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PREMATURITY AND NUTRITIONAL RECOVERY AFTER DUODENAL ATRESIA REPAIR: A STRUCTURED NARRATIVE REVIEW

PREMATURIDADE E RECUPERAÇÃO NUTRICIONAL APÓS REPARO DE ATRESIA DUODENAL: UMA REVISÃO NARRATIVA ESTRUTURADA

PREMATURIDAD Y RECUPERACIÓN NUTRICIONAL DESPUÉS DE LA REPARACIÓN DE ATRESIA DUODENAL: UNA REVISIÓN NARRATIVA ESTRUCTURADA

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ABSTRACT

Introduction: Duodenal atresia is usually corrected successfully in the neonatal period, but nutritional recovery is heterogeneous. Prematurity adds gastrointestinal immaturity, higher protein and energy requirements, limited nutrient reserves, respiratory vulnerability, and delayed suck–swallow–breath coordination. Consequently, restoration of intestinal continuity does not necessarily coincide with enteral autonomy or safe full oral feeding. Objective: To evaluate how prematurity influences enteral, oral, and growth recovery after duodenal atresia repair and to synthesize evidence on postoperative feeding advancement, parenteral nutrition, transanastomotic feeding, enhanced-recovery pathways, surgical approach, and multidisciplinary oral-feeding support. Methods: A structured narrative review was conducted using PubMed/MEDLINE, Scopus, Web of Science, and the Cochrane Library from database inception through August 27, 2026. Primary studies of congenital duodenal obstruction were prioritized when they reported gestational age, birth weight, feeding milestones, parenteral nutrition, growth, gastrostomy, complications, or length of stay. High-quality neonatal nutrition studies were included when direct evidence in repaired duodenal atresia was unavailable. Because definitions and interventions were heterogeneous, findings were synthesized

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narratively. Results and Discussion: Prematurity was consistently associated with slower nutritional recovery, particularly at gestational age 35 weeks or less, and the combination of birth before 34 weeks with birth weight below 1,500 g identified a subgroup with more complications and delayed full oral intake. The evidence supports separating readiness for enteral feeding from readiness for oral feeding. Faster, protocolized feed advancement and selected use of transanastomotic feeding can reduce parenteral nutrition and central-line exposure, although contemporary cohorts are discordant and benefits depend on an accompanying nutritional protocol. Growth recovery is generally favorable by one year, but delayed initiation of parenteral nutrition in infants who subsequently require it may contribute to loss of weight z score. Comorbid congenital heart disease, trisomy 21, respiratory support, and additional gastrointestinal anomalies substantially modify outcomes. Conclusion: Prematurity should be treated as a modifier of the nutritional pathway rather than as a reason for routine prolonged fasting. A gestational-age–adapted strategy should provide early adequate parenteral nutrition when prolonged enteral insufficiency is expected, introduce human milk enterally as soon as physiological stability and anastomotic assessment allow, advance feeds through a standardized protocol, fortify according to preterm nutritional requirements, and develop oral skills independently of enteral volume targets. The central clinical objective is safe nutritional autonomy with preserved growth, not merely early first feeding.

Keywords: Infant. Premature. Duodenal Atresia. Enteral Nutrition. Parenteral Nutrition.

RESUMO

Introdução: A atresia duodenal é geralmente corrigida com sucesso no período neonatal, mas a recuperação nutricional é heterogênea. A prematuridade acarreta imaturidade gastrointestinal, maiores necessidades proteicas e energéticas, reservas limitadas de nutrientes, vulnerabilidade respiratória e atraso na coordenação entre sucção, deglutição e respiração. Consequentemente, a restauração da continuidade intestinal não coincide necessariamente com a autonomia enteral ou com a alimentação oral completa e segura. Objetivo: Avaliar como a prematuridade influencia a recuperação enteral, oral e do crescimento após a correção da atresia duodenal e sintetizar as evidências sobre o avanço da alimentação pós-operatória, nutrição parenteral, alimentação transanastomótica, protocolos de recuperação acelerada, abordagem cirúrgica e apoio multidisciplinar à alimentação oral. Métodos: Foi realizada uma revisão narrativa estruturada utilizando PubMed/MEDLINE, Scopus, Web of Science e a Cochrane Library, desde o início das bases de dados até 27 de agosto de 2026. Estudos primários sobre obstrução duodenal congênita foram priorizados quando relatavam idade gestacional, peso ao nascer, marcos de alimentação, nutrição parenteral, crescimento, gastrostomia, complicações ou tempo de internação. Estudos de alta qualidade sobre nutrição neonatal foram incluídos quando não havia evidências diretas sobre a atresia duodenal reparada. Como as definições e intervenções eram heterogêneas, os resultados foram sintetizados narrativamente. Resultados e Discussão: A prematuridade foi consistentemente associada a uma recuperação nutricional mais lenta, particularmente na idade gestacional de 35 semanas ou menos, e a combinação de nascimento antes da 34ª semana com peso ao nascer inferior a 1.500 g identificou um subgrupo com mais complicações e atraso na alimentação oral completa. As evidências apoiam a separação entre a prontidão para a alimentação enteral e a prontidão para a alimentação oral. O avanço mais rápido e protocolizado da alimentação e o uso seletivo da alimentação transanastomótica podem reduzir a nutrição parenteral e a exposição à linha central, embora as coortes atuais apresentem resultados discordantes e

os benefícios dependam de um protocolo nutricional associado. A recuperação do crescimento é geralmente favorável ao completar um ano, mas o início tardio da nutrição parenteral em bebês que posteriormente a necessitem pode contribuir para a perda do escore z de peso. Comorbidades como cardiopatia congênita, trissomia do cromossomo 21, suporte respiratório e anomalias gastrointestinais adicionais modificam substancialmente os resultados. Conclusão: A prematuridade deve ser considerada um fator que modifica a trajetória nutricional, e não um motivo para o jejum prolongado de rotina. Uma estratégia adaptada à idade gestacional deve proporcionar nutrição parenteral adequada desde o início quando for prevista uma insuficiência enteral prolongada, introduzir o leite materno por via enteral assim que a estabilidade fisiológica e a avaliação da anastomose o permitirem, avançar na alimentação por meio de um protocolo padronizado, fortificar de acordo com as necessidades nutricionais do prematuro e desenvolver habilidades orais independentemente das metas de volume enteral. O objetivo clínico central é a autonomia nutricional segura com crescimento preservado, e não apenas a primeira alimentação precoce.

