Surgical repair of neonatal total anomalous pulmonary venous connection: A single institutional experience with 241 cases

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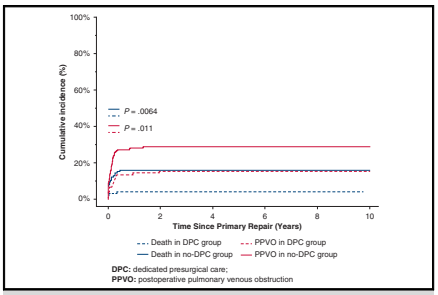
ABSTRACT

Objective: Challenges persist in surgery for neonatal total anomalous pulmonary venous connection (neoTAPVC), with the high mortality risk not mitigated over time.

Methods: A prospectively collected single-center database containing all neonates with TAPVC undergoing biventricular repair in 2012 to 2020 was retrospectively reviewed. The primary outcome was death or postoperative pulmonary venous obstruction (PPVO). Based on the preoperative admission location in our hospital, patients were classified into those being admitted to cardiac intensive care unit versus neonatal intensive care unit or general pediatric intensive care unit. Access to dedicated presurgical care (DPC) was defined as patients who were preoperatively admitted to the cardiac intensive care unit.

Results: Overall, 241 patients with a median age at surgery of 14 days (interquartile range [IQR], 9-21 days) were included. Anomalous return was supracardiac in 38.6%, cardiac in 26.1%, infracardiac in 28.6%, and mixed in 6.6%. Patients receiving DPC had better survival (96.3% vs 84.3%; $P = .0028$) and lower incidence of PPVO (15.2% vs 28.6%; $P = .011$) compared with those without DPC. Patients in the DPC group were less likely to undergo operation within 24 hours on presentation (27.1% vs 40.3%; $P = .041$), had improved lactate clearance (1.5 [IQR, 1.0-2.2] vs 2.8 [IQR, 1.8-4.1]; $P < .001$), and had lower incidence of postoperative pulmonary hypertension crisis (2.8% vs 18.7%; $P < .001$) compared with those in no-DPC group. After matching, no difference in PPVO could be observed in patients undergoing conventional versus sutureless repair (22.6% vs 12.9%; $P = .29$).

Conclusions: Access to DPC potentially improves outcomes in the neoTAPVC setting; freedom from PPVO were similar using conventional versus sutureless repair. (JTCVS Open 2023;16:739-54)



Competing risk analysis of events (death and restenosis) in DPC versus no-DPC group.

CENTRAL MESSAGE

Dedicated preoperative care is associated with better survival and lower restenosis rate following surgical repair of total anomalous pulmonary venous connection (TAPVC) in the neonatal patients.

PERSPECTIVE

Dedicated care aimed at preoperative stabilization may bridge the time to surgery in selected newborns with TAPVC, and is associated with better survival. Such specialist care facilitates completion of preoperative computed tomography scan, mitigating the risk of restenosis. Our study suggests that delivering tertiary care for this subpopulation may be considered in the quality improvement initiatives.

See Discussion on page 755.

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Funding sources: This study is supported by the Chinese National Natural Science Foundation of China (grant nos. 81801777, 82170307, and 81970267), Shanghai Rising-Star Program (grant no. 22QA1405800), Interdisciplinary Program of Shanghai Jiao Tong University (grant nos. YG2022QN094, ZH2018ZDA24), Program of Shanghai Shen-kang Hospital Development Centre (grant no. SHDC2022CRD014), and Program of Science and Technology Commission of Shanghai Municipality (grant nos. 19411964000, 20025800300).

Drs Shentu, Shi, and Zhang contributed equally to this article.

Read at the 103rd Annual Meeting of The American Association for Thoracic Surgery, Los Angeles, California, May 6-9, 2023.

Received for publication March 3, 2023; revisions received May 31, 2023; accepted for publication June 20, 2023; available ahead of print Sept 8, 2023.

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2666-2736

https://doi.org/10.1016/j.xjon.2023.07.021

Abbreviations and Acronyms

CI = confidence interval
CICU = cardiac intensive care unit
CPB = cardiopulmonary bypass
CTA = computed tomography angiography
DPC = dedicated presurgical care
HR = hazard ratio
IQR = interquartile range
LA = left atrium
neoTAPVC = neonatal total anomalous pulmonary venous connection
NICU = neonatal intensive care unit
PBF = pulmonary blood flow
PHC = pulmonary hypertensive crisis
PICU = pediatric intensive care unit
PPVO = postoperative pulmonary venous obstruction
PSM = propensity score matching
PV = pulmonary vein
PVO = pulmonary venous obstruction
PVR = pulmonary vascular resistance
TAPVC = total anomalous pulmonary venous connection

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Surgery for total anomalous pulmonary venous connection (TAPVC) in neonates remains challenging and is classified as a high-complexity and high-risk procedure based on several universally accepted risk score tools.[1-3] Despite the relentless efforts in pursuit of operative refinements[4,5] and better risk stratification,[6,7] outcome improvements have not been observed in this subpopulation,[8,9] driving the unmet need for identifying relative modifiable risk/protective factors for continued improvement.

There are some suggestions that a cardiac program including trained or experienced medical and nurse providers may be beneficial for neonates with critical congenital heart disease, especially in those undergoing high-complexity operations.[10,11] The rationale for the requirement of such specialist care is mainly based on the previous finding that patient-specific preoperative status has a strong association with mortality and morbidity, which can be influenced by preoperative management strategy.[12,13] Neonates with TAPVC represent the most severe end of the disease spectrum, as these patients are usually hemodynamically comprised largely because of preoperative pulmonary venous obstruction (PVO). American College of Physicians Task Force on requirements for pediatric cardiac critical care has underscored the importance of sufficient training and experience in managing neonates with TAPVC.[14] However, to what extent dedicated presurgical care (DPC) would influence the outcomes after neonatal TAPVC (neoTAPVC) surgery has not been well investigated. We performed this study to test the hypothesis that DPC may improve the outcomes following neoTAPVC surgery, in terms of survival and freedom from postoperative pulmonary venous obstruction (PPVO).

METHODS

Design and Population

This was a retrospective observational study enrolling consecutive neonatal patients who underwent TAPVC surgery between January 2012 and October 2020 in Shanghai Children's Medical Center (Figure 1), which is a free-standing children's hospital taking care of postnatal patients whose ages ranging from 0 to 18 years old. This study was approved by the institutional review board (SCMCIRB-K2022105-1; July 26, 2022), and all participants' guardians provided informed consent for publication of study data.

Dedicated Cardiac Intensive Care Unit (CICU) Care Model

Based on the preoperative admission location in our hospital, patients were classified as those being admitted to CICU versus neonatal intensive care unit (NICU) or general pediatric intensive care unit (PICU), irrespective of the referring hospitals (Figure 1). Compared with the NICU/PICU care model, CICU care has several unique features. First, the medical staff in CICU has sufficient training in congenital heart disease. The cardiac intensivists are dual-trained in both pediatric cardiology and critical care. The nurses assigned to care for newborns with TAPVC have at least 5 years' experience in cardiac patient care. Second, the CICU is a 45-bed closed ICU that is located in the same building with the in-patient department of cardio-thoracic surgery and cardiology. Such geographic grouping enables timely communication and discussion among the cardiac intensivists, surgical team members, and cardiologists. In addition, we have allied providers (nutritionists, pharmacists) with expertise in newborns in the CICU. Third, there is a standardized protocol for managing neonatal patients with TAPVC in the CICU (Figure 2). Fourth, our CICU provides a transportation service for newborns with TAPVC whose location is within 1500 miles from Shanghai. The cardiac transport team comprises 1 cardiac intensivist, 1 surgeon, and 1 CICU nurse. If the patients are diagnosed as TAPVC in the outside hospitals, and their parents decide to come to our hospital for further treatment and have a demand for transportation service, our transport team will be used. Of note, the transport team is routinely equipped with a surgeon in case that emergent life-saving extracorporeal membrane oxygenation may be required. This surgeon does not necessarily participate in the subsequent operation. Patients who were preoperatively cared in the CICU setting were defined as patients receiving DPC. Postoperatively, all patients were cared in the CICU.

