doi: 10.67071/3093-9038-2026-25-06-65-72

FEATURES OF STRUCTURAL AND HEMODYNAMIC CHANGES IN THE LIVER IN CHILDREN WITH ACUTE KIDNEY INJURY

T.Sh. Ikromi, Kh.I. Ibodzoda, A. Abdulaliev, A.K. Salomov

State Educational Institution Institute of Postgraduate Education in Healthcare of the Republic of Tajikistan, Dushanbe, Tajikistan

Annotation. Purpose of the study consisted of assessing the structural and hemodynamic changes in the liver in children with AKI.

Material and methods. The study included 26 patients with a clinically verified diagnosis of AKI complicated by acute liver failure (ALF), divided by AKI stage. The control group consisted of 20 healthy children matched for age and gender. A comprehensive noninvasive ultrasound examination was performed, including Doppler ultrasonography of the hepatic vessels and bioimpedance spectroscopy.

Research results showed a progressive and statistically significant increase in the craniocaudal dimensions of the right and left lobes of the liver, as well as the portal vein (PV) diameter, with increasing severity of acute liver failure in all age subgroups. A decrease in the systolic velocity characteristic in the PV and an increase in the congestion index in the PV were noted. The splenic artery diameter (SAD) and arterial blood flow velocity parameters (Vmax, Vdias, Vav) significantly decreased, accompanied by a compensatory increase in the integral resistance indices (PI, IR, SDO). The identified changes were more pronounced in patients with concomitant acute liver failure.

Conclusions. The obtained data indicate significant and progressive structural and hemodynamic changes in the liver in children with AKI, reflecting the systemic nature of renal dysfunction.

Keywords: *acute kidney injury, liver, hemodynamics, children, liver failure, ultrasound.*

Introduction. Acute kidney injury (AKI) remains one of the most pressing and challenging issues in modern pediatrics and intensive care. This condition is characterized by a sudden decline in renal function, leading to disruption of homeostasis and a high risk of developing multiple organ dysfunction, as well as a significant increase in mortality rates [1]. The prevalence of AKI among hospitalized children varies widely, ranging from 5% to 25% according to global statistics [2, 3]. Mortality associated with AKI can reach 15%, and in cases complicated by concomitant multiple organ failure, this figure increases significantly [4, 5].

Despite significant advances in the diagnosis and treatment of AKI, key issues related to timely risk assessment and effective prevention of complications remain unresolved [6]. This situation dictates an urgent need to identify and implement new markers and diagnostic approaches that will improve early detection and management of patients [7]. The understanding that AKI is not simply a local kidney lesion, but a powerful trigger for systemic organ dysfunction, emphasizes the critical importance of a comprehensive approach to the management of such patients. The high risk of multiple organ failure, clearly identified in clinical practice, requires clinicians not only to focus on renal function but also to carefully monitor the condition of other vital organs [8].

Among all the possible complications of AKI, liver damage occupies a special place, as it significantly worsens the prognosis of the disease. Modern studies convincingly demonstrate a close relationship between structural and hemodynamic changes in the liver developing in the context of AKI and the severity of systemic inflammation, general hemodynamic disturbances, and venous congestion. The development of acute liver failure (ALF) in the context of AKI is a critical prognostic factor, increasing mortality in children to 35–40% [9]. This indicator emphasizes that early detection and timely intervention for liver dysfunction are as critical as the management of renal function itself in improving overall outcomes in children with AKI.

However, despite the obvious clinical significance, the mechanisms of development and progression of structural and hemodynamic changes in the liver in children with AKI remain poorly understood. In the literature of recent years, there are only a few studies devoted to the relationship between liver and kidney dysfunction in pediatric practice [10]. These studies are often limited by a small number of observations and, most importantly, do not offer a comprehensive approach to assessing hemodynamics in the hepatic blood flow system. The existing gap in knowledge about the

dynamics of these changes and their relationship with the severity of AKI and the general condition of the patient creates significant difficulties for early diagnosis and the development of effective prevention and treatment strategies.

Thus, a comprehensive study of the structural and hemodynamic parameters of the liver in children with AKI is a highly relevant area of scientific research. Such studies are necessary for the development of more accurate and early diagnostic methods, as well as for the creation of effective approaches to the prevention and treatment of multiple organ failure, which will ultimately improve the survival and quality of life of young patients [11]. Particular attention to the search for new markers and diagnostic approaches, especially non-invasive ones, is key to the transition from reactive treatment to predictive and preventive medicine [4, 12]. This will allow for the timely identification of children at high risk of developing severe multiple organ dysfunction and the initiation of more aggressive interventions before irreversible changes occur.

