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Author for correspondence:
Dilek Yavuzcan Ozturk, MD, Department of Neonatology, Istanbul Saglik Bilimleri University Basaksehir Cam and Sakura Hospital, Başakşehir Mahallesi G-434 Caddesi No: 2L Başakşehir, Istanbul, Turkey.
Tel: +90 212 909 60 00; Fax: +90 212 909 60 00.
E-mail: drdileky@gmail.com

Evaluation of perfusion index and left ventricular output changes in low cardiac output syndrome after arterial switch operation

Dilek Yavuzcan Öztürk[1], Erkut Öztürk[2], Rahime Tuten Dıkmen[2], Hatice Dilek Ozcanoglu[3], Hamza Halil Toprak[2], Behzat Tuzun[4], İbrahim Cansaran Tanıdır[2], Ali Can Hatemi[4] and Merih Cetinkaya[1]

[1]Department of Neonatology, Istanbul Saglik Bilimleri University Basaksehir Cam and Sakura Hospital, Istanbul, Turkey; [2]Department of Pediatric Cardiology, Istanbul Saglik Bilimleri University Basaksehir Cam and Sakura Hospital, Istanbul, Turkey; [3]Department of Anaesthesiology and Reanimation, Istanbul Saglik Bilimleri University Basaksehir Cam and Sakura Hospital, Istanbul, Turkey and [4]Department of Pediatric Cardiovascular Surgery, Istanbul Saglik Bilimleri University Basaksehir Cam and Sakura Hospital, Istanbul, Turkey

Abstract

Introduction: Transposition of great arteries is one of newborns' most common cyanotic CHDs, and its treatment is arterial switch operation in the first days of life. Low cardiac output syndrome may develop in the early postoperative period. In this study, we evaluated perfusion index and left ventricular output blood flow changes in patients who underwent arterial switch operation and developed low cardiac output syndrome. *Methods:* This study was conducted prospectively in newborns with transposition of great arteries who underwent arterial switch operation between 1st August 2020 and 1st August 2022. Low cardiac output syndrome score and left ventricular output were investigated. Initially, 6th, 12th, 18th, and 24th hour perfusion index and left ventricular output values of patients with and without low cardiac output syndrome were recorded. The results were evaluated statistically. *Results:* A total of 60 patients were included in the study. Sex distribution was equal. The median age at the time of surgery was 5 days (interquartile range 3–7 days), and the median weight was 3.1 kg (interquartile range 2.9–3. 4). Low cardiac output syndrome was detected in 30% (n = 18) of cases. The median perfusion index of patients who developed low cardiac output syndrome was significantly lower at the 12th, 18th, and 24th hours ($p < 0.05$) (0.99 versus 1.25, 0.86 versus 1.21, and 0.96 versus 1.33, respectively). Similarly, the median left ventricular output of patients who developed low cardiac output syndrome was significantly lower at 12th, 18th, and 24th hours ($p < 0.05$) (95 versus 110 ml/kg/min, 89 versus 109 ml/kg/min, and 92 versus 112 ml/kg/min, respectively). There was a significant correlation between perfusion index values and left ventricular output at all measurements ($r > 0.500$, $p < 0.05$). *Conclusion:* Perfusion index and left ventricular output measurements decreased in newborns who developed low cardiac output syndrome after arterial switch operation, especially at 12th and 18th hours. Serial perfusion index and left ventricular output measurements can be instructive in predicting low cardiac output syndrome development.

Transposition of great arteries is one of the most common cyanotic CHDs in newborns. Today, the standard treatment of transposition of great arteries is arterial switch operation in the first days of life.[1] Complications such as arrhythmia, acute kidney injury, and low cardiac output syndrome may be observed in patients after arterial switch operation. Low cardiac output syndrome is a clinical and biochemical condition in which the oxygen supply is insufficient to meet the patient's metabolic demands, either secondary to inadequate transport and distribution or increased oxygen consumption.[2] Mathematically, it is also expressed as a cardiac output of less than 2 L/min/m^2. Low cardiac output syndrome, a well-known and common postoperative complication, is one of the most important problems in paediatric cardiac ICUs; its incidence varies between 10 and 40% (2). Early diagnosis of low cardiac output syndrome can prevent mortality and morbidity.

