Thrombosis Research 162 (2018) 15–21

Contents lists available at ScienceDirect

Thrombosis Research

journal homepage: www.elsevier.com/locate/thromres

Full Length Article

Increasing rates of thrombosis in children with congenital heart disease undergoing cardiac surgery

Michael Silvey[a,*], Matt Hall[b], Eryn Bilynsky[a], Shannon L. Carpenter[a]

[a] Department of Pediatrics, Children's Mercy Hospital, Kansas City, MO, United States
[b] Children's Hospital Association, Lenexa, KS, United States

ARTICLE INFO

Keywords:
Congenital heart disease
Thrombosis
Mortality

ABSTRACT

Objective: To determine thrombosis incidence, morbidities, and mortality of children with congenital heart disease who develop thrombosis after cardiac surgery.
Materials and methods: This retrospective study reviewed patients < 18 years old within the Pediatric Health Information System (PHIS) database who underwent cardiac surgery from 2004–2012. Thrombosis rates were compared for each procedure. Mortality was modeled using proportional hazards, adjusting for important clinical and demographic factors.
Results: Of 91909 CHD patients who underwent surgery, 2655 (2.9%) developed thrombosis within the ensuing 12 months. The rate of thrombosis increased 253% ($p < 0.001$), from 1.7% in 2004 to 4.4% in 2012. Systemic to pulmonary shunt placement (34.3%) and septostomy (26.1%) had the highest thrombosis percentages. Children < 28 days had the highest prevalence (61%). Those with thrombosis had longer lengths of stay than those without [median 27 hospital days and 10 ICU days vs. 6 and 2 ($p < 0.001$)]. Mean risk-adjusted cost was higher with thrombosis; $126,257 vs. $40,773 ($p < 0.001$). Thrombosis was also associated with higher rates of bacteremia [8.3% vs. 3.4%, $p < 0.001$], endocarditis [0.7% vs. 0.2%, $p < 0.001$], and mortality [12.3% vs. 0.8%, $p < 0.001$]. The adjusted hazard ratio for mortality in patients with thrombosis was 5.5 (95% CI: 4.6–6.5).
Conclusions: Thrombosis rates in CHD patients after cardiac surgery is increasing. Thrombosis is associated with longer hospital stay, increased ICU days, and cost. CHD patients with thrombosis also have increased bacteremia and mortality rates. More research is needed to understand contributors to thrombosis which may help develop strategies to mitigate morbidity and mortality.

1. Introduction

Congenital heart disease (CHD) affects approximately 1% of all live births. The majority of these conditions occur in healthy children, and once these cardiac lesions are corrected, the patients can live a normal lifespan. However, CHD has a wide range of severity and some lesions may need several surgical procedures to achieve a complete repair [1]. Some lesions are not amenable to complete repair and therefore surgical approaches are focused on palliation. Due to these procedures, children with CHD are at risk for developing significant morbidity and mortality.

Venous thromboembolism (VTE) is an increasing complication in hospitalized pediatric patients. A previous study demonstrated that from 2001 to 2007 the incidence of VTE for pediatric patients increased by 70% [2]. Patients with CHD are at high risk of developing thrombosis due to the disruption of blood flow, inflammation, and platelet activation secondary to surgical procedures which can initiate thrombus formation. Also, many of these procedures are dependent on the formation of shunts from foreign material, which can increase the risk of developing a thrombus [3].

The incidence of thrombosis in patients with CHD reported in the literature ranges widely from 1 to 31% [4–8]. These are mostly single institution studies examining patients over several years. The objective of our study was to determine the incidence of thrombosis after cardiac surgery for congenital heart disease at children's hospitals and also examine the morbidities and mortality in this population.

Abbreviations: CHD, Congenital heart disease; PHIS, pediatric health information system; ICU, Intensive care unit; VTE, Venous thromboembolism; ICD-9, International Classification of Diseases 9th revision; RACHS, Risk Adjustment in Congenital Heart Surgery; rFVIIa, Recombinant factor VIIa; CPB, cardiopulmonary bypass

* Corresponding author at: Children's Mercy Hospital, Division of Hematology/Oncology, 2401 Gillham Rd, Kansas City, MO 64108, United States.
E-mail address: msilvey@cmh.edu (M. Silvey).

https://doi.org/10.1016/j.thromres.2017.12.009
Received 2 October 2017; Received in revised form 7 December 2017; Accepted 11 December 2017
Available online 13 December 2017

Table 1
Patient Characteristics.

