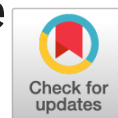


DOI: <https://doi.org/10.17816/uroved70857>

Clinical and laboratory features of the course of obstructive pyelonephritis in a patient with quantitative kidney abnormality



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The kidney duplication is the most common abnormality of the urinary system. In most cases, this condition is an accidental finding on prenatal ultrasound or can be diagnosed when the first clinical manifestations occur. Abnormalities of the upper urinary tract can be detected when examining a patient with arterial hypertension, proteinuria, or renal failure. As an example of the complicated course of the inflammatory process in a patient with quantitative kidney abnormality, a clinical observation of the course of obstructive pyelonephritis against the background of complete obliteration of the lower third of the ureter with the formation of terminal changes in the upper half of the doubled kidney, which led to renovascular hypertension and clinically significant renal failure, is presented. The article describes the clinical manifestations of the disease, laboratory and diagnostic screening, as well as the stages of surgical treatment in a multidisciplinary hospital.

Keywords: kidney duplication; hydronephrosis; renovascular hypertension; urinary tract abnormalities; pyelonephritis.

To cite this article:

Suleymanov SI, Kadyrov ZA, Dilanyan OE, Ramishvili VSh, Musohranov VV, Babkin AS. Clinical and laboratory features of the course of obstructive pyelonephritis in a patient with quantitative kidney abnormality. *Urology reports (St. Petersburg)*. 2021;11(2):183-190. DOI: <https://doi.org/10.17816/uroved70857>

Received: 20.05.2021

Accepted: 09.06.2021

Published: 23.06.2021

DOI: <https://doi.org/10.17816/uroved70857>

Клинико-лабораторные особенности течения обструктивного пиелонефрита у пациента с количественной аномалией почек

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Удвоение почки — самая распространенная аномалия мочевыводящей системы. В большинстве случаев данное состояние является случайной находкой при пренатальном ультразвуковом исследовании или может быть диагностировано при возникновении первых клинических проявлений. Аномалии развития верхних мочевых путей могут быть обнаружены при обследовании пациентов с артериальной гипертензией, протеинурией или почечной недостаточностью. В качестве примера осложненного течения воспалительного процесса у пациента с аномалией количества почек представлено клиническое наблюдение за течением обструктивного пиелонефрита на фоне полной облитерации нижней трети мочеточника с формированием терминальных изменений верхней половины удвоенной почки, приведшее к реноваскулярной гипертензии и клинически значимой почечной недостаточности. В статье подробно описаны клинические проявления заболевания, лабораторный и диагностический скрининг, а также этапы хирургического лечения в условиях многопрофильного госпиталя.

Ключевые слова: удвоение почки; гидронефроз; нефрогенная артериальная гипертензия; аномалии развития мочевых путей; пиелонефрит.

Как цитировать:

Сулейманов С.И., Кадыров З.А., Диланян О.Э., Рамишвили В.Ш., Мусохранов В.В., Бабкин А.С. Клинико-лабораторные особенности течения обструктивного пиелонефрита у пациента с количественной аномалией почек // Урологические ведомости. 2021. Т. 11. № 2. С. 183–190. DOI: <https://doi.org/10.17816/uroved70857>

INTRODUCTION

Urinary and genital organ abnormalities are widespread and account for about 40% of all congenital malformations. Autopsy data reveal that about 10% of people have such defects [1]. Studies have shown a wide range of congenital abnormalities of the kidneys and urinary tract, from mild, relatively asymptomatic malformations, such as ureter duplication, to severe, sometimes non-survivable ones, such as bilateral renal agenesis [2, 3]. Some of these kidney abnormalities can be detected by multidisciplinary approaches, including fetal ultrasound examination and vesicoamniotic shunting to facilitate drainage during the prenatal period or by other imaging techniques [4, 5].

One common malformation of the urinary system is kidney duplication, which is a risk factor for a number of diseases leading to disability. Kidney duplication is incomplete in about 1 in 25 people, whereas it is complete in 1 in 125 people. Complete duplication of the kidney on both sides is found in 40% of cases. A study has found that about 10% of siblings have complete kidney duplication [6].

