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Abnormal Abdominal Aorta Hemodynamics Are Associated with Necrotizing Enterocolitis in Infants with Hypoplastic Left Heart Syndrome

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Abstract

Background—The risk of necrotizing enterocolitis (NEC) in association with congenital heart disease is highest in patients with hypoplastic left heart syndrome (HLHS). Within the HLHS population, however, risk factors for NEC remain debated. We hypothesized that some infants with HLHS have vascular changes that contribute to gut hypoperfusion, independent of diastolic run-off and low cardiac output.

Methods—We analyzed the abdominal aorta pulsatility index and right ventricular function on routine pre-operative and post-operative echocardiograms for all infants who underwent stage I palliation for HLHS from January 2007 to January 2012. The echocardiography findings and clinical course were compared between those with and those without an episode of NEC.

Results—Of the 61 cases reviewed, 11 (18%) developed NEC during a mean follow-up of 3.8 ± 1.3 years. Those with NEC had a lower abdominal aorta pulsatility index compared to those without NEC both on stage I pre-operative (3.38 ± 0.15 vs. 3.89 ± 0.09 , $p < 0.05$) and post-operative echocardiograms (2.21 ± 0.28 vs. 3.05 ± 0.78 , $p = 0.01$) despite similar ventricular function and operative risk.

Conclusions—Abdominal aorta Doppler pulsations are lower in patients with HLHS whose clinical course is complicated by NEC. This finding suggests that the systemic vasculature in a subset of neonates with HLHS may be inherently abnormal. Further investigation is warranted to determine if this is secondary to structural changes in the mesenteric and/or systemic vasculature.

Keywords

Congenital heart disease; hypoplastic left heart syndrome; necrotizing enterocolitis

INTRODUCTION

Necrotizing enterocolitis (NEC) significantly increases the morbidity and mortality in patients with hypoplastic left heart syndrome (HLHS). Among neonates with congenital heart disease (CHD), those with HLHS have the highest incidence of NEC ranging between 11 and 20% [1,2]. For those with HLHS who are unfortunate enough to develop NEC,

mortality has been reported near 40%, with a mortality odds ratio close to 6. When NEC is severe enough to cause pneumatosis intestinalis, mortality increases to as high as 75% [3].

Low cardiac output, diastolic run-off from aorto-pulmonary shunts, and aggressive post-operative feeding regimens have all been implicated in the increased risk of NEC in HLHS, but their roles remain controversial. For example, while an episode of low cardiac output has been shown to be a risk factor for NEC, the majority of cases occur in the face of normal cardiac output [2]. The role of diastolic run-off remains poorly defined given the similar incidence of NEC between infants who underwent stage I palliation with a right ventricle to pulmonary artery (RV-PA) vs. an aorto-pulmonary shunt [4]. Finally, the role of post-operative feeding regimens and their link to NEC has recently been challenged [5]. Post-feeding resistance indices and flow velocities in the mesenteric arteries are abnormal in infants with HLHS regardless of shunt type [6]. This raises the possibility of abnormalities in the systemic vasculature of the neonate with HLHS that contribute to the development of NEC.

The prenatal drivers of systemic vascular development in fetuses with CHD are poorly understood. They are likely multifactorial and include both prenatal features unrelated to the cardiac lesion as well those resulting from physiology altered by the structural heart defect [7,8]. This interplay between maternal, placental and fetal factors may lead to variations in vascular development and predispose a subset of infants to NEC. The purpose of this study was to determine if abdominal aorta Doppler pulsations would give us insight into abnormal vascular development in infants with HLHS who develop NEC.

MATERIAL AND METHODS

Study Population

In this retrospective cohort study, we collected data for all consecutive infants who underwent stage I palliation for HLHS at our institution between January 2007 and January 2012. The study was approved by the Institutional Review Boards of both the University of Utah and Primary Children's Medical Center with a waiver for consent. Inclusion criteria were HLHS or other single right ventricle anomaly requiring stage I palliation (including double outlet right ventricle with mitral atresia and critical aortic stenosis with left ventricular hypoplasia) and survival to initiation of feeds following surgery. Exclusion criteria were unbalanced atrioventricular septal defect, known chromosomal abnormality, surgical intervention prior to stage I palliation, and first NEC event occurring after cardiac transplantation.

Clinical Outcomes

The modified Bell staging criteria were used to determine NEC incidence [9]. These criteria classify NEC as either suspected (IA and IB), proven (IIA and IIB) and advanced (IIIA and IIIB). In this study, classification of stage IB (clinical signs of abdominal distention, feeding intolerance, along with grossly bloody stool) or higher was considered positive for NEC. A classification of stage II or higher was defined by radiographic evidence of pneumatosis intestinalis. The anatomic classification of HLHS was determined by the post-natal echocardiogram. Small for gestational age (SGA) was defined as birth weight <10th percentile for gestational age based on recently published normative data [10]. Weight for age z-score was calculated with the World Health Organization anthropometric calculator (WHO Anthro v3.2.2).