	All CHD patients who had corrective repair	CHD patients who did not develop thrombosis after corrective repair	CHD patients who developed thrombosis after corrective repair	P-value
N	91,909	89,254 (97.1)	2655 (2.9)	< 0.001
Sex				
Male	49,775 (54.2)	48,245 (54.1)	1530 (57.6)	< 0.001
Female	42,127 (45.8)	41,002(45.9)	1125 (42.4)	
Race				
Non-Hispanic White	47,777(52)	46,387 (52)	1390 (52.4)	0.064
Non-Hispanic Black	11,025 (12)	10,709 (12)	316 (11.9)	
Hispanic	16,603 (18.1)	16,088 (18)	515 (19.4)	
Asian	2639 (2.9)	2558 (2.9)	81 (2.1)	
Other	13,865 (15.1)	13,512 (15.1)	353 (13.3)	
Payor				
Government	41,565 (45.2)	40,194 (45)	1371 (51.6)	< 0.001
Private	37,134 (40.4)	36,176 (40.5)	958 (36.1)	
Other	43,372 (47.2)	42,448 (47.6)	924 (34.8)	
Age				
< 28 days	25,901 (28.2)	24,257 (27.2)	1644 (61.9)	< 0.001
29 days– < 1 yr.	28,807 (31.3)	28,268 (31.6)	569 (21.4)	
1 yr– < 6 yrs.	20,287 (22.1)	20,046 (22.5)	241 (9.1)	
6 yrs.– < 13 yrs.	10,311 (11.2)	10,195 (11.4)	116 (4.4)	
13 yrs.–18 yrs.	6603 (7.2)	6518 (7.3)	85 (3.2)	
RACHS				
Missing	16,013(.)	15,559 (.)	454(.)	< 0.001
0	8559 (11.3)	2567 (11.2)	292 (13.3)	
1	8538 (11.2)	8489 (11.5)	49 (2.2)	
2	24,918 (32.8)	24,626 (33.4)	292 (13.3)	
3	22,731 (30)	21,962 (29.8)	769 (34.9)	
4	8700 (11.5)	8323 (11.3)	977 (17.1)	
5	109 (0.1)	98 (0.1)	11 (0.5)	
6	2341 (3.1)	1930 (2.6)	411 (18.7)	

P-value from chi-square test comparing the distribution of each characteristic between CHD patients who developed thrombosis and those that did not.

2. Materials and methods

2.1. IRB approval

This study was reviewed and approved by the Children's Mercy Hospital Institutional Review Board.

2.2. Data source

Data was obtained from the Pediatric Health Information System (PHIS) database which contains resource utilization data from 48 tertiary care children's hospitals. The participating hospitals are located in 27 states and the District of Columbia. These hospitals provide inpatient and observation data, including patient demographics, up to 41 *International Classification of Diseases, 9th revision* (ICD-9) diagnoses, and up to 41 ICD-9 procedures as well as encrypted unique identifiers to track individual patients across admissions. Daily billing data is also available for every encounter. The PHIS database is maintained by the Children's Hospital Association (CHA; Lenexa, KS). Data quality and reliability are assured through CHA and participating hospitals. 43 hospitals that consistently provided data into PHIS throughout the study period were included in this study.

2.3. Subjects

CHD patients were included if they were admitted to a PHIS hospital from January 2004 to December 2012. Children with CHD were identified using ICD-9 discharge codes. Patients were then identified from this set that had cardiac surgery. Patients were tracked during the initial surgery admission and for one year post discharge from their initial cardiac surgery for a thrombosis diagnosis, making the date of the last data collection December 2013. Diagnostic and procedures codes are listed in Appendix 1.

2.4. Patient demographics

Age at admission, age at the time of surgical procedure, race, and gender were recorded for all patients. Ages were classified as the following: birth to 28 days, 29 days to < 1 year, 1 year to < 6 years, 6 years to < 13 years, and 13 years to 18 years. The number of ICU days, length of stay, adjusted cost, and disposition were also recorded. Patients were categorized by the Risk Adjustment in Congenital Heart Surgery (RACHS) classification system [9]. In PHIS, costs are estimated from charges based on hospital and year specific cost to charge ratios and adjusted for regional variation using the wage index from the Center for Medicare & Medicaid Services (Centers for Medicare & Medicaid Services Wage Index website https://www.cms.gov/Medicare/Medicare-Fee-for-Service-Payment/AcuteInpatientPPS/wageindex.html).

