Acute renal failure (pediatrics)

Acute renal failure (ASL) is a sudden deterioration of kidney function that has not been seriously damaged before. The laboratory finding is an increase in non-protein nitrogen in the serum (urea, creatinine, uric acid). Urinary excretion disorder (oliguria, anuria) is often present. Hyponatremia, hyperkalemia, hypocalcemia, and metabolic acidosis develop. During sonographic examination, both kidneys are enlarged (in chronic renal failure they are "shriveled"), the corticomedullary differentiation is often obliterated. More recently , the term *acute kidney* **injury** is used .

The so-called RIFLE criteria are used for adults, modified as the so-called pRIFLE criteria for children. This classification evaluates the decrease in diuresis and the dynamics of serum creatinine (calculated creatinine clearance according to the Schwartz formula).

- **RIFLE** (risk, injury, failure, loss, end);
- **R** : mild forms of damage to the renal parenchyma;
- **F** : renal failure requiring elimination treatment, reversible condition;
- **L** : loss of kidney function for more than 4 weeks;
- **E** : loss of renal function for more than 3 months; dialysis-transplant program candidates; *end stage renal disease* .

Impairment of renal function in newborns: rapid decrease in glomerular filtration rate, diuresis < 1 ml/kg/h for min. 24 hours, creatinine > 80 µmol/L (or > than maternal values), urea > 10 mmol/L, tendency to hyperkalemia.

Etiology

The etiology is multifactorial.

Prerenal type of ASL

- pathophysiology: reduction of blood pressure on the glomerular capillaries (below the normal approx. 30 mm Hg) due to a decrease in systemic pressure and the so-called emergency distribution;
- etiology: acute disorder of water and ion management – dehydration during diarrhea, vomiting, severe infection, shock state ;
- development of ASL and risk of metabolic derangement, hyperkalemia, pulmonary and brain edema.

Intrarenal type of ASL

- pathophysiology: lesions of small intrarenal vessels in front of the glomerulus and directly in the glomerular loops or direct damage to the renal tubules;
- etiology: hemolytic-uremic syndrome (mostly D+HUS), rapidly progressing glomerulonephritis , tubulointerstitial nephritis (e.g. hantaviruses), significant hematuria in severe hemolysis, myoglobinuria in rhabdomyolysis, bilateral thrombosis of renal veins, ethylene glycol (Fridex) poisoning, green toadstool poisoning.

Iatrogenic ASL

- in aggressive cytostatic treatment of malignancies and bone marrow transplantation (cisplatin, cyclophosphamide, ifosfamide);
- the sudden breakdown of cells in hemato-oncology patients leads to a rapid rise in uricemia with the risk of urate nephropathy and then ASL (prevention: high diuresis, urine alkalinization, inhibition of uric acid formation with allopurinol).

Postrenal (obstructive) type of ASL

- bilateral obstruction of the hollow system, rare in children (bilateral urolithiasis, coagulum, candida bezoars).

Metabolic changes

- hypoxia and lack of substrate at the level of the tubules → impairment of tubular functions → morphological changes up to necrosis of the epithelium of the ducts.

Clinical picture

The clinical picture is given by the basic diagnosis, on the basis of which ARS developed.

- oliguria to anuria → edemas → edema of lungs and brain.

Diagnostics

- rapidly progressing glomerular filtration disorder → rise in urea, creatinine and uric acid; tendency to hyponatremia, hyperkalemia, and hypocalcemia;

- acidification disorder leads to a drop in pH (metabolic acidosis).

However, the interpretation of these parameters in children is problematic:

- age, sex, muscle mass, hydration affect serum creatinine level; 48-72 hours after birth, it is comparable to the mother's level and decreases only during the following days, depending on the weeks of gestation.
- newborns have a very low glomerular filtration rate (GFR).
- a rise in serum creatinine will only become apparent when GFR drops by more than 30-50% (risk of late capture).

