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Is routine preoperative screening echocardiogram indicated in all children with congenital duodenal obstruction?

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Abstract

Introduction—Congenital duodenal obstruction (DO) is frequently associated with congenital heart disease (CHD). Operative repair of DO is often postponed until an echocardiogram is completed, which may result in unnecessary delays. We aimed to identify and characterize CHD in children with DO to determine if appropriately selected patients could forego preoperative echocardiogram.

Methods—A two-center retrospective review of all infants with DO undergoing operative repair with completed echocardiograms was included (2003–2011). Demographics, co-morbid

conditions, clinical exam findings, radiologic imaging, and need for cardiac surgery were recorded.

Results—67 children were identified. 47 (70.1 %) had CHD on echocardiogram of which 19 (40.5 %) had significant CHD. Children without clinical findings, abnormalities on physical examination, and/or abnormal chest x-ray were unlikely to have CHD; i.e., no asymptomatic child had significant CHD. Sensitivity and specificity of clinical findings, physical exam, and/or chest x-ray for significant CHD were 100 % (95 % CI 0.79–1.0) and 37.5 % (95 % CI 0.24–0.53), respectively, for major CHD and 87.2 % (0.74–0.95) and 60 % (0.36–0.80) for any CHD.

Conclusion—Careful clinical assessment, evaluation with pulse oximetry, and chest x-ray may be sufficient to exclude significant CHD in children with DO. Identifying children at low risk for cardiac lesions may prevent unnecessary delays to operative intervention and may limit medical expenses.

Keywords

Congenital duodenal obstruction; Duodenal atresia; Congenital heart disease; Echocardiogram

Introduction

Congenital duodenal obstruction (DO) is a common cause of congenital intestinal obstruction with an estimated incidence of 1 in 6,000 to 1 in 10,000 live births [1, 2]. It is frequently associated with trisomy 21 (35–46 %), cardiac anomalies (31–39 %), malrotation (10 %), additional intestinal atresias (7 %), vertebral anomalies (5 %), tracheo-esophageal fistula (5 %), renal anomalies (5 %), and anorectal malformations (3 %) [2–4]. Of these conditions congenital heart disease (CHD) is thought to present the greatest morbidity [5]. As a result, operative repair for DO is often delayed until echocardiogram is completed even in the setting of normal clinical findings.

Recent reports suggest that routine preoperative echocardiogram in all neonates with conditions predisposing for CHD may be unnecessary as the operative plan is seldom altered [6]. Studies have indicated that cardiac interventions are rarely needed among children with DO that is not associated with trisomy 21 [2]. We hypothesized that children with DO who have no clinical or radiologic features suggestive of CHD would not have significant underlying cardiac disease. We aimed to evaluate need for preoperative echocardiogram in all patients with congenital duodenal obstruction.

Methods

A retrospective chart review of all infants with a diagnosis of congenital DO undergoing operative repair at two major academic centers between January 2003 and January 2011 was performed. Relevant patient demographics, co-morbid conditions, operative details, and outcomes were recorded. Primary outcome was presence of any significant cardiac lesion. Institutional review board approval was obtained at both institutions.

Patient identification and selection

Patients were identified through hospital registries. ICD-9 codes were utilized to identify children with a discharge diagnosis of intestinal atresia (Code 751.1) or congenital anomalies of the pancreas (Code 751.7). Operative reports and discharge summaries were reviewed to exclude patients with jejunal, ileal, or colonic atresia, as well as pancreatic anomalies not leading to duodenal obstruction. In addition, surgical case logs were reviewed for procedure description of “duodenoduodenostomy or duodenojejunosomy.” All patients with evidence of congenital DO (duodenal atresia, annular pancreas, duodenal web/stenosis) were included. Patients without echocardiogram and those who underwent operative repair for DO at a referring facility were excluded from analysis.

