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ACUTE RENAL FAILURE IN NEWBORN CHILDREN

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ABSTRACT

This article represents a study of 20 new born children, hospitalized for a period of 5 years in two different profile units. The Fourth Clinic of Pediatric Nefrology Iasi and The Compartment of Intensive Care of Pediatric Clinic "SF. Ioan Galati". The Children have been diagnosticated, according to the RIFLE pediatric criteria, which allow a better classification of the disease and better assumptions on the evolution of the disease. The IRA etiology on new born children is shown, the genetic cause of children with IRA risk. Clinical and biological parameters where taken into attention, the treatment and evolution of the disease, being known that IRA episode while very young, may cause chronic renal disease.

KEYWORDS*: acute renal failure, acute kidney injury, newborn*

1.Introduction

In the newborn the precise incidence and prevalence of acute renal failure is unknown, several studies have shown the acute renal failure is comman in the neonatal intensive care unit. In a developing contry the incidence was 3,9 of 1000 live birts and 34.5 of 1000 newborns admitted to the neonatal unit.

In the newborn, renal failure may have a prenatal onset in congenital diseases such as renal dysplasia with or without obstructive uropathy and in genetic diseases such as autosomal recessive polycystic kidney disease. Is also commonly acquired in the postnatal period because of hypoxic ischemic injury and toxic insults associated with aminoglycoside antobiotics and nonsteroidal anti-inflammatory medications (used to mother or child).

In the newborn alterations in renal function occur in approximately 40% of premature newborn who have received indomethacin and such alterations are usually reversible.

Sice nephrogenesis proceeds through approximately 34 weeks' gestation, ischemic/hypoxic and toxic insults to the developing kidney in a premature newborn can result in not only in acute renal failure but long-term complication associated with potatial interrupted nephrogenesis.

After birth, the serum creatinine in the new born in a reflection of maternal renal function and cannot be used as a measure of rena function in the newborn shortly after birth. In full-term healthy

newborns, the glomerular filtration rate rapidly increases and the serum creatinine declines to about 0.4 to 0.6 mg/dL at about 2 weeks of age as the serum creatinine declines at a slower rate in premature infants.

Tabel 1. *Etiology of Acute Renal Failure in Newborns*

Prerenal Failure
Decreased true intravascular volume
Dehydration
Gastrointestial losses
Salt wasting renal or adrenal disease
Central or nephrogenic diabetes insipidus
Third space losses (sepsis, traumatized tissue)
Decreased effective intravascular volume blood volume
Congestiv heart failure
Pericardis, cardiac tamponade
Intrinsic Renal Disease
Acute tubular necrosis
Ischemic/ hypoxic insults
Drug induced
-Aminoglycosides
-Intravascular contrast
-Nonsteroidal anti-inflammatory drugs
Toxin mediated
-Endogenous toxins
-Rhabdomyolysis, hemglobinuria
Interstitial nephritis
Drug induced-antibiotics, anticonvulsant
Idiopathic
Vascular lesions
Cortical necrosis
Renal artery thrombosis
Infectious causes
Sepsis
Pyelonephritis
Obstructive Uropathy
Obstruction in a solitary kidney
Bilateral uretral obstruction
Urethral obstruction
Congenital Renal Diseases
Dysplasia/hypoplasia
Cystic renal diseases
-Autosomal Recessive polycystic kidney disease
-Autosomal Dominant polycystic kidney disease
-Cystic dysplasia

Thus, use of the serum creatinine asa a determinate of renal insufficiency requires that the gestational age of time of birth and the postnatal age as well as maternal factors needs to be taken in to account.

Some newborns may have genetic risks factors for acute renal failure (polymorphism of the angiotensin converting enzyme, polymorphisms of tumor necrosis factor alpha, interleukin 1, 6, 10, the heat shock protein 72 genetic variation)

2. Materials and methods

For a period of four years, between 2004 and 2008, 104 children were hospitalized with renal insufficiency at the Pediatric Nephrology Clinic IV in Iasi – group A and at the intensive care unit of the Pediatric Hospital in Galati – group B. Among these, 20 patients aged between 1 and 30 days have been subject for a study, in order to analyze the etiological spectrum, the evolution and the influencing factors. The period of study lasted between 2 months and 4 years. We have analyzed: the ARI (Acute Renal Insufficiency) etiology, clinical and lab parameters (edemas, T.A., diuresis, digestive and neurological disorders, nitrate retention and E.C.G. modifications, treatment, disease complications and evolution)

3. Results and discussion

The new-born subject for the study, have been divided into two groups, according to their city of provenance (figure 1). Age varied between 1 day and 1 month (figure 2, 3). 15 children were from the rural area and 5 children were from the urban area.

