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Key learning points from a CHD necrotising enterocolitis learning collaborative across high- and low-performing centres

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Abstract

Objective: Patients with CHD are at risk for developing necrotising enterocolitis. Currently, no standardised approaches for identification, diagnosis, and treatment of necrotising enterocolitis exists, and there are varying rates and management strategies of necrotising enterocolitis across centres. We used the Paediatric Cardiac Critical Care Consortium to identify high- and low-performing centres based on necrotising enterocolitis rates and convened a necrotising enterocolitis working group. The aims of the group were to understand why variability exists, identify risk factors, and create a foundation for a prospective improvement project. *Methods:* Nine centres participated, and collaborative learning sessions were held with multidisciplinary input. REDCap surveys were disseminated to centres to create consensus among site practices and recommendations. *Results:* The following topics were discussed: diagnosis, risk factors, and management. Diagnosis consensus suggests (1) Diagnosis would benefit from a comprehensive scoring tool, and (2) ultrasound may serve as a highly sensitive diagnostic tool for those at high risk with the absence of other radiologic findings of necrotising enterocolitis. Risk factor consensus suggests (1) those with ductal-dependent systemic blood flow are the highest risk, and (2) vasopressors with splanchnic constriction should be used with caution. Management consensus suggests (1) breastmilk be used first-line for feeding, 2) resume feeds 24–48 hours after a necrotising enterocolitis rule-out, and 3) surgical deference to physical examination and laboratory evaluation above radiographic findings. *Conclusion:* Variability exists in diagnosing necrotising enterocolitis and feeding approaches for at-risk patients. Opportunities exist for collaboration to standardise definitions, compare outcomes, identify risk factors, and create consensus on the management of necrotising enterocolitis.

Introduction

Patients with CHD are at risk for necrotising enterocolitis, an inflammatory condition of the intestine with risk of bacterial translocation, intestinal ischaemia, and subsequent sepsis and death.[1] The [1] estimated incidence of necrotising enterocolitis in patients with CHD can range up to 7.1% among term infants and up to 13% among low birthweight preterm infants.[2]

Across paediatric congenital heart centres necrotising enterocolitis rates vary, suggesting the presence of potentially modifiable factors. Furthermore, a standardised approach for the diagnosis and treatment of necrotising enterocolitis in children with cardiac disease does not exist. However, implementation of prescriptive multi-site treatment algorithms is challenging due to the lack of evidentiary support and the need for multi-disciplinary collaboration from diagnosis to resolution in the setting of a low-rate, high-risk diagnosis.

As an alternative model of improvement, the chylothorax work group standardised diagnosis and management of a low-rate high-risk disease by utilising the data-driven infrastructure provided by the Paediatric Cardiac Critical Care Consortium [3] in combination with structured

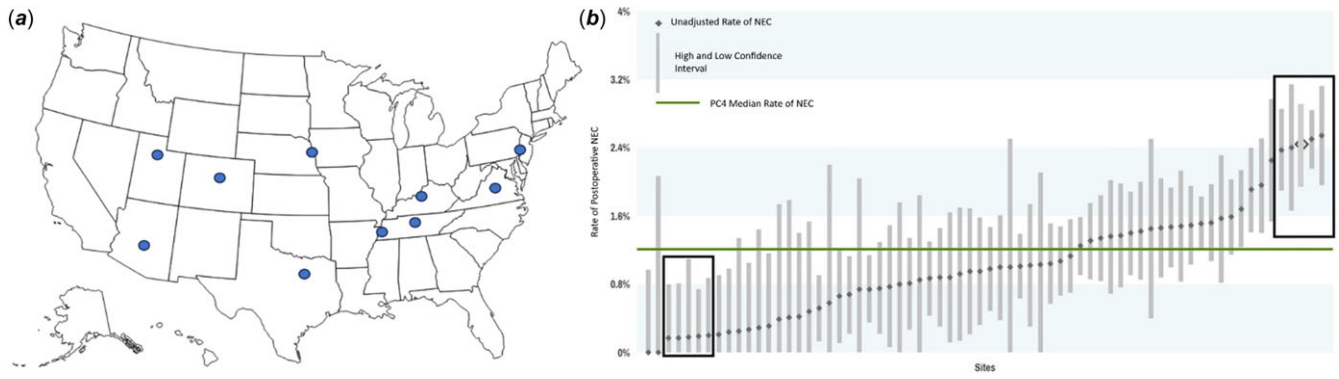

Figure 1. (***a***) geographic distribution of participating centres and (***b***) representation of high and low performing necrotising enterocolitis sites based on Paediatric Cardiac Critical Care Consortium post-operative necrotising enterocolitis Version 3 Arbormetrix data.

