Predictors of persistent pulmonary hypertension of the newborn in D-transposition of the great arteries: experiences at Songklanagarind Hospital, Songkhla

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Article

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Abstract

Transposition of the great arteries (TGA) with persistent pulmonary hypertension has high preoperative mortality. We aimed to define the predictive factors for persistent pulmonary hypertension of the newborn (PPHN) in TGA by comparing patients with TGA with and without PPHN in this retrospective cohort study, which recruited patients with TGA admitted to the hospital. Demographic and clinical information, and mortality, were compared between the two groups. Eighty-four neonates were diagnosed with TGA. The majority were boys (70.2%). The median birth weight was 3,095 g (IQR 2807-3425 g). The oxygen index (OI) was 18.2 ± 11.7. Seventy patients (83.3%) presented with TGA with an intact ventricular septum (IVS). TGA with PPHN occurred in 19% (16 of 84 patients) and 15 were the TGA with IVS type. The severity of PPHN was mild to moderate in 87.5% and severe in 12.5%. Multivariate analysis, comparing the two groups, showed that a higher OI (>21) and vasoactive-inotropic score (VIS) (>50) were associated with an increased odds ratio (OR) for TGA/PPHN (OR: 45.9, 95% CI: 8.9-236.38, P=0.003; OR: 61, 95% CI: 11.4-325.8, P=0.004), respectively.

The combination of TGA and PPHN has an increased risk of preoperative mortality despite successful balloon atrial septostomy for adequate mixing and optimal ventilator support. However, the development of PPHN in TGA requires aggressive PPHN treatment strategies, and can be predicted, based on our results, by a VIS >50 and OI >21.

Introduction

Transposition of the great arteries (TGA) in the newborn is the most common cause of cyanotic heart disease, accounting for 5–7% of cases [1]. Preoperative mortality in the newborn with TGA has been reported to be 4%. Moreover, persistent pulmonary hypertension occurred in 3–12% of newborns with TGA [2]. Persistent pulmonary hypertension of the newborn (PPHN) is the failure of the expected decline in pulmonary vascular pressure after birth, resulting in constant pulmonary vascular resistance. In addition, clinically, TGA/PPHN results in a higher degree of cyanosis because of decreased pulmonary blood flow, with unoxygenated blood supplying the upper part of the body and rich oxygenated blood preferentially going from the pulmonary artery to the descending aorta through a patent ductus arteriosus (PDA). The clinical presentation is reverse differential cyanosis with a higher degree of cyanosis in the upper than the lower part of the body. Roofthoft et al. found that 12.5% of TGA cases were associated with PPHN. They defined mild to moderate PPHN as an O_2 saturation difference of 5–15% between the upper and lower parts of the body and severe PPHN as an O_2 saturation difference > 15%. Additionally, preoperative mortality reached 28.6% in severe PPHN [3]. Sallaam et al. performed a retrospective analysis of 93 infants with D-TGA; 21.5% had associated PPHN, significantly associated with lower birth weight (2.8 [0.56] VS 3.33 [0.61]), gestational age (37.3 vs. 38.9 [1.7]), and mortality, which was 20% in the PPHN group vs. 1.4% in the group without PPHN [4]. Karimi et al. reported one case of TGA with an intact ventricular septum (IVS), with severe PPHN, where the patient survived after successful early surgical repair in the first three days of life without ECMO support; this showed the success of early intervention to correct hypoxia and preserve ventricular function [5]. Awareness of predictors of PPHN in

D-TGA would enable good management, which is challenging. Preoperative management is a crucial part of the treatment strategy, with adequate mixing after balloon atrial septostomy (BAS) and prostaglandin E1. However, this is insufficient to improve oxygenation in some infants with TGA and PPHN. El-Segaier et al. showed a positive effect with inhaled nitric oxide (iNO), with rapid improvement in oxygenation and the clinical condition [6]. Goissen et al. reported two cases in the high-risk group that had developed PPHN that failed to respond to usual management but benefited from bosentan [7]. We aimed to define the predictive factors of the occurrence and mortality of PPHN in TGA patients at our center.