Diagnosis of TAPVC

Echocardiogram is the first-line choice for the diagnosis of TAPVC. In addition, we recommend a preoperative computed tomography angiography (CTA) for better understanding of the detailed morphology, including (1) the confluence size, shape, orientation as well as its positional relationship with the left atrium (LA); (2) the entire anomalous draining pathway; and (3) individual pulmonary vein (PV) size. Diagnosis of preoperative PVO was made by a combined evaluation of oxygen saturation (<90%) and echocardiography (nonphasic PV flow velocity >1.8 m/s)[15]

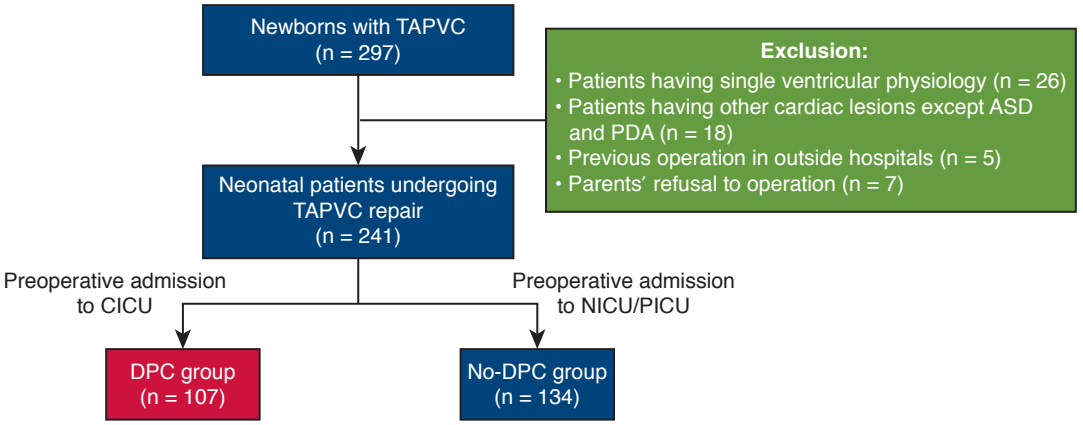


FIGURE 1. Flow diagram of the participants. A total of 241 neonates who underwent TAPVC repair between January 2012 and October 2020 were included. Fifty-six patients were excluded from the study, among whom 7 were excluded due to parents' refusal to consent to operation. Notably, 3 of the 7 developed circulation collapse, and we strongly recommended life-saving ECMO before surgery. However, their parents refused any treatment. *TAPVC*, Total anomalous pulmonary venous connection; *ASD*, atrial septal defect; *PDA*, patent ductus arteriosus; *CICU*, cardiac intensive care unit; *DPC*, dedicated presurgical care; *NICU*, neonatal intensive care unit; *PICU*, pediatric intensive care unit; *ECMO*, extracorporeal membrane oxygenation.

and was further subcategorized into extrinsic stenosis (obstruction occurred within the anomalous connecting vein or at its connection to the systemic circulation), intrinsic stenosis (pulmonary ostial stenosis, pulmonary venous stenosis/hypoplasia, or both), and restrictive atrial septal defect (<3 mm).[16] Similarly, PPVO was diagnosed on documentation as abnormal pulmonary venous flow patterns (described previously), and was further subcategorized into anastomotic restriction and intrinsic stenosis.

Timing of Surgery

Newborns with TAPVC will undergo surgery within 2 to 4 days after admission, which is a general principle in both DPC and no-DPC group. Specifically, preoperative CTA will be arranged within the first 1 to 2 days after admission; then, patients will undergo surgery within the next 1 to 2 days. However, if the patient has clinical instability or deterioration of the disease unresponsive to the targeted medical therapies (eg, worsening pulmonary edema, hypoxemic multiorgan dysfunction), emergent surgery will be performed without delay, despite whether CTA is completed. In some extreme cases (sudden circulation collapse), we recommend life-saving extracorporeal membrane oxygenation to achieve sufficient systemic perfusion and organ regeneration before surgery.

Definitions

Preoperative arterial lactate level was divided into 0 to 1.9 mmol/L, 2 to 3.9 mmol/L, and ≥4 mmol/L, given that (1) patients with critical illness can be considered to have normal lactate concentrations of <2 mmol/L and (2) an initial lactate ≥4 mmol/L increases the probability of mortality in patients admitted to ICU.[17,18] Diagnosis of pulmonary hypertension crisis (PHC) includes (1) the ratio of mean pulmonary arterial pressure to mean systemic arterial pressure of >1 accompanied by a rapid drop in saturation of peripheral oxygen in patients with a pulmonary arterial line and (2) a combination of echocardiographic calculation (Doppler measurement of maximal tricuspid regurgitation velocity[19]) and clinical judgment on basis of abruptly decreased systemic arterial pressure (decrease >20%) with high central venous pressure (>15 mm Hg), a rapid drop in saturation of peripheral oxygen, and decreased lung compliance in those without a pulmonary arterial line.[20,21] When there is suspicion of right ventricular failure, other parameters are taken into account (eg, tricuspid annular plane systolic excursion, ventricular septal position, biventricular size and function).[22] A pulmonary arterial line is routinely placed when the chest is left open and is removed when delayed sternal closure is performed.

Surgery

Procedural details have been described in our previous publications.[15,23-26] In brief, for direct atrio-pericardial (conventional repair), a side-to-side anastomosis between the confluence and LA was performed with a sufficient long incision in the confluence and a corresponding incision in the LA; for atrio-pericardial (sutureless repair), rechanneling of the anomalous pulmonary venous return was performed through anastomosing the LA to the posterior pericardium adjacent to the PV entrance to the pericardium. A routine cutback technique was performed in the cardiac type. The surgeons who were qualified for performing neoTAPVC surgery and the aforementioned techniques used in conventional and sutureless repair were not changed during the study period. Preoperative CTA plays an important role in adoption of the sutureless repair. If there is an unfavorable confluence–LA relationship (eg, great distance between LA and confluence) or intrinsically small individual PV that would make direct atrio-pericardial anastomosis difficult, a sutureless repair is chosen.

Outcome and Follow-up

The primary outcome was death or PPVO. Patients are required to have protocolized follow-up (postoperative 1, 3, 6, 9, and 12 months and then annually) after discharge in our outpatient cardio-thoracic department. Echocardiography was required at each follow-up, and CTA was further performed if there was a suspicion of PPVO. Reintervention for PPVO was recommended upon the evaluation of patients' clinical manifestations and imaging evidences. For patients with subclinical PPVO or whose parents refused receiving immediate reintervention, close follow-up visit (1- to 3-month intervals) was required.

Statistics

Skewed data were summarized in medians and interquartile range (IQR) and analyzed using the nonparametric Mann–Whitney *U* tests and Spearman rank correlation. Outcomes were reported as hazard ratio (HR) with 95% confidence intervals (CIs). Univariable risk factors for mortality were explored using Cox proportional hazards test. The Schoenfeld residuals test was used to verify the assumption of proportional hazard assumption, which was satisfied for the primary outcome from the index operation to the last follow-up. Multivariable analysis was not performed, as there were too few deaths (n = 25) to have multiple variables. We assessed the potential association between DPC and survival after respectively adjusting for TAPVC subtype, weight, operation era, or preoperative PVO. A