Purpose of the study. To assess structural and hemodynamic changes in the liver in children with acute kidney injury (AKI).

Material and methods of research. A prospective study was conducted to comprehensively assess the structural and hemodynamic characteristics of the liver in children with AKI.

The study included 26 pediatric patients (aged 3 to 16 years) with a clinically verified diagnosis of acute kidney injury (AKI). This cohort constituted the main study group. All patients in the main group had concomitant manifestations of acute liver failure (ALF). To ensure the objectivity and representativeness of the obtained data, patients in the main group were additionally stratified by the severity of AKI according to generally accepted criteria: 6 patients (23.1%) were diagnosed with stage I of the disease, 9 patients (34.6%) with stage II, and 11 patients (42.3%) with stage III. For a more detailed analysis of age characteristics, all study participants were additionally divided into three age subgroups: 3–7 years, 8–11 years, and 12–16 years.

To comprehensively assess the biometric, structural, and Doppler characteristics of the liver in children with AKI, a multistage ultrasound analysis was performed. The study was conducted at the hospital using standard clinical protocols at the time of admission. The primary diagnostic equipment used was an ALOKA SSD-3500 ultrasound system (Japan), supplemented with a UST-9123 multifrequency convex transducer (operating range 2–6 MHz), as well as a Panasonic Multivisor ADR-2000 system, which ensured high resolution and versatility of the technique. In addition to the basic ultrasound assessment, the study included color duplex scanning of the renal vasculature, including examination of the main renal trunk, interlobar (parenchymal), arcuate, and interlobular arteries. This approach allowed for the analysis of blood flow parameters in various parts of the organ's vascular system, which is especially important in pediatric renal pathology. Color Doppler mapping was used to obtain a clear visualization of the direction and intensity of blood flow.

In the pulsed-wave Doppler mode, key hemodynamic parameters were determined, such as maximum systolic (Vmax), minimum diastolic (Vdias) and average (Vav) blood flow velocities, measured in cm/s. Based on the obtained data, in accordance with the methodological approaches proposed by Nazarenko G.I. et al. (2002), as well as Lopatkina N.A. (1998), integral indicators of the state of the renal vascular bed were calculated: the resistance index (RI), the pulsatility index (PI) and the systolic-diastolic ratio (SDR). These integrative parameters allowed for an objective assessment of changes in vascular hemodynamics in pediatric patients with acute renal pathology.

Due to the diverse etiologies that lead to the development of AKI in children, an additional step was added to the study: calculation of the renal circulating fraction. Renal function in patients who experienced AKI and its associated complications was assessed both early after the illness and at later stages of follow-up, using the previously described diagnostic approaches. Diagnostic criteria outlined by the international NKF/KDOQI-200 guidelines were used to identify CKD.

A multi-level noninvasive monitoring system was used to conduct an in-depth analysis of central, pulmonary, and hepatic hemodynamics in pediatric patients with AKI, depending on the severity of the pathological process and the presence of concomitant organ complications. Monitoring was performed continuously throughout the diagnostic and therapeutic phases, enabling the collection of dynamic data in real time. The Diamant-R hardware and software system was used in the study to quantitatively characterize central hemodynamic parameters and fluid distribution within the body. It was used to perform bioimpedance spectroscopy, developed according to the methodological principles of M.I. Tishchenko, allowing for the collection of objective information on physiological changes.

The use of such regular and comprehensive monitoring is informative for tracking clinical dynamics and assessing the effectiveness of treatment.

Statistical processing of all quantitative parameters was performed using Statistica 10.0 (StatSoft, USA). Mean values (M) and standard errors of the mean (±m) were calculated for all parameters. The statistical significance of differences between the compared groups was assessed using Student's t-test. Differences were considered statistically significant at a probability of error ($p < 0.05$).

Research results. The study revealed significant and progressive changes in liver structural and hemodynamic parameters in children with acute kidney injury (AKI), especially in the presence of concomitant acute liver failure (ALF) or systemic inflammatory response syndrome (SIRS). These changes were closely related to the severity of renal dysfunction and were observed across all age subgroups.

Structural changes in the liver. A study of the main cohort of patients revealed that, as the severity of acute kidney injury worsened, there was a consistent increase in the craniocaudal dimensions of both the right and left lobes of the liver. This increase in size was observed across all age groups studied (3 to 7 years, 8 to 11 years, and 12 to 16 years) and was statistically significantly different from similar parameters in the control group.