Materials And Methods

Study design and participants

Our center is a referral university-based hospital in southern Thailand for all newborns presenting with cyanosis and diagnosed with congenital heart disease. Neonatologists support our team in treating them. This retrospective study was conducted with consecutive TGA newborn patients between January 2005 and December 2020 in Songklanagarind Hospital. TGA cases are diagnosed by a pediatric cardiologist on echocardiogram. If the patient is still hypoxemic after optimal treatment, we consider complications such as PPHN. The diagnosis of PPHN was made on physical examination, showing reverse differential cyanosis, and confirmed by echocardiogram. The exclusion criteria in this study were TGA with right or left ventricular obstruction.

Patient management

We usually prescribe prostaglandin E1 (PGE1) infusion at a dose of 0.01–0.2 mcg/kg/min to maintain oxygen saturation of more than 70% or PaO2 more than 30 mmHg in suspected TGA cases before transport to our center. Mechanical ventilation is started if oxygen saturation is less than 70%. Moreover, we recorded oxygen saturation in the upper and lower extremities first, for early detection of PPHN. If the O_2 saturation indicates reverse differential cyanosis between lower and upper extremities of more than 5–10%, PPHN may develop later. The diagnosis was confirmed by echocardiogram, which also illustrated possible risk factors for PPHN. For instance, restrictive atrial septal defect (ASD), D shape left ventricle (LV), and PDA flow direction may indicate a risk of PPHN in TGA cases. All TGA cases diagnosed with PPHN were given conventional treatment that included optimal ventilation, such as High-frequency oscillatory ventilation (HFOV), sedative medication (morphine or midazolam), volume support with normal saline solution(NSS), or blood transfusion if the hematocrit was less than 45%. Inotropic drugs, such as milrinone, were administered if blood pressure was normal but there was ventricular dysfunction. However, hypotension in critically ill infants with TGA and PPHN requires immediate treatment. Systemic blood pressure was supported with vasoactive drugs such as epinephrine, norepinephrine, dobutamine, and dopamine. This helped increase pulmonary blood flow due to increased systemic vascular resistance and raised the left to right shunt bypassing the PDA (systemic to pulmonary flow), resulting in improved cardiac output as well. The vasoactive-inotropic score (VIS) was calculated for some inotropic drugs. Emergency BAS was performed in cases with a restrictive atrial septum and clinical signs of hypoxia, in

the cardiac catheterization laboratory. After BAS, the patient is re-evaluated for increased oxygen saturation, no reverse differential in oxygenation, and arterial blood gas (ABG) and undergoes an echocardiogram demonstrating PDA flow and cardiac function. In addition, iNO was started, at 20 parts per million (ppm), if patients with TGA and PPHN developed severe respiratory failure (OI > 25). Therefore, iNO doses could be decreased if there is no reverse differential and the OI level is less than 15, before arterial switch operation (ASO). Other pulmonary vasodilator drugs, such as bosentan, sildenafil, and iloprost, have also been used [8, 9]. In cases of TGA with PPHN diagnosis was confirmed by echocardiogram to identify the PDA flow direction. We suggest that monitoring oxygen saturation in the upper and lower extremities are a crucial examination.

Data collection

The clinical data from the electronic medical records were reviewed. We collected the demographic and clinical information of newborn patients, including gender, gestational age, birth weight, type of TGA, age at presentation of cyanosis, initial oxygen saturation, oxygen index (OI), age, PEG1 drugs doses, arterial blood gas, echocardiogram result, severity of PPHN, inotropic drugs, VIS score, shock, medication for the treatment of PPHN, age at surgery, postoperative complications, and mortality. PPHN mortality was defined as patients dying before and after surgery.

Defining PPHN by severity [3]

TGA patients presenting with mild to moderate PPHN can be diagnosed by echocardiography showing bidirectional flow through the PDA that reverses differential saturation from pre- to post-ductal between 5% and 15%. Patients with severe PPHN presented with profound reverse differential cyanosis, with the echocardiogram revealing continuous right to left flow through a PDA and reverse differential saturation of 15% or higher.

Definition of the value of VIS [10, 11]

The value of VIS was calculated by a standard formula where the amount of inotrope used was measured by VIS, which was defined as dopamine dose (ug. kg_1. min_1) + dobutamine dose (ug. kg_1 min_1) + 100 x epinephrine dose (ug. kg_1 min_1) + 10 x milrinone dose (ug. kg_1 min_1) + 10,000 x vasopressin dose (U. kg_1. min_1) + 100 x norepinephrine dose (ug .kg_1. min_1).