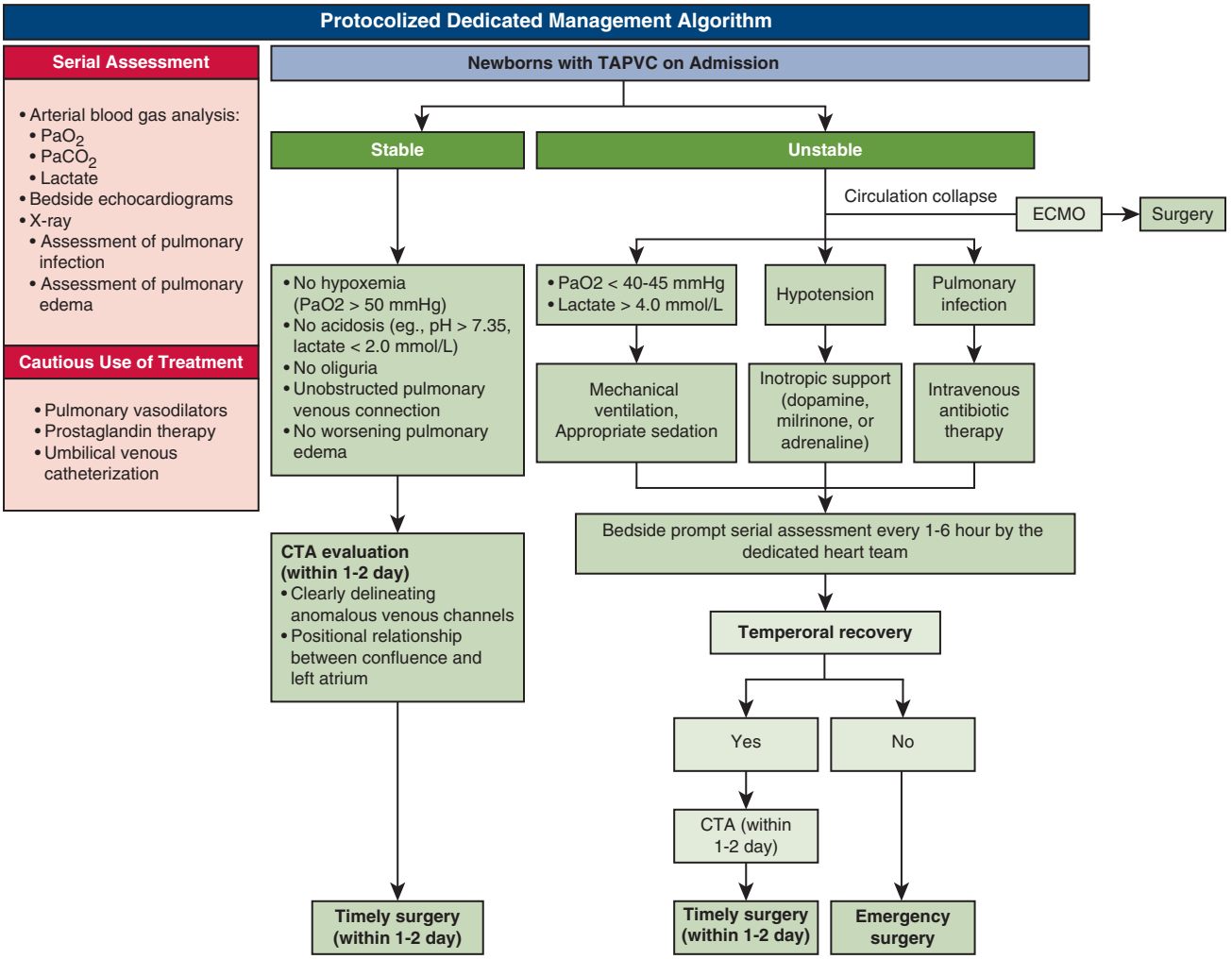


FIGURE 2. Protocolized management algorithm. *Pao_2*, Partial pressure of oxygen in arterial blood; *$Paco_2$*, partial pressure of carbon dioxide in arterial blood; *TAPVC*, total anomalous pulmonary venous connection; *ECMO*, extracorporeal membrane oxygenation; *CTA*, computed tomography angiography.

competing risk analysis was used to explore the association between potential predictors and PPVO. Variables of *P* value less than .05 in univariable analyses were entered into a multivariable model. Propensity score matching (PSM) analysis was performed to compare outcomes in patients undergoing atrio-pericardial versus atrio-pericardial repair. Variables used to generate the propensity score were the factors associated with mortality or PPVO identified in univariable or multivariable analyses, or regarded as clinically relevant (sex, age at operation, preoperative PVO, concomitant cardiac lesions, and preoperative management). Matching on the generated propensity scores was performed using 1:1 matching with a fixed caliper width set at 0.1 standard deviation of the logit of the propensity score. Survival was analyzed using Kaplan–Meier method, whereas freedom from PPVO was analyzed with death as a competing risk using the Fine and Gray method. All analyses were performed using R, version 4.1.3 (The R Foundation).

RESULTS

Baseline Characteristics

A total of 241 neonates (DPC: 107 vs no-DPC: 134) with a median age at surgery of 14 days (IQR, 9.0-21.0) were included. Specifically, no patient was directly sent to the operation room. Nine percent of the patients (22/241) were transferred by the cardiac transport team (Figure E1); for the remaining patients, their parents took them to visit our hospital by their own. Anomalous return was supracardiac in 38.6%, cardiac in 26.1%, infracardiac in 28.6%, and mixed type in 6.6%. Overall, 166 neonates (69%) had preoperative PVO, and 45% of the patients (75/166) with preoperative PVO underwent operation within 24 hours on presentation. Between-group comparisons showed statistical differences in several perioperative variables, including operation within 24 hours on presentation, preoperative CTA completion rate, preoperative arterial blood gas results (partial pressure of oxygen in arterial blood, partial pressure of carbon dioxide in arterial blood, and lactate level), and aortic crossclamping time (Table 1). Notably, there was shorter crossclamping time in patients who underwent presurgical CTA in DPC group than those who did not undergo presurgical CTA in no-DPC group (42.0 [IQR, 30.0-54.0] minutes vs 50.0 [IQR,

TABLE 1. Baseline characteristics and perioperative details

Characteristic	All patients N = 241	DPC vs no DPC DPC (N = 107)	No DPC (N = 134)	*P* value
Patient demographics				
Male sex, n (%)	159 (66.0%)	70 (65.4%)	89 (66.4%)	.89
Age, median, d (IQR)				
At surgery	14.0 (9.0-21.0)	14.0 (10.0-21.0)	14.0 (7.0-22.0)	.25
At admission	12.0 (6.5-19.0)	12.0 (8.0-19.0)	12.0 (5.0-19.0)	.34
Duration of preoperative care, d (IQR)	3.0 (1.0-3.0)	3.0 (1.0-3.0)	2.0 (1.0-3.0)	.18
Weight, kg, median (IQR)	3.4 (3.0-3.6)	3.5 (3.0-3.7)	3.3 (3.0-3.6)	.36
Associated cardiac lesion, n (%)				
ASD	241 (100%)	107 (100%)	134 (100%)	NA
PDA	161 (66.8%)	67 (62.6%)	94 (70.1%)	.27
Subtypes of TAPVC, n (%)				.65
Supracardiac	93 (38.6%)	37 (34.6%)	56 (41.8%)	
Cardiac	63 (26.1%)	31 (28.0%)	32 (23.9%)	
Infracardiac	69 (28.6%)	31 (28.0%)	38 (28.4%)	
Mixed	16 (6.6%)	8 (7.5%)	8 (6.0%)	
Operation era, n (%)				<.001
2012-2014	67 (27.8%)	13 (12.1%)	54 (40.3%)	
2015-2017	89 (36.9%)	45 (42.1%)	44 (32.8%)	
2018-2020	85 (35.3%)	49 (45.8%)	36 (26.9%)	
Preoperative PVO, n (%)				
Intrinsic stenosis	8 (3.3%)	3 (2.8%)	5 (3.7%)	>.99
External stenosis	93 (38.6%)	42 (39.3%)	51 (38.1%)	.89
Restrictive ASD	72 (29.9%)	27 (25.2%)	45 (33.6%)	.20
Spo_2, median (IQR), %	85 (80-90)	85 (80-90)	85 (79-90)	.49
Last arterial blood gas analysis before operation				
Lactate, median (IQR), mmol/l	2.0 (1.2-3.0)	1.5 (1.0-2.2)	2.7 (1.8-4.1)	<.001
Pao_2, median (IQR)	44.6 (37.0-54.6)	47.0 (41.1-56.8)	42.9 (34.8-52.2)	.004
$Paco_2$, median (IQR)	41.4 (36.7-48.5)	40.2 (34.9-45.6)	43.7 (38.5-51.5)	<.001
Preoperative management, n (%)				
Serial assessment of arterial blood gas*	107 (44.4%)	107 (100%)	0 (0%)	<.001
Inotropes	92 (38.2%)	40 (37.4%)	52 (38.8%)	.89
Mechanical intubation	59 (24.5%)	22 (20.6%)	37 (27.6%)	.23
UVC	47 (19.5%)	0 (0.0%)	47 (35.1%)	<.001
PGE1 therapy	33 (13.7%)	1 (0.9%)	32 (23.9%)	<.001
Pulmonary vasodilators	27 (11.2%)	0 (0.0%)	27 (20.1%)	<.001
Operation within 24 h on presentation, n (%)	83 (34.4%)	29 (27.1%)	54 (40.3%)	.041
CTA completion, n (%)	144 (59.8%)	96 (89.7%)	48 (35.8%)	<.001
Surgical details				
Sutureless repair	44 (18.3%)	19 (17.8%)	25 (18.7%)	.87
CPB time, median (IQR), min	85.0 (64.0-115.0)	83.0 (61.0-106.0)	88.0 (64.5-117.0)	.081
AXC time, median (IQR), min	43.0 (31.0-55.0)	39.0 (27.5-54.0)	46.0 (33.0-59.0)	.022
Duration of mechanical ventilation, h, median (IQR)	92.0 (51.0-134.6)	82.0 (48.0-123.5)	96.0 (64.8-138.0)	.21
Postoperative information				
Delayed sternal closure, n (%)	112 (46.5%)	44 (41.1%)	68 (50.7%)	.15
Pulmonary artery pressure line, n (%)	112 (46.5%)	44 (41.1%)	68 (50.7%)	.15
Duration of pulmonary artery pressure line, d, median (IQR)	3.0 (2.0-3.0)	3.0 (2.0-3.0)	3.0 (2.0-3.0)	.64
Low cardiac output, n (%)	81 (33.6%)	30 (28.0%)	51 (38.1%)	.13
PHC, n (%)	28 (11.6%)	3 (2.8%)	25 (18.7%)	<.001
Postoperative CICU stay, d, median (IQR)	7.0 (5.7-9.8)	6.8 (5.8-8.8)	7.4 (5.6-10.8)	.28
Hospital stay, d, mean (SD)	19.0 (8.6)	19.5 (10.1)	18.5 (7.1)	.36