Renal ultrasound: both kidneys are enlarged (if not, ASL is unlikely).

Diuresis < 1 ml/kg/hour is referred to as *oliguria* , while a decrease in diuresis < 0.5 ml/kg/hour is considered *anuria* . The term *azotemia* is reserved for the accumulation of nitrogenous substances (creatinine and urea) in the blood. *Uremia* then represents azotemia with already manifest clinical symptoms of retention of nitrogenous substances.

Renal damage usually results from ischemic or toxic injury and results in acute tubular necrosis (ATN). In this case, the decrease in glomerular filtration is an adaptive response, because in ATN there is insufficient reabsorption of filtrate in the renal tubules, and this would lead to a massive loss of water and salts without GF restriction. Acute tubular necrosis (ATN) thus represents the dysfunction of tubular cells when they are directly damaged or indirectly by ischemia (the most common mechanism).

Therapy

- monitoring in the intensive care unit (indications: oliguria persisting after adjustment of circulating volume, signs of hyperhydration, rising potassium, hypertension, etc.); in particular, monitoring the dynamics of urine excretion;
- prerenal ASL: volume therapy, furosemide, fluid balance, sufficient energy with low water and potassium content, antihypertensives (calcium channel blockers); correction of hyperkalemia (β2-mimetic; calcium gluconicum; bicarbonate; furosemide; ion exchangers; elimination therapy);
- indications for elimination treatment (hemodialysis; continuous venovenous hemofiltration; peritoneal dialysis in small children): potassium above 7 mmol/l, natremia < 120 or > 160 mmol/l, creatinine > 700 µmol/l, uricemia > 30 mmol/l, hyperphosphatemia, acidosis with pH < 7,, pulmonary edema, cerebral edema;
- after diuresis is restored, a polyuric phase may appear (glomerular filtration has improved, but the kidney's ability to concentrate remains impaired) - monitor fluid balance and urine composition, compensate for fluid and ion losses.

Etiology of ASL by age

Causes in newborns

Anatomical

- congenital anomalies of the kidneys;
- bladder obstruction – posterior urethral valves, catheter obturation, orifice stenosis;
- neurogenic bladder ;
- ureteral obstruction;
- thrombosis of renal veins;
- renal artery occlusion;
- urate nephropathy .

Pathophysiological

- alteration of hemodynamics – asphyxia , hypotension, shock , sepsis , bleeding;
- congenital heart defects ;
- ECMO ;
- hemolytic disease of the newborn ;
- indomethacin ;
- polycythemia ;
- persistent fetal circulation .

Direct cellular toxicity

- exogenous toxins – aminoglycosides , amphotericin B , contrast agents ;
- endogenous toxins – hemoglobin , uric acid .

Causes in later childhood

Prerenal type

- decrease in effective circulating volume: dehydration – vomiting, diarrhea, excessive sweating, excessive diuresis; burns ; adrenal insufficiency ; bleeding ; sepsis ; anaphylaxis ;
- heart failure: congestive heart failure ; massive pulmonary embolism .

Renal type

- ischemic-hypoxic form – acute tubular necrosis (direct damage to the kidney parenchyma, most often ischemic or toxic);
- myoglobinuria – crush syndrome , rhabdomyolysis ;
- hemoglobinuria – hemolysis of various etiologies;
- hypercalciuria ;
- tumor lysis syndrome – urate nephropathy;
- hepatorenal syndrome ;
- nephrotoxic form: salts of heavy metals, carbon tetrachloride , methanol , ethylene glycol , Fridex , drugs - aminoglycosides , sulfonamides , cephalosporins , tetracycline , X-ray contrast agents , mushroom poisoning, organophosphates ;
- inflammatory form: acute glomerulonephritis , rapidly progressive glomerulonephritis (RPGN), tubulointerstitial nephritis (TIN)
- vascular form: hemolytic uremic syndrome (HUS), renal vein thrombosis, disseminated intravascular coagulation (DIC).

