

Effects of Maternal and Socio-economic Characteristics on Infantile Hypertrophic Pyloric Stenosis

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Abstract

Background

Infantile hypertrophic pyloric stenosis (IHPS) is one of the leading causes of gastric outlet obstruction among young infants, and potential etiological factors include the child's sex, maternal parity, maternal age, social class, and other maternal conditions, although their significance differs between communities. This study aimed to assess whether socio-demographic, maternal, and clinical factors are associated with the development of IHPS.

Methods

A total of 90 mother-infant dyads were included in this study. IHPS was confirmed by clinical assessment and abdominal ultrasound. Data on maternal age, infant sex, parity, socioeconomic status, maternal education, maternal occupation, maternal disease (diabetes, hypertension), and maternal residence were recorded. Associations were analyzed using logistic regression to determine odds ratios (OR) and 95% confidence intervals (CI).

Results

The prevalence of IHPS was 11.1% (10/90). All cases occurred among mothers aged ≥ 26 years, with equal proportions in the 26–30 and ≥ 31 years groups (5.6% each). Most affected infants were male (8.9%), first-born (6.7%), from lower socioeconomic families (8.9%), and from rural areas (10.0%). All cases occurred among employed mothers. Maternal diabetes (10.0%) and hypertension (1.1%) were common among positive cases. Binary logistic regression identified maternal age ≥ 26 years (OR = 22.06, $p < 0.035$), primary education (OR = 4.76, $p < 0.048$), employment (OR = 3381.00, $p < 0.001$), diabetes (OR = 3059.00, $p < 0.001$), and hypertension (OR = 483.00, $p < 0.004$) as significant predictors of IHPS. Male sex (OR = 3.40) and lower socioeconomic status (OR = 2.16) showed increased odds but were not statistically significant.

Conclusions

This study demonstrated a relatively high prevalence of IHPS (11.1%) among the infants studied. Maternal factors, particularly age ≥ 26 years, employment status, diabetes, hypertension, and lower educational attainment, emerged as significant determinants of IHPS risk. The observed predominance of male infants, first-born children, and infants from lower socioeconomic and rural backgrounds further suggests demographic disparities in disease occurrence.

Background

Infantile hypertrophic pyloric stenosis (IHPS) is the leading cause of gastric outlet obstruction in infancy, characterized by progressive hypertrophy and hyperplasia of the circular and longitudinal muscular layers of the pylorus, resulting in a narrowed lumen and functional obstruction. Infants with IHPS typically present between 3 and 6 weeks of age with progressively worsening, non-bilious, projectile vomiting, which can rapidly lead to dehydration, electrolyte imbalance (hypochloremic, hypokalemic metabolic alkalosis), and failure to thrive if not promptly diagnosed and treated. The definitive treatment is surgical pyloromyotomy, which is associated with excellent outcomes, although delayed presentation may lead to increased morbidity and prolonged hospital stay [1].

The incidence of IHPS varies geographically and among ethnic groups. In Western populations, the reported incidence ranges from 2 to 5 per 1,000 live births. In the United States, a large population-based study using data from 11 birth defect surveillance programs estimated the overall prevalence of IHPS at 20.09 per 10,000 live births (95% CI 19.87–20.32) between 1999 and 2010. Marked racial and ethnic differences have been observed, with the highest incidence reported in white infants (2.4 per 1,000), followed by Hispanic (1.8 per 1,000), Black (0.7 per 1,000), and Asian (0.6 per 1,000) infants [2].

Despite a well-recognized clinical picture, the exact etiology of IHPS remains incompletely understood. Current evidence suggests a multifactorial pathogenesis involving both genetic predisposition and environmental influences. The strong male predominance, elevated recurrence risk among siblings, and higher concordance rates in monozygotic versus dizygotic twins indicate a substantial genetic component, yet no single causative gene has been consistently identified, suggesting considerable genetic heterogeneity. Environmental risk factors that have been repeatedly implicated include maternal smoking during pregnancy, bottle/formula feeding, early exposure to macrolide antibiotics (e.g., erythromycin), and certain perinatal characteristics such as preterm birth and cesarean delivery [3, 4].

Given the multifactorial nature of IHPS, a comprehensive understanding of its epidemiological profile—including the influence of demographic, socioeconomic, and parental factors—is essential for early recognition, prompt referral, and optimal management of affected infants. The present study was therefore undertaken to describe the epidemiological characteristics of IHPS at a single institution, with specific focus on maternal age, infant sex, parity, socioeconomic status, maternal educational level, child's age at diagnosis, and maternal occupation [5–7].