(Continued)

TABLE 1. Continued

Characteristic	All patients N = 241	DPC vs no DPC DPC (N = 107)	No DPC (N = 134)	*P* value
Follow-up time, y, median (IQR)	4.1 (1.6-6.9)	4.3 (1.9-7.0)	3.8 (1.4-6.5)	.29
PPVO	54 (22.4%)	16 (15.0%)	38 (28.4%)	.019
Death	25 (10.4%)	4 (3.7%)	21 (15.7%)	.003

DPC, Dedicated presurgical care; *IQR*, interquartile range; *ASD*, atrial septal defect; *NA*, not available; *PDA*, patent ductus arteriosus; *TAPVC*, total anomalous pulmonary venous connection; *PVO*, pulmonary venous obstruction; Spo_2, saturation of peripheral oxygen; Pao_2, partial pressure of oxygen in arterial blood; $Paco_2$, partial pressure of carbon dioxide in arterial blood; *UVC*, umbilical venous catheterization; *PGE1*, prostaglandin E1; *CTA*, computed tomography angiography; *CPB*, cardiopulmonary bypass; *AXC*, aortic crossclamping; *PHC*, pulmonary hypertensive crisis; *SD*, standard deviation; *CICU*, cardiac intensive care unit; *PPVO*, postoperative pulmonary venous obstruction. *Serial assessment of arterial blood gas every 1 to 6 hours.

34.0-67.3] minutes; $P = .007$); however, no difference in crossclamping time could be observed in those who undergo presurgical CTA in both DPC and no-DPC group (42.0 [IQR, 30.0-54.0] minutes vs 38.5 [IQR, 31.5-52.0] minutes; $P = .87$).

Risk Factors of Death and PPVO

Overall, death occurred in 25 patients (10%), at a median of 0.26 months (IQR, 0.12-0.89); PPVO occurred in 54 patients (22.4%), at a median of 1.26 (IQR, 0.23-2.54) months. Of the 54 patients developed PPVO, 8 underwent reinterventions (7 had relief of PPVO whereas the other 1 died of recurrent PVO), 38 with subclinical PPVO had ongoing close surveillance, and the remaining 8 did not undergo reoperation due to the parents' refusal despite our strong recommendations, all of whom died during the follow up. Details of postoperative PVO were shown in Table E2.

Lower weight, greater last arterial lactate level before surgery, emergency surgery, noncardiac connection type, long CPB, and crossclamping time were associated with death, whereas access to DPC was a protective factor (Table 2). Further, DPC consistently remained a protective factor for survival after respectively adjusting for weight (hazard ratio [HR], 0.24; 95% confidence interval [CI], 0.10-0.60; $P = .002$), TAPVC subtype (HR, 0.24; 95% CI, 0.08-0.68; $P = .008$), preoperative PVO (HR, 0.23; 95% CI, 0.08-0.68; $P = .008$) and operation era (HR, 0.23; 95% CI, 0.08-0.69; $P = .009$). There was a better survival in the DPC group compared with no-DPC group ($P = .0028$, Figure 3, *A*). Competing risk analysis showed that both noncardiac (supracardiac, infracardiac, and mixed) subtype (subdistribution HR, 2.84; 95% CI, 1.11-7.24; $P = .029$) and access to DPC (subdistribution HR, 0.52; 95% CI, 0.28-0.98; $P = .041$) were independent predictors for PPVO. The cumulative incidence of PPVO was significant lower in DPC group than no-DPC group ($P = .011$, Figure 3, *B*).

Lactate Clearance in DPC Versus No-DPC Group

All patients had at least one lactate test. Patients in the DPC group all had at least >1 lactate test (median of 9 tests [IQR, 6-18]) due to the protocolized management algorithm whereas only approximately two-thirds of the patients (83/134) in no-DPC group had more than 1 lactate test (median of 3 tests [IQR, 2-5]). Arterial lactate level improved in 57% of the patients (61/107) in DPC group. In contrast, arterial lactate level deteriorated or remained unchanged in 78% of the patients (65/83) in the no-DPC group (Figure E2). Furthermore, serial bedside arterial blood gas analysis every 1 to 6 hours is strongly correlated with improved lactate clearance ($r = 0.36$, $P < .001$).

Sutureless Versus Conventional Repair

PSM resulted in 2 well-balanced groups consisting of 31 patients undergoing sutureless repair and 31 patients undergoing conventional repair (Table E1). There was no significant difference in the cumulative incidence of PPVO in the 2 matched subcohort (12.9% vs 22.6%, Grey test $P = .29$; Figure 4).

DISCUSSION

This study, including 241 patients completing a median of 4.1 years' follow-up after the initial surgery, represents the largest cohort of neonates with TAPVC. The main findings are as follows: (1) DPC helps to optimize preoperative clinical status in neonatal patients with TAPVC and is associated with better survival; (2) DPC facilitates preoperative CTA completion, which is helpful in better understanding of the detailed morphology, and is associated with decreased PPVO; and (3) after PSM, similar incidences of PPVO were observed in patients undergoing conventional versus sutureless repair.

This study first provides an insight into the impact of presurgical care models on outcomes in the neoTAPVC setting. It is noteworthy that the access to DPC conferred an approximately 4-fold decrease in death, particularly resulting in the postoperative 1-year mortality of 3.7% that was remarkably lower than previously published data.[1,6-9] Therapeutic plan associated with serial lactate monitoring in DPC group is related to the improved lactate clearance, which is a possible explanation for the better survival, as emerging evidence has shown that lactic acidosis can decrease

TABLE 2. Univariable and competing risk analysis of predictors for primary outcomes

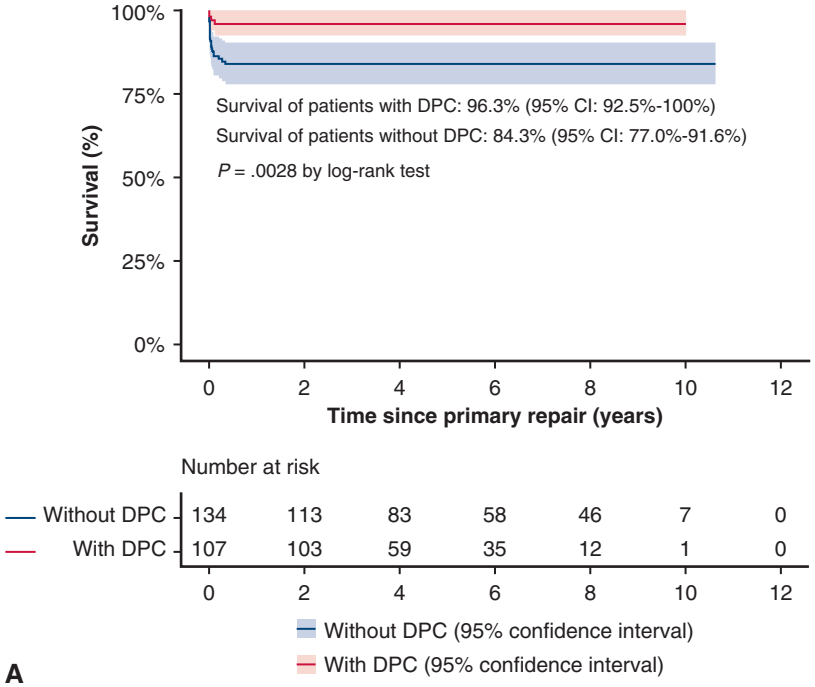
Variable	Death			PPVO					
	Univariable Cox regression			Univariable subdistribution hazard regression			Multivariable subdistribution hazard regression		
	HR	95% CI	*P* value	sHR	95% CI	*P* value	sHR	95% CI	*P* value
Male sex	0.53	0.24-1.16	.11	0.86	0.50-1.49	.59			
Age at surgery	0.95	0.90-1.01	.083	0.98	0.94-1.02	.24			
Weight	0.25	0.10-0.59	.002	0.97	0.53-1.78	.92			
Operation era									
2012-2014	Ref			Ref					
2015-2017	0.73	0.29-1.83	.50	0.93	0.53-1.62	.80			
2018-2020	0.59	0.22-1.60	.30	0.69	0.38-1.25	.22			
Noncardiac type	4.33	1.02-18.37	.047	3.97	1.60-9.86	.003	2.95	1.15-7.53	.024
Preoperative PVO	1.84	0.69-4.91	.22	1.17	0.67-2.06	.58			
Last lactate before surgery	1.24	1.14-1.34	<.001	1.05	0.96-1.15	.27			
Spo_2	0.98	0.95-1.01	.13	0.98	0.96-1.00	.083			
Pao_2	1.00	0.97-1.03	.88	0.99	0.97-1.01	.16			
$Paco_2$	1.01	0.99-1.04	.36	1.01	0.99-1.04	.31			
DPC	0.23	0.08-0.66	.006	0.47	0.27-0.84	.011	0.53	0.30-0.93	.027
CPB time	1.01	1.01-1.02	<.001	1.01	1.01-1.02	<.001	1.00	0.98-1.01	.73
AXC time	1.02	1.01-1.03	.001	1.02	1.01-1.03	<.001	1.02	1.00-1.04	.070
Operation within 24 h on presentation	2.55	1.16-5.62	.020	0.93	0.53-1.64	.81			
Sutureless repair	0.84	0.29-2.43	.74	0.53	0.23-1.21	.13			