Palavras-chave: Bebê. Prematuro. Atresia Duodenal. Nutrição Enteral. Nutrição Parenteral.

RESUMEN

Introducción: La atresia duodenal suele corregirse con éxito durante el periodo neonatal, pero la recuperación nutricional es heterogénea. La prematuridad conlleva inmadurez gastrointestinal, mayores necesidades proteicas y energéticas, reservas limitadas de nutrientes, vulnerabilidad respiratoria y un retraso en la coordinación entre la succión, la deglución y la respiración. En consecuencia, el restablecimiento de la continuidad intestinal no coincide necesariamente con la autonomía enteral ni con una alimentación oral completa y segura. Objetivo: Evaluar cómo influye la prematuridad en la recuperación enteral, oral y del crecimiento tras la corrección de la atresia duodenal, y sintetizar la evidencia sobre el avance de la alimentación postoperatoria, la nutrición parenteral, la alimentación transanastomótica, los protocolos de recuperación acelerada, el abordaje quirúrgico y el apoyo multidisciplinar a la alimentación oral. Métodos: Se llevó a cabo una revisión narrativa estructurada utilizando PubMed/MEDLINE, Scopus, Web of Science y la Biblioteca Cochrane, desde el inicio de las bases de datos hasta el 27 de agosto de 2026. Se dio prioridad a los estudios primarios sobre la obstrucción duodenal congénita cuando informaban sobre la edad gestacional, el peso al nacer, los hitos de alimentación, la nutrición parenteral, el crecimiento, la gastrostomía, las complicaciones o la duración de la estancia hospitalaria. Se incluyeron estudios de alta calidad sobre nutrición neonatal cuando no se disponía de evidencia directa sobre la atresia duodenal reparada. Dada la heterogeneidad de las definiciones y las intervenciones, los resultados se sintetizaron de forma narrativa. Resultados y discusión: La prematuridad se asoció de forma sistemática con una recuperación nutricional más lenta, especialmente en el caso de una edad gestacional de 35 semanas o menos, y la combinación de un parto antes de las 34 semanas con un peso al nacer inferior a 1.500 g permitió identificar un subgrupo con más complicaciones y un retraso en la ingesta oral completa. La evidencia respalda la separación entre la preparación para la alimentación enteral y la preparación para la alimentación oral. Un avance más rápido y protocolizado de la alimentación, junto con el uso selectivo de la alimentación transanastomótica, puede reducir la nutrición parenteral y la exposición a la vía central, aunque los resultados de las cohortes actuales son contradictorios y los beneficios dependen del protocolo nutricional que se aplique. La recuperación del crecimiento suele ser favorable al cabo de un año, pero el inicio tardío de la nutrición parenteral en los lactantes que posteriormente la requieran puede contribuir a la pérdida de la puntuación z de peso. La

cardiopatía congénita comórbida, la trisomía 21, el soporte respiratorio y otras anomalías gastrointestinales modifican sustancialmente los resultados. Conclusión: La prematuridad debe considerarse un factor que modifica la vía nutricional, más que una razón para un ayuno prolongado rutinario. Una estrategia adaptada a la edad gestacional debe proporcionar una nutrición parenteral adecuada desde el principio cuando se prevea una insuficiencia enteral prolongada, introducir la leche materna por vía enteral tan pronto como lo permitan la estabilidad fisiológica y la evaluación de la anastomosis, avanzar en la alimentación siguiendo un protocolo estandarizado, enriquecer la alimentación según las necesidades nutricionales de los bebés prematuros y desarrollar las habilidades orales independientemente de los objetivos de volumen enteral. El objetivo clínico principal es lograr una autonomía nutricional segura que preserve el crecimiento, y no simplemente iniciar la alimentación lo antes posible.

Palabras clave: Lactante. Prematuro. Atresia duodenal. Nutrición Enteral. Nutrición Parenteral.

1 INTRODUCTION

Congenital duodenal obstruction, which includes duodenal atresia, stenosis, web, and obstructive annular pancreas, has an estimated incidence of 1.22 per 10,000 live births and usually requires repair during the first days of life.[1] In a prospective population-based British cohort, 69% of affected infants had at least one associated anomaly, 42% received a transanastomotic tube, and 88% received parenteral nutrition, demonstrating that postoperative nutritional management remains highly variable despite excellent short-term surgical survival.[1]

The operation restores luminal continuity, but it does not immediately normalize gastric emptying, proximal duodenal motility, enteral tolerance, oral competence, or growth.[2–4] In a cohort of 87 infants with intrinsic duodenal obstruction, the median time to full enteral nutrition was 12 days, with an interquartile range of 9 to 17 days, illustrating clinically important dispersion even within one diagnostic category.[2] At one year, most infants have favorable gastrointestinal and growth outcomes, yet early recovery may involve prolonged parenteral nutrition, central venous access, weight loss, and extended hospitalization.[3,5]

