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MULTIPLE RENAL INJURIES LEAD TO DEATH IN POSTOPERATIVE CARDIAC SURGERY EVEN WITH PRECOCIOUS HEMODIAFILTRATIONS

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ABSTRACT

We present the case of a newborn diagnosed with perinatal asphyxia and secondary renal injuries, transposition of the great vessels and low systemic blood flow, treated with Prostaglandin, atrioseptostomy, followed by arterial switch surgery After the cardiac surgery the patient is oliguric and requires hemodiafiltration for 12 days, after which renal function is restored. In evolution, however, AVB (atrioventricular block) grade III occurs, followed by implantation of permanent pacemaker, but another postoperative complication – chylothorax – leads to stopping electrical stimulation followed by severe cardiac dysfunction and, consequently, recurrent renal injury and anuria. Re-establishing hemodiafiltration for another 7 days without recovery of renal function. Perinatal asphyxia, cardiac heart disease with low systemic blood flow, prostaglandin, atrioseptostomy, cardiac rhythms disturbances, chylothorax, sepsis, cardiac arrest are intriguing factors that bring renal injury. Their association greatly decreases the chance of survival even if the patient benefits from supportive treatment and early hemodiafiltration.

Keywords: hemodiafiltration, acute kidney injury, newborn, transposition of the great vessels, permanent pacemaker malfunction, chylothorax

INTRODUCTION

The acute kidney injury (AKI) in neonates continues to be an important cause of morbidity and mortality in intensive care because approximately 25-30% of newborns in NICU (Newborn Intensive Care Unit) have acute kidney injury (1). Although no consensus has been reached, more and more clinics are using the modified KDIGO (Kidney Disease Improving Global Outcomes) criteria for newborns (n-KDIGO) which are based on the decrease of diuresis below 1 ml/kg/hour and the increase of serum creatinine by more than 0.3 mg/dl or more than 50% of the previous value (2). The difficulties encountered in establishing the diagnosis of AKI in newborns are due to the fact that in the first 72 hours of life the creatinine level can be increased, in accordance with the serum level of the mother and most acute kidney injury in newborns, in the early stages are non-oliguric (3). This is due to the fact that newborns have an immature renal tubular function, with a limited ability to concentrate urine and a high percentage of water in body composition (4). In 85% of cases neonatal AKI has prerenal etiology, becoming intrinsic if it is prolonged. Dehydration, perinatal asphyxia, kidney malformation, thrombosis of the renal vessels, sepsis and cardiovascular surgery are among the most common causes of kidney injury in newborns (5), but there are also less common causes, such as metabolic disease, cardiac rhythm disorders or chylothorax.

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CASE PRESENTATION

This case is about a term female newborn, extracted through an emergency caesarean section for acute fetal asphyxia, BW = 3,000 g, who presents postnatal seizures and cerebral changes specific to hypoxic-ischemic encephalopathy. She is diagnosed immediately after birth with transposition of the great vessels, a high muscular ventricular septal defect and a minimal oval foramen. Prior to the cardiac surgery, she receives Prostaglandin and Raskind atrial septostomy is administered through the umbilical vein. During this procedure, cardiac rhythm disorders occur spontaneously, without hemodynamic imbalances, while SpO2 increases from 78% to 85% post-septostomy. In the following days, she presents progressive edema and decreased diuresis (Figure 1), for which she receives diuretic treatment (Furosemide) and Aminophylline, initially in intermittent doses, then by continuous infusion until reaching maximum doses. The renal ultrasound excludes kidney malformation or vascular thrombosis at this stage. The cardiac surgery takes place on the 12-th day of life, and the following are practiced: Jatene arterial switch, arterial canal ligation and the closure of both the muscular ventricular defect and the post-Raskind atrial defect with bovine pericardial patch. ECC (extracorporeal circulation) duration is 283 minutes and the aorta clamp time is 145 minutes. Post-surgery she remains in a critical general condition with generalized edema, mechanically ventilated, SpO2 = 96-98%, AV = 150 bpm with temporary pacemaker on epicardial electrodes and inotropic support (Adrenalin and Milrinone). In the following hours the patient presents a tendency to hypotension that requires inotropic dose escalation and supplementation with Dopamine, Dobutamine and Noradrenalin. Gradually the patient becomes oligo-anuric with important edemas and increasing serum creatinine values, so two days post-surgery she requires a decompressive sternotomy and CRRT-CVVHDF hemodiafiltration (continuous renal replacement therapy-continuous veno-venous hemodiafiltration).