A growing body of evidence indicates that several demographic and parental factors are associated with the risk of developing IHPS. Understanding the distribution of these factors in a local cohort is critical for identifying high-risk groups, guiding clinical suspicion, and informing public health strategies [8]. The strong male predominance of IHPS is one of the most consistent epidemiological findings, with reported male-to-female ratios ranging from 4:1 to 5:1 and as high as 7:1 in some series. Most affected infants are also first-born, with firstborn status reported in 30% to 40% of all cases and as high as 57.8% in some cohorts. This predilection for first-born males suggests that factors related to birth order and sex-specific biological mechanisms may play important roles in pathogenesis [9]. Maternal age has emerged as a significant independent risk factor for IHPS. Young maternal age, particularly younger than 20 years,

has been associated with a 29% to 42% increased risk of IHPS. Conversely, some studies have reported a decreased risk among older mothers, although the evidence has not been entirely consistent. A large, population-based US study found that the adjusted prevalence ratio for IHPS increased with decreasing maternal age [10, 11].

The role of socioeconomic status (SES) in IHPS has received increasing attention in recent years. Low SES, often measured using median household income, has been directly correlated with a higher incidence of pyloric stenosis. In a case-control study from Illinois, as median household income decreased, the odds of pyloric stenosis significantly increased, with a protective effect of breastfeeding observed even after controlling for SES. Moreover, disparities in insurance status and social determinants of health have been shown to affect clinical outcomes: infants with public insurance or lower SES are more likely to present with metabolic derangements, require longer hospitalization, and experience delays in diagnosis and treatment [12, 13].

Maternal educational level is a proxy for health literacy and access to care, and its association with IHPS has been examined in several studies. A systematic review and meta-analysis reported that lower maternal educational level is a significant risk factor for IHPS. In the large US birth defects surveillance study, adjusted prevalence ratios for IHPS increased with decreasing maternal education. Maternal occupation, especially occupational exposures during pregnancy, has been less studied but may provide insight into environmental or occupational risk factors that contribute to IHPS pathogenesis [14, 15].

The classical age at presentation for IHPS is between 3 and 8 weeks of life, with a mean age at diagnosis of approximately 4 to 5.5 weeks in contemporary series. Earlier or later presentation may have implications for diagnostic delay and clinical outcome. Delayed diagnosis is associated with worse metabolic derangement, longer hospital stay, and increased healthcare costs. While numerous studies have described the epidemiology of IHPS in North American and European populations, data from many regions—particularly low- and middle-income countries—remain sparse. Furthermore, the interplay between demographic factors (sex, parity, maternal age), socioeconomic variables (SES, maternal education, maternal occupation), and clinical characteristics (age at diagnosis) has seldom been examined simultaneously in a single cohort.

The present study was therefore designed to provide a comprehensive, contemporary description of these key epidemiological parameters in a defined patient population. The main aim of this research was to assess whether socio-demographic, maternal, and clinical factors are associated with the development of IHPS.

Methods

Study design and setting

This case-control study was conducted over a 1-year period at Rajshahi Medical College Hospital (RMCH), Bangladesh.

Study population

Patients admitted to RMCH with suspected infantile hypertrophic pyloric stenosis were eligible for inclusion.

Inclusion and exclusion criteria

Infants aged 0–6 months whose parents/guardians were willing to undergo ultrasonographic examination and provide informed consent were included. Patients whose parents/guardians were unwilling to participate or provide informed consent were excluded.

Sample size and sampling method

A sample of 90 mother-infant dyads was selected based on the prevalence of IHPS and patient turnover at RMCH, using standard prevalence estimates and statistical confidence intervals. Purposive sampling was used to select patients meeting the inclusion criteria, aiming for diverse representation across risk factors (e.g., timing and type of delivery, sex, medication history, feeding history).

Data collection

Clinical data, including demographic details (age, sex) and maternal medical history (hypertension, diabetes, cardiovascular disease) and lifestyle factors (smoking, alcohol consumption, physical activity), were recorded from a detailed patient and maternal history. All patients underwent high-resolution abdominal Doppler ultrasonography to assess pyloric length and diameter, pyloric muscle thickness, vascularity of the pyloric muscle and mucosa, and the presence of other lesions.