The conducted analysis of the dynamics of changes in liver size at different stages of acute kidney injury revealed a clearly expressed relationship between the degree of renal dysfunction and the degree of increase in the craniocaudal size of its lobes. Already at stage I of AKI, a statistically significant increase in this indicator was recorded in all age categories: in patients aged 3–7 years, the sizes of the right and left lobes of the liver increased by 12.3% ($p < 0.05$) and 11.2%, respectively; at the age of 8–11 years – by 12.6% ($p < 0.05$) and 10.8%; among adolescents aged 12–16 years, the increase was 13.5% ($p < 0.001$) and 12.1%. Upon transition to stage II of the pathology, further progression of liver size growth was noted: for children aged 3–7 years, the increase reached 26.7% ($p < 0.001$) and 24.3% ($p < 0.01$), for children aged 8–11 years – 24.8% ($p < 0.05$) and 22.2% ($p < 0.05$), for the adolescent group – 25.2% ($p < 0.001$) and 23.3% ($p < 0.01$). The maximum changes were detected at stage III of the disorder: in children aged 3–7 years, the increase was 34.4% ($p < 0.001$) and 32.6% ($p < 0.001$), in 8–11 years old, the indicators increased by 36.1% ($p < 0.001$) and 33.7% ($p < 0.01$), and in patients aged 12–16 years, by 34.2% ($p < 0.001$) and 31.9% ($p < 0.001$). These results indicate a direct relationship between the severity of renal dysfunction and the extent of morphological changes in the liver in children.

Portal vein hemodynamic parameters. During the analysis of the main blood flow parameters, special attention was paid to the portal vein (PV) diameter in patients with varying degrees of renal dysfunction severity. It was revealed that as AKI progresses, a consistent increase in this parameter is observed in all age groups compared to normal age values. An increase in PV diameter was already recorded at stage I of acute kidney injury: in children aged 3–7 years, it increased by 6.7%, in the group aged 8–11 years–by 7.3%, while in adolescents aged 12–16 years, an increase of 7.8% was observed. Upon transition to stage II of the disease, an even more pronounced expansion of the portal vein diameter was noted: the increase reached 13.4% in children aged 3–7 years, 14.1% in patients aged 8–11 years, and 15.6% among adolescents. In stage III renal dysfunction, the diameter of the hepatic vein increased to 20.2% in children aged 3–7 years, 22.6% in children aged 8–11 years, and 24.7% in adolescents aged 12–16 years. These changes were considered indirect signs of impaired intrahepatic hemodynamics and the development of signs of venous congestion.

The study found that the observed increase in the craniocaudal dimensions of both liver lobes, along with the dilation of the portal vein diameter, was closely associated with changes in key hemodynamic parameters in children with AKI. Specifically, these morphological changes were due to a simultaneous decrease in SVF and an increase in the volumetric blood flow rate in this vessel. This combination of changes led to a significant increase in the IZ VV, and the obtained values for this parameter demonstrated a clear progression with increasing stage of acute kidney injury–from stages I to III in all age groups of the pediatric population studied.

The conducted quantitative analysis of portal vein SCC demonstrated a pronounced decrease in this indicator as the clinical picture of acute kidney injury becomes more complex in children of different age groups. Already at stage I of the disease, a reliable decrease in portal vein SCC was observed: in children aged 3–7 years, the decrease was 15.6% ($p < 0.05$), in the age group 8–11 years – 18.7% ($p < 0.01$), and among adolescents aged 12–16 years – 21.4%. As the disease progressed to stage II, this indicator decreased even more significantly, reaching 28.7% ($p < 0.01$), 32.5% ($p < 0.001$), and 36.3% ($p < 0.001$) for the indicated age categories. During the development of the most

severe stage III of AKI, the maximum decrease in the vascular vascular resistance was observed, amounting to 40.4% ($p < 0.001$) in patients aged 3–7 years, 38.8% ($p < 0.001$) in children aged 8–11 years, and 41.2% ($p < 0.001$) in adolescents. The obtained results indicate progressive dilation and impaired vascular responses in children against the background of increasing renal dysfunction.

A study of the BV's blood cell count revealed mixed changes. At stage I, a significant decrease in this indicator was recorded: by 12.2% ($p < 0.001$), 32.7% ($p < 0.001$), and 43.8% ($p < 0.001$) in the age groups 3–7 years, 8–11 years, and 12–16 years, respectively. At the same time, at stage II, an increase in the total blood cell count by 7.9% ($p < 0.01$), 19.3% ($p < 0.001$) and 30.6% ($p < 0.001$) was observed, and at stage III – by 21.0% ($p < 0.001$), 4.6% ($p < 0.001$) and 17.8% ($p < 0.001$), respectively, by age category.1 The indicators of the IZ VV significantly increased ($p < 0.001$) in children with AKI and OPEF both with increasing age and with increasing degree of organ dysfunction – from stage I to stage III.