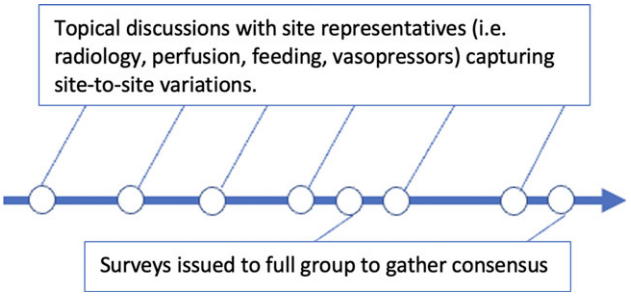

Figure 2. The process of learning sessions, including topic discussions and consensus surveys.

multi-site collaboration. Following this proof-of-concept approach, a necrotising enterocolitis working group was formed, comprised of both high- and low-necrotising enterocolitis incidence centres.

The necrotising enterocolitis working group's aims were to understand the variability in practice of management strategies for necrotising enterocolitis across a range of centres.

Methods

The working group was formed by considering Paediatric Cardiac Critical Care Consortium centres with the highest and lowest rates of necrotising enterocolitis between 2021 and 2023 in surgical patients less than 3 months of age. Ultimately, nine centres participated (four high performing, five low performing), collectively represented by more than 20 providers, including intensivists, cardiologists, nurses, dieticians/nutritionists, and advance practice practitioners (Figures 1a and b).

In addition, input from outside of the collaborative was provided by other stakeholders including general surgeons, cardiothoracic surgeons, and radiologists. The group met for a series of 10 monthly "Learning Sessions." Each session included discussion of a topic related to necrotising enterocolitis and was followed by a literature review (Figure 2).

To accompany each session, protocols and site-specific pathways were shared and reviewed. After six sessions and again after the final session, a Research Electronic Data Capture (REDCap) survey was disseminated for working group members to assemble a consensus understanding of site practices within each topic.[4] The compilation of site practices was not compared to outcomes, nor was the intent to be analysed statistically to understand best practice. The goal is to disseminate a range of practice patterns across a variably successful range of centres, to highlight the opportunities for future clinical decision-making, quality improvement, and research.

Results

The findings of each session are summarised as topics that were discussed, classified into themes of diagnosis (definition, diagnostic imaging), risk factors (vasoactive infusions, transfusions, anatomy, anticoagulation), and management (formula/nutrition, treatment, and surgical perspective). The learning points were not intended to be recommendations or guidelines, but as findings from the collaborative that can contribute to a broader consensus.

Diagnosis

Definition and diagnosis of necrotising enterocolitis

The Paediatric Cardiac Critical Care Consortium defines necrotising enterocolitis by the modified Bell's criteria.[5] Adjuncts to diagnosis not included in Bell's criteria include serum markers for inflammation and infection (i.e. white blood cell count, c-reactive protein) and alternative imaging techniques such as abdominal ultrasound and the use of near infra-red spectroscopy.[6,7 8] The role of biomarkers in necrotising enterocolitis diagnosis, treatment guidance, and prognostication is expanding [1] yet, to date, universally adopted standard biomarkers do not exist in routine clinical practice.

Necrotising enterocolitis pathophysiological features and risk factors in infants with CHD do not completely overlap with those of necrotising enterocolitis in premature neonates. Therefore, applying necrotising enterocolitis diagnostic criteria (either Bell's criteria or adjunct diagnostic criteria) to the CHD population is complex. Traditional Bell's criteria are not tailored to accommodate the unique risk factors and presentation of CHD patients, such as impaired mesenteric ischaemia from dynamic and transient episodes of hypoperfusion, and high abdominal venous pressures limiting organ perfusion.