2.5. Infectious complications and factor VIIa exposure

We examined the bacteremia and endocarditis rates for CHD patients who did and did not develop thrombosis utilizing ICD-9 codes for these diagnoses (Appendix 1). Recombinant factor VIIa (rFVIIa) usage for CHD patients was determined using pharmacy billing data. These specific variables were chosen as it is known both serious infections and rFVIIa use can increase the risk of thrombosis [10,11].

2.6. Statistical analysis

Demographic characteristics were summarized with frequencies and percentages for categorical variables and with medians and interquartile ranges for continuous variables. These were compared between those with thrombosis and those without using Wilcoxon-rank sum or chi-squared analysis as appropriate. By year, thrombosis rates were computed in aggregate and separately for each surgical procedure and presented with exact binomial 95% confidence intervals. Trends were

assessed with the Cochran-Armitage test for trend. We modeled the likelihood of developing thrombosis using generalized estimating equations with factors significant at $p < 0.10$ in bivariate analyses, and accounting for patient clustering within hospital. Mortality was modeled using proportional hazards and adjusted for important clinical and demographic factors including gender, race, payer, disposition, length of stay, use of anticoagulation and antibiotic drugs, imaging, and cardiac surgical group (Appendix 1). All modeling accounted for patient clustering within hospital. Statistical analyses were performed using SAS v. 9.4 (SAS Institute, Cary, NC), and p-values < 0.05 were considered statistically significant.

3. Results

During the study period, there were a total of 91,909 CHD patients identified who underwent corrective surgical repair (Table 1). Among these patients, 2655 (2.9%) developed thrombosis within 12 months following their operation. Rates of arterial and venous thrombosis were similar (1.7% vs. 1.5%, respectively). Patients who developed graft thrombosis had a thrombosis rate of 1.6%. Only one patient developed a mural thrombus during the study period. The thrombosis incidence over the study period (2004–2013) increased 253% from 1.7% to 4.4% ($p < 0.001$) (Fig. 1). Venous thrombosis incidence increased by 400% from 0.6% to 2.5% ($p < 0.001$) and the incidence of arterial thrombosis increased by 203% from 1.2% to 2.5% ($p < 0.001$). Graft thrombosis decreased 33% from 2% to 1.5% ($p < 0.001$).

The systemic to pulmonary shunt procedure (34.3%) and septostomy (26.1%) were associated with the highest thrombosis percentages (Table 2). The patients who had RACHS-3 surgeries had the highest percentage of thrombosis (34.9%) followed by RACHS-6 (18.7%). Patients < 28 days old were more likely to have thrombosis (61.9%) followed by infants 29 days to one year of age (21.4%).

CHD patients with thrombosis experienced longer ICU stays, more hospital days, and higher adjusted costs during their initial cardiac surgical admission. CHD patients who developed thrombosis had a median of 10 ICU days [Interquartile range (IQR) 2, 25] vs. 2 days [IQR 1, 6] for those who did not develop thrombosis ($p < 0.001$). The median length of hospital stay was 27 days [IQR 13, 56] for those who developed thrombosis vs. 6 days [IQR 3, 16] for those who did not develop thrombosis ($p < 0.001$). Adjusted cost for CHD patients with thrombosis was 3-fold higher at $126,257 [IQR $86,995, $328,440] vs. $40,773 [IQR $25,355, $84,193] for patient who did not develop thrombosis ($p < 0.001$).

We evaluated the use of recombinant factor VIIa (rFVIIa) and the incidence of thrombosis. Recombinant factor VIIa was used in 500 patients, and the thrombosis rate among these children was 30% (152/500) vs. 2.8% (2503/88906) for those who did not receive rFVIIa ($p < 0.001$). The use of rFVIIa also significantly increased during the study period ($p < 0.001$). Recombinant factor VIIa use was highest in 2010 with 0.8% of CHD patients being exposed.

The overall rate of bacteremia or endocarditis for CHD patients was 1.3%. The bacteremia rate for CHD patients who developed thrombosis was 8.3% vs. 0.7% for those who did not ($p < 0.001$). The endocarditis rate for CHD patients with thrombosis was also higher compared to the patients who did not develop thrombosis (3.4% vs. 0.2%, $p < 0.001$).