A structured questionnaire, designed for this study, was used to assess potential risk factors contributing to IHPS, including timing and type of delivery, maternal illness, socioeconomic status, maternal smoking and alcohol habits, formula feeding, and family history.

Statistical analysis

Descriptive statistics and the chi-square test were used to analyze associations between categorical variables, including the presence of IHPS and candidate risk factors. Binary logistic regression analysis was used to identify independent risk factors for IHPS. Data were analyzed using IBM SPSS Statistics, version 29.

Results

Of the 90 participants, 10 (11.1%) were diagnosed with IHPS, while 80 (88.9%) were negative for the disease. No cases of IHPS were observed among mothers aged ≤ 20 years or 21–25 years; positive

cases were identified only among mothers aged 26–30 years (5.6%) and ≥ 31 years (5.6%), indicating that higher maternal age groups had comparatively more IHPS cases. Male infants showed a higher prevalence of IHPS than female infants: 8 of 90 (8.9%) male infants were positive, compared with 2 of 90 (2.2%) female infants. First-born children had the highest proportion of IHPS cases (6/90, 6.7%); the frequency decreased with increasing parity, with second parity accounting for 3 (3.3%) cases and third parity for 1 (1.1%) case, while no cases were observed among fourth-parity births.

Infants from lower socioeconomic families had a greater proportion of IHPS cases (8/90, 8.9%) than those from middle-class families (2/90, 2.2%). Maternal education also appeared to influence IHPS occurrence: mothers with primary education had 5 (5.6%) positive cases, higher than mothers with secondary, higher-secondary, or graduate-level education, among whom only one positive case (1.1%) was observed in each category, alongside one case among mothers with no formal education. All positive IHPS cases occurred among employed mothers (10/90, 11.1%); no cases were observed among self-employed mothers. IHPS was more common among mothers with diabetes (9/90, 10.0%) than among those with hypertension (1/90, 1.1%); no positive cases were reported among mothers without either condition. Rural participants had a higher prevalence of IHPS (9/90, 10.0%) than urban participants (1/90, 1.1%).

Binary logistic regression was used to evaluate the independent effects of socio-economic and demographic factors on IHPS occurrence, with odds ratios (OR) and 95% confidence intervals (CI) calculated for each predictor. Maternal age ≥ 26 years was significantly associated with IHPS (OR = 22.06, $p < 0.035$). Sex was not significantly associated with IHPS, although male infants had higher odds than female infants (OR = 3.40, $p = 0.104$). Lower socioeconomic status was not significantly associated with IHPS relative to middle-class status (OR = 2.16, $p = 0.306$). Among maternal education categories, only primary education showed a statistically significant association with IHPS (OR = 4.76, $p < 0.048$); no formal education, higher-secondary education, and graduate-level education were not significantly associated with the disease. Maternal employment was a highly significant predictor of IHPS relative to self-employment (OR = 3381.00, $p < 0.001$). Maternal diabetes (OR = 3059.00, $p < 0.001$) and hypertension (OR = 483.00, $p < 0.004$) were both significantly associated with increased odds of IHPS. Rural residence was not significantly associated with IHPS relative to urban residence, despite higher odds among rural participants (OR = 2.59, $p = 0.297$).

Discussion

The present study investigated the association between socio-economic and demographic factors and IHPS. Maternal age, maternal education, maternal occupation, and maternal illness were significantly associated with IHPS, whereas sex, socioeconomic status, and residence were not statistically significant in the regression analysis. These findings are relevant to understanding the multifactorial etiology of IHPS and to identifying vulnerable populations for early diagnosis and intervention.

In this study, maternal age ≥ 26 years was significantly associated with increased odds of IHPS, with approximately 22-fold higher odds compared with younger mothers. Previous studies have similarly reported that advanced maternal age may contribute to an increased risk of congenital and gastrointestinal disorders among infants, potentially through genetic and environmental interactions or hormonal and metabolic changes during pregnancy that affect fetal gastrointestinal development [14, 15].

Sex distribution in the present study showed a higher proportion of IHPS among male infants, although the association was not statistically significant on regression analysis. This finding is consistent with the existing literature, which has consistently demonstrated a strong male predominance in IHPS, with male infants reported to be approximately four to five times more likely to develop IHPS than females [16]. The exact mechanism underlying this sex difference remains unclear; genetic susceptibility, hormonal influences, and differences in smooth-muscle physiology have been proposed [16, 17].

