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Pediatrics 2009;123;e901-e906
DOI: 10.1542/peds.2008-3216

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ARTICLE

Short- and Long-Term Outcomes of Necrotizing Enterocolitis in Infants With Congenital Heart Disease

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

What's Known on This Subject

Although prematurity accounts for 90% of case subjects with NEC, 10% occurs in term infants. Congenital heart disease is a risk factor for the development of an NEC-like clinical presentation in term neonates.

What This Study Adds

Infants with CHD have a significantly better NEC prognosis compared with neonates without CHD, independent of gestational age. This study provides evidence that NEC in the cardiac patient is a distinct disease process and should be labeled cardiogenic NEC.

ABSTRACT

OBJECTIVE. Congenital heart disease is a significant risk factor for necrotizing enterocolitis in the term infant. We compared the short- and long-term necrotizing enterocolitis–specific outcomes of infants with congenital heart disease with those of neonates without congenital heart disease.

PATIENTS AND METHODS. A retrospective study of 202 patients with necrotizing enterocolitis treated at our center from May 1999 to August 2007 was conducted. Infants with necrotizing enterocolitis were grouped according to the presence ($n = 76$) or absence ($n = 126$) of congenital heart disease. Demographic and necrotizing enterocolitis–specific outcomes were recorded. The groups were compared by nonparametric and χ^2 analyses. Univariate and multivariate odds ratios were determined for each outcome.

RESULTS. The average birth weight and gestational age of the 2 groups were not significantly different. The initial necrotizing enterocolitis severity, as determined by Bell stage, was less for necrotizing enterocolitis subjects with congenital heart disease compared with those without congenital heart disease. When controlling for birth weight and gestational age, the congenital heart disease group had decreased risk of perforation, need for a bowel operation, strictures, need for a stoma, sepsis, and short bowel syndrome compared with the non–congenital heart disease group. Although not statistically significant, subjects with congenital heart disease had a trend toward decreased risk of death from necrotizing enterocolitis, recurrent necrotizing enterocolitis, and need for peritoneal drainage.

CONCLUSIONS. Infants with congenital heart disease and necrotizing enterocolitis have decreased risk of major short- and long-term negative outcomes associated with necrotizing enterocolitis compared with neonates without congenital heart disease. Differences in initial severity, range of age at diagnosis, and prognoses between subjects with necrotizing enterocolitis with and without cardiac disease suggest that necrotizing enterocolitis in the cardiac patient is a distinct disease process and should be labeled cardiogenic necrotizing enterocolitis. *Pediatrics* 2009;123:e901–e906

www.pediatrics.org/cgi/doi/10.1542/peds.2008-3216

doi:10.1542/peds.2008-3216

Key Words
necrotizing enterocolitis, congenital heart disease, patent ductus arteriosus

Abbreviations
NEC—necrotizing enterocolitis
CHD—congenital heart disease
PDA—patent ductus arteriosus
SBS—short bowel syndrome
OR—odds ratio
CNEC—cardiogenic necrotizing enterocolitis

Accepted for publication Jan 9, 2009

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PEDIATRICS (ISSN Numbers: Print, 0031-4005; Online, 1098-4275). Copyright © 2009 by the American Academy of Pediatrics

NECROTIZING ENTEROCOLITIS (NEC) is a disease predominantly of preterm neonates. The intestinal injury of NEC occurs in association with pathogenic enteric bacteria and leads to bowel ischemia, necrosis, perforation, sepsis, and, in severe cases, death. Although prematurity and the associated immaturity of the gut mucosa account for 90% of cases, 10% of NEC occurs in term infants. There is ample evidence that congenital heart disease (CHD) is a risk factor for the development of an NEC-like clinical presentation in term neonates.[1–7] Episodic or chronic decreased mesenteric perfusion with CHD contributes to the development of this clinical picture in the term infant.[1,2] Persistent diastolic flow reversal in the abdominal aorta of neonates with NEC and CHD substantiates the hypothesis that certain heart defects result in a diastolic steal.[3] The steal phenomenon has also been reported previously for hemodynamically significant patent ductus arteriosus.[4]