Prematurity changes this postoperative trajectory because intestinal motor function, digestive capacity, nutrient stores, thermoregulation, respiratory stability, and immune defenses remain developmentally immature.[6,7] Preterm infants also require more protein and energy per kilogram to approximate fetal tissue accretion while simultaneously tolerating smaller fluid volumes and greater metabolic instability.[7] Therefore, a feeding schedule that is adequate for a term neonate after uncomplicated repair may deliver insufficient nutrition or impose inappropriate oral expectations in a very preterm infant.[7,8]

Direct evidence supports gestational age as more than a background characteristic.[2,6] Gestational age of 35 weeks or less independently predicted delayed full enteral nutrition in the 87-infant cohort, together with congenital heart disease and malrotation.[2] In a separate 82-infant series, the combination of birth before 34 weeks and birth weight below 1,500 g was associated with surgical and nonsurgical complications and a prolonged time to full oral intake.[6] These findings also show why gestational age and birth weight should be reported separately rather than collapsed into a single label of neonatal fragility.[2,6]

Nutritional recovery contains at least four distinct milestones: initiation of enteral milk, achievement of full enteral volume, discontinuation of parenteral nutrition, and achievement of full oral feeding.[2,3,8] The first three primarily reflect intestinal access, tolerance, and adequacy of nutrient delivery, whereas the fourth also requires neurological maturation and

coordinated sucking, swallowing, and respiration.[8] In preterm infants without major complications, oral feeding has commonly begun near 33.6 weeks postmenstrual age and full oral feeding near 35.1 weeks, with respiratory-support duration and birth weight influencing progression.[8]

Comorbidities further complicate attribution.[1,2,9] Congenital heart disease increases energy expenditure and may limit feed advancement, while trisomy 21 can add hypotonia, dysphagia, and aspiration risk.[2,9] In one duodenal atresia cohort, 82.4% of infants with trisomy 21 ultimately received a gastrostomy, and every infant with both trisomy 21 and structural heart disease required gastrostomy placement.[9] Thus, delayed oral feeding after anatomically successful repair should not automatically be interpreted as anastomotic dysfunction.[6,9]

Postoperative strategies have evolved from prolonged decompression and cautious volume increments toward earlier enteral exposure, transanastomotic feeding, human-milk prioritization, standardized feeding algorithms, minimally invasive surgery, and enhanced-recovery pathways.[10] Neonatal Enhanced Recovery After Surgery recommendations support multimodal care, avoidance of unnecessary tubes and opioids, maintenance of physiological stability, and timely enteral nutrition, while emphasizing that evidence in surgical neonates remains limited.[10] The central uncertainty is not whether nutrition is necessary, but how to match route, timing, volume, nutrient density, and oral skill demands to the infant's gestational maturity and comorbidity profile.[2,7,10]

This review therefore examines prematurity as a modifier of nutritional recovery after duodenal atresia repair.[2,6] It distinguishes enteral from oral autonomy, integrates growth and parenteral-nutrition outcomes, and evaluates the extent to which feeding protocols, transanastomotic tubes, operative approach, and multidisciplinary oral-motor support can reduce the nutritional disadvantage associated with preterm birth.[3,5,10]

2 OBJECTIVES

2.1 PRIMARY OBJECTIVE

To determine how prematurity influences nutritional recovery after surgical correction of duodenal atresia, with specific attention to time to full enteral nutrition, duration of parenteral nutrition, acquisition of full oral feeding, postnatal growth, and hospital length of stay.

2.2 SECONDARY OBJECTIVES

1. To distinguish gestational age, birth weight, small-for-gestational-age status, and associated anomalies as separate risk modifiers.
2. To compare early and slower postoperative feeding advancement strategies.
3. To assess the benefits and limitations of transanastomotic feeding and central venous parenteral nutrition.
4. To evaluate whether enhanced-recovery pathways and operative approach modify nutritional milestones.
5. To integrate human-milk provision, fortification, oral-motor maturation, swallowing safety, and growth monitoring into a practical prematurity-adapted pathway.

3 METHODOLOGY

3.1 DESIGN

This article was developed as a structured narrative review. The approach was selected because the available literature combines retrospective cohorts, prospective observational studies, quality-improvement pathways, surgical comparisons, and indirect studies of preterm oral-feeding maturation, with substantial heterogeneity in populations, interventions, and outcome definitions.

3.2 INFORMATION SOURCES AND SEARCH STRATEGY

PubMed/MEDLINE, Scopus, Web of Science, and the Cochrane Library were searched from database inception through August 27, 2026. Search concepts combined duodenal atresia, congenital duodenal obstruction, prematurity, preterm birth, low birth weight, very low birth weight, enteral feeding, oral feeding, full feeds, parenteral nutrition, growth, transanastomotic tube, gastrostomy, enhanced recovery, and laparoscopic repair. Reference lists and forward citations of pivotal studies were examined to identify additional primary reports.

3.3 ELIGIBILITY AND PRIORITIZATION

Primary studies were prioritized when they included neonates undergoing repair of duodenal atresia or intrinsic congenital duodenal obstruction and reported at least one nutritional outcome. Studies were retained when they evaluated prematurity, gestational age,

birth weight, feeding advancement, time to full enteral or oral feeding, parenteral nutrition, central venous access, growth, gastrostomy, length of stay, or a perioperative intervention with direct nutritional implications. Studies of preterm infants without duodenal obstruction were used only for mechanisms or interventions related to oral-feeding maturation and nutrient requirements that had not been studied directly after duodenal repair.

3.4 OUTCOME DEFINITIONS

Full enteral feeding was interpreted according to each study's definition, commonly 120 to 150 mL/kg/day. Full oral feeding meant that prescribed nutrition was taken safely by mouth without gavage support. Parenteral-nutrition duration, time to first enteral feed, time to first oral feed, weight or weight-for-age z score, gastrostomy placement, central venous catheter use, and postoperative length of stay were treated as distinct outcomes rather than interchangeable indicators of recovery.