Patient evaluation

Patient history including gestational age, weight, and age at operation were recorded. The preoperative clinical history was reviewed in detail and any reports of hypoxia and/or desaturation, auscultative murmur, hepatosplenomegaly, abnormal pulse exam, tachypnea, intercostal recession or retraction, desaturation, precordial thrill, and cardiac-induced respiratory embarrassment were recorded. Chest x-rays were reviewed to evaluate for evidence of cardiomegaly, pulmonary vascular congestion, interstitial edema, situs abnormalities, or congenital anomalies in the preoperative period. Pulse oximetry was reviewed to evaluate for cyanosis, which has previously shown to be useful in the screening for CHD [7]. Other clinical factors including need for ventilation, prostaglandin therapy, and use of inotrope or vasopressor therapy were recorded. Lastly, the age at DO repair and need for cardiac surgery during any admission were assessed.

Echocardiogram and categorization of cardiac defects

All echocardiograms were reviewed and anatomic details recorded. Children were considered to have significant cardiac disease if they had evidence of a cyanotic heart lesion (e.g. truncus arteriosus), a ductal dependent lesion (e.g. transposition of the great vessels), a lesion that required prostaglandin therapy, or a lesion requiring cardiac surgery. Non-significant defects were those that did not alter the surgical course or did not require cardiovascular intervention.

Comparative groups

To determine if clinical findings were sufficient to identify CHD, we compared those with abnormal clinical findings (abnormal cardiac, respiratory, chest x-ray findings) to those without and compared clinical differences between those with and without significant CHD. Additional analysis was performed to determine the specificity, sensitivity, positive predictive value (PPV), and negative predictive value (NPV) of exam findings.

Statistical analysis

All data were recorded into a standardized spreadsheet (Microsoft Excel®, Seattle, WA, USA). Numerical variables were summarized by mean and standard deviation (SD) or median and interquartile range (IQR). Binary variables were summarized by frequency and percent. Numerical variables were compared across groups by the Wilcoxon rank sum test.

Categorical variables were compared across groups by Fisher's exact test. Our primary outcome measure was presence of CHD. A p value > 0.05 per comparison was considered statistically significant.

Results

Sixty seven children (37-boys, 30-girls) with DO met inclusion criteria during the study period. Eight children were excluded; seven underwent operative repair at a referring facility and one child did not undergo operative repair secondary to significant associated co-morbidities. The median gestational age (EGA) was 36 weeks with a median weight of 2.46 kg. Forty seven (70.1 %) children had an associated disorder thought to place them at high risk for CHD: trisomy 21 ($n = 32$), heterotaxy syndrome ($n = 8$), and VACTERL complex ($n = 7$). Forty three (64.2 %) manifested findings suggestive of CHD including murmur (49.3 %), desaturation (29.8 %), cyanosis (13.4 %), tachypnea (13.4 %), CHF (11.9 %), hepatosplenomegaly (10.5 %), and abnormal pulse exam (7.5 %). Thirty three (49.3 %) had abnormal chest x-ray findings (cardiomegaly, pulmonary edema, etc.) and several children required ventilatory support (22.4 %) as well as vasoactive medications (17.9 %). Echocardiogram revealed significant CHD in 28.3 % of children and 22.4 % required cardiac surgery (Tables 1, 2).

Of the 47 identified defects on echocardiogram 19 were significant and 28 were not. Most common significant defects included AV canal ($n = 6$), single ventricle physiology ($n = 5$), and Tetralogy of Fallot ($n = 4$). Common minor defects included ASD $\pm$ PDA ($n = 10$), PDA ($n = 10$), and VSD $\pm$ PDA ($n = 6$) (Table 2). Presence of dextrocardia or situs inversus was not considered CHD without associated structural heart defects.

Overall, 49 (73 %) of children demonstrated abnormal clinical or chest x-ray findings. These children were more likely to require vasoactive medical therapy, ventilatory support, have underlying CHD (significant or non-significant), and cardiac surgery. Importantly, clinical and chest x-ray findings were able to identify all cases of significant CHD, and presence of a normal clinical exam and chest x-ray did not exclude any children with significant CHD with median follow-up time of 5.0 years (Table 3).