In multivariable analyses, factors which statistically increased the likelihood of thrombosis in our patient population were age < 28 days (OR 1.68, CI 1.15–2.45, $p = 0.0073$), discharge to a long term care facility (OR 1.76, CI 1.07–2.91, $p \leq 0.001$), length of stay > 15 days (OR 10.6, CI 6.94–16.2, $p \leq 0.001$), length of stay 8–14 days (OR 4.42, CI 2.86–6.84, $p \leq 0.001$), length of stay 4–7 days (OR 2.25, CI 1.51–3.36, $p \leq 0.001$), RACHS-4 classification (OR 1.26, CI 1.05–1.51, $p = 0.0139$), bacteremia (OR 4.26, CI 3.32–5.45, $p \leq 0.001$), endocarditis (OR 6.15, CI 3.99–9.46, $p \leq 0.001$) and factor VIIa exposure (OR 5.77, CI 4.26–7.82, $p < 0.001$).

The overall in-hospital mortality rate for patients with CHD who underwent surgical repair was 1.2% (Fig. 2). Mortality was significantly higher in patients who developed thrombosis vs. those who did not (12.3% vs. 0.8%, $p < 0.001$). This was consistent across all RACHS categories ($p < 0.001$). Mortality in the RACHS categories for patients who developed thrombosis ranged from 8.2%–18.2% with the RACHS-5 group having the highest mortality percentage (18.2%). In multivariable proportional hazards models, the adjusted hazard ratio for mortality for thrombosis was 5.5 (95% confidence interval: 4.6–6.5, $p < 0.001$).

The thrombosis group was then divided between arterial versus venous thrombosis to determine whether there were differences in ICU days, length of hospital stay, hospital costs, infectious complications, and mortality. When examining all of these factors, there were no significant differences between patients who developed an arterial vs. venous thrombus.

4. Discussion

To date, these are the most comprehensive data regarding the incidence, morbidities, and mortality of CHD patients who develop

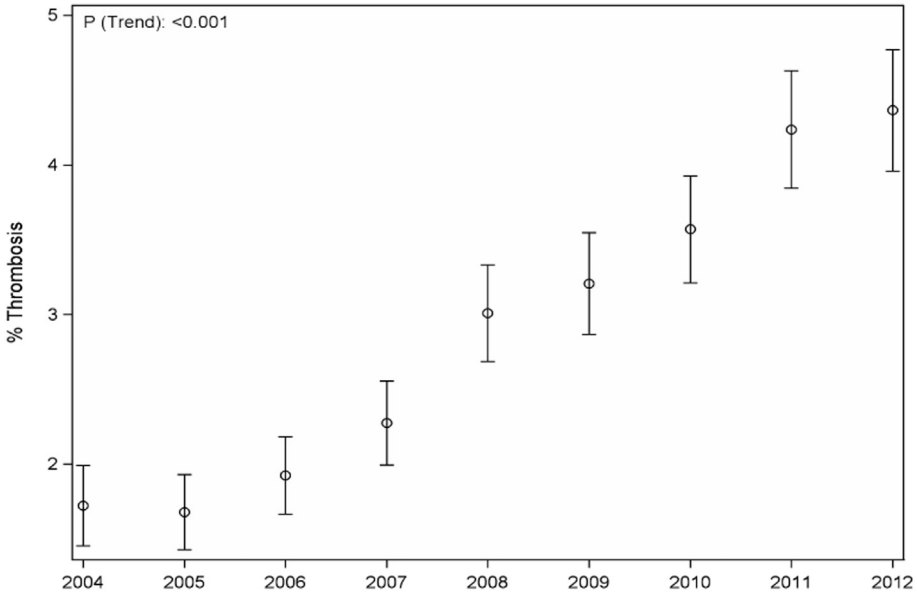

Fig. 1. Thrombosis incidence in congenital heart disease (CHD) patients after cardiac surgery from 2004 to 2012.

Table 2
Cardiac surgery types and thrombosis.

