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Association of Unexplained Feeding Intolerance with Congenital Hypothyroidism in Preterm Neonates: A Missing Link

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ABSTRACT

Objective: This study investigates the association between unexplained feeding intolerance (FI) and congenital hypothyroidism (CH) in otherwise healthy preterm neonates.

Methodology: This descriptive study was conducted in the Neonatology Department, Fatima Memorial Hospital, Lahore to assess the association between thyroid function and feeding intolerance in 102 preterm neonates. Maternal, neonatal, and feeding variables were recorded, and data were analyzed using SPSS version 16.0. Categorical variables were expressed as frequencies and percentages, while continuous variables were summarized as mean ± SD or median (IQR) based on distribution. Fisher's exact test, Student's *t*-test, and Mann–Whitney *U* test were applied as appropriate.

Results: Of the 102 preterm neonates with FI, 37.3% had CH. FI characteristics such as type of feed, time to establish full feeding, and abdominal distention (isolated or combined with increased gastric residue or reflux) were significantly associated with hypothyroidism ($p < 0.005$). Significant risk factors for CH including APGAR score at 5 minutes ($p < 0.001$), jaundice ($p < 0.001$), patent ductus arteriosus ($p = 0.006$), duration of hospital stay ($p = 0.002$), parity ($p < 0.001$), chorioamnionitis ($p = 0.024$) and antepartum hemorrhage ($p = 0.002$).

Conclusion: Approximately one-third of preterm neonates with FI were diagnosed with hypothyroidism. Abdominal distention, whether isolated or associated with increased gastric residue or reflux, was the most prominent feature of FI linked to CH. Parity, antepartum hemorrhage, chorioamnionitis, low APGAR scores, jaundice, and hsPDA, were significantly associated with CH.

Key words: Abdominal Distention; Feeding Intolerance; Hypothyroidism; NICU; Prematurity; Reflux, vomiting

Authors' Contribution:
[1,2]Conception; Literature research; manuscript design and drafting; [2,3]Critical analysis and manuscript review; [3,4]Data analysis; Manuscript Editing.

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Introduction

Preterm birth, defined as delivery before 37 weeks of gestation, affects approximately 10% of deliveries worldwide.[1] Pakistan bears a significant burden of preterm births (15.8% of all deliveries).[2] Prematurity-related problems include temperature instability, respiratory distress syndrome (RDS), infections, apnea, hypoglycemia, jaundice, hemodynamically significant patent ductus arteriosus (hsPDA), feeding intolerance, necrotizing enterocolitis (NEC), periventricular leukomalacia (PVL), and repeated hospitalizations.[3]

In preterm neonates feed establishment is crucial to promote growth, support immune function, and facilitate digestive system maturation.[4] Feed intolerance is difficulty in digesting enteral feeding and continues to be a significant challenge in this process. Its diagnostic criteria include the triad of abdominal distension, increased gastric residuals, and reflux.[4] FI in newborns can be a sign of a variety of problems, ranging from minor, self-limiting illnesses to serious, life-threatening systemic illnesses.[5] FI affects approximately 2.6% to 75% of all preterm neonates.[3,6,7]

FI in preterm neonates is linked to structural and functional immaturity of GIT.[8] The scenario is further complicated in settings of other prematurity related comorbidities like NEC, congenital heart disease (CHD), hsPDA, sepsis, respiratory failure, intraventricular hemorrhage (IVH), and electrolyte imbalances.[9,10] Gastrointestinal symptoms in hypothyroidism are underreported in neonates.[11] About 1:3000 neonates have congenital hypothyroidism globally.[12]

In our clinical practice, a small group of otherwise healthy preterm neonates experience feeding intolerance without an identifiable cause. This raises the question: is feeding intolerance solely due to an immature gut, or are there other overlooked causes in preterm neonates. Research on the incidence, the clinical presentation, and comorbidities associated with feeding intolerance in preterm infants is limited in developing countries such as Pakistan. Additionally, there is minimal data on the association between unexplained feeding intolerance in otherwise healthy preterm neonates and congenital hypothyroidism. Identifying congenital hypothyroidism early in these neonates is essential to prevent complications like feeding intolerance, reduce unnecessary investigations, shorten hospital stays, and lower the risk of hospital-acquired infections. Our study aims to interrogate the link between unexplained feeding intolerance with congenital hypothyroidism in otherwise healthy preterm neonates.

Methodology

A descriptive study was conducted during period spans over 12 months from May 2023 to April 2024 in the Department of Neonatology, Fatima Memorial Hospital, Lahore.