TAPVC was divided into cardiac versus noncardiac (including supra-, infra-, and mixed TAPVC) connection type. *PPVO*, Postoperative pulmonary venous obstruction; *HR*, hazard ratio; *CI*, confidence interval; *sHR*, subdistribution hazard ratio; *PVO*, pulmonary venous obstruction; Spo_2, saturation of peripheral oxygen; Pao_2, partial pressure of oxygen in arterial blood; $Paco_2$, partial pressure of carbon dioxide in arterial blood; *DPC*, dedicated presurgical care; *CPB*, cardiopulmonary bypass; *AXC*, aortic crossclamping; *TAPVC*, total anomalous pulmonary venous connection.

cardiac contractility, tissue perfusion, and sensitize the myocardium to cardiac arrhythmias, and it is associated with increased mortality in pediatric patients with cardiac disease.[17,27] Several specific factors of the CICU care model are responsible for the improvement in the preoperative health: first, personnel has sufficient training in congenital heart disease; second, the team-based multidisciplinary approach enables optimal decision-making in terms of preoperative planning and surgical timing; third, development and implementation of a standardized protocol can allow for rapid patient risk-stratification and timely targeted therapies (therapeutic plan associated with the monitor), all of which cater to the preoperative needs of these vulnerable newborns better than the NICU/PICU (Figure 5).

Of note, isolated TAPVC has one of the lowest prenatal detection rates (~2%-10%) for congenital heart disease.[28] Thus, these newborns probably less benefit from a planned delivery and a dedicated postnatal care. Findings from this study modestly suggest that transporting newborns with TAPVC by a dedicated heart team may be an alternative option when they have no access to a specialized congenital heart center.

In general clinical opinion, it appears to be better to perform surgery as soon as possible in newborns with TAPVC, especially in those with obstructed TAPVC. However, there is no consensus on the optimal timing regarding the neoTAPVC surgery. Findings from this study suggest that care by a dedicated cardiac team can allow for tiered patient triage and helps to optimize presurgical clinical status and bridge the time to surgery in the selected patients. Patients with obstructed TAPVC can have different underlying pathophysiology. Patients with severe obstruction usually have increased pulmonary vascular resistance (PVR) and decreased pulmonary blood flow (PBF). These patients present with severe hypoxemia and low cardiac output that are unresponsive to the targeted medical therapies; thus, emergent operation should be required. Although those with moderate/mild obstruction can have an increase in both PVR and PBF, the increased PVR and PBF balance one another out so that hypercirculation through the lung probably results in congestive heart failure. These patients usually present with moderate/mild hypoxemia; tertiary care potentially produces the clinical benefit and helps to bridge the time to surgery. This is partially in line with the large experience on surgical care for neonatal patients



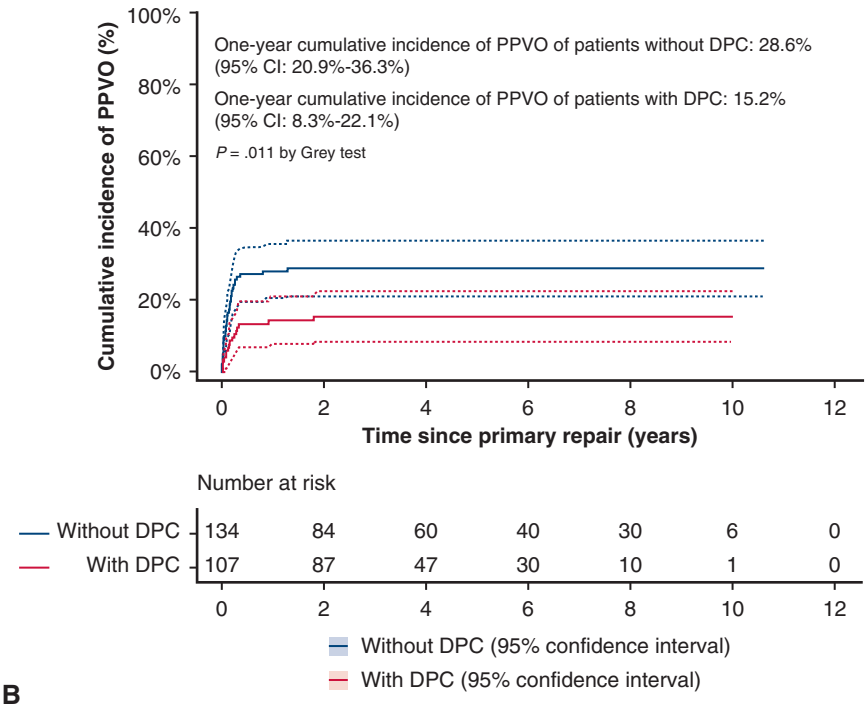


FIGURE 3. Between-group comparison of primary outcomes in the DPC and no-DPC group. A, Kaplan–Meier curves showed that access to DPC was associated with a significantly better survival; B, competing risk analysis showed that access to DPC was associated with a significantly lower cumulative incidence of PPVO. *DPC*, Dedicated presurgical care; *CI*, confidence interval; *PPVO*, postoperative pulmonary venous obstruction.

with TAPVC (sample size: 175) from the Melbourne group,[9] in which less than one half of the patients (43%, 49/115) with preoperative PVO underwent emergency operation. Plausibly, it can be speculated that a portion of the "obstructed" patients can be triaged to elective surgery if appropriately managed. Nonetheless, we do not recommend pursuing ongoing preoperative management with a delay of surgery because the PVs are structurally abnormal in

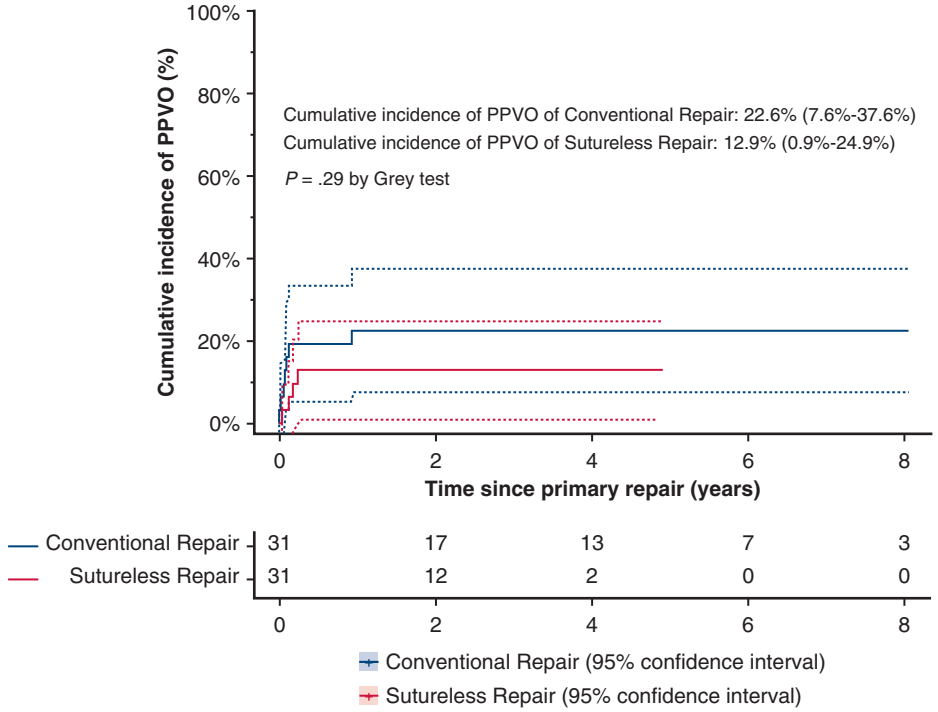


FIGURE 4. Comparison of cumulative incidence of PPVO in sutureless- versus conventional-repair group. No difference was observed in occurrence of PPVO between sutureless- and conventional-repair group ($P = .29$). *PPVO*, Postoperative pulmonary venous obstruction.

