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Original Article

Predictors of enteral tube dependence in pediatric esophageal atresia

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SUMMARY. **Children with esophageal atresia (EA) may require enteral tube feedings in infancy and a subset experience ongoing feeding difficulties and enteral tube dependence. Predictors of enteral tube dependence have never been systematically explored in this population. We hypothesized that enteral tube dependence is multifactorial in nature, with likely important contributions from anastomotic stricture. Cross-sectional clinical, feeding, and endoscopic data were extracted from a prospectively collected database of endoscopies performed in EA patients between August 2019 and August 2021 at an international referral center for EA management. Clinical factors known or hypothesized to contribute to esophageal dysphagia, oropharyngeal dysphagia, or other difficulties in meeting caloric needs were incorporated into regression models for statistical analysis. Significant predictors of enteral tube dependence were statistically identified. Three-hundred thirty children with EA were eligible for analysis. Ninety-seven were dependent on enteral tube feeds. Younger age, lower weight *Z* scores, long gap atresia, neurodevelopmental risk factor(s), significant cardiac disease, vocal fold movement impairment, and smaller esophageal anastomotic diameter were significantly associated with enteral tube dependence in univariate analyses; only weight *Z* scores, vocal fold movement impairment, and anastomotic diameter retained significance in a multivariable logistic regression model. In the current study, anastomotic stricture is the only potentially modifiable significant predictor of enteral tube dependence that is identified.**

KEY WORDS: **esophageal stricture, feeding difficulties.**

INTRODUCTION

Anastomotic stricture is widely accepted as a cause of dysphagia and feeding difficulties in patients with a history of esophageal atresia (EA), though the degree to which anastomotic stricture versus other clinical factors contributes to feeding difficulties is unclear. There is no consensus around any age-based goals for esophageal anastomotic diameter, and it is unclear if severity of narrowing correlates with severity of feeding difficulty symptoms.[1] We were interested in understanding the degree to which severity of stricture contributes to significant feeding difficulties, as measured by partial or full enteral tube dependence. We hypothesized that enteral tube dependence in patients with EA is likely multifactorial in nature, with likely important contributions from stricture. In this prospectively collected study of endoscopies in 330 patients with repaired EA, we examined the relative importance of stricture and other clinical predictors in enteral tube dependence.

METHODS

This study was approved by our institutional review board. We analyzed cross-sectional data from a prospectively collected database of all endoscopies performed by our tertiary care international referral center for EA from August 2019 through August 2021. All patients with a history of EA were eligible for inclusion for this study. Patients with jejunal ($n = 25$) or colonic ($n = 2$) interpositions, esophageal discontinuity ($n = 6$), or unrepaired tracheoesophageal fistula at the time of evaluation ($n = 8$) were excluded. Data collected included demographic and clinical characteristics, feeding status at the time of endoscopy, and detailed endoscopic information. Clinical, feeding, and endoscopic information were recorded for the time point of the patient's most recent endoscopy within the study period if more than one endoscopy was performed. Feeding status was summarized as a feeding score based on a modified Functional Oral Intake Scale

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Table 1 The modified Functional Oral Intake Scale

Score	Feeding status description
1	Enteral tube dependent for nutrition with no attempts to feed by mouth
2	Enteral tube dependent for nutrition with inconsistent attempts to feed by mouth
3	Partially enteral tube dependent for nutrition (receiving any prescribed amount of tube feeds per day) with consistent (at least daily) successful attempts to feed by mouth
4	Minimal to no tube feeds but require thickened liquids due to aspiration
4.5	Minimal to no tube feeds, no thickened liquids, but mash/blending solids or using high calorie formula by mouth to supplement
5	No tube feeds, no thickened liquids, eats all age appropriate solid foods with minor accommodation (e.g. cutting food into smaller pieces, or requires sips of liquids in between solids)
6	No tube feeds, no thickened liquids, eats all age-appropriate solids without special accommodation

The modified Functional Oral Intake Scale assessed by clinical interview of the patient and/or caregiver at the time of presentation for endoscopy.[2,3] Enteral tube refers to any tube-assisted method of feeding including gastrostomy, gastrojejunostomy, jejunostomy, nasogastric tube, or nasojejunal tube.

(MFOIS)[2,3] as described in Table 1. Enteral tube dependence was defined as an MFOIS score of 1–3, and scores of 4–6 were considered reflective of oral feeding independence.

