INTESTINAL LOCATION OF NECROTIZING ENTEROCOLITIS AMONG INFANTS WITH CONGENITAL HEART DISEASE

Poster Contributions
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Background: Infants with congenital heart disease (CHD) are at increased risk for developing necrotizing enterocolitis (NEC). Pathophysiology of NEC among infants with CHD is largely unknown although an alteration in intestinal blood flow remains a leading theory. Previous reports of NEC in non-CHD patients have documented primarily small intestinal involvement in preterm infants (< 37 weeks) versus primarily colonic involvement in full-term infants (> 37 weeks). Predominant location of NEC in CHD patients has not been previously described. Goal of this study was to determine if differences exist in the location of NEC in infants with CHD versus those without CHD.

Methods: Retrospective cohort study was performed on patients who underwent a surgical exploration for NEC between 1/2000 to 12/2011. The presence or absence of CHD was noted via echocardiograms. Surgical and/or pathology reports were reviewed to identify the location of NEC. NEC involvement was separated into small intestine, large intestine, ileocecal, NEC totalis, and multi-segment. T-tests and Chi square were used for analysis. P < 0.05 was considered significant.

Results: One hundred sixty-seven patients met inclusion criteria (149 non-CHD, 18 CHD). Non-CHD patients had significantly lower gestational age (28 + 4 vs. 34 + 5 weeks), birth weight (1178 + 58 vs. 2106 + 252 grams), and age at surgery (21 + 1 vs. 40 + 10 days) compared to CHD patients. CHD infants had a higher percentage of mortality (72% vs 29%). There was no significant difference in location of NEC between non-CHD and CHD patients with the predominant location being small intestine in both (31% vs 33%, respectively). In addition, there was no significant difference in location of NEC between preterm non-CHD patients (n = 137) and full-term CHD patients (n = 9) with the small intestine again being the primary site (34% vs. 44%, respectively).

Conclusions: Despite differences in gestational age between non-CHD and CHD patients, location of NEC in these infants did not differ. This suggests that the pathophysiology of NEC in the CHD population might be more similar to that of the preterm population than previously believed.