In numerous studies, low cardiac output syndrome has been associated with serious organ failures, prolonged mechanical ventilator and ICU time, and increased mortality risk in congenital cardiac surgery. Timely diagnosis and precise management of this serious complication are crucial. Although various parameters such as pulse index contour cardiac output (system), echocardiographic measurements, serum biomarkers, heart rate, blood pressure, central venous pressure, near-infrared spectroscopy, urinary output, and serum lactate level are used for low cardiac output syndrome score calculation, there is no ideal method to make a definitive diagnosis of low cardiac output syndrome.[2,3]

Thanks to the recently developed technology, the role of non-invasive tests in diagnosing and managing diseases is gradually increasing. Perfusion index is one of those parameters used in newborns. Perfusion index reflects the ratio of pulsatile (arterial) to non-pulsatile (static) blood flow peripheral tissues. Thus, perfusion index monitors the perfusion of the specific part continuously in real-time and in a non-invasive manner.[4,5] Some studies claimed that low perfusion index values (perfusion index < 1.24) could be a useful indicator to determine the presence and severity of disease in newborns.[4]

Left ventricle output can be estimated by functional echocardiography using various systemic blood flow measurement methods. The estimates of cardiac blood flow can offer a clearer understanding of the pathophysiology which underlies the various clinical conditions, and they may guide the management of these conditions.[6]

In the literature, data on peripheral perfusion index values and left ventricular output changes in newborns with low cardiac output syndrome are limited. In this study, we aimed to evaluate the perfusion index and left ventricular output changes over time in newborns diagnosed with low cardiac output syndrome by using clinical scoring system after arterial switch operation.

Materials and Methods

Patient selection

This study was conducted prospectively in newborns with transposition of great arteries who underwent arterial switch operation between 1st August 2020 and 1st August 2022. Premature babies (n = 6) and patients with complex transposition of great arteries (subaortic stenosis, pulmonary stenosis) (n = 8) were excluded from the study. The study was planned according to the Declaration of Helsinki after obtaining approval from the local ethics committee.

A study form including preoperative data (demographic characteristics, cardiac pathology, and echocardiographic findings), *operative* data (cardiopulmonary bypass and surgery time) and *postoperative data* [time for extubation, length of intensive care and hospital stay, mortality, vasoactive inotropic score, blood gas analysis, cerebral and renal near-infrared spectroscopy changes, major complications (low cardiac output syndrome, arrhythmia, infection, acute kidney injury), perfusion index, left ventricular output measurements] was organised for each patient.

Intensive Care Unit

Patients were transferred from the operating room to the ICU as intubated and followed on mechanical ventilatory support. All patients were monitored for central venous pressure, electrocardiogram, invasive arterial blood pressure, end-tidal carbon dioxide, and cerebral near-infrared spectroscopy.

Typical inotropic support in the first postoperative hours was milrinone (0.5 μg/kg/min) and low-dose norepinephrine (0.05 μg/kg/min). Epinephrine was administered only if clinically necessary. Fentanyl and midazolam were used for analgesia and sedation; 100 mg/kg/day of cefazolin sodium was initiated for postsurgical antibiotic prophylaxis. Antibiotic treatment was started according to blood culture results and acute phase reactants. On the second postoperative day, total parenteral nutrition and minimal enteral feeding by nasogastric tube were started for all patients.[1]

Definitions

The primary outcome of this study was to determine the incidence of low cardiac output syndrome within 24 hours of arterial switch operation and its relation with perfusion index and left ventricular output. Modified Ulate's low cardiac output syndrome score was modified for low cardiac output syndrome diagnosis.[3] The low cardiac output syndrome score was calculated by assigning one point for each of the following:

tachycardia (>20% above postinduction heart rate in the operating room),
oliguria (<1 ml/kg/hour),
capillary filling time >3 seconds,
need for volume expansion (on top of maintenance IV fluids) (>30 ml/kg/day),
decreased near-infrared spectroscopy measurements (cerebral and renal near-infrared spectroscopy < 50 and 75% of arterial saturations, respectively),
elevated arterial lactate levels (>2 mmol/L or > 0.75 mmol/L/hour increase), and
need for vasoactive-inotropic infusions over 0.5 μg/kg/minute milrinone.

Each parameter was given one point, and the low cardiac output syndrome score was calculated hourly. If the total low cardiac output syndrome score was 3 > at any time in the first 24 hours, the patient was considered as low cardiac output syndrome.