Splenic artery hemodynamic parameters. The study of the splenic artery (SAP) parameters, in particular changes in their diameter in children of the main group, revealed a reliable decrease in this parameter compared to normal age values in all subgroups. At stage I of AKI, the SAP diameter decreased by 10.2% ($p < 0.001$) in children aged 3–7 years, by 9.1% ($p < 0.01$) in children aged 8–11 years, and by 12.4% ($p < 0.001$) in adolescents aged 12–16 years. At stage II of AKI, the decrease was by 16.2% ($p < 0.001$), 14.7% ($p < 0.001$), and 16.2% ($p < 0.001$), respectively. At stage III – by 25.4% ($p < 0.001$), 23.2% ($p < 0.001$) and 21.3% ($p < 0.001$), respectively, by age category.

Analyzing the obtained hemodynamic data, it can be concluded that a gradual decrease in the diameter of the hepatic artery, which becomes increasingly pronounced with increasing severity of liver and kidney disease, is a significant factor in the disruption of various arterial blood flow velocity parameters (Vmax, Vdias, Vav). The accompanying reduction in both systemic and intrahepatic blood flow significantly worsens the microcirculatory disorder. As a result of the deterioration in blood flow, key biochemical and detoxifying functions of the liver are impaired, and the ability of other organs and systems to adapt to pathological changes is significantly reduced, further complicating the disease course in this group of patients.

A study of blood flow parameters in the hepatic arteries (HA) in children with acute kidney injury (AKI) and acute liver injury (ALI) revealed a significant decrease in all key velocity parameters compared to age-appropriate norms in the control group. This trend was observed across all age categories studied.

In early stage I of AKI, a decrease in maximum systolic blood flow velocity (Vmax) was recorded, ranging from 14.8% ($p < 0.05$) in children aged 3–7 years to 18.9% ($p < 0.01$) in 8–11-year-olds and 22.6% ($p < 0.001$) in adolescents aged 12–16 years. Similarly, minimum diastolic velocity (Vdias) decreased by 18.4% ($p < 0.01$), 21.6% ($p < 0.001$) and 23.5% ($p < 0.001$), respectively, and mean blood flow velocity (Vav) – by 17.3% ($p < 0.01$), 21.1% ($p < 0.001$) and 22.4% ($p < 0.001$) in the same age subgroups.

As the patients progressed to stage II of AKI, a more pronounced suppression of hemodynamic parameters was observed: the decrease in Vmax reached 20.6% ($p < 0.01$), 25.7% ($p < 0.001$), and 29.3% ($p < 0.001$); Vdias values decreased by 26.5% ($p < 0.001$), 28.9% ($p < 0.001$), and 30.6% ($p < 0.001$). The average blood flow velocity (Vav) decreased by 23.0% ($p < 0.01$), 28.1% ($p < 0.001$), and 27.4% ($p < 0.001$) with increasing age.

The maximum decrease in all studied parameters was observed at stage III of AKI, where for the age groups of 3–7, 8–11 and 12–16 years, the decrease in Vmax was 28.5%, 34.2% and 38.7% ($p < 0.001$), respectively. Vdias decreased in these groups by 34.9%, 37.3% and 38.6% ($p < 0.001$), and the average velocity Vav – by 31.0%, 36.2% and 38.3% ($p < 0.001$). These results indicate a progressive impairment of arterial hemodynamics of the liver, characteristic of the severity of the pathological process, regardless of the patient's age.

A comprehensive analysis revealed that pediatric patients with AKI and acute liver disease exhibited a significant decrease in blood flow velocity parameters in the hepatic artery (Vmax, Vdias, Vcp). These circulatory disturbances were accompanied by an increase in integral hemodynamic indices–PI, IR, and SOD–indicating the development of a compensatory response in the vascular system. In subsequent stages of AKI and APHF progression, increasingly significant impairment of vascular tone, weakening of the adaptive potential of the vascular wall of the hepatic artery, and worsening impairment of intrahepatic arterial microcirculation occurred. The severity of these abnormalities directly correlated with the increasing severity of organ dysfunction, emphasizing the connection between liver pathology and hemodynamic changes at various stages of the disease.

An in-depth study of PI dynamics in children with acute renal and hepatic impairment revealed a significant increase in this parameter across all age groups as the pathological process progresses.

This increase in PI indicates a decrease in the compensatory capacity of the vascular wall in the patients studied.

