Published in final edited form as:

Pediatr Blood Cancer. 2009 January ; 52(1): 75–79. doi:10.1002/pbc.21714.

Risk Factors for Umbilical Venous Catheter-Associated Thrombosis in Very Low Birth Weight Infants

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Abstract

Background—Thrombosis in neonates is a rare but serious occurrence, usually associated with central catheterization. The objective of this study was to investigate the risk factors associated with catheter related thrombosis in very low birth weight (VLBW) infants.

Procedure—The present retrospective study was performed using data from a randomized trial of duration of umbilical venous catheters (UVC) placement among infants <1250 g birth weight. Twenty-two cases of UVC-associated thrombosis were identified in this sample. The remaining study sample (n=188) served as the comparison group. Data on thrombosis, platelets, gestational age, birth weight, hematocrit, serum sodium, maternal preeclampsia, blood group, infant of diabetic mother and demographic factors were collected using database and record review.

Results—Among the total subjects (n=210), 112 (53%) were males and 126 (60%) were Caucasians, with mean gestational age of 27.7 ± 2.1 weeks (standard deviation) and mean birth weight of 923 ± 195 grams. Bivariate analysis revealed significant association of thrombosis with hematocrit >55% in the first week (odds ratio [OR] 5.4; 95% confidence interval [CI] 2.0-14.6; p=0.0003), being small for gestational age (OR, 2.9; 95% CI, 1.2-7.4; p=0.02) and maternal preeclampsia (OR, 3.97; 95% CI, 1.6-9.84; p=0.0017). In multivariate logistic regression analysis, only hematocrit >55% was independently associated with thrombus (OR, 3.7; 95% CI 1.1-11.8; p=0.03).

Conclusions—This study demonstrates a significant, independent association between elevated hematocrit and development of UVC-associated thrombosis. Careful monitoring for catheter-associated thrombosis may be indicated in VLBW infants who have hematocrit >55% in the first week of life.

Keywords

neonate; thrombosis; risk factors; umbilical venous catheters

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Introduction

Thrombosis in neonates is rare but associated with high morbidity¹. The overall incidence of symptomatic neonatal thromboembolism as high as 2.4 per 1,000 neonatal intensive care unit admissions was reported in a Canadian registry report¹. The venous and arterial systems are equally involved^{1, 2}. Ninety percent of neonatal thrombi are associated with central catheterization³. In addition to the newborn's natural decreased ability to inhibit thrombin and relatively deficient fibrinolysis, several additional factors may place some neonates at an increased risk of thrombosis^{4- 5}. Among the congenital risk factors are deficiency of natural anticoagulants (AT III, Proteins C and S), elevated lipoprotein a levels, Factor V Leiden, prothrombin gene mutation and hyperhomocysteinemia⁵⁻¹⁰. Acquired risk factors include indwelling catheters, dehydration, polycythemia, asphyxia, septicemia, non-O blood group type, maternal diabetes and extracorporeal membranous oxygenation^{1, 5, 11-20}. We and others have reported that being small for gestational age (SGA) is associated with an increased risk of catheter related thrombosis in neonates¹¹. In addition to the aforementioned potential acquired risk factors, thrombocytosis has been shown to be associated with increased risk of thrombosis in other settings²¹. Most of the data regarding neonatal thrombosis are based on case reports, case series or registries^{1, 22}. There is a paucity of investigational studies to study the risk factors for neonatal thrombosis.

We assessed the risk factors for umbilical venous catheter (UVC) associated thrombosis among subjects from a randomized controlled trial of very low birth weight (VLBW) infants, in which infants were regularly evaluated by echocardiography for the presence of thrombosis. We hypothesized that VLBW neonates with UVC-associated thrombosis would have higher platelet counts in the first week after birth than the neonates without thrombosis and that neonates with umbilical catheter-related thrombosis would be more likely to be small for gestational age (SGA) compared to neonates without thrombosis.