The kidneys develop through several sequential stages: pronephros, mesonephros, and metanephros. First, the pronephros tubules merge and flow into the mesonephric duct. Afterwards, a metanephrogenic blastema branches further from the tail end of the duct, which represents an accumulation of mesodermal cells. Metanephrogenic blastema also generates parts of the pelvicalyceal system. Kidney duplication occurs when the protrusion of the mesonephric duct splits before the latter grows into metanephrogenic tissue and then reaches the metanephrogenic blastema, forming independent renal structures. An incomplete duplication of the kidney most often occurs without the creation of its own accessory kidney, with a common fibrous capsule, but with its own vessels, ureter, and pelvicalyceal system. Because of this, the lack of adequate urine excretion, more often from the upper part of the duplicated kidney, increases the risk of pyelonephritis, hydronephrosis, pyonephrosis, and lithogenesis [7, 8].

Fortunately, the duplication of the collector systems of the kidneys in most cases is asymptomatic and is diagnosed occasionally through first-line examination, such as the ultrasound examination (US) of the kidneys and bladder, to assess the upper urinary tract and identify the expansion of the collector system and renal parenchyma thickness. Additionally, intravenous contrasting urography "delineates" the anatomy of the kidney (e.g., dilatation of the collector system, displacement of the kidneys or ureters, characteristics of the bladder wall) and provides a subjective assessment of renal function. Conversely, nephroscintigraphy provides an objective assessment of renal function and the drainage effectiveness

of the dilated pelvicalyceal system. Meanwhile, magnetic resonance imaging (MRI) provides anatomical and functional assessment of the renal parenchyma, collector system, and vascular system without X-ray irradiation [9, 10]. However, MRI requires sedation in younger children since it is sensitive to motion artifacts.

Renal duplication abnormalities with concomitant pathology require appropriate drug therapy and possible surgical correction. Clinical symptoms, usually related to complete kidney duplication, are caused by complications such as obstruction, vesicoureteral reflux, ureterocele, urinary tract infection, arterial hypertension, proteinuria, and renal failure [3, 12]. While surgical treatment is mainly performed only when clinically significant symptoms appear in childhood, similar surgeries are performed in adults. Treatment depends on the function of the upper half of the duplicated kidney. If the function is adequate, an ureteropyelostomy is performed (the ureter of the upper half of the duplicated kidney to the pelvis of the lower half of the kidney). However, if vesicoureteral reflux is not noted in the lower half of the kidney, ureteroureterostomy is done (the ureter of the upper half of the kidney to the ureter of the lower one). Contrarily, if the upper half of the kidney is dysfunctional, a heminephroureterectomy is performed, preferably using a laparoscopic approach [7, 11, 13].

CLINICAL CASE

This work aims to demonstrate our own experience in the surgical treatment of a patient with obstructive uropathy in the presence of a quantitative kidney anomaly that led to renovascular hypertension and clinically significant renal failure. We performed the treatment of the patient in stages in the urological department of the City Clinical Hospital No. 13 of the Moscow City Health Department from 17.12.2020 to 21.12.2020 and from 21.01.2021 to 27.01.2021.

On a routine check-up with a cardiologist at the consultative and diagnostic department of the hospital, a 39-year-old patient complained of a persistent increase in blood pressure (up to 180/100 mm Hg), peripheral edema on the skin of the upper half of the body, general weakness, rapid fatigue, and lack of appetite with subfebrile fever (up to 37.2°C). Electrocardiography and echocardiography ruled out acute coronary pathology. Express laboratory screening of the main clinical and biochemical parameters indicated the presence of moderate leukocytosis (up to $13.8 \times 10^9/l$), anemia (hemoglobin level = 108 g/l), and an increase in creatinine level (up to 128 $\mu\text{mol/l}$). The patient was referred to a nephrologist. Further diagnostic tests required sequential ultrasound of the urinary system, and since unilateral dilatation of the pelvicalyceal system, with a decrease in the parenchymal thickness of the left kidney upper pole, was found,

multispiral computed tomography with intravenous contrast (MSCT) was performed. Since comprehensive X-ray examination of the urinary system indicated obstructive uropathy in the presence of a congenital abnormality of the upper urinary tract with negative laboratory changes (an increase in blood creatine up to 169 $\mu\text{mol/L}$), the patient was referred to a urologist for further therapeutic treatment.

