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Complicated forms of pyelonephritis in children

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The article is dedicated to purulent renal lesion which is considered to be very rare in children. Case reports on complicated and purulent pyelonephritis forms in children are only singular. The authors present their own experience of observing 10 such clinical cases. Clinical manifestations, peculiarities of laboratory parameters' change and also the visualization efficiency of kidney changes discovered with instrumental methods are considered in detail. Antibacterial therapy peculiarities are also shown. The authors offer the criteria, according to which a complicated urinary tract infection course may be suspected with high confidence.
Keywords: *urinary tract infection, complicated pyelonephritis, course, etiology, diagnostics peculiarities, treatment, children.*

INTRODUCTION

Urinary tract infections (UTI) are one of the main forms of bacterial infection in children; it is most often observed in infancy and early childhood. UTI are revealed in 5-12% of all children of 0-2 years of age admitted to reception wards of different medical institutions with fever [1, 2]. UTI rate in the Russian Federation (RF) is estimated at 1,000 cases per 100,000 people [3].

UTI are more often registered in boys of the first months of age (up to 3%); by 1 year of age the rate drops down to 1-2%, in preschool age – down to 0.5%, in puberty – down to 0.1%. UTI rate in girls rises: in girls of 0-1 years of age it may be as high as 2.7%; in preschool age UTI are revealed in 1.2-1.9% of girls [4]. Several studies reveal even higher figures of UTI rate in girls.

Usually, UTI take a mild course with clinical symptoms of low intensity; however, pyretic fever may appear in some patients; this is considered to indicate pyelonephritis. The ascending infection affects kidneys with separate nidi, which may later undergo fibrosis and cicatrization revealed at isotopic examination. Between nidi there are segments of normal renal parenchyma. Such a development is the most typical of urinary tract obstruction; when revealed, it indicates the necessity of a more intensive therapy and even surgical treatment in some cases. Non-obstructive pyelonephritis caused by an ascending or hematogenous renal infection develops rarer.

Focal inflammation in renal parenchyma was previously known as "focal bacterial nephritis" (before that it had been known as "focal nephronia"); this term covered both the infiltration and suppuration stages nidi are inclined to. Formation of an abscess out of such a nidus may be revealed by computed tomography (CT) [5]. Purulent multicompartment (usually – corticomedullary) processes in renal parenchyma is known as "renal carbuncle". If there is a mass of suppurative foci in a parenchyma, we talk about apostematous nephritis (the process often spreads to the paranephric body).

In case of a hematogenous infection way, abscess or carbuncle appear due to the obstruction of a great end renal vessel with a septic embolus or due to the fusion of several abscesses.

Size of corticomedullary nidi, abscess or carbuncle may reach 6cm or more; sometimes a purulent process may affect the whole kidney. It is considered that nidi of 2-3cm may involve in case of timely and adequate antibiotic therapy (in adults). Nidi of bigger size usually require drainage [6].

In case of a hematogenous affection resulting in suppuration, the causative agent enters the kidney with blood flow from the primary extrarenal suppurative focus or due to bacteremia, especially frequent in early childhood [7]. In a series of observations by Y.D. Prokopenko, extrarenal suppurative foci were revealed in half of children with "primary" (i.e. not associated with renal or urinary tract pathology) purulent pyelonephritis (60% - skin furuncle or carbuncle; 35% - tonsillitis and its complications); the predisposing factor in 33% was pancreatic diabetes. Ca. 40% of the observed in these series were boys [8]. At the same time, such foci were not revealed at all in a study by German authors [9].

Purulent renal lesion in childhood is a rare pathology; only individual observations have been described so far [10, 11]. The data of the series of observing such patients mentioned below have been being collected for 20-30 years.

Data on the relative role of obstruction and hematogenous infection in the development of purulent nephritis forms in children are scarce. According to the data of inpatient hospitals, in adults they are revealed in 23-59% of cases at non-obstructive and in 40-43% of cases at obstructive forms [12]. Y.D. Prokopenko [8] observed 122 patients with purulent pyelonephritis forms at the Novokuznetsk pediatric surgery department; only 26 (21%) of those children had renal malformations. At the same, purulent forms were revealed in 15% of children with pyelonephritis hospitalized to the Minsk pediatric urology department; obstructive forms were prevalent (93.1%) [13]. In a series of observations conducted in 2 pediatric clinics of Munster and Celle (Germany), anomalies causing urinary tract obstruction were revealed in 12 out of 25 hospitalized patients with bacterial nephritis [9].