Clinical factors that were hypothesized or known to contribute to feeding difficulties through esophageal dysphagia, oropharyngeal dysphagia, or other difficulties in orally meeting caloric needs were collected from the database. Specifically, data regarding esophageal comorbidities including esophagitis (eosinophilic or erosive peptic), co-existing congenital esophageal stenosis, and fundoplication were collected. Extra-esophageal comorbidity data were also collected, including neurodevelopmental comorbidities, severe unrepaired tracheomalacia, and significant cardiac defects. Neurodevelopmental risk factors included genetic syndromes (e.g. CHARGE syndrome, trisomy 21), history of prematurity less than 34 weeks gestation, and unrepaired Chiari malformations. Severe tracheomalacia was defined as the combination of severe respiratory symptoms (e.g. blue spells, recurrent pneumonias, dependence on positive pressure ventilation) and significant tracheal collapse identified by dynamic airway assessment during direct laryngoscopy and bronchoscopy that led to recommendation for surgical correction of tracheomalacia at our institution. Cardiac defects included hemodynamically significant atrial or ventricular septal defects, double outlet right ventricle, hypoplastic ventricle, tetralogy of Fallot, transposition of the great arteries, pulmonary vein stenosis, absent pulmonary valve, and pulmonary hypertension. Clinical factors thought to contribute to increased risk of aspiration were also collected, including the presence of unrepaired or residual type 1 or greater laryngeal clefts and vocal fold movement impairment detected by flexible fiberoptic nasolaryngoscopy (which are routinely performed pre- and postoperatively for patients undergoing esophageal surgery at our institution, regardless of symptoms, due to risk of intraoperative recurrent laryngeal nerve injury). Vocal fold movement impairments included unilateral or bilateral hypomobility or immobility of the vocal cord(s). Modified barium swallow (MBS) results were also collected if ever performed and were positive for aspiration risk if aspiration and/or deep penetration was observed during the study. Demographic data including age, weight *Z* score at the time of endoscopy, and type of esophageal atresia were also collected for analysis. Weight *Z* score was defined as the number of standard deviations above or below the mean weight for age as calculated using the world health organization (WHO) child growth standards for children $\leq$2 years old, centers for disease control and prevention (CDC) growth standards for children $>$2 years old, or condition-specific growth standards (e.g. Trisomy 21 growth standards) as appropriate. Long gap atresia was defined functionally as any EA type not able to be repaired primarily with one surgical procedure.

All endoscopies were performed by one of three pediatric gastroenterologists (JY, PN, and MM) with subspecialization in the management of esophageal atresia using either Olympus XP-190N or Olympus GIF-H190 under general anesthesia. Measurements of esophageal diameter at the surgical esophageal anastomosis were performed by introducing the biopsy forceps through the working channel of the endoscope and using known dimensions of the forceps as a visual reference. Approximately half of the cases also had an intraoperative esophagram performed during the endoscopy, which permitted validation of the endoscopically estimated measurement of esophageal dimensions by comparison with the dimensions of the endoscope and radiographic ruler.

Statistical analysis

Within the feeding tube dependent and the oral feeding groups, continuous data are presented as medians and interquartile ranges and categorical data are presented as frequencies and percentages. Univariate and multivariable logistic regression analyses were implemented to identify independent predictors of feeding tube dependence. Variables with

Table 2 Univariate logistic regression analysis of feeding tube dependence versus oral feeding

Variable	Feeding tube dependence ($n = 97$)	Oral feeding ($n = 233$)	Odds ratio for feeding tube dependence	95% CI	*P* value
Age at endoscopy (months)	16 (9–49)	54 (24–97)	0.99	(0.98, 1.00)	**<0.001***
Weight *Z* score	−0.98 (−3.32, 0.64)	−0.42 (−1.32, 0.3)	0.69	(0.56, 0.86)	**0.001**
Gender (male)	57 (59%)	132 (57%)	0.92	(0.57, 1.48)	0.724
Long gap	46 (47%)	79 (34%)	1.76	(1.09, 2.85)	**0.022**
Neurodevelopmental risk	64 (66%)	71 (30%)	4.43	(2.67, 7.32)	**<0.001***
Cardiac defect	52 (54%)	95 (41%)	1.68	(1.04, 2.70)	**0.033**
Laryngeal cleft†	18 (19%)	46 (20%)	0.93	(0.51, 1.70)	0.804
MBS‡	35 (74%)	75 (64%)	1.67	(0.79, 3.56)	0.182
Vocal fold movement impairment§	28 (54%)	22 (28%)	2.97	(1.42, 6.19)	**0.004***
Tracheomalacia	9 (9%)	24 (10%)	0.89	(0.57, 1.48)	0.778
Fundoplication	21 (22%)	35 (15%)	1.56	(0.86, 2.85)	0.146
Congenital esophageal stricture	9 (9%)	17 (7%)	1.30	(0.56, 3.02)	0.544
Esophagitis	2 (2%)	16 (7%)	0.29	(0.06, 1.27)	0.099
Esophageal diameter	10 (8–12.5)	15 (12–16)	0.76	(0.71, 0.82)	<0.001*

