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**Case Report / Приказ болесника**

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# Challenges in the treatment of hypotension in an extremely preterm neonate

## Изазови у лечењу хипотензије код екстремно незрелог новорођенчета

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## Изазови у лечењу хипотензије код екстремно незрелог новорођенчета

**SUMMARY**

**Introduction** Hypotension is often in preterm neonates, as a result of various factors such as immature myocardium, transitional circulation, perinatal hypoxia, positive pressure ventilation and relative adrenal insufficiency. The leading causes of hypotension in preterm neonates are sepsis and septic shock, patent ductus arteriosus and necrotizing enterocolitis.

**Case outline** The female preterm neonate delivered at $24^{1/7}$ weeks of gestation with birth weight of 710 g and Apgar score of 3 in first minute after birth. The hypotension was registered from the first day of life, so dopamine was administered. The neonate was admitted to Institute of Neonatology on the fourth day of life and we continued dopamine and dobutamine. Despite increasing the dose, the hypotension was still persistent and we started epinephrine. Echocardiography showed patent ductus arteriosus and signs of heart failure. The neonate's condition was complicated by acute renal injury. Therefore, ibuprofen was not used to close the patent ductus arteriosus, so we continued its conservative treatment. In order to correct the hypotension, hydrocortisone was added to the therapy. Despite conservative treatment of the patent ductus arteriosus, hypotension was persistent and severe, and its treatment lasted for four weeks of hospitalization. After achieving hemodynamic and respiratory stability, surgical ligation of the patent ductus arteriosus was performed.

**Conclusion** Hypotension is a complication in early neonatal period in extremely preterm neonate. Knowing the cause, as well as the pathophysiology, allows the selection of the appropriate drug and proper treatment of hypotension in preterm neonates, which is very challenging for neonatologists.

**Keywords**: premature newborn; ductus arteriosus; hypotension; cardiovascular agents

**САЖЕТАК**

**Увод** Хипотензија је честа код претерминске новорођенчади, као последица различитих фактора као што су незрели миокард, транзиторна циркулација, перинатална хипоксија, вентилација позитивним притиском и релативна адренална инсуфицијенција. Водећи узроци хипотензије код претерминске новорођенчади су сепса и септични шок, отворен артеријски канал и некротизирајући ентероколитис.

**Приказ болесника** Женско претерминско новорођенче рођено је у $24^{1/7}$ недељи гестације, са телесном масом на рођењу од 710 *g* и Апгар скором од 3 у првом минуту после рођења. Хипотензија је регистрована од првог дана живота, па је примењиван допамин. Новорођенче је примљено у Институт за неонатологију у четвртом дану живота и ми смо наставили допамин и добутамин. Упркос повећању дозе, хипотензија је и даље била упорна, због чега је примењен адреналин. Ехокардиографски преглед је показао постојање отвореног артеријског канала и знакова срчане инсуфицијенције. Стање новорођенчета је било компликовано акутним оштећењем бубрега. Због тога ибупрофен није коришћен у затварању отвореног артеријског канала, па је настављено његово конзервативно лечење. У циљу корекције хипотензије у терапију је додат хидрокортизон. Упркос конзервативном лечењу отвореног артеријског канала, хипотензија је била упорна и тешка, а њена терапија је трајала четири недеље хоспитализације. Након постизања хемодинамске и респираторне стабилности, урађена је хируршка лигација отвореног артеријског канала.

**Закључак** Хипотензија је компликација у раном неонаталном периоду код екстремно незреле новорођенчади. Познавање узрока, као и патофизиологије, омогућава одабир одговарајућег лека и правилно лечење хипотензије код претерминске новорођенчади, што је за неонатологе веома изазовно.

**Кључне речи:** претерминско новорођенче; артеријски канал; хипотензија; кардиоваскуларни агенси



## INTRODUCTION

Hypotension is often encountered in preterm neonates. Various factors are associated with the hypotension in preterm neonates such as immature myocardium, transitional circulation, perinatal hypoxia, positive pressure ventilation (PPV) after birth and transient or relative adrenal insufficiency. The leading causes of hypotension in preterm neonates are sepsis and septic shock, patent ductus arteriosus (PDA) and necrotizing enterocolitis (NEC) [1, 2, 3]. Patent

 

ductus arteriosus is the common cause of low diastolic blood pressure (BP) as a result of the diastolic outflow of blood into the pulmonary circulation from the left-to-right (L-R) shunt, which makes it difficult to treat [4]. Therefore, pharmacotherapy should be adjusted according to the pathophysiology of the condition that caused hypotension [1, 5, 6]. We report a case of the extremely preterm (EPT) neonate with severe and persistent hypotension caused by hemodynamically significant PDA.