Despite a 10- to 100-fold increased risk of NEC in the population with CHD compared with the entire preterm and term neonatal population, little is known about the prognosis of NEC in the context of CHD.[2,5] A small report of 30

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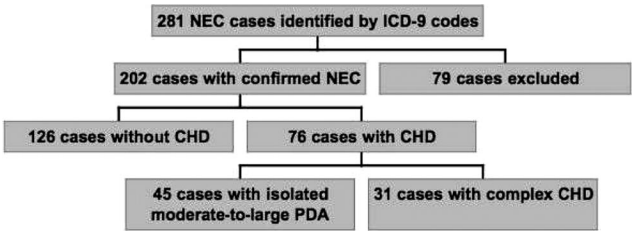

FIGURE 1
Flow chart of selection of patients with NEC with and without CHD. ICD-9 indicates *International Classification of Diseases, Ninth Revision.*

neonates with CHD and NEC from 1981 to 1997 in Hong Kong found increased NEC-related mortality compared with subjects with NEC without CHD.[6] However, term infants with NEC, including infants with CHD, other congenital anomalies, and no known risk factors for NEC, were reported to have lower mortality compared with the traditional preterm population with NEC.[7] The small sample sizes and limited measures of outcome of previous studies necessitate a more extensive examination of the short- and long-term outcomes of NEC in the unique setting of cardiac disease.

Our aim was to determine the natural history and prognosis of NEC in the CHD population compared with the traditional preterm population. A better understanding of NEC outcomes in infants with CHD may help improve clinical decision-making and our understanding of the pathophysiology of this disorder in the CHD population. We believe that this study represents the largest sample of children with CHD and NEC studied to date.

PATIENTS AND METHODS

Cohort Identification

We retrospectively identified a cohort of neonates with NEC treated at Lucile Packard Children's Hospital from May 1999 to August 2007. This time period was chosen because of comparable and accessible records at Lucile Packard Children's Hospital. Patients were identified by the *International Classification of Diseases, Ninth Revision*, codes for NEC (777.5) or acute vascular insufficiency of the intestine (557.0). The original search identified 281 patients, including those with congenital heart defects (Fig 1). Seventy-nine case subjects were excluded because they did not fit the criteria for NEC. Frequent alternative diagnoses excluded from the study were intestinal atresia, gastroschisis, isolated intestinal perforation, and malrotation. Each case subject was staged according to modified Bell stage criteria for NEC.[8]

The type and severity of CHD were confirmed through review of cardiologist reports, echocardiography reports, and consultation with a pediatric cardiologist. The resulting cohort consisted of 126 patients with NEC and no evidence of CHD and 76 patients with NEC and ≥1 significant congenital heart defect (Table 1). Of the 76 case subjects with CHD, 45 had an isolated moderate-to-large patent ductus arteriosus (PDA), leaving 31 with a significant cardiac lesion other than or in addition to a PDA (Fig 1). This distinction was made to explain the lower average gestational age of our CHD group compared with the CHD group of previous studies. The analysis of risk of outcomes included all 76 patients with CHD and did not differentiate based on type of lesion.

TABLE 1 Types and Frequencies of CHDs in the Group of 76 Infants With NEC

CHD	*n*	Additional Defects
Isolated PDA (moderate to large)	45	—
Tetralogy of Fallot/PA	6	5 MAPCAs
Hypoplastic left heart	4	—
AV canal	3	1 PDA
VSD	3	1 PVO, 1 PDA
Transposition of the great arteries	3	2 VSD, PS; 1 AS
Hypoplastic right ventricle	2	1 PS, 1 PA
Truncus arteriosus	2	—
PS	1	1 PDA, 1 PFO
COA	1	—
Ebstein anomaly	1	1 VSD, PS
Interruption of aortic arch (type B)	1	1 VSD, ASD, PDA, AS
Double outlet right ventricle	1	1 COA, ASD, PDA
Aortopulmonary window	1	—
Dilated cardiomyopathy	1	—
ASD	1	1 PVO, PDA
Total	76	—

MAPCAs indicates major aorto-pulmonary collateral arteries; PVO, pulmonary vein obstruction; AS, aortic stenosis; PFO, patent foramen ovale; ASD, atrial septal defect; COA, coarctation of aorta; PS, pulmonic stenosis; VSD, ventricular septal defect; PA, pulmonary atresia; —, no additional defects.