Perfusion indexes were monitored simultaneously using two motion-resistant pulse oximeter monitor (IntelliVue MX800, Philips Healthcare, Best, The Netherlands). Disposable probes were placed in the right palm or wrist. Data were considered valid for analysis if perfusion index, heart rate, and SpO2 were simultaneously present at a time point and the plethysmography pulse wave was confirmed to be artefact free. Limits of perfusion index values identified to be invalid were ≤0.02 and ≥20 and simultaneous absence of heart rate and SpO2 values.[7] Perfusion index values at the transfer time to the ICU after arterial switch operation (0. hour), at the 6^{th}, 12^{th}, 18^{th}, and 24^{th} hours in intensive care and the lowest perfusion index values in the first 24 hours, were recorded from the monitor system (Fig 1).

Echocardiography was performed by one of the four paediatric cardiologists (EO, RTD, HHT, ICT) using Philips EPIQ CVx Ultrasound System (Philips Healthcare, Best, The Netherlands), with an S9-2 or S12–4 sector array transducer simultaneously with the pulse oximeter measurements. EO was blinded about perfusion index values. All images were analysed offline.

Left ventricular output was evaluated, as previously reported,[8] using the following formula:

Left ventricular output blood flow (ml/kg/min) = left ventricular output velocity time integral (cm) × π × (left ventricular output diameter/2)^2 (cm^2) × heart rate (bpm) / body weight (kg).

Left ventricular output diameter was measured in 2D mode, just below the aortic valve during systole in a cardiac long axis view. Left ventricular output velocity time integral was measured with pulse Doppler tracing in a five-chamber apical view with the box positioned inside the left ventricular output tract and with an angle <15° (Fig 2 a-b).

A four-channel trend monitor (Somanetics 5100B, Troy, MI, United States of America) was used for cerebral monitoring. The near-infrared spectroscopy sensor was placed on the right frontal region for children. Baseline near-infrared spectroscopy

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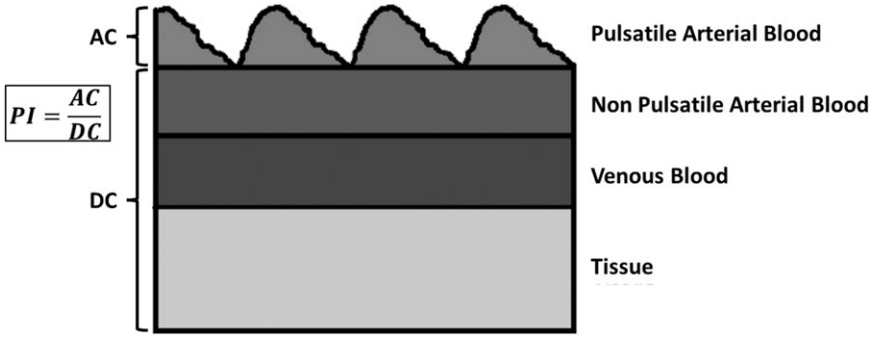

Figure 1. Formula for calculation of perfusion index.