Parity analysis revealed that first-born children had the highest proportion of IHPS cases, consistent with previous epidemiological studies reporting an increased risk of pyloric stenosis among first-born infants relative to later-born children [18, 19]. This increased risk may be associated with maternal physiological adaptation during the first pregnancy, feeding practices, or environmental exposures during the neonatal period.

Socioeconomic status in the current study showed higher odds of IHPS among infants from lower socioeconomic families than middle-class families, although the relationship was not statistically significant. Similar observations have been reported in developing countries, where lower socioeconomic conditions are often associated with limited healthcare access, poor nutritional status, and delayed diagnosis; however, evidence regarding socioeconomic status and IHPS remains inconsistent across studies, and some investigators have reported no direct relationship, suggesting that biological and genetic factors may play a more dominant role than economic conditions alone [20].

Maternal education demonstrated an important association with IHPS in this study: mothers with primary education had significantly higher odds of having infants with IHPS than mothers with secondary education. Lower educational attainment may influence maternal awareness of infant feeding practices, early disease symptoms, and timely healthcare-seeking behavior. Educated mothers are generally more likely to recognize symptoms early and seek prompt medical care, potentially reducing disease complications [21, 22].

One of the most notable findings of this study was the strong association between maternal occupation and IHPS: infants of employed mothers had markedly increased odds of IHPS compared with infants of self-employed mothers. Although few studies have directly examined maternal occupation and IHPS, occupational stress, altered feeding practices, and reduced maternal-infant interaction due to work commitments may indirectly influence infant health outcomes; employed mothers may also rely more on formula feeding, which has been suggested as a potential risk factor for IHPS in some studies [23, 24].

Maternal illness, particularly diabetes and hypertension, showed strong associations with IHPS in this study. Maternal metabolic disorders may affect fetal growth and smooth-muscle development during pregnancy; maternal diabetes can alter fetal gastrointestinal motility and increase the risk of neonatal complications, while maternal hypertension may impair placental circulation, thereby influencing fetal development and contributing to gastrointestinal abnormalities [25, 26].

The present study also demonstrated a higher prevalence of IHPS among rural residents than urban residents, although the association was not statistically significant. Rural populations often experience barriers to healthcare access, delayed diagnosis, and lower health awareness; similar findings have been observed in resource-limited settings, where rural residence contributes to delayed treatment and poorer neonatal outcomes. Nevertheless, the lack of statistical significance suggests that residence alone may not independently predict IHPS when other socio-demographic variables are considered [27, 28].

These findings are clinically relevant because they emphasize the importance of maternal and socio-demographic characteristics in the occurrence of IHPS. Early identification of high-risk groups—particularly infants born to older mothers, mothers with diabetes or hypertension, and mothers with lower educational status—may facilitate timely diagnosis and management. Health education programs focusing on maternal awareness, antenatal care, and neonatal monitoring may help reduce disease burden and improve infant outcomes [29, 30].

This study has several limitations. The sample size was relatively small, which may have affected the precision of estimates and contributed to the wide confidence intervals observed in the regression analysis. The design also limits the ability to establish causal relationships [31, 32]. Additionally, some variables may have been influenced by recall bias or incomplete clinical documentation. Future multicenter studies with larger sample sizes and prospective designs are recommended to further clarify the role of socio-economic and maternal factors in the etiology of IHPS.

Conclusions

This study demonstrated a relatively high prevalence of IHPS (11.1%) among the infants studied. Maternal factors, particularly age ≥ 26 years, employment status, diabetes, hypertension, and lower educational attainment, emerged as significant determinants of IHPS risk. The observed predominance of male infants, first-born children, and infants from lower socioeconomic and rural backgrounds further suggests demographic disparities in disease occurrence. Strengthening maternal healthcare services, improving management of chronic maternal conditions during pregnancy, and enhancing awareness among healthcare providers may contribute to earlier identification and better outcomes for infants at risk of IHPS.

Abbreviations

IHPS: Infantile hypertrophic pyloric stenosis; OR: Odds ratio; CI: Confidence interval; USG: Ultrasonography; SES: Socioeconomic status; SPSS: Statistical Package for the Social Sciences

Declarations

Ethics approval and consent to participate

The study was conducted in accordance with the Declaration of Helsinki. Ethical approval was obtained from the Institutional Review Board/Ethics Committee of the Institute of Biological Sciences, University of Rajshahi, Bangladesh. Written informed consent was obtained from all participating mothers or legal guardians before enrollment.