Based on MSCT results, congenital quantitative renal anomaly complicated by terminal hydronephrosis of the upper half of the duplicated kidney with dilatation of the ureter up to the lower third was diagnosed in the patient for the first time in his life (Fig. 1). Additionally, timed renal contrasting of the pelvicalyceal system was done, and the subsequent passage of the contrast agent along the ureter to the bladder was clearly traced from the lower half of the duplicated left kidney only, indicating the presence of a single vascular pedicle. Because the contrast agent did not pass along the dilated ureter from the upper half of the left kidney during the entire study period, the nature of the quantitative renal anomaly based on MSCT alone could not be determined. Based on the results of the preliminary clinical and instrumental examinations and the presence of subfebrile condition, the patient was admitted to the urology department to clarify the diagnosis and determine a possible method of draining the upper left urinary tract.

We attempted a retrograde drainage of the upper half of the duplicated left kidney in the urological department; however, we examined only the left orifice during cystoscopy. In this regard, we performed the first stage involving percutaneous puncture drainage of the upper half of the duplicated left kidney, draining up to 200 ml of purulent discharge, which was sent for microbiological

analysis. The patient underwent a course of detoxification, infusion, and antibacterial therapy with broad-spectrum drugs with a positive clinical and laboratory effect postoperatively. The serum creatinine level and leukocyte count were stabilized (108 $\mu\text{mol/l}$ and $8.8\text{--}9.0 \times 10^9/\text{l}$, respectively). The body temperature has completely normalized. Daily urine output through nephrostomy drainage did not exceed 100–120 ml, and blood pressure persistently decreased to prescriptive values during follow-up. Antegrade pyelography revealed complete obliteration of the lower third of the ureter of the upper half of the duplicated left kidney. The patient was discharged on day 4 with full course drug therapy recommendation for purulent pyelonephritis and follow-up visit 1 month after dynamic nephroscintigraphy to determine the scope of the final stage of surgical treatment.

During the planned repeat hospitalization in the same department, palpation of the abdomen and lumbar region did not provoke pain sensation typical of acute urological pathology in the patient, and symptoms of peritoneal irritation and costovertebral angle tenderness were negative. The left renal fistula area had no signs of inflammation, the nephrostomy drainage was functioning, and the volume of excreted urine was the same (≤ 100 ml per day).

Laboratory results revealed erythrocyte sedimentation rate at 12 mm/h (reference values 2–8 mm/h), hemoglobin level at 118.0 g/l, and an insignificant increase in creatinine level (169 $\mu\text{mol/l}$, with reference values of 62–106 $\mu\text{mol/L}$). Additionally, Jelliffe creatinine clearance was 43.31. Contrarily, urine analysis showed turbid transparency, quantitative protein at 1.72 g/l, and leukocyte count at 40–50. Coagulogram indices were also within reference values.

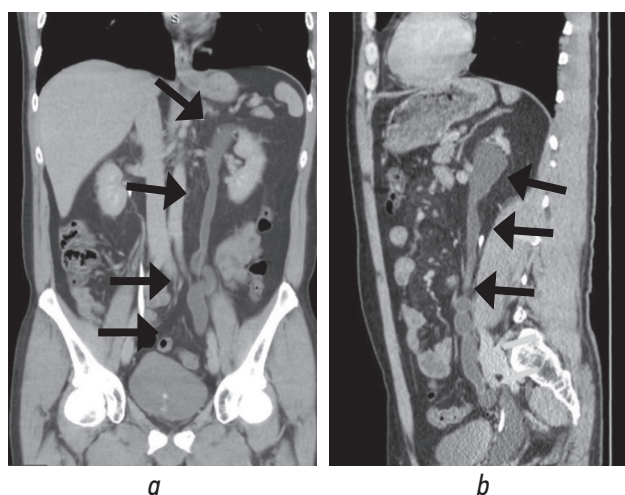


Fig. 1. MSCT of the kidneys with intravenous bolus contrast in a patient with congenital quantitative kidney anomaly complicated by terminal ureterohydronephrosis of the upper half of an incompletely doubled left kidney: *a* – frontal projection; *b* – sagittal projection. Arrows indicate a dilated ureter