Definition of the OI formula [12, 13]

OI was calculated using the standard formula (FiO2 x mean airway pressure x 100) / PaO2. Previously, the OI was widely used to initiate management in neonatal patients with PPHN, with OI > 25 being treated with iNO. However, recently, OI > 20–25 is the criterion for the use of iNO, and OI > 40 identifies a candidate for extracorporeal membrane oxygenation (ECMO).

Statistical analysis

The R program version R4.2.0 (R Core Team (2022). R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria) was used to analyze the data. Descriptive data are presented as numbers and percentages. Continuous data are described as means ± standard deviations (SD) and medians (interquartile range [IQR]) depending on the distribution of data. The data were initially analyzed by univariate analysis. Factors with a P-value < 0.05 in univariate analysis were further analyzed by multivariate logistic regression analysis. The Mann–Whitney U or Fisher's exact tests compared data between the two groups. Accuracy measures, including sensitivity, specificity, positive predictive value, negative predictive value, and area under the curve (AUC) were analyzed and used to determine the optimal cutoff values of peak VIS and OI for PPHN in TGA patients. Differences were considered statistically significant at a P-value < 0.05.

Results

Our study presents the result of a single-center experience of predictive factors and treatment outcomes between January 2005 and 2020 in 84 neonates diagnosed with TGA. Patient characteristics and clinical data are displayed in Table 1. The majority were boys (70.2%). The median birth weight was 3095 g (IQR 2807–3425 g). Median gestational age was 38 weeks (IQR 38–40 weeks). The median age (IQR) at cyanosis presentation was 3 hours (1, 24), and initial oxygen saturation was 72.9 ± 10.5. The OI was 18.2 ± 11.7. All patients with oxygen saturation less than 70% started PGE1, with the median age of initiation of PEG1 (IQR) being 13 hours (5, 27). Initial arterial blood gas analyses were performed, with the results showing a pH of 7.3 (IQR 7.3,7.4), PaO2 of 36.1 ± 8.2, PCO2 of 36 ± 9.7, and Base excess (BE) of -5 (-9, -2.8). Echocardiography confirmed the diagnosis of TGA and excluded left and right ventricular obstruction. Seventy patients (83.3%) presented with TGA/IVS. Nineteen percent had TGA/PPHN (16 of 84 patients) and 15 of these patients had TGA with IVS. The severity of PPHN was mild to moderate (reverse differential 5–15%) in 87.5% and the other 12.5% had severe PPHN (reverse differential > 15%). Also, PDA flow displayed bidirectional and right to left shunt flow in 23 (28.7%) and 8 (10%), respectively, through the PDA to the descending aorta (DAO), suggesting pulmonary hypertension. Fifty patients had restrictive ASD, but only 48 patients needed BAS. Of the other two, one had a large ventricular septal defect (VSD), and the other went on to an arterial switch operation within 24 hours. Twenty-one patients (27.6%) presented with shock. We prescribed inotropic drugs to support cardiac output. The median (IQR) VIS was 5 (0, 39). A total of 12 (14.3) infants required iNO, and 20 ppm was given to the majority. The mortality rate of all the patients was 26 (31%). The TGA with PPHN group had 5 (31%) patients who died; four patients underwent ASO; two patients died due to complications post-operation; one patient died from a postoperative PHT crisis; one patient developed a PHT crisis, received ECMO after surgery, came off ECMO, but then developed severe sepsis and died; and one patient died preoperatively from a severe PHT crisis.

Table 1
Characteristics and clinical condition data of TGA patients.

Demographic data	D-TGA (n = 84), n (%)
Gestational age (days)	38 ± 1.7
Birth weight (g)	3101 ± 624
Male	59 (70.2)
APGAR < 7 at 5 minutes	2 (2.4)
Age of presentation of cyanosis (hours) (median (IQR))	3 (1,24)
Initial oxygen saturation (mean ± SD)	72.9 ± 10.5
Oxygen index (mean ± SD)	18.2 ± 11.7
Age at initiation of PEG1 (hours) (median (IQR))	13 (5,27)
Initial arterial blood gas	
pH (median (IQR))	7.3 (7.3,7.4)
PaO_2 (mean ± SD)	36.1 ± 8.2
PCO_2 (mean ± SD)	36 ± 9.7
BE [median (IQR)]	-5 (-9, -2.8)
Type of TGA	
TGA/IVS or TGA/small VSD	70 (83.3)
TGA/VSD	14 (16.7)
TGA with PPHN	16(19)
Balloon atrial septostomy	48(57.1)
Shock	21(27.6)
Vasoactive- inotropic score (VIS)	5(0,39)
Need for iNO	12(14.3)
Death	26(31)
*PPHN; persistent pulmonary hypertension in newborn, **iNO; inhaled nitric oxide	