Postrenal type = obstruction and disorders of urinary emptying

- VVV of the urinary tract – valves of the posterior urethra, agenesis of the kidneys;
- urolithiasis ;
- tumors ;
- blood clot;
- retroperitoneal fibrosis;
- neurogenic bladder ;
- bladder neck sclerosis.

Drug-induced acute renal failure

Most often, drug-induced ARF is caused by toxic damage to the tubules. ARF due to aminoglycoside toxic damage typically presents with nonoliguric failure. Intravascular contrast agents can also induce ARF, especially in children at risk of "contrast nephropathy" (dehydration, diabetes mellitus, preexisting kidney disease, hypergammaglobulinemia). The risk of "contrast nephropathy" can be reduced by the use of non-ionized contrast medium with low osmolality. NSAIDs cause ARF by affecting intrarenal hemodynamics. Other more frequently used nephrotoxic drugs are acyclovir , amphotericin B , paracetamol (acetaminophen), some cytostatics .

Acute renal failure caused by exogenous toxins

Toxins, either in their natural state or their toxic metabolites, can lead to ARF. The most common are the toxic metabolites of ethylene glycol and methanol . In the treatment of intoxication with both substances, we use intravenously concentrated ethyl alcohol or, newly, fomepizole, which displaces toxic substances by competitive inhibition. Competitive inhibition of alcohol dehydrogenase results in a decrease in the formation of toxic metabolites of ethylene glycol and methanol. In patients with moderate to severe intoxication with severe MAC or a high initial level of ethylene glycol or methanol (> 50 g/dl), urgent hemodialysis is indicated. Both substances are removed by hemodialysis before their further metabolism, at the same time the MAC is adjusted. Of the exogenous substances, intoxication with tetrachloromethane (CCl4 – chlorinated hydrocarbon), which is used as a fat solvent, is even more common. Intoxication is by inhalation, oral or percutaneous. The simultaneous consumption of alcohol potentiates the toxic effect. Intoxication affects the CNS, liver and kidneys. Renal involvement is characterized by ATN. Similar clinical symptoms are also caused by toluene poisoning (drug addicts). Poisoning by heavy metals (e.g. mercury) is less common.

Nephropathy in hemolysis and rhabdomyolysis

Hemolysis and rhabdomyolysis can, under certain circumstances, result in marked hemoglobinuria and myoglobinuria, leading to toxic tubule involvement and ARF. Vasoconstriction, precipitation of pigments in the tubular lumen and heme protein-induced oxidative stress are used in the mechanism of tubule damage. If rhabdomyolysis occurs as a result of extensive tissue damage (crush syndrome) with significant leakage of fluids into the extracellular space, rapid and adequate rehydration may prevent or alleviate renal impairment. Mannitol or a loop diuretic along with intravascular volume adjustment will improve flow and prevent precipitation of heme proteins in the tubules. Urine alkalinization (administration of bicarbonate) increases the solubility of both hemoglobin and myoglobin and reduces the rate of tubular damage. MAC, hyperkalemiaand imbalance of other electrolytes as a manifestation of ARF require adequate treatment.

Urate nephropathy

ARF in children may occur in association with leukemia or lymphoma due to infiltration of the kidney by tumor cells or due to urate nephropathy. Children with acute lymphoblastic leukemia and B-cell lymphoma have a higher risk of urate nephropathy and *tumor lysis syndrome*. In the pathogenesis of urate nephropathy, precipitation of uric acid crystals in the distal tubules, collecting ducts, calyces, pelvis and ureter is an important mechanism of damage, with subsequent obstruction of urine flow and obstruction of blood flow through the renal microcirculation. Crystal deposits in the renal tubules can directly damage the tubular cells due to their composition. The most common cause of ARF in leukemia is the breakdown of tumor cells during chemotherapy ("tumor lysis syndrome") with an extreme increase in the level of uric acid in the blood (> 1000 umol/l). Allopurinolreduces the excretion of uric acid during chemotherapy, but significantly increases the excretion of uric acid precursors (hypoxanthine, xanthine). Precursors have a lower solubility than uric acid, and their precipitation plays a role in the development of ARF in tumor lysis syndrome, which is accompanied by a rapid rise in serum potassium, urea, products of purine

metabolism, phosphorus along with a decrease in serum calcium. The prognosis of ARF due to the breakdown of tumor cells is good with possible improvement of renal function. However, dialysis or hemofiltration treatment is often needed due to hyperkalemia and other metabolic changes.