Рис. 1. Мультиспиральная компьютерная томограмма почек с внутривенным болюсным контрастированием у пациента с врожденной количественной аномалией почек, осложненной терминальным уретерогидронефрозом верхней половины неполно удвоенной левой почки: *a* – фронтальная проекция; *b* – сагиттальная проекция. Стрелками указан дилатированный мочеточник

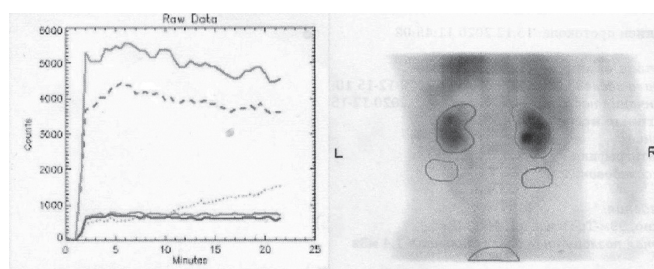


Fig. 2. Nephrosцинтиграм of a patient with congenital quantitative kidney anomaly complicated by terminal ureterohydronephrosis of the upper half of an incompletely duplicated left kidney

Рис. 2. Нефросцинтиграмма пациента с врожденной количественной аномалией почек, осложненной терминальным уретерогидронефрозом верхней половины неполно удвоенной левой почки

Dynamic nephrosцинтиgraphy results revealed that kidney functions on both sides were moderately reduced, which could be largely due to the deterioration of urine passage through the ureters with intact parenchymal function. The functional contribution ratio of the left and right kidneys was 38%:62% (Fig. 2).

We removed the nephrostomy drainage, corrected the nephrogenic hypertension, and prevented vascular complications to improve the patient's quality of life. We also decided to do a heminephroureterectomy on the left side laparoscopically on the patient.

We performed the surgery in the Trendelenburg position on the right side using combined anesthesia with artificial pulmonary ventilation. The surgery protocol included the following stages. We installed a temporary urethral Foley catheter No. 16 in the bladder in order to control urine output. We made a skin incision at a distance of 2 cm from the umbilical ring, inserted a central trocar (5–12) pararectally according to Hudson's technique, and

induced a 12-mm Hg pneumoperitoneum. Additionally, we installed three working trocars with a diameter of 5 and 10 mm. Diagnostic laparoscopy revealed no abdominal pathology. Using a Sonicision ultrasonic dissector, we then dissected the parietal layer of the peritoneum in the transition area of the transverse colon and descending colon. We mobilized the descending colon in a blunt and sharp manner to the level of the proposed projection of the lower pole of the left duplicated kidney and to the projection of the corona mortis and then bluntly separated the Toldi and Gerot fascia. We saw that the paranephric fat on the left was in a state of vitreous edema, tightly adhered to the renal capsule (presentation of armor paranephritis). We then opened the paranephrium, mobilized electroscurgically from the anterior surface and hilum of the left kidney. We also mobilized both ureters of the duplicated kidney bluntly while dilating the extension from its upper half to 1.5 cm, thickening the wall and infiltrating the surrounding tissue (Fig. 3, a).

