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ORIGINAL ARTICLE

Postoperative Chylothorax Development Is Associated with Increased Incidence and Risk Profile for Central Venous Thromboses

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Abstract This study tested the hypothesis that pediatric patients who develop chylothorax (CTX) after surgery for congenital heart disease (CHD) have an elevated incidence and risk profile for central venous thrombosis (CVT). We evaluated 30 patients who developed CTX after surgery for CHD. All but one CTX patient were surgery-, anatomy-, and age-matched with two controls (NON-CTX) to compare their relative risk and incidence of CVT. Using conditional logistic regression analyses, CTX development was associated with significantly longer ventilator dependence (14.8 ± 10.9 vs. 6.1 ± 5.9 days, $p = 0.003$) and a non-significant trend towards more days of central venous catheters (CVC) (19.1 ± 16.6 vs. 12.2 ± 10.0 days; $p = 0.16$) when comparing the period prior to CTX development with the entire hospitalization in NON-CTX patients. CTX development was associated with a significantly elevated mortality risk (Odds Ratio 6.2, 95% CI 1.3–30.9). Minimum and mean daily central venous pressures were significantly higher in the CTX group. Post operative need for extracorporeal membrane oxygenation conferred an increased risk of CTX development in this sample of patients (Odds Ratio 9.9, 95% CI 2.2–44.8). Incidence of documented CVT was 26.7% in the CTX group versus 5.1% in the NON-CTX group. Prospective screening for CVT risk and formation, combined with early removal of CVC may help reduce the incidence of CTX.

Keywords Chylothorax · Pediatrics · Thrombosis · Surgery · Congenital heart disease

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Chylothorax (CTX) occurs postoperatively in up to 4% of all pediatric patients undergoing surgery for congenital heart disease [1, 10, 11, 27, 31]. It has been associated with prolonged ventilator dependence, increased length of hospital stay, malnutrition, nosocomial infection, and death [2, 6, 9, 10, 28, 30, 34]. Though multiple case series and case reports [4, 14, 16, 19] have suggested an association between central venous thromboses (CVT) and CTX development, no large, controlled studies have verified this association.

Pediatric patients undergoing surgery for congenital heart disease are at considerably elevated risk for CVT. In the perioperative period, these patients routinely experience all three factors [21] asserted to independently predispose to CVT formation: vascular injury is induced by central venous catheterization (CVC), cardiopulmonary bypass (CPB) produces a hypercoagulable state, and blood flow stasis occurs in the setting of central venous hypertension. Further, it has been suggested that children with congenital heart disease have an increased prevalence of genetic coagulopathies [2, 24, 25].

Given the lack of proven treatment strategies for CTX, risk factor analysis is needed to optimize the management of postoperative pediatric congenital heart surgery patients in order to reduce the incidence of CTX and its complications. Therefore, we performed a retrospective case-control study to identify factors potentially contributing to or associated with CTX in pediatric congenital heart surgery patients. We hypothesized that pediatric patients who develop CTX after surgery for congenital heart disease have a worse risk profile for and an increased incidence of CVT compared with anatomy-, surgery-, and age- matched patients who do not develop CTX.

Materials and Methods

After obtaining approval by the Institutional Review Board, the University of Virginia's Pediatric Intensive Care Unit (UVa PICU) database was queried for all patients who underwent surgery for congenital heart disease between January 1999 and November 2006. The search was narrowed for those who also developed a pleural effusion and then for those who were diagnosed with a CTX. In addition, the UVa Pediatric Pharmacy produced a list of inpatients receiving octreotide therapy. This combined search resulted in 30 such patients diagnosed with CTX, of whom 9 were treated with octreotide. The UVa Pediatric Cardiology database was then searched in an effort to surgery-, anatomy-, and age-match each CTX subject with two, non-CTX controls. In the event of multiple matches, the CTX subject was paired with non-CTX controls who underwent surgery within the same year. When exact non-CTX matches were not available for CTX patients with complex single ventricle physiology and anatomy, subjects were matched with controls who had undergone similar operations. Four biventricular patients with a combination of tetralogy of Fallot and atrioventricular canal defects (TOF/AVC) were repaired during the study period, of whom three developed CTX. The remaining five non-CTX matches were subjects who underwent repair of AVC (four) and TOF (one) defects alone. This search process produced 59 non-CTX controls (data were available for only 1 non-CTX subject status post neonatal heart transplant).