Nephropathy in hypercalcemia

ARF occurs with acute hypercalcemia, and the degree of damage depends on the degree of hypercalcemia . The clinical picture is dominated by polydipsia , a marked impairment of concentration, polyuria with losses of electrolytes and water. The most common cause in children is hypervitaminosis D (vitamin D poisoning), as well as diseases associated with hyperparathyroidism and long-term immobilized patients. Treatment consists of hyperhydration, administration of furosemide and correction of ion imbalance by administration of calcitonin and possibly corticoids. Administration of bisphosphonates is not recommended in children. If the conservative procedure fails, hemodialysis with a low concentration of calcium in the dialysis solution is indicated.

Acute interstitial nephritis

Acute interstitial nephritis can lead to ARF as a result of a drug reaction or from idiopathic causes. In children, it is clinically manifested by exanthema, fever, arthralgias, eosinophilia. There is leukocyturia with a predominance of eosinophils in the urine (eosinophiluria). Sonographic examination shows enlarged, hyperechoic kidneys. To establish the diagnosis, it is often necessary to perform a renal biopsy with the characteristic finding of interstitial infiltration with a large number of eosinophils. Acute interstitial nephritis pathogenetically caused by drugs belongs to hypersensitivity reactions. In some cases, it is associated with the formation of anti-TBM antibodies. Among the drugs most often associated with its development are methicillin and other penicillin analogues, sulfonamides, rifampicin, NSAIDs. A manifestation of interstitial nephritis, especially in connection with NSAIDs, can be significant proteinuria up tonephrotic syndrome . Immediate cessation of administration of the suspected drug together with corticoid treatment is one of the basic treatment measures.

Rapidly progressive glomerulonephritis

All forms of glomerulonephritis can lead to Rapidly Progressive Glomerulonephritis (RPGN) and ARF in severe cases. The clinic is dominated by severe hypertension, edema, macroscopic hematuria, proteinuria with a rapid increase in azotemia. RPGN is histologically characterized by the finding of so-called crescents in more than 50% of glomeruli. In some forms of GN (post-infectious, membranoproliferative, Henoch-Schönlein purpura , SLE), the picture of RPGN is less frequent, in others (ANCA positive, anti-GBM) RPGN with the development of ARF is a typical picture. An immunological examination of the complement is important to clarify the diagnosis, its components and autoantibodies (ANA, ANCA, anti-ds-DNA, anti-GBM). In order to start treatment in time, it is necessary to establish the diagnosis histologically (character and extent of changes) and early renal biopsy is indicated in all children with suspected RPGN.

Thrombosis of the renal artery and vein

Thrombosis of the renal artery and vein, in addition to cortical necrosis, most often occurs in newborns and young children. Thrombosis of the renal artery is closely associated with affection in the area of the arteria umbilicalis and ductus arteriosus . In ARF on the basis of thrombosis of large vessels, hypertension is present, macroscopic or microscopic hematuria, thrombocytopenia appears. In the treatment, anticoagulants are given in addition to the removal of the provoking cause (umbilical catheter).and fibrinolytics. In renal artery thrombosis, the sonographic image is initially normal or very poor. A radionuclide examination shows significantly reduced or absent blood flow through the kidney. In the case of renal vein thrombosis, sonography shows an enlarged, edematous kidney with a finding of reduced blood flow through the kidney and reduced function of the affected kidney during radionuclide examination.