	All CHD patients who had cardiac surgery	CHD patients who did not develop thrombosis after cardiac surgery	CHD patients who developed thrombosis after cardiac surgery	*p*-value
Missing	28,418 (.)	27,605 (.)	813 (.)	< 0.001
ASD/VSD Repair (RACHS-2)	37,178 (58.6)	36,770 (59.6)	408 (22.1)	
Fontan Procedure (RACHS-3)	2234 (3.5)	2185 (3.5)	49 (2.7)	
TAPVR Repair (RACHS-4)	1455 (2.3)	1403 (2.3)	52 (2.8)	
Tetralogy of Fallot Repair (RACHS-3)	3944 (6.2)	3881 (6.3)	63 (3.4)	
Transposition of Great Arteries Repair (RACHS-4)	494 (0.8)	485 (0.8)	9 (0.5)	
Truncus Arteriosus Repair (RACHS-4)	499 (0.8)	454 (0.7)	45 (2.4)	
Septostomy Procedure (RACHS-4)	5699 (9)	5218 (8.5)	481 (26.1)	
Systemic to Pulmonary Shunt Placement (RACHS-3)	4759 (7.5)	4128 (6.7)	631 (34.3)	
Valve Repair/Replacement (RACHS-2)	7229 (11.4)	7125 (11.6)	104 (5.6)	

p-value from chi-square test comparing the distribution of each characteristic between CHD patients who developed thrombosis and those that did not.

thrombosis. Our study is also the first to utilize data from multiple institutions.

The overall incidence of thrombosis in our study was 2.9%, which is on the lower end of what has been reported in previous studies. This result was expected as our study looked at every type of cardiac surgery. Two studies which reported high thrombosis incidence were done by Tzanetos, et al. (31%) and Cholette, et al. (22.7%). Both of these studies had very few study subjects (19 and 22 patients respectively) and both only examined patients with single ventricle physiology [5,6]. Coon, et al. examined thrombosis in patients after the Fontan procedure and found an incidence of 8.8%. Though this incidence is lower than the previous studies mentioned in single ventricle physiology patients, this study examined only intracardiac thrombosis [12]. Three studies examined thrombosis in all CHD patients who had cardiac surgery and their reported incidence is much lower than the single ventricle physiology patient studies. Hanson, et al. (3.8%) and Petaja, et al. (1.1%) reported low thrombosis incidence while Manlhiot, et al. reported a thrombosis an incidence of 11% [4,7,13]. One possible explanation of the higher thrombosis incidence in the Manlhiot study compared to the Hanson and Petaja studies is Manlhiot captured both arterial and venous thrombosis while Hanson and Petaja only focused on venous thrombosis.

The 253% increase in thrombosis in the CHD population is much higher than the 70% increase in VTE rate reported in general pediatric hospitalized patients from 2001 to 2007 [2]. When separating thrombosis in our study to arterial and venous thrombosis, the increase in VTE incidence is even higher at 400%. Several factors could explain this higher rate of increase: increased central venous access compared to the general hospitalized pediatric population, increased awareness of thrombosis as a morbidity in CHD patients, as well as sicker patients surviving longer with improved surgical techniques and critical care. Though these reasons could account for a portion of the increased rate, they are unlikely to account for the entire thrombosis rate increase.

Long-term complications of VTE are numerous and include post-thrombotic syndrome, recurrent VTE, and increased risks of bleeding due to prolonged exposure to anticoagulation. Another complication which may be particularly important for CHD patients is loss of venous access for cardiac catheterizations [14,15]. Though there is less significant morbidity with arterial thrombosis, Rizzi, et al. reported significant blood pressure discrepancies and reduced ankle/ankle index in patients with persistent arterial occlusion of the femoral artery. It was also found these patients did also have a significant leg length difference, though the absolute difference was < 10 mm in all children except for 2 [16].

When analyzing the incidence of thrombosis by surgery, we found that patients who had the pulmonary to systemic shunt and septostomy procedures had the highest percentage of thrombosis and also the highest increase in thrombosis rate. We also found that patients younger than 28 days of age had the highest thrombosis prevalence followed by infants 29 days to 1 year of age. These results are concordant with most of the previous studies that have shown the highest incidence of thrombosis is in patients with single ventricle physiology

