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THE ROLE OF ACUTE KIDNEY INJURY IN CHRONIC KIDNEY DISEASE – CLINICAL CASE

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SUMMARY

Key words: acute kidney injury, chronic kidney disease, renal recovery, hemodialysis.
Acute kidney injury (AKI) is described as a spectrum of abruptly compromised renal functions that result in impaired balance of fluid, electrolytes, and waste products. It is recognized as an increasingly common cause of morbidity, mortality and long-term renal sequelae in children. Studies report that survivors of pediatric AKI are at risk for chronic kidney disease (CKD) including hypertension and end stage renal disease (ESRD).

РЕЗЮМЕ

РОЛЬ ОСТРОГО ПОВРЕЖДЕНИЯ ПОЧЕК ПРИ ХРОНИЧЕСКОЙ БОЛЕЗНИ ПОЧЕК – КЛИНИЧЕСКИЙ СЛУЧАЙ

Ключевые слова: острое повреждение почек,хроническая болезнь почек,восстановление почечной функции, гемодиализ.
Острое повреждение почек (ОПП) описывается как внезапное снижение почечной функции, приводящее к водно-электролитному дисбалансу и накоплению токсических продуктов обмена в крови. У детей ОПП признана частой причиной заболеваемости, смертности и долговременных почечных осложнений. Исследования показывают, что выжившие дети после ОПП подвергаются риску развития хронической болезни почек (ХБП), включая артериальная гипертензия и терминальную ХБП.

REZUMAT

ROLUL LEZIUNII RENALE ACUTE ÎN BOALA RENALĂ CRONICĂ – CAZ CLINIC

Cuvinte-cheie: leziune renală acută, boală cronică renală, recuperarea funcției renale, hemodializă.
Leziunea renală acută (LRA) este descrisă ca scăderea bruscă a funcției renale, rezultând dezechilibrul hidroelectrolitic și acumularea produselor reziduale. La copii, LRA este recunoscută drept cauză frecventă de morbiditate, mortalitate și sechele renale pe termen lung. Studiile raportează că copiii supraviețuitori ai LRA prezintă risc de boală cronică renală (BCR), inclusiv, hipertensiune arterială și BCR terminală.

Introduction.
Acute renal injury (AKI) represents an abrupt decrease in renal function, resulting in reduced glomerular filtration rate, syndrome of nitrogen retention, and hydroelectrolytic disorders [2]. Acute renal dysfunction has been defined differently in various studies.
According to Grandham J. is a syndrome characterized by a relative decline of renal function, with water, crystalloid solutions and nitrogenous metabolites retention in the body. Blantz C. R. defined it as a clinical syndrome marked by a relatively abrupt reduction of the glomerular filtration rate (GFR)[1].The experimental data complete the metabolic and molecular processes, as from the renal tubular cells and their glomerular consequences. In acute renal dysfunction, two concepts are developed: the toxic genesis and ischemia-reperfusion injury. Ischemic factor is claimed to be decisive in the genesis of the renal injury. The incidence of AKI in children is constantly increasing. In the AWARE study, the incidence of AKI was 26.9%, and the incidence of severe AKI (KDIGO stage 2 or 3) - 11.6% [6]. In children, AKI is associated with high mortality or long-term sequelae. In severe cases, mortality constitutes 30-50% of cases [2,3]. Over 47 - 60% of survivors present signs of progression to chronic kidney disease (CKD): renal fibrosis, high blood pressure, proteinuria or decreased GFR, and 5-9% develop the ESRD [2,3]

The aim of this study is to present a clinical case characterized by the evolution of AKI to CKD with the subsequent recovery of the renal function after the chronic hemodialysis therapy individually adapted to the given case.

Materials and methods.

We present the case of a 9-year-old boy from the rural area, who is transferred to a tertiary pediatric clinic with: pronounced edematous syndrome, rare voiding, quantitatively reduced, accompanied by regurgitation, nausea, repeated vomiting (7- 8 times / 24 hours), abdominal pain, moderate headache, irritability, emotional lability.

From the history of present illness we note that 9 days earlier, the child had dyspeptic digestive syndrome with repeated vomiting, abdominal pain, diarrhoea stools. The child was treated at home with oral rehydration solution and the enterosorbents.

On examination, the patient is miserable, fussy, toxic, with oedema, and high blood pressure up to 142/80 mmHg. The skin is pale-grey, dry, with generalized pastosity, with pronounced periorbital oedema, cold lower extremities with soft oedema. On auscultation, there are no crackles or wheezes, but the pulmonary respiration is attenuated in the right basal segments. Urine output - 0.28 ml/min/h.

Results.

CBC showed anemia (Hb – 103 g/l, RBC – 3.1 x 10^{12}/l), leucocytosis (16.7 x 10^{9}/l) with left shift (bands – 11%), elevated ESR (26 mm/h).