3.5 DATA SYNTHESIS AND CRITICAL APPRAISAL

Study design, sample size, gestational-age and birth-weight distribution, intervention or exposure, feeding definitions, quantitative findings, adjustment strategy, and clinically important limitations were extracted. Meta-analysis was not undertaken because of incompatible outcome definitions, historical changes in practice, overlapping cohorts, nonrandom treatment allocation, and substantial confounding by prematurity, associated anomalies, and surgical era. The strength of each conclusion was calibrated according to consistency, directness, precision, and risk of bias.

4 RESULTS AND DISCUSSION

4.1 EVIDENCE LANDSCAPE

The evidence base is dominated by single-center retrospective cohorts, although prospective population-based data and recent multicenter or protocol-implementation studies provide important external context.[1,3,10] Direct analyses of prematurity are scarce, and many intervention studies contain few extremely preterm or very-low-birth-weight infants.[2,6,11] Table 1 summarizes the principal studies that inform the relationship between prematurity, feeding strategy, parenteral nutrition, growth, and oral recovery.

Table 1

Principal studies of nutritional recovery after congenital duodenal obstruction repair

Study	Design and population	Risk factor / strategy	Nutritional endpoint	Quantitative finding	Clinical interpretation
Bairdain et al., 2014[2]	Retrospective cohort; 87 infants with intrinsic duodenal obstruction	Prematurity and comorbidity	Time to full enteral nutrition	Median 12 days (IQR 9–17). GA ≤35 weeks (P<0.001), congenital heart disease (P=0.02), and malrotation (P=0.03) independently predicted delay.	Prematurity is a direct predictor of delayed enteral autonomy, not merely a marker of lower birth weight.
Bishay et al., 2013[5]	Retrospective cohort; 54 repaired infants	Initial PN, delayed PN, or no PN	Growth and suspected sepsis	Of 35 infants not started on PN, 13 (37%) later required it. Suspected sepsis occurred in 41% receiving PN vs 14% never receiving PN (P=0.04). Delayed-PN infants lost weight z score and did not recover it by last follow-up.	PN has catheter-related cost and risk, but waiting too long in an infant who ultimately needs PN may permit avoidable nutritional deficit.
Bethell et al., 2020[1]	Prospective UK population cohort; 103 infants	Contemporary practice variation	TAT and PN use; early outcomes	TAT was used in 42%; PN in 88%. Associated anomalies occurred in 69%; repair-related reoperation by day 28 was 3%.	Nutritional practice varies widely even when early surgical outcomes are favorable.

Study	Design and population	Risk factor / strategy	Nutritional endpoint	Quantitative finding	Clinical interpretation
Smith and Landman, 2019[9]	Retrospective cohort; 43 infants; 19 with trisomy 21	Trisomy 21 and congenital heart disease	Gastrostomy requirement	Ultimately, 82.4% of infants with trisomy 21 had a gastrostomy. All 9 infants with trisomy 21 plus structural heart disease required gastrostomy.	Oral-feeding failure may reflect neuromotor and cardiac comorbidity rather than impaired intestinal continuity.
Xu et al., 2020[12]	Prospective nonrandomized study; 23 ERAS vs 26 controls	Neonate-specific ERAS pathway	Length of stay, feeding intolerance, 1-month weight	LOS 9.70±1.22 vs 12.65±1.69 days (P<0.001); 1-month weight was higher with ERAS, without more complications or feeding intolerance.	Coordinated perioperative care may improve recovery and early growth, but allocation by prenatal diagnosis creates selection bias.
Deguchi et al., 2022[6]	Retrospective cohort; 82 infants	Early preterm birth and very low birth weight	Complications and time to full oral intake	Thirty infants (37%) were preterm; 11 (13%) were born <34 weeks. The combination of GA <34 weeks and birth weight <1,500 g predicted surgical complications (P=0.0028), nonsurgical complications (P=0.021), and delayed full oral intake (P=0.013).	The highest-risk group is defined by both developmental immaturity and limited body mass.

Study	Design and population	Risk factor / strategy	Nutritional endpoint	Quantitative finding	Clinical interpretation
Aroonsaeng et al., 2022[13]	Retrospective cohort; 26 fast-fed vs 25 slow-fed infants	Oral or gavage increments ≥2.5 mL/kg more than once daily	First oral feed and full enteral feed	First oral feed: POD 7.7±3.2 vs 16.1±7.7 (P<0.001). Full feeds: POD 12.5±5.3 vs 18.8±9.7 (P<0.01).	Faster advancement was feasible in predominantly near-term infants, but results should not be transferred unchanged to very preterm infants.
Bethell et al., 2021[3]	Prospective population follow-up; 80/100 infants with 1-year data	Natural history after repair	Enteral autonomy, PN, growth, CVC complications	Full enteral feeding at 1 year: 67/68 (99%); median time 13 days. Median PN 11 days. Median birth-to-1-year weight z-score change −0.12; 91% had a 1-year z score between −2 and +2. CVC-related complications occurred in 23%.	Long-term growth is usually reassuring, but central-line exposure carries substantial morbidity.
Treider et al., 2022[14]	Retrospective cohort; 100 infants; 37% received TAFT	Transanastomotic feeding	First enteral feed, PN, CVC	TAFT infants started enteral feeding 1.5 days earlier, received PN 2 days less (both P<0.001), and had fewer CVCs (65% vs 89%; P=0.008).	A tube may accelerate distal nutrient delivery and reduce invasive access, but allocation was not randomized.