Sample size was 102 and calculated with a 95% confidence level along with 10% margin of error, and 5% level of significance by taking feeding related problems as 7%.[3] It was done by using openepi.com

All preterm neonates (gestational age <37 weeks) admitted to the NICU during the study period were considered for inclusion. Only those who were otherwise healthy but presented with unexplained feeding intolerance were reviewed. However, all those neonates having major anomalies incompatible with life, sepsis, electrolytes imbalance, bloody gastric aspirates, antenatal diagnosis of gut malformations, surgical abdomen (radiological diagnosis), suspected case of inborn error of metabolism or parents who refused permission to participate in the study were excluded. The serum fT_4 and TSH were measured by chemiluminescent immunoassay (CLIA). Consent was obtained from parents or guardians before inclusion. Feeding intolerance was labelled if the neonate had one or any combination leading to feed interruption and include

1. Abdominal distension (abdominal girth increment as ≥ 2 cm between 2 consecutive feedings)
2. Increased gastric residuals (more than 50% gastric residue of the previous feeding)
3. Reflux/vomiting

Congenital hypothyroidism was diagnosed based on either low Free T4 (fT_4) and increased TSH levels or both TSH & fT_4 low.

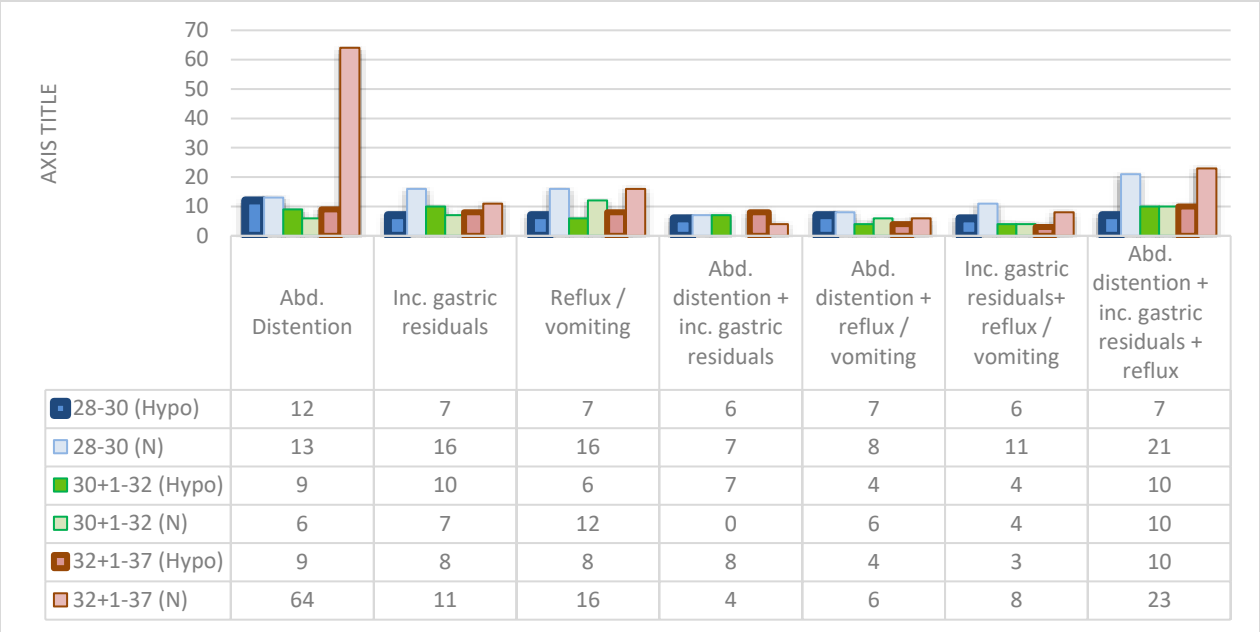
All relevant data (maternal and neonatal) was collected on a specially designed proforma. Maternal demographic and clinical data included age, parity, educational qualification, residence, previous preterm baby, job status, diabetes, hypertensive disorders, chronic ailment, multifetal gestation. Neonatal data included gender, gestation

age, birth weight, growth centiles, diagnosis, APGAR score, non-invasive (NIV) and invasive ventilation with duration, and caffeine. Parameters assessed for feed establishment were the type of milk as maternal own milk (MOM) vs formula milk and mixed, amount and route of milk (oral, gavage), age (gestational, chronological, and postmenstrual age (PMA), daily increments and signs of feed intolerance. Data was collected in predesigned proforma and analysis was done by using Statistical Package for the Social Sciences (SPSS) software, version 16.0 (SPSS Inc., Chicago, IL). Categorical variables were expressed as absolute and relative frequencies. Shapiro Wilk test was used to assess the normality of the distribution of continuous variables. Symmetrically distributed variables were described as mean and standard deviation (+SD), while asymmetrically distributed variables were described by means of medians and interquartile range (IQR). Fisher exact test was used to compare categorical variables. Student's t-test was used to compare symmetrically distributed quantitative variables, while the Mann- Whitney test was used for symmetrically distributed variables.