Diagnostic imaging:

Abdominal radiography has long been a mainstay of the diagnosis of necrotising enterocolitis. While pneumatosis intestinalis and/or portal venous gas are considered pathognomonic, more subtle signs may be noted on radiographs. These include dilated loops of bowel, paucity of bowel gas, and gas-filled loops of bowel that are

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unchanged on follow-up examinations.[9] Because radiographs can be normal in the earliest stages and findings may be equivocal, serial examinations are often warranted.

Abdominal ultrasound can be useful when radiographs are non-diagnostic. Abdominal ultrasound can be used to differentiate intraluminal stool from pneumatosis, which can be difficult to distinguish on radiographs.[10] Abdominal ultrasound may also have higher sensitivity for portal venous gas, pneumoperitoneum, and ascites; however, specificity is inconsistent.[11] Though first described over 40 years ago, adoption of abdominal ultrasound for diagnosis of necrotising enterocolitis remains limited due to resource availability, technical skills, and a lack of diagnostic consensus among neonatologists, intensivists, radiologists, and surgeons.[10,11] Concern has also been raised about high false positive rates, though this has not been well documented.[12]

Across the necrotising enterocolitis working group, the availability of abdominal ultrasound was variable and inconsistent, used at five of the nine sites to some extent. According to general surgeons from the group sites, abdominal ultrasound is not used as a determinant for surgical intervention. Overall, the availability and consistency of abdominal ultrasound were variable, but abdominal ultrasound can serve as a highly sensitive tool for individuals at high risk or with suggestive clinical presentation, even in the absence of radiographic findings.

Risk Factors

Anatomy

In patients with CHD, necrotising enterocolitis is a multifactorial disease process distinct from preterm necrotising enterocolitis.[13,14] CHD-associated necrotising enterocolitis has a mixture of inflammatory and vascular injury involving mesenteric hypoperfusion and/or ischaemia. Proposed mechanisms for abnormal perfusion in CHD include diastolic steal and flow reversal in the abdominal aorta. Multiple types of CHD can lead to necrotising enterocolitis, including a patent ductus arteriosus with significant left-to-right shunting leading to systemic hypoperfusion, generalised hypoxaemia in cyanotic CHD, compromised diastolic gut perfusion pressures restricting oxygenated blood flow in patients with ductal dependent CHD (i.e. coarctation of the aorta), or lesions such as complete atrioventricular canal defect with accompanying atrioventricular valve regurgitation leading to pulmonary over circulation and compromised systemic output.[15,16] Newborns with truncus arteriosus and lesions with ductal-dependent pulmonary blood flow also carry significantly increased risk of developing necrotising enterocolitis.[17,18] In premature or very low birth weight infants, the highest risk of necrotising enterocolitis development is associated with the presence of atrioventricular canal defect.[15,16] Despite this, caution should be made when comparing incidences of necrotising enterocolitis among specific cardiac lesions. In a systemic review and meta-analysis of cardiogenic necrotising enterocolitis in infants with CHD by Asztalos et al, evidence for the incidence of cardiogenic necrotising enterocolitis in specific populations of cardiac anomalies was extremely limited and subject to bias.[2]

Within the necrotising enterocolitis working group, pre-operative CHD lesions that were deemed high risk of developing necrotising enterocolitis included ductal dependent systemic blood flow lesions as well as truncus arteriosus, independent of primary anatomy. This was based on institutional experience despite literature reviews on the topic. In addition, the working group's consensus is that patients who have ductal stents and pulmonary arterial bands, populations with little data as it relates to necrotising enterocolitis, tend to have high rates of necrotising enterocolitis at their sites.

Vasoactive infusions

There is limited literature regarding how vasoactive support in infants with CHD impacts the development of cardiac necrotising enterocolitis. Hemodynamic abnormalities and stressors of gastrointestinal health are common occurrences for infants hospitalised for cardiac surgery. Poor gut perfusion and an imbalance between vasodilatory and vasoconstrictor signalling in the mesenteric microvasculature are factors that can be influenced by vasoactive medications and further exacerbate an at-risk mesentery vascular bed. Episodes stressing the mesentery, including feeds during hypoperfusion states and episodes of diastolic runoff, then add to the risk of developing necrotising enterocolitis. For example, early post-operative hypoperfusion, independent of a low cardiac output state, makes an infant more susceptible to developing necrotising enterocolitis days later in their course.[18,19] There is limited literature on vasoactive infusions and necrotising enterocolitis in patients with CHD.

