J Surg Med. 2026;10(2):40-42.
https://doi.org/10.28982/josam.8270

JOSAM

Research article
https://jsurgmed.com/

Journal of Surgery and Medicine

e-ISSN: 2602-2079

Determinants of infantile hypertrophic pyloric stenosis among infants attended in tertiary referral hospital, Addis Ababa, Ethiopia

Abebe Habtamu [1], Fikreab Bisrat [1], Tenagne Million [2]

[1] Department of Pediatrics and Child Health, Addis Ababa University, College of Health Sciences, Addis Ababa, Ethiopia
[2] Out-patient Department, Meron Clinic (Private health Facility), Addis Ababa, Ethiopia

ORCID of the author(s)

AH: https://orcid.org/0009-0005-3005-4821
FB: https://orcid.org/0009-0006-4660-5672
TM: https://orcid.org/0009-0006-1375-732X

Corresponding Author
Abebe Habtamu
Department of Pediatrics and Child Health, Addis Ababa University, College of Health Sciences, Addis Ababa, Ethiopia
E-mail: tamireabebe05@gmail.com

Ethics Committee Approval
Ethical clearance was obtained from the department research ethical committee and Addis Ababa University medical faculty college of health sciences institutional review board (IRB). A formal letter was written to the registrar office from the department of pediatrics and child health to get permission to retrieve the charts.
All procedures in this study involving human participants were performed in accordance with the 1964 Helsinki Declaration and its later amendments.

Conflict of Interest
No conflict of interest was declared by the authors.

Financial Disclosure
The authors declared that this study has received no financial support.

Published
2026 February 7

Abstract

Background/Aim: Infantile hypertrophic pyloric stenosis (IHPS) is a common surgical condition in early infancy, affecting approximately 2 per 1,000 live births. Despite its prevalence, limited data are available regarding the factors contributing to its development. This study aimed to identify determinants associated with IHPS among infants treated at Tikur Anbessa Specialized Hospital in Addis Ababa, Ethiopia, in 2021.
Methods: A retrospective, hospital-based case-control study was conducted from June to September 2021. A total of 466 infants—233 cases and 233 controls—were randomly selected. Data were collected using a structured checklist via the Open Data Kit (ODK) platform and analyzed using SPSS version 25. Bivariable and multivariable binary logistic regression analyses were performed to identify significant predictors of IHPS. Statistical significance was set at $P<0.05$, and adjusted odds ratios (AORs) with 95% confidence intervals (CIs) were reported.
Results: Five independent determinants of IHPS were identified: male sex (AOR=2.09; 95% CI: 1.38–3.17), first-born status (AOR=2.02; 95% CI: 1.36–3.00), cesarean delivery (AOR=1.74; 95% CI: 1.05–2.89), bottle feeding (AOR=6.08; 95% CI: 2.85–12.98), and blood group O (AOR=2.40; 95% CI: 1.05–5.49).
Conclusion: Male sex, first-born status, cesarean delivery, bottle feeding, and blood group O were significantly associated with IHPS. These findings suggest that both genetic and environmental factors contribute to its development. Promoting exclusive breastfeeding is recommended unless contraindicated. Further research is warranted to explore additional etiological factors and inform preventive strategies.

Keywords: infantile hypertrophic pyloric stenosis, IHPS, determinants, case-control study, Ethiopia

How to cite: Tamire A, Bisrat F, Million T. Determinants of infantile hypertrophic pyloric stenosis among infants attended in tertiary referral hospital, Addis Ababa, Ethiopia. J Surg Med. 2026;10(2):40-42.

Introduction

Infantile hypertrophic pyloric stenosis (IHPS) is a gastrointestinal disorder primarily affecting infants, characterized by hypertrophy of the pyloric muscle, which leads to partial gastric outlet obstruction. Clinically, IHPS presents with projectile, non-bilious vomiting, a palpable olive-shaped mass in the mid-epigastrium, and occasionally visible gastric peristalsis during feeding. Diagnosis is typically confirmed via ultrasonography, which allows direct visualization of the thickened pylorus. The standard treatment is pyloromyotomy, performed either through a periumbilical incision or laparoscopically, with excellent outcomes and minimal complications [1].