Study	Design and population	Risk factor / strategy	Nutritional endpoint	Quantitative finding	Clinical interpretation
Cresner et al., 2022[15]	Retrospective cohort; 96 infants; 54 received TAT	Transanastomotic feeding	Full enteral feed and CVC	Full enteral feeding: 6 days (IQR 5–8) vs 10 days (7.5–12), P<0.001. First feed: 2 vs 3 days, P<0.001. CVC use: 15% vs 76%, P<0.001.	Large apparent benefit, but historical practice and patient selection may explain part of the effect.
Røkkum et al., 2025[11]	Prospective implementation cohort; 21 ERP infants vs 40 historical controls	Diagnosis-specific enhanced recovery protocol	First enteral and breast feed; complications	Overall protocol compliance was 80%. Time to first enteral and breast feed decreased, while complication rate and LOS remained unchanged.	A standardized multidisciplinary pathway can advance feeding without a safety penalty; a dedicated protocol for GA <32 weeks is still needed.
Nowak et al., 2026[16]	Retrospective cohort; 71 infants; 25 received TAT	TAT without an institutional feeding-advancement protocol	Full enteral and full oral feeding; PN; growth	Full enteral: 13 vs 12 days (adjusted HR 0.98, 95% CI 0.44–2.21). Full oral: 22 vs 16 days (adjusted HR 0.74, 95% CI 0.31–1.77). PN: 5 vs 6 days; growth similar.	Tube placement alone did not improve recovery when feeding progression was not protocolized.

Study	Design and population	Risk factor / strategy	Nutritional endpoint	Quantitative finding	Clinical interpretation
Türker Çolak et al., 2026[17]	Retrospective cohort; 36 analyzable infants; 21 TAT vs 15 no TAT	Transanastomotic feeding	First/full enteral feed, PN, CVC, LOS	First feed 3.01±2.05 vs 11.1±5.4 days (P=0.001); full feeds 7.6±3.8 vs 19.2±15.6 (P=0.002); PN 6.5±4.1 vs 19.8±12.4 days (P=0.019); CVC 23.8% vs 73.3% (P=0.004); LOS 12.6±4.9 vs 25.2±21.1 days (P=0.03).	Contemporary favorable signal, but exclusions, small sample, and very large group differences raise residual-confounding concerns.
Li et al., 2026[18]	Retrospective cohort; 152 infants; 60 matched pairs	Laparoscopic vs open repair	Gastrointestinal recovery, PN, LOS	After propensity matching, laparoscopic repair accelerated gastrointestinal recovery and shortened postoperative PN; LOS and costs were not significantly different. Overall complications: 5.92%.	Operative approach may influence early milestones, but expertise, selection, and feeding protocols remain inseparable modifiers.

Source: Prepared by the authors.

Abbreviations: CDO, congenital duodenal obstruction; CVC, central venous catheter; ERAS, Enhanced Recovery After Surgery; ERP, enhanced recovery protocol; GA, gestational age; HR, hazard ratio; IQR, interquartile range; LOS, length of stay; PN, parenteral nutrition; POD, postoperative day; TAT/TAFT, transanastomotic feeding tube.

4.2 PREMATURITY IS A GRADED RISK MODIFIER, NOT A BINARY DIAGNOSIS

The most consistent direct finding is a gradient of delayed recovery with decreasing gestational maturity.[2,6] In Bairdain *et al.*, gestational age of 35 weeks or less independently

delayed full enteral nutrition even when congenital heart disease and malrotation were considered.[2] In Deguchi *et al.*, the combination of gestational age below 34 weeks and birth weight below 1,500 g identified infants with more complications and slower full oral intake, whereas small-for-gestational-age status alone was not a significant predictor.[6]

These studies imply that prematurity and low birth weight should not be used interchangeably.[2,6] Gestational age captures neuromotor, respiratory, gastrointestinal, and metabolic maturity, while birth weight also reflects fetal growth and the absolute reserve available during postoperative catabolism.[6,7] A 1,500-g infant born at 33 weeks and a growth-restricted 1,500-g infant born later may therefore have different feeding physiology despite the same weight.[6,7]

Prematurity also interacts with the anatomy and associated disease rather than acting alone.[2,19] Complete obstruction is associated with lower gestational age, lower operative weight, more congenital heart disease, and higher morbidity than incomplete obstruction.[19] Once prematurity and cardiac disease are removed from subgroup analyses, the anatomical degree of obstruction still influences time to first enteral feeding, showing that developmental and anatomical factors contribute simultaneously.[19]

The practical consequence is risk stratification before surgery.[2,6] A stable late-preterm infant with isolated atresia may follow a near-term pathway, while a very-low-birth-weight infant born before 34 weeks, particularly with heart disease or another gastrointestinal anomaly, should be expected to need longer nutritional bridging and more conservative oral goals.[2,6] This expectation should trigger earlier nutritional planning, not a presumption of surgical failure.[2,6]

4.3 ENTERAL AUTONOMY AND ORAL AUTONOMY MUST BE SEPARATED

Full enteral feeding can be achieved through gastric or postpyloric gavage before an infant is developmentally ready to take all nutrition by mouth.[3,8] This distinction is especially important in preterm infants because suck–swallow–breath coordination, endurance, and airway protection mature mainly across the mid-30-week postmenstrual period.[8] In uncomplicated preterm cohorts, full oral feeding near 35 weeks postmenstrual age is common, but respiratory support and lower birth weight shift that milestone later.[8]

After duodenal atresia repair, a falling gastric aspirate, absence of progressive distension, hemodynamic stability, and a clinically reassuring abdomen can support enteral progression, but none of these findings proves oral safety.[4,10] Conversely, coughing,

desaturation, bradycardia, poor state regulation, fatigue, or inefficient milk transfer during oral attempts should not automatically lead to withdrawal of well-tolerated tube feeding.[8],[10] The route can be changed while nutritional delivery continues.[7],[8]

