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Frequency of Superior Vena Cava Obstruction in Pediatric Heart Transplant Recipients and Its Relation to Previous Superior Cavopulmonary Anastomosis

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Abstract

The risk factors for superior vena cava (SVC) obstruction after pediatric orthotopic heart transplantation (OHT) have not been identified. This study tested the hypothesis that pretransplant superior cavopulmonary anastomosis (CPA) predisposes patients to SVC obstruction. A retrospective review of file Pediatric Cardiac Care Consortium registry from 1982 through 2007 was performed. Previous CPA, other cardiac surgeries, gender, age at transplantation, and weight at transplantation were assessed for the risk of developing SVC obstruction. Death, subsequent OHT, or reoperation involving the SVC were treated as competing risks. Of the 894 pediatric OHT patients identified, 3.1% (n = 28) developed SVC obstruction during median follow-up of 1.0 year (range: 0 to 19.5 years). Among patients who developed SVC obstruction, 32% (n = 9) had pretransplant CPA. SVC surgery before OHT was associated with posttransplant development of SVC obstruction (p < 0.001) after adjustment for gender, age, and weight at OHT and year of OHT. Patients with previous CPA had increased risk for SVC obstruction compared with patients with no history of previous cardiac surgery (hazard ratio 10.6, 95% confidence interval: 3.5 to 31.7) and to patients with history of non-CPA cardiac surgery (hazard ratio 4.7, 95% confidence interval: 1.8 to 12.5). In conclusion, previous CPA is a significant risk factor for the development of post-heart transplant SVC obstruction. Published by Elsevier Inc. (*Am J Cardiol* 2013;112:286–291)

Orthotopic heart transplantation (OHT) is used successfully for patients with end-stage cardiac failure or irreparable congenital heart disease. Superior vena cava (SVC) obstruction has emerged as a notable complication following OHT, particularly with the bicaval method of graft implantation.^{1–3} It has been postulated that younger age at transplantation, donor—

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recipient caval mismatch,^{4,5} and previous surgery involving the SVC,⁶ and previous superior cavopulmonary anastomosis (CPA)⁷ may be risk factors for posttransplant SVC obstruction. However, the incidence of SVC obstruction in pediatric heart transplant patients and the effect of pretransplant exposure to previous superior CPA or other types of non-CPA SVC surgeries remain unknown. This study describes the incidence of SVC obstruction in pediatric transplant recipients from a large multicenter database and investigates its association with previous cardiac surgeries and other risk factors. We hypothesized that patients with previous superior CPA, younger age, and smaller weight at transplantation would indicate higher risk for SVC obstruction.

Methods

We conducted a retrospective cohort study using data from the Pediatric Cardiac Care Consortium (PCCC), a voluntary multi-institutional registry collecting outcome data after cardiac procedures, including surgeries and cardiac catheterizations, for pediatric or adult congenital heart diseases. The registry includes data from 50 institutions in United States and Canada. All cardiac catheterizations and operations performed during the years a center was participating in PCCC are included in the database. Each site maintains its own evaluation and management approach for heart transplantation. Details about the nature and operation of the PCCC have been described before.^{8,9} The study was approved by the Institutional Review Board at the University of Minnesota without requirement for patient consent. We identified pediatric patients (<18 years at transplantation) who underwent OHT in a PCCC center from 1982 to 2007. Data reviewed included age and weight at transplant, patient's gender, diagnoses, and types and timing of interventional procedures. CPA was defined as any superior CPA, regardless of whether simultaneous or subsequent inferior CPA was performed. Patients without CPA were grouped by whether they had had previous cardiac surgery. We considered that patients with non-CPA surgery involving the SVC, such as partial anomalous pulmonary vein repair and atrial baffle procedures might also be at increased risk, but these represented only 3.8% of our cohort, and none developed SVC obstruction, so we grouped them with other non-CPA cardiac surgery patients in the analysis. Diagnosis of SVC stenosis was made by the center's managing team and may have included a combination of clinical, imaging, and hemodynamic data, which may not be available to PCCC. All diagnoses of SVC obstruction were included when made at the time of the first cardiac procedure reported in the PCCC (either cardiac catheterization or surgery), independent of when or whether an intervention to relieve took place. Cardiac catheterizations were performed at the discretion of the treating team, either as a routine posttransplantation surveillance, or secondary due to other clinical indications. SVC obstructions identified in autopsy were not included in this study.