Although the clinical features and management of IHPS are well-established, its etiology remains poorly understood. Evidence suggests a multifactorial origin, involving both genetic and environmental influences. Male infants are disproportionately affected, and a maternal family history significantly increases risk—particularly among male offspring. Additional familial patterns include increased incidence in twins and associations with blood groups B and O [2].

Environmental exposures such as early administration of macrolide antibiotics (e.g., erythromycin, azithromycin) and formula feeding have also been implicated. Preterm birth is another potential risk factor, though its role remains unclear [3, 4].

Understanding these risk factors is essential for early identification and prevention. This study investigates the determinants of IHPS among infants in Ethiopia, aiming to inform clinical strategies and contribute to the broader body of pediatric gastroenterological research.

Materials and methods

This retrospective case-control study aimed to identify risk factors associated with infantile hypertrophic pyloric stenosis (IHPS) in a hospital-based setting. Medical records of infants admitted to Tikur Anbessa Specialized Hospital in Addis Ababa, Ethiopia, between January 2018 and December 2022 were reviewed.

Study Population and Case Definition

Cases were defined as infants diagnosed with IHPS based on clinical presentation and confirmed via ultrasonography. A total of 233 cases were identified.

Control Group Selection

An equal number of controls (n=233) were selected from infants admitted during the same period for conditions unrelated to gastrointestinal obstruction or congenital anomalies. Controls were matched to cases by age (±2 weeks) and sex to reduce confounding. Randomization was performed using a computer-generated sequence applied to the hospital admission registry. Infants with incomplete records or diagnoses potentially related to IHPS (e.g., feeding intolerance, unexplained vomiting) were excluded.

Data Collection and Variables

Data were collected using a structured checklist, capturing: Demographics: age at admission, sex, birth order, Birth history: gestational age, birth weight, mode of delivery, Feeding practices: exclusive breastfeeding, formula feeding, mixed feeding, Family history: IHPS in siblings or parents, Medication exposure: erythromycin or azithromycin within the first two weeks of life, Blood group: ABO and Rh typing, Clinical data: comorbidities, symptom duration before admission

Data abstraction was performed by trained pediatric residents under supervision, and any discrepancies were resolved through consensus.

Statistical Analysis

Data were analyzed using SPSS version 25.0. Descriptive statistics summarized case and control characteristics. Bivariate logistic regression identified variables associated with IHPS (P<0.20), which were then included in a multivariate logistic regression model. Adjusted odds ratios (AORs) with 95% confidence intervals (CIs) were reported. Statistical significance was set at P<0.05.

Ethical Considerations

Ethical approval was obtained from the Department of Research Ethical Committee. The Institutional Review Board (IRB) of Addis Ababa University waived the need for informed consent. Access to patient charts was granted by the hospital registrar's office. All procedures adhered to institutional and ethical guidelines.

Results

A total of 466 infants were included: 233 IHPS cases and 233 controls. The median age at presentation was 38 days for cases and 23 days for controls. Most IHPS cases presented between 1 and 5 months of age. Male infants constituted 63.9% of both groups, and urban residency was predominant (69.3%).

Clinical Characteristics

All IHPS cases presented with vomiting, and most showed signs of dehydration. Visible gastric peristalsis and a palpable olive-shaped mass were noted in some cases. Congenital anomalies were less frequent among IHPS cases (5.2%) compared to controls (38.2%). No cases reported maternal smoking or a family history of IHPS (Table 1).

Risk Factors

Multivariate logistic regression analysis revealed five independent risk factors significantly associated with IHPS. Male infants were more than twice as likely to develop IHPS compared to females (adjusted odds ratio [AOR]=2.09; 95% confidence interval [CI]: 1.38–3.17). First-born infants had a twofold increased risk (AOR=2.02; 95% CI: 1.36–3.00). Cesarean delivery was associated with a modest increase in risk (AOR=1.74; 95% CI: 1.05–2.89). Bottle feeding showed the strongest association, with infants who were formula-fed being over six times more likely to develop IHPS compared to those exclusively breastfed (AOR=6.08; 95% CI: 2.85–12.98). Additionally, infants with blood group O had a significantly higher risk (AOR=2.40; 95% CI: 1.05–5.49) (Table 2).

