

# Multiple renal injuries lead to death in postoperative cardiac surgery even with precocious hemodiafiltrations

Mihaela Bizubac<sup>1</sup>, Catalin Cirstoveanu<sup>1,2</sup>, Cristina Filip<sup>3</sup>, Alin Nicolescu<sup>3</sup>,  
Ileana Barascu<sup>1</sup>, Ruxandra Chirca<sup>1</sup>, Alina Gaiduchevici<sup>1</sup>, Doina Anca Plesca<sup>2,4</sup>

<sup>1</sup>Neonatal Intensive Care Unit, "M.S.Curie" Emergency Children's Hospital, Bucharest, Romania

<sup>2</sup>"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

<sup>3</sup>Pediatric Cardiology, "M.S.Curie" Emergency Children's Hospital, Bucharest, Romania

<sup>4</sup>"Dr. Victor Gomoiu" Clinical Children's Hospital, Bucharest, Romania

## 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.

*Corresponding author:*  
Catalin Cirstoveanu  
E-mail: cirstoveanu@yahoo.com

*Article History:*  
Received: 13 March 2021  
Accepted: 17 March 2021

## 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 SpO<sub>2</sub> 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, SpO<sub>2</sub> = 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 venovenous 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.

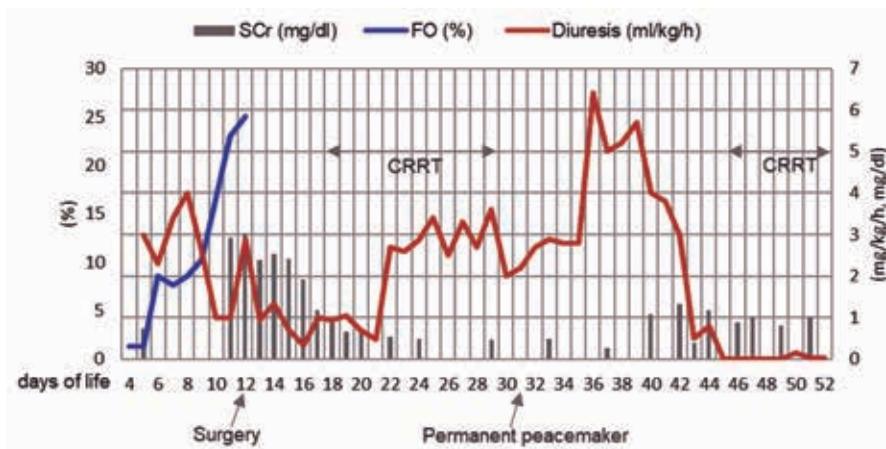
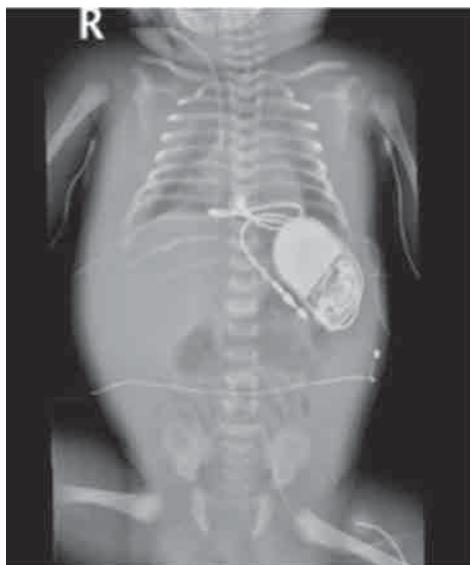


FIGURE 1. Daily evolution of diuresis, fluids overload (FO) and serum creatinine



**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 perfusion. 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 replacement 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 replacement 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  $(\text{Current weight} - \text{Birth weight}) / \text{Birth weight} \times 100$  (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 pre-term 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.