The difference between the two milestones explains apparently contradictory results across studies.[6],[9],[16] A transanastomotic tube may shorten time to enteral delivery yet have little effect on full oral feeding, as seen in the 2026 cohort in which TAT placement changed neither endpoint after adjustment.[16] Similarly, trisomy 21 and cardiac disease can produce persistent oral-feeding dependence despite adequate intestinal absorption and successful anastomotic function.[9]

Clinical documentation should therefore report first enteral feed, full enteral volume, PN discontinuation, first nutritive oral attempt, and full oral feeding separately.[2],[3],[16] Corrected gestational age at each oral milestone is more informative than postoperative day alone in preterm infants.[6],[8] This reporting framework also prevents a term infant who reaches oral feeds on postoperative day 12 from being compared uncritically with a 30-week infant who reaches enteral volume on day 10 but appropriately remains gavage-dependent for developmental reasons.[6],[8]

4.4 PARENTERAL NUTRITION: AVOID BOTH ROUTINE EXPOSURE AND DELAYED RESCUE

Parenteral nutrition is often necessary after duodenal atresia repair, but its benefit depends on timing and predicted duration of inadequate enteral intake.[1],[5] In the UK population cohort, 88% received PN, whereas a subsequent one-year analysis showed a median PN duration of 11 days and at least one central venous catheter complication in 23%.[1],[3] These data support reducing avoidable central-line days without permitting a prolonged protein and energy deficit.[3],[7]

Bishay *et al.* provide the clearest warning against a simple policy of withholding PN from everyone.[5] Among 35 infants not started promptly on PN, 13 later required it; this delayed-PN subgroup lost weight z score between surgery and full enteral feeding and had not recovered the loss at last follow-up.[5] The study was observational and confounding by severity is likely, but it suggests that infants predicted to feed slowly should receive adequate nutritional support early rather than accumulate a deficit before rescue.[5]

The same study also documents the tradeoff: clinically suspected sepsis occurred in 41% of infants exposed to PN compared with 14% of those never receiving PN.[5] This

association does not prove PN caused every infection because sicker infants were more likely to receive it, yet the one-year population cohort confirms that central venous access itself produces material morbidity.[3,5] The best strategy is therefore neither mandatory PN nor automatic avoidance, but early individualized estimation of how long enteral intake will remain insufficient.[3,5,7]

Premature infants are more likely to cross the threshold at which PN is justified because their nutrient requirements are higher and their enteral and oral progression may be slower.[2,7] PN should provide age-appropriate amino acids, glucose, lipid, electrolytes, minerals, vitamins, and trace elements while glucose tolerance, triglycerides, acid-base status, liver tests, and catheter integrity are monitored.[7] As enteral milk increases, PN should be reduced according to delivered protein and energy rather than a volume-only rule.[7]

4.5 EARLY ENTERAL FEEDING AND STANDARDIZED ADVANCEMENT

The traditional rationale for slow feeding is postoperative dysmotility in the dilated stomach and proximal duodenum.[4] Manometric studies confirm abnormal transit and motor patterns in the dilated segment, but the stomach and distal intestine may still support controlled nutrient delivery.[4,13] Therefore, dysmotility justifies monitoring and route adaptation; it does not necessarily justify prolonged complete fasting.[4,13]

In the 51-infant study by Aroonsaeng *et al.*, a fast-fed strategy using oral or gavage increments of at least 2.5 mL/kg, advanced more than once daily, moved the first oral feed from postoperative day 16.1 to 7.7 and full feeds from day 18.8 to 12.5.[13] No signal of anastomotic disruption or necrotizing enterocolitis was identified, but most infants were near term and weighed more than 1,500 g.[13] The exact increment should therefore not be copied into extremely preterm infants without adjustment for gestational age, milk type, cardiorespiratory stability, and local neonatal feeding standards.[7,13]

Enhanced-recovery studies support the broader concept of protocolized care.[11,12] Xu *et al.* reported a reduction in hospital stay from 12.65 to 9.70 days and higher weight at one month without more feeding intolerance, although allocation according to prenatal diagnosis created important selection bias.[12] Røkkum *et al.* achieved 80% protocol compliance and earlier first enteral and breast feeds without more complications, but length of stay did not change and the authors specifically recognized the need for a separate pathway below 32 weeks.[11]

These findings suggest that standardization is more important than a universal fixed speed.[11–13] A useful protocol specifies prerequisites for starting milk, preferred milk, initial route, starting volume, advancement steps, objective reasons to pause, a plan for aspirates and emesis, criteria for PN reduction, and escalation triggers.[10,11] For a preterm infant, the protocol should permit enteral advancement through gavage while oral attempts remain cue-based and developmentally limited.[7,8,10]

4.6 TRANSANASTOMOTIC FEEDING: A BRIDGE WHOSE EFFECT DEPENDS ON THE PATHWAY

A transanastomotic feeding tube delivers milk distal to the repair while proximal decompression and motility recover.[14,15,20] Earlier cohorts found substantial benefits: Hall *et al.* reported earlier enteral feeding and less central venous access, Treider *et al.* found feeding began 1.5 days earlier with two fewer PN days, and Cresner *et al.* reported full enteral feeding at 6 rather than 10 days with CVC use of 15% rather than 76%.[14,15,20] These observations are biologically plausible and clinically relevant for infants in whom central-line avoidance is particularly valuable.[14,15,20]

The population-based analysis by Bethell *et al.* was more cautious.[21] TAT use was associated with earlier enteral feeding and shorter PN in univariable analysis, but only the shorter PN association persisted after multivariable adjustment, and most infants with TATs still received both a CVC and PN.[21] This shows that tube placement can be neutralized when the surrounding feeding culture remains conservative.[21]