When comparing significant to non-significant CHD, abnormal clinical findings were much more prevalent in the significant cohort, with all patients demonstrating abnormal clinical or chest x-ray findings. Specifically, these patients had higher incidence of cyanosis (42.1 vs. 2.1 %), hepatosplenomegaly (31.6 vs. 2.1 %) and evidence of congestive heart failure (31.6 vs. 4.2 %) on exam, and the majority had cardiac murmurs (94.7 vs. 31.2 %). These patients were also more likely to require cardiovascular support (36.8 vs. 10.4 %) and nearly 95 % (vs. 31.3 %) had abnormal chest x-ray findings including cardiomegaly ($n = 16$), pulmonary congestion ($n = 8$), interstitial edema ($n = 1$), and congenital diaphragmatic hernia ($n = 1$). This group was also more likely to require cardiac surgery (79 vs. 0 %). Despite increased prevalence of co-morbid conditions in children with significant CHD, difference in time to DO repair was not significant (7 vs. 4.5 days, $p = 0.06$) (Table 4).

The sensitivity, specificity, PPV, and NPV of cardiac, respiratory, and chest x-ray findings in detecting CHD on echocardiogram were determined to assess ability to discern CHD. The combined sensitivity of these exam findings was 100 % (95 % CI 0.79–1.0) with a specificity of 37.5 % (95 % CI 0.24–0.53), and PPV and NPV of 73.1 and 38.8 % for significant CHD, respectively. For all cases of CHD, the sensitivity was 87.2 % (0.74–0.95) and specificity was 60 % (0.36–0.80) (Table 5).

Discussion

Our study demonstrates that in the absence of clinical findings, children with DO are unlikely to have significant CHD that necessitates precluding or delaying plans for establishment of intestinal continuity. Despite the relatively high frequency of CHD in this study, no children were found to have significant underlying CHD requiring future cardiac surgery in the absence of an abnormal clinical picture at a median follow-up of 5 years.

Few studies have evaluated the role of routine physical examination and its ability to detect underlying CHD in high-risk children. A recent multicenter study evaluating screening methods in over 20,000 newborn infants 35 weeks gestation or older found that use of pulse oximetry with clinical exam will identify most critical or significant CHD [7]. Others have reported equivalent detection rates of CHD with the use of clinical exam in combination with pulse oximetry compared to exam with adjunctive echocardiogram at decreased economic cost [8, 9]. A recent study evaluated 86 neonates with congenital diseases commonly associated with CHD, including esophageal atresia, omphalocele, and anorectal malformations. Similar to our study, they were able to demonstrate that normal exam findings in conjunction with chest x-ray effectively excluded presence of significant CHD in 100 % of cases [6].

Nonetheless, others have recommended routine echocardiogram in all children with congenital DO due to the relative frequency of CHD and semi-elective nature of duodenal atresia repair [10]. Further, at least one study reported increased detection of CHD by echocardiogram compared to physical exam including lesions that required cardiac surgery [11]. These papers highlight the need for cautious and thorough evaluation of these patients prior to surgical intervention to identify children at high risk for CHD.

Keckler and colleagues previously evaluated children with or without trisomy 21, a known risk factor for CHD, in the setting of DO. They found children with DO without trisomy 21 to have similar risk of CHD requiring cardiac surgery as children without DO [2]. While our study did not determine trisomy 21 to be a predictor of CHD in children with DO we, like NASR and colleagues, did find those without clinical findings to be at low risk of CHD [6]. Identifying patients at low risk of CHD is needed to develop algorithms to avoid unnecessary costs of care, delay of surgical intervention, and to minimize risk of inadequate evaluation. This may diminish NICU costs, which average more than \$1,400 per day and reduce the incremental cost of routine screening echocardiogram [8, 12].