It is installed using the right femoral vein approach. The Prismaflex Gambro HF 20 Set and systemic anticoagulation with Heparin is used. The evolution is favorable with the resumption of diuresis, the decrease of the edema and the decrease of serum creatinine values (Figure 1). The sternum is closed after 5 days and hemodiafiltration is interrupted after 12 days. Since AVB grade III is maintained, at 18 days post-surgery, the permanent cardiac pacemaker is mounted by implanting the electrode at the RV (right ventricle) and the device is installed anterior to the abdominal right muscle on the left side of the body. Four days since this procedure, throughout which the patient receives enteral nutrition, massive bilateral chylothorax are observed.

Treatment with Octreotide 10 µg/kg/h is established, but the pacemaker is in intimate contact with the fluid (Figures 2 and 3), so 14 days since implantation, permanent pacemaker malfunction with severe bradycardia, arterial hypotension and desaturation and cardiac arrest are observed.

After cardiopulmonary resuscitation, the temporary pacemaker is restarted. As a result, oligoanuria and important edemas nonresponsive to diuretic treatment reappear. The hemodiafiltration is reinitiated and is maintained for another 7 days. The same machine is used, the approach is on the left femoral vein and the same technique is used. This time, however, oligoanuria is maintained and the normalization of serum creatinine values is no longer observed (Figure 1). On the 41st day post-surgery, she has irreversible cardiac arrest during cardiopulmonary resuscitation.

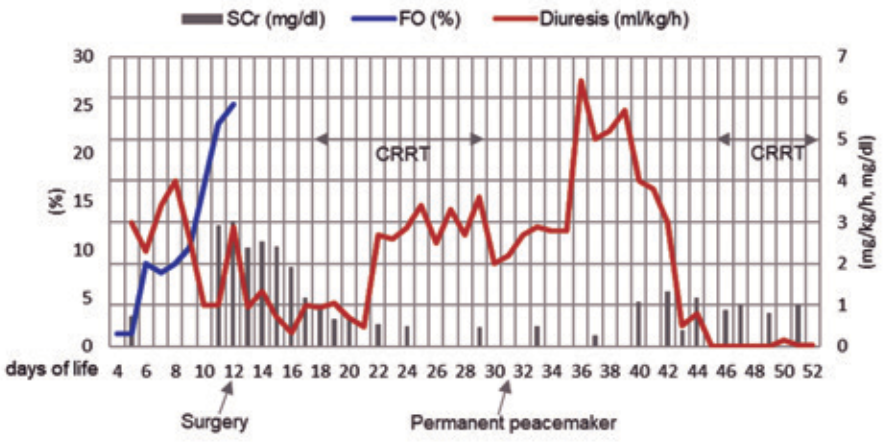

FIGURE 1. *Daily evolution of diuresis, fluids overload (FO) and serum creatinine*