Ethical approval was taken from the Institutional Review Board of Fatima Memorial Hospital (FMH-14/03/2023-IRB-1190)

Results

In our study, 475 preterm neonates got admitted to the neonatal ICU, of which 217 were excluded, leaving 153 initially enrolled. From this group, 10 were further excluded due to issues such as the absence of fT4 alongside TSH, 9 experienced electrolyte imbalances later, 3 developed surgical abdominal issues, 16 had incomplete data, and 13 withdrew their participation after giving initial consent. Consequently, the data of 102 preterm neonates with feeding intolerance were analysed. Of these, 38 (37.3%) were diagnosed with hypothyroidism, while 64 had euthyroid status. All neonates diagnosed with hypothyroidism were started on thyroxine therapy. They were monitored for clinical response as “feeding tolerance”. The relationship between gestational age, thyroid status (hypothyroidism vs. normal), and feeding intolerance features was further illustrated in Graph 1. Characteristics of feeding intolerance, such as the kind of feed, total time taken to establish full feeding, and abdominal distention (whether isolated or combined with increased residue or reflux) were significantly associated with hypothyroidism, all with p-values < 0.005 (table I). Statistically insignificant factors were age at commencement of feeding PMA, feeding route, time of onset of FI, and feed amount at intolerance. Among neonatal factors, the APGAR score at 5 minutes ($p < 0.001$), jaundice (JNN) ($p < 0.001$), patent ductus arteriosus (hsPDA) ($p = 0.006$), and length of hospital stay ($p = 0.002$) were significantly associated with hypothyroidism (table II). Factors such as ventilation (invasive as well as non-invasive), hypoglycemia, congenital heart disease, surfactant, caffeine, inotropic support, mode of delivery and antenatal abnormal doppler studies. With reference to birth weight, gestation age, weight for gestation age and gender, both are identical. The univariate analysis identified maternal parity ($p < 0.001$), antepartum hemorrhage ($p = 0.002$), and chorioamnionitis ($p = 0.024$) as statistically significant risk factors for neonatal hypothyroidism (table III). Statistically insignificant maternal factors were Maternal age, residence, education, job status, Previous preterm, GDM, PIH, anemia, chronic ailment, singleton pregnancy, PPROM>24 hours, antenatal steroids and mode of delivery. The comparison of maternal, neonatal, and feeding-related characteristics based on thyroid status revealed no significant differences in maternal age, neonatal weight, gestational age, postmenstrual age (PMA), and chronological age (ChA) at the start of feeding between the hypothyroid and euthyroid groups (table I-III).



Graph1: Gestation age vs feed intolerance vs thyroid status

Table I: Feeding intolerance characteristics in neonates with hypothyroidism				
Feeding characteristics		**Hypothyroidism**		**p value**
		Yes	**No**	
Chronological age at commencement of feeding (hours)	<48	0(0%)	1(100%)	**0.148**
	48-96	27(33.3%)	54(66.7%)	
	>96	11(55%)	9(45%)	
Type of feed	Mother feed	24(53.3%)	21(46.7%)	**0.004**
	Formula	5(16.1%)	26(83.9%)	
	Mixed	9(34.6%)	17(65.4%)	
Time taken for full feed establishment (days)	3 days	3(60%)	2(40%)	**0.010**
	4-6 days	12(30%)	28(70%)	
	7-9 days	12(28.6%)	30(71.4%)	
	10-12 days	7(63.6%)	4(36.4%)	
	13-15 days	4(100%)	0(0.0%)	
Abdominal distention	Yes	30(47.6%)	33(52.4%)	**0.006**
	No	8(20.5%)	31(79.5%)	
Increased gastric residue	Yes	25(42.4%)	34(57.6%)	**0.210**
	No	13(30.2)	30(69.8%)	
Reflux/vomiting	Yes	21(32.3%)	44(67.7%)	**0.171**
	No	12(45.9%)	20(54.2%)	
Abdominal distention + increased residue	Yes	21(65.6%)	11(34.4%)	**0.000**
	No	17(24.3%)	53(75.7%)	
Abdominal distention + reflux	Yes	15(42.9%)	20(57.1%)	**0.398**
	No	23(34.3%)	44(65.7%)	
Reflux + increased residue	yes	13(36.1%)	25(37.9%)	**0.860**
	No	23(63.9%)	41(62.1%)	
Abdominal distension + gastric residues + reflux	Yes	27(31.5%)	59(68.6%)	**0.005**
	No	11(68.8%)	5(31.2%)	