Our study has a number of limitations. Despite excellent sensitivity for significant CHD, our data demonstrates relative poor specificity for the evaluation of CHD amongst all children

with duodenal atresia, which is something that previous authors have demonstrated when screening large populations of trisomy 21 [13]. Further, our study is retrospective in nature and is limited by the accuracy of the medical chart and the description of clinical findings. Other relevant clinical factors and diagnostic tools, including use of EKG, may have relevance and were not specifically included in this study. There may be selection bias given children without echocardiograms were excluded from this study; thereby increasing the proportion of children with significant co-morbidity. In addition, other children may have had normal prenatal echocardiogram precluding repeat postnatal echocardiogram. Despite a median followup time of 5.0 years, it is possible that some children may have undergone cardiac surgery following conclusion of our study and could have been incorrectly classified as having non-significant CHD. Our centers are quaternary care centers with one center having no in-born population and many children present with advanced disease and/or complicated co-morbid conditions, which may not be representative of populations seen at other centers. This may account for the high rate of cesarean births (69 %) as well as frequency of CHD (70.1 %). Lastly, it is impossible to determine if delays to surgery were directly related to echocardiography and not other perinatal issues or operating room availability.

Conclusion

Identifying children at low risk for significant cardiac lesions may prevent unnecessary delays to operative intervention and may limit excess medical expenses. As demonstrated by our study and others, careful clinical assessment, evaluation with pulse oximetry, and simple chest x-ray may be sufficient to exclude significant underlying CHD. As such, one general approach promulgated by the CHD working group for use in well infant and intermediate nurseries may warrant prospective evaluation in conjunction with careful physical exam and chest x-ray in the DO population. They recommend pulse oximetry screening after 24 h of life to allow for transition from fetal circulation. An oxygen saturation >90 % warrants further work-up for CHD while a saturation [95 % with >3 % differential between the right hand and foot is considered a negative work-up. Children with intermediate readings undergo repeat evaluations [14]. Pending prospective evaluation for the role of echocardiography in children with DA, an echocardiogram should be obtained when available but should not delay operative intervention in the stable patient with normal clinical findings.

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Table 1

Patient demographics and diagnoses

Demographics	
Gender (% male)	55 %
Weight (kg)	2.46 (2.1, 2.8) ^a
Estimated gestational age (weeks)	36 (33.5, 37) ^a
Median follow-up (years)	5.0 (3.2, 6.6) ^a
<i>Delivery</i>	
C-section	68.9 %
<i>Ethnicity</i>	
Hispanic	60%
Non-hispanic	40%
<i>Race</i>	
Asian	10.4 %
Black	5.9 %
White	22.4 %
Other	61.2 %
<i>Primary diagnosis</i>	
Duodenal atresia	55 %
Annular pancreas	22%
Duodenal web	12%
Duodenal stenosis	9%
Predoduodenal portal vein	1.5 %
<i>Secondary diagnosis</i>	
CHD (any)	70.1 %
Down syndrome (trisomy 21)	43.2 %
Malrotation	31.3 %
Significant CHD	28.3 %
Heterotaxy syndrome	11.9 %
Multiple GI atresias	10.5 %
VACTERL complex	7.4 %
Esophageal atresia (±TEF)	4.4 %
Situs inversus	6%
CDH	3 %
Biliary atresia	1.5 %
Abdominal wall defect	1.5 %
Imperforate anus	1.5 %
<i>Procedures</i>	
Age at DO repair (days)	6 (3, 22) ^a
Duodenoduodenostomy	85 %
Duodenojejunostomy	15 %

Demographics	
Required cardiac surgery	22.4 %

CDH congenital diaphragmatic hernia, *CHD* congenital heart disease

^aMedian (IQR)