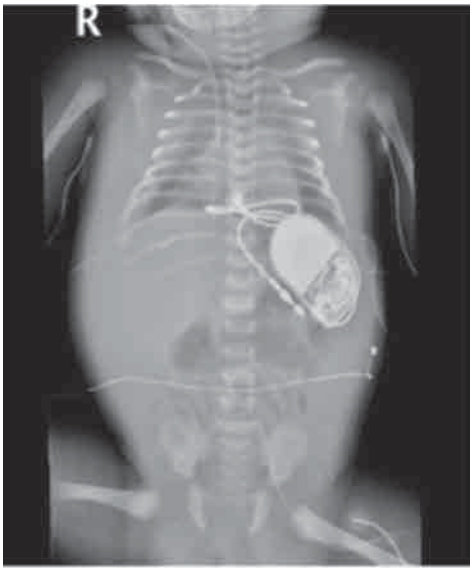

FIGURE 2. *The thoracoabdominal radiography. Right pleural effusion*

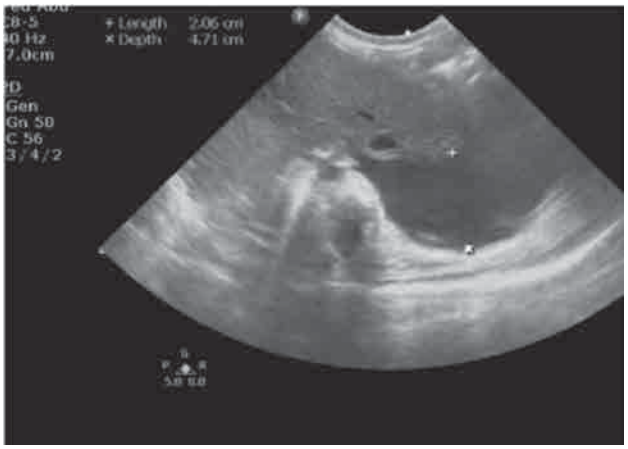

FIGURE 3. *Ultrasound. Left pleural effusion*

DISCUSSION

The acute kidney injury in our patient occurred step by step and there were several aggravating factors. Acute fetal asphyxia for which cesarean surgery was performed was continued postnatally with hypoxic-ischemic encephalopathy and post hypoxic renal impairment. The incidence of AKI in patients with neonatal encephalopathy is 41.6%, as evidenced by the Analysis of Awaken (Assessment of Worldwide Acute Kidney Epidemiology in Neonates) (4). In the first moments after a hypoxic event there is a redistribution of blood flow to maintain cerebral, cardiac and adrenal flow to the detriment of renal, gastrointestinal flow and skin infusion. The release of adenosine increases acting as a vasoconstrictor, which reduces glomerular filtration. The aggressive management of asphyxia through fluid restriction, parenteral nutrition and therapy with antibiotics could be an additional risk for AKI (4). Even congenital heart disease due to low oxygen saturation and low glucose levels in the ascending aorta can cause perinatal brain injuries (6). The renal function is achieved, in this phase, with help of diuretic treatment.

Transposition of great vessels is associated with a low systemic blood flow and decreased splanchnic blood perfusion, which may change depending on mixing in the arterial canal or interatrial septum (7). As an indicator of organ perfusion, Klauwer et al. recommend monitoring diuresis, preferably in the absence of diuretic treatment (8).

The prostaglandin used to keep the arterial canal open may cause vasodilatation leading to secondary effects such as arterial hypotension (9) and AKI. Our patient has received this treatment since her first day of life.

The septostomy is a maneuver that can cause AKI by cardiac injury or heart rhythm disorders. During the procedure, our patient experienced cardiac rhythm disorders but without hemodynamic impact. Hamer et al. conducted a study involving 71 newborns with transposition of great vessels. 50.7% of them developed AKI and 11% of them needed CRRT. 70% of patients had previously been subjected to balloon atrioseptostomy, but it is not found that atrioseptostomy is associated with an increased need for renal repletion therapy (10).

The renal function, however, was deteriorated significantly after surgery. In the literature it is specified that AKI after cardiac surgery can occur globally in 30-40% of the cases (10,11) and in neonates in an even higher percentage, up to 64% (12), depending on the complexity of the cardiac malformation. AKI generally occurs in the first 3 days postoperatively (13) but only 1% of patients operated require renal repletion therapy (10). Over the past 7 years, 62% of the total cases requiring CRRT (25 cases) in our clinic were post-operative cardiac surgical patients (14). Numerous studies have shown that renal damage after cardiac surgery has both preoperative risk factors (low age, preexisting kidney injury, complexity of malformation or preoperative mechanical ventilation) (12) as well as intraoperative risk factors (arterial hypotension, low hemoglobin, aorta surgery, ECC and aorta clamp time). The causes are multiple, such as: ischemia-reperfusion injury, mechanical trauma of blood cells in the bypass circuit, oxidative stress, myocardial edema and oxygenation deficiency. Reduction of mean arterial pressure and non-pulsatile flow during cardiopulmonary bypass leads to activation of apoptosis and death of endothelial and tubular cells (15). An ECC duration greater than 180 minutes causes AKI in about 70% of patients (11) and an aorta clamp time of over 57 minutes causes

AKI in 64% of patients (12). Transposition of the great vessels with ventricular sept defect is categorized as 4/6 RACHS-1 (Risk Adjustment for Congenital Heart Surgery) (16) and is usually associated with prolonged operating time as reported by Wetter et al. who found that the average time of ECC is 204 min (range 120-1554 min) and average aorta clamp time is 115 min (range 78-244 min) (17). In our patient´s case, the time were 283 min and respectively 145 min. Postoperatively, the main risk factors for AKI occurrence are volume overload and the need for circulatory support. Genetic predisposition may be an aggravating factor, identifying the presence of two alleles (interleukin 6-572C and angiotensinogen 842C) associated with AKI in the Caucasian population after aortic-coronary surgery (18). Because AKI after cardiac surgery is associated with increased mortality, many centers insert the peritoneal dialysis catheter during surgery or even perform prophylactic peritoneal dialysis (19,20) although lately hemodiafiltration is preferred as method of renal repletion after cardiovascular surgery.