Table 2 compares baseline characteristics between patients with D-TGA, with and without PPHN. The OI mean ± SD was 31.1 ± 8.7 vs 14 ± 9. Arterial blood gas pH (median (IQR)) was 7.2 (7.2,7.3) vs 7.3 (7.3,7.4) and PCO_2 was 40.2 ± 10.4 vs 35 ± 9.3 mmHg and were significant ($P < 0.05$). There were bidirectional and right to left shunt flows of the PDA in 8 (50%) and 8 (50%) cases, respectively. The shock rate was higher in patients with TGA and PPHN in 13 (81.2%) vs 8 (13.3%) in those without PPHN. The VIS was [median

(IQR)] 283 (151, 430) vs 5 (0,10). The TGA with PPHN group were given iNO in 12 (75%) cases. The postoperative complication rate of PHT crisis was 6 (54.4%) vs 2 (5.4%). This factor was significant ($P < 0.001$). The receiver operation characteristic (ROC) curve for the OI was used to predict outcomes in TGA with PPHN. The AUC was 0.91, and the optimal cutoff value for peak OI was 21. The highest points of specificity and sensitivity were 0.93 and 0.81, respectively (Fig. 1). Also, the ROC curve for peak VIS in predicting the outcome of PPHN showed an AUC for the peak VIS of 0.919 (Fig. 2), and the optimal cutoff value of peak VIS was 50 (sensitivity 0.88, specificity 0.90). When gestational age, birth weight, gender, presenting age of cyanosis, OI, TGA type, arterial blood gas, echocardiogram, shock, and the VIS score were entered in the logistic regression for predictive factors of PPHN with TGA, multivariate analysis showed that higher OI (> 21) and VIS (> 50) were associated with an increase odds ratio for TGA/PPHN (OR: 45.9, 95% CI: 8.9-236.38, $P = 0.003$; OR: 61, 95% CI: 11.4-325.8, $P = 0.004$), respectively.

Table 2
Comparison of baseline characteristics between patients with D-TGA, with and without PPHN

Demographic data	D-TGA with PPHN (n = 16) N (%)	D-TGA without PPHN (n = 67) N (%)	P-value
Gestational age (weeks)	38.6 ± 0.8	38.4 ± 1.9	0.44
Birth weight (g)	3186 ± 479	3081 ± 655	0.60
Male	14(88.2)	45(66.2)	0.13
Age at presentation of cyanosis (median (IQR))	1.5(1,4)	3(1,24)	0.06
Oxygen index (mean ± SD)	31.1 (8.7)	14 (9)	< 0.001
TGA/IVS physiology	15 (93.8)	55 (80.9)	0.290
Age at initiation of PEG1	5(4,17.8)	19(5.8,36)	0.01
Arterial blood gas			
pH (median (IQR))	7.2(7.2,7.3)	7.3(7.3,7.4)	0.005
PaO_2 (mean ± SD)	34.3 ± 7.2	36.5 ± 8.3	0.335
PCO_2 (mean ± SD)	40.2 ± 10.4	35 ± 9.3	0.046
BE (median (IQR))	-5.5(-9.5, -2.9)	-4.9(-7, -2.8)	0.556
Echocardiogram			0.109
D shape LV	11(68.8)	29(42.6)	< 0.001
PDA flow	0	49(76.6)	0.074
Left to right flow	8(50)	0	
Right to left flow	8(50)	15(23.4)	
Bidirectional flow	14(87.5)	36 (52.9)	
Restrictive ASD			
BAS	14(87.5)	34(50)	0.054
Shock	13 (81.2)	8(13.3)	< 0.001