The primary (up to 80%) causative agent of UTI, including pyelonephritis, is *E. coli*, which has become amoxycillin-tolerant in the RF (more than 50% of strains), though it has remained usually susceptible to amoxycillin + clavulanic acid, III generation cephalosporins and amino glycosides. In 10-20% of cases the infection is caused by *Klebsiella pneumoniae, Pseudomonas, Proteus* etc. Species composition of flora causing purulent renal process development depends largely on the lesion form. Intestinal group bacteria are usually revealed at obstructive pyelonephritis forms, aurococcus is prevalent at hematogenous forms. The same causative agent is identified in most patients with known infection source [8].

Violent disease onset with symptoms indicating intoxication (fever of up to 40-41^{o}C, usually with shaking chills and abundant perspiration) is characteristic of purulent pyelonephritis forms in adults. As a rule, there is sharp renal palpatory tenderness and muscle tension. The most frequent signs of purulent renal lesion in children are anorexia (92%), emesis (86%), excitation (70%), constipation (60%), diarrhea (10%) and convulsion (6%). Dysuric disorders (pollakiuria, dysuria, oliguria) were noted in 28% of patients. Scarcity of local symptoms results in diagnostic mistakes leading to delay in prescription and correction of therapy [14].

Seeding of causative agents in urine of more than 10^5 CFU/ml and leukocyturia are criteria of any URI. However, there may be no direct correlation between the degree of leukocyturia and severity of pyelonephritis. It is assumed that the number of blood leukocytes of more than 10-12x10^9/l may indicate acute pyelonephritis better than leukocyturia [15]. As for thrombocytosis of >500x10^9/l, it is characteristic both for renal abscess and apyetous obstructive pyelonephritis [16]. More than 60g/l of C-reactive protein (CRP) and more than 10ng/ml of procalcitonin correlate with the indication of parenchymatous alterations of kidneys (usually, they also increase in size) at ultrasound examination (US) [2].

According to a range of authors, US is highly informative (80-100%) in revealing purulent renal lesions in adults [17]. However, there are also works that insist on the low US resolution in case of renal parenchyma lesion (without involving paranephric body) [8]. This is absolutely true about children; examination of parenchyma in them usually ends with measuring size of kidneys and, considerably rarer, with detecting altered density nidi or focal blood flow disorder.

Intravenous urography gives sufficient data to diagnose purulent pyelonephritis: reduced contrast accumulation zones and deterioration of caliceal contours. It is also possible to verify diagnosis using static renal radionuclide imaging, although specificity and sensitivity are insufficient [18].

CT (especially using contrast enhancement) and magnetic resonance imaging give a clearer presentation of parenchymatous alterations of kidneys. Small children require narcosis in order to conduct these examinations; obviously, this requires additional clinical and paraclinic examinations [19-21].

Study aim: reveal peculiarities of clinical-laboratorial and visualization data in children with acute inflammatory renal processes causing concern in terms of purulent complications, definition of indications to CT.

PATIENTS AND METHODS

In 2009-2010, 10 out of 98 children hospitalized to the RAMS FSBI SCCH research institute with URI were distinguished by the unusually severe condition, stable pyretic fever and CRP content of more than 100mg/l. Acute purulent renal process was verified in 9 patients using CT with contrast enhancement, in 1 patient – using US. 3 children out of them required operative intervention, which confirmed diagnosis. The data are given in tb. 1 and 2. Age of patients was from 2 months to 14 years: 4 patients of 0-1 years of age, 3 – 1-5 years of age, 3 – over 7 years of age; 4 boys and 6 girls. It was the first URI episode in 9 children; indications of a URI, diagnosed as pyelonephritis, at 7 months of age were noted only in 1 8-year-old patient (patient #9). Urinary tract obstruction signs were not revealed in any case. Pyelocaliceal system's duplication in both kidneys may have been a predisposing factor in a 3-month-old patient (patient #10), hypoimmunoglobulinemia (IgG – 1.19g/l, IgM – 0.24g/l and IgA – 0.18g/l) – in the other 3-month old patient (patient #2). 2 children (patients #5 and #9) had had infections, treated using antibiotics, 2-3 weeks before the disease.