Within the necrotising enterocolitis working group, most centres feed patients while on a vasopressin infusion but at low doses (Table 1). Most of the centres do not feed patients while on norepinephrine infusions but all centres do feed patients while on low-dose epinephrine infusions. With regards to dopamine, three of the nine centres involved in the working group use dopamine and each of those centres included centres with high and low rates of necrotising enterocolitis, while four of those five centres fed patients while on dopamine infusion. Instead of attempting to reach a goal nutritional feed rate while the patient is on vasopressor support, all centres with low rates of necrotising enterocolitis hold feeds at a lower-than-goal rate.

Transfusions

There is conflicting literature assessing the association between red blood cell transfusions and necrotising enterocolitis in the premature infant population.[20] The exact mechanism of transfusion-associated necrotising enterocolitis or acute gut injury is unknown and may result from an alteration in intestinal circulation [e.g. dynamic balance between vasoconstrictive and vasodilatory mechanisms (nitric oxide)] with subsequent tissue hypoxia and reoxygenation that may stimulate gut inflammation (predominately macrophages) and mucosal barrier damage.[21,22] Given this data, approaches to necrotising enterocolitis mitigation have included preventing intestinal circulatory stress during times of transfusions (holding feeds).

Within the working group, two of the nine centres withheld enteral feeds for the duration of the red blood cell transfusion in infants deemed to be at high risk of developing necrotising enterocolitis; both were centres with low incidence of necrotising enterocolitis. The effectiveness of withholding enteral feeding periprandial should be further evaluated in neonates with CHD during red blood cell transfusion. The role of red blood cell transfusion in necrotising enterocolitis aetiology for infants with complex CHD is unclear, but a conservative treatment approach may include withholding enteral feeds during transfusion.[23]

Anticoagulation

Data are limited linking anticoagulation strategy with necrotising enterocolitis in CHD, either as a risk or protective factor. No consensus regarding the use of systemic anticoagulation as a risk

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Table 1 Number of centres that feed while on vasoactive drips

Vasopressor	Number of centres that feed while on vasopressor	centres that feed on low-dose vasopressor and no other pressors	centres that feed on low-dose vasopressor and if all other pressors at low dose	centres that feed on vasopressor that is not dose dep
Vasopressin	5/9	2/5	3/5	0/5
Norepinephrine	3/9	1/3	2/3	0/3
Epinephrine	9/9	3/9	5/9	1/9
Dopamine	3/9	1/3	2/3	0/3
Milrinone	9/9	1/9	0/9	8/9

factor for necrotising enterocolitis was reached by the working group, owing to the multifactorial pathophysiology of necrotising enterocolitis in CHD. Several centres employ treatment guidelines that seek to lower the risk of systemic thromboembolism in general, which in theory ameliorates risk of mesenteric thrombosis and ischaemic gut injury.

The protocolized use of anticoagulation with therapeutic-dosed enoxaparin to prevent systemic thromboembolic disease in patients with indwelling venous central lines is employed at five of the nine centres. While this protocol is for thrombus prophylaxis, and independent of necrotising enterocolitis assessment, four of these five centres were high performing for necrotising enterocolitis. One centre holds anticoagulants when a patient has hematochezia or true necrotising enterocolitis given concern that it increases risk for worsened bleeding after an ischaemic bowel injury.

While all centres in our necrotising enterocolitis working group used laboratory values (e.g. low-molecular weight heparin assays) to assess the effectiveness of their anticoagulation; these levels are not used to guide feeding strategies. In all participating centres, routine anticoagulation was achieved with either heparin infusion or subcutaneous enoxaparin.