Among 90,124 patients operated in the PCCC, 971 pediatric cases with OHT were identified. Only the first OHT per patient was included. We excluded patients with incomplete or conflicting data (n = 22), pretransplant SVC obstruction (n = 11), first OHT outside of PCCC (n = 9), left or bilateral SVCs, as well as patients requiring additional SVC-related surgery at the time of transplant, such as partial anomalous pulmonary venous repair (n = 10) or placed on mechanical circulatory support (n = 23) and patients who met

multiple exclusion criteria (n = 2). After exclusions 894 patients were available for analysis (age range: 1 day to 18 years, median: 3.4 years).

Primary outcome was time from OHT to SVC obstruction before death, subsequent OHT, or reoperation involving the SVC. Follow-up data were available each time a patient had a subsequent cardiac procedure in a PCCC center. Because of the multiple outcomes following OHT, we used a competing risk analysis with SVC obstruction, death, retransplantation, and additional nontransplant cardiac surgery treated as competing risks.^{10,11} These competing risks were used to analyze the relationship between SVC obstruction and CPA or otii» previous cardiac surgery, adjusting for gender, age at OHT, weight at OHT, and decade of OHT. Statistical significance was tested using Wald chi-square statistics at the $p < 0.05$ level.¹² Age, year of OHT, and weight were allowed to be nonlinear using restricted cubic splines, with 3 knots each set at the 10th, 50th, and 90th percentiles.¹² The proportional hazard assumption was assessed by plotting scaled Schoenfeld residuals. The hazard ratios (HRs) and 95% confidence intervals (CIs) comparing the 75th percentile and the 50th percentile to the 25th percentile for each continuous variable were computed along with the HR and 95% CI for the categorical variables.

Results

The characteristics of patients undergone OHT are described in Table 1. The median follow-up time was 1 year (range: 0 to 19.5 years). During follow-up, 28 patients (3%) developed SVC obstruction, 122 (14%) patients died, 57 (6%) had a second OHT or a subsequent surgery involving the SVC, and 687 (77%) were observed for variable lengths of time without reaching any of these end points. The cumulative incidence of SVC obstruction in OHT patients was 2.6% (95% CI: 1.4 to 3.7) at 6 months, 3.4% (95% CI: 2.0 to 4.7) at 1 year, 3.6% (95% CI: 2.2 to 5.0) at 3 years, and 4.1% (95% CI: 2.5 to 5.7) at 5 years (Figure 1). Characteristics of patients with and without SVC obstruction are available in Table 2.

History of surgery involving the SVC before OHT was associated with an increased risk of SVC obstruction following OHT ($p = 0.001$) after adjustment for gender, age, and weight at OHT and year of OHT (Table 3). Furthermore, age appeared to have a nonlinear association with risk of SVC obstruction following OHT. There was no consistent relationship between risk of SVC obstruction and age or weight. Patients who underwent CPA were at significantly higher risk of SVC obstruction compared with those patients with no pre-OHT cardiac surgical history (HR: 10.6, 95% CI: 3.5 to 31.7; Table 4). Similarly, CPA patients had a higher risk of SVC obstruction after transplant than patients with a history of all other types of cardiac surgery (HR: 4.7, 95% CI: 1.8 to 12.5). Patients who underwent non-CPA cardiac surgery were at higher risk of SVC obstruction compared with those patients with no cardiac surgical history (HR: 2.2, 95% CI: 0.79 to 6.4), although this was not statistically significant (Table 4). From the non-CPA patients who developed SVC obstruction after OHT (n = 19), 13 had undergone bicaval technique versus 5 biatrial, and in one case, no information was available about the surgical technique. However, without knowing the overall number of patients undergoing bicaval versus biatrial technique, this information cannot infer relationship with SVC obstruction. Of the 28 patients with SVC obstruction following OHT, 54% (n = 15) of the patients required SVC intervention. Of these patients,

73% (n = 11) underwent percutaneous intervention (balloon angioplasty, n = 2; or stent placement, n = 9) and 27% (n = 4) underwent surgical patch augmentation (Table 5).