Methods

Study Design

We conducted a retrospective analysis using data gathered from infants who participated in a randomized controlled trial conducted at University of Rochester's Level III neonatal intensive care unit (NICU) between 1998 and 2004¹¹. The University of Rochester Institutional Review Board approved the study, and parents or legal guardians of the patients gave informed consent prior to enrollment. Very low birth weight infants were studied to evaluate whether long-term UVC use (up to 28 days) resulted in fewer infections than short-term UVC use (7-10 days) followed by peripherally inserted central catheter (PICC) placement. Eligibility criteria included birth weight ≤ 1250 grams and UVC placement upon NICU admission. Neonates requiring UVC for exchange transfusion, infants with GI anomalies or congenital heart disease with intra-cardiac shunting were excluded. Detailed methods and results of the trial have been described previously¹¹.

Echocardiography for the short-term group was performed upon study entry, before removal of the UVC, at placement of the PICC, at 7 and 21 days after PICC placement, and 1-2 weeks after the PICC was discontinued. For the long-term group, echocardiography was performed upon study entry, at days 7, 14 and 28 of age, before UVC removal and 1-2 weeks after removal of the UVC. Twenty-four (11%) of the 210 infants in the primary trial were found to have thrombi, and 22 of these thrombi were associated with the UVC. We analyzed data from the 22 infants with UVC-associated thrombi, compared to the rest of the population (n=188). The tips of PICC were poorly visualized by echocardiography, precluding systematic analysis of PICC-related thrombosis¹¹. The two infants with thrombi detected at the PICC tip were thus included in "no thrombus" group.

Data Collection

The primary study database and electronic and paper medical records were reviewed for factors associated with thrombophilia in neonates¹¹. 5th, 18th, 20th, 21st. The data for highest platelets in the first week of life, SGA status, septicemia (defined as signs and symptoms plus either a single positive blood culture for definite pathogens or multiple cultures [≥ 2 within 48 hours] for possible pathogens), highest serum sodium >150 mmol/L in the first week of life to assess for dehydration, highest hematocrit $> 55\%$ in the first week to assess for polycythemia, infant of diabetic mother (IDM) status, maternal preeclampsia and blood group types was collected. A hematocrit of 55% was chosen because it is at approximately the 95th percentile and 2 standard deviations above the mean for a 26-29 week gestation newborn but represents the median for hematocrit in a full term infant in the first week of life²³. Data about race, ethnicity, gender and gestational age were also collected. As previously described, thrombus onset was defined either as the date when thrombus was first detected on echocardiogram or the date of UVC removal, whichever occurred earlier¹¹. Thrombi were grouped by size as small (≤ 5 mm longest dimension), moderate (5-10 mm) or large (>10 mm). Only the first detected thrombus was evaluated for each subject.

Statistical Analysis

Student t-tests were performed to evaluate association between thrombus formation and maximum platelet count in the first week, birth weight and gestational age. Odds ratios and χ^2 statistics were calculated to quantify associations between thrombus formation and each of hematocrit, sodium, SGA, IDM, non-type O blood group, maternal preeclampsia, infection of the line, race and gender. A multivariate logistic regression analysis was performed using the significant risk factors from the bivariate analysis. $P < 0.05$ was considered to indicate statistical significance. SAS version 9.1 (SAS Institute, Cary, NC) was used for data analysis.

Results

Thrombi Characteristics

All thrombi were sessile or “sheath like” (Figure 1)¹¹. Sixteen were small (≤ 5 mm) and 6 were moderate sized (5-10 mm). None of the thrombi was clinically suspected to be present by the patient care team. Eleven thrombi were judged to be clinically significant by the cardiologist reading the echocardiogram, but none caused hemodynamic compromise. These thrombi were reported by the cardiologist to the patient care team; the remainder were not. No subject had more than one thrombus detected. Nineteen thrombi were noted on echocardiogram 1 to 2 weeks after UVC removal and 50% were located at the RA-IVC junction. The time of thrombus onset is shown in Figure 2.

Patient Demographics

Of the 210 neonates in the study, 53% were male and 60% were Caucasian. The mean gestational age of the entire cohort was 27.7 ± 2.1 weeks (standard deviation) and mean birth weight was 923 ± 195 grams. Demographics of the thrombus and no-thrombus groups are shown in Table I. The neonates without thrombus had lower gestational ages than the group those with thrombus; however, there was no significant difference between the birth weights in the two groups.