Consent for publication

Not applicable.

Availability of data and materials

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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The authors received no specific funding for this work.

Authors' contributions

A.K.M.A.K. conceived and designed the study, collected data, performed statistical analyses, interpreted the results, and drafted the manuscript. M.A.H. contributed to study design, supervised the research activities, interpreted the findings, and critically revised the manuscript for important intellectual content. M.M.R. contributed to study design, data interpretation, manuscript preparation, critical revision of the manuscript, and overall supervision of the study. All authors read and approved the final manuscript.

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References

1. Krogh C, Gørtz S, Wohlfahrt J, Biggar RJ, Melbye M, Fischer TK. Pre- and perinatal risk factors for pyloric stenosis and their influence on the male predominance. *Am J Epidemiol*. 2012;176(1):24–31.
2. Pedersen RN, Garne E, Loane M, Korsholm L, Husby S. Infantile hypertrophic pyloric stenosis: a comparative study of incidence and other epidemiological characteristics in seven European regions. *J Matern Fetal Neonatal Med*. 2008;21(9):599–604.
3. Ranells JD, Carver JD, Kirby RS. Infantile hypertrophic pyloric stenosis: epidemiology, genetics, and clinical update. *Adv Pediatr*. 2011;58(1):195–206.
4. Sommerfield T, Chalmers J, Youngson G, Heely C, Fleming M, Thomson G. The changing epidemiology of infantile hypertrophic pyloric stenosis in Scotland. *Arch Dis Child*. 2008;93(12):1007–11.
5. Wang J, Waller DK, Hwang LY, Taylor LG, Canfield MA. Prevalence of infantile hypertrophic pyloric stenosis in Texas, 1999–2002. *Birth Defects Res Clin Mol Teratol*. 2008;82(10):736–67.
6. Obaid YY, Toubasi AA, Albustanji FH, Al-Qawasmeh AR. Perinatal risk factors for infantile hypertrophic pyloric stenosis: a systematic review and meta-analysis. *J Pediatr Surg*. 2023;58(3):458–66.
7. 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.
8. Sørensen HT, Nørgard B, Pedersen L, Larsen H, Johnsen SP. Maternal smoking and risk of hypertrophic pyloric stenosis: 10 year population based cohort study. *BMJ*. 2002;325(7371):1011–2.
9. Applegate MS, Druschel CM. The epidemiology of infantile hypertrophic pyloric stenosis in New York State, 1983 to 1990. *Arch Pediatr Adolesc Med*. 1995;149(10):1123–9.
10. Persson S, Ekblom A, Granath F, Nordenskjöld A. Parallel incidences of sudden infant death syndrome and infantile hypertrophic pyloric stenosis: a common cause? *Pediatrics*. 2001;108(4):e70.
11. Fadista J, Skotte L, Geller F, Bybjerg-Grauholm J, Gørtz S, et al. Genome-wide meta-analysis identifies *BARX1* and *EML4-MTA3* as new loci associated with infantile hypertrophic pyloric stenosis. *Hum Mol Genet*. 2019;28(2):332–40.
12. Everett KV, Chioza BA, Georgoula C, Reece A, Gardiner RM, Chung EMK. Infantile hypertrophic pyloric stenosis: evaluation of three positional candidate genes, *TRPC1*, *TRPC5* and *TRCP6*, by association analysis and re-sequencing. *Hum Genet*. 2009;126(6):819–31.
13. MacMahon B. The continuing enigma of pyloric stenosis of infancy: a review. *Epidemiology*. 2006;17(2):195–201.