Discussion

SVC obstruction is a rare complication following heart transplantation.⁴ The original biatrial transplantation technique¹⁴ incurred risk of important complications including impaired right heart hemodynamics, atrioventricular valve insufficiency, sinus node dysfunction, and atrial arrhythmias.^{15,16} The bicaval technique, introduced in 1991,¹⁷ has replaced the biatrial technique in many institutions because of significant reduction in the aforementioned morbidities.¹⁸⁻²² Despite its advantages, the bicaval technique has been associated with increased ischemic time, higher rates of anastomotic stricture, and higher rates of venous thrombosis.^{23,24} There are few published reports on the incidence of SVC obstruction in transplant recipients and on the role of previous CPA as an independent risk factor for SVC obstruction after OHT. Although some reports highlight the association of SVC obstruction following CPA in both transplant^{7,25} and nontransplant patients, other publications do not confirm this association.^{27,28}

Our study demonstrates an increased risk for posttransplant SVC obstruction in patients with history of previous superior CPA compared with patients with no history of previous cardiac surgery and a history of other cardiac surgeries, with or without SVC involvement. We speculate that this could be the result of previous manipulation of the SVC, resulting in vascular scarring or limited mobility and shortening of the SVC and thus in increased tension on the SVC after OHT. Patients with history of CPA routinely have bicaval OHT because the SVC has already been disconnected from the right atrium. This would potentially increase the risk of SVC obstruction given that this is a second surgical manipulation of the SVC. In addition, any SVC compromise could be more symptomatically or hemodynamically significant in the CPA group; these patients may lack the ability to decompress SVC congestion because the azygos vein is often ligated at the time of CPA. Modifying the anastomotic technique may limit this complication. For example, use of an interrupted rather than continuous suture technique can prevent a purse string effect at the anastomosis. When the SVC is short, an interposition graft can be used to reconstruct the superior vena cava. A nonvalved segment of bovine jugular vein conduit can be used for this purpose. If the SVC has sufficient length for a tension-free anastomosis, it can be implanted in the right atrial appendage. In this case, it is important to resect pectinate muscles in the appendage to ensure unobstructed path.

An increased incidence of SVC obstruction in younger and smaller transplant patients has been reported.^{7,28} Our study population showed an association between younger age at transplantation with the development of SVC obstruction after OHT, but it failed to show similar association with patient weight. Several factors may contribute to a higher rate of SVC complications in younger patients. The short length of the donor SVC may contribute to postimplantation SVC obstruction, particularly when coupled with other potential risk factors such as those mentioned. Other factors that may increase the risk for SVC obstruction in younger patients include smaller SVC size and increased donor-recipient SVC

size mismatch. These important variables are not routinely available in the PCCC registry and therefore could not be analyzed.

Our data show a gradually increased rate of SVC obstruction over time, coinciding with the spread of the bicaval technique across surgical centers. Of interest, the majority of SVC obstruction in non-CPA patients occurred with this technique. However, to address the effect of the surgical technique on the incidence of SVC obstruction after OHT, a review of all surgical procedures without SVC obstruction, a review of all surgical procedures without SVC obstruction is needed. This is outside of the scope of this exploratory study and will be the subject of a future targeted investigation.

Our study limitations include its retrospective nature and the nature of information available in the PCCC database as described earlier. Loss to follow-up is a limitation of all registry studies but should have been reduced because all heart transplant patients require close clinical follow-up. Because some patients appear to have received posttransplantation care outside the PCCC, long-term follow-up data are incomplete, and the absolute incidence of SVC obstruction may be underestimated. Variability in duration of follow-up was accounted for in our analysis, but we cannot rule out the possibility of systematic bias due to differences between groups. The role of the transplant technique (bicaval vs biatrial) as a risk factor for SVC stenosis post-OHT cannot be addressed by this study without reviewing all surgical procedures. In addition, the long study period and the different institutional SVC obstruction definition and approach introduce confounding variables such as changing surgical technique.

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Figure 1.
Cumulative incidence plot of SVC obstruction, death, OHT, or reoperation.

Table 1.

Characteristics of orthotopic heart transplant recipients in the Pediatric Cardiac Care Consortium

Characteristics	n(%) [*]
Gender	
Male	494(55)
SVC-related surgery before OHT	
Previous CPA	84(9%)
Previous SVC surgery/other	284 (32%)
Age at OHT (yrs), median (min, max)	3.4 (0, 17.9)
Weight at OHT (kg), median (min, max)	12.9 (2.1,113)
Decade	
1982–1989	52 (6%)
1990–1999	411 (46%)
2000–2007	431 (48%)
Total	894

^{*} Unless otherwise noted

Table 2.