## CASE REPORT

The preterm female neonate was delivered vaginally at $24^{1/7}$ weeks of gestation with birth weight of 710 g and Apgar score of 3 in first minute after birth. The pregnancy was properly controlled, complicated by the mother's vaginal bleeding one month before delivery. There was no infection during pregnancy. Immediately after birth, the neonate was intubated. In Neonatal Intensive Care Unit (NICU) in Maternity Hospital, the neonate was placed on conventional mechanical ventilation (MV) and surfactant was administered. The neonate received dual antibiotic therapy (ampicillin and gentamicin) and total parenteral nutrition (TPN). In order to stimulate of breathing, a preparation of methylxanthine, caffeine, was used. Because of hypotension, the neonate received a fluid bolus and during the first three days of life (DOL), inotropic support by dopamine was applied. Also, the neonate received red blood cell (RBC) transfusion in order to correct anemia.

The neonate was admitted to Institute of Neonatology on the 4th DOL, in severe condition, orotracheally intubated, with a weight of 590 g, heart rate (HR) of 126 beats/min, BP of 61/28/41 mmHg and respiratory rate (RR) of 52 breaths/min. Oxygen saturation ($SpO_2$) was 94% on mechanical ventilation with fraction of inspired oxygen ($FiO_2$) of 0.4. In our NICU, conventional MV was continued. Blood gas analysis showed metabolic acidosis. Chest X-ray showed signs of respiratory distress syndrome (RDS) with moderate cardiomegaly. In the following hours after admission to the Institute, hypotension, predominantly diastolic hypotension, and oliguria were registered in the neonate, so inotropic support by dopamine and dobutamine in minimal doses was started. At the same time, cardiac auscultation was normal, without heart murmurs. Despite increasing the dose of dopamine and dobutamine, the hypotension was still persistent (minimal diastolic BP was 10 mmHg), so on the second day of hospitalization, the administration of epinephrine was started. Echocardiography showed PDA measuring 2 mm with a L-R shunt of blood flow, as well as signs of heart failure. There was a massive regurgitation on both AV valves. Also, patent foramen ovale (PFO) was also present, with L-R shunt



 

of blood flow. Because of acute kidney injury (stage 3 KDIGO classification) and green gastric residuals, so ibuprofen was not used to close PDA. Restriction of total fluid intake (120 ml/kg per day) was applied. In accordance with BP values and diuresis, the dose of epinephrine was gradually increased to the maximum. After a short phase of maintaining stable BP values and diuresis at maximum doses of epinephrine, a gradual reduction in the dose of epinephrine was started. Diastolic hypotension then occured again, so at the end of the first week of hospitalization, in addition to epinephrine, hydrocortisone was introduced for the therapy of hypotension. In the following days, the doses of epinephrine and hydrocortisone were gradually titrated. Control echocardiography was performed four days later after first examination and the findings were without significant changes. Because of heart failure, intermittent administration of loop diuretics such as furosemide was started. In order to treat hypotension and stimulate diuresis, the neonate still required very high doses of inotropes and hydrocortisone. Control echocardiography findings were without significant changes. At the end of the third week of hospitalization, we started to gradually reduce the doses of inotropes based on BP values and diuresis. In order to maintain normal BP values, hydrocortisone was administered until the end of the fourth week of hospitalization in minimal daily doses. Blood pressure values, as well as the inotropic and/or vasopressor therapy applied in our case, are shown in Table 1. When the neonate was hemodynamically stabilized, a cardiosurgical evaluation was performed. Surgical ligation of PDA was performed on the 43$^{rd}$ DOL without complications.

The neonate was on conventional MV for 46 days, then on non-invasive respiratory support (nasal Continuous Positive Airway Pressure, nCPAP) for 10 days and further on oxygen therapy until the age of three months (corrected 36 weeks of gestation). Sepsis screening was positive for late-onset sepsis for *Staphylococcus haemolyticus*. The head ultrasound (HUS) at 7$^{th}$ DOL showed cerebellar hemorrhage. During control HUS registered a gradual reduction of cerebellar hemorrhage. Because of neonatal seizures, the neonate was treated with phenobarbital and midazolam. Electroencefalography (EEG) showed irregularity and depression of basic activity with epileptic discharges. On control EEG examinations, there was no epileptic discharges, the neonate had no neonatal seizures, so discharged home without antiseizure medications. During hospitalization the neonate received five RBC transfusions and one platelet transfusion. Minimal enteral nutrition was started at the end of the 2$^{nd}$ week of life. Full enteral nutrition was achieved in the 6th week of hospitalization. At the age of 2 months and 10 days, due to retinopathy of prematurity (ROP) intravitreal application of anti-VEGF was performed. The



 

neonate was discharged home in the 4th month of life (corrected 40 weeks of gestation) with a weight of 2700 g (less than the 10$^{th}$ percentile for the gestational age).