Patient Characteristics

We documented the following characteristics for each patient: gestational age, birth weight, gender, presence and type of congenital heart defect, Bell stage, age at NEC diagnosis, and whether the cardiac defect was repaired or palliated before NEC diagnosis. Based on echocardiography reports, we included patients with tiny-to-small PDA or patent foramen ovale in the non-CHD group because of the ubiquity of small ductuses in the neonatal population and the lack of evidence of decreased splanchnic perfusion from ductal steal.[9] The echocardiographers consistently used the terms "tiny," "small," "moderate," and "large" throughout the study period. The sensitivity and specificity of two-dimensional echocardiography in the diagnosis of PDA using catheterization as the gold standard have been reported previously to be 83% and 100%, respectively.[10]

Outcomes

Short-term outcome measures included the following: (1) time to initiate feeds; (2) bowel perforation; (3) peritoneal drainage; (4) bowel operation; (5) need for stoma; (6) progression to sepsis; and (7) death resulting from NEC. Long-term outcome measures included the following: (1) stricture development; (2) development of recurrent NEC; and (3) development of short bowel syndrome (SBS). Death from NEC did not include those patients who died as a consequence of their SBS or small bowel transplant resulting from NEC ($n = 6$). SBS was defined as total parenteral nutrition dependence >3 months because of enteral nutritional insufficiency.[11]

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Time to initiate feeds was measured as time from the diagnosis of NEC to the date that feeds were initiated and maintained for ≥7 days. Sepsis was determined by review of infectious disease consultation reports. Line infections resulting in bacteremia were not included. The need for bowel operation, peritoneal drainage, or stoma depended on the judgment of the pediatric surgeon. During our study period, a multicenter, randomized trial of peritoneal drainage versus laparotomy for perforated NEC was being conducted. Only preterm neonates (<34 weeks) were included in the randomized trial, which may have affected the percentage of case subjects in our study receiving peritoneal drainage versus bowel operation.[12]

Statistical Analysis

We compared birth weight, gestational age, and age at diagnosis between the NEC case subjects with CHD and NEC case subjects without CHD using a Mann-Whitney test for nonparametric distributions. Gender and Bell stage were compared by χ^2 analyses. We also compared gestational age, birth weight, gender, age at diagnosis, and Bell stage of case subjects with isolated moderate-to-large PDA ($n = 45$) with case subjects with more complex CHD ($n = 31$). Statistical significance was set at a P value of <.05.

To determine whether NEC case subjects with CHD had decreased risk of NEC-associated morbidities and mortality, we conducted univariate analyses and multivariate logistic regression for each outcome with adjustment for birth weight, gestational age, and age at diagnosis. In addition, subjects with CHD were further divided into those with an isolated PDA and those with a more complex cardiac lesion. The univariate and multivariate risks of each outcome were then analyzed by comparing non-CHD with isolated PDA and by comparing non-CHD with CHD other than isolated PDA. Risk was represented as an odds ratio (OR) with 95% confidence intervals. Time to initiate feeds was analyzed by Cox regression controlled for birth weight and gestational age. SPSS 16.0 software (SPSS Inc, Chicago, IL) was used for statistical analysis.