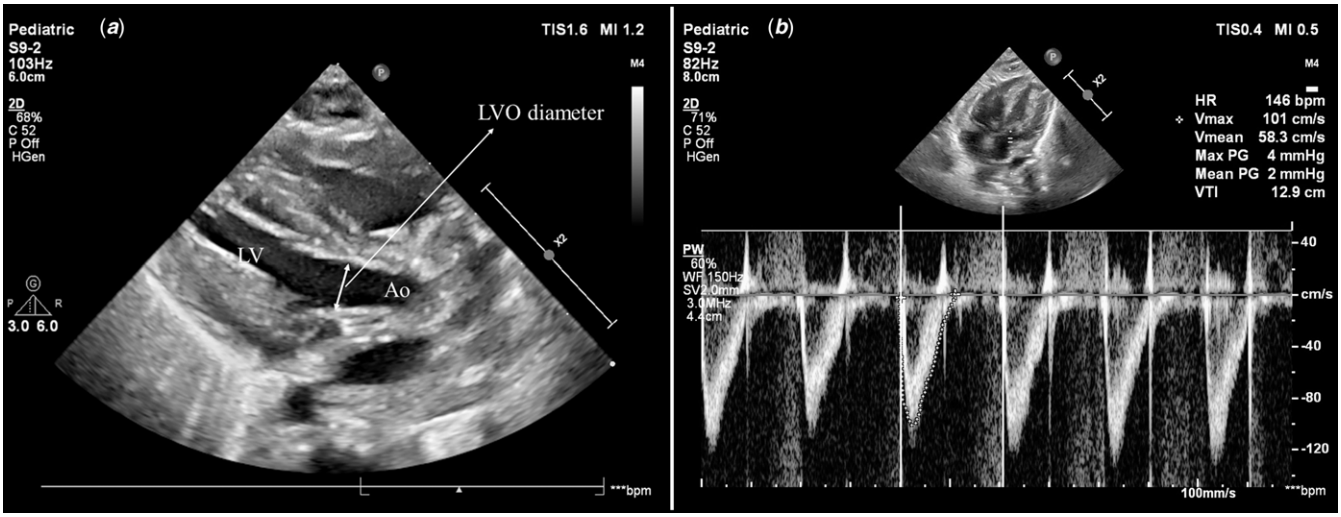

Figure 2. Left ventricular outflow measurement on echocardiography. (***a***) Left ventricular outflow diameter. (***b***) Left ventricular outflow velocity time integral (VTI).

values were recorded as the first measured after admission to the ICU, and cerebral oxygenation changes were evaluated.

Blood sample for lactate level was collected from the arterial cannula inserted during surgery. Blood lactate levels are routinely collected on cardiac ICU admission and frequently thereafter (i.e., 6, 12, 24, 48 hours, and more often if clinically indicated) during the postoperative period.[9]

Vasoactive inotropic score values were calculated for each patient by a standard formula for the first 24 postoperative hours, and the maximum score was recorded: vasoactive inotropic score: dopamine dose (μg/kg/min) + dobutamine dose (μg/kg/min) + 100 × epinephrine dose (μg/kg/min)] + 10 × milrinone dose (μg/kg/min) + 10,000 × vasopressin dose (units/kg/min) + 100 × norepinephrine dose (μg/kg/min).[10]

Statistical analysis

Statistical analysis was performed using SPSS for Windows (version 21.0, SPSS Inc., Chicago, IL, United States of America). Results for continuous variables with normal distribution were presented as mean (SD), and non-normally distributed data were reported as median (interquartile range). Categorical variables were presented as numbers and percentages. Demographic characteristics and perioperative variables were compared with Mann–Whitney U and Chi-square test. The effect of parameters in predicting low cardiac output syndrome was assessed by the receiver operating characteristic (ROC) curve. $P < 0.05$ was considered statistically significant.

Results

A total of 60 patients were enrolled in the study. Sex distribution was equal; 50% were male. The median age was 5 days (interquartile range 3–7 days), and the median weight was 3.1 kg (interquartile range 2.9–3.4). One patient had a genetic syndrome. Among all patients, 36% had coronary artery anomalies. The median cardiopulmonary bypass time was 110 minutes (interquartile range 100–140 min). Low cardiac output syndrome was diagnosed in 30% of patients (n = 18) according to low cardiac output syndrome scores within the first 24 hours. The general characteristics of the cases are shown in Table 1.

Acute kidney injury (44% versus 4%), patients with open chest management (38 % versus 11%), mortality in postoperative 30 days (33% versus 4%), and median cardiopulmonary bypass time (145 versus 120 minutes) were higher in patients with low cardiac output syndrome ($p < 0.05$). The clinical outcomes of the patients according to low cardiac output syndrome are shown in Table 2.

The change in median perfusion index of patients with and without low cardiac output syndrome at paediatric cardiac ICU admission, 6th, 12th, 18th, and 24th hours, is shown in Fig 3a. The median perfusion index of patients who developed low cardiac output syndrome was significantly lower at the 12th, 18th, and 24th hours ($p < 0.05$) (0.99 versus 1.25, 0.86 versus 1.21, and 0.96 versus 1.33, respectively).

The change in median left ventricular output (ml/kg/min) of patients with and without low cardiac output syndrome at paediatric cardiac ICU admission, 6th, 12th, 18th, and 24th hours, is shown

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Table 1. General characteristics of patients