The prerenal etiology mostly affected the neonatal age.

A). Prerenal causes:

a. decrease of the plasmatic volume:

-Acute dehydration - 9 cases;

-Hemorrhages - 2 cases (post hemorrhagic shock);

-Neonatal septicemia – 7 cases;

b. other conditions that induced the renal hypo-perfusion:

-Neonatal respiratory distress – 2 cases;

-Congestive cardiac insufficiency – 3 cases.

c. medicines – inhibitors of the prostaglandin synthesis:

-Administration to the mother, during labor, of the nonsteroidal antiinflammators - 1 case;

-paracetamol intoxication of the child – 1 case;

-i.v. diazepam administration as anticonvulsive induced the transitory decrease of the renal sanguine flux - 1 case;

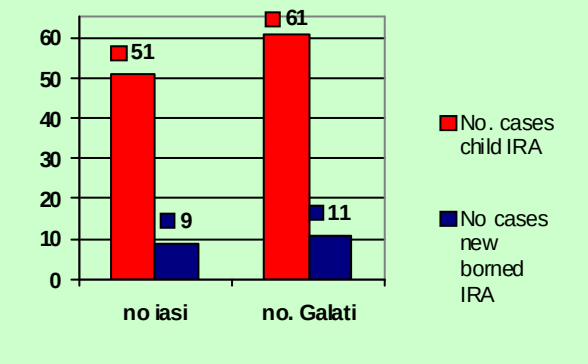

Figure 1. *Case division according to the city of provenance. A – ARI children hospitalized in the Pediatric Nephrology Clinic IV in Iasi ; B – ARI children hospitalized at the intensive care unit Galati*

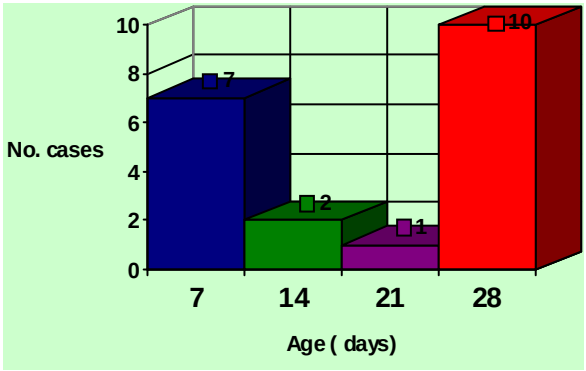

Figure 2. Case division according to age

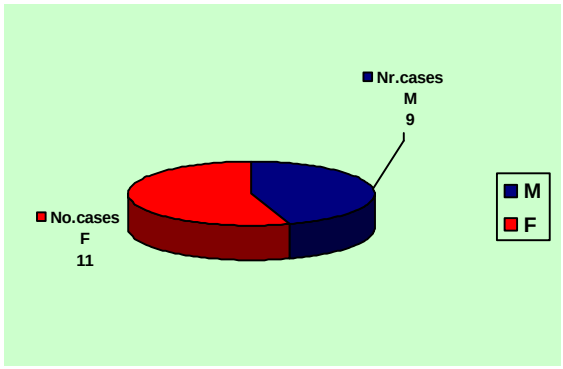

Figure 3. Case division according to sex

B). ARI because of renal causes:

-cortical necrosis has been encountered in one case who had hypoxic-ischemic lesions because of the hemorrhage shock (placenta praevia). This case led to the differential diagnosis between prerenal ARI induced by hypovolemia secondary to the massive hemorrhage and intrinsic ARI ; the clinical evolution of the lab parameters after the recovery of the volemic equilibrium allowed us to find the ARI intrinsic cause.

-the renal tubular necrosis has been encountered to a new-born with perinatal asphyxia that induced the decrease of the sanguine flux and also the decrease of the renal parenchyma perfusion;

-renal congenital anomalies:

- 1 case of bilateral renal hypoplasia.
- 1 case of right renal agenesis.

-neonatal hyperuricuria – 1 case.

C). Postrenal causes

Obstructions of the urinary tract:

-pielo-ureteral duplication – 1 case;

-hydronephrosis, pielo-ureteral junction stenosis – 1 case;

We should mention that in most cases the ARI pathogeny was a cumulus of factors. For example: - child with right renal agenesis and left hydronephrosis, presents neonatal asphyxia and SDA; child with cord congenital malformation who presents

perinatal hemorrhages and SDA; child with Down syndrome, intoxicated with nitrites, presents SDA and evolution towards multiple organic insufficiency.