Characteristics of patients with and without superior vena cava obstruction

Variable	SVC Obstruction (n = 28)	Death (n = 122)	O HT/Reop eration (n = 57)	No Event (n = 687)
Age at OHT (yrs)	4.9 ± 6.4, 0.85	4.8 ± 6.1, 1.3	5.4 ± 5.3, 4.2	6.7 ± 6.3, 4.1
Weight at OHT (kg)	16.3 ± 16.1, 9.4	18.1 ± 21.2, 8.3	20.7 ± 22.5, 12.0	23.9 ± 21.5, 14.7
Male	16 (57%)	65 (53%)	30 (53%)	383 (56%)
Previous CPA	9 (32%)	14 (11%)	7 (12%)	54 (8%)
Previous SVC/other surgery Decade	9 (32%)	36 (30%)	20 (35%)	219 (32%)
1982–1989	2(7%)	16 (13%)	7 (12%)	27 (4%)
1990–1999	9 (32%)	63 (52%)	33 (58%)	306 (45%)
2000–2007	17 (61%)	43 (35%)	17 (30%)	354 (52%)

Mean ± SD along with medians (mean ± SD, Median) are shown for continuous variables and counts and percentages, N (%), are shown for categorical variables.

Table 3.

Wald statistics for competing risks modeling time to superior vena cava obstruction after orthotopic heart transplant

Variable	Chi-Square	df	p Value
History of surgery involving SVC before OHT	19.10	2	0.0010 *
Gender	0.03	1	0.8701
Age (yrs)	8.02	2	0.0181 *
Nonlinear component	3.22	1	0.0730
Surgical weight (kg)	4.48	2	0.1062
Nonlinear	0.18	1	0.6689
Yr of transplant	1.74	2	0.4180
Nonlinear	1.30	1	0.2548
Total Nonlinear	9.33	3	0.0252
Total	26.59	7	0.0004

Overall tests for each factor are presented along with the test for each nonlinear part and the overall tests for the model, df, degrees of freedom.

* p value <0.05.

Table 4.

Hazard ratios for development of superior vena cava obstruction following orthotopic heart transplant

Variable	HR	Lower 95% CI	Upper 95% CI
History of surgery associated with SVC before OHT			
CPA vs none	10.6 *	3.5	31.7
CPA vs SVC surgery/other	4.7 *	1.8	12.5
SVC surgery/other vs none	2.2	0.79	6.4
Gender			
Female vs male	1.1	0.50	2.3
Age			
50th vs 25th percentile (3.4 vs 0.4 yrs)	0.27	0.03	2.5
75th vs 25th percentile (13.1 vs 0.4 yrs)	0.79	0.01	43.5
Surgical weight			
50th vs 25th percentile (12.9 vs 5.4 kg)	0.97	0.19	5.11
75th vs 25th percentile (37.5 vs 5.4 kg)	0.43	0.01	23.3
Transplant yr			
50th vs 25th percentile (1999 vs 1995)	1.49	0.82	2.71
75th vs 25th percentile (2004 vs 1995)	1.27	0.57	2.85

*
p value <0.05.

Table 5.

Details of patients who developed superior vena cava obstruction after orthotopic heart transplant