Management

Nil per os time and antibiotics

Despite necrotising enterocolitis in infants with cardiac disease having different pathophysiology and outcomes compared to necrotising enterocolitis in premature infants, there are currently no evidence-based treatment guidelines specific to this population and management plans are often extrapolated from the neonatal literature. "Suspected necrotizing enterocolitis" is as frequent as confirmed necrotising enterocolitis [24] and most centres will treat with broad-spectrum antibiotics with coverage for enteric Gram-negative and anaerobic organisms, as well as keeping the patients *nil per os* for 24–48 hours. For medical necrotising enterocolitis, treatment courses of both nil per os time and antibiotics are most often 7–10 days, which is consistent with neonatal guidelines, though more recent studies in premature infants suggest earlier refeeding may be beneficial.[25]

Surgical intervention

The proportion of patients with CHD and necrotising enterocolitis that progresses to surgical intervention for necrotising enterocolitis is estimated to be 11% among term infants and 21% among preterm infants.[2] Compared to premature patients, those with CHD-associated necrotising enterocolitis had lower incidences of perforation, need for bowel operation, strictures, need for a stoma, sepsis, and short bowel syndrome.[26] Conflicting data suggests that intestinal necrosis was present with greater frequency intraoperatively in infants with CHD and necrotising enterocolitis in comparison to infants without CHD.[27] This offers insight for earlier consideration for surgical intervention for patients with CHD.[28]

Surgical treatments for necrotising enterocolitis include bedside peritoneal drain placement or standard laparotomy. The primary goals of surgical intervention in necrotising enterocolitis are to control enteric spillage and/or resect necrotic intestine while maximising the length of viable intestine. Ideally, the optimal time for intervention would be after the onset of severe ischaemia but before intestinal perforation and/or progression of physiologic derangement.[29]

Determining optimal treatment strategy (surgical intervention vs. medical management) based on patient comorbidities remains controversial. Currently, the absolute criterion for operative intervention is evidence of pneumoperitoneum on radiography.[29] Clinical deterioration despite maximum medical therapy tends to guide surgical intervention.

Within the working group, the decision for surgical intervention is most often based on pneumatosis on abdominal X-ray or continued bleeding per rectum. Among our necrotising enterocolitis working group, with input from general surgeons, the greatest determinant of surgical versus medical necrotising enterocolitis, aside from clinical examination, is serial laboratories. Cohort studies have identified severe thrombocytopenia as an indicator of underlying intestinal necrosis [30] and proposed criteria for metabolic derangements including acidosis and low bicarbonate may predict timely operative intervention.[31] However, this has been limited in its generalizability, due to the lack of evidence from prospective or randomised trials.[29] Abdominal ultrasound was not suggested as a determinant for operating.

Formula/Nutrition

There is a lack of consensus about optimal nutrition progression for post-cardiac surgery patients post-hematochezia or necrotising enterocolitis. Limited data exist to guide how best to advance nutrition support for high-risk infants in the post-operative period, which results in wide variability between centres and possibly detrimental delays in nutrition advancement. For those at lower risk for necrotising enterocolitis, the decision to withhold feeds for cases of hematochezia must be balanced by considering the other aetiologies of hematochezia, such as cow's milk allergy.

After an occurrence of necrotising enterocolitis, some nutrition strategies employed are nil per os with parenteral nutrition, a switch in feed type (breastmilk/formula), selection of a different formula or fortifier (standard, semi-elemental, elemental), altered feeding tube placement (gastric/transpyloric), adjusted caloric density (at feed initiation and goal), change in feeding regimen (bolus/continuous), or modified goal for enteral volume (mL/kg). These modifiable factors are common practice despite no clear

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relationship between enteral feeding patterns (day of initiation of feeds, feeding velocity, caloric density, formula type) and necrotising enterocolitis incidence in the post-operative period.[32]

Without the evidence of how best to advance feeds after necrotising enterocolitis, an institution-based feeding protocol targeting calories with standard monitoring is a reasonable approach to reduce feeding complications and incidence of necrotising enterocolitis.[33,34] Balanced against a conservative approach of limiting feeds is the risk of excessive nutrition delay, as this may contribute to faltering growth. Utilising a nils per os approach requires incremental advancement of energy delivery (both parenteral and enteral nutrition), which can take several days, contributing to the accumulation of caloric deficits. Having a standardised feeding algorithm is one way to facilitate safe feeding advancement.