Cortical necrosis

Cortical necrosis is the most common cause of ARF in neonates. Cortical necrosis is associated with hypoxic involvement due to severe perinatal hypoxia , placental abruption , feto-fetal or fetomaternal transfusion with subsequent activation of the coagulation cascade. Newborns and young children with cortical necrosis usually have hypertension, macroscopic or microscopic hematuria, oliguria. In addition to azotemia, thrombocytopenia is present as a result of impaired microcirculation. The prognosis of cortical necrosis is grave. Children with cortical necrosis require different lengths of dialysis treatment. In some children, there may be a partial adjustment of renal functions, but in further development they are at risk of developing chronic renal insufficiency. In comparison with renal vein thrombosis, the sonographic examination is initially normal, in the later period the kidneys atrophy, gradually decreasing in size. Radionuclide examination shows reduced or absent renal perfusion and a picture of functional kidneys (without signs of uptake of radionuclide by the renal parenchyma).

Postrenal type ARF

ARF of postrenal origin is characterized by a urodynamic disorder. The most common causes that lead to obstruction of the urinary tract are congenital anatomical malformations (valve of the posterior urethra in boys, tight phimosis, stenosis of the urethra, bilateral stenosis of the ureters, most often in the area of the ureterovesical junction and in the pyeloureteral transition), functional disorders (severe degree of bilateral VUR) _ _ .

Disruption of outflow in the outlet urinary tract (both organic and functional) leads to a rise in pressure and subsequently to dilatation of the urinary tract above the obstruction. Even short-term obstruction (hours) can lead to the development of ARF. The so-called reflex anuria of the unaffected kidney may also develop (e.g. with renal

colic). Long-term obstruction leads to the development of hydronephrosis and severe pressure damage to the kidney parenchyma. A frequent and very serious complication is infection with the development of inflammatory nephropathy with the formation of irreversible scars (reflux nephropathy). At the beginning of the blockage of the outflow of urine, the urine formed in the affected kidney is hyperosmolar with a low sodium content. There is increased blood flow through the kidney as a result of prostaglandin dilation of the afferent arterioles. In the further course, blood flow through the kidney decreases and GF decreases. The slight decrease in GF at the beginning of the obstruction is a consequence of the increased hydrostatic pressure above the obstruction. After the obstruction is removed, both kidney blood flow and GF are adjusted. The degree of adjustment of renal functions depends on the speed of adjustment of the increased pressure in the outlet urinary tracts, either by definitive removal of the obstruction, or by temporary diversion of the urine flow with a definitive solution at a later period (after normalization of renal functions). The process is therefore very often reversible after removal of the obstruction (the exception is the very poor long-term prognosis in congenital obstructive defects - e.g. posterior urethral valve in boys). The postrenal type of ARF is caused by impaired urine outflow, when increased pressure in the urinary tract causes dilatation with subsequent progression to nephron destruction. RTA IV is characteristically present at initial tubular cell damage. type.

Pathogenesis

The pathophysiological mechanisms of ARF can be divided into 3 groups: changes in renal hemodynamics, nephron factors and cellular/metabolic changes:

1. **Changes in renal hemodynamics** : The presence of renal vasoconstriction in clinical ARF is included in the term vasomotor nephropathy. An insult acting on the tubular epithelia leads to the release of vasoactive substances that increase cortical vascular resistance. There is a decrease in blood flow through the kidney and continued damage to the tubules (the most important factor in the development of acute tubular necrosis is the release of vasoconstrictor substances). The release of vasoactive substances leads to a decrease in GF by constriction of both afferent and efferent arterioles. The result is reduced diuresis. The system of vasoactive substances and components of epithelial damage includes the RAAS , prostaglandins, adenosine, endothelin and nitric oxide. In the prerenal type of ARF, the common denominator is a decrease in the volume of circulating fluid and a decrease in blood pressure, which cause a significant decrease in blood flow through the kidneys. The most sensitive to hypoxia and ischemia is the proximal tubule and the ascending part of the loop of Henle. EGFT= effective glomerular filtration pressure, EGFT= BP in vas afferens − (oncotic BP + pressure in Bowman's capsule) = 70 − (30 + 10) = 30 torr. A decrease in the effective circulating volume leads to a decrease in EGFT, a decrease in blood flow through the glomerulus. Glomerular hypoperfusion represents a reduction in the supply of glucose and amino acids to the tubules, which leads to damage to the proximal tubules. Damaged cells of the proximal tubules cannot reabsorb sodium in the glomerular infiltrate, there is an increase in the sodium load to the juxtaglomerular apparatus, followed by renin washout and activationRAAS with vas afferens vasoconstriction. This represents a further reduction in renal blood flow and the circulus vitiosus closes.
2. **Changes in nephrons** : In the direct section of the proximal tubule, there is a high activity of oxidative phosphorylation, which is demanding on the supply of energy. In case of long-term ischemia, cell edema and rupture of the tubular basement membrane occur, which means the death of the cell. As a result of these changes, ultrafiltrate leaks through broken tubular cells into the interstitium and peritubular capillaries. The result of this process is then oliguria.
3. **Cellular and metabolic changes** : Disruption of adenine nucleotide metabolism as a result of ischemia within 5-10 minutes causes a decrease in ATP of up to 90%. Reactive oxygen radicals are responsible for reperfusion injury after ischemia. A rise in intracellular calcium concentration then ultimately leads to cell death. There is a significant activation of endogenous phospholipase, which breaks down membrane phospholipids and thus ruptures the cell membrane. Last but not least, there is a reversal of cell polarity, sodium retention and cell edema.

Clinic

The most common type of prerenal ARF occurs, which can cause all conditions of dehydration and shock in children. We observe signs of dehydration in children. Oligoanuria is present. With nephrotic syndrome, children have swelling and are very pale. In ATN, the clinical picture does not differ from states of prerenal failure (except for the usual dehydration), but the difference is in the duration of acute insufficiency. In pre-renal failure, renal function is usually corrected within a few days, while in ATN the failure persists for several weeks and tubular functions in particular are corrected for up to several months, even so some patients progress to chronic renal insufficiency. The most common cause of parenchymal involvement that leads to ARF in infancy is the hemolytic-uremic syndrome. Clinically, it manifests as marked pallor, mild swelling, oliguria, anemia, thrombocytopenia with signs of bleeding. In older children, acute GN and acute TIN are the most common cause of ARF of renal origin. In acute GN, patients are pale, tired, edema, accompanying hypertension and oliguria are present. On the contrary, patients with TIN, despite the manifestations of renal insufficiency, often have polyuria, allergic exanthema is common, other manifestations such as fatigue, pallor, malaise are similar. In postrenal failure, the cause of which is urinary stasis, the clinical picture is dominated by anuria, the child is pale, tearful, during palpation we feel resistance in the abdominal cavity (abdominal mass). During the clinical examination, it is necessary to focus on the state of hydration, manifestations of bleeding, cardiac compensation, BP (cave ! - hypertension) and thorough palpation of the abdominal cavity. Common clinical signs of uremia include symptomatology:

- CNS: increased neuromuscular irritability, lethargy, confusion, convulsions... uremic coma,
- cardiovascular system: hypertension, peripheral edema, pericarditis,
- lungs: dyspnoea, tachypnea,
- GIT problems: anorexia, nausea, vomiting, foetor uremicus,

- hematology: anemia, hemorrhagic diathesis ,
- general: pallor, fatigue, nausea, MAC.

The clinical course of ARF is divided into several phases:

Initial phase

This phase, which sometimes lasts 24-48 hours, can manifest itself with symptoms of shock and symptoms of the underlying cause (poisoning, renal colic, etc.). The patient does not yet have clearly altered renal functions, the urine contains a small amount of protein, sometimes even blood.