ARI etiology to the new-born in the analyzed groups.

In 5 cases we have encountered precarious nutrition, out of which 3 with atrepsy.

In 4 cases we have encountered prematurity.

We have analyzed the following clinical parameters in the ARI group of children: edemas, oligoanuria, hypo or hypertension, digestive disorders, neurological disorders, fever:

-edemas - 6 cases, with a generalized character;

-oligoanuria - 6 cases at 3 children, older than 2 days (these children died);

-arterial hypotension - 2 patients who needed treatment with vasopresor amines; both children died, hypotension being an aggravating factor;

-digestive disorders (nausea and vomiting) - 13 children;

-respiratory distress, neonatal hypoxia - 5 cases;

-neurological disorders – 5 cases (convulsions - 1 case, coma - 2 cases, obnubilation - 2 cases);

-fever was present in 10 cases.

Electrolytic disorders - 8 patients:

-hyperpotassium – 4 cases;

-hyponatrium – 2 cases;

-hypernatrium – 2 cases;

The higher potassium values were of 9 mEq/l, 8 and 7,6 mEq/l, all of them leading to the patient's death. Metabolic acidosis was revealed in 9 cases. The nitrate retention level measured considering the clearance to creatinine at the beginning had values between 3 and 25 ml/min/1,73m^2.(figure 4)

The presence of comorbidities (figure 5)

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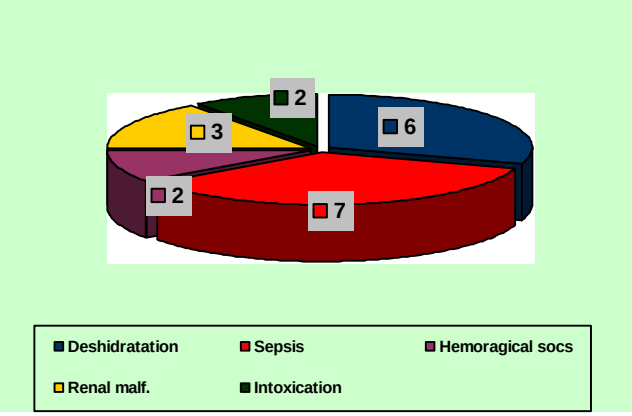

Figure 4. – Case division according to the etiology

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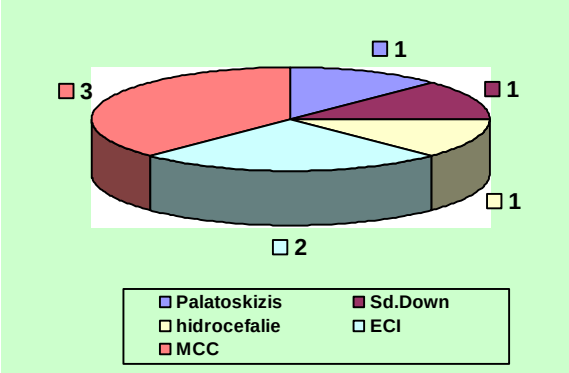

Figure 5. *Representation of the comorbidities*

Hypo-proteinuria was revealed in 6 cases.

Anemia was mentioned in 3 cases, leukocytosis in 6 cases, trombocitopeny in 4 cases (tabel 1,2).

Tabel 1. *Biological modifications*

Biological modifications	No. of cases
Anemia	3
Leukocytosis	6
Trombocitopeny	4
Hypo-proteinuria	6
Metabolic acidosis	9
Hyper K	4
Hypo Na	2
Hyper Na	2

The ECG modifications detected to our patients were high T waves, symmetric and sharp in 4 cases of hyperpotassium, confirmed by the sanguine ionogram.

The creatinine values increased in 2 – 10 days from the beginning of the disease. Death occurred in 1 – 11 days after establishing the diagnostic and it was generally induced by the comorbidities, the renal insufficiency having a main role in the aggravation of the disease progress.

Table 2. *Diagnostic according to the Rifle Criteria*

RIFLE Criteria	GROUP A		GROUP B	
	S	D	S	D
R	2	1	3	2
I	1	-	1	1
F	5	-	1	2
L	-	-	-	-
E	-	-	-	-

Treatment was a conservatory one in all cases and its main purpose was to maintain the hydroelectric and nutritional equilibrium. Fluids were calculated according to the diuresis and the digestive losses. Liquid administration in PEV was carefully checked.