*IVS; intact ventricular septum

**PPHN; Persistent pulmonary hypertension

Demographic data	D-TGA with PPHN (n = 16) N (%)	D-TGA without PPHN (n = 67) N (%)	P-value
Vasoactive-inotropic score (median (IQR))	283(151,430)	5 (0,10)	< 0.001
Need for iNO	12(75)	0	< 0.001
Age at surgery (days) (median (IQR)]	14(10.5,18.2)	13(8,15.5)	0.36
Postoperative complications	1(10)	7(19.4)	0.664
Bleeding	3(30)	6(15.5)	0.364
Arrhythmia	6(54.4)	2(5.4)	< 0.001
PPHN**	4(40)	8(21.6)	0.251
Lung problems			
*IVS; intact ventricular septum			
**PPHN; Persistent pulmonary hypertension			

Table 3
Literature Review on predictors of PPHN and management outcomes in newborns with TGA/PPHN

First Author	Year	No. of patients	%PPHN	Risk PPHN	%BAS**	%iNO	%ECMO	%Death from PHT
El-Segaier[6]	2005	22	13.6	TGA/IVS	67/32	100	0	0
Goissen[7]	2007	2	100	TGA/IVS	100	100	0	0
Roofthooft[3]	2007	112	12.5	TGA/IVS	93/54	77	0	29
Karimi[5]	2014	1	100	TGA/IVS	100	100	0	0
Sallaam[4]	2016	93	21.5	LBW* and preterm infant	55/64	95	45	20
This article	2022	84	19	OI*** >21, VIS**** >50	87.5/50	75	18.8	12.5
*LBW; Low birth weight, **BAS; balloon atrial septostomy procedure was performed in TGA/PPHN and TGA without PPHN, ***OI; oxygen index, ****VIS; vasoactive-inotropic score								

Discussion

This retrospective cohort study researched TGA, which is a congenital heart disease, fatal due to progressive hypoxia and acidosis, particularly when combined with PPHN. The incidence of TGA with PPHN in our study was 16/84 (19%), similar to previous reports in the literature (12.5–21.5%) [3, 4, 6]. Patients with a reduced mixing shunt, TGA with IVS, and restrictive open foramen ovale have been suggested as inherently likely to lead to PPHN. Jaillard et al. demonstrated that neonates diagnosed with TGA with IVS, associated with PPHN, need ECMO during the preoperative period [14]. Additionally, Roofthooft et al. reported a correlation between D-TGA and PPHN in 112 infants. Moreover, TGA with IVS was more commonly associated with PPHN (15.7%) than TGA with VSD (3.4%) [3]. Table 3 describes previous studies supporting the fact that TGA/IVS was an essential factor in PPHN. Our study compares D-TGA, with and without PPHN, to identify predictive factors for the risk of later occurrence of PPHN.

In our data, in contrast, TGA/IVS was not associated with PPHN because the vast majority of TGA cases were TGA/IVS (83.3%). Indeed, TGA/PPHN patients were TGA/IVS in 15 of 16 patients (93.8%), while TGA/VSD was 1/16 (6.2%), P = 0.290. Sallaam et al. suggest that TGA with PPHN is significantly associated with low birth weight (2.8 [0.56] vs. 3.33 [0.61]) and gestational age (37.7 [2.1] vs. 38.9 [1.7]) compared to TGA without PPHN. However, our study showed no correlation with gestational age (38.6 ± 0.8 VS 38.4 ± 1.9, P = 0.44) or birth weight (3186 ± 479 VS 3081 ± 655, P = 0.60). Our study population was almost term infants of average weight. The mechanism of TGA with PPHN is diminished pulmonary blood flow due to high pulmonary vascular resistance after birth. We demonstrated reverse differential oxygen saturation on physical examination and bidirectional flow or right to left PDA flow on echocardiogram. Although PGE1 was given, patients also underwent BAS to increase mixing, and optimal ventilator support, as well as sedative drugs as the therapy of choice in TGA with PPHN cases. The recent acceptance of iNO for pulmonary vasodilatation means that iNO is a first-line treatment for PPHN. Therefore, our center started using iNO in 2013, and the indication at that time was an OI of more than 25; TGA with PPHN was considered for the use of iNO too. The reviewed literature in Table 3 revealed the preference for iNO therapy, similar to our center, at 75%. Our article demonstrated the ROC curve for the OI that predicted TGA cases that could develop PPHN later. The AUC was 0.91. The optimal cutoff value for peak OI was 21 and indicated the highest points of sensitivity and specificity at 0.93 and 0.81, respectively. Moreover, multivariable analysis showed that OI > 21 raised the odds ratio of PPHN with TGA (OR: 45.9, 95% CI: 8.9-236.38, P = 0.003).