RESULTS

Clinical manifestations

4 children were hospitalized 2-4 days after the disease onset, 3 – 5-6 days after, 1 – 8 days after, 1 – 11 days after; 1 child was hospitalized after 3 weeks of unsuccessful treatment using antibiotics in a different inpatient hospital. The main complaint and reason for hospitalization in all children was high body temperature (39.7-41^{o}C), which suddenly appeared in the setting of full health. Hectic fever was accompanied by an intense chill in 6 children: in 4 – from the 1st disease day, in 2 – on the 5th and 9th fever day. 1 child, who had been receiving cefuroxime for 4 days, had fever up to 38.5^{o}C (patient #8).

No patients had catarrhal signs on the part of respiratory tract; together with high fever and other symptoms it allowed suspecting a severe bacterial disease, possibly accompanied by bacteremia. Almost all children refused food, repeated emesis was noted in 4 patients. Small children had anxiety and skin pallor; 2 out of them had icteritiousness (patients #3 and #6); 1 patient (patient #10) had mottled skin, bloating and altered stool.

All children over 4 years of age complained about abdominal pains; palpatory tenderness was more pronounced on the affected kidney's side. Tender spot was also noted in the costovertebral angle on the affected side. CVA tenderness was also positive in these children. Anxiety and inconsolable crying may be equivalent to pain syndrome in infants.

No children experienced dysuric syndrome. Intense leukocyturia (more than 100 leukocytes per field of vision) was revealed in the first urine analyses only in 5 children; number of leukocytes in all the other did not exceed 25 per field of vision. Rapid leukocyturia decrease (down to total disappearance) after just 1 day of antibacterial treatment with the remaining on the same level or even increasing fever and inflammation markers was very distinctive.

Data of laboratory studies

Urine cultures in 7 children who did not receive antibiotics gave increase in causative agents of more than 10^5 CFU/ml per titer: *E. coli* – in 6 children, *Kl. pneumoniae* – in 1 patient. They were positive in blood cultures of 4 out of 6 children: in 2 – *E. coli* (in 1 patient – together with *Enterobacter*), in 2 – positive blood and urine cultures corresponded (*Kl. pneumoniae*).

Multiple resistance to antibiotics was revealed in 2 urine isolates when studying sensitivity of the isolated strains. *Kl. pneumoniae* separated from blood of a 7-month-old child, who had been receiving antibacterial therapy for a long period of time, appeared resistant to all penicillins, cephalosporins and amino glycosides (except amikacin) and sensitive to meropenem (extended-spectrum β-lactamase +). Other strains were sensitive to amoxycillin + clavulanic acid, III-IV generation cephalosporins and amino glycosides.

Sharp increase in laboratory inflammation markers was noted in all children. Hyperleukocytosis was revealed in most children: in 4 – within 20-30x10^9/l, in 5 – within 30-39x10^9/l, as a rule, with 75-85% of neutrophils and left shift in leukogram (stab forms – up to 7x10^9/l). Thrombocytosis higher than 500x10^9/l at hospitalization was noted in 3 children; the number of platelets exceeded this level in the setting of treatment in 5 patients. As was mentioned above, an increase in CRP content within 130-250mg/l was revealed. Increased procalcitonin concentration (3-82ng/ml) was revealed in all 5 patients with positive blood cultures. Procalcitonin concentration was within 1.2-2.2ng/l in 3 children with negative blood cultures and only in 2 patients who had received antibiotic treatment earlier it was lower than 0.5ng/ml. Leukocytosis in these patients was 14x10^9/l and 27x10^9/l, CRP concentration – 110 and 85mg/l, accordingly. ESR exceeding 20mm/h at hospitalization was noted in 4 children. Blood urea indices remained normal in all children; creatinine level slightly exceeded age norm in 2 children.

Severity of clinical manifestations and high inflammatory potential indicated the possibility of a complicated form of pyelonephritis. All children underwent renal US at hospitalization and in dynamics. We were interested when and to what extent may the US data indicate focal changes, which are only rarely revealed at the first examination.