RESULTS

Patient Characteristics

Comparisons of patient characteristics of NEC case subjects with and without CHD are summarized in Table 2. There were no significant differences in gender, gestational age, and birth weight between the neonates with CHD and those without CHD. The majority of case subjects were boys in both groups. In addition to comparing characteristics of neonates with and without CHD, the CHD group was further divided into 2 groups: infants with isolated moderate-to-large PDA and infants with a more complex cardiac lesion. The gestational age and birth weight of the complex CHD group were significantly greater than those in the isolated PDA group (Table 3). There were no significant differences in gender, age at diagnosis, or Bell stage.

The median age at NEC diagnosis was 10 days for non-CHD infants compared with 14 days for infants with CHD ($P = .016$). The age range for the CHD group was 0 to 246 days compared with 0 to 77 days for the non-CHD group (Table 2). Bell stages were significantly different between groups ($P = .009$). The majority of case subjects without CHD were diagnosed as stage III (44%), whereas the majority of case subjects with CHD were considered stage II (49%).

Of those case subjects with CHD, 48% of them were diagnosed with NEC before medical (ie, indomethacin) or surgical intervention. Of the patients who were diagnosed with NEC after a CHD-related intervention, 36% were surgically repaired (eg, coarctation repair), 31% were surgically palliated (eg, Norwood procedure for hypoplastic left heart), and 33% were medically treated (eg, indomethacin for PDA).

TABLE 2 Demographics, Age at Diagnosis, and Bell Stage of Subjects With NEC With and Without CHD

Variable	NEC Without CHD ($N = 126$)	NEC With CHD ($N = 76$)	*P*
Male gender, *n* (%)	71 (56)	45 (60)	NS
Gestational age, mean ± SD, wk	29.9 ± 4.37	30.6 ± 5.43	NS
Birth weight, mean ± SD, kg	1.44 ± 0.69	1.49 ± 0.87	NS
Age at diagnosis, median (minimum to maximum), d	10 (0–77)	14 (0–246)	.002
Bell stage I, *n* (%)	27 (21)	22 (29)	.009
Bell stage II, *n* (%)	44 (35)	37 (49)	.009
Bell stage III, *n* (%)	55 (44)	17 (22)	.009

NS indicates not significant.

TABLE 3 Demographics, Age at Diagnosis, and Bell Stage of Subjects With NEC With Isolated PDA and With CHD Other Than Isolated PDA

Variable	NEC With Isolated PDA ($N = 45$)	NEC With CHD Other Than Isolated PDA ($N = 31$)	*P*
Male gender, *n* (%)	28 (62)	17 (55)	NS
Gestational age, mean ± SD, wk	27.3 ± 2.56	35.8 ± 4.60	<.001
Birth weight, mean ± SD, kg	0.985 ± 0.340	2.340 ± 0.820	<.001
Age at diagnosis, median (minimum to maximum), d	14 (2–61)	16 (0–246)	NS
Bell stage I, *n* (%)	16 (36.0)	6 (19.3)	NS
Bell stage II, *n* (%)	18 (40.0)	19 (61.3)	NS
Bell stage III, *n* (%)	11 (24.0)	6 (19.3)	NS

NS indicates not significant.

Outcomes

Univariate analyses revealed better NEC-related short-term and long-term outcomes for neonates with CHD (Table 4). Infants with CHD had a statistically significant decreased risk of perforating (OR: 0.42 [95% confidence interval: 0.22–0.81]), needing a bowel operation (OR: 0.30 [95% confidence interval: 0.15–0.58]), developing a stricture (OR: 0.06 [95% confidence interval: 0.01–0.50]), needing a stoma (OR: 0.46[95% confidence in-

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TABLE 4 Risk of NEC-Specific Outcomes for Subjects With and Without CHD