Univariate logistic regression analysis of clinical predictors of feeding tube dependence. †Unrepaired or residual laryngeal cleft of any grade. ‡MBS with aspiration or deep penetration; MBS results were available for 165 patients. §Vocal fold movement impairment refers to unilateral or bilateral hypomobility or immobility of the vocal cord(s); vocal cord mobility assessments were available for 130 patients. See 'Methods' section for full definitions of each clinical variable listed. Asterisk (*) and bold face type denote statistical significance. CI, confidence interval.

$P < 0.05$ upon univariate analysis were included in multivariable modeling. Regression analysis results are presented as adjusted odds ratios, 95% confidence intervals, and *P* values. Stratified by age group, receiver operating characteristic curve analyses were implemented to determine the optimal cutoff value for esophageal diameter in distinguishing between patients with versus without feeding tube dependence. The optimal cutoff was identified as the value that maximizes Youden's J index, to maximize the sum of sensitivity and specificity. All statistical analyses were performed using Stata (version 16.1, StataCorp LLC, College Station, Texas). A two-tailed $P < 0.05$ was considered as statistically significant.

RESULTS

A total of 330 unique EA patients (125 long-gap EA) met inclusion criteria for analysis. Ninety-seven patients (29%) were dependent on partial or full enteral tube feeds. Patients with feeding tube dependence were significantly younger than independent oral feeders (16 vs. 54 months, $P < 0.001$), had lower median weight *Z* scores (−0.98 vs. −0.42, $P = 0.001$), and were significantly more likely to have long-gap atresia (47% vs. 34%, $P = 0.022$) (Table 2). Children with feeding tube dependence were also more likely to have neurodevelopmental risk factor(s) (66% vs. 30%, $P < 0.001$) or cardiac disease (54% vs. 41%, $P = 0.033$). Vocal fold movement impairments were also significantly associated with enteral tube dependence (54% tube dependent vs. 28% oral feeders, $P = 0.004$); MBS identifying aspiration and presence of laryngeal cleft were not significantly associated with enteral tube dependence.

Severity of esophageal anastomotic stricture was significantly associated with feeding tube use, with smaller anastomoses more likely to be seen in feeding tube dependence (median 10 mm vs. 15 mm, $P < 0.001$). No other esophageal factor (esophagitis, fundoplication, congenital esophageal stenosis) was significantly associated with feeding tube dependence.

A multivariable logistic regression analysis was performed using significant univariate predictors and identified only weight *Z* score, vocal fold movement impairment, and esophageal diameter as significantly independently associated with feeding tube dependence when adjusted for age, neurodevelopmental risk, long-gap EA status, and cardiac disease (Table 3).

As esophageal diameter is expected to increase with age, we attempted to identify statistically optimal diameter cutoffs to discriminate between patients who were dependent on enteral tube feeds versus independent oral feeders (Table 4). Most cutoffs were statistically significant, though limited in clinical utility by poor to fair sensitivity and specificity.[4]

DISCUSSION

Despite most EA patients eventually achieving full oral feeding, many experience ongoing dysphagia and a subset experience prolonged dependence on enteral tube feeds.[5–7] The current study is the largest to our knowledge to systematically explore clinical factors and their relative importance in contributing to feeding difficulties leading to enteral tube dependence in a large cohort of over 300 children with EA.

Consensus guidelines state that there is no correlation between severity of anastomotic narrowing

Table 3 Multivariable logistic regression analysis of feeding tube dependence

Covariate	Adjusted odds ratio	95% CI	*P* value
Age at endoscopy (months)	0.99	(0.97, 1.01)	0.351
Weight *Z* score	0.64	(0.42, 0.99)	**0.046***
Long gap EA	1.34	(0.54, 3.32)	0.533
Neurodevelopmental risk	1.54	(0.61, 3.87)	0.356
Cardiac defect	1.33	(0.57, 3.10)	0.502
Vocal fold movement impairment	3.52	(1.46, 8.49)	**0.005***
Esophageal diameter	0.82	(0.71, 0.94)	**0.005***

Multivariable logistic regression analysis of significant univariate predictors of feeding tube dependence. Asterisk (*) and bold face type denote statistical significance. CI, confidence interval.