Table 1. Clinical Features of Infants with IHPS at Tikur Anbessa Specialized Hospital, Addis Ababa, Ethiopia (n=466)

Variable	Controls n (%)	Cases n (%)	Total n (%)
Vomiting	68 (29.2)	233 (100)	301 (64.6)
No Vomiting	165 (70.8)	0 (0)	165 (35.4)
Dehydration	32 (13.7)	160 (68.7)	192 (41.2)
No Dehydration	201 (86.3)	73 (31.3)	274 (58.8)
Visible Peristalsis	12 (5.2)	51 (21.9)	63 (13.5)
No Visible Peristalsis	221 (94.8)	182 (78.1)	403 (86.5)
Olive-Shaped Mass	0 (0)	91 (39.1)	91 (19.5)
No Olive-Shaped Mass	233 (100)	142 (60.9)	375 (80.5)
Congenital Anomaly	89 (38.2)	12 (5.2)	101 (21.7)
No Congenital Anomaly	144 (61.8)	221 (94.8)	365 (78.3)

Table 2. Binary and Multivariate Logistic Regression of Risk Factors Associated with IHPS

Variable	Cases n (%)	Controls n (%)	COR (95% CI)	AOR (95% CI)	*P*-value
Sex					
Male	166 (71.2)	132 (56.7)	1.89 (1.29–2.78)	2.09 (1.38–3.17)	0.01*
Female	67 (28.8)	101 (43.3)	1.0	1.0	—
Birth Order					
First-born	136 (58.4)	90 (38.6)	2.22 (1.53–3.22)	2.02 (1.36–3.00)	0.001*
Second-born and above	97 (41.6)	143 (61.4)	1.0	1.0	—
Mode of Delivery					
Cesarean Section	62 (26.6)	36 (15.5)	1.98 (1.25–3.14)	1.74 (1.05–2.89)	0.033*
Vaginal Delivery	171 (73.4)	197 (84.5)	1.0	1.0	—
Feeding Practice					
Exclusive Breastfeeding	185 (79.4)	223 (95.7)	1.0	1.0	—
Formula Feeding	48 (20.6)	10 (4.3)	5.78 (2.84–11.75)	6.08 (2.85–12.98)	<0.001*
Blood Group					
A	54 (23.2)	50 (21.5)	1.54 (0.70–3.37)	0.77 (0.30–1.90)	0.06
B	48 (20.6)	44 (18.9)	1.55 (0.70–3.45)	1.54 (0.65–3.65)	
AB	14 (6.0)	20 (8.6)	1.0	1.0	0.12
O	19 (8.2)	47 (20.2.8)	1.94 (0.92-4.10)	2.4 (1.05, 5.49)	0.038*

* Statistically significant at p-value<0.05

Discussion

The study identified male sex, first-born status, cesarean delivery, bottle feeding, and blood group O as independent determinants of IHPS. These findings are consistent with previous studies conducted in Ethiopia, China, Cameroon, and Canada, which have reported a higher prevalence of IHPS among male infants [5–8]. The sex-modified inheritance pattern proposed by Mitchell and Risch [9] may explain this predisposition, although the precise genetic mechanisms remain unclear.

The increased risk among first-born infants aligns with meta-analyses and international literature [1, 3], possibly reflecting differences in maternal experience, hormonal factors, or feeding practices during initial pregnancies.

Cesarean delivery was associated with a higher likelihood of IHPS, potentially due to delayed initiation of breastfeeding. Studies have shown that infants born via cesarean section are less likely to be breastfed early [10], and since breastfeeding is protective against IHPS, this delay may contribute to increased risk.

Bottle feeding demonstrated the strongest association with IHPS, consistent with findings from Canada, Australia, Italy, and the United States [5, 11–13]. Proposed mechanisms include the absence of pyloric-relaxing hormones in formula, elevated plasma gastrin levels, and reduced gastric motility in formula-fed infants.

The association between blood group O and IHPS supports earlier studies from the UK, Iraq, and Denmark [14–16]. One hypothesis involves increased production of alkaline phosphatase in response to fatty meals, which may influence pyloric muscle activity in individuals with blood groups O and B.