Outcome	NEC Without CHD ($N = 126$), n (%)	NEC With CHD ($N = 76$), n (%)	Univariate OR (95% CI)	Multivariate OR (95% CI)[a]	Multivariate OR (95% CI)[b]
Perforation	51 (40)	17 (22)	0.42 (0.22–0.81)	0.42 (0.21–0.84)	0.51 (0.22–1.18)
Peritoneal drainage	21 (17)	10 (13)	0.76 (0.34–1.71)	0.84 (0.35–2.00)	0.66 (0.22–2.00)
Bowel operation	57 (45)	15 (20)	0.30 (0.15–0.58)	0.26 (0.12–0.55)	0.29 (0.12–0.71)
Sepsis	28 (22)	8 (11)	0.41 (0.18–0.96)	0.36 (0.15–0.89)	0.46 (0.13–1.55)
Recurrent NEC	11 (9)	4 (5)	0.58 (0.18–1.89)	0.45 (0.12–1.66)	0.67 (0.17–2.70)
Stricture	22 (17)	1 (1)	0.06 (0.01–0.48)	0.07 (0.01–0.50)	0.06 (0.01–0.47)
SBS	13 (10)	0 (0)	Not estimable[c]	Not estimable[c]	Not estimable[c]
Need for stoma	39 (31)	13 (17)	0.46 (0.23–0.93)	0.43 (0.20–0.93)	0.50 (0.20–1.26)
Death from NEC	18 (14)	6 (8)	0.51 (0.20–1.36)	0.42 (0.14–1.21)	0.37 (0.12–1.10)

CI indicates confidence interval.

[a] Multivariate model includes CHD, birth weight, and gestational age.

[b] Multivariate model includes CHD, birth weight, gestational age, and age at diagnosis.

[c] OR cannot be estimated because of 0 occurrences of SBS in the CHD group.

terval: 0.23–0.93]), becoming septic (OR: 0.41 [95% confidence interval: 0.18–0.96]), and developing SBS (OR not estimable because there were 0 cases of SBS in the CHD group). Case subjects with CHD also had a trend toward decreased risk of death from NEC (OR: 0.51 [95% confidence interval: 0.20–1.34]), need for peritoneal drainage (OR: 0.76 [95% confidence interval: 0.37–1.80]), and recurrence of NEC (OR: 0.58 [95% confidence interval: 0.18–1.89]; however, these differences lacked statistical significance. Every measured outcome demonstrated a trend toward decreased risk of NEC-related mortality and morbidity for NEC case subjects with CHD.

The univariate risk of each outcome was also analyzed by comparing the non-CHD group with the isolated moderate-to-large PDA group. The purpose of this analysis was to determine whether the presence of isolated PDA in premature neonates decreased the risks of negative NEC outcomes with the same magnitude as the primary analysis, including all CHD. Isolated PDA case subjects had a similar reduction in risk as the entire CHD cohort; however, sepsis did not meet statistical significance because of insufficient sample size ($N = 45$; Table 5). An additional comparison of complex CHD cases with non-CHD cases revealed similar risk estimates as the primary analysis and the isolated PDA analysis with the exception of the risk of peritoneal drainage (Table 5). Again, the point estimates were in the same direction and of similar magnitude as the combined CHD analysis but lacked statistical significance because of small sample size ($N = 31$). Risk of peritoneal drainage was decreased for complex CHD and unchanged for isolated PDA, although neither achieved statistical significance.

In the multivariate analysis, the group with CHD and NEC had a significantly decreased risk of the following outcomes: perforation, need for a bowel operation, stricture, need for a stoma, and sepsis (Table 4). When age at diagnosis was added to the multivariate model, only need for a bowel operation (OR: 0.32 [95% confidence interval: 0.15–0.65]) and development of a stricture remained significantly reduced for the CHD-related NEC group (OR: 0.06 [95% confidence interval: 0.01–0.46]; Table 4). The OR for SBS cannot be estimated by multivariate analysis, because there were no subjects with SBS in the CHD cohort. Of the 13 subjects with SBS in the non-CHD group, 4 received small bowel with or without liver transplant (31%); 6 died waiting for, or subsequent to, transplantation (46%); and 2 were lost to follow-up.