Patient number (n)	60
Age at surgery (day)	5 (3–7)
Weight (kg)	3.1 (2.9–3.4)
Gender (male/female)	30/30
Genetic syndrome	1 (1.6%)
Prostaglandin E1 infusion	48 (80%)
Inotropic support	9 (15%)
Mechanical ventilation	5 (9%)
Balloon atrial septostomy	11 (18%)
Usual coronary artery pattern	38 (64)
Aortic valve position relative to pulmonary artery	
Anterior and right	43 (71.6)
Side-by-side, aorta to the right	9 (15)
Directly anterior	4 (6.6)
Posterior and right	2 (3.3)
Anterior and left	2 (3.3)
Ventricular septal defect	20 (33%)
Atrial septal defect (non-restrictive)	26 (44%)
Patent ductus arteriosus	52 (88)
Cardiopulmonary bypass time, minutes	130 (110–140)
Chest re-opening within 72 hours	12 (20%)
Low cardiac output syndrome within 24 hours	18 (30%)
Peak lactate levels within 24 hours (mmol/l)	4.4 (3.2–5.5)
Peak vasoactive inotrope score within 24 hours	10 (7–12)
Cerebral near infrared spectroscopy <50% of arterial saturations	15 (25)
Acute kidney injury	10 (16%)
Arrhythmia	4 (6.6%)
Extracorporeal membrane oxygenation	3 (5%)
Intra-hospital mortality within 30 days	8 (13.3%)

Results were given in median (IQR) or n (%).

Table 2. Relation of variables with low cardiac output syndrome

Variable	LCOS(+) n = 18	LCOS(–) n = 42	p
Age at surgery (day)	5 (3–7)	5 (3–7)	NS
Weight (kg)	3 (2.8–3.2)	3.2 (3–3.5)	NS
Sex (male/female)	8/10	22/20	NS
Inotropic support	3 (16)	6 (14)	NS
Mechanical ventilation	2 (11)	3 (7)	NS
Usual coronary artery pattern	12 (66)	20 (48)	NS
Cardiopulmonary bypass time, minutes	145 (120–160)	120 (105–140)	0.008
Chest re-opening within 72 hours	7 (38)	5 (11)	0.01
Acute kidney Injury	8 (44)	2 (4)	0.001
Arrhythmia	2 (11)	2 (4.8)	NS
Extracorporeal membrane oxygenation	3 (16)	–	0.002
Intra-hospital mortality within 30 days	6 (33)	2 (4)	0.001
Lactate			
Lactate at PICU arrival (mmol/l)	3.1 (2.5–3.8)	3 (2.4–3.6)	NS
Lactate at 6 hours (mmol/l)	3.7 (3.2–4.2)	2.9 (2.3–3.5)	0.040
Lactate at 12 hours (mmol/l)	4.2 (3.5–4.7)	2.8 (2.4–3.2)	0.001
Lactate at 18 hours (mmol/l)	3.8 (3.2–4.5)	2.7 (2.5–2.9)	0.025
Lactate at 24 hours (mmol/l)	3.5 (3–4.2)	2.5 (2.1–2.9)	NS
Vasoactive inotropic score (VIS)			
VIS at PICU arrival (mmol/l)	7 (5–10)	7 (5–10)	NS
VIS at 6 hours (mmol/l)	10 (7–12)	7 (5–10)	NS
VIS at 12 hours (mmol/l)	12 (10–15)	7 (5–10)	0.005
VIS at 18 hours (mmol/l)	15 (10–18)	7 (5–10)	0.001
VIS at 24 hours (mmol/l)	12 (10–15)	7 (5–10)	0.005
Cerebral near infrared spectroscopy <50% of arterial saturations			
At PICU arrival	6 (33)	10 (25)	NS
At 6 hours	5 (27)	5 (11)	0.040
At 12 hours	8 (44)	4 (9)	0.001
At 18 hours	7 (38)	4 (9)	0.001
At 24 hours	3 (16)	3 (7)	NS

in Fig 3b. The median left ventricular output of patients who developed low cardiac output syndrome was significantly lower at the 12th, 18th, and 24th hours ($p < 0.05$) (95 versus 110 ml/kg/min, 89 versus 109 ml/kg/min, and 92 versus 112 ml/kg/min, respectively).

For all measurements, the perfusion index and left ventricular output values correlated significantly ($r > 0.5$, $p < 0.05$) (Table 3).

ROC analysis of perfusion index parameters in predicting low cardiac output syndrome was summarised in Fig 4a. Perfusion index <0.9 was an independent risk factor [area under the curve (AUC) 0.82 confidence interval (0.76–0.88), $p < 0.001$, sensitivity 86%, specificity 80%, positive predictive value 84%] and strongly predicted low cardiac output syndrome [odds ratio 1.2 (confidence interval 0.9–5)].

ROC analysis of left ventricular output parameters in predicting low cardiac output syndrome was summarised in Fig 4b. Left ventricular output <100 ml/kg/min was an independent risk factor [AUC 0.78, confidence interval (0.70–0.84), $p < 0.001$, sensitivity 80%, specificity 70%, positive predictive value 78%] and strongly predicted low cardiac output syndrome [odds ratio 0.9 (confidence interval 0.5–3)].