TAPVC and there is a high likelihood of pulmonary vascular changes.[29]

Interesting, DPC was associated with lower incidence of PPVO, which intuitively appears to be more related to the underlying anatomy and surgical adequacy. An explanatory contributor to this observation may be the greater preoperative CTA completion rate in the DPC group. There is a great morphologic heterogeneity in TAPVC, for example, the confluence can feature an inherent variation in not only size but also shape and orientation, introducing patient-specific particularities.[30] Emerging evidence has stressed the crucial role of CTA in clearly delineating the anomalous venous channels and providing information of the positional relationship between confluence and LA.[15,23,24,31,32] This helps the surgeon better comprehend the detailed morphology and assists in decision-making, which is associated with surgical success (eg, surgical perfection, faster surgery) and accordingly lower rate of PPVO. In addition, the finding that access to presurgical CTA is associated with shorter cross-clamping time partially supports this notion.

The lower preoperative CTA completion in the no-DPC group was probably ascribed to the fact that these patients were prone to developing clinical instability and proceeded to emergent operation. Reasons are multifactorial. First, lack of expertise in caring for patients with critical congenital heart diseases potentially results in poorer recognition of the relevant cardiopulmonary physiology and induces some inappropriate treatments. For example, when in the face of fixed obstruction, pulmonary vasodilators or prostaglandin therapy may result in worsening pulmonary edema and can be detrimental. In addition, placement of umbilical venous catheters in those with infracardiac TAPVC can increase the risk of obstruction because the placed catheters may enter the pulmonary venous confluence and descending vein, therefore resulting in obstruction. Second, lack of serial evaluation precludes a dynamic blood gas parameter-guided treatment, which may be an obstacle to treatment effectiveness. However, we cannot rule out the possibility that patients in the no-DPC group might be more critically ill.

A significant lower rate of PHC was observed in the DPC group compared with no-DPC group. Previous studies[27,33-35] have shown that the occurrence of postoperative PHC could be an early indication of undetected level of micro-obstruction (intrinsic hypertrophy and fibrosis of the pulmonary vessel) which would potentially progress. Yamaki and colleagues[29] have found that both media and intima of PVs are thickened in patients with TAPVC (especially obstructed TAPVC). Such inherently abnormal pulmonary vessels are prone to vasoconstriction in response to preoperative hypoxia, hypercapnia, and/or acidosis, which potentially leads to hyperreactivity in the pulmonary vasculature predisposing to

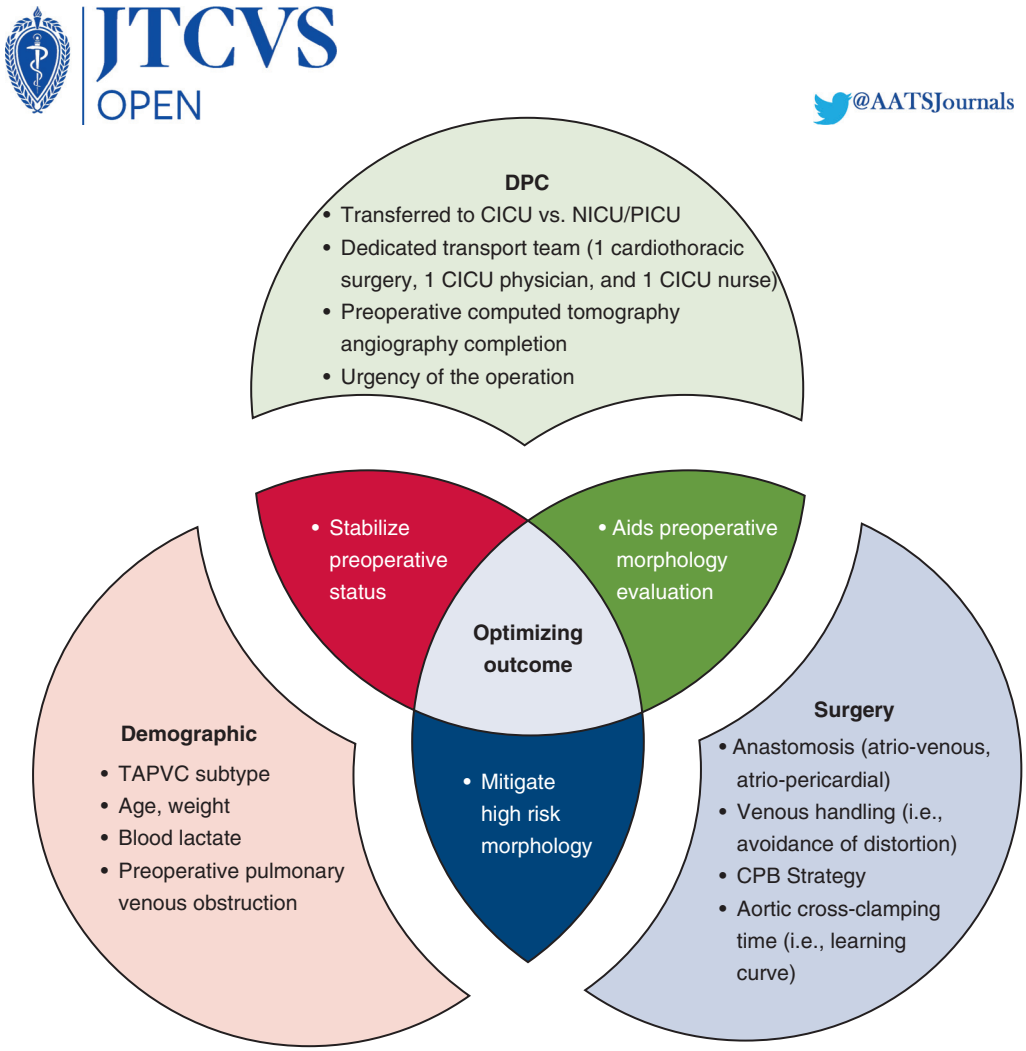


FIGURE 5. Patient demographics, surgical techniques, and DPC are 3 predictors for outcomes after neoTAPVC surgery. DPC is helpful for stabilization of patient preoperative status, and allows preoperative CTA, which aids individual morphologic evaluation. *DPC*, Dedicated presurgical care; *CICU*, cardiac intensive care unit; *NICU*, neonatal intensive care unit; *PICU*, pediatric intensive care unit; *CPB*, cardiopulmonary bypass; *TAPVC*, total anomalous pulmonary venous connection; *neoTAPVC*, neonatal total anomalous pulmonary venous connection; *CTA*, computed tomography angiography.

the onset of postoperative PHC. In addition, as aforementioned, more patients underwent preoperative CTA in the DPC group. It is plausible that CTA-based surgical strategy may contribute to the procedural perfection. This is helpful for maintaining a low pressure and keeping laminar flow in the individual PVs, which reduces the risk of vascular remodeling triggered by locoregional hemodynamic factors.

No clear superiority of sutureless repair to direct atrio-pericardial in minimizing PPVO was observed in the PSM analysis, contrary to the general thinking that prophylactic sutureless repair can potentially mitigate the risk of PPVO in difficult TAPVC subgroup (ie, neonatal patents).[5] Preoperative CTA-guided surgery can produce clinical benefits in terms of lowering PPVO[31] and is a probable explanatory factor. In contrast, the purse-string effect of the suture line or fulcrum effect of the pericardial reflection adjacent to the individual PVs could potentially cause PV angulation, which are the mechanism of PPVO in patients undergoing sutureless repair.[36] Nevertheless, we cannot rule out the possibility that there were fewer patients with infracardiac and mixed TAPVC, as previous studies have revealed striking advantages of sutureless repair for these 2 subtypes versus no tangible benefit in supracardiac or cardiac subtype.

Limitations

This study has several limitations. First, patients in this cohort tended to be older at presentation (median age at admission of 12 days), and there was possibility that more critically ill newborns may die before referral, potentially

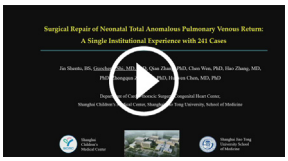
reflecting a selection bias. Second, there may be underestimation or overestimation of the postoperative PHC, in which situation the diagnosis was only based on a combination of echocardiographic calculation and clinical judgement. Third, there was some changes in postoperative care during the study period that potentially influenced the results. For example, inhaled nitric oxide was not available until August, 2019 in our center, which has been recommended as the first choice in mechanically ventilated patients as a pulmonary hypertension targeted therapy.[20,21] Hence, we cannot rule out the possibility that access to inhaled nitric oxide after 2019 might have beneficial effects on those who developed postoperative PHC, which might be a confounding factor. In addition, other unmeasured variables influenced by the era effect may potentially affect the results.

CONCLUSIONS

Dedicated care directed at preoperative stabilization, if feasible, is potentially worthwhile before surgery in neonatal patients with TAPVC, in terms of better survival and freedom from PPVO (Figure 5). Our finding suggests that freedom from PPVO were similar using conventional versus sutureless repair.

Webcast

You can watch a Webcast of this AATS meeting presentation by going to: https://www.aats.org/resources/surgical-repair-of-neonatal-total-anomalous-pulmonary-venous-return-a-single-institutional-experience-with-241-cases.