Patient data included age at admission and surgery, weight on admission, original cardiac anatomy and surgical intervention. When applicable, total CPB time was noted. Durations of intensive care unit (ICU) and total hospital stay were calculated. Daily flow sheets were examined for the entire hospital course to collect the following data: periods of no enteral feedings (NPO), periods of total parenteral nutrition, periods of low-fat or medium-chain triglyceride (MCT) formula use, periods of ventilator dependence, periods and location of CVC, periods of extracorporeal membrane oxygenation (ECMO), periods of octreotide therapy, daily volume and site of drainage of chylous fluid, and daily ranges of central venous pressure (CVP; mm Hg). Daily progress notes were evaluated in the event of uncertainty in the daily flow sheets. When overlap of two opposing periods (i.e., NPO and low-fat diet) occurred during a hospital day, the day was credited to the entity lasting >12 h. The UVa clinical data repository was searched for chest radiograph frequency and the following laboratory data: pleural WBC, percentage lymphocytes, and triglyceride (TAG) content. The repository was also evaluated for all fluoroscopy, ultrasound, MRI, CT, cardiac catheterization, and interventional radiology results to determine the date of diagnosis and incidence of documented CVT.

The date of CTX diagnosis was determined by both a change in medical management (e.g. addition of octreotide therapy, change to NPO status, change to low-fat diet) and the initial inclusion of the diagnosis "chylothorax" in the assessment and plan section of the attending physician's daily progress note. All but two CTX subjects were diagnosed and treated at our institution after either placement of a pleural drainage tube ($n = 27$) or thoracentesis alone ($n = 1$). One CTX subject who received surgical intervention was ultimately diagnosed with CTX at an outside hospital prior to being transferred back for further management, and a second CTX subject was started on the MCT formula Portagen 2 days prior to the removal of chylous pleural fluid.

Statistical Methods

To account for the 2:1 matching of non-CTX to CTX patients, conditional logistic regression was used for analysis. Multivariate conditional logistic regression analysis was performed using the continuous variables of CVC duration and CVP values prior to CTX diagnosis and the binary variable of need for ECMO prior to CTX diagnosis. These variables were chosen a priori to evaluate their association with the development of CTX. Statistical significance was defined as $p < 0.05$. All statistics were performed using the SAS statistical package (Cary, NC).

Results

Demographic data are reported in Table 1. CTX patients were ventilator dependent approximately seven times longer than non-CTX patients; this difference remained significant when comparing entire hospitalization non-CTX ventilator dependence to ventilator dependence prior to

Table 1 Demographics

Demographic variable	CTX (n = 30)	Non-CTX (n = 59)	p value/odds ratio
Age, days (median) At admission	116.3 ± 190.3 (16)	160.6 ± 225.2 (93)	0.33
At surgery	119.7 ± 186.9 (22)	164.2 ± 223.2 (93)	0.32
Admission weight, kg	4.67 ± 2.63 (3.48)	5.21 ± 2.81 (4.66)	0.15
Vent duration, days	31.9 ± 36.8 (21)	6.1 ± 5.9 (4)	0.005
Total CPB duration, min	89.5 ± 79.4 (101)	81.9 ± 69.6 (84)	0.59
Total no. CXR	69.2 ± 54 (59)	16.3 ± 10.6 (14)	0.002
Total days NPO	23.1 ± 20.9 (22)	5.9 ± 4.4 (4)	0.005
Total days HAL	28.2 ± 25.3 (23.5)	5.2 ± 6.8 (0)	0.03
ICU duration, days	38.7 ± 34.4 (28.5)	7.4 ± 6.7 (5)	0.005
Hospital duration, days	52.1 ± 34.2 (49)	16.8 ± 14.4 (10)	<0.001
Total hospital costs, $K	424 ± 393 (311)	121 ± 92 (90)	0.006
No. deaths	7 (23%)	3 (5%)	6.2 (95% CI, 1.3–30.9)

Note. CXR, chest radiograph; ICU, Intensive Care Unit; CPB, cardiopulmonary bypass; NPO, no enteral foods; HAL, hyperalimentation. All continuous data are listed as mean ± SD. Medians given in parentheses (except for no. deaths)

Table 2 CTX-group description

Anatomy	Surgery	No.
Hypoplastic left heart Syndrome	Norwood/Sano	3
Hypoplastic left heart syndrome	Hybrid procedure	1
Hypoplastic left heart syndrome	Glenn anastamosis	3
Hypoplastic right ventricle	Damus-Kaye Stansel/BT shunt	3
Severe Ebstein's	Starne's procedure/BT shunt	1
Hypoplastic right ventricle/VSD	VSD repair/ BT shunt/ASD formation	1
Tetralogy of Fallot	Complete repair	2
Tetralogy of Fallot/pulmonary atresia	Complete repair	1
Tetralogy of Fallot/pulmonary atresia	BT shunt	1
Tetralogy of Fallot/complete AV canal	Tetralogy and AV canal repair	3
Complete AV canal	AV canal repair	1
Coarctation of the aorta	Extended end-to-end reanastamosis	3
Coarctation of the aorta/VSD	VSD and coarctation repair	1
Critical aortic stenosis/hypoplastic left ventricle	Neonatal heart transplant	1
Total anomalous pulmonary venous return (TAPVR)	TAPVR repair	2
PAPVR	PAPVR/ASD repair	1
Inlet-type ventricular septal defect	VSD repair	1
Partial AV canal defect	ASD repair	1