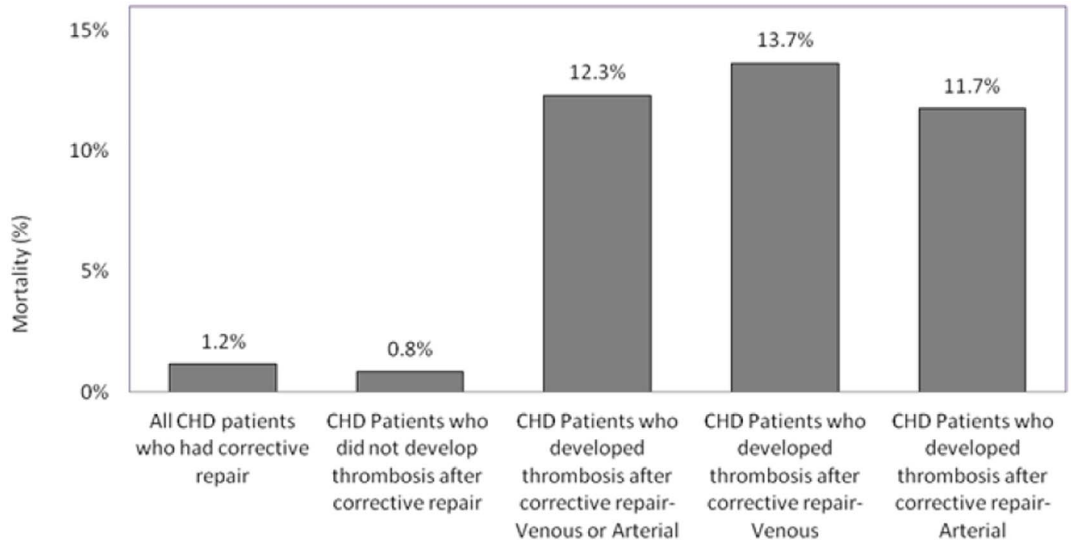

Fig. 2. Mortality in congenital heart disease (CHD) patients who did/did not develop thrombosis after cardiac surgery.

during and after the first palliation procedure [4–7].

It has been shown previously that CHD patients who develop VTE have an increased number of ICU days and length of hospitalization [4,5,12]. Manlhiot, et al. showed ICU stay increased from 3 to 11 days and the hospital stay increased from 7 to 25 days [4] with the presence of VTE. Emani, et al. also found neonates who developed thrombosis had an increased number of ICU days and a longer hospital stay [8]. These data corresponds to what we found in our study. In our study, we also found the adjusted cost of hospitalization was significantly increased which corresponds with the increased length of stay.

Since most CHD patients require cardiopulmonary bypass (CPB) during their corrective operations, high dose anticoagulation is used intra-operatively. Neonates who undergo surgical repair and are placed on CPB are at increased risk of bleeding due to the high doses of anticoagulation and their immature coagulation system [17]. One option used to control the amount of hemorrhage and decrease blood product transfusion is administration of recombinant factor VIIa. There have been data suggesting that recombinant factor VIIa is helpful in preventing blood product transfusion and has minimal side effects [18–20]. Over the period of this study, we observed an increased use of rFVIIa in CHD patients after surgical repair. The increased use was associated with an increased thrombosis rate in these patients as well.

To help stratify risks for mortality in patients with CHD who undergo surgical repair, an expert panel reviewed data from the Pediatric Cardiac Care Consortium and created the risk adjustment for congenital heart disease system (RACHS) [9]. Using this system, corrective surgical procedures are categorized for adjusted risk of operative mortality. Patients undergoing a higher RACHS classification procedure have increased adjusted risk for operative mortality. This risk adjustment allows for meaningful comparisons for mortality for CHD patients who undergo cardiac surgery. In our study we evaluated the thrombosis rate of each risk category to determine if thrombosis rates were higher in higher risk categories. Patients who underwent surgical procedures which fall in the RACHS-3 category had the highest number of thrombosis. This finding is mostly explained by the fact RACHS-3 surgeries were the most commonly performed in the entire cohort.

Logistic regression analysis did identify the following factors which increased the thrombosis risk in our study: age $<$ 28 days, length of stay, discharge to a long term care facility, RACHS-4 classification, bacteremia, endocarditis, and factor VIIa exposure. It was not surprising to note the increased thrombosis risk in the patients in the $<$ 28 days of age group due to previously published work showing increased VTE prevalence in that age group [2]. Though discharge to a long term care facility as well as length of stay did show increased risk, these cannot be used for risk assessment/prediction of thrombosis as these are unknown variables until the patient is actually discharged.