## REFERENCES

1. Jetton JG, Boohaker LJ, Askenazi DJ; Neonatal Kidney Collaborative (NKC). Incidence and outcomes of neonatal acute kidney injury (AWAKEN): a multicentre, multinational, observational cohort study. *Lancet Child Adolesc Health*. 2017 Nov;1(3):184-194.
2. Selewski DT, Akcan-Arikan A, Guillet R; Neonatal Kidney Collaborative. The impact of fluid balance on outcomes in critically ill near-term/term neonates: a report from the AWAKEN study group. *Pediatr Res*. 2019 Jan;85(1):79-85.
3. Askenazi DJ, Stojanović V. Neonatal Critical Care Nephrology. In: Deep A, Goldstein SL. *Critical Care Nephrology and renal replacement therapy in children*. Springer International Publishing AG 2018:63-71.
4. Kirkley MJ, Boohaker L, Griffin R, Soranno DE, Gien J, Askenazi D, Gist KM; Neonatal Kidney Collaborative (NKC). Correction to: Acute kidney injury in neonatal encephalopathy: an evaluation of the AWAKEN database. *Pediatr Nephrol*. 2019 Feb;34(2):363.
5. Selewski DT, Charlton JR, Jetton JG, Guillet R, Mhanna MJ, Askenazi DJ, Kent AL. Neonatal Acute Kidney Injury. *Pediatrics*. 2015 Aug;136(2):e463-73.
6. Owen M, Shevell M, Majnemer A, Limperopoulos C. Abnormal brain structure and function in newborns with complex congenital heart defects before open heart surgery: a review of the evidence. *J Child Neurol*. 2011 Jun;26(6):743-55.
7. Files MD, Arya B. Preoperative Physiology, Imaging, and Management of Transposition of the Great Arteries. *Semin Cardiothorac Vasc Anesth*. 2015 Sep;19(3):210-22.
8. Klauwer D, Neuhaeuser C. Renal Aspects of Cardiac Intensive Care. In: Klauwer D, Neuhaeuser C, Thoul J, Zimmermann R. *A Practical Handbook on Pediatric Cardiac Intensive Care Therapy*. Springer International Publishing AG 2019:103-136.
9. Dorfman AL. Transposition of the Great Arteries. In: Alboliras ET, Hijazi ZM, Lopez L, Hagler DJ. *Visual Guide to Neonatal Cardiology*. John Wiley and Sons Ltd 2018:194-198.
10. Harmer MJ, Southgate G, Smith V. Acute kidney injury and short-term renal support in the post-operative management of neonates following repair of transposition of the great arteries. *Progress in Pediatric Cardiology*. 2019;(52):26-32.
11. Li S, Krawczeski CD, Zappitelli M; TRIBE-AKI Consortium. Incidence, risk factors, and outcomes of acute kidney injury after pediatric cardiac surgery: a prospective multicenter study. *Crit Care Med*. 2011 Jun;39(6):1493-9.
12. Morgan CJ, Zappitelli M, Robertson CM; Western Canadian Complex Pediatric Therapies Follow-Up Group. Risk factors for and outcomes of acute kidney injury in neonates undergoing complex cardiac surgery. *J Pediatr*. 2013 Jan;162(1):120-7.e1.
13. Zappitelli M, Bernier PL, Saczkowski RS. A small post-operative rise in serum creatinine predicts acute kidney injury in children undergoing cardiac surgery. *Kidney Int*. 2009 Oct;76(8):885-92.
14. Cirstoveanu C, Bizubac M, Plesca A. Hemodiafiltration at neonates and small infants: a small retrospective case series. *American College of Chest Physicians* 2020.
15. Cooper DS, Ricci Z. Post Cardiac Surgery. Acute Kidney Injury and Cardiorenal Syndromes. In: Deep A, Goldstein SL. *Critical Care Nephrology and renal replacement therapy in children*. Springer International Publishing AG 2018:99-110.
16. Cavalcanti PE, Sá MP, Santos CA. Stratification of complexity in congenital heart surgery: comparative study of the Risk Adjustment for Congenital Heart Surgery (RACHS-1) method, Aristotle basic score and Society of Thoracic Surgeons-European Association for Cardio-Thoracic Surgery (STS-EACTS) mortality score. *Rev Bras Cir Cardiovasc*. 2015 Mar-Apr;30(2):148-58.
17. Wetter J, Belli E, Sinzobahamvya N. Transposition of the great arteries associated with ventricular septal defect: surgical results and long-term outcome. *Eur J Cardiothorac Surg*. 2001 Oct;20(4):816-23.
18. Stafford-Smith M, Podgoreanu M, Swaminathan M; Perioperative Genetics and Safety Outcomes Study (PEGASUS) Investigative Team. Association of genetic polymorphisms with risk of renal injury after coronary bypass graft surgery. *Am J Kidney Dis*. 2005 Mar;45(3):519-30.
19. Webb TN, Basu R, Askenazi D. Diagnosis and Management of Acute Kidney Injury in Critical Illness. In: Mastropietro CW, Valentine KM. *Pediatric Critical Care – Current Controversies*. Springer Nature Switzerland AG 2019:177-192.