Thrombotic Risk Factors

A lower platelet count in the first week was observed in neonates with thrombus (Figure 3). A significant, strong association between thrombus and hematocrit more than 55% in the first week of life was observed (Table II). As previously reported, more SGA infants than

non-SGA infants were noted to have thrombus¹¹. Maternal preeclampsia was also associated with risk of thrombus formation. No relationship was observed between thrombosis and elevated serum sodium, septicemia, IDM or non-O blood groups.

In a multivariate regression analysis, including the factors associated with thrombosis on bivariate analysis, hematocrit more than 55% in the first week of life was the only significantly, independently associated factor (Table III).

Discussion

The present retrospective study was designed to evaluate the risk factors associated with UVC-related thrombosis in very low birth weight infants. Associations were shown between thrombosis and hematocrit greater than 55% in the first week of life, being SGA, maternal preeclampsia, higher gestational age and lower platelet count. However, when those factors were combined in a regression model, only hematocrit greater than 55% in the first week of life remained independently associated with thrombus. The risk factors were assessed only during the first week of life based on the assumptions that early findings would allow the clinician to make decision about whether or not to place a catheter and leave in place. Moreover, early steps in the pathogenesis of catheter related thrombosis probably occur quite early in a catheter's lifespan, making the early clinical variables the most relevant.

The independent association between hematocrit more than 55% in the first week of life and thrombosis in newborns is an important finding. Neonatal polycythemia has been reported to be associated with an increased risk of thrombosis in several reports but seldom have been evaluated systematically as a risk factor.^{5, 24, 25} Thrombosis in neonates with polycythemia is most likely due to hyperviscosity resulting in reduced blood flow within microcirculation and chronic activation of platelets²⁶. Polycythemia has also been reported as an associated factor in neonates with spontaneous superior sagittal sinus thrombosis²⁵.

Our results also showed that neonates with thrombosis were more likely to be SGA than neonates without thrombosis. Maternal acquired and congenital thrombophilias contribute to pathophysiology of being SGA^{27–31}. SGA infants have been found to have persistent antithrombin III deficiency and polycythemia, which may be major risk factors for thromboembolic complications in SGA neonates³².

A significant difference was also found between neonates with and without thrombosis with respect to maternal preeclampsia. This finding is in agreement with the report of Hunt et al that found a strong association between preeclampsia and neonatal venous sinus thrombosis³³. As is true for SGA, there seems to be an association between preeclampsia and inherited maternal predisposition to thrombosis^{34, 35}. In addition, preeclamptic women are also noted to have higher thrombin-antithrombin (TAT) complex concentrations than normal pregnant women³².

We also noted significant difference between the platelet counts in the two groups. However, contrary to our hypothesis, very low birth weight neonates with UVC-associated thrombosis did not have higher platelet counts than neonates without UVC-associated thrombosis. In fact, there was a trend towards an independent negative association between platelets and thrombosis. Thrombocytopenia has often been noted in low birth weight infants and SGA infants^{32, 36}. The lack of association with elevated platelet count suggests that increased thrombotic risk in neonates may be primarily due to abnormalities in secondary hemostasis; primary hemostasis may contribute only little to the process.

Several mechanisms may be involved in the association of polycythemia and preeclampsia with thrombosis. It is possible that placental insufficiency results in chronic hypoxic state of

the fetus that bolsters erythropoiesis and hence results in polycythemia. Being SGA probably also has similar effect of intrauterine hypoxia on both fetal growth and erythropoiesis, with resultant polycythemia. As mentioned previously, hyperviscosity and chronic activation of platelets are likely to be the immediate antecedents to thrombosis²². Thrombosis could also be the result of direct effects on coagulation in SGA infants.

There are limitations to our study. First, the retrospective, observational design of the study allows us to determine relationships among variables, but not causality. Further evaluation of the significant risk factors identified in our study in a prospective analysis is warranted. Second, extrapolation of the results to full term neonates should be performed carefully due to numerous physiologic differences between full term neonates and VLBW infants. Third, despite the relatively large size of the study, only a few infants developed thrombi. This gave us low power to assess potential associated risk factors. Fourth, since our study evaluated only umbilical venous catheter-associated thrombi, care is warranted in extrapolating the data to peripherally inserted central catheters or other central catheters. Fifth, thrombi were evaluated by echocardiogram, which is limited in evaluating thrombi outside of the thoracic great vessels. Finally, our study did not evaluate congenital thrombophilic risk factors, such as Factor V Leiden or congenital anticoagulant protein deficiency in neonates, which may contribute to thrombophilic risk during neonatal period.