Blood biochemical examination highlighted the syndrome of nitrogen retention (serum creatinine - 353 µmol / l, BUN - 16.5 mmol/l, eGFR - 18.2 ml/min/1.73 m^2), hyponatremia (125 mmol/l), hypocalcemia - 1.9 mmol/l, also syndrome of hepatic cytolysis (ALT - 135 IU/l, AST -176 IU/l). The urinalysis revealed proteinuria (4 g/l), hematuria (8-13 hpf). Kidney ultrasound exam showed normal-sized kidneys with increased echogenicity and blurred outline. Kidney pelvis are not dilated. Empty bladder. Abdominal ultrasound: moderate hepatomegaly. Ultrasound of the pleural and abdominal cavities: paravesical - a liquid collection about 30 cm^3; free fluid in the right pleural cavity.Negative stool tests.

Considering the complaints, the history of illness, the clinical and laboratory examination was established the diagnosis: Acute tubulointerstitial nephritis. Acute renal injury, stage III. Anuria. Ascites. Pleurisy on the right. Hypertension, stage II. Toxic hepatitis.

Due to persistence of repeated vomiting, refractory hypertension to treatment, syndrome of nitrogen retention and anuria we started supportive treatment with antihypertensive, vasoprotective and antiaggregant therapy, and the therapy for renal function replacement by hemodialysis.

During the hospitalization, about a month and a half, the child supported 18 hemodialysis sessions, with the improvement of the general state, but with a weak positive dynamic in clinical and paraclinical parameters: blood pressure - 128/90 mmHg, diuresis - 0.8 - 0.91 ml/kg/h, serum creatinine - 535 µmol/l, BUN - 16.5 mmol/l, eGFR - 12 ml/min/1.73 m2. We decided to continue the renal replacement therapy by hemodialysis in outpatient settings.

The patient underwent renal replacement therapy by intermittent hemodialysis, 2 sessions per week, supported by antihypertensive, vasoprotective and antiaggregant treatment, with recombinant human erythropoietin, calcium preparations and vitamin D3, antioxidants with a periodical evaluation of renal function.

At 6th month of intermittent hemodialysis, the child was re-evaluated with the following clinical and paraclinical indices: blood pressure - 110/70 mmHg, diuresis - 1.26 ml/kg / h, serum creatinine - 105 µmol/l, urea - 8.5 mmol/l, eRFG - 61.6 ml/min/1.73 m2. Considering the normal diuresis and the effective control of blood pressure, renal replacement therapy by intermittent hemodialysis was stopped, but the patient continued the renoprotective and antioxidant treatment, .

Over 1 year and 8 months after the onset of the disease the blood pressure is about 105/70 mmHg, diuresis - 1.75 ml/kg/hour, serum creatinine -72 µmol/l, urea - 5.2 mmol/l, and eGFR - 92 ml/min/1.73 m2. The child is under the pediatric nephrologist's surveillance, with monitoring of renal function.

Discussions.

AKI is defined as persistence of renal dysfunction for up to 7 days after renal injury. Traditional, AKI is classified into three distinct categories depending on the level of injuring factor. Thus, are defined pre-renal, renal or intrinsic and postrenal AKI [5]. At the same time, AKI is preceded and followed by a series of events that define acute renal disease, lasting between 7 and 90 days from acute renal injury. Actually, AKI is a self-limiting process with subsequent morphofunctional restoration. However, in severe cases, pronounced ischemia and reperfusion injury can lead to interstitial fibrosis, tubular atrophy and long-term renal dysfunction, respectively.

The persistence of renal dysfunction over a longer period determines the progression to CKD of different degrees [4,7,9]. Numerous studies and observations show that a single episode of AKI is not sufficient for progression to CKD, and two scenarios are possible: 1. repeated episodes of AKI, which cumulatively lead to CKD or 2. the overlap of an episode of AKI with CKD in the initial stages and its progression [8].

In our case, given the lack of information about the initial levels of serum creatinine and about a renal impairment, is difficult to highlight one of the scenarios. Renal

function can be fully restored, with GFR > 90 ml / min and RFR> 30 ml / min, or partially - GFR > 90 ml / min and RFR <30 ml / min. The third option is progression to CKD with GFR <60 ml/min [10].

Following the history of our patient's disease, we can see a complete recovery of renal function, including normal levels of serum creatinine with RFG of 92 ml/min/1,73 m^2 and restoration of adequate diuresis [6].

Another important moment to mention is the effective blood pressure control, as high blood pressure is an independent factor for CKD progression.

Theoretically, the recovery of renal function can occur at any time interval from the occurrence of acute renal injury. However, it is considered that its absence for a period of more than 90 days, during renal replacement therapy, is an independent prognostic factor for progression to CKD [4,9]. Therefore, cases with recovery of renal function after long-term hemodialysis require careful monitoring from the perspective of long-term sequelae [4,11].

Thus, the patient follows a permanent follow-up by the pediatric nephrologist with the assessment of blood pressure, urinalysis and serum creatinine levels, GFR, as well as the assessment of risk factors for CKD recurrence and progression.

Conclusions.

The association between AKI and CKD is complex and multidirectional, because CKD can evolve after undergoing unrecovered AKI, it can also appear again after recovery from AKI or can be a progression of pre-existing CKD.Thus, we reiterate the necessity of careful monitoring after recovery of renal function following intermittent hemodialysis from the perspective of long-term sequelae.

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