14. Panteli C. New insights into the pathogenesis of infantile pyloric stenosis. *Pediatr Surg Int*. 2009;25(12):1043–52.
15. [UNRESOLVED. — see note] Clinical presentation and diagnosis.
16. Hernanz-Schulman M. Infantile hypertrophic pyloric stenosis. *Radiology*. 2003;227(2):319–31.
17. Aspelund G, Langer JC. Current management of hypertrophic pyloric stenosis. *Semin Pediatr Surg*. 2007;16(1):27–33.
18. Apostolidis K, Cury J, Aslanidis T. A rare case of late presentation of infantile hypertrophic pyloric stenosis in a 5-year old child. *Greek E J Perioper Med*. 2018;17(a):71–5.
19. Chalya PL, Manyama M, Kayange NM, Mabula JB, Massenga A. Infantile hypertrophic pyloric stenosis at a tertiary care hospital in Tanzania: a surgical experience with 102 patients over a 5-year period. *BMC Res Notes*. 2015;8:690.
20. [UNRESOLVED. — see note] Environmental and medication exposures.
21. Abdellatif M, Ghozy S, Kamel MG, Elawady SS, Ghorab MME, et al. Association between exposure to macrolides and the development of infantile hypertrophic pyloric stenosis: a systematic review and meta-analysis. *Eur J Pediatr*. 2019;178(3):301–14.
22. Honein MA, Paulozzi LJ, Himelright IM, Lee B, Cragan JD, Patterson L, et al. Infantile hypertrophic pyloric stenosis after pertussis prophylaxis with erythromycin: a case review and cohort study. *Lancet*. 1999;354(9196):2101–5.
23. Krogh C, Fischer TK, Skotte L, Biggar RJ, Øyen N, Skytthe A, et al. Familial aggregation and heritability of pyloric stenosis. *JAMA*. 2010;303(23):2393–9.
24. Panteli C. New insights into the pathogenesis of infantile pyloric stenosis. *Pediatr Surg Int*. 2009;25(12):1043–52.
25. Sørensen HT, Nørgård B, Pedersen L, Larsen H, Johnsen SP. Risk of infantile hypertrophic pyloric stenosis after maternal postnatal use of macrolides. *Scand J Infect Dis*. 2003;35(2):104–6.
26. Applegate MS, Druschel CM. The epidemiology of infantile hypertrophic pyloric stenosis in New York State, 1983 to 1998. *Arch Pediatr Adolesc Med*. 1995;149(10):1123–9.
27. To T, Wajja A, Wales PW, Langer JC. Population demographic indicators associated with incidence of pyloric stenosis. *Arch Pediatr Adolesc Med*. 2005;159(6):520–5.
28. MacMahon B. The continuing enigma of pyloric stenosis of infancy: a review. *Epidemiology*. 2006;17(2):195–201.
29. Chung E. Infantile hypertrophic pyloric stenosis: genes and environment. *Semin Pediatr Surg*. 2008;17(3):152–8.
30. Carter CO, Evans KA. Inheritance of congenital pyloric stenosis. *J Med Genet*. 1969;6(3):233–54.
31. Svenningsson A, Green A, Svensson JF, et al. Maternal risk factors and infantile hypertrophic pyloric stenosis. *Acta Paediatr*. 2016;105(11):1328–33.
32. Mitchell LE, Risch N. The genetics of infantile hypertrophic pyloric stenosis. *Am J Dis Child*. 1993;147(11):1203–11.