Patient	Diagnosis	Pre-OHT Surgeries		Pre-OHT Risk	OHT Era*	Wt(kg) at OHT	OHT Technique	Age (yrs) at OHT	Age (yrs) at dx of SVC	Intervention(s)
1	PATVS	Shunt, Glenn + ECMO, shunt + ECMO	Glenn	Glenn	3	10	Bicaval	0.5	1	Stent, balloon
2	TGA, mitral stenosis	DKS + Glenn	Glenn	Glenn	3	23	Bicaval	0.8	0.8	Stent
3	Tricuspid atresia, coarctation of aorta	Norwood, Glenn	Glenn	Glenn	2	8	Bicaval	0.9	1.4	Stent
4	Heterotaxy, complex SV, TAPVC	TAPVC repair, Glenn	Glenn	Glenn	2	15	Unknown	7	7.8	Stent
5	Complex SV, coarctation of aorta	Glenn, Fontan, multiple reoperations	Glenn	Glenn	3	26	Bicaval	10	10.3	None as of 9 mo f/u
6	Heterotaxy, complex SV, intIVC	Glenn, Fontan	Glenn	Glenn	3	36	Bicaval	10.3	12.8	None as of 3 yr f/u
7	Heterotaxy, complex SV, TAPVC	Glenn, AV valve repair	Glenn	Glenn	3	27	Bicaval	15.6	15.7	None at dx
8	HLHS	DKS, Glenn, Fontan, ASD creation	Glenn	Glenn	3	34	Bicaval	15.6	15.7	None as of 4 mo f/u
9	Heterotaxy, complex SV, intIVC	Kawashima	Glenn	Glenn	3	51	Bicaval	16.9	17.1	Stent
10	Unbalanced AVC, arch hypoplasia	Norwood	Cardsurg	Cardsurg	3	4	Bicaval	0.1	0.1	Surgical patch
11	Complex SV, coarctation of aorta	Create ASD+VSD, AWR	Cardsurg	Cardsurg	2	4	Atrial	0.3	0.8	Surgical patch
12	Complex SV, arch hypoplasia	Multiple arch repairs	Cardsurg	Cardsurg	2	5	Atrial	0.4	0.4	None as of 1 mo f/u
13	HLHS	Norwood	Cardsurg	Cardsurg	1	3	Atrial	0.5	6.4	None as of 10 yr f/u
14	Multiple VSDs, coarctation of aorta	VSD repair	Cardsurg	Cardsurg	2	6	Bicaval	0.8	0.8	Balloon, stent
15	Situs inversus, DORV, left SVC	VSD baffle and RVOT patch	Cardsurg	Cardsurg	2	10	Bicaval	1.6	1.6	Surgical patch
16	Heterotaxy, partial AVC, intIVC	ASD repair, AV valve replacement	Cardsurg	Cardsurg	3	9	Bicaval	1.7	2.3	None at dx
17	IAA/VSD, subAS	IAA/VSD repair, Ross-Kono, CABG	Cardsurg	Cardsurg	3	15	Atrial	5.9	6.3	None as of 9 mo f/u
18	Complex SV	Fontan	Cardsurg	Cardsurg	3	45	Bicaval	16.1	16.9	None as of 4 mo f/u
19	HLHS	Fontan	Cardsurg	Cardsurg	3	11	Bicaval	2.4	2.5	None as of 7 yr f/u
20	PATVS		Cardsurg	Cardsurg	3	4	Bicaval	0	0.3	Surgical patch, stent
21	HLHS		Cardsurg	Cardsurg	3	3	Unknown	0.1	3.2	None as of 3 yr f/u
22	HLHS		Cardsurg	Cardsurg	1	3	Atrial	0.1	3.7	Stent, balloon
23	Cardiomyopathy		Cardsurg	Cardsurg	3	5	Bicaval	0.2	0.4	Balloon
24	HLHS		Cardsurg	Cardsurg	2	4	Bicaval	0.2	6.3	None as of 3 yr 17u
25	HLHS		Cardsurg	Cardsurg	2	4	Bicaval	0.2	0.6	Stent, stent
26	Cardiomyopathy		Cardsurg	Cardsurg	3	6	Bicaval	0.3	0.9	None as of 4 yr f/u
27	Cardiomyopathy		Cardsurg	Cardsurg	2	44	Bicaval	13.2	13.5	Stent
28	Pulmonary hypertension		Cardsurg	Cardsurg	3	52	Bicaval	16.3	16.5	Stent

ASD = atrial septal defect; AVC = atrioventricular canal defect; AWR = atrioventricular valve replacement; Cardsurg = non-CPA cardiac surgery; CABG = coronary artery bypass graft; DKS = Damus Kaye-Stansel procedure; DORV = double-outlet right ventricle; dx = diagnosis; ECMO = extracorporeal membrane oxygenation; f/u = follow-up; HLHS = hypoplastic left heart syndrome; IAA = interrupted aortic arch; intI VC = congenitally interrupted inferior vena cava; PATVS = pulmonary atresia with intact ventricular septum; RVOT = right ventricular outflow tract; subAS = subvalvar aortic stenosis; SV = single ventricle; TAPVC = total anomalous pulmonary venous connection; TGA = transposition of great arteries; VSD = ventricular septal defect

* OHT era: 1, 1982–1989; 2, 1990–1999; 3, 2000–2007.