Table 4 Age-based esophageal anastomotic diameters with increased likelihoods of tube dependence

Age	Diameter (mm)	Sensitivity	Specificity	Odds ratio	95% CI	*P* value
<9 months	<9	74% (17/23)	67% (10/15)	5.7	(1.37, 23.5)	**0.017***
9–18 months	<11	66% (19/29)	68% (17/25)	4	(1.30, 12.6)	**0.016***
19–36 months	<13	73% (11/15)	56% (27/48)	3.5	(0.98, 12.7)	0.053
>36 months	<14	53% (16/30)	88% (128/145)	8.6	(3.58, 20.7)	<**0.001***

Statistically optimal cutoffs that increase the likelihood of tube dependence, by age. Age cutoffs were chosen to approximate anticipated developmental progression of feeding skills from liquids to purees, to small bites, and finally to larger bites of food. Asterisk (*) and bold face type denote statistical significance. CI, confidence interval.

and severity of feeding difficulties, though the evidence supporting this statement is limited to expert opinion.[1] We find in the current study that stricture severity is in fact significantly associated with feeding tube dependence, contrary to current dogma. Esophageal diameter remained a significant predictor of feeding tube dependence even after adjusting for confounders such as age, nutritional status, gap length, cardiac disease, and neurodevelopmental risk factors. We were particularly interested in the role of stricture severity in feeding difficulties, as stricture is a potentially modifiable risk factor via endoscopic treatment.

Our findings have potential critical implications for management of pediatric esophageal strictures. Many centers do not treat strictures until they become symptomatic, largely citing the results of a single small, retrospective study of routine dilations versus symptom-based dilations in EA patients where there were significantly fewer dilations in the symptom-based dilation group with no difference in feeding outcomes.[8] However, this study was limited to a single institution's experience with a cohort of mostly short-gap EA patients where routine dilations were started in early infancy and repeated until stable achievement of an esophageal diameter of 10 mm, which may have led to an excessive number of dilations in the routine group. On the other hand, in our clinical experience, waiting to dilate until the development of dysphagia or feeding difficulties in an infant who is taking primarily a liquid diet may sometimes delay treatment of a stricture until it has advanced and scarred to a considerable degree, becoming potentially more difficult to successfully treat endoscopically.[9]

In addition, one source of confusion in understanding predictors of feeding difficulties in EA is the subjective nature and variability in patient perception of dysphagia as a problem. Studies of feeding difficulties in esophageal atresia are often fundamentally limited by reliance on subjective measures such as patient reported symptoms of dysphagia. It is well established that EA patients either do not recognize symptoms as abnormal given their life-long nature or learn adaptive measures to minimize symptoms with age (e.g. liberal use of liquids, avoidance of problematic textures, small bites, etc.).[5,6,10] It is therefore unsurprising that studies using dysphagia as a measure often fail to find associations between esophageal size and feeding or nutritional outcomes given this variability in patient perception of symptom importance in their everyday lives.[6]

Studies that instead use objective feeding or nutritional outcome measures suggest that EA patients may be at risk for difficulties that persist into adulthood. Individuals with esophageal atresia are more likely to be small, with up to 41% of EA children below the 25th percentile at age 10 years and 46% of EA adults with body mass index <18.5.[5,11,12] EA patients are also more likely to be short, with children at risk of stunting[13] and about a third of adults achieving an adult height less than 2 standard deviations below the mean.[12] Though some older studies are limited by lack of adjustment for extra-esophageal comorbidities, a recent study of 4-day dietary records in adolescents with repaired esophageal atresia found that even full oral feeders—regardless of comorbidities—had an energy intake that was lower than age-appropriate recommendations, with 71% reporting avoidance of specific foods mostly due to challenging texture.[13] (Of note, dysphagia questionnaire scores did not correlate with energy intake, which the authors hypothesized was due to learned adaptive eating behaviors to avoid symptoms).[13]

While the current study is limited by the use of enteral tube dependence as a proxy marker for feeding difficulties, we have adjusted for other potential confounders that contribute to oropharyngeal or esophageal dysphagia that may result in tube dependence. As patients with EA are often able to compensate for symptoms with feeding adaptations, the use of enteral tube dependence is likely an overly stringent outcome that underestimates the true prevalence of clinically significant feeding difficulties in our cohort. Moreover, we do not incorporate any objective measures of esophageal dysmotility as a contributor to feeding difficulties. Dysmotility likely interacts with esophageal diameter, as even mild strictures may become obstructive in a child with impaired motility. However, dysmotility has been repeatedly shown to be universal in EA, making this less relevant to incorporate into a predictive model, and there are currently no effective treatments for esophageal dysmotility making it an unmodifiable risk factor at the present time.[14–21]