Upon transition to stage I of AKI, the increase in PI value was 4.3% in children aged 3–7 years, 5.1% among the 8–11 year old group, and reached 9.3% in adolescents aged 12–16 years ($p < 0.05$). At stage II of pathological changes, these indicators continued to increase: the increase in PI reached 7.7% ($p < 0.05$) in the younger age group, 5.7% in children aged 8–11 years, and already 10.2% ($p < 0.01$) in adolescents. The maximum PI values were recorded at stage III of the disease, where the increase was 7.7% ($p < 0.05$) in children aged 3–7 years, 7.6% ($p < 0.05$) for the 8–11 year old group, and 8.5% ($p < 0.05$) among patients in the older age category.

During the study, IR, used to assess the vascular tone of the hepatic artery, arterioles, and capillaries of the microcirculatory bed, demonstrated a clear increase in children with AKI and acute liver dysfunction in all age groups. Already at stage I of AKI, a tendency towards an increase in IR was noted: in children aged 3–7 years and adolescents aged 12–16 years, the indicator increased by 1.5%, and by 3.0% in the group aged 8–11 years. At stage II of the pathological process, a further increase was observed: the indices increased by 3.0% in children of both younger groups and by 2.9% in adolescents. The most stable increase values were also recorded at stage III of the disease: 1.5% for the age groups 3–7 and 12–16 years, as well as 3.0% for patients aged 8–11 years.

A study of the systolic-diastolic ratio (SDR) dynamics in children with acute renal and hepatic dysfunction revealed a clear upward trend in this indicator across all age subgroups studied, as organ pathology worsens. The increase in SDR is interpreted as reflecting increased vascular resistance, loss of arterial wall elasticity, and weakening of the adaptive mechanisms of the hepatic arterial bed as the severity of dysfunction increases.

In stage I of acute kidney injury, the SOD level increased by 4.5% in the group of children aged 3–7 years, by 3.7% in patients aged 8–11 years, and by 2.5% among adolescents aged 12–16 years. Upon transition to stage II, the value of this indicator increased by 12.3% ($p < 0.001$), 5.0%, and 3.1% in the corresponding age categories. The most pronounced change in SOD was observed at stage III: in the age group of 3–7 years, the increase was 9.6% ($p < 0.001$), for children aged 8–11 years – 5.3%, and among adolescents aged 12–16 years – 0.9% (Table 1). These results demonstrate a progressive deterioration in vascular reactivity and elasticity of the arterial bed of the liver in pediatric patients as organ failure worsens.

The identified pathological changes are associated not only with severe impairments of both arterial and venous blood supply, but also with direct damage to the parenchymal tissues of the corresponding organs. Taken together, these factors predetermine a more severe course of the disease in these patients, accompanied by a significant risk of developing multiple organ dysfunction syndrome. It should be emphasized that the development of multiple organ dysfunction syndrome is directly associated with increased mortality in this patient population.

The data presented in Table 1 demonstrate the complex nature of the detected abnormalities. The combination of liver enlargement with signs of venous congestion (vasodilation, increased IZ vasodilation) and arterial hypoperfusion (sparse stenting, decreased arterial blood flow velocity) suggests a dual hemodynamic impact on the liver. This means that the liver in AKI is severely stressed by both obstructed venous outflow and insufficient arterial blood flow. This complex pathophysiological picture likely contributes significantly to hepatocyte damage and overall liver dysfunction, necessitating therapeutic strategies that address both aspects of hepatic blood flow.

The progression of structural and hemodynamic changes in the liver as AKI worsens suggests that these parameters may serve as potential prognostic markers. Measurements obtained using noninvasive ultrasound and Doppler imaging techniques make them attractive for use as real-time biomarkers. They may help identify children at increased risk of developing severe multiorgan dysfunction, allowing for timely initiation of more intensive interventions and improved outcomes.

Of particular note is the observation that the portal vein volumetric flow characteristic (PVVC), which initially decreased in stage I, then increased in stages II and III of AKI, despite general signs of congestion and decreased arterial inflow. This apparent contradiction may indicate a complex compensatory or maladaptive mechanism. For example, it may result from increased splanchnic blood flow due to systemic vasodilation or shunting, which, despite increasing volume, does not adequately improve perfusion due to increased intrahepatic resistance. Understanding this phenomenon requires further research, as it may reflect a critical shift in systemic and hepatic circulation that impacts the overall prognosis.