The two 2026 cohorts sharpen the uncertainty.[16,17] Nowak *et al.* found no adjusted difference in full enteral feeding, full oral feeding, PN duration, growth, or complications in a center without a formal feed-advancement protocol.[16] By contrast, Türker Çolak *et al.* reported markedly earlier feeding, 13 fewer PN days, fewer CVCs, and shorter hospitalization with TAT placement.[17] The latter study excluded deaths before feeding and early tube removals, and the magnitude of benefit is vulnerable to selection and era effects.[17]

A 2024 meta-analysis of observational studies estimated that TAT feeding reduced time to full enteral feeding by 3.34 days and PN duration by 6.32 days, but certainty remained low because high-risk studies had to be excluded and no randomized trials were available.[22] The most defensible interpretation is that TAT feeding can be useful when paired with a defined early-enteral protocol, particularly in infants expected to require prolonged PN, but it is not an isolated guarantee of faster recovery.[16,17,22] Tube dislodgement, blockage, mucosal

injury, and rare perforation must also be balanced against the central-line complications it may prevent.[20,22]

4.7 OPERATIVE APPROACH IS ONE COMPONENT, NOT THE NUTRITIONAL PRESCRIPTION

Laparoscopic repair may reduce tissue trauma and hasten early gastrointestinal recovery, but feeding outcomes are strongly influenced by surgeon experience, case selection, anastomotic technique, and postoperative protocols.[18,23,24] Early concurrent series reported faster advancement and shorter hospitalization after laparoscopic repair, while later studies produced less uniform results.[23]

Holler *et al.* found that a laparoscopic miniature-stapler group began feeds at 5 rather than 11.9 days and reached full feeds at 14.5 rather than 24.4 days compared with open repair.[24] The stapled group was older and heavier at surgery, however, making nutritional benefit difficult to separate from patient selection.[24] This limitation is particularly important for the present topic because the smallest premature infants are more likely to undergo open repair.[18,24]

The 2026 Chinese cohort used propensity matching to address baseline gestational-age and birth-weight imbalance.[18] Among 60 matched pairs, laparoscopy retained advantages in gastrointestinal recovery and PN duration, but not in postoperative length of stay, cost, or complications.[18] This pattern suggests a modest early recovery effect rather than a universal improvement in the entire nutritional course.[18]

Choice of operative approach should therefore be based on neonatal stability, anatomy, body size, available instruments, and team expertise.[10,18] A well-constructed anastomosis followed by a coherent nutritional pathway is more important than selecting an approach solely to achieve an earlier feed date.[10,18] For very-low-birth-weight infants, the evidence does not justify delaying necessary repair simply to reach a size considered more convenient for a particular technique, but preoperative stabilization and comorbidity evaluation remain essential.[6,10]

4.8 HUMAN MILK, FORTIFICATION, ORAL SKILL DEVELOPMENT, AND GROWTH SURVEILLANCE

Human milk should be the preferred enteral substrate because it is adapted to neonatal digestion and is central to contemporary preterm nutrition practice.[7] In a premature infant

recovering from duodenal repair, maternal milk expression should begin early, and donor human milk may be considered according to local policy when maternal milk is unavailable.[7,10] The absence of direct duodenal-atresia trials comparing milk types should be acknowledged rather than converted into a disease-specific efficacy claim.[7]

Unfortified human milk alone often does not meet the protein, mineral, and energy requirements of a growing preterm infant once full volumes are reached.[7] Fortification should be introduced according to gestational age, current weight, fluid allowance, biochemical monitoring, and tolerance, with attention to calcium, phosphorus, sodium, iron, vitamin D, and overall protein delivery.[7] Increasing caloric density can improve nutrient delivery when volume is limited, but excessive osmolality or rapid concentration changes may worsen intolerance.[7]

Oral feeding should develop in parallel through cue-based opportunities, nonnutritive sucking, breastfeeding support, positioning, pacing, and early speech-language or occupational-therapy assessment when instability or inefficiency appears.[8,25] In a randomized trial of 84 preterm infants, a structured premature infant oral-motor intervention shortened transition to full oral feeds by approximately 2 days, reduced hospitalization by 8 days, and increased weight gain by 4.9 g/kg/day relative to routine oral stimulation.[25] Because these infants were not recovering from duodenal surgery, the result supports feasibility of oral-motor support but does not prove the same magnitude of benefit after atresia repair.[25]

Trisomy 21, congenital heart disease, respiratory support, and neurological abnormalities should lower the threshold for a formal swallowing and airway-safety assessment.[6,9,26] Population-based data suggest that trisomy 21 alone does not uniformly worsen all short-term surgical outcomes, yet single-center feeding data show a high gastrostomy rate when trisomy 21 coexists with duodenal atresia, especially with structural heart disease.[9,26] The discrepancy likely reflects differences in gastrostomy practice, outcome definitions, and comorbidity mix, and it argues against using the chromosome diagnosis as an automatic indication for permanent enteral access.[9,26]

Growth monitoring should include daily weight during acute fluid shifts, weekly length and head circumference when feasible, and serial weight-for-age or weight-for-length z scores using an appropriate preterm or syndrome-specific reference.[3,7] In the population cohort, the median change in weight z score from birth to one year was only −0.12, and 91% of measured infants were between −2 and +2 at one year.[3] This reassuring average should not obscure the subgroup that loses centiles during prolonged inadequate intake or the infant whose weight gain reflects fluid rather than tissue accretion.[3,5,7]

4.9 A PREMATURITY-ADAPTED CLINICAL PATHWAY

The evidence supports a staged pathway in which anatomical recovery, nutrient delivery, and oral maturation are assessed separately.[2,6,10] Table 2 translates the review findings into a practical framework; it is intended for multidisciplinary adaptation rather than as a fixed prescriptive protocol.[7,10,11]