TABLE 5 Comparison of Univariate Risk (OR) of NEC-Specific Outcomes for All Subjects With CHD, for Isolated Medium-to-Large PDA, and for CHD Other Than Isolated PDA

Outcome	CHD vs No CHD, OR (95% CI)	PDA vs No CHD, OR (95% CI)	CHD Other Than Isolated PDA vs No CHD, OR (95% CI)
Perforation	0.42 (0.22–0.81)	0.42 (0.19–0.92)	0.43 (0.17–1.07)
Peritoneal drainage	0.76 (0.34–1.71)	1.08 (0.44–2.65)	0.35 (0.08–1.56)
Bowel operation	0.30 (0.15–0.58)	0.22 (0.09–0.54)	0.42 (0.18–1.01)
Sepsis	0.41 (0.18–0.96)	0.54 (0.21–1.40)	0.24 (0.05–1.07)
Recurrent NEC	0.58 (0.18–1.89)	0.49 (0.10–2.28)	0.72 (0.15–3.43)
Stricture	0.06 (0.01–0.48)	0.11 (0.01–0.82)	Not estimable[a]
SBS[b]	Not estimable	Not estimable	Not estimable
Need for stoma	0.46 (0.23–0.93)	0.34 (0.13–0.88)	0.65 (0.26–1.64)
Death from NEC	0.51 (0.20–1.36)	0.75 (0.26–2.15)	0.20 (0.03–1.56)

[a] OR cannot be estimated because of 0 occurrences of stricture in the CHD other than in the isolated PDA group.

[b] OR cannot be estimated because of 0 occurrences of SBS in CHD group.

The hazard ratio for time to initiate feeds was 1.03 (95% confidence interval: 0.73–1.44). Inconsistent record keeping and transfers from outside our institution resulted in missing 13% of dates that feeds were initiated, consequently limiting the analysis.

DISCUSSION

This study shows that preterm and term infants with CHD have decreased NEC-related morbidity and mortality compared with infants without CHD. The CHD cohort had a statistically significant decreased risk of perforation, need for a bowel operation, stricture, need for a stoma, sepsis, and SBS. There was also a trend toward decreased risk of NEC-related death among infants with CHD. The incidences of the measured outcomes among

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the non-CHD cohort are consistent with previous studies.[13,14]

The lack of statistical significance for risk of death and recurrent NEC is likely a function of the low incidence of both of these outcomes and our sample size, despite a relatively large number of case subjects compared with previous studies. However, with multivariate adjustment, the point estimates are still in the same direction and approximately similar in magnitude, which suggests that a larger cohort would reveal similar trends in reduction in morbidity and mortality for infants with CHD and NEC. A multicenter, randomized trial comparing laparotomy with peritoneal drainage in the management of NEC took place at our institution during the same time period as this study. The trial only included neonates <34 weeks' gestational age, which likely affected our assessment of the risk of peritoneal drainage in the entire CHD group and the subgroup analysis (isolated PDA and complex CHD).

The presence of CHD is the major determinant of improved NEC outcomes in the CHD population. Adjustment for gestational age and birth weight did not significantly affect the risk estimates for all of the measures of outcome. Isolated subjects with moderate-to-large PDA had better outcomes than the non-CHD group, despite lower gestational age (27.3 vs 29.9 weeks, respectively). These data indicate that cardiac comorbidity is the main determinant of outcome in the cardiac population, not gestational age. Previous studies of CHD in the context of NEC have been restricted to term infants. The exclusion of preterm infants with cardiac disease is likely a consequence of the source of patients for the study. McElhinney et al[2] only included CHD case subjects admitted to their cardiac ICU; consequently preterm subjects with isolated PDA managed in the NICU were not evaluated. We felt that it was important to include these patients, because abundant evidence exists for decreased mesenteric perfusion, retrograde diastolic flow, and low diastolic pressure resulting from PDA and the subsequent development of NEC, similar to other significant structural lesions.[9,15,16] The better outcomes of preterm neonates with significant ductuses compared with noncardiac neonates suggest that the role of a heart defect should be considered in the etiology and outcome of a subject with NEC in all age groups, not just the term infant. In these preterm infants with PDAs, we may be seeing an NEC-like presentation that is a consequence solely of the PDA and not of gut immaturity, as in the classic NEC case. Consequently, disease severity is less in many preterm infants with PDA than in those without.