Tables

Tables 1 and 2 are available in the supplementary files section

Figures

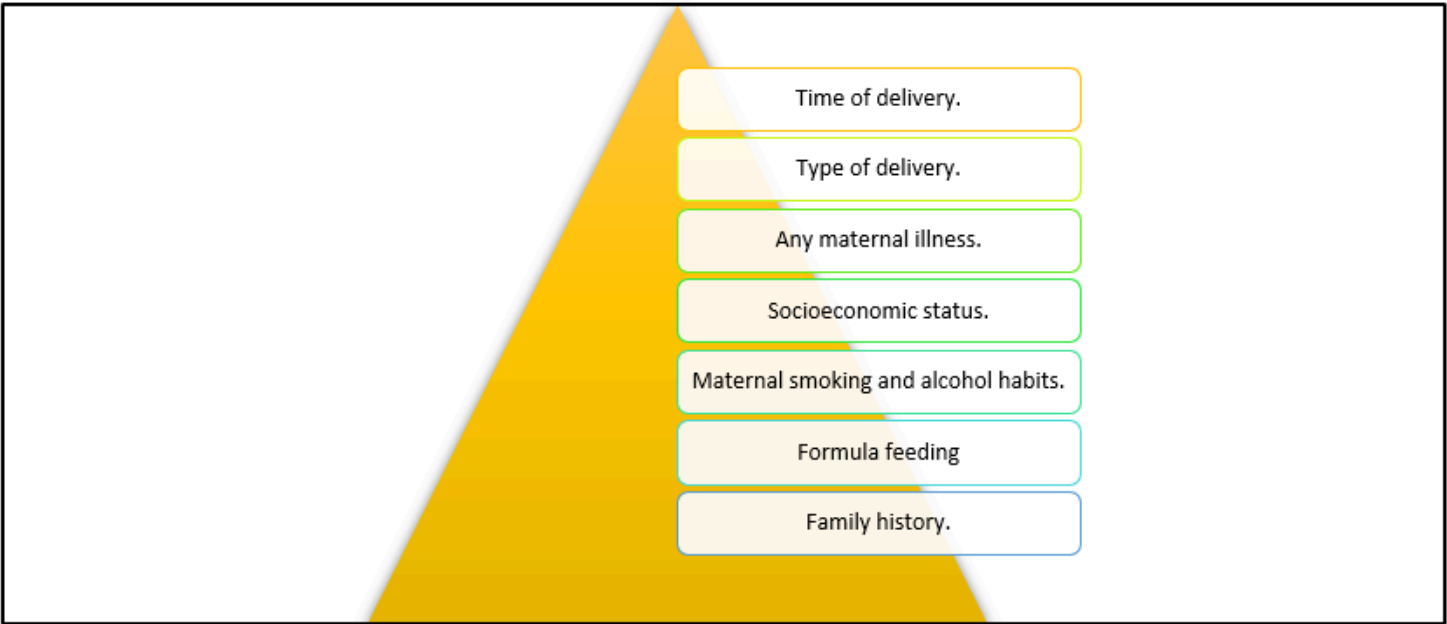


Figure 1

Type of delivery. Any maternal illness