Since admission in our NICU (day 4 of life) the patient has progressive edema, with the maximum fluid overload, calculated by (Current weight – Birth weight) / Birth weight x 100 (3), at 25% (Figure 1). Piggot et al. affirm that fluid overload greater than 30% is associated with 100% mortality (11,21) and therefore it is recommended that until meeting the criteria n-KDIGO to take into account that small variations in fluid overload can be an alarm signal and to initiate early diuretic treatment or CRRT (22).

Reopening the sternum or leaving the sternum open is one of the therapeutic options to decrease cardiac compression and to avoid hemodynamic instability (23). In our patient´s case although after the decompression of the sternum the blood pressure values were within normal limits, no increase in diuresis was obtained and serum creatinine continued to soar and the edemas were accentuated.

The complete atrioventricular block, also responsible for hemodynamic instability, is a possible complication, especially in transposition of the great vessels, that also associate a ventricular sept defect (9). In an analysis by the Pediatric Cardiac Critical Care Consortium, it is estimated that it can occur in up to 6% of cardiovascular interventions, and that in 25-60% of cases it requires a permanent pacemaker (24). The American College of Cardiology, the American Heart Association and the Heart Rhythm Society currently recommend implanting the definitive pacemaker if AVB Grade II-III is persistent after 7-10 days after surgery or if expected to be definitive (24,25). In our patient´s case, the permanent pacemaker was fitted on the 18th day post-surgery.

The chylothorax with low cardiac output, protein loss and increased infection risk are important risk factors for worsening AKI. The chylothorax occurs in 0.5-6.5% (26) of cardiovascular surgery, reaching maximum level on day 2 postoperatively, but can occur up to the 29th day, especially if it is accompanied by increased venous pressure (27). Arterial switch surgery was the most incriminated (28%) in a study conducted by Biewer ES et al. on 26 patients out of 282 heart interventions (26). When chylothorax occurs, the most important factors are: increased venous pressure, injury to the thoracic duct and formation of a thrombus in a central vessel. Increased venous pressure is a very important factor and there are authors who recommend prophylactic total nutrition if the central venous pressure is higher >15 mmHg (28). There is a strong correlation between increased CVP and increased operating times (26). In most patients with congenital heart defects, who are operated, the chylothorax is not posttraumatic but caused by lymphatic flow abnormalities (29). The thoracic duct is typically located posterior to the pericardium and is damaged only when interventions occur in the aortic arch (29). Fluid overload is also associated with increased central venous pressure and increased pressure in the vascular wall at lymphatic-venous junction (30,31). Deep thrombosis of the vessels is involved in the occurrence of chylothorax, not only postoperatively, but especially if thrombosis is present on the vessels located on the left side of the body (30,31). Reopening of the sternum is an additional risk factor, supporting the mediastinal lymph injury and impairing postoperative hemodynamics.

The aortic surgery, increased lymph production by initiating nutrition, arrhythmia that increased central venous pressure through atrioventricular asynchronism (30,32), and reopening of the sternum, could all be causes for the appearance of chylothorax in our patient.

During the second hemodiafiltration course, done without any positive hemocultures, but in the present of thrombocytopenia and increased inflammatory markers, has also directed us to the coexistence of an infection that may play an important role in worsening renal failure. The sepsis through cytokine-mediated systemic vasodilatation and capillary leak syndrome causes renal hypoperfusion and acute ischemic tubular necrosis, but it can also cause direct injury through multiple vascular and glomerular microthrombi, inflammation and vasoconstriction (33).

The nephrotoxic medication administrated (Amikacin, Vancomycin, Furosemide) should not be ignored and may have an aggravating effect in the context of kidney disease (34) although the serum level

was not dosed but however, we administered dose-adjusted from AKI.

Regarding the permanent pacemaker malfunction, Kwak et al. found, in a study involving 48 children under one year of age, 16 of whom were newborns, that the most common causes are: fracturing migration, puncture of the probe, generator dysfunction, infections, generator migration or skin necrosis (35). Postcardiac injury syndrome may occur in adults after assembling the pacemaker (36) which associates exudative pleural fluid with immune etiology (37), but very rarely in the pediatric population. We have not found any cases of permanent stimulator and chylothorax in the literature and the association of chylothorax and AVB is mentioned only in the context of genetic syndromes (38). In the case of our patient, it was not possible to determine exactly the nature of the stimulator dysfunction and it was replaced with a temporary one with epicardial threads.