Data of visualization methods

Data of renal US and CT are given in tb. 3. Focal changes at US were not revealed in any of the 5 children who had been hospitalized in the first days of disease (in days 2-4). Kidney was increased in 2 of these patients, blood flow in cortical layer was stripped in 1 patient. Hypoechoic segment of 7x12mm was revealed in left kidney following the US examination in dynamics of a 7-year-old child (pic. 1) on the 3rd and 5th day of disease. Abscess formation was revealed on the 3rd day using contrast-enhanced CT.

No parenchymatous alterations were revealed by US in the other 2-month-old child (pic. 2) on the 4th day of disease; 2 segments in right kidney (8x5mm and 6x4mm) with altered structure and blood flow could be defined on the 6th day. Increased density nidi of slight opacification with no encapsulation signs were revealed in both kidneys on the 4th day using CT.

No focal changes were revealed at the first and subsequent US in 2 children hospitalized on the 2nd-4th day of disease (both – with positive hemoculture). Reduced opacification zones were revealed in both kidneys in a 3-month-old child with PCS duplication and *E. coli* discharge (septicopyemia) using CT. According to the CT data, the other 3-month-old patient with

hypoimmunoglobulinemia and *Kl. pneumoniae* discharge from urine and blood had 2 slight opacification nidi in both kidneys. These 2 radiopharmaceutical accumulation decrease nidi of 8.5x26mm and 17x21mm in the left kidney's superior and inferior segments remained (acute corticomedullary pyelonephritis) 2 months later when static renal radionuclide imaging using technetium (Tc^{99}) was conducted.

Hypoechoic formation in an 8-year-old girl (pic. 3) hospitalized on the 2nd day of disease was revealed by US only on the 5th day; CT revealed carbuncle and multiple apostematous nidi requiring drainage on the 4th day of disease.

US at hospitalization revealed increase in size of both kidneys only in a 14-year-old boy out of 3 children hospitalized on the 5th-6th day of disease. US in dynamics revealed a heterogeneous segment of parenchyma (49x25mm) on the 9th day of disease; it had been reducing under the influence of treatment for 6 days and could not have been visualized on the 15th day of disease. CT (7th day of disease) revealed signs of abscess forming in 1 kidney in this patient. US revealed renomegaly and blood flow decrease in cortical zone in 2 other patients at hospitalization. Segment of 15x15mm with reduced blood flow was revealed in a 15-month-old child on the 7th day of disease. Focal alterations obtained using CT in this patients were regarded as acute focal pyelonephritis.

Multifocal corticomedullary abscess was revealed in a girl of 3 years 11 months of age (pic. 4) on the 5th day of disease using CT; positive dynamics – on the 8th day. No focal alterations were revealed by US.

US revealed sharp increase in size of 1 kidney with no focal alterations in a 7-month-old child who had been ill for 3 weeks, whereas CT and intravenous urography revealed sharp alterations of the right kidney's interstitial tissue and lack of blood flow in it due to multiple abscesses. Diagnosis "renal apostematosa" was confirmed surgically (nephrectomy). US revealed hypoechoic nidus (abscess) of 64x34mm confirmed by the CT data in an increased kidney a 4-year-old girl (pic. 5) on the 11th day of disease.

Lesions of the left kidney were prevalent in the patients described (5 patients); right kidney was affected in 3 patients, both kidneys – in 2 children.

Clinical course

Disease course in the setting of antibacterial treatment was different. 2 children with clear purulent inflammation presentation were operated soon after hospitalization; the subsequent course was smooth. A patient with renal carbuncle and discharge of sensitive *E. coli* and *Enterobacter* received a 2-week therapy course consequently using protected penicillin (amoxycillin + clavulanic acid) and then meropenem + amikacin + linezolid; fever remained in place despite certain improvement in well-being and laboratory indices. The girl had to be operated again, although not because of an additional purulent nidus in the kidney, but because of ruinous infiltration, which appeared after the first operation.

Out of 7 children healed conservatively 4 patients had been receiving ceftriaxone treatment (60-80mg/kg OD); immediate effect (after 1 day) was achieved only in 1 child; 3 patients required drug replacement as high body temperature remained. 3 children had been receiving amoxycillin + clavulanic acid as treatment (90mg/kg per day); they required therapy enhancement in 2-3 days of treatment. Thus, monotherapy is not always efficient in the first 2-3 days of treatment in children with focal lesions, even when started early; this necessitates the antibiotic replacement as the pronounced clinical symptoms remain.