Discussion

This study aimed to evaluate perfusion index and left ventricular output changes in low cardiac output syndrome after arterial switch operation in newborns with transposition of great arteries. In 30% of our patient population, low cardiac output syndrome

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Table 3. Correlation between left ventricle output and perfusion index

Values	R	p	Estimate
PI-0 hr / LVO-0 hr	0.845	0.001	y = 60.1 + 40.2*PI
PI-6 hr / LVO-6 hr	0.780	0.001	y = 61.5 + 37.8*PI
PI-12 hr / LVO-12 hr	0.680	0.001	y = 65.1 + 34.8*PI
PI-18 hr / LVO-18 hr	0.686	0.001	y = 59.7 + 39.6*PI
PI-24 hr / LVO-24 hr	0.608	0.001	y = 68.5 + 31.1* PI

PI=perfusion index; LVO=left ventricle output.

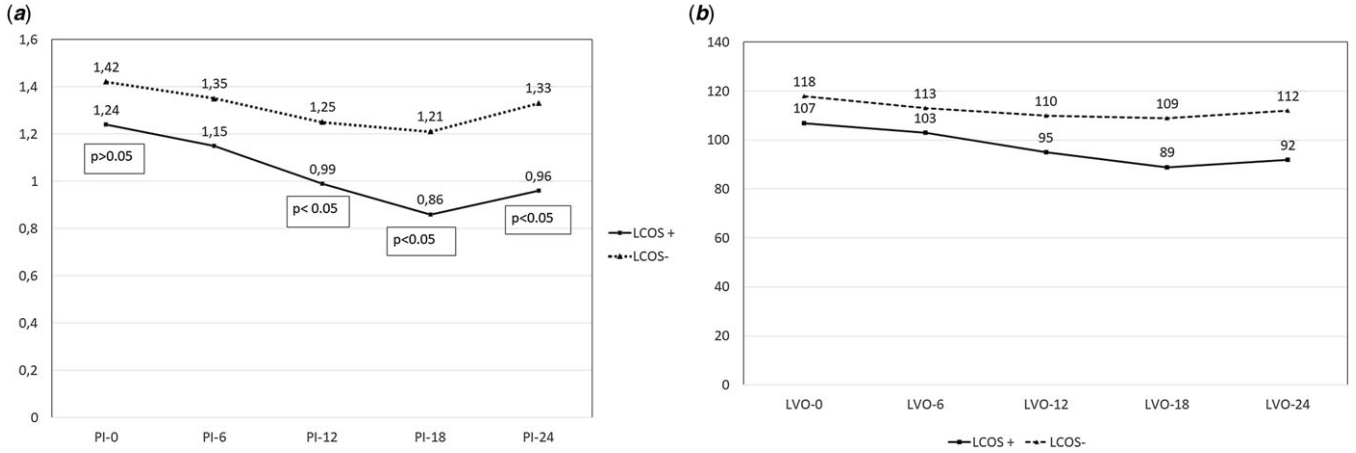

Figure 3. According to LCOS presence. (***a***) PI change against time. (***b***) LVO blood flow change against time.