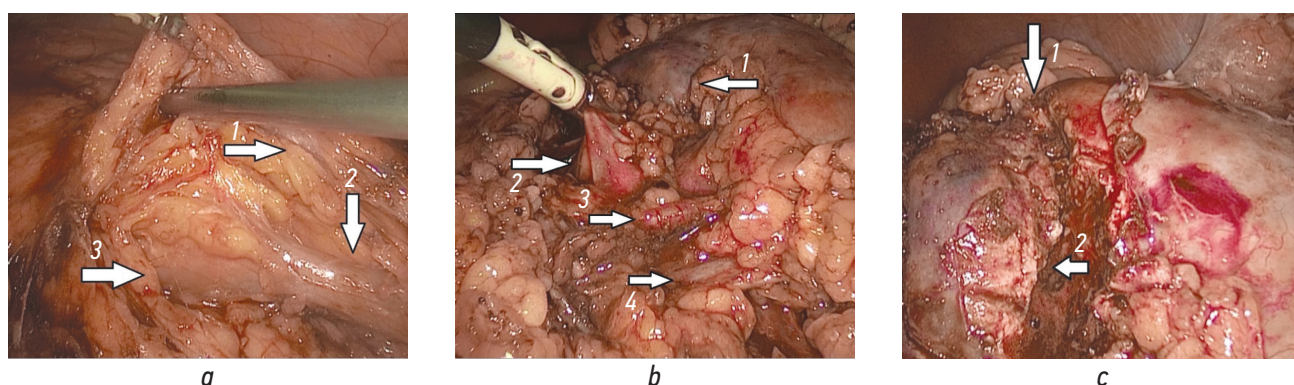


Fig. 3. Videoendoscopic heminephroureterectomy on the left: *a* – the stage of mobilization of the ureters (1 – unchanged ureter of the lower half of the doubled left kidney, 2 – dilated pelvis of the upper half of the doubled left kidney, 3 – dilated ureter of the upper half of the doubled left kidney); *b* – the stage of mobilization of the terminally changed segment of the left kidney (1 – upper pole of the doubled left kidney, 2 – pelvis of the upper half of the doubled left kidney; 3 – left renal artery, 4 – left renal vein); *c* – the stage of resection of the terminally changed segment of the upper half of the doubled left kidney (1 – the edge of the resection, 2 – the resected segment of the doubled left kidney)

Рис. 3. Видеоэндоскопическая геминефроуретерэктомия слева: *a* – этап мобилизации мочеточников (1 – неизменный мочеточник нижней половины удвоенной левой почки, 2 – дилатированная лоханка верхней половины удвоенной левой почки, 3 – дилатированный мочеточник верхней половины удвоенной левой почки); *b* – этап мобилизации терминально измененного сегмента левой почки (1 – верхний полюс удвоенной левой почки, 2 – лоханка верхней половины удвоенной левой почки, 3 – левая почечная артерия, 4 – левая почечная вена); *c* – этап резекции терминально измененного сегмента верхней половины удвоенной левой почки (1 – край резекции, 2 – резецированный сегмент удвоенной левой почки)

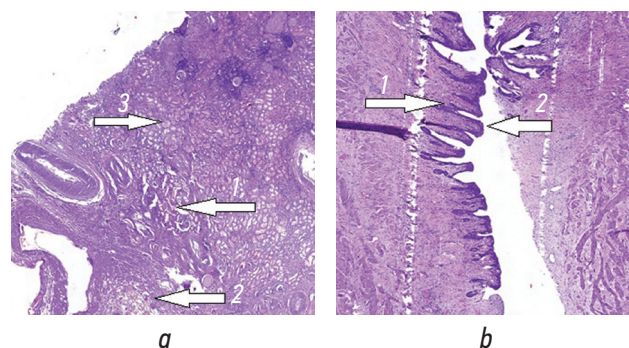


Fig. 4. Microscope specimen: *a* – the resected area of the left kidney, hematoxylin-eosin, $\times 100$ (1 – focal fibrosis, 2 – hemorrhages, 3 – foci of lymphohistiocytic infiltration with an admixture of segmented leukocytes; *b* – the resected section of the ureter, hematoxylin-eosin, $\times 100$ (1 – the wall of the ureter with hemorrhages, 2 – granulations, flattened mucosa)

Рис. 4. Микропрепарат: *a* – резецированный участок левой почки, окраска гематоксилином и эозином, $\times 100$ (1 – очаговый фиброз, 2 – кровоизлияния, 3 – фокусы лимфогистиоцитарной инфильтрации с примесью сегментоядерных лейкоцитов; *b* – резецированный участок мочеточника, окраска гематоксилином и эозином, $\times 100$ (1 – стенка мочеточника с кровоизлияниями, 2 – грануляции, уплощенная слизистая)