Table 2

Clinical and chest x-ray findings

Exam findings	%
Murmur	49.3
Desaturation	29.8
Cyanosis	13.4
Tachypnea	13.4
Congestive heart failure	11.9
Hepatosplenomegaly	10.5
Intercostal retractions	7.5
Abnormal pulse exam	7.5
Clinical factors	
Ventilated	22.4
Inotrope/vasopressor	17.9
Ductal dependent lesion	9
Prostaglandin therapy	7.5
Radiologic findings	
Abnormal chest x-ray	49.3
Cardiomegaly	35.8
Pulmonary congestion	16.4
Interstitial edema	10.4
Congenital diaphragmatic hernia	3.0
Echocardiogram	
<i>Significant CHD</i>	28.3
AV canal	8.9
Single ventricle physiology	7.5
Tetralogy of fallot	5.9
Large VSD	3
Caval anomalies	3
Patent ductus arteriosus requiring intervention	3
Hypoplastic aortic arch	1.5
<i>Minor CHD</i>	41.8
ASD ± PDA	14.9
PDA	14.9
VSD ± PDA	8.9
SVC anomalies	3
ASD + VSD	1.5

ASD atrial septal defect, VSD ventricular septal defect, PDA patent ductus arteriosus, AV canal atrioventricular Canal, SVC superior vena cava

Table 3

Symptomatic vs. asymptomatic children with DO

	Symptomatic ^a (n = 49)	Asymptomatic (n = 18)	P
Gender (% male)	53.1	61.1	0.56
EGA (median)	36 (34, 37)	35 (33, 36.8)	0.27
Age at repair (median)	6 (3.5, 55)	5 (3, 17.5)	0.43
Down (%)	50	27.8	0.12
VACTERL (%)	10.2	0	0.16
Heterotaxy (%)	16.3	0	0.07
Inotrope/vasopressor (%)	24.5	0	0.02
Ventilated (%)	28.6	5.6	0.04
Any CHD (%)	83.7	33.3	<0.001
Significant CHD (%)	38.8	0	0.002
Cardiac surgery (%)	30.6	0	0.008

EGA estimated gestational age

^aSymptoms: abnormal cardiac, pulmonary, or chest x-ray findings

Table 4

Significant CHD vs. no significant CHD in DO children

	Sig. CHD (n = 19)	No sig. CHD^a (n = 48)	P
Gender (% male)	42.1	60.4	0.19
C-section delivery	47.1 %	25 %	0.13
Birth weight (kg)	2.5 ± 0.66	2.42 ± 0.65	0.61
Estimated gestational age	35.2 ± 2.6	35.8 ± 2.4	0.43
Age at DA repair	7 (2–207)	4.5 (0–485)	0.06
Age at echo	1.1 ± 1.2	2 ± 2.7	0.29
Associated anomalies (%)			
Down syndrome	47.4	41.7	0.79
VACTERL complex	5.3	8.3	1.0
Heterotaxy	26.3	6.3	0.04
Malrotation	47.4	25	0.09
Clinical findings (%)			
Dysmorphic features or associate anomaly (e.g. imperforate anus, trisomy 21, etc)	78.9	56.3	0.10
Cyanosis	42.1	2.1	<0.001
Congestive heart failure	31.6	4.2	0.005
Murmur	94.7	31.2	<0.001
Hepatosplenomegaly	31.6	2.1	0.002
Abnormal pulse exam	15.8	4.2	0.13
Tachypnea	15.8	12.5	0.71
Intercostal retraction	15.8	4.2	0.13
Vasopressor/inotrope requirements (%)	36.8	10.4	0.03
Abnormal CXR findings (%)	94.7	31.3	<0.001
Cardiac surgery (%)	79	0	<0.001

EGA estimated gestational age, CXR chest x-ray

^aIncludes those without CHD and those with minor CHD

Table 5

Sensitivity, specificity, PPV, and NPV for clinical and/or radiology findings in detecting cardiac lesions

Cardiac lesion	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
<i>Radiologic evaluation</i>				
Major	94.7	68.8	54.5	97.1
Major and minor	61.7	80	87.8	47.1
<i>Cardiac evaluation</i>				
Major	100	64.5	52.8	100
Major and minor	70.1	70.2	91.7	54.8
<i>Respiratory evaluation</i>				
Major	42.1	70.8	36.4	75.6
Major and minor	38.3	80	32.8	67.2
<i>Combined evaluations</i>				
Major	100	37.5	73.1	38.8
Major and minor	87.2	60	83.7	66.7