The addition of age at diagnosis in the regression model slightly affected risk estimates. When the multivariate analysis included gestational age, birth weight, and age at diagnosis, CHD case subjects had a significantly decreased risk of bowel operation, stricture, and SBS. This indicates that age at diagnosis is a confounder for risk of perforation, sepsis, and need for stoma. Although the CHD infants' average gestational age is equivalent to that of the non-CHD infants, the median age at diagnosis of the CHD group was 4 days greater. Chronologically older neonates may have greater physiologic stability resulting in less negative sequelae from the NEC.

NEC in infants with CHD may have a unique pathophysiology. The decreased risk of negative NEC-related outcomes in the context of cardiac disease suggests that 2 separate pathophysiologies are grouped under the broad categorization of NEC. The exact etiology of NEC is unknown. NEC in the preterm, very-low-to-low-birth-weight neonate is thought to result from the initiation of enteral feeds in the context of functionally immature gut mucosa, bowel injury, and pathogenic organisms.[14] In CHD-related NEC, decreased mesenteric perfusion resulting from cardiovascular abnormalities, cardiac surgery, and bypass may be the predominant initiating factor rather than the initiation of feeds and immature bowel. An animal model of NEC in asphyxiated newborn pigs demonstrated mesenteric flow insufficiency rather than gut immaturity as the predisposing factor for NEC development.[17] In addition to improved outcomes, the range of ages at NEC diagnosis among the CHD cohort supports an alternative pathophysiology. One case subject was diagnosed with Bell stage II NEC at 246 days of life after the development of severe dilated cardiomyopathy. Kurbegov et al[18] found that 12.5% of pneumatosis intestinalis, a characteristic finding of NEC, in the nonneonatal period occurs in infants with decompensated CHD and reported better outcomes among all of the subjects with nonneonatal pneumatosis intestinalis compared with those with neonatal NEC.

Another potential explanation for better outcomes in children with CHD is the close in-hospital monitoring of perfusion and cardiovascular stability that these children receive. Consequently, subjects with NEC in the context of CHD management may be identified earlier than the traditional preterm case. Earlier identification likely results in decreased gut injury, sepsis, and negative sequelae. The initial decreased severity of NEC among subjects with CHD supports this explanation; however, our data cannot directly answer this question, because the exact time of NEC development is unknown.

This study was limited by lack of access to complete medical charts for all of the subjects, because some patients were transferred to our center from outside hospitals for management of cardiac issues. The time that feeds were initiated was unknown in 13% of subjects. This study did not measure all of the known independent risk factors for NEC in the traditional preterm neonate, including maternal cocaine use, absence of prenatal steroid use, low 1- and 5-minute Apgar scores, persistent respiratory distress, and septicemia.[19,20] McElhinney et al[2] did not find Apgar scores to be a significant risk factor among infants with CHD. There is no evidence that these risk factors would have affected the comparison of outcomes in our study.

Our study demonstrates better short- and long-term outcomes for infants with CHD-related NEC compared with the preterm neonatal NEC, independent of gestational age. These outcomes, in conjunction with previous studies supporting the role of decreased mesenteric flow in NEC pathogenesis, suggest that the constellation of signs and symptoms labeled NEC in the cardiac patient

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should be distinguished from traditional NEC. We believe that CHD-related NEC should be labeled as cardiogenic NEC (CNEC).

CONCLUSIONS

A change in nomenclature to distinguish cardiac and noncardiac patients who present with this similar clinical constellation may be a step in the right direction toward a better understanding of the disease processes and individualized management. It is possible that the CNEC patient may benefit from strict control of cardiac output and enhancement of splanchnic perfusion in contrast to more aggressive measures, such as laparotomy. Additional studies are needed to assess the efficacy of different treatment modalities in the care of CNEC.

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Pediatrics 2009;123;e901-e906
DOI: 10.1542/peds.2008-3216

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