We then mobilized the latter 3 cm below the intersection zone with the iliac vessels, resected it, and clipped the stump. The next stage involved examining the main renal pedicle, represented by one artery and two veins. During visual examination of the anterior surface of the left kidney, we noted that the parenchyma deficit in the upper pole region was clearly defined, the kidney tissue was hydronephrotically substituted, and the surface was dirty gray in color. We also noted that the pelvis of the upper half of the left kidney was infiltrated, tightly adhered to the surrounding tissue, and significantly dilated. We then performed a phased mobilization of the pelvis and ureter in the upper third, accompanied by moderate hemorrhage in the presence of an infiltrative process (Fig. 3, *b*). We also temporarily blocked the renal artery with an ischemia time of 11 min. We then electrosurgically resected the hydronephrotically altered upper pole of the duplicated kidney within the visually intact renal tissue (Fig. 3, *c*). We placed a fragment of the adjacent mobilized tissue to the bottom of the resected part of the kidney, followed by approximation suture of the edges of the resection zone using Vicryl 0 sutures. While controlling hemostasis, we placed the resected part of the kidney and ureter in a separate Endobag container and removed them through the working port. We also installed a safety drain in the left lateral flank to the area of the resected ureter stump and removed the trocars while suturing the skin wounds, followed by aseptic dressings. The surgery duration lasted 145 min. No complications occurred during the surgery, and total blood loss was kept at 70 ml.

The histological examination revealed fibrotic kidney tissues with the uriniferous tubules either compressed or atrophic in different areas, as well as hyaline-like masses in the lumen of the tubules. We noted focal hemorrhages and lymphohistiocytic infiltration in areas with an admixture of segmented leukocytes (Fig. 4, *a*).

Additionally, we noted hemorrhages and granulations on the ureter wall as well as flattened ureteral mucosa (Fig. 4, *b*).

The patient underwent replacement infusion, antibacterial, gastroprotective, and anticoagulant therapy under dynamic clinical and laboratory control after the surgery. After confirming that the patient's condition has stabilized, we transferred him to the specialized urological department, where ongoing therapy continued. We removed the urethral catheter and safety drain on day 2 after the surgery. Follow-up ultrasound examination did not find free fluid in the abdominal cavity, and the pelvicalyceal system did not expand on either side. Since the patient's daily urine output did not exceed 2,000 ml and the patient was fully activated and self-adapted, he was discharged from the hospital on day 5 after the surgery.

CONCLUSION

Data analysis from international and Russian literature shows that quantitative malformations of the kidneys requiring surgical correction rarely occur in adults. The range of surgical interventions used for pathological conditions in the presence of duplication of the upper urinary tract includes both preservation of a functioning kidney segment and organ-resecting surgeries. The prevalent introduction of minimally invasive endoscopic techniques is currently practiced in Russian surgery.

The complicated course of obstructive uropathy in a patient with quantitative renal anomaly in this case study, which led to renovascular hypertension and clinically significant renal failure, indicates the absence of clear diagnostic criteria for assessing its severity in adult urological practice at the outpatient-polyclinic stage. Considering the frequency of the asymptomatic nature of the disease, the present algorithm for examining patients

with suspected chronic obstruction of the upper urinary tract should include highly informative methods of medical imaging. The high-resolution capability of multispiral computed tomography and MRI, as well as the possibilities of endourological methods of optical control, not only detects any urinary tract obstruction, but also assesses its causes, which is of decisive importance in planning the surgical intervention method.

Because current literature presents no detailed descriptions of surgical approaches and techniques based on the specific nosological data, the question of the indications for ureterectomy and the permissible level of resection of a nonfunctioning ureter remains unsolved. This implies the need to develop and optimize laparoscopic heminephrectomy techniques to reduce the probability of complications at each stage of the surgical intervention.

Combining the use of modern radiology and video endoscopy and integrating interdisciplinary approaches in interpreting high-quality diagnostics of obstructive conditions of the upper urinary tract will enhance and enable the personalization of treatment methods while taking into account the risk of complications.

Given the limited amount of data on the results of laparoscopic heminephrectomy, analyzing the immediate and long-term results of laparoscopic surgeries and comparing them with the results of traditional open interventions are important.

ADDITIONAL INFORMATION

Conflict of interest. The authors declare no conflict of interest.

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