Table II: Neonatal characteristics with feeding intolerance				
Neonatal characteristics		**Hypothyroidism**		**p value**
		Yes	**No**	
APGAR @5 minutes (out of 10)		8 (7 - 9)	7 (6 – 8)	**0.000**
Jaundice (JNN)	Yes	24(66.7%)	12(33.3%)	**0.000**
	No	14(21.2%)	52(78.8%)	
	No	15(32.6%)	31(67.4%)	
Hemodynamically significant Patent ductus arteriosus (HsPDA)	Yes	16(59.3%)	11(40.7%)	**0.006**
	No	22(29.3%)	53(70.7%)	
Sepsis	Yes	17(29.3%)	41(70.7%)	**0.057**
	No	21(47.7%)	23(52.3%)	
Intraventricular hemorrhage (IVH)		38(37.3%)	64(62.7%)	**NA**
Seizures	Yes	1(100%)	0(0.0%)	**0.192**
	No	37(36.6%)	62(63.4%)	
Pack cell transfusion	Yes	11(52.4%)	10(47.6%)	**0.108**
	No	27(33.3%)	54(66.7%)	
Post-natal steroids	Yes	5(71.4%)	2(28.6%)	**0.053**
	No	33(34.4%)	62(65.3%)	
Duration of stay (days)		12 (9 – 20)	15 (10 – 16)	**0.002**

Table III: Maternal characteristics with feeding intolerance				
Maternal characteristics		**Hypothyroidism**		**p value**
		Yes	**No**	
Parity	Primigravida	30(53.6%)	26 (46.4%)	**0.000**
	Multigravida	8 (17.4%)	38 (82.6%)	
Antepartum hemorrhage	Yes	14(66.7%)	7(33.3%)	**0.002**
	No	24(29.6%)	57(70.4%)	
Chorio amnionitis	Yes	9(64.3%)	5(35.7%)	**0.024**
	No	29(33.0%)	59(67.0%)	
Antenatal steroids	Complete	33(41.2%)	47(58.8%)	**0.112**
	Incomplete	5(22.7%)	17(77.3%)	
	No	2(40.0%)	3(60.0%)	
Maternal thyroid (euthyroid)		38(37.3%)	64(62.7%)	
Maternal thyroxine intake	Yes	2 (100%)	0(0.0%)	**0.064**
	No	36(36.0%)	64(64.0%)	

Discussion

Following birth, neonates must rapidly shift from “placental” to “enteral” nutrition, necessitating a crucial phase of gastrointestinal adaptation to sustain ex-utero life. FI is difficulty in digesting enteral feedings that present as abdominal distension, increased gastric residuals, and/or

reflux.[4] FI is a frequent complication in preterm neonates, associated with prolonged parenteral nutrition, increased risk of sepsis, and adverse clinical outcomes. Identifying modifiable factors contributing to feeding intolerance in hemodynamically stable preterm infants is essential to optimize nutritional management and improve outcomes.[13]

In our study, we have tried to determine the association of unexplained feeding intolerance with congenital hypothyroidism in preterm neonates.

Globally, the prevalence of congenital hypothyroidism (CH) in developed countries is approximately 1 in 3,000 to 4,000, with a 127% increase over the past 50 years.[12] In India, CH prevalence is 0.97 per 1,000 neonates in non-endemic areas and 79 per 1,000 in endemic regions.[14] A study from Pakistan reports a prevalence rate of 16%.[15] In our study, the incidence of hypothyroidism among preterm neonates between 28-34 weeks of gestation with feeding intolerance was 37.3%. This highlights the considerable variation in hypothyroidism incidence across different studies. The relatively high incidence observed in our study may be attributed to the study protocol, which specifically included neonates between 28-34 weeks gestation and excluded those outside this range (either <28 weeks or >34 weeks), potentially influencing the reported incidence.

