієнтів з дисфункцією сечового міхура важливим є послідовність у роботі стаціонарного та амбулаторного етапів надання медичної допомоги, мультидисциплінарна взаємодія дитячого нефролога, дитячого уролога, педіатра та сімейного лікаря.

Ключові слова: діти; хронічна хвороба нирок; пошкодження нирок; маркери пошкодження нирок, швидкість клубочкової фільтрації, нейрогенна дисфункція сечового міхура за гіпотонічним типом.

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