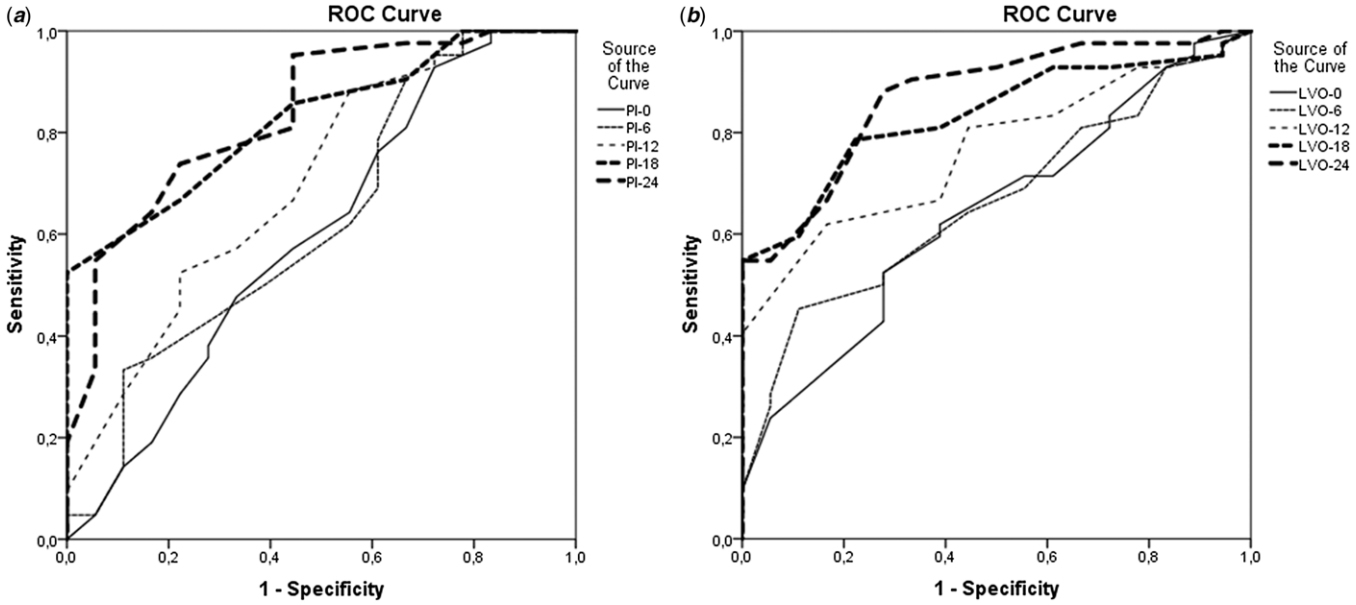

Figure 4. LCOS prediction based on ROC analysis. (***a***) PI. (***b***) LVO blood flow.

developed following arterial switch operation. There was a significant decrease in perfusion index and left ventricular output, especially after the 12th hour. Perfusion index and left ventricular output values correlated strongly with each other, and these two parameters could help to predict the development of low cardiac output syndrome. Our study is one of the limited studies evaluating perfusion index and left ventricular output in congenital cardiac surgery patients.

Wernovsky et al. found that cardiac output fell below 2 L/min/m^2 in 25% of patients in the postoperative period after

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arterial switch surgery, and it occurs mainly between 6 and 18 hours after admission to the ICU.[11] Navero et al. found low cardiac output syndrome incidence of 29%.[12] A newborn study expressed its incidence as 42%.[13] It has been stated that myocardial dysfunction associated with cardiopulmonary bypass, ischaemia-reperfusion damage, arrhythmia, and residual lesions may have an aetiological role in low cardiac output syndrome. Factors such as decreased preload, increased pulmonary and systemic vascular resistance, and increased metabolic demand have been accused of low cardiac output syndrome development.[2,12,13] We found low cardiac output syndrome incidence of 30%. The difference in low cardiac output syndrome incidence between studies might be due to different diagnostic criteria for low cardiac output syndrome definition.

Low cardiac output syndrome usually presents clinically with tachycardia, oliguria secondary to high systemic vascular resistance, inadequate tissue perfusion, high blood lactate levels, and metabolic acidosis.[2,3] Parr et al. demonstrated a positive correlation between low central venous oxygen saturation and low cardiac output syndrome development.[14] Also, increased lactate levels were associated with increased mortality and extracorporeal membrane oxygenation support.[15] In a large multicentre study, patients with low cardiac output syndrome had prolonged mechanical ventilatory support and ICU stay.[2] However, differences between centres pose difficulties in defining and managing low cardiac output syndrome. In this study, we used the low cardiac output syndrome score created by Ulate et al. using different clinical and laboratory data. Also, there were significant changes in blood lactate level, and near-infrared spectroscopy and vasoactive inotropic score scores after 6 hours in patients with low cardiac output syndrome.