There are various reports regarding the mortality rate of CHD patients who develop thrombosis after their corrective repair. The findings of our study showing a significant mortality for CHD patients who develop thrombosis vs. CHD patients who did not develop thrombosis after cardiac surgery (12.3% vs. 0.8%) correlate to previously published studies. Petaja, et al. found that in children after cardiac procedures the mortality rate for those with thrombosis was increased to 40% vs. those patients who did not develop thrombosis (8.3%) [7]. Manlhiot, et al. also showed an increased mortality rate of 5.3% for CHD patients with thrombosis vs. 0.6% for those who did not develop thrombosis [4].

The primary limitation of our study is that we have relied on administrative data. As this database is ICD-9 code based, there is the possibility that some patients had the improper diagnosis code assigned. Also, patients who have CHD and undergo corrective repair typically have many ICD-9 codes assigned. As there is a limit of 41 ICD-9 discharge codes, some patients might not have been classified as having a thrombosis when they in fact had one. This would have led us to underestimate the rate of thrombosis. The PHIS database also only collects inpatient and emergency room data, so we were unable to collect any data from the clinic setting regarding thrombosis. Since we can expect that most CHD patients with thrombosis would have been hospitalized, this limitation likely had little impact on our results. By using this administrative data we also were unable to determine whether thrombosis were related to venous or arterial line placement. Also, our study was done retrospectively without any access to medical records, so we are unable to correlate infection rates, hospital stay length, and rFVIIa usage and thrombosis rate.

Future strategies should be explored on how to identify and treat patients who are at risk for developing thrombosis. CHD patients are at risk for developing thrombosis due to several disruptions in their hemostatic system. It has been shown CHD patients have been found to have decreased levels of several pro-coagulation proteins as well as naturally occurring anticoagulants [21]. Factor VIII has also been shown to be increased in CHD patients after the Fontan Procedure [22]. Identifying risk factors can be challenging, as patients who undergo cardiopulmonary bypass also can have profound imbalances between the pro- and antithrombotic pathways [23]. Examining coagulation proteins, thrombophilic gene mutations, and other acquired risk factors may be beneficial as they have been found to possibly play a role in the formation of thrombosis [24,25]. Prevention strategies should also be explored in this population. Aspirin is commonly used in CHD patients after surgery to help prevent thrombosis [26]. Alternate pharmacologic therapies also should be examined as patients who have been shown to have aspirin resistance had an increased rate of thrombosis after their corrective surgery [27–29].

5. Conclusion

The thrombosis rate for congenital heart disease patients who undergo corrective surgical repair has increased over the past 10 years. This rate of increase is considerably higher than published rates regarding the general hospitalized pediatric population [2]. The surgeries which were associated with the highest percentage of thrombosis were the septostomy procedure and the systemic to pulmonary shunt procedure. Thrombosis development was associated more ICU days, longer hospital length of stay, higher hospital costs and mortality. Risk factors identified for increased thrombosis risk were age $<$ 28 days, length of stay, discharge to a long term care facility, RACHS-4 classification, bacteremia, endocarditis, and factor VIIa exposure. Further prospective studies are needed to validate these risk factors and examine other possible risk factors associated with thrombosis formation in the CHD population and to determine optimal treatments to address this complication.

Financial disclosure

The authors have no financial relationships relevant to this article to disclose.

Funding source

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Potential conflicts of interest

The authors have no conflicts of interest relevant to this article to disclose.

Appendix 1

ICD − 9 codes used for patient Identification

Congenital Heart Disease Codes:

a. 745.0- Common Truncus
b. 745.0- Common Truncus

c. 745.1x- Transposition of the Great Vessels
d. 745.2- Tetralogy of Fallot
e. 745.3 Common Ventricle
f. 745.4- Ventricular Septal Defect
g. 745.5- Ostium Secundum type Atrial Septal Defect
h. 745.6x- Endocardial Cushion Defects
i. 745.7- Cor Biloculare
j. 745.8- Other
k. 745.9- Unspecified Defect of Septal Closure
l. 746.0x- Anomalies of the Pulmonary Valve
m. 746.1- Tricuspid Atresia
n. 746.2- Ebstein's Anomaly
o. 746.3- Congenital Stenosis of the Aortic Valve
p. 746.4- Congenital Insufficiency of Aortic Valve
q. 746.5- Congenital Mitral Stenosis
r. 746.6- Congenital Mitral Insufficiency
s. 746.7- Hypoplastic Left Heart Syndrome
t. 746.8x- Other specified anomalies of Heart
u. 747.1x- Coarctation of the Aorta
v. 747.2x- Other Anomalies of Aorta
w. 747.3x- Anomalies of Pulmonary Artery
x. 747.4x- Anomalies of Great Veins