Note. BT, Blalock-Taussig; VSD, ventricular septal defect; ASD, atrial septal defect; AV, atrioventricular; TAPVR, total anomalous pulmonary venous return; PAPVR, partial anomalous pulmonary venous return

diagnosis of CTX in the CTX group (14.8 ± 10.9 days [median, 13 days]; $p = 0.003$). The median number of postoperative days until CTX diagnosis was 11.5 (mean, 13.4 ± 8.2 days).

Table 2 reports the original cardiac anatomy of the CTX group. Over the study period, a total of 650 surgeries performed at UVa were similar to those shown, with an overall CTX incidence of 4.6%. This incidence increased to 5.8% among patients with single ventricle physiology and anatomy. Of the 30 patients with CTX, 8 had effusions from the left pleural space, 3 from the right pleural space, 16 demonstrated bilateral pleural effusions, 1 had peritoneal fluid only, and 2 patients produced chylous fluid from the peritoneal cavity and both pleural spaces.

Univariate, conditional logistic regression analysis demonstrated a 10-fold increased risk of CTX development in patients who required ECMO in the immediate postoperative period, as reported in Table 3. Matched CVPs were significantly higher for both the minimum and the mean values and near-significant for maximum daily values. On average, the CTX group had CVC in place 5 and 6 days longer in their upper and lower extremities, respectively, than the non-CTX group; this difference did not reach statistical significance. It is important to note that the CVC and CVP data in Table 3 are all prior to CTX diagnosis in the CTX group, as compared to the entire hospitalization for the non-CTX group.

Table 4 lists the average values of each pleural fluid lab most commonly used to verify the diagnosis of CTX at our

Table 3 Risk factors for central venous thrombosis

	CTX	Non-CTX	Odds ratio/*p* value
Postoperative ECMO	18 (60%)	5 (8%)	9.9 (95% CI, 2.2–44.8)
Max CVP (median)	15.3 ± 3 (14.9)	14.3 ± 3.5 (13.6)	0.058
Min CVP (median)	8.6 ± 2.6 (8.2)	7.4 ± 2.5 (7.3)	0.011
Average CVP (median)	12.0 ± 2.7 (11.6)	10.8 ± 2.9 (10.4)	0.019
Days of UE CVC (median)	12.2 ± 10.4 (11)	7.1 ± 9.8 (4)	0.067
Days of LE CVC (median)	13.0 ± 14.9 (9.5)	7.1 ± 9.2 (3)	0.062

Note. CTX, chylothorax; ECMO, extracorporeal membrane oxygenation; UE, upper extremity; LE, lower extremity; CVC, central venous catheter. All continuous data are listed as mean ± SD. Medians given in parentheses (except for postoperative ECMO)

Table 4 Diagnostic chylous fluid lab values

CTX diagnostic lab data	Value
Total pleural WBC, per μL	2534 ± 3990 (870)
Pleural WBC lymph, %	80.9 ± 17.6% (85%)
Pleural TAG, mg/dL	413 ± 724 (233)

Note. WBC, white blood cells; TAG, triglyceride. Data are expressed as mean ± SD. Medians given in parentheses. These values were all obtained on the day of chylothorax diagnosis or the day prior to diagnosis

institution. These studies were obtained either on or 1 day prior to the date of CTX diagnosis.

Multivariate conditional logistic regression analysis was performed using the continuous variables of total CVC duration (including both upper and lower extremity locations) and mean CVP prior to CTX diagnosis, and the binary variable of need for ECMO prior to CTX diagnosis, compared with the entire hospitalization for the non-CTX group. This constellation of variables was significantly different between the CTX and the non-CTX groups ($p < 0.0001$). Only elevated mean CVP independently increased CTX risk, with an odds ratio of 1.59 (95% CI, 1.30–2.40).

There were eight documented cases of CVT in the CTX group (26.7%), of which five were supracardiac in location, one was infracardiac in location, and two patients had CVT in both locations; four of these patients had single ventricle physiology and anatomy. With the exception of one CTX patient, whose CVT was diagnosed 14 days prior to CTX diagnosis, the remaining seven CVTs were diagnosed between 4 and 36 days after CTX diagnosis. The non-CTX group had three documented cases of CVT (5.1%), of which one was infracardiac in location and two were supracardiac; all three patients had single ventricle anatomy.