Abdominal Aorta Pulsatility Index

All echocardiograms were performed on commercially available systems (Acuson Sequoia 512, Siemens Medical Solutions, Mountain View, CA, USA; and Phillips IE33, Royal Phillips Electronics, Amsterdam, The Netherlands). All images were stored digitally and reviewed on the local archiving system (Syngo, Siemens Medical Solutions, Mountain View, CA, USA). We calculated the pulsatility index in the abdominal aorta from the spectral Doppler on the first complete pre-operative echocardiogram and routine complete stage I post-operative echocardiogram (typically performed just prior to hospital discharge) for each subject. Spectral Doppler interrogation of the abdominal aorta was obtained from the subcostal view. Pulsatility index (PI) was calculated as $PI = (\text{Peak Systolic Velocity} - \text{End Diastolic Velocity}) / \text{Mean Antegrade Velocity}$. Utilizing the mean antegrade velocity rather than the mean overall velocity was an adaptation of the standard PI equation [11] as reversal of flow in the abdominal aorta, seen in all of the pre-operative studies, was not calculated into the mean velocity (Figure 1). This method was used to calculate the PI for all studies. Echocardiograms were excluded from analysis if abdominal aortic Doppler interrogation was not performed or fidelity was inadequate for an accurate trace. We also excluded pre-operative echocardiograms if they were performed while the infant was hemodynamically unstable, defined clinically by late presentation with acidosis and evidence of end-organ failure, and objectively as a time to peak velocity >100 msec on Doppler interrogation of the abdominal aorta.

Right Ventricular Function

We recorded ventricular function only for those infants whose echocardiogram was deemed adequate to calculate a pulsatility index. Subjective right ventricular systolic function was recorded as either normal or abnormal based on the report generated for clinical use at the time of the study. Objective right ventricular systolic function was prospectively acquired with velocity vector imaging (VVI) using commercially available software (Syngo, Siemens Medical Solutions, Mountain View, CA, USA) on the stored echocardiograms. The endocardium of the right ventricle was manually traced at end-diastole using a point-click approach. The curves of velocity, strain and strain rate were averaged across two cardiac cycles. Global longitudinal strain was defined as relative deformation of the myocardium and expressed as a percentage.

Statistical Analysis

Normally distributed outcomes between the two groups were compared using unpaired t-test. Categorical variables were compared using Chi-square test. Statistical significance level was set at a $p < 0.05$. Analysis was performed with Graph Pad Prism (Graph Pad Software, San Diego, CA, USA).

RESULTS

Study Population

Of the 67 infants who met anatomic inclusion criteria, 63 (94%) survived to the initiation of feeds. Two patients developed NEC after subsequent cardiac transplantation and were excluded from analysis leaving 61 infants to form the study cohort regardless of whether their echocardiograms were adequate for PI measurements. Mean follow-up was 3.8 ± 1.4 years. During the study period, 18% (11/61) of subjects developed NEC with 73% (8/11) of the NEC events graded Bell stage II. The clinical timing of NEC fell into multiple time frames including pre-operative (n=1), prior to stage I discharge (n=3), between stage I and stage II (n=5), post-stage II (n=1), and post-Fontan (n=1). There was no difference in gender, birth weight, baseline anatomy, surgical procedure, bypass time or cross-clamp time

between the two groups (Tables 1 and 2). Both groups had similar intensive care unit (ICU) and hospital length of stay as well as discharge weight for age z-score. Those with NEC had significantly fewer days to chest closure (2.9 vs. 4.5 days, $p<0.05$, Table 2).

Abdominal Aorta Pulsatility Index and Adequacy of Echocardiograms

The abdominal aorta pulsatility index on the pre-operative stage I echocardiogram was lower for infants who developed NEC at any time during their follow-up compared to the group with no NEC (3.38 ± 0.15 vs. 3.89 ± 0.09 , $p<0.05$, Figure 2). A similar difference in pulsatility index was seen between the NEC and no NEC groups at the time of the routine stage I post-operative echocardiogram (2.21 ± 0.28 vs. 3.05 ± 0.78 , $p=0.01$, Figure 3). A significantly lower percentage of the pre-operative echocardiograms were adequate for retrospective analysis in the NEC group (64%) compared to the group with no NEC (94%, Table 2). This difference was not secondary to exclusion because of hemodynamic instability. Only two pre-operative echocardiograms were excluded because of unstable hemodynamics at the time of study and both were in the no NEC group. Of the post-operative echocardiograms, there was no difference in the percentage of adequate studies between those with NEC (55%) and those with no NEC (56%, Table 2). The use of vasoactive medications at the time of echocardiogram was similar between those patients in the NEC and no NEC groups. At the time of the pre-operative echocardiogram no patients in the NEC group and 5% of the no NEC group were on inotropic support ($p=1.0$). At the time of the post-operative echocardiogram, the percentages of patients in the NEC and no NEC groups who were on oral afterload reduction was also similar (50% and 44%, respectively, $p=1.0$).