Pulse oximeter waveforms contain additional information that have not been fully exploited despite its great clinical potential. Most studies have focused on establishing perfusion index parameters for the early detection of critical conditions in patients. Perfusion index is a non-invasive assessment that reflects the ratio of pulsatile to non-pulsatile blood flow in peripheral tissue; lower perfusion index values correspond to reduced peripheral perfusion in conditions such as specific CHD that reduce the stroke volume in arterial circulation.[16,17] The perfusion index decreases in cases of sympathetic predominance and/or low cardiac output states; therefore, it is a useful predictor of patient outcomes followed in critical care units. The perfusion index could be a surrogate for cardiac output in tests for fluid responsiveness, as an objective measure of pain, especially in non-cooperative patients, and as a predictor of successful weaning from mechanical ventilation. The perfusion index is simple to measure, easy to interpret, and has continuously displayed variables, making it a convenient parameter for detecting the adequacy of blood flow and sympathetic–parasympathetic balance. It is a common finding to have fluctuating perfusion index values as they reflect a brief momentary measurement of one's peripheral perfusion at the site of the pulse oximeter. Peripheral perfusion is dynamically affected by a variety of factors. For this reason, a single transient measurement has limited value because it may not accurately reflect one's true perfusion status. Instead, the median perfusion index calculated from monitoring perfusion index over a period of time after the pulse oximeter tracing stabilises may provide a more accurate depiction of true perfusion status.[16,17]

The use of perfusion index in clinical practice has some limitations. Perfusion index is characterised by skewness and a wide range of measurements among normal persons; therefore, it is better to evaluate its changes in comparison to the bassline readings from the same person. Care should always be paid to the possibility of poor signals especially in cold extremities, low body temperature, and high doses of vasopressors.[18]

In critically ill patients, the perfusion index was evaluated for predicting several outcomes. Takahashi et al. proposed that perfusion index <0.44 was consistent with <40 ml/min SVC flow.[19] Granelli et al. suggested using a perfusion index value of 0.7 as a method to evaluate whether newborns had left heart obstructive lesions, which was also a supplementary way to screen for CHD.[20] However, it is necessary to know the normal range for infants of specific gestational age at specific postnatal periods. Another study stated that perfusion index <1.24 may indicate a clinically poor condition.[5] Relying on its relation to the sympathetic tone, low perfusion index was able to predict hypotension during intermittent and continuous haemodialysis.[21] A pre-dialysis perfusion index ≤1.8 can predict hypotension during dialysis with a positive predictive value of 80% and a negative predictive value of 100%. In our study, perfusion index values were lower than the healthy population. Specifically, the low cardiac output syndrome group had lower 12th, 18th, and 24th hour perfusion index values. Perfusion index <0.9 was the cut-off point to predict low cardiac output syndrome.

Left ventricular output flow changes have been reported in obstructive left heart disease and low cardiac output syndrome.[19–20] Corsini et al. showed a positive correlation between left ventricular output and perfusion index in healthy newborns for the first time in their preliminary study in 2017.[7] Janaillac et al. proposed that cerebral regional tissue oxygen saturation and preductal perfusion index were strongly correlated with echocardiographically measured cardiac output during low cardiac output syndrome. We found that left ventricular output decreased significantly between 6 and 18 hours and started to rise after the 24th hour. We also had a similar observation to Corsini et al., simultaneous left ventricular output and perfusion index measurements correlated well.

This article provides two important and interesting assessment methods of cardiac output: directly evaluating the myocardium (specifically left ventricular output) in contrast to laboratory parameters such as lactate and MVO2, which are indirect indicators of myocardial health. Besides, perfusion index might provide additional information about peripheric tissue perfusion. Left ventricular output and perfusion index together might support conventional methods to monitor global (both central and peripheric) haemodynamics and evaluate low cardiac output syndrome in postoperative arterial switch patients. Employing both non-invasive haemodynamic monitorisation methods in a structural protocol might provide additional clinical benefit in the future.

Limitations

First, this is a single-centre study including a limited number of patients. Second, low cardiac output syndrome diagnosis is based on a scoring system. At last, a comparison of our results with the healthy control group could have made the results more meaningful.

Conclusion

In newborns with low cardiac output syndrome, diagnosed with low cardiac output syndrome scoring system, after arterial switch operation, perfusion index and left ventricular output measurements show a decrease, especially in the 12th and 18th hours.

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There is a strong correlation between simultaneous perfusion index and left ventricular output measurements; these measurements were also correlate with the forthcoming low cardiac output syndrome as well. In this regard, randomised prospective studies, including more patients, are needed.

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Conflict of interest. None.

Authors' contribution. DO, EO, RTD, HDO, HHT, and BT : Conception or design of the work, drafting the work, final approval of the version to be published, any part of the work is appropriately investigated and resolved.

ICT, AH, and MC: Acquisition and analysis, final approval of the version to be published, any part of the work is appropriately investigated and resolved.

Ethical standards. The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national guidelines on human experimentation and with the Helsinki Declaration of 1975 and Cam And Sakura Hospital Institutional Review Board (143-2022) approved the study.

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