Anuric phase

Diuresis decreases < 0.5 ml/kg/hour. This phase lasts for several days to weeks, sometimes the production of urine does not resume at all. The concentration of nitrogenous substances in the blood rises rapidly (urea by > 8 mmol/l during 24 hours, creatinine by > 100 umol/l during 24 hours). In hypercatabolism, which is significant in burn trauma, crush syndrome, but also in a low-energy diet, the concentration of creatinine and especially urea rises much faster. Such a course is more often associated with serious and life-threatening complications. Potassium also increases, and at a concentration > 8 mmol/l there is a risk of arrhythmia or cardiac arrest. Water is retained in the body, serum osmolality drops, hyponatremia occursand with the accumulation of interstitial fluid with the clinical manifestation of edema of the lungs (dyspnea, tachypnea) and brain (impaired consciousness, convulsions). Peripheral edema and arterial hypertension occur, the patient's weight increases (excellent evidence of hyperhydration). Patients complain of fatigue, loss of appetite, dry mouth, nausea, and there is often a singultus . We find disturbances of consciousness of varying degrees, sleep inversion, increased neuromuscular excitability up to tetany/convulsions, and in extreme cases a uremic coma develops. The development of uremia is characterized by vomiting, foetor uremicus and Kussmaul respiration in severe MAC. Terminally, significant normochromic anemia develops, manifestations of hemorrhagic diathesis (bleeding from the nose, GIT, into the skin), pericarditis, infectious complications. About 1/5 of patients have polyuria instead of anuria. It is a so-called hyperfiltration ARF, most often in cases where ARF occurs on the basis of acute TIN. Despite the presence of polyuria, there is an increase in serum urea and creatinine. In shock states, it is therefore absolutely necessary to check not only the diuresis, but also the concentration of urea and creatinine at appropriate intervals, so that this polyuric variant of ARF does not escape.

Phase of early diuresis

Kidney function begins to recover in a favorable course, but it is not enough to remove catabolites from the blood.

Stages of polyuria

The urea and creatinine levels begin to drop, the diuresis begins to recover, which in the course of several days can reach values of around 10 liters/day. However, the osmolality of the urine is low, we are talking about the so-called **hyposthenuria** . The patient loses a large amount of water and electrolytes, there is a risk of dehydration, depletion of potassium and other ions, thrombosis and embolization to internal organs.

The recovery phase

It means gradual adjustment of glomerular filtration and azotemia. This period can last up to 3 months, and the impaired concentration ability can last up to 6 months. During this phase, it is still necessary to monitor the patient's hydration, protein catabolism, changes in ABR and minerals.

Diagnostics

When a patient with oliguria and suspected ARF is hospitalized, it is necessary to determine the type of acute failure based on laboratory tests and to reveal the cause that led to it. The basis is the examination of urine, where in the sediment we demonstrate erythrocytes, leukocytes, debris, casts of cylinders, in chemistry then myoglobin or hemoglobin , various amounts of protein. Urine findings vary according to the etiology of ARF. We always determine waste Na, K, creatinine and urea in urine, we determine osmolality in serum and urine. In ARF, the examination of urea concentration is more important than creatinine for monitoring progression, because urea concentration more quickly reflects acute changes in renal function. Typical laboratory findings in ARF include:

- blood count – anemia, thrombocytopenia ,
- hemocoagulation disorders,
- azotemia – elevation of urea and creatinine,
- ABR – metabolic acidosis,
- ion imbalance – hyponatremia/hypernatremia, hyperkalemia, hypocalcemia, hyperphosphatemia,
- hyperuricemia,
- proteinuria – tubular or glomerular.

In general, urea and creatinine concentrations should always be checked for any dehydration, loss of consciousness of unclear etiology or changes in the internal environment!