Albraik and Gupta have documented vomiting as the most common symptoms observed in neonates with FI (about 90%) followed by abdominal distension (8.7%).[3,4] On the other hand, Chen has reported combination of vomiting and abdominal distention as sign of feed intolerance.

In our study of preterm neonates with feeding intolerance, abdominal distention (whether isolated or combined with increased residue or reflux) were significantly associated with hypothyroidism. This variation could be attributed to protocol base population selection involving only preterm neonates. Literature showing relationship of hypothyroidism and pattern of feed intolerance is scare.[16]

The "thyroid-gut axis" connects thyroid and gastrointestinal system. Experimental animal studies and adult populations research have shown that hypothyroidism is linked to both anatomical and physiological impairments of the gastrointestinal tract. An animal study inducing in utero hypothyroidism via fetal thyroidectomy showed reduced stomach and small intestine weights, thinner mucosa, shorter villi, shallower crypts, and decreased brush border enzyme activity.[17]

Xu et al. noted that hypothyroidism in adults leads to reduced GIT motility.[18] Yaylali et al. found that hypothyroidism reduces lower esophageal sphincter pressure and contraction amplitude, causing dysmotility and reflux.[19] Wang et al. observed delayed gastric emptying in hypothyroid patients, likely due to smooth muscle dysfunction.[20]

It is believed that hypothyroidism may cause glycosaminoglycan accumulation in the GI tract, contributing to delayed bowel transit. In hypothyroidism, there is impaired “thyroid-enteroendocrine axis”. As a result, hormones like GLP-1 (glucagon-like peptide-1), GIP (gastric inhibitory polypeptide), and PYY (peptide YY) are down regulated resulting in impaired digestive functions and motility.[21]

Hypothyroidism can result in gut dysbiosis that stimulates Toll-like receptors signalling pathway hence leads to "leaky gut." As a result, endotoxins such as lipopolysaccharides (LPS) from Gram-negative bacteria enter the bloodstream can induce systemic inflammation, leading to "metabolic endotoxemia" and exacerbating feed intolerance.[22]

Several maternal and neonatal factors have shown to contribute to neonatal hypothyroidism in preterm neonates with feeding intolerance.

Factors leading to perinatal stress, such as antepartum haemorrhage, chorioamnionitis, and an APGAR score below 7 at 5 minutes all influence neonatal thyroid status. These stressors influence

placental function, trigger inflammatory responses, and increase stress during pregnancy, potentially contributing to hypothyroidism.[23] Our study affirms this relationship antepartum hemorrhage, chorioamnionitis, APGAR score <7 at 5 minutes and hypothyroidism in preterm neonates with feed intolerance.

There is a link between patent ductus arteriosus (PDA) and hypothyroidism. Thyroid hormone promotes PDA closure by enhancing the synthesis of proteins like von Willebrand factor, fibronectin, and endothelin-1, through the induction of mRNA expression for these endothelial proteins.[24] In our study of preterm neonates with feeding intolerance, significant association between hypothyroidism and PDA was observed.

Dai C et al have reported a strong association between hypothyroidism and neonatal jaundice. In hypothyroidism, prolonged jaundice occurs due to delayed maturation of liver enzymes, impaired bilirubin clearance, slowed gastrointestinal motility, and changes in red blood cell turnover.[25] In our cohort, significant association between hypothyroidism and neonatal jaundice was also noted.

Suggestions / Recommendations: Further research could focus on exploring the underlying mechanisms of the thyroid-gut axis in preterm neonates with feeding intolerance. Investigating potential interventions for managing hypothyroidism and feeding intolerance, as well as examining the impact of early thyroid treatment, may provide valuable insights. Additionally, exploring the role of genetic factors and biomarkers could help identify at-risk populations for targeted interventions. Long-term follow-up is needed to assess the impact of hypothyroidism on growth, feeding, and neurodevelopment. These findings emphasize that congenital hypothyroidism should be considered in the differential diagnosis of preterm neonates presenting with persistent, unexplained feeding intolerance, especially when associated with abdominal distention.

Conclusion

Among preterm neonates with feeding intolerance, 37.3% were diagnosed with hypothyroidism. Abdominal distention, whether isolated or combined with increased residue or reflux, showed a significant association with hypothyroidism. Neonatal hypothyroidism was also linked to maternal factors such as parity, antepartum hemorrhage, and chorioamnionitis, as well as neonatal characteristics like lower APGAR scores, jaundice, and hsPDA. Prompt screening and treatment for hypothyroidism in preterm neonates with feeding intolerance is essential to improve feeding outcomes and reduce morbidity.

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