Conflict of Interest Statement

The authors reported no conflicts of interest.

The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling or reviewing manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

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Key Words: atriopericardial repair, dedicated care, neonate, PVO, TAPVC

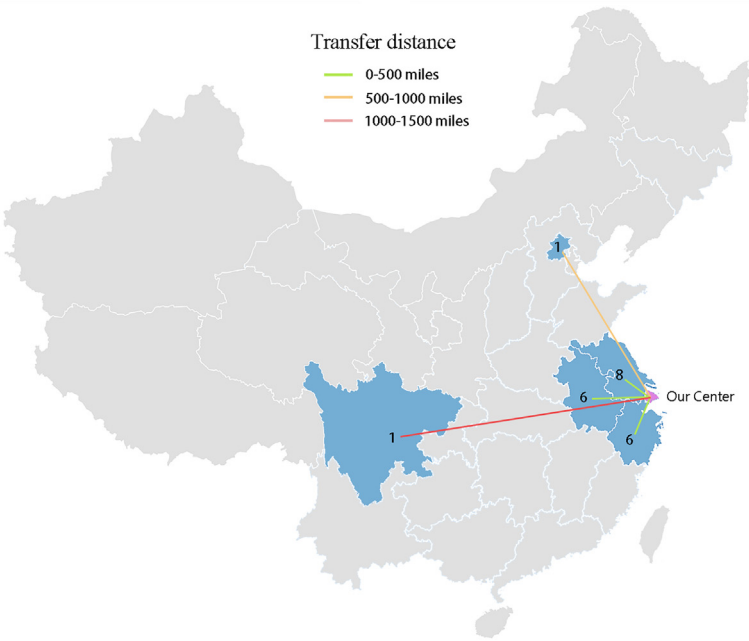

FIGURE E1. Geographic distribution of patients who transferred with a dedicated heart team. A total of 22 patients in the DPC group (21%, 22/107) transferred from other provinces through a dedicated heart team, among whom 22 were from the neighboring area ranging from 85 to 402 miles. The remaining 2 patients were from the distant areas (751 and 1242 miles, respectively). *DPC*, Dedicated presurgical care.

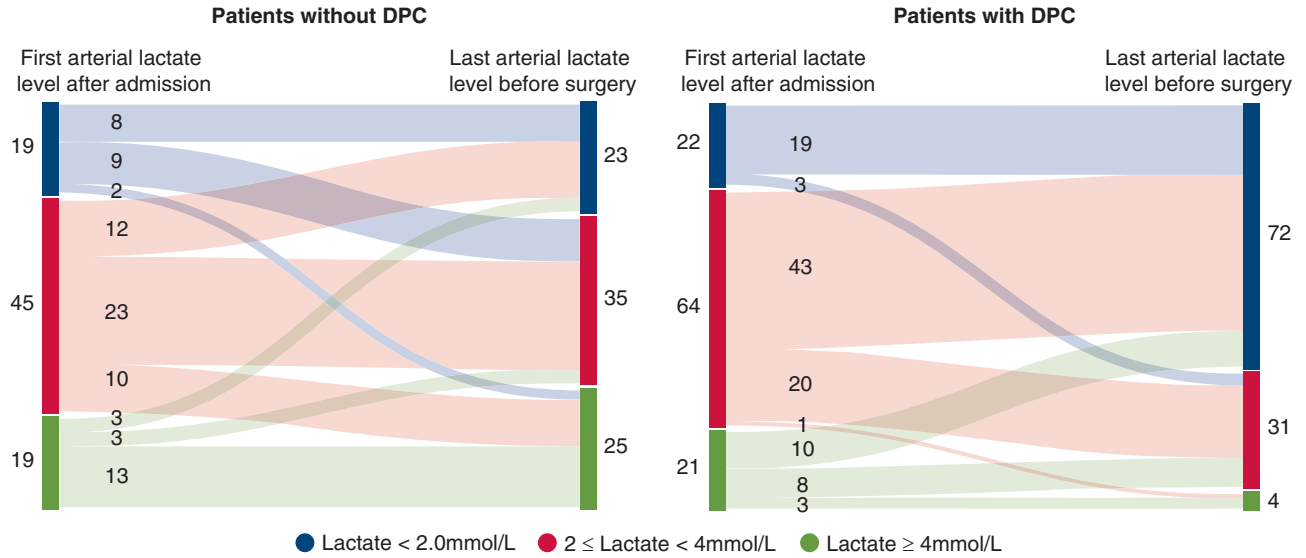

FIGURE E2. Dynamic changes in preoperative arterial lactate level stratified by different severity. Serial monitoring of preoperative arterial lactate level was available in all of the patients with DPC and in 83 of the 134 patients without DPC. Downgrade of the preoperative arterial lactate level was found in 61 of the 107 patients receiving DPC, including 43 from moderate to normal, 10 from severe to normal, and 8 from severe to moderate. However, arterial lactate level remained unchanged in 44 of the 83 patients receiving no DPC; of note, deterioration of the preoperative arterial lactate level was found in 21 of the 83 patients, including 9 from normal to moderate, 2 from normal to severe, and 10 from moderate to severe. *DPC*, Dedicated presurgical care.

TABLE E1. Characteristics of 174 patients who received conventional versus sutureless repair before and after propensity-score matching

Characteristic	Before propensity-score matching			After propensity-score matching		
	Conventional repair (n = 130)	Sutureless repair (n = 44)	*P* value	Conventional repair (n = 31)	Sutureless repair (n = 31)	*P* value
Male, n (%)	78 (60.0%)	33 (75.0%)	.10	24 (77.4%)	25 (80.0%)	>.99
Age, d, median (IQR)	16.0 (9.0-22.3)	10.0 (6.0-13.8)	<.001	11 (8-15)	11 (7-15)	.77
Weight, kg, median (IQR)	3.5 (3.0-3.7)	3.3 (3.1-3.5)	.12	3.4 (2.9-3.8)	3.3 (3.2-3.5)	.91
Associated cardiac lesion, n (%)						
ASD	130 (100%)	44 (100%)	>.99	31 (100%)	31 (100%)	>.99
PDA	85 (65.4%)	32 (72.7%)	.46	22 (71.0%)	23 (74.2%)	>.99
Subtype of TAPVC, n (%)			<.001			.51
Supracardiac	43 (33.1%)	20 (45.5%)		14 (45.2%)	16 (51.6%)	
Cardiac	47 (36.2%)	1 (2.3%)		1 (3.2%)	1 (3.2%)	
Infracardiac	27 (20.8%)	21 (47.7%)		16 (51.6%)	12 (38.7%)	
Mixed	13 (10.0%)	2 (4.5%)		0 (0%)	2 (6.5%)	
Operation era, n (%)			<.001			.12
2015-2017	80 (61.5%)	9 (20.5%)		16 (51.6%)	9 (29.0%)	
2018-2020	50 (38.5%)	35 (79.5%)		15 (48.4%)	22 (71.0%)	
Preoperative PVO, n (%)	92 (70.8%)	31 (70.5%)	>.99	23 (74.2%)	22 (71.0%)	>.99
Spo_2, median (IQR)	85.0 (80.0-90.0)	88.0 (79.3-94.0)	.055	85.0 (75.0-90.0)	85.0 (78.0-94.0)	.26
Last lactate before surgery, mmol/L, median (IQR)	2.0 (1.2-3.1)	1.8 (0.9-2.9)	.21	1.6 (0.8-2.4)	1.8 (0.9-2.8)	.93
Preoperative management, n (%)						
Inotropes	52 (40.0%)	25 (56.8%)	.056	18 (58.1%)	17 (54.8%)	>.99
Mechanical Intubation	40 (30.8%)	11 (25.0%)	.57	11 (35.5%)	9 (29.0%)	.79
UVC	21 (16.2%)	15 (34.1%)	.017	8 (25.8%)	8 (25.8%)	>.99
PGE1	14 (10.8%)	8 (18.2%)	.29	6 (19.4%)	3 (9.7%)	.47
Pulmonary vasodilators	13 (10.0%)	4 (9.1%)	>.99	3 (9.7%)	2 (6.5%)	>.99
DPC, n (%)	75 (57.7%)	19 (43.2%)	.12	19 (61.3%)	16 (51.6%)	.61
Operation within 24 h on presentation, n (%)	43 (33.1%)	16 (36.4%)	.72	13 (41.9%)	12 (38.7%)	>.99

Variables listed were used to generate the propensity score. Matching on the generated propensity scores was performed using 1:1 matching with a fixed caliper width set at 0.1 standard deviation of the logit of the propensity score. A total of 72 patients could be matched according to the propensity score. *IQR*, Interquartile range; *ASD*, atrial septal defect; *PDA*, patent ductus arteriosus; *TAPVC*, total anomalous pulmonary venous connection; *PVO*, pulmonary venous obstruction; *UVC*, umbilical venous catheterization; *PGE1*, prostaglandin E1; *DPC*, dedicated presurgical care.