**Ethics:** Manuscript has been written in accordance with the ethical standards of the Declaration of Helsinki. The publication of the data in the manuscript was approved by the Ethics Committee of the Institute of Neonatology (No. 2398/3).

## DISCUSSION

After birth, there are complex and sudden changes that mostly affect the cardiovascular and respiratory system. In healthy full-term neonates, rapid closure of the ductus arteriosus (DA) occurs at birth, which is one of the key changes during the transition from fetal to neonatal circulation. However, in preterm neonates, constriction and permanent closure of the DA at birth are delayed, which may be associated with neonatal morbidity and mortality. The risk for PDA is inversely proportional to the gestational age (GA). It is estimated that about 87% of neonates born at 24 weeks of gestation have a symptomatic PDA [3, 7, 8].

Hypotension is especially common in preterm neonates during the first 72 hours of life, as a result of large transient adaptive changes in the respiratory, cardiovascular and neuroendocrine systems. There is wide variation in the definition of hypotension in preterm neonates. One of the most common definitions of hypotension in preterm neonates used in clinical practice is given by the British Association of Perinatal Medicine. According to this association, hypotension is defined as a value of mean BP that is less than the weeks of gestation. According to the second, hypotension in preterm neonates is defined as mean BP below the 5th or 10th percentile for the weeks of gestation and postnatal age. Also, hypotension in preterm neonates is defined as a mean BP value below 30 mmHg. This definition is particularly useful for neonates after 72 hours of life [1, 6]. Hypotension associated with PDA can be challenging and difficult to treat [4, 9].

Preterm neonates are usually treated for hypotension in the first 24 hours of life. Batton et al. [10] in prospective observational study showed that 55% of EPT neonates received therapy for hypotension and 28% received a vasoactive medication. Our patient was EPT female neonate and had a hypotension within 24 hours of life, so she received a fluid bolus and dopamine in Maternity Hospital. The first-line therapy for hypotension in the preterm neonates in many NICU is administration of a fluid bolus followed by inotropes such as dopamine. However, in neonates with PDA and heart failure a fluid bolus in hypotension therapy might be harmful.



 

Also, dopamine, although it increases BP can have a negative effect on cerebral perfusion and cerebral autoregulation in very low birth weight (VLBW) neonates (birth weight < 1500 g), which can increase the risk of intraventricular hemorrhage (IVH) [6, 9, 11].

In accordance with the guidelines and recommendations [12], if hypotension is caused by myocardial dysfunction, the first-line therapy is dobutamine, which achieves its effect by stimulating β-1 adrenergic receptors. Dobutamine increases myocardial contractility, HR, right and left ventricular output and mean BP. As a second-line therapy, consider dopamine, whose effect on BP and different organs is dose-dependent. In doses that are most often used in NICU, 3-10 mcg/kg/min, by stimulating adrenergic and dopaminergic receptors, dopamine increases HR, myocardial contractility and BP [1, 6].

Epinephrine is the third-line therapy for severe and persistent hypotension caused by myocardial dysfunction. Its hemodynamic effects, which is achieved through alpha and beta adrenergic receptors, are dose-dependent. In low doses (0.01 – 0.1 mcg/kg/min) epinephrine stimulates the α-2, β-1 and β-2 adrenergic receptors, which causes peripheral vasodilatation, increased miocardial contractility and HR, and finally increases BP. At higher doses (> 0.1 mcg/kg/min) epinephrine achieves effects through α-1 adrenergic receptors whose activation causes peripheral vasoconstriction and increased HR [1, 6, 12]. Also, corticosteroids, such as hydrocortisone, are used as adjunctive or rescue therapy for refractory hypotension in preterm neonates, i.e. who are hypotensive despite the administration of two inotropes. Their short-term use seems safe; however, questions is raised about their impact on neurodevelopmental outcome in preterm neonates [13, 14].

In addition to dopamine, dobutamine and epinephrine, norepinephrine is also used in the therapy of hypotension in neonates. Norepinephrine is an endogenous sympathomimetic that is a potent alpha-1 agonist, with moderate to weak effects on beta-1 and beta-2 adrenergic receptors. Because the effect on β-2 adrenergic receptors is minimal, norepinephrine has combined inotropic and peripheral vasoconstrictive effects. Norepinephrine is recommended as first-line treatment for septic shock in adults, children, and full-term neonates [15]. It is used in less than 5% of preterm neonates with shock as second- or third-line therapy [16, 17]. Preterm neonates have been shown to have higher levels of catecholamines, which may account for a weaker response to norepinephrine [18]. Although the effect of norepinephrine on blood pressure in preterm and full-term neonates with septic shock, pulmonary hypertension or isolated systemic hypotension has been confirmed in several retrospective studies with significant improvement



 

in diuresis, arterial blood gas values and tissue perfusion, in clinical practice it is traditionally used as second- or third-line therapy or as an adjuvant anti-hypotensive drug [19].