It is rare in Neonatal Intensive Care Unit for a patient to require repeated and spaced hemodiafiltration sessions during the same admission. In our clinic it is the only case out of the 25 hemodiafiltrations performed in the last 6 years. A second AKI episode is usually expected in at-risk populations such as preterm newborns, kidney malformation, thrombosis of renal vessels, necrotizing enterocolitis, multiple cardiovascular surgery (39).

CONCLUSIONS

AKI is one of the major causes of death in patients with congenital heart disease. The association of several consecutive causes with different etiology of renal injury may be fetal in this group of patients.

This is a complex case, with multiple risk factors for kidney injury: acute fetal asphyxia and hypoxic-ischemic encephalopathy, cardiac malformation, prostaglandin, atrial-septostomy, cardiovascular surgery, reopening of the sternum, complete atrioventricular block, chylothorax, sepsis, heart stimulator malfunction and which despite maximum treatment, the patient did not survive.

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LEZIUNILE RENALE MULTIPLE DUC LA DECES ÎN CHIRURGIA CARDIACĂ CHIAR ŞI CU HEMODIAFILTRARE PRECOCE

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REZUMAT

Prezentăm cazul unui nou-născut diagnosticat cu suferință perinatală și injurie renală secundară, transpoziție de vase mari și debit sistemic scăzut, tratat cu prostaglandină, atrioseptostomie, urmate de operația de switch arterial. Postoperator, pacientul este oliguric și necesită hemodiafiltrare pentru 12 zile, după care funcția renală este restabilită. În evoluție, apare bloc atrioventricular (BAV) grad III, urmat de implantare de stimulator cardiac permanent, dar o altă complicație postoperatorie – chilotorax – duce la oprirea stimulării electrice, urmată de disfuncție cardiacă severă și, consecutiv, injurie renală recurentă și anurie. Se reinstituie hemodiafiltrarea pentru încă 7 zile, fără recuperarea funcției renale. Asfixia perinatală, malformația cardiacă cu debit sistemic scăzut, prostaglandina, atrioseptostomia, circulația extracoroporeală, tulburările de ritm, chilotoraxul, sepsisul, stopul cardiac sunt factori intricaţi care aduc injurie renală importantă. Asocierea lor scade major șansele de supraviețuire chiar dacă pacientul beneficiază de tratament suportiv și hemodiafiltrare precoce.

Cuvinte cheie: hemodiafiltrare, insuficiență renală acută, nou-născut, transpoziţie de vase mari, malfuncţie stimulator cardiac permanent, chilotorax

INTRODUCERE

Insuficiență renală acută (IRA) la nou-născut continuă să fie o cauză importantă de morbiditate și mortalitate, 25-30% dintre nou-născuții din TINN (Terapie Intensivă nou-născuți) având afectare renală (1). Deși nu este un consens în ceea ce privește diagnosticul, tot mai multe clinici utilizează criteriile KDIGO (Kidney Disease Improving Global Outcomes) modificate pentru nou-născut (n-KDIGO), care au la bază scăderea diurezei sub 1 ml/kg/oră și creșterea creatininei serice cu mai mult de 0,3 mg/dl sau peste 50% din valoarea anterioară (2). Dificultătile sunt legate de faptul că, în primele 72 ore, nivelul creatininei poate fi crescut, în concordanţă cu nivelul seric al mamei, iar cele mai multe injurii renale la nou-născut, în fazele incipiente, sunt nonoligurice (3). Acest lucru se datorează faptului că nou-născuții au o imaturitate a funcției tubulare renale, cu o capacitate limitată de concentrare a urinei și un procent mare de apă în compoziția corpului (4). În 85% dintre cazuri, IRA neonatală este de etiologie prerenală, putând deveni intrinsecă în contextul prelungirii injuriei renale. Deshidratarea, asfixia perinatală, malformațiile renale, trombozele vaselor renale, sepsisul și chirurgia cardiovasculară sunt printre cele mai frecvente cauze de afectare renală la nou-născut (5), dar există și cauze mai puțin frecvente, precum bolile metabolice, tulburările de ritm sau chilotoraxul.

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