20. DeSena HC, Nelson DP, Cooper DS. Cardiac intensive care for the neonate and child after cardiac surgery. *Curr Opin Cardiol*. 2015 Jan;30(1):81-8.
21. Piggott KD, Soni M, Decampli WM. Acute Kidney Injury and Fluid Overload in Neonates Following Surgery for Congenital Heart Disease. *World J Pediatr Congenit Heart Surg*. 2015 Jul;6(3):401-6.
22. Ueno K, Shiokawa N, Takahashi Y. Kidney Disease: Improving Global Outcomes in neonates with acute kidney injury after cardiac surgery. *Clin Exp Nephrol*. 2020 Feb;24(2):167-173.
23. Elassal AA, Eldib OS, Dohain AM, Abdelmohsen GA, Abdalla AH, Al-Radi OO. Delayed Sternal Closure in Congenital Heart Surgery: A Risk-Benefit Analysis. *Heart Surg Forum*. 2019 Aug 27; 22(5):E325-E330.
24. Romer AJ, Tabbutt S, Etheridge SP. Atrioventricular block after congenital heart surgery: Analysis from the Pediatric Cardiac Critical Care Consortium. *J Thorac Cardiovasc Surg*. 2019 Mar; 157(3):1168-1177.e2.
25. Liberman L, Silver ES, Chai PJ, Anderson BR. Incidence and characteristics of heart block after heart surgery in pediatric patients: A multicenter study. *J Thorac Cardiovasc Surg*. 2016 Jul; 152(1):197-202.
26. Biewer ES, Zürn C, Arnold R. Chylothorax after surgery on congenital heart disease in newborns and infants -risk factors and efficacy of MCT-diet. *J Cardiothorac Surg*. 2010 Dec 13;5:127.
27. Czobor NR, Roth G, Prodán Z. Chylothorax after pediatric cardiac surgery complicates short-term but not long-term outcomes-a propensity matched analysis. *J Thorac Dis*. 2017 Aug; 9(8):2466-2475.
28. Panthongviriyakul C, Bines JE. Post-operative chylothorax in children: an evidence-based management algorithm. *J Paediatr Child Health*. 2008 Dec;44(12):716-21.
29. Savla JJ, Itkin M, Rossano JW, Dori Y. Post-Operative Chylothorax in Patients With Congenital Heart Disease. *J Am Coll Cardiol*. 2017 May 16;69(19):2410-2422.
30. Perry T, Bora K, Bakar A, Meyer DB, Sweberg T. Non-surgical Risk Factors for the Development of Chylothorax in Children after Cardiac Surgery-Does Fluid Matter? *Pediatr Cardiol*. 2020 Jan;41(1):194-200.
31. Ratnayake CBB, Escott ABJ, Phillips ARJ, Windsor JA. The anatomy and physiology of the terminal thoracic duct and ostial valve in health and disease: potential implications for intervention. *J Anat*. 2018 Jul;233(1):1-14.
32. Chua Chiaco JM, Parikh NI, Fergusson DJ. The jugular venous pressure revisited. *Cleve Clin J Med*. 2013 Oct;80(10):638-44.
33. Schrier RW, Wang W. Acute renal failure and sepsis. *N Engl J Med*. 2004 Jul 8;351(2):159-69.
34. Hanna MH, Askenazi DJ, Selewski DT. Drug-induced acute kidney injury in neonates. *Curr Opin Pediatr*. 2016 Apr;28(2):180-7.
35. Kwak JG, Cho S, Kim WH. Surgical Outcomes of Permanent Epicardial Pacing in Neonates and Young Infants Less Than 1 Year of Age. *Heart Lung Circ*. 2019 Jul;28(7):1127-1133.
36. Kumar S, Madanieh A, Patel H, Srinivasa Murthy R, Goyas JM, Milunski MR. Large Unilateral Pleural Effusion with Pacemaker-associated Post-cardiac Injury Syndrome. *Cureus*. 2018 Jul 8;10(7):e2946.
37. Lee YJ, Mubasher M, Zainal A, Syed T, Mohamed MFH, Ferrantino M, Hoefen R. Pacemaker-Associated Post-cardiac Injury Syndrome Presenting with Tamponade and Recurrent Pleural Effusion. *Clin Med Insights Case Rep*. 2020 Oct 30;13:1179547620965559.
38. Fujino S, Maruyama H, Tsukamoto K, Ono H, Isayama T, Ito Y. Chylothorax Associated with Congenital Complete Atrioventricular Block. *AJP Rep*. 2020 Oct;10(4):e403-e407.
39. Charlton JR, Boohaker L, Askenazi D, Brophy PD, D'Angio C, Fuloria M, Gien J, Griffin R, Hingorani S, Ingraham S, Mian A, Ohls RK, Rastogi S, Rhee CJ, Revenis M, Sarkar S, Smith A, Starr M, Kent AL; Neonatal Kidney Collaborative. Incidence and Risk Factors of Early Onset Neonatal AKI. *Clin J Am Soc Nephrol*. 2019 Feb 7; 14(2):184-195.

*Conflict of interest:* none declared

*Financial support:* none declared