Right Ventricular Function

Ventricular function was similar between the NEC and no NEC groups. On the pre-operative stage I echocardiogram, 14% of the NEC group and 9% of the no NEC group had abnormal subjective right ventricular function reported at the time of the study ($p=0.54$). Similarly, there was no difference in average global longitudinal strain between the NEC ($-11.0 \pm 1.3\%$) and the no NEC ($-10.1 \pm 0.5\%$, $p=0.52$) groups. On the post-operative stage I echocardiogram, 33% of the NEC group and 11% of the no NEC group had abnormal subjective right ventricular function reported at the time of the study ($p=0.21$). The average global longitudinal strain was $-8.9 \pm 0.9\%$ in the NEC and $-9.0 \pm 0.6\%$ in the no NEC groups ($p=0.99$). The pulsatility index was similar for those with subjectively normal vs. those with subjectively abnormal function on the stage I pre-operative (3.86 ± 0.08 vs. 3.49 ± 0.22 , $p=0.18$), and post-operative echocardiograms (2.95 ± 0.14 vs. 2.44 ± 0.21 , $p=0.15$).

DISCUSSION

We present the first report showing that HLHS patients who developed NEC had a lower abdominal aorta pulsatility index both before and after their stage I palliation, compared to those who did not develop NEC. Importantly, the decreased pulsatility index was not associated with decreased right ventricular systolic function.

The clinical characteristics of our cohort challenge the significance of traditionally discussed cardiac and peri-operative risk factors for NEC in HLHS. In our study, peri-operative risk was similar between the NEC and no NEC groups. In contrast to expected findings, patients with NEC had earlier chest closure and tended to have less complicated stage I hospital courses with respect to days of mechanical ventilation, as well as hospital and ICU length of stay. They also had similar discharge weight for age z-scores. Similar to other reports [4,12], our findings failed to support diastolic run-off as an explanation for NEC as all of the subjects in the NEC group had a Sano conduit that limits diastolic run-off [13] and two had

NEC after stage II palliation. The timing of NEC varied considerably within our cohort. This created a wide range of time between the echocardiogram and the NEC event. This variability further strengthens the argument that the abnormal pulsatility index is due to intrinsic vascular abnormalities and not acute clinical changes. Finally, a pre-operative episode of low cardiac output was not responsible for NEC in our cohort. Patients who presented in extremis and had evidence of hemodynamic instability on their pre-operative echocardiogram from were excluded from our analysis of pulsatility index. There were only two patients who met these criteria and neither of them developed NEC during the study period.

The decreased pulsatility index in the setting of comparable right ventricular function and surgical course implicates differences in the vasculature between those who develop NEC and those who do not. It is unclear whether such differences result from primary pathology of the systemic and/or mesenteric vasculature or from gastrointestinal tract inflammation that secondarily affects the mesenteric vasculature. In animal models of NEC, intestinal pre-mucosal arterioles vary in size and have abnormal flow patterns [14]. In other animal studies, augmenting mesenteric blood flow with captopril decreases the severity of NEC related intestinal damage [15]. Neither of these models, however, provides a causal explanation of the vascular abnormalities. Our finding of lower pulsatility index at the time of neonatal admission, however, suggests that vascular perturbations are present prior to the NEC event and its associated inflammation in some individuals. Therefore, a risk factor for NEC that is inherent to the vasculature may be present and warrants further investigation.

We can only speculate on the development of abnormal vasculature in the population with a lower pulsatility index. Fetal vascular development is a complex process subject to maternal, placental and fetal factors. In HLHS, fetal systemic afterload increases while cerebral artery resistance decreases in what is thought to create a ‘brain sparing’ circulatory adaptation to the structural heart defect *in utero* [16–18]. Placental pathology is associated with its own set of fetal vascular adaptations also aimed at increasing cerebral blood flow [19]. In the case of uteroplacental insufficiency (UPI), the fetal adaptation has prolonged post-natal effects. A history of UPI correlates with increased aortic stiffness in infancy [7] and higher blood pressures in childhood [20]. The inter-play between fetal adaptations to UPI and structural heart disease are not well understood; neither are the heterogeneity of the fetal adaptations to HLHS nor their long term effects on the vasculature. Our findings suggest that better understanding of these areas will improve the fidelity by which we stratify risk within the HLHS cohort and help guide patient-specific therapies.