In these patients we used meropenem (50-60mg/kg per day), amikacin (15mg/kg OD) and combinations of them. Replacement of ceftriaxone for meropenem in a 3-month-old child with multiresistant *E. coli* in blood and urine (sensitivity to meropenem was preserved) led to a gradual body temperature decrease on the 5th therapy day. The initial drug was replaced by a combination of meropenem with amikacin in 3 children. Apyrexia with smooth course was obtained within 1-2 days in all cases. The same rapid effect we obtained in 2 patients adding amikacin to the initial combination of amoxycillin + clavulanic acid.

DISCUSSION

Given observations of 10 children showed that focal renal processes are not as rare pathology as is commonly assumed. Our data do not allow stating the hematogenous character of infection in all cases; however, it is most probable, as we may suspect urodynamic disorder in the setting of pyelocaliceal system's duplication only in 1 child. No other purulent nidi formed in any patient. Moreover, bacteremia revealed in 4 out of the 6 examined children indicates the chance of the hematogenous infection.

Clinically, severe bacterial disease/bacteremia may have been suspected in most children on the basis of an acute beginning of the disease, high fever and chill (with no catarrhal syndrome), anorexia. Emesis, which is considered distinctive of pyelonephritis, appeared in 4 children; no patients had dysuric disorders. Renal palpatory tenderness and costovertebral angle pains were typical of children over 4 years of age.

In most children we revealed very moderate leukocyturia, quickly passing over in the setting of antibiotics with the condition severity remaining and absence of other positive effects of the treatment conducted. We assume that it also results from the hematogenous character of renal lesion in the setting of smaller degree of inflammation in urinary tract.

Apart from the severity of clinical manifestations, patients attracted attention by extremely high indices of inflammation markers. We may consider neutrophilic leukocytosis higher than $20x10^9$/l, all the more higher than $30x10^9$/l, a sign that may indicate the purulent process. The same goes for the CRP concentration of more than 100mg/l, all the more higher than 150mg/l. Procalcitonin concentration was less consistent: it was very high (10, 43 and 82.7 ng/ml) in 3 children with proved bacteremia and moderately increased in half of the other children; this necessitates careful interpretation of low indices. At the same time, we cannot confirm the diagnostic value of thrombocytosis, which more often appears in the setting of treatment in case the patient's condition is improving.

Our data demonstrate that ultrasonic alterations that allow suspecting focal purulent process usually appear not earlier than on the 5^{th}-6^{th} day of disease. That is why negative US results should carefully be interpreted given the time that had passed since the disease onset in case there are clinical-laboratorial data in favor of this diagnosis. US did not reveal clear data on the focal process in 5 children examined on the 2^{nd}-4^{th} day of disease, however, they were revealed in 3 patients by the repeated US. We deem reasonable to repeat US every day in case there is no possibility to conduct contrast-enhanced CT.

CT allowed revealing nidi indicating the possibility of a purulent process developing in 9 children. US results were deemed sufficient to enhance antibacterial therapy in 1 child. We may confirm literary data on the possibility of conservative tactics of managing patients and total recovery in case renal corticomedullary nidi do not exceed 30mm in size. 3 children with nidi of 64 and 65 mm in maximal size and with a purulent nidus occupying the whole kidney had to undergo operation. In our practice, only 1 14-year-old adolescent with nidus of 49mm quickly responded to the conservative treatment.

Peculiarity of the process course in the setting of antibacterial therapy (in children with small nidi) is an absence of quick response (within 2-3 days) to monotherapy using ceftriaxone and amoxycillin + clavulanic acid. Moreover, flora of most patients appeared sensitive to these drugs. Monotherapy is usually efficient within 1-2 days at non-complicated URI forms and pyelonephritis. Shift to using amikacin and/or meropenem, which gave rapid body temperature decrease in all cases, was probably excessive in a range of cases (given the flora sensitivity). This tactics is justified both by severe condition of patients and by delay in obtaining antibiogram results and most importantly – by the chance of preventing disease from progressing towards the development of a purulent microbial-inflammatory process in renal parenchyma.

CONCLUSUION

Purulent focal renal processes signify not the ascending infection of urinary organs, but hematogenous infection.

Bacterial inflammation of renal parenchyma should be suspected in case of pronounced changes in clinical-laboratorial indices.