No meaningful patterns were demonstrated with regard to the effect of octreotide, a MCT-only diet, or NPO management on daily volumes of chylous chest tube output. As a result, the effectiveness of these strategies was not amenable to statistical analysis in this study.

Discussion

To our knowledge, this is the largest case-control study to demonstrate an increased incidence of and risk profile for CVT in pediatric patients who developed CTX after surgery for congenital heart disease. Management strategies aimed at minimizing CVT risk and decreasing CVP may help decrease the incidence of CTX in this patient population. However, the retrospective nature of this study precludes definitive statements on causality; prospective studies screening for CVT formation in high-risk patients are needed.

It is important to note that all patients in this study were at increased risk for CVT formation. By matching for anatomy and surgery type, there were no differences in the use of intraoperative CPB or need for CVC access: both are associated with an increased risk of CVT [5, 17, 18, 24–26, 28, 29]. Congenital heart disease alone is associated with an increased risk of genetic coagulopathies [2, 24, 25]. However, those in the CTX group had longer CVC duration, significantly more exposure to ECMO, higher CVP, and a fivefold increased incidence of CVT by radiologic examination. In the period leading up to CTX diagnosis, these case patients already had an average of 5 to 6 more days of CVC exposure compared to the entire hospitalization of the non-CTX group. Though this difference did not reach statistical significance, it may have become clinically significant when coupled with the exposures to both ECMO and higher CVPs.

Our suggestion of a causative link between CVT and CTX development has been supported by several case reports and series [3, 14, 16, 19] as well as a case report documenting resolution of CTX with thrombolysis of an upper extremity thrombosis [22]. While <27% of the CTX group in our study had a documented CVT, this is likely an underrepresentation since routine screening or surveillance is not the standard of care in this patient population. Conversely, seven of eight CTX patients were diagnosed with a CVT after the diagnosis of CTX. While this is suggestive that CTX development plays a causal role in the

development of CVT, the retrospective design of this study allows only for assessment of association, not causality. Prospective research screening for CVT development is necessary to evaluate whether there is a causal relationship and, if present, its direction.

Statistical analysis demonstrated elevated CVP as the only independent risk factor for CTX development. This finding is supported by a case report of resolution of chylous effusions in a neonate treated with nitric oxide [4]. However, the absolute CVP difference between the two groups in this study was minimal and its clinical significance is unclear. It is possible that a marginally elevated CVP was a causative factor in the development of a CVT, or was the result of one. Regardless, we propose that patients needing corrective surgery for tetralogy of Fallot, single ventricle physiology and anatomy, and congenital heart disease complicated by elevated pulmonary vascular resistance are at increased risk for development of CTX due to the postoperative central venous hypertension commonly seen with these lesions. Campbell and colleagues [9] reported a similarly elevated incidence of chylous effusions in this patient population. Elevated CVPs, together with increased need for ECMO and longer CVC exposure, may be surrogates for decreased cardiac output, though this was not addressed in our study. Management strategies aimed at decreasing CVC duration and CVP levels as well as maximizing cardiac output may be warranted in these patients.

Though our study was not adequately powered to address the issue of therapeutic efficacy, the lack of any identifiable change in daily chylous fluid drainage suggests that they are ineffective. Many small case series report resolution of chylous effusions with octreotide [1, 7, 11, 13, 30, 31–33], with only a few questioning its efficacy [20] or safety [23]. Similarly, few have evaluated MCT therapy [12, 15]. As with many treatments, however, there is likely a reporting bias favoring positive results. Our data emphasize the need for prospective, randomized, controlled studies evaluating the efficacy of octreotide, NPO, parenteral nutrition, and low-fat diets for the treatment of patients with CTX.

Study Limitations

A universally accepted set of diagnostic criteria for CTX does not exist. This poses a significant challenge for any retrospective evaluation of this entity. Our pleural effusate labs are in keeping with the guidelines of Buttiker et al. [8], which suggest that the diagnosis of CTX be made in the setting of a pleural TAG count >1.1 mmol/L (100 mg/dL), a pleural WBC count of >1000 cells/μL, and a lymphocyte predominance of >80%. As shown in Table 4, the standard deviations associated with our CTX group are large, which attests to the significant heterogeneity of these patients. Further, these guideline values are not indexed to serum values and do not account for present feeding status, somewhat limiting their utility. Until more stringent guidelines have been established, the diagnosis and appropriateness of therapy for CTX will be subject to question.

Conclusions

We have demonstrated that pediatric patients who develop CTX after surgery for congenital heart disease have a significantly increased incidence of and risk profile for CVT. The combination of this finding with inadequate evidence for the efficacy of the available conservative treatment strategies underscores the need for management strategies aimed at preventing the development of CTX. Prospective research into the association between CVT and CTX development is needed to confirm independent risk factors and inform evidence-based interventions.

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