Severe anemia was treated in a substitutive way in 3 cases with ME 6-10 ml/kg, in 2 cases patients were administrated plasma and in 2 other cases patients needed an inotropic treatment.

In 4 cases, oliguria, hyponatrium and hyperpotassium put to the issue the necessity of the substitution renal therapy.

In another case with hyperpotassium – 9 mEq/l to a child with multiple organic insufficiency, Down syndrome and nitrite intoxication, we have chosen an anti-shock treatment (inotropic treatment, plasma), but the disease evolution soon became unfavorable, the patient manifesting respiratory distress, mechanic ventilation and death.

Another 2 cases with malformative syndromes, characterized with hyponatrium and hyperpotassium (consequences of the ARI) had an unfavorable evolution induced by the comorbidities.

Following the patient’s evolution, we can easily observe that most of them fully regained their renal function (table 3).

In group B, 2 children have severe neurological sequelae within the pale of comorbidities.

Table 3. *Evolution of the patients from the two groups*

Evolution	Group A	Group B
Good	6	6
Chronicity	2	0
Death	1	5

In 8 cases, it was possible to get a fair valuation, from the distance.

The recovery period lasted between 5 and 30 days.

A quick recovery, in less than 7 days was observed in the ARI cases through SDA. Most of the patients normalized their seric creatinine values in 5-7 days.

The nitrite retention exceeded 30 days at the 2 patients with obstructive malformations of the urinary tracts and vesica and ureteral reflux. Evolution towards chronicity was observed to one of these cases after 3 years and to the other one after one year from the debut, when the corrective surgical operation was delayed.

Death occurred in 6 cases:

-3 cases with severe MCC;

-1 case of mielo-meningocele;

-2 cases of multiple organic suffering.

Mortality – 30%. The medical treaties refer to values between 10-61%, higher to the children with multiple organic insufficiency.

Prognostic depends on the ARI etiology. The factors which lowered the prognostic were: multiple organic insufficiency, hypotension, pressure agents, homodynamic instability and mechanic ventilation.

The death rate was higher for patients with multiple organic insufficiencies.

Although the prognosis of the cortical necrosis is unfavorable, our case that didn't require dialysis therapy had a better evolution 1 year later. There is however the risk to later develop a chronic renal insufficiency.

The child diagnosed with renal tubular necrosis had a good progress one year after the diagnostic.

The newborn with ARI request a long term medical surveillance of the renal function, TA, proteinuria. Typically, the late evolution towards the CRI (Chronic Renal Insufficiency) is characterized by the development of the HTA, proteinuria and increase of the ureic azoth.

Near 5% of the newborn hospitalized patients develop ARI. The ARI etiology may be of a prerenal, intrinsic or postrenal cause. Most of the very young patients develop ARI, having a prerenal cause or acute tubular necrosis. A minute anamnesis and a complete clinical examination, the sanguine and urinary tests and the logistic evaluation allow us to establish the diagnostic and to specify the ARI etiology. Near 0.5% of the patients with ARI need dialysis, required by the inability of the renal function, the need to maintain the hemodynamic equilibrium, the correction of the electrolytic disorders or the assurance of a corresponding nutritional supply.[4] The cases we have presented had a different diagnostic: prerenal ARI induced by hypovolemia and intrinsic ARI. Studies have revealed that the long term prognostic is better when the renal disease is isolated, compared to the multiple organic diseases.[5] the anuric or oliguric patients show a higher death rate than the patients with constant diuresis.[8]

4. Conclusions

ARI has complicated the evolution of many newborn hospitalized in the intensive care units and etiology was, in general, multifactor (ischemic, hypoxic and nephrotoxic) dominated by comorbidities.

Division on sexes was relatively equal and 75% of the cases came from the rural area.

Treatment was supportive in most of the above cases, but for 3 of them, dialysis was put to the issue. The decision of a dialysis treatment in the intensive care unit is taken by the pediatrician - nephrologist, the resuscitator and the neonatologist, this modality depending on the clinical estate and the local resources.

Following the patient’s evolution, we can easily observe that most of them fully regained their renal function. Children who survive the renal insufficiency present the risk to later develop a chronic renal insufficiency and must be carefully put under medical observation.

The death rate was high (30%), especially for children who have developed multiple organic insufficiency. In most of the cases, mortality was due to extra-renal causes. The factors that lowered the prognostic were: multiple organic insufficiency, pressure agents, homodynamic instability.

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