Discussion of the research results. Our study demonstrated a progressive increase in the craniocaudal dimensions of the right and left liver lobes in children with AKI, particularly in those

Table 1. Structural and hemodynamic parameters of the liver in children at different stages of acute kidney injury complicated by organ dysfunctions

Indicators	Age in years	Control group	Group 1 26 (49.1%) AKI and ALI,		
			Stage 1, n=6	Stage 2 n=9	Stage 3, n=11
Cranio-cau-distal size of PD, mm	3-7	73,6±2,3	93,5±2,8***	106,4±6,2***	124,8±7,9***
	8-11	89,3±2,8	112,3±4,6***	127,4±5,5***	147,8±6,3***
	12-16	105,1±2,8	128,5±3,6***	145,6±4,2***	165,4±4,4***
Cranio-co-distant LD size, mm	3-7	48,0±2,4	60,1±3,3**	68,5±3,7***	80,1±4,1***
	8-11	63,6±3,2	78,4±3,2**	89,4±3,7***	103,9±4,6***
	12-16	72,6±3,4	87,5±4,1*	98,9±4,9***	112,7±5,0***
Diameter of the explosive, mm	3-7	4,1±0,5	4,6±0,6	5,1±0,4	5,4±0,6
	8-11	5,5±0,6	6,3±0,5	6,9±0,7	7,5±0,7
	12-16	7,0±0,6	8,1±0,5	9,0±0,7*	9,8±0,6**
SSC in BB, cm/s	3-7	8,6±0,5	7,3±0,7	6,1±0,5**	5,3±0,4***
	8-11	11,7±0,7	9,5±0,6*	7,9±0,4***	6,9±0,3***
	12-16	15,1±0,8	11,9±0,6**	9,6±0,5***	7,9±0,5***
OSK in BB, ml/min	3-7	113,5±8,6	99,7±5,2	122,5±6,3	137,3±7,3*
	8-11	277,8±13,4	186,9±8,6***	224,2±8,9**	264,9±9,3
	12-16	550,0±28,5	309,0±12,6***	381,5±12,7***	452,3±13,3**
FROM BB, cm s	3-7	0,02±0,01	0,09±0,01***	0,23±0,02***	0,27±0,02***
	8-11	0,02±0,01	0,26±0,02***	0,31±0,02***	0,37±0,03***
	12-16	0,03±0,01	0,32±0,03***	0,42±0,04***	0,50±0,04***
SPA diameter, mm	3-7	2,4±0,02	2,15±0,04***	2,0±0,05***	1,80±0,03***
	8-11	3,1±0,03	2,82±0,04***	2,64±0,03***	2,38±0,04***
	12-16	3,8±0,04	3,33±0,04***	3,18±0,03***	3,0±0,04***
Vmax, cm/s in SPA	3-7	69,0±2,4	58,8±3,2*	54,8±2,9**	49,3±2,7***
	8-11	63,3±1,8	51,3±2,8**	47,0±2,7***	41,7±2,3***
	12-1 6	58,4±1,0	45,2±2,4***	41,3±2,1***	35,8±2,0***
Vdias., cm/s in SPA	3-7	23,6±0,9	19,3±0,8**	17,3±0,7***	15,4±0,6***
	8-11	21,0±0,7	16,5±0,7***	14,9±0,6***	13,2±0,6***
	12-16	18,0±0,5	13,8±0,6***	11,5±0,5***	11,1±0,5***
Vavg., cm/s in SPA	3-7	38,7±1,1	32,5±1,3**	29,8±1,1***	26,7±1,0***
	8-11	35,6±1,1	28,1±1,0***	25,6±0,9***	22,7±0,8***
	12-16	31,3±0,9	24,3±0,6***	22,1±0,7***	19,3±0,6***
PI in SPA	3-7	1,17±0,02	1,22±0,04	1,26±0,04*	1,26±0,03*
	8-11	1,18±0,03	1,24±0,03	1,25±0,03	1,27±0,03*
	12-16	1,18±0,04	1,29±0,03*	1,30±0,02**	1,28±0,02*
IR in SPA	3-7	0,66±0,02	0,67±0,01	0,68±0,02	0,67±0,02
	8-11	0,66±0,03	0,68±0,02	0,68±0,02	0,68±0,02
	12-16	0,68±0,02	0,69±0,02	0,70±0,02	0,69±0,03
SDO PA	3-7	2,92±0,04	3,05±0,06	3,28±0,05***	3,20±0,06***
	8-11	3,0±0,065	3,11±0,05	3,15±0,05	3,16±0,04
	12-16	3,2±0,07	3,28±0,05	3,30±0,05	3,23±0,04

Note: value *p <0.05; **p<0.01; ***p<0.001 reliability of indicators in relation to the control group

with concomitant acute hepatic insufficiency, across all age subgroups. This hepatomegaly became more pronounced with increasing AKI stages. Concurrently, we observed a consistent dilation of the portal vein (PV) diameter, indicating impaired intrahepatic hemodynamics and the development of venous congestion. These structural changes were further confirmed by a decrease in the portal vein blood flow (PVBF) and portal vein blood flow (PVBF), while the portal vein occlusion (PVI) significantly increased. Taken together, these data indicate compromised portal circulation, leading to passive congestion in the liver parenchyma.