Moderate leukocyturia or leukocyturia, which quickly passes over in the setting of therapy, may be a sign of parenchymatous lesions; in its turn, it does not allow ruling out formation of a renal purulent nidus.

Leukocytosis of more than $30x10^9$/l and CRP level exceeding 150mg/l indicate possibility of a renal purulent process. Low procalcitonin indices do not always correlate with the severity of renal lesion.

Ultrasonic presentation characteristic of focal process sometimes lags in comparison with renal CT.

Conservative management of patients with the CT-confirmed corticomedullary abscesses is possible; however, it depends on their size.

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Table 1. Clinical description of patients

#	Age	Day of disease	t, ^{o}C	Abdominal pain	Emesis
1	15 months	5	40	+	-
2	3 months	3	39.5	?	-
3	9 months	30	39.7	+	++
4	7 years	2	40	-	+
5	14 years	5	40	++	++
6	2 months	4	40	?	-
7	4 years	11	40	++	-
8	3 years 11 months	5	39.5	+	-
9	8 years	2	41	++	++
10	3	3	40	-	-

Table 2. Laboratory description of the material

#	Leukocyturia, per field of vision	Leukocytosis, x10^9/l	CRP, mg/l	Procalcitonin, ng/ml	Urine culture	Hemoculture
1	50	22.56	227.14	2.17	*E. coli*	-
2	417	23.03	213.81	43.58	*Kl. pneumoniae*	*Kl. pneumoniae*
3	500	34	160	10	*Kl. pneumoniae*	*Kl. pneumoniae*
4	1,137	19.9	137.8	3.58	*E. coli*	No growth
5	2	31	111	0.56 (a/b)	- (a/b)	-
6	500	30.08	123.33	1.8	*E. coli*	-
7	25	26.81	77.85 (a/b)	0.39 (a/b)	- (a/b)	- (a/b)
8	20-25	39	200	1.19	- (a/b)	- (a/b)
9	20	27.58	250	82.72	*E. coli*	*E. coli*
10	6 + clusters	41.5	137	2.98	*E. vulneris*	*E. coli*

Note. a/b – in the setting of using an antibacterial drug.

Table 3. Visualization methods

#	Renal US I, day of disease	Renal US II, day of disease	Renal CT, day of disease
1	Renomegaly, 5	Heterogeneous segment, 7	-
2	Renomegaly, 3	Normal blood flow	Nidus in lower pole, 7
3	Renomegaly, concrements, 30	-	Abscess, 30
4	Renomegaly, 2	Hypoechoic segment, 4	Nidus in lower pole, 4
5	Heterogeneous segment, 6	-	Nidi, 6
6	Norm, 5	2 hypoechoic segments, 6	Nidi, 6
7	Renomegaly, abscess, 11	-	Multiple abscesses, 11
8	Renomegaly, nidus, 6	-	Nidus, 6
9	Stripped blood flow, 2	Hypoechoic formations, 4	Carbuncle, 4
10	Pyelocaliceal system's duplication, 3	-	Wedge-shaped hypoechoic segment, 8

Captions to pictures.

Pic. 1. *Patient #4.* Renal computed tomography (CT) of a 7-year-old child P. on the 2nd day of febrile disease. Blood analysis: leukocytes – $19.9x10^9$/l, neutrophils – 76%, CRP – 138mg/l, PCT – 3.6ng/ml, culture – no growth. Urine analysis at hospitalization: leukocytes – 1,137/ml, *Escherichia coli* $>10^5$/ml, sensitive to β-lactams; no leukocyturia after 1 day. Ultrasonic examination (US) on the 2nd day: increased left kidney, parenchyma of increased echogenicity, stripped subcapsular blood flow. US on the 3rd day of disease revealed hypoechoic segment of ca. 14mm in the left kidney's lower pole. CT on the 2nd day of disease: reduced accumulation segment of 20x20mm with indistinct contours could have been determined in the left kidney's lower pole – focal pyelonephritis with abscess formation. The child intravenously received amoxycillin + clavulanic acid, which were 48 hours later replaced by meropenem + amikacin due to the remaining temperature: temperature decreased within 1 day. Subsequent course was smooth. Renal US and CT on the 15th day of disease – pronounced positive dynamics.