Surgical Corrective Procedures

a. 35.0x- Closed Heart Valvotomy or Transcatheter Replacement of heart valve
b. 35.1x- Open heart valvuloplasty without replacement
c. 35.2x- Open and other replacement of heart valve
d. 35.3x- Operations on structures adjacent to heart valves
e. 35.4x- Production of septal defect in heart
f. 35.5x- Repair of atrial and ventricular septa with prosthesis
g. 35.6x- Repair of atrial and ventricular septa with tissue graft
h. 35.7x- Other and unspecified repair of antrial and ventricular septa
i. 35.8x- Total repair of certain congenital cardiac anomalies
j. 35.9x- Other operations on valves and septa of heart
k. 36.9x- Other operations on vessels of heart
l. 37.3x- Pericardiectomy and excision of lesion of heart
m. 37.5x- Heart Replacement Procedures
n. 38.3x- Resection of vessel with anastomosis
o. 38.4x- Resection of vessel with replacement
p. 38.6x- Other excision of vessel
q. 38.8x- Other surgical occlusion of vessels
r. 39.0x- Systemic to pulmonary shunt
s. 39.2x- Other shunt or vascular bypass
t. 39.4x- Revision of vascular procedure
u. 39.5x- Other repair of vessels
v. 39.6x- Extracorporeal Circulation and procedures auxiliary to heart surgery

Venous Thrombosis Codes:

a. 325.0- Phlebitis and thrombophlebitis of intracranial venous sinuses
b. 415.1x- Pulmonary embolism and infarction
c. 437.6- Nonpyogenic thrombosis of intracranial venous sinus
d. 453.0- Budd-Chiari syndrome/Hepatic vein thrombosis
e. 453.1- Thrombophlebitis migrans
f. 453.2- Venous embolism and thrombosis of inferior vena cava
g. 453.3- Venous embolism and thrombosis of renal vein
h. 453.4x- Acute venous embolism and thrombosis of deep vessels of lower extremity
i. 453.5x- Chronic venous embolism and thrombosis of deep vessels of lower extremity
j. 453.8x- Acute venous embolism and thrombosis of other specified veins
k. 453.9- Venous embolism and thrombosis of unspecified site
l. 557.0- Vascular insufficiency of intestine
m. 452.0- Portal vein thrombosis
n. 593.81- Vascular Disorders of the Kidney
o. 671.5- other phlebitis and thrombosis/cerebral venous thrombosis/ Thrombosis of intracranial venous sinus
p. 996.72- embolism of graft
q. V12.51- Venous thrombosis and embolism
r. V12.55- Pulmonary embolism

Arterial Thrombosis Codes:

a. 433.x- Occlusion and stenosis of precerebral arteries
b. 434.x- Cerebral thrombosis
c. 444.0- Arterial embolism and thrombosis of abdominal aorta
d. 444.1x- Arterial embolism and thrombosis of thoracic aorta
e. 444.2x- Arterial embolism and thrombosis of arteries of the extremities
f. 444.8x- Arterial embolism and thrombosis of other specified artery
g. 444.9x- Arterial embolism and thrombosis of unspecified artery

Other Thrombosis Codes:

a. 429.79- Mural thrombus

Infectious Disease Codes:

a. 421.x- Acute and Subacute Endocarditis
b. 790.7- bacteremia

Medication Codes:

a. 161327—enoxaparin
b. 161307—heparin
c. 161345—antithrombin
d. 112158—aspirin
e. 161361—warfarin
f. 161511—alteplase

Imaging Codes:
Ultrasound Codes:

1. Lower Extremity codes:
   a. 425,041 425,042 464,245 464,246
2. Upper Extremity codes
   a. 428,141 428,142 464,145 464,146
3. Abdominal codes
   a. 441,045 441,145 441,245 464,445
4. Neck Codes
   a. 464,345 464,346

CT Scan Codes

1. Chest codes
   a. 433,012 433,051 433,951 473,018 473,051 473,052
2. Head Codes
   a. 471,051 480,051 481,100

MRI Codes

1. Brain/arteriogram/venogram
   a. 471,052 481,052

Echocardiogram

1. 463,040 463,041 463,042 463,043 463,046

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