TABLE E2. Details of postoperative PVO

Age, d	Weight, kg	Preoperative PVO	TAPVC subtype	Time interval,* d	Details of PPVO	Reintervention	Late death
26	3.2	No	Supracardiac	7	Restrictive communication between PV and LA, velocity: 1.90 m/s	No; ongoing surveillance	No
13	3.3	Yes	Supracardiac	12	Restrictive communication between PV and LA, velocity: 2.10 m/s	No; ongoing surveillance	No
7	3.3	Yes	Infracardiac	7	Obstructed RPV, velocity: 1.95 m/s	No; ongoing surveillance	No
3	4.5	Yes	Supracardiac	5	Restrictive communication between PV and LA, velocity: 1.88 m/s	No; ongoing surveillance	No
25	3.0	No	Infracardiac	483	Obstructed RPV, velocity: 1.86 m/s	No; ongoing surveillance	No
29	3.0	Yes	Cardiac	32	Restrictive communication between PV and LA, velocity: 2.08 m/s Obstructed LIPV, velocity: 2.42 m/s	No; ongoing surveillance	No
28	3.8	Yes	Infracardiac	73	Restrictive communication between PV and LA, velocity: 2.28 m/s	No; ongoing surveillance	No
10	3.2	Yes	Supracardiac	2	Obstructed RPV, velocity: 1.80 m/s	No; ongoing surveillance	No
19	3.5	No	Infracardiac	132	Restrictive communication between PV and LA, velocity: 1.85 m/s	No; ongoing surveillance	No
14	3.3	No	Mixed	91	Obstructed LUPV, velocity: 1.94 m/s	No; ongoing surveillance	No
7	3.5	Yes	Infracardiac	3	Restrictive communication between PV and LA, velocity: 2.67 m/s	No; ongoing surveillance	No
16	3.5	No	Infracardiac	90	Restrictive communication between PV and LA, velocity: 2.40 m/s	No; ongoing surveillance	No
19	4.0	Yes	Cardiac	300	Obstructed LIPV, velocity: 2.40 m/s	No; ongoing surveillance	No
12	2.4	Yes	Supracardiac	17	Restrictive communication between PV and LA, velocity: 2.38 m/s	No	Yes
7	3.5	Yes	Infracardiac	7	Restrictive communication between PV and LA, velocity: 1.93 m/s	No	Yes
24	3.6	No	Infracardiac	78	Restrictive communication between PV and LA, velocity: 2.33 m/s	No; ongoing surveillance	No
14	4.5	Yes	Supracardiac	341	Restrictive communication between PV and LA, velocity: 1.90 m/s	No; ongoing surveillance	No
11	3.1	Yes	Infracardiac	60	Restrictive communication between PV and LA, velocity:1.80 m/s	No; ongoing surveillance	No
16	2.4	Yes	Supracardiac	118	Restrictive communication between PV and LA, velocity: 2.30 m/s	No; ongoing surveillance	No
16	3.5	No	Cardiac	55	Restrictive communication between PV and LA, velocity: 2.64 m/s	Yes	No
19	3.2	Yes	Supracardiac	8	Restrictive communication between PV and LA, velocity: 2.40 m/s	No; ongoing surveillance	No
8	3.7	No	Supracardiac	60	Restrictive communication between PV and LA, velocity: 1.83 m/s	No; ongoing surveillance	No
27	3.8	Yes	Infracardiac	26	Restrictive communication between PV and LA, velocity: 2.20 m/s	No; ongoing surveillance	No
23	4.0	Yes	Infracardiac	107	Restrictive communication between PV and LA, velocity: 1.95 m/s	No; ongoing surveillance	No
5	3.5	Yes	Mixed	2	Restrictive communication between PV and LA, velocity: 2.00 m/s	No; ongoing surveillance	No
15	3.4	Yes	Infracardiac	18	Restrictive communication between PV and LA, velocity: 1.90 m/s	No; ongoing surveillance	No
28	3.7	Yes	Supracardiac	1	Obstructed LPV, velocity: 2.69 m/s Obstructed RPV, velocity: 2.02 m/s	No; ongoing surveillance	No
18	3.9	No	Infracardiac	25	Restrictive communication between PV and LA, velocity: 1.80 m/s	Yes	No
30	3.6	No	Infracardiac	103	Restrictive communication between PV and LA, velocity: 2.25 m/s Obstructed RPV, velocity: 2.00 m/s Obstructed LPV, velocity: 1.96 m/s	No; ongoing surveillance	No

(Continued)

TABLE E2. Continued

Age, d	Weight, kg	Preoperative PVO	TAPVC subtype	Time interval,* d	Details of PPVO	Reintervention	Late death
1	3.2	Yes	Infracardiac	26	Obstructed LPV, velocity: 2.96 m/s	No	Yes
27	4.2	Yes	Mixed	0	Obstructed LPV, velocity: 2.00 m/s	Yes	No
11	3.2	Yes	Supracardiac	14	Restrictive communication between PV and LA, velocity: 3.00 m/s Obstructed RPV, velocity: 2.30 m/s Obstructed LPV, velocity: 1.80 m/s	No; ongoing surveillance	No
23	3.5	No	Mixed	673	Restrictive communication between PV and LA, velocity: 1.80 m/s	No; ongoing surveillance	No
10	2.7	Yes	Supracardiac	43	Restrictive communication between PV and LA, velocity: 2.00 m/s	No; ongoing surveillance	No
4	4.2	No	Cardiac	65	Restrictive communication between PV and LA, velocity: 2.82 m/s	No; ongoing surveillance	No
7	3.2	Yes	Supracardiac	51	Restrictive communication between PV and LA, velocity: 1.90 m/s	No; ongoing surveillance	No
4	3.5	No	Supracardiac	77	Obstructed RPV, velocity: 2.60 m/s Obstructed LPV, velocity: 3.00 m/s	Yes	No
7	3.1	Yes	Infracardiac	6	Restrictive communication between PV and LA, velocity: 2.70 m/s	No	Yes
12	3.3	Yes	Infracardiac	45	Restrictive communication between PV and LA, velocity: 1.80 m/s	Yes	Yes
19	2.9	Yes	Infracardiac	24	Restrictive communication between PV and LA, velocity: 2.60 m/s	Yes	No
5	3.2	Yes	Supracardiac	2	Restrictive communication between PV and LA, velocity: 1.90 m/s	No	Yes
13	2.3	Yes	Supracardiac	4	Restrictive communication between PV and LA, velocity: 2.40 m/s	No	Yes
22	3.6	Yes	Cardiac	29	Obstructed LUPV, velocity: 1.85 m/s	No; ongoing surveillance	No
8	3.0	Yes	Infracardiac	5	Restrictive communication between PV and LA, velocity: 3.00 m/s	No; ongoing surveillance	No
0	3.5	Yes	Supracardiac	3	Restrictive communication between PV and LA, velocity: 2.57 m/s	No	Yes
12	3.5	Yes	Supracardiac	47	Restrictive communication between PV and LA, velocity: 2.50 m/s Obstructed LPV, velocity: 2.30 m/s	Yes	No
23	3.0	Yes	Mixed	27	Obstructed RPV, velocity: 2.00 m/s Obstructed LIPV, velocity: 2.10 m/s	No	Yes
7	3.0	No	Mixed	49	Restrictive communication between PV and LA, velocity: 2.02 m/s	No; ongoing surveillance	No
7	3.5	Yes	Supracardiac	32	Obstructed RIPV, velocity: 2.21 m/s Obstructed LUPV, velocity: 2.00 m/s	No; ongoing surveillance	No
3	3.0	No	Infracardiac	51	Restrictive communication between PV and LA, velocity: 2.16 m/s	No; ongoing surveillance	No
6	3.1	No	Infracardiac	87	Restrictive communication between PV and LA, velocity: 1.88 m/s	No; ongoing surveillance	No
0	3.5	Yes	Supracardiac	65	Obstructed LIPV, velocity: 1.83 m/s	Yes	No
21	3.3	Yes	Supracardiac	105	Restrictive communication between PV and LA, velocity: 2.32 m/s	No; ongoing surveillance	No
20	3.3	Yes	Supracardiac	47	Restrictive communication between PV and LA, velocity: 2.37 m/s	No; ongoing surveillance	No

PVO, Pulmonary venous obstruction; *TAPVC*, total anomalous pulmonary venous connection; *PPVO*, postoperative pulmonary venous obstruction; *PV*, pulmonary vein; *LA*, left atrium; *RPV*, right pulmonary vein; *LIPV*, left inferior pulmonary vein; *LUPV*, left upper pulmonary vein; *LPV*, left pulmonary vein. *The time interval from the operative day to the onset of postoperative PVO.