Furthermore, our study revealed a significant decrease in the hepatic arterial diameter and a marked reduction in its arterial blood flow velocities (Vmax, Vdias, Vavg). This arterial hypoperfusion was accompanied by a compensatory increase in PI, IR, and SDO in the hepatic arterial system. These changes indicate an increase in vascular resistance and a decrease in arterial compliance in the hepatic arterial system, potentially contributing to liver ischemia and further impairment of liver function. The observed deterioration in both arterial and venous hepatic blood flow indicates a complex interplay of systemic and local factors contributing to liver injury in the context of AKI.

It is noteworthy that these structural and hemodynamic abnormalities were more pronounced in children with AKI combined with AHF (group one) compared to children with AKI and SIRS (group two), which emphasizes the serious impact of concomitant liver failure on the integrity of the liver vessels. Impaired hepatic blood flow in the examined patients did not represent a separate pathological process, but reflected a complex set of systemic disorders, including disruptions of central hemodynamics, deterioration of cardiac function and changes in pulmonary circulation, and also accompanied various stages of impaired MV. Such multilevel interaction is confirmed not only by our results, but is also comparable with modern literature data, which emphasize a pronounced association between AKI and the state of liver function in children. Previous studies have shown that AKI can lead to various systemic complications, including liver injury, often referred to as "hepatorenal syndrome" in severe cases [13]. Our observation of hepatomegaly and portal vein dilation echoes findings in adult patients with AKI, where similar changes are associated with volume overload and right heart dysfunction, leading to hepatic venous congestion [14]. The progressive nature of these changes with increasing AKI severity, as demonstrated in our study, is consistent with the understanding that persistent renal impairment exacerbates systemic complications.

The decrease in hepatic artery diameter and arterial flow velocities, coupled with increased resistance indices, suggests an adaptive or maladaptive response of the hepatic arterial system to altered systemic hemodynamics in AKI. This is supported by studies indicating that systemic inflammation and endothelial dysfunction, often present in AKI, can lead to vasoconstriction in various vascular territories, including the hepatic circulation [15]. While some studies in critically ill adults report increased hepatic artery blood flow as compensatory mechanisms during systemic shock (eg, early sepsis), our data in pediatric patients with AKI suggest a predominant vasoconstrictive response, possibly reflecting a later stage of injury or a unique pediatric physiologic response [16]. Further studies are needed to define these nuances in responses across different etiologies and stages of AKI in children.

Although our results are generally consistent with the literature on systemic organ involvement in AKI, the observed initial decrease in IR in some subgroups followed by an increase may be unexpected. This biphasic response may reflect early compensatory vasodilation aimed at maintaining liver perfusion in the face of reduced systemic blood flow, followed by a later, dominant phase of vasoconstriction caused by inflammatory mediators, sympathetic hyperactivity, or advanced stages of endothelial dysfunction [17]. It is also possible that these patterns were influenced by differences in the etiology of AKI (e.g., primary kidney disease versus sepsis-induced AKI) or age-related hemodynamic responses in children. The relatively small sample size in each age and severity subgroup may also affect the statistical significance of subtle early changes.

Our study also has several limitations. The cross-sectional nature of the initial assessment at admission limits our ability to establish a direct causal relationship between the onset of AKI and the observed liver changes; follow-up was conducted, but the initial assessment is key. Although we examined the impact of AKI and SIRS, other confounding factors, such as comorbidities, nutritional status, and specific pharmacological interventions, may also have influenced liver hemodynamics and were not fully accounted for in the analysis. Furthermore, the use of ultrasound, while noninvasive and highly valuable, has inherent limitations in fully quantifying liver microcirculation or assessing parenchymal injury at the cellular level. Future studies using advanced imaging techniques, such as elastography, or biomarkers of liver injury may provide additional insights.