In conclusion, the present study evaluated some of the potential risk factors for umbilical venous-catheter-associated thrombosis in very low birth weight infants, and suggested a strong association between polycythemia and UVC-related thrombosis. This association may represent the final common pathway of other previously reported associations, such as maternal preeclampsia and SGA that may result in hypoxic state in fetus triggering polycythemia. Although no infant in our study suffered an adverse event related to thrombus, the risk-benefit ratio of UVC may be altered in very low birth weight infants who have hematocrit more than 55% in first week of life. This report suggests that measurement of hematocrit should be performed in all VLBW infants with central venous catheters. It may be reasonable to consider close ultrasound follow up in polycythemic VLBW infants with central venous catheters.

Acknowledgments

This research was supported by Ruth L. Kirschstein National Research Service Award through NIH T32 FY0506: Hematology Training Grant, PI: Charles Francis, M.D., and in part by grant 5 M01 RR00044 from the National Center for Research Resources, National Institutes of Health, Bethesda, MD for the University of Rochester General Clinical Research Center, and by a Strong Memorial Hospital Innovations in Patient Care grant. Dr. Stevens is funded by a grant from the National Institute of Child Health and Human Development (K23 Award # HD050646).

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Figure 1.
Echocardiograph representation of a sheath-like thrombus at UVC tip.

Figure 2.

Days of age at thrombus onset. Thrombus onset was defined as the date thrombus was first detected on echocardiogram or time of UVC removal, whichever occurred earlier. Number of catheters remaining in italics at bottom.

Figure 3. Platelet Counts in infants with and without UVC-associated thrombus. Mean ($\pm$ standard deviation) platelet counts were $238 \pm 70 \times 10^3/\mu\text{L}$ in infants without thrombus and $193 \pm 57 \times 10^3/\mu\text{L}$ in those with thrombus; $p=0.005$. Box represents 50% of the data; whiskers represent range of the data; * indicates mean; — indicates median.

Table I

Demographic Characteristics

	Thrombus (n=22)	No Thrombus (N=188)	P-VALUE
Race			0.97
White	14(64)	112(60)	
Black	7(32)	62(33)	
Other	1(4)	14(7)	
Gender (Male)	14(64)	98(52)	0.31
Birth Weight (g)	890±219	927±193	0.41
Gestational Age (wk)	28.8±2.5	27.6±2.0	0.02
Data presented as n (%) or mean ± SD			

Table II

Risk factors for thrombosis (bivariate analysis)

	Number with thrombus		OR (95% CI)	P-value
	Risk Factor Present	Risk Factor Absent		
Hematocrit >55%	8/26 (31%)	14/184 (8%)	5.4 (2.0-14.6)	0.0003
Maternal Preeclampsia	12/55 (22%)	10/145 (7%)	4.1 (1.64-10)	0.001
SGA	9/45 (20%)	13/165 (9%)	2.92 (1.16-7.37)	0.02
Dehydration	5/50 (10%)	17/160 (11%)	0.94 (0.33-2.68)	0.90
IDM	1/8 (13%)	21/202 (10%)	1.23 (0.14-10.5)	0.85
Non-type O blood group	13/127 (10%)	8/73 (11%)	0.93 (0.37-2.35)	0.87
Septicemia	5/36 (14%)	17/174 (10%)	1.49 (0.51-4.34)	0.46
Odds ratio (OR); confidence Interval (CI); small for gestational age (SGA); infant of diabetic mother (IDM)				

Table III

Risk factors for thrombosis (multivariate logistic regression analysis)

	OR	95% CI	P-value
Hematocrit >55%	3.657	1.134-11.791	0.03
Platelets (per $10 \times 10^3/\mu\text{L}$)	0.940	0.856-1.033	0.197
SGA	1.572	0.473-5.220	0.46
Gestational Age	1.018	0.784-1.323	0.89
Maternal Preeclampsia	1.770	0.612-5.125	0.29
Odds ratio (OR); confidence interval (CI); small for gestational age (SGA)			