This study is limited by its small sample size and retrospective design. The percentage of acceptable echocardiograms highlights the limitations of retrospective analysis of spectral Doppler. While there were fewer acceptable pre-operative studies in the NEC group versus the no NEC group, the reasons for exclusion in the NEC group were technical and not clinically based. Therefore, the likelihood of this difference being clinically relevant is low. Since there was reversal of flow from the large patent ductus arteriosus on the pre-operative echocardiograms, the pulsatility index equation was modified to minimize confounding by the retrograde flow. This method has not been previously validated so there are no reference values for comparison. Although this method may not allow for consideration of variability in diastolic run-off, it was consistently used for all studies in both groups. The small study size increases the risk of type II error and limited our ability to detect significant differences between the groups. Given the trends that we found in outcomes, however, a larger study group with sufficient power would also have the potential to further support our hypothesis.

In summary, we found that HLHS patients who develop NEC had a lower abdominal aorta pulsatility index despite comparable surgical and functional cardiac risk. While the

pathogenesis of NEC is multifactorial, the role of vascular development warrants further investigation to explore potential congenital vascular perturbations that may allow for better risk-stratification and patient-specific treatment to decrease morbidity and mortality in this vulnerable population.

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Figure 1. Abdominal aorta pulsed wave Doppler signal and representative trace for the modified pulsatility index (PI). PI was measured as the peak systolic velocity (a) minus the end-diastolic velocity (b) divided by the mean antegrade velocity (c).

Figure 2. Pulsatility index as measured in the abdominal aorta on pre-operative echocardiogram for patients who later developed necrotizing enterocolitis (NEC) and those that did not (No NEC). * $p < 0.05$

Figure 3. Pulsatility index as measured in the abdominal aorta on echocardiogram at the time of Norwood discharge for patients who developed necrotizing enterocolitis (NEC) and those that did not (No NEC). ** $p=0.01$

Table 1

Characteristics and risk factors of patients who developed necrotizing enterocolitis (NEC) and those that did not (no NEC).

	No NEC	NEC	p
Patients	50	11	
Male	30 (60%)	7 (64%)	1.00
Anatomy			
AA, MA	16 (32%)	6 (55%)	0.18
AA, MS	9 (18%)	1 (9%)	0.67
AS, MA	4 (8%)	0	1.00
AS, MS	11 (22%)	3 (27%)	0.70
HLHS variant	10 (20%)	1 (9%)	0.67
Birth Weight (g)	3130 ($\pm$ 439)	3126 ($\pm$ 450)	0.98
SGA	4 (8%)	1 (9%)	1.00

AA=aortic atresia, MA=mitral atresia, AA=aortic atresia, AS=aortic stenosis, HLHS variant includes critical AS with left ventricular hypoplasia and double outlet right ventricle with mitral atresia.,

Table 2

Clinical findings and peri-operative risk factors of patients who developed necrotizing enterocolitis (NEC) and those that did not (no NEC).

	No NEC	NEC	p
Age at Norwood (days)	5.3 ($\pm$ 3.1)	5.3 ($\pm$ 2.9)	0.96
Sternal Closure (post-op day)	4.5 ($\pm$ 2.4)	2.9 ($\pm$ 0.9)	0.03*
Bypass Time (min)	143 ($\pm$ 35)	127 ($\pm$ 28)	0.14
Cross Clamp Time (min)	56 ($\pm$ 16)	52 ($\pm$ 9)	0.46
Shunt Type			
Sano	45 (90%)	11 (100%)	0.57
Modified BT	5 (10%)	0	0.57
Mechanical Ventilation (days)	15.7 ($\pm$ 15.8)	7.5 ($\pm$ 3.2)	0.10
ICU Length of Stay (days)	21.9 ($\pm$ 17.6)	14 ($\pm$ 4.5)	0.15
Hospital Length of Stay (days)	31.6 ($\pm$ 26.4)	22.7 ($\pm$ 7.6)	0.28
Discharge Weight for Age z-score	-1.64 ($\pm$ 0.89)	-1.69 ($\pm$ 1.07)	0.87
Adequate Pre-operative Echo for PI	47 (94%)	7 (64%)	0.02*
Adequate Post-operative Echo for PI	28 (56%)	6 (55%)	1.00
Age at Adequate Pre-operative Echo (days)	0.67 ($\pm$ 0.2)	0.57 ($\pm$ 0.2)	0.87
Age at Adequate Post-operative Echo (days)	20.6 ($\pm$ 3.3)	18.0 ($\pm$ 2.5)	0.72

BT=Blalock-Taussig, ICU=intensive care unit, PI=pulsatility index.