There is some literature to suggest that breastmilk can be a protective factor against necrotising enterocolitis among patients with CHD.[35,36] Within the necrotising enterocolitis working group, breastmilk was the preferred feeding. In addition, two centres with low incidence of necrotising enterocolitis have a post-op goal of only unfortified breastmilk. The use of elemental formulas was reported in two centres as a nutritional strategy after necrotising enterocolitis.[37] The osmotic loads of amino-acid-based formulas are significantly higher, which can contribute to feeding intolerance. The pros and cons of using these formulas for patients with CHD after necrotising enterocolitis need further investigation.

Deciding whether to remove mother's milk or modify her diet in the setting of suspected cow's milk intolerance or allergy remains challenging. There is no consistent guideline on how to handle this recurring issue and when to consider developing a feeding protocol.

Discussion

Necrotising enterocolitis is associated with significant morbidity among the CHD population and the incidence rates vary across necrotising enterocolitis among centres. Our collaboration was formed to better understand why variability exists and determine a consensus around practices that improve prevention, diagnosis, and managing care. Consensus was determined after a comprehensive multi-disciplinary review of the literature and review of existing feeding protocols among high- and low-performing centres.

In general, the most notable variability among centres is on the criteria for diagnosing necrotising enterocolitis, use of imaging modalities for diagnosis, and feeding decisions for patients on vasopressors or receiving transfusions. While there is a limited literature on near infra-red spectroscopy monitoring and necrotising enterocolitis rates in the CHD population, near infra-red spectroscopy monitoring is a feasible tool to guide feeding in high-risk patients. A comprehensive scoring tool that included biomarkers would be beneficial for diagnosing and grading necrotising enterocolitis as well as helping guide management.

Overall, there were more similarities than differences among centres within the collaborative between rates of feeds, caloric goals, types of formula, and nil per os timing. In terms of feeding protocols, consensus suggests that neonatology expertise should be considered when developing protocols, regardless of where patients are admitted.

Limitations

Limitations of this collaborative consensus include limited granularity given the substantial practice variation among centres. Given the small volume, comparative statistical analysis was not intended to be obtained between centres as this was not a hypothesis-generating study and sites were not case-mix or adjusted at the population level. Rather, the process disseminates a wide range of practice patterns across a variably successful range of sites, offering a template for future work as a prospective research or collaborative quality improvement opportunity. In the current state, the lack of prospective trials to guide management of necrotising enterocolitis makes it difficult to reach a consensus among all aspects of management and diagnosis of necrotising enterocolitis, and therefore, anecdotal practices were likely incorporated into the results of the survey. In some cases, consensus was built based on institutional practices, given the lack of literature that exists for some aspects (i.e. cardiac lesions). Given that the criteria for a centre to participate in this collaborative included having a statistically significant number of cases and fitting the profile of a high- or low-performing centre in terms of necrotising enterocolitis rates, selection bias could have confounded the results. In addition, because diagnostic work up of necrotising enterocolitis varied among centres, and while the Paediatric Cardiac Critical Care Consortium was used to determine high- and low-performing centres based on Paediatric Cardiac Critical Care Consortium definition of necrotising enterocolitis, some low-performing centres may have more sensitive criteria for necrotising enterocolitis than other centres, a limitation that is important to highlight for any future study.

Next steps

Although the results of this collaborative working group do not constitute a guideline, it provides critical background for clinicians to review and for future collaboration to create quality improvement initiatives to address necrotising enterocolitis rates and outcomes. Similar to previous work groups in the field, we hope that this would inform sites to convene and, utilising the modified Delphi method, procure consensus recommendations for diagnosis, management, and treatment recommendations for post-operative necrotising enterocolitis in patients with CHD. Given the variability that exists in necrotising enterocolitis, the next steps for a multicenter prospective quality project likely involve committing to a common pathway or at least diagnostic scoring mechanism, and rigorously tracking outcomes and practice patterns around that common pathway for collaborative learning. At the individual site level, the results of this collaboration can guide centres towards more aggressive or conservative approach to necrotising enterocolitis relative to their current practice.

Conclusion

We created a multi-centre collaborative to identify why variability exists and to determine a consensus that may guide improvement in necrotising enterocolitis rates. Substantial variability exists in diagnosing necrotising enterocolitis and feeding approaches for at-risk patients. Opportunities exist for future collaboration to create quality improvement initiatives to reduce necrotising enterocolitis rates and to guide management of necrotising enterocolitis in patients with CHD.

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