Unsurprisingly, we found that lower weight *Z* scores were associated with feeding tube use, which likely reflects tube use for the purpose of providing supplemental calories to correct malnutrition rather than low weight itself causing feeding difficulties. Vocal fold movement impairments were also significantly associated with tube dependence, which may be related to poor airway protection and subsequent aspiration; documented aspiration on MBS was not significantly associated with our feeding outcome, possibly due to lack of power from low numbers of patients with documented MBS studies, failure of the MBS study to capture aspiration in all aspirating patients (e.g. those who refuse to fully participate due to aversion, or those who aspirate intermittently and are not captured on MBS by chance), and/or due to patient's ability to compensate with thickened liquids or modified textures in many cases of aspiration limited to thinner consistencies.

While tracheomalacia and the associated competition from work of breathing during feeding may contribute to feeding difficulties in populations cared for elsewhere, we failed to find a significant effect of tracheomalacia on tube dependence in our cohort, likely due to lack of power in the setting of low numbers of children followed at our institution with unrepaired severe malacia. In other settings where surgical correction of tracheomalacia is less common, respiratory symptoms from tracheal collapse may indeed contribute to feeding difficulties; this is supported by a recent French study that identified a significant link between respiratory symptoms and feeding difficulties, though the etiologies (e.g. aspiration, tracheomalacia, etc.) of respiratory symptoms are not further delineated.[7] Standardization of respiratory morbidity assessment in children with EA and further study are needed.

Even adjusting for important confounders such as nutritional status, extra-esophageal comorbidities, and age (as older children are expected to benefit from both expected developmental progression of oral feeding skills and acquisition of compensatory mechanisms to minimize dysphagia), we found that anastomotic diameter remained a significant, independent predictor of feeding tube dependence. Esophageal diameter was the only potentially modifiable predictor identified by our analysis. However, we have identified in our analysis and it is important to note that even children with larger anastomoses may experience enteral tube dependence for a variety of reasons (e.g. oral aversion and delayed acquisition of feeding skills in the surgically intense but developmentally critical infancy period; oropharyngeal dysphagia; increased metabolic needs from comorbid conditions such as cardiac disease; etc.) and that dilations alone may be insufficient to reach enteral tube independence in a child who may have multiple contributing factors toward tube dependence.

Understanding contributors to poor feeding and poor nutritional status is critical in the long-term management of children with EA. The current study identifies stricture severity as a potentially modifiable risk factor in EA children with feeding difficulties and is the first study to explore age-based diameters that are statistically predictive of feeding outcome, though our age-based diameters analysis is limited by poor sensitivity and specificity and should not be used to extrapolate rigid cutoffs to a clinical setting.

Relying on the combination of our collective clinical experience with strictures and emerging evidence from formal investigation of our EA cohort such as in the current study, we advocate for a more tailored approach to identifying and handling strictures in children with repaired EA as we have previously described,[22] rather than applying a one-size-fits-all approach of simply waiting for symptoms to emerge before considering treatment. Our approach—especially for long gap or complex surgical patients at high risk for stricture—is proactive rather than reactive. All high-risk children are evaluated by our center with a postoperative endoscopy at 1 month to permit initiation of dilations for evolving stricture; in low risk asymptomatic infants, our general practice is—at minimum—to assess the anastomosis with an esophagram within 3–6 months of repair to ensure severe stricture is not occultly developing.[22] Randomized, prospective study would help settle the debate over the optimal approach to stricture management but presents logistical and ethical challenges to feasibly complete. Additional studies using other objective feeding outcome measures or dysphagia tools that can be validated in EA will be essential in defining best practices in the feeding and nutritional management of individuals with EA.

ABBREVIATIONS

EA esophageal atresia
MFOIS modified Functional Oral Intake Scale

AUTHOR CONTRIBUTIONS

Dr Jessica L. Yasuda conceptualized and designed the study, designed the data collection instruments, collected data, performed statistical analysis, drafted the initial manuscript, and reviewed and revised the manuscript. Ms Gabriela N. Taslitsky designed the data collection instruments, collected data, drafted the initial manuscript, and reviewed and revised the manuscript. Mr Steven J. Staffa contributed to study design, performed statistical analysis, drafted the initial manuscript, and reviewed and revised the manuscript. Drs Jay Meisner, Peter D. Ngo, Thomas Hamilton, Somala Mohammed, and Benjamin Zendejas contributed to study design, reviewed, and revised the manuscript. Dr Michael A. Manfredi conceptualized and designed the study, supervised data collection, and critically reviewed the manuscript for important intellectual content. All authors approved the final manuscript as submitted and agreed to be accountable for all aspects of the work.

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