Recommendations

Healthcare professionals should maintain heightened clinical awareness when evaluating infants who present with risk factors identified in this study. Promoting exclusive breastfeeding is particularly important, given its strong protective effect against IHPS. Public health messaging should be culturally sensitive and avoid prescriptive language, recognizing that feeding choices may be influenced by medical, social, or economic factors.

Obstetric and pediatric care teams should support vaginal delivery when medically appropriate and encourage timely initiation of breastfeeding following both vaginal and cesarean births. Further research is warranted to explore the potential role of macrolide antibiotics in IHPS development and to investigate genetic predispositions that may inform risk stratification and preventive strategies.

Conclusion

Male sex, first-born status, cesarean delivery, bottle feeding, and blood group O were independently associated with IHPS. These findings underscore the multifactorial nature of IHPS and highlight the importance of early recognition and preventive strategies.

Although macrolide use was not significant in multivariate analysis, its association in univariate analysis warrants further investigation.

References

1. Zhu J, Zhu T, Lin Z, Qu Y, Mu D. Perinatal risk factors for infantile hypertrophic pyloric stenosis: A meta-analysis. J Pediatr Surg. 2017;52(9):1389–97.
2. Chung E. Infantile hypertrophic pyloric stenosis: Genes and environment. Arch Dis Child. 2008;93(12):1003–4.
3. Tadesse A. Infantile hypertrophic pyloric stenosis: A retrospective study from a tertiary hospital in Ethiopia. East Cent Afr J Surg. 2014;19(1):120–4.
4. Eberly MD, Eide MB, Thompson JL, Nylund CM. Azithromycin in early infancy and pyloric stenosis. Pediatrics. 2015;135(3):483–8.
5. Krogh C, Biggar RJ, Fischer TK, Lindholm M, Wohlfahrt J, Melbye M. Bottle-feeding and the risk of pyloric stenosis. Pediatrics. 2012;130(4):e943–9.
6. Li J, Gao W, Zhu J, Zuo W, Liu X. Epidemiological and clinical characteristics of 304 patients with infantile hypertrophic pyloric stenosis in Anhui Province of East China, 2012–2015. J Matern Fetal Neonatal Med. 2018;31(20):2742–7.
7. Lulseged D. Infantile hypertrophic pyloric stenosis in a children's hospital: A retrospective study. Ethiop Med J. 1986;24(4):169–73.
8. Ndongo R, Tolefac PN, Tambo FFM, Abanda MH, Ngowe MN, Fola O, et al. Infantile hypertrophic pyloric stenosis: A 4-year experience from two tertiary care centres in Cameroon. BMC Res Notes. 2018;11(1):18–22.
9. Mitchell LE, Risch N. The genetics of infantile hypertrophic pyloric stenosis: A reanalysis. Am J Dis Child. 1993;147(11):1203–11.
10. Svenningsson A, Svensson T, Akre O, Nordenskjöld A. Maternal and pregnancy characteristics and risk of infantile hypertrophic pyloric stenosis. J Pediatr Surg. 2014;49(8):1226–31.
11. Wayne C, Hung JHC, Chan E, Sedgwick I, Bass J, Nasr A. Formula-feeding and hypertrophic pyloric stenosis: Is there an association? A case-control study. J Pediatr Surg. 2016;51(5):779–82.
12. Pisacane A, De Luca U, Criscuolo L, Vaccaro F, Valiante A, Inglese A, et al. Breastfeeding and hypertrophic pyloric stenosis: Population-based case-control study. BMJ. 1996;312(7033):745–6.
13. McAteer JP, Ledbetter DJ, Goldin AB. Role of bottle feeding in the etiology of hypertrophic pyloric stenosis. JAMA Pediatr. 2013;167(12):1143–9.
14. Dodge JA. ABO blood groups and infantile hypertrophic pyloric stenosis. BMJ. 1967;4(5582):781–2.
15. Hagos C, Mengistu H. Congenital (infantile) hypertrophic pyloric stenosis (IHPS). Ethiop J Pediatr Child Health. 2006;3(1):5–8.
16. Rasmussen L, Green A, Hansen LP. The epidemiology of infantile hypertrophic pyloric stenosis in a Danish population, 1950–84. Int J Epidemiol. 1989;18(2):413–7.