Furthermore, patients with TGA with PPHN have profound hypoxemia and a progressive effect on myocardial function. Our study showed that 81.2% of this group presented with shock and required high doses of inotropic drugs to maintain cardiac output. Wernovsky et al., who initially reported inotropic scores, described quantifying inotropic drugs providing postoperative support in arterial switch operations in the neonate. Moreover, the inotropic score has been used as a marker of illness severity [10]. Consequently, a high inotropic score can imply the risk of PPHN in TGA. Gaies et al. proposed the inotropic score called the vasoactive-inotropic score (VIS). This study revealed that children < 6 months of age, admitted to PICU, who undergo congenital heart surgery with cardiopulmonary bypass, have a

strongly associated poor outcome if prescribed high maximum VIS, with an odds ratio (OR) of 8.1 [11]. In addition, Gaies et al. demonstrated that high VIS (≥ 20) was significantly associated with the odds of a poor outcome in neonates and infants who undergo cardiac surgery with bypass [15]. Dilli et al. reported that a high VIS score (> 15.5) correlated with higher mortality in newborns undergoing cardiac surgery, and the AUC was 0.83 (P < 0.001, CI: 95% 0.7–0.9) for VIS to identify mortality [16]. Nowadays, the VIS score is frequently used as an outcome measure of treatment, but we have insufficient data to support its use as a predictive factor for PPHN in TGA patients who presented with shock and needed treatment with many inotropic drugs. PPHN may present with impaired right ventricular function, especially in severe PPHN (oxygen diff > 15%), which can lead to left ventricular dysfunction because of interventricular function independence. Moreover, low cardiac output could develop and lead to poor tissue perfusion and decompensated shock. For this reason, we also use inotropic drugs to prevent shock and maintain blood pressure. Usually, we monitor BP, using neonatal blood pressures based on gestational age, and adjust accordingly, maintaining blood pressure at the 50th-75th centile for mean arterial pressure or systolic blood pressure. Our data indicated the optimal cutoff high VIS, and we sought to maximize VIS into a high and low variable, based on the sensitivity and specificity as a predictor of TGA/PPHN cutoff points. The optimal VIS value was 50, with a sensitivity and negative predictive value for PPHN in TGA of 88% and 97%, respectively (Fig. 2). Higher VIS was associated with an increased odds ratio (OR: 61, 95% CI: 11.4-325.8, P = 0.004) for TGA/PPHN. Our study reflected that patients who had PPHN before surgery presented with postoperative persistent pulmonary complications in 6 (54.4%) cases post ASO. Awareness of this is crucial.

Limitations

We acknowledge the limitations of our retrospective study. We did not have data on the predictors of PPHN in D-transposition of TGA because this condition was rare. Most importantly, our results provide predictors, in that a VIS score > 50 or OI > 21 may be associated with PPHN in infants with D-TGA.

Conclusion

The combination of TGA with PPHN causes serious illness and higher mortality preoperatively and the data concerning the prognosis of the disease are limited despite successful BAS for adequate mixing and optimal ventilator support. However, patients may develop PPHN in TGA and prediction allows aggressive PPHN treatment strategies, such as early, prompt administration of iNO and additional treatment modalities to treat resistant PPHN, including ECMO. The predictive factors identified in our study, VIS > 50 or OI > 21, serve as an alert for prompt treatment.

Declarations

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Declaration of competing interests

The authors declare no potential conflicts of interest with respect to the research, authorship, and publication of this article.

Ethics statement

Our Institutional Medical Ethics committee approved the study (REC:63-105-1-1) following The Declaration of Helsinki and The International Conference on Harmonization in Good Clinical Practice ICH-GCP. The institutional review board (IRB) does not require written informed consent in a medical chart review as the principal investigator is the attending physician, and all participants' identities are anonymous.