Milrinone is a type III phosphodiesterase inhibitor that acts on the myocardium by increasing intracellular concentrations of cyclic AMP and calcium by inhibiting the breakdown of cAMP. It has positive inotropic and lusitropic effects on the myocardium, while reducing systemic and pulmonary vascular resistance [20]. Clinical studies on the use of milrinone in neonates have mainly focused on the treatment of persistent pulmonary hypertension of newborn (PPHN), postoperative use after corrective cardiac surgery, and prevention and treatment of low left ventricular output syndrome after DAP ligation. However, the use of this drug also carries a risk of severe hypotension [21, 22].

Vasopressin is also used in the treatment of hypotension. It is synthesized as a prohormone, primarily in hypothalamic neurons. It plays a key role in the control of blood pressure, osmotic balance, kidney function and sodium homeostasis. More recent clinical studies have confirmed the efficacy of vasopressin as rescue therapy in neonates with PPHN and catecholamine-refractory hypotension [23].

Levosimendan, a pyridazinone-dinitrile derivative, is a calcium sensitizer with positive inotropic and vasodilatory properties. It achieves its inotropic effect by selectively binding to cardiac troponin, improves myocardial contractility by increasing the sensitivity of contractile myofilaments to intracellular calcium. Data on the use of levosimendan in neonates and children are limited. Relevant systematic reviews have reported have been shown good effects on cardiac functions, but there is no significant difference in hemodynamic effects compared to standard inotropic therapy (including dopamine, dobutamine and milrinone). Some studies have shown a potentially positive effect on cardiovascular function in preterm neonates with pulmonary hypertension and its safety. Although in some studies it has shown promising effects in improving hemodynamics in neonates, its specific use for DAP induced hypotension is not well established. Additional research is needed to determine its effectiveness and safety in the treatment of hypotension associated with DAP. For now, levosimendan remains an innovative therapeutic option for the treatment of severe cardiac dysfunction and pulmonary hypertension in preterm neonates [24, 25].

Clinical and laboratory findings in our case showed hemodynamically significant PDA, which was also confirmed by echocardiographic examination. There were contraindications for the use of ibuprofen in order to close the PDA. At that time, acetaminophen was not introduced in the Institute as a therapy for closing the PDA, so conservative treatment was applied. First of



 

all, along with the previously started antihypotension therapy, fluid restriction was implemented with optimal ventilation, i.e. increasing positive end-expiratory pressure (PEEP). Hemodynamic assessment of the patient was performed daily based on clinical picture, blood gas analysis, serum lactate concentration, BP values and diuresis, with occasional echocardiographic examinations. Despite early recognition and conservative treatment of the PDA in our case, hypotension was persistent and severe, and its treatment lasted for four weeks of hospitalization. There was no effect on closing the PDA by conservative treatment, so after achieving hemodynamic and respiratory stability, surgical ligation of the PDA was performed.

With advances in technology and neonatal intensive therapy and care, the survival of preterm neonates, especially EPT neonates, has increased. Along with their survival, as a consequence of immaturity, neonatologists are increasingly encountering complications of premature birth in the early neonatal period, among which is hypotension. Knowing the cause, as well as the pathophysiology, allows the selection of the appropriate drug and proper treatment of hyotension in preterm neonates. In order to reduce the adverse effects of drugs, a gradual titration of the dose is required along with the monitoring of the hemodynamic state of the neonate.

**Conflict of interest:** None declared.



 

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**Table 1.** Blood pressure values and inotropic and/or vasopressor therapy in our patient

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **First week of hospitalization** | **BP** | 37/12 | 37/11 | 43/15 | 42/15 | 32/10 | 41/16 | 40/17 |
| | **Th** | dop + dob | dop + dob | epi | epi | epi | epi | epi + hydr |
| **Second week of hospitalization** | **BP** | 36/17 | 46/20 | 58/22 | 37/18 | 46/23 | 49/26 | 44/15 |
| | **Th** | epi + hydr | epi + hydr | epi + hydr | epi + hydr | epi + hydr | epi + hydr | dop + dob + hydr |
| **Third week of hospitalization** | **BP** | 42/19 | 50/25 | 63/32 | 56/21 | 44/22 | 51/29 | 43/16 |
| | **Th** | dop + dob + hydr | dop + dob + hydr | dop + dob + hydr | dop + dob + hydr | dop + dob + hydr | dop + dob + hydr | dop + dob + hydr |
| **Fourth week of hospitalization** | **BP** | 56/20 | 52/30 | 56/24 | 51/20 | 59/27 | 64/35 | 64/34 |
| | **Th** | dop + dob + hydr | dop + dob + hydr | dop + dob + hydr | dop + dob + hydr | hydr | hydr | |

BP – blood pressure; dop – dopamine; dob – dobutamine; epi – epinephrine; hydr – hydrocortisone



 