Display methods

- USG : we always perform a sonographic examination (it is stress-free, painless and quickly available). It is an excellent method for excluding a postrenal cause of ARF, when it demonstrates dilatation of the lower urinary tract. We evaluate the size of the kidneys according to the Dinkel nomogram. In ARF, the kidneys are usually enlarged, on the other hand, wrinkled kidneys are found in chronic renal insufficiency , which could have escaped attention for a long time and the first clinical manifestation was a picture of acute failure.
- nephrogram,
- dynamic scintigraphy – DTPA, MAG 3,
- DMSA ,
- CT , MRI of kidneys and urinary tract.

Differential diagnosis

Differential diagnosis of prerenal and renal failure		
	Prerenal cause	**Renal cause**
nitrogenous substances	III urea, I creatinine	II urea, II creatinine
U-Na in mmol/l (single sample)	< 20	> 40
U-osmolality in mosmol/l	> 500	< 300
specific gravity of urine	> 1020	< 1010
FE for sodium	< 1%	> 2%
U-osmolality / S-osmolality	> 1.5	< 1.2

As part of the differential diagnosis of increased urea and creatinine, we consider the following causes:

- increase in urea,
  - increased protein intake,
  - GIT bleeding,
  - catabolism,
  - steroids,
  - ARF,
- increase in creatinine:
  - increased muscle relaxation,
  - ARF.

Differential diagnosis of acute and chronic renal failure		
	acute failure	**chronic failure**
kidney sonography	normal or larger kidney size	small kidney size
changes in the background of the eye	0	0
changes on X-ray of the wrist	+	+
rise in S-creatinine	daily increase of > 45 umol/l	slow rise

Therapy

The standard provision is the insertion of a urinary catheter to monitor the exact water balance. The therapeutic approach to a patient with ARF depends mainly on the type of renal failure. In pre-renal failure, the basis is adequate and timely rehydration with the restoration of renal perfusion and diuresis, in the renal type of failure, then specific treatment of RPGN , TIN , hyperuricemia, hypercalciuria, etc. The basis of post-renal type treatment is to restore the patency of the urinary tract, possibly to ensure the diversion of urine in an alternative way - catheterization, epicystostomy. The therapeutic procedure is fundamentally based on 3 considerations:

1. the cause of ARF is hypovolemia/hypoperfusion and the patient is still in a reversible state where he needs adequate volume therapy and the condition will be corrected,
2. it was primarily hypovolemia/hypoperfusion, but the patient is already in the stage of acute tubular necrosis. GF does not recover (perhaps partially) after fluid replacement, but the patient needs an adequate fluid supply to improve perfusion of other organs,
3. the primary cause is renal or postrenal, and intravascular volume and perfusion of other organs are normal. Fluids are not necessary, on the contrary, it is urgent to address the specific cause that led to the renal type of failure or to relieve the obstruction of the urinary tract.

If we initially diagnose renal failure, but neither the clinical condition nor the anamnesis are helpful in determining the type of failure, the basic milestone is to perform a sonographic examination. If sono shows obstruction, acute urinary diversion is required. If sono does not show obstruction, we will try to improve renal perfusion. The goal of conservative therapy is to adjust the volume of fluids, adjust sodium and potassium disorders, adjust calcium phosphate metabolism, correct MAC and hypertension , and adjust the diet.

Complications

- Lungs: ARDS , pulmonary edema, bronchopneumonia,

- heart and circulation: heart failure, arrhythmia, pericardial effusion, hypertension,
- GIT: bleeding, stress ulceration, gastroenteritis,
- CNS: brain edema, intracranial hypertension ,
- hematopoiesis: anemia , thrombocytopenia, coagulopathy.

Links

Related Articles

- Chronic kidney function disorders (pediatrics) • Renal failure (neonatology)
- Acute kidney failure • Chronic kidney disease • Acute kidney failure/Repetitor
- Acute glomerulonephritis • Rapidly progressing glomerulonephritis • Chronic glomerulonephritis

Source

- MD HAVRÁNEK, Jiří: Acute kidney failure