Credit authorship contribution statement

Kanjarut Wongwaitaweewong: conceptualization, methodology, formal analysis, investigation, data curation, writing –review & editing, supervision

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Figures

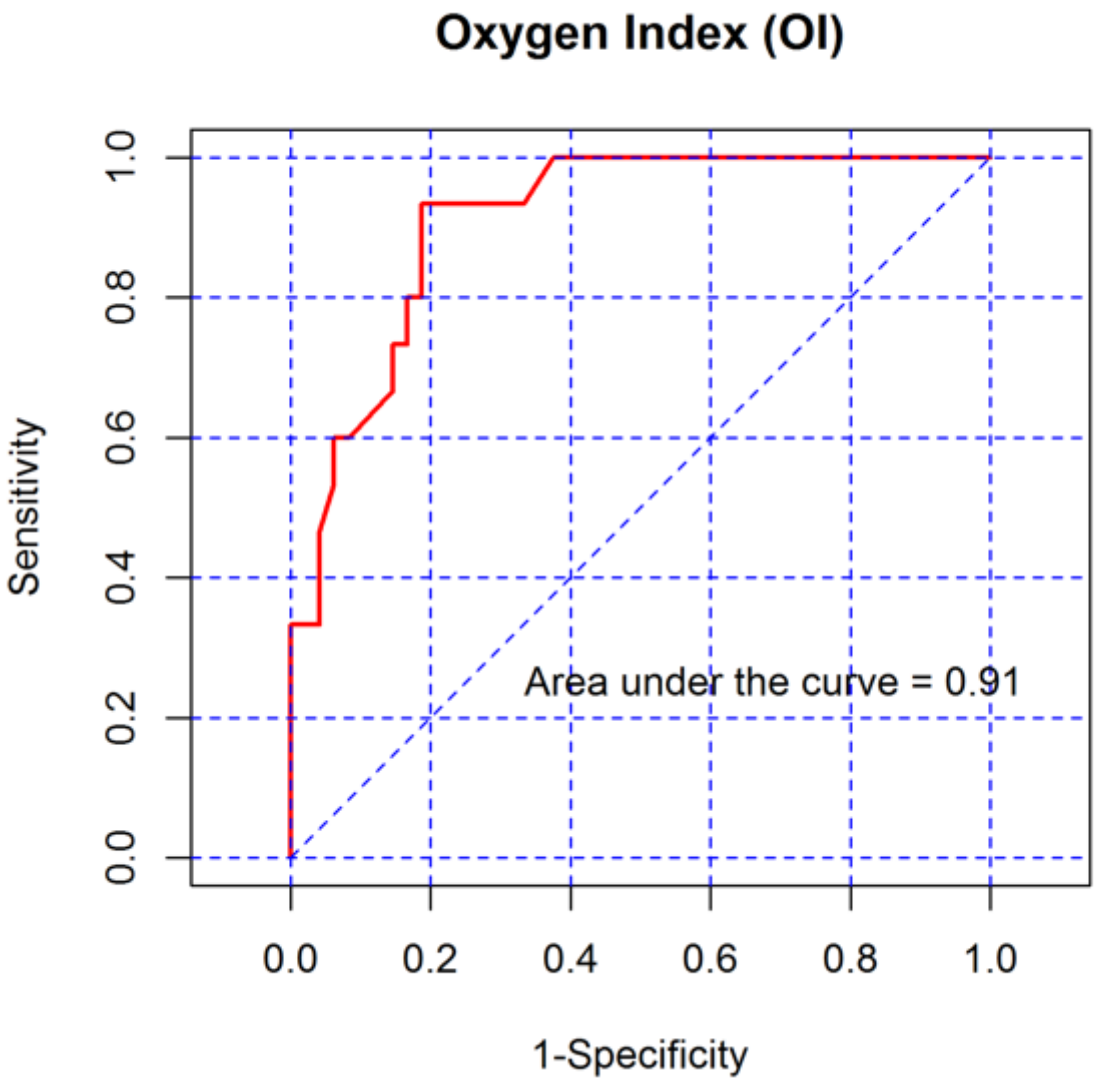

Figure 1

The receiver operating characteristic (ROC) curve for the oxygen index in predicting outcomes in TGA with PPHN. The area under the curve was 0.91. The optimal cutoff value for peak OI was 21. Max OI (red plotted) indicates the highest sensitivity and specificity of 0.93 and 0.81, respectively.