Our results highlight the importance of early and comprehensive monitoring of structural and hemodynamic liver parameters in children with AKI, especially given the high risk of developing MOD and associated mortality. Observed changes in portal vein diameter and arterial flow characteristics may serve as early noninvasive biomarkers for predicting the severity of liver injury and promptly initiating therapeutic interventions. However, to fully realize this potential, further studies are needed to validate ultrasound data with biochemical markers of liver injury and, where ethically permissible, histological assessment. This will allow for the establishment of clear cutoff values for these noninvasive markers and their integration into clinical protocols for prognosis and risk stratification.

Conclusions:

1. Acute kidney injury (AKI) in children leads to significant and progressive structural and hemodynamic changes in the liver, manifested by an increase in the craniocaudal size of the liver lobes and portal vein dilation, as well as changes in the velocity and resistivity of portal and arterial blood flow. The severity of these changes directly correlates with the severity of renal dysfunction and the presence of concomitant organ complications, such as acute liver failure and systemic inflammatory response.

2. The identified disturbances in intrahepatic hemodynamics and liver morphology are not isolated manifestations, but rather an integral part of multiple organ dysfunction syndrome (MODS), characteristic of severe acute kidney injury in children. These changes reflect interdependent and mutually aggravating pathophysiological mechanisms of disruption of central and regional circulation, microcirculation, and homeostasis, highlighting the need for a comprehensive approach to diagnosis and treatment of this patient population.

Literature/References

1. Savenkova ND. Pediatric classifications and epidemiology of acute kidney injury. Russian Bulletin of Perinatology and Pediatrics. 2018;63(5):36-42.Menon S, Tarrago R, Carlin K, Wu H, Yonekawa K. Acute kidney injury in pediatric critical care: epidemiology, risk factors, and outcomes. Pediatr Crit Care Med. 2021;22(11):945-956.

2. Hoste EA, Kellum JA, Selby NM, et al. Global epidemiology and outcomes of acute kidney injury. Nat Rev Nephrol. 2018;14(10):607-625.

3. Vanmassenhove J, Kielstein J, Jörres A, Biesen WV. Management of patients at risk of acute kidney injury. Lancet. 2017;389(10084):2139-2151.

4. Kellum JA, Romagnani P, Ashuntantang G, Ronco C, Zarbock A, Anders HJ. Acute kidney injury. Nat Rev Dis Primers. 2021;7(1):52.

5. Смирнов АВ, Румянцев АШ.. Острое повреждение почек. Часть II. Нефрология. 2020;24(2):96-133.

6. Makris K, Spanou L. Acute kidney injury: diagnostic approaches and controversies. Clin Biochem Rev. 2016;37(4):153-175.

7. Ronco C, Bellomo R, Kellum JA. Acute kidney injury. Lancet. 2019;394(10212):1949-1964.

8. Ostermann M, Lumlertgul N, Jeong R, See E, Joannidis M, James M. Acute kidney injury. The Lancet. 2025;405(10474):241-256.

9. Raina R, Chakraborty R, Sethi SK, Bunchman T. Kidney replacement therapy in pediatric acute kidney injury: Indications and modalities. Pediatr Res. 2020;88(3):323-331.

10. Yakovleva N.A., Ignatyeva E.G. Assessment of the structural and functional state of the liver in acute kidney diseases in children. Pediatrics. 2021;(5):102-108.

11. Smirnova NN, Galkina OV, Novikova VP, Prokopyeva NE. Modern biomarkers of kidney injury in pediatrics. Nephrology. 2019;23(4):112–118.

12. Liu PMF, de Carvalho ST, Fradico PF, Cazumbá MLB, Campos RGB, Simões E Silva AC. Hepatorenal syndrome in children: a review. Pediatr Nephrol. 2021 Aug;36(8):2203-15.

13. Beaubien-Souligny W, Galarza L, Buchannan B, Lau VI, Adhikari NKJ, Deschamps J, et al. Prospective Study of Ultrasound Markers of Organ Congestion in Critically Ill Patients With Acute Kidney Injury. Kidney Int Rep. 2023 Dec 29;9(3):694-702.

14. Manrique-Caballero CL, Del Rio-Pertuz G, Gomez H. Sepsis-Associated Acute Kidney Injury. Crit Care Clin. 2021 Apr;37(2):279-301.

15. Zhao NB, Chen Y, Xia R, Tang JB, Zhao D. Prognostic value of ultrasound in early arterial complications post liver transplant. World J Gastrointest Surg. 2024 Jan 27;16(1):13-20.

16. Brazzelli M, Aucott L, Aceves-Martins M, Robertson C, Jacobsen E, Imamura M, et al. Biomarkers for assessing acute kidney injury for people who are being considered for admission to critical care: a systematic review and cost-effectiveness analysis. Health Technol Assess. 2022;26(7):1–286.