Table 2

Prematurity-adapted nutritional recovery pathway after duodenal atresia repair

Phase	Primary objective	Readiness / decision points	Prematurity-adapted strategy	Monitoring and escalation
Preoperative	Restore physiological stability and define risk	Correct fluid, electrolyte, acid-base, temperature, glucose, and respiratory instability; assess heart disease, malrotation, additional atresia, GA, birth weight, and growth status	Begin individualized PN early when prolonged enteral insufficiency is likely; initiate maternal milk expression; avoid delaying repair solely for weight gain when the infant is otherwise ready	Daily weight and fluid balance; electrolytes, glucose, acid-base status; echocardiography and anomaly work-up as indicated
POD 0–2	Protect repair while preventing cumulative nutrient deficit	Hemodynamic stability, reassuring abdomen, controlled gastric losses, no evidence of leak or distal obstruction	Continue adequate PN; use human-milk oral care or minimal enteral exposure according to local protocol; consider TAT only with a defined advancement plan	Gastric output trend, abdominal findings, perfusion, lactate when indicated, glucose, triglycerides, catheter site
Enteral progression	Reach adequate enteral protein and energy safely	Tolerance assessed by clinical trajectory rather than isolated residual volume; distinguish benign emesis/dysmotility from obstruction, ischemia, infection, or leak	Advance human milk by standardized gestational-age–adapted increments; gastric or postpyloric gavage may progress before oral readiness; fortify as volume and tolerance permit	Delivered mL/kg/day, kcal/kg/day, protein g/kg/day, stool/emesis, abdominal examination, electrolytes, phosphorus, alkaline phosphatase, liver tests

Phase	Primary objective	Readiness / decision points	Prematurity-adapted strategy	Monitoring and escalation
Oral maturation	Develop safe and efficient oral feeding	Stable respiratory support; organized alert state; coordinated sucking, swallowing, and breathing; no recurrent cough, desaturation, bradycardia, or fatigue	Cue-based breast or bottle attempts; pacing and positioning; nonnutritive sucking; early therapy assessment. Do not reduce tolerated gavage nutrition merely because oral skills are immature	Volume and quality per oral attempt, cardiorespiratory events, feeding duration, aspiration signs; instrumental swallowing assessment when clinically indicated
Discharge and follow-up	Maintain growth and reduce device-related harm	Sustainable nutrition plan, trained caregivers, medication and micronutrient plan, safe tube strategy if needed, coordinated surgical/neonatal follow-up	Use corrected age and an appropriate preterm or syndrome-specific growth reference; reassess need for PN, central access, gavage, and gastrostomy at each milestone	Weight, length, head circumference, z-score trajectory, micronutrients, liver function after prolonged PN, vomiting, distension, stooling, oral aversion, tube complications

Abbreviations: GA, gestational age; PN, parenteral nutrition; POD, postoperative day; TAT, transanastomotic feeding tube. This framework does not replace local neonatal surgical, nutrition, or infection-control protocols.
Source: Prepared by the authors.

5 LIMITATIONS

The literature specifically linking prematurity to nutritional outcomes after duodenal atresia repair is small and primarily retrospective. Gestational-age thresholds vary, very preterm infants are underrepresented, and some reports combine atresia, stenosis, web, annular pancreas, and other forms of congenital duodenal obstruction.

Feeding outcomes are inconsistently defined. Full enteral feeding may mean 120, 140, or 150 mL/kg/day; some studies report first oral feeding, others any enteral feeding, and still others full oral autonomy. Hospital length of stay is especially confounded in premature infants because discharge also depends on respiratory stability, thermoregulation, apnea resolution, and corrected gestational age.

Treatment allocation was rarely randomized. Infants selected for laparoscopic repair or TAT placement may differ in birth weight, anatomy, era of care, and clinician preference, while PN exposure is strongly confounded by illness severity. Historical cohorts also span

major changes in neonatal nutrition, central-line care, human-milk access, and surgical technique.

Evidence on human milk, fortification, and oral-motor therapy is partly extrapolated from preterm populations without duodenal atresia. These interventions are physiologically relevant, but disease-specific effect sizes cannot be assumed. Long-term body composition, linear growth, neurodevelopment, feeding behavior, and family-reported burden remain insufficiently studied.

6 CONCLUSION

Prematurity is associated with slower nutritional recovery after duodenal atresia repair, but its effect is graded and strongly modified by birth weight, congenital heart disease, additional gastrointestinal anomalies, respiratory support, and trisomy 21. The clearest high-risk phenotype is the infant born before 34 weeks with birth weight below 1,500 g, for whom complications and delayed full oral feeding are more likely.

The central clinical distinction is between intestinal readiness and oral readiness. A premature infant may tolerate full enteral nutrition by tube while appropriately lacking the neurological and respiratory maturity for full oral intake. Enteral volume, parenteral-nutrition discontinuation, and oral autonomy should therefore be tracked as separate outcomes using corrected gestational age.

Protocolized early enteral feeding and selected transanastomotic feeding may reduce PN and central-line exposure, but tube placement alone is insufficient. Benefits are most plausible when the intervention is embedded in a multidisciplinary advancement protocol that defines milk choice, volumes, pause criteria, fortification, and escalation. Evidence does not support routine prolonged fasting, nor does it support routine avoidance or routine use of PN for every infant.

A practical prematurity-adapted strategy combines early adequate PN when delayed enteral autonomy is predicted, prompt human-milk enteral exposure once stable, gestational-age–appropriate advancement and fortification, independent cue-based oral-skill development, and serial growth assessment. Future multicenter prospective studies should stratify by gestational age and birth weight, use standardized definitions of full enteral and full oral feeding, record nutrient delivery rather than volume alone, and measure growth and neurodevelopment beyond discharge.

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