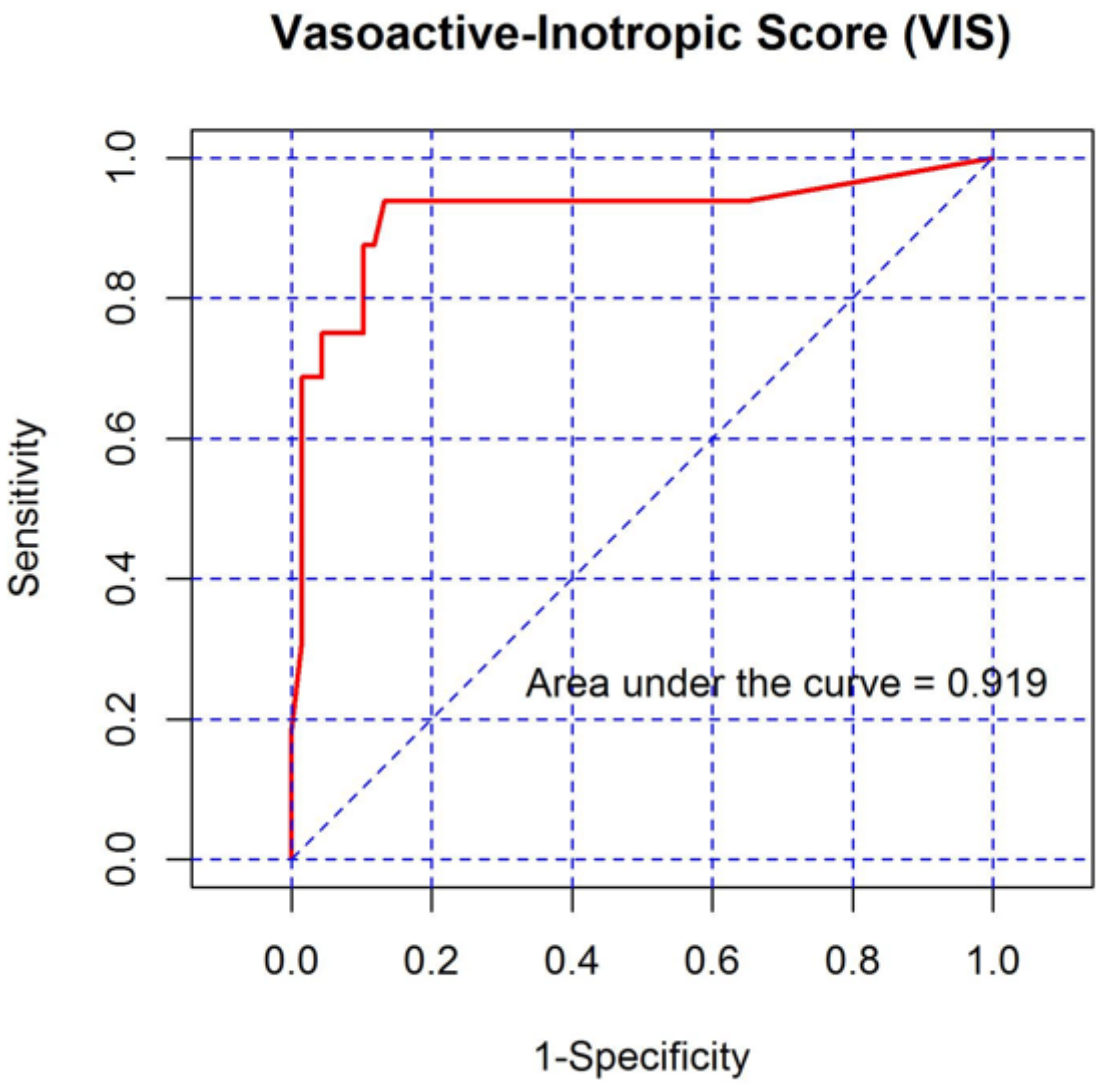

Figure 2

The receiver operating characteristic (ROC) curve for the peak vasoactive-inotropic score (VIS) predicts the outcome in TGA with PPHN. The area under the curve was 0.919, and the optimal cutoff value for peak VIS was 50. Specificity and sensitivity were 0.88 and 0.90, respectively.