Pic. 2. *Patient #6.* Computed tomography (CT) of a 2-month-old child P. hospitalized on the 4th day of febrile disease. Blood analysis: leukocytes – $30.8x10^9$/l, neutrophils – 60%, platelets – $600x10^9$/l, CRP – 123mg/l, PCT – 3.6ng/ml, culture – no growth. Urine analysis at hospitalization: leukocytes cover the whole field of vision, *Escherichia coli* $>10^5$/ml, sensitive to β-lactams; no leukocyturia after 2 days. Ultrasonic examination: no pathology on the 5th day, renomegaly of both kidneys on the 6th day, 2 segments of heterogeneous structure and blood flow alteration were revealed in the right kidney's lower pole. CT (without breath-holding) on the 6th day of disease revealed segments with slight opacification without capsule in the right

kidney's lower segment (ca. 6 and 12 mm) and in the left kidney's posterior segment (10mm). The child intravenously received ceftriaxone; amikacin was added after 48 hours due to the remaining temperature: temperature decrease within 36 hours. Subsequent course was smooth.

Pic. 3. *Patient #9*. Computed tomography (CT) of an 8-year-old girl F. hospitalized on the 2nd day of disease. At 7 months of age she had a urinary tract infection, 1 week before the disease described – acute respiratory viral infection, maxillary sinusitis. Blood analysis: leukocytes – $27.5x10^9$/l, neutrophils – 93%, platelets – $600x10^9$/l, CRP – 125mg/l, PCT – 82ng/ml, culture – no growth. Urine analysis at hospitalization: leukocytes – 1,600/ml, no leukocyturia after 1 day. *Escherichia coli* and enterobacter $>10^5$/ml, sensitive to β-lactams, were separated from urine and blood. Ultrasonic examination: no renal focal diseases revealed on the 2nd and 3rd days of disease; on the 5th day – hypoechoic formation of 65x38x47mm. CT on the 4th day of disease: renomegaly of the left kidney, especially of its outer surface and in the area of upper pole; multiple segments not accumulating contrast agent were revealed in cortical and cerebral substance. The largest segment is of 47x38x66mm; subcapsular fluid collection up to 5mm was noted on this level.

Conservative treatment attempt (consequent 2-week course: 2 days – amoxycillin/clavulanate, then – meropenem + amikacin + linezolid) was ineffective; 2 drainages were conducted as carbuncle cavities were not communicating.

Pic. 4. *Patient #8*. Computed tomography (CT) of a girl of 3 years 11 months hospitalized on the 5th day of febrile disease. Before hospitalization she had intravenously been receiving cefuroxime for 4 days. Blood analysis: leukocytes – $39x10^9$/l, neutrophils – 53%, ESR – 58mm/h, CRP – 219mg/l, PCT – 1.2ng/ml, culture – no growth. Urine analysis at hospitalization: 5 leukocytes per field of vision. Ultrasonic examination on the 6th day revealed renomegaly of the left kidney, stripped blood flow in its lower pole; CT on the same day – presentation of focal pyelonephritis of the left kidney with abscess formation. Given the inefficacy of cefuroxime, the girl was treated by ceftriaxone and amikacin: normalization of temperature and general condition was achieved on the next day.

Pic. 5. *Patient #7*. Computed tomography (CT) of a 4-year-old girl hospitalized on the 11th day of febrile disease accompanied by abdominal pains. Blood analysis: leukocytes – $22x10^9$/l, neutrophils – 83%, ESR – 70mm/h, CRP – 85mg/l, PCT – 0.4ng/ml, culture – no growth. Urine analysis at hospitalization: 25 leukocytes per field of vision, separated *Escherichia coli* $>10^5$/ml sensitive to β-lactams.

Ultrasonic examination: renomegaly of the left kidney, nidus of 64x34mm with hypoechoic zone in the medial segment. CT: multiple different-sized (2-18mm across) formations of fluid density with walls thickened up to 4mm and perirenal spread (total size –

38x45x63mm) and infiltrations of paranephric body with a small amount of fluid can be observed in the projection of the left kidney's superior anterior segment. These formations accumulate contrast only on the wall side; no connection with pyelocaliceal system is noted. Right kidney – no pathology. Multiple para-aortic lymph nodes thickened up to 9mm on the left.

Operation: dissection of carbuncle, plastic restitution of the pyelocaliceal system's integrity in the left kidney. Smooth postoperative course.