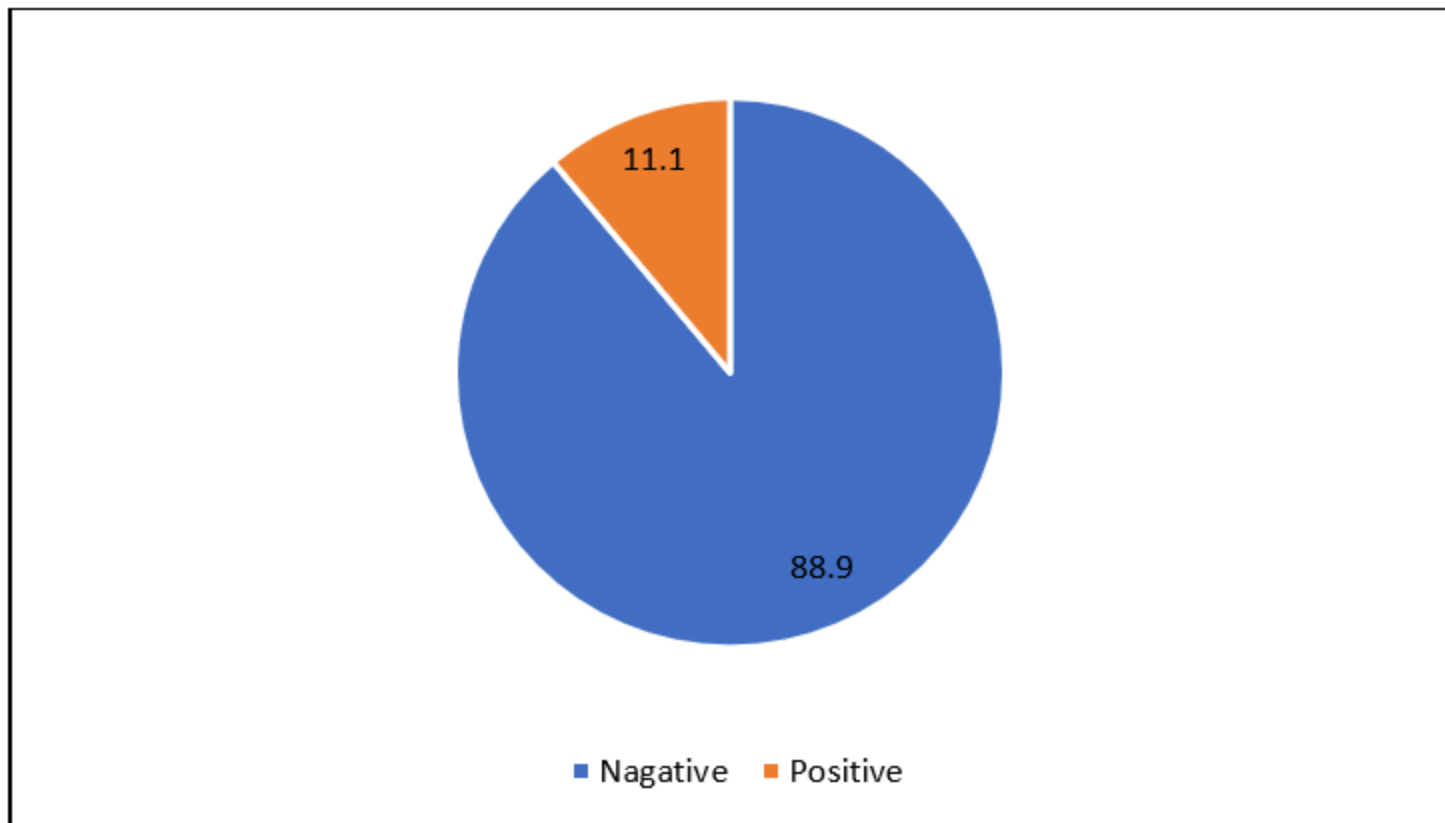


Figure 2

Infantile hypertrophic pyloric stenosis

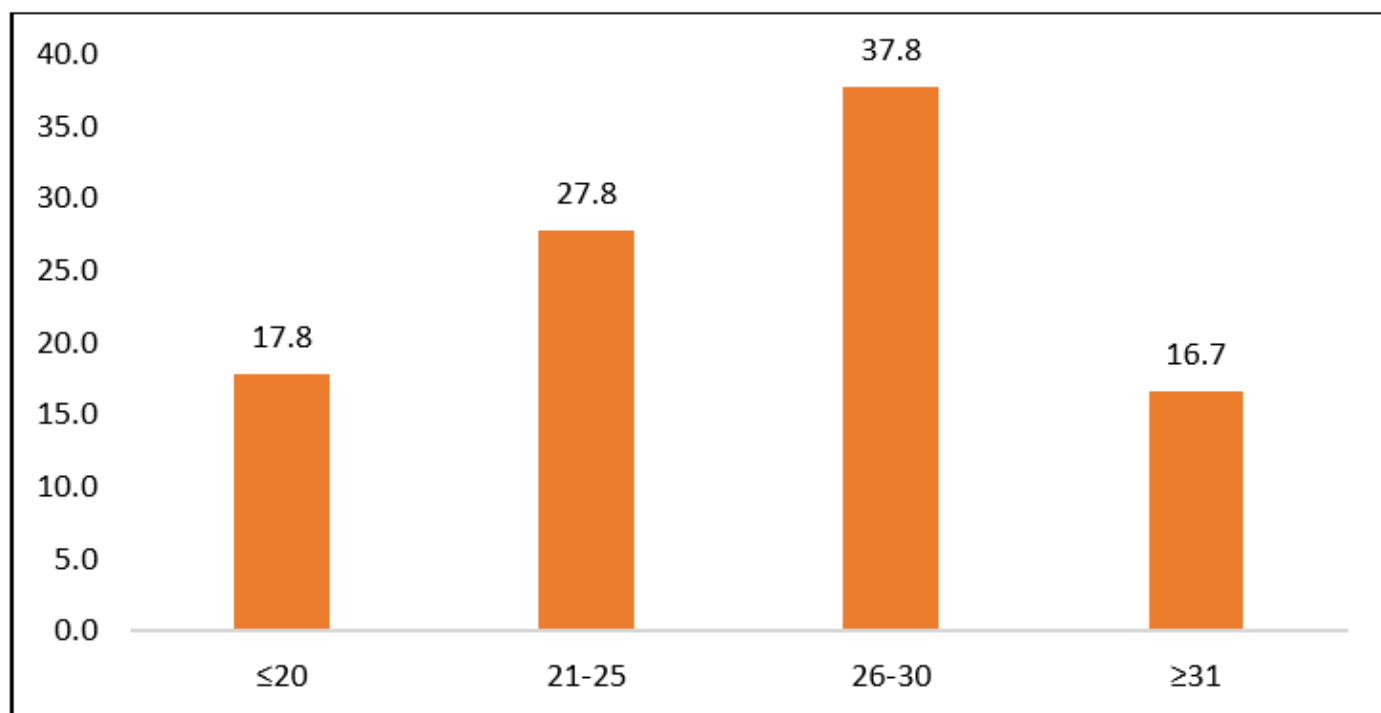


Figure 3

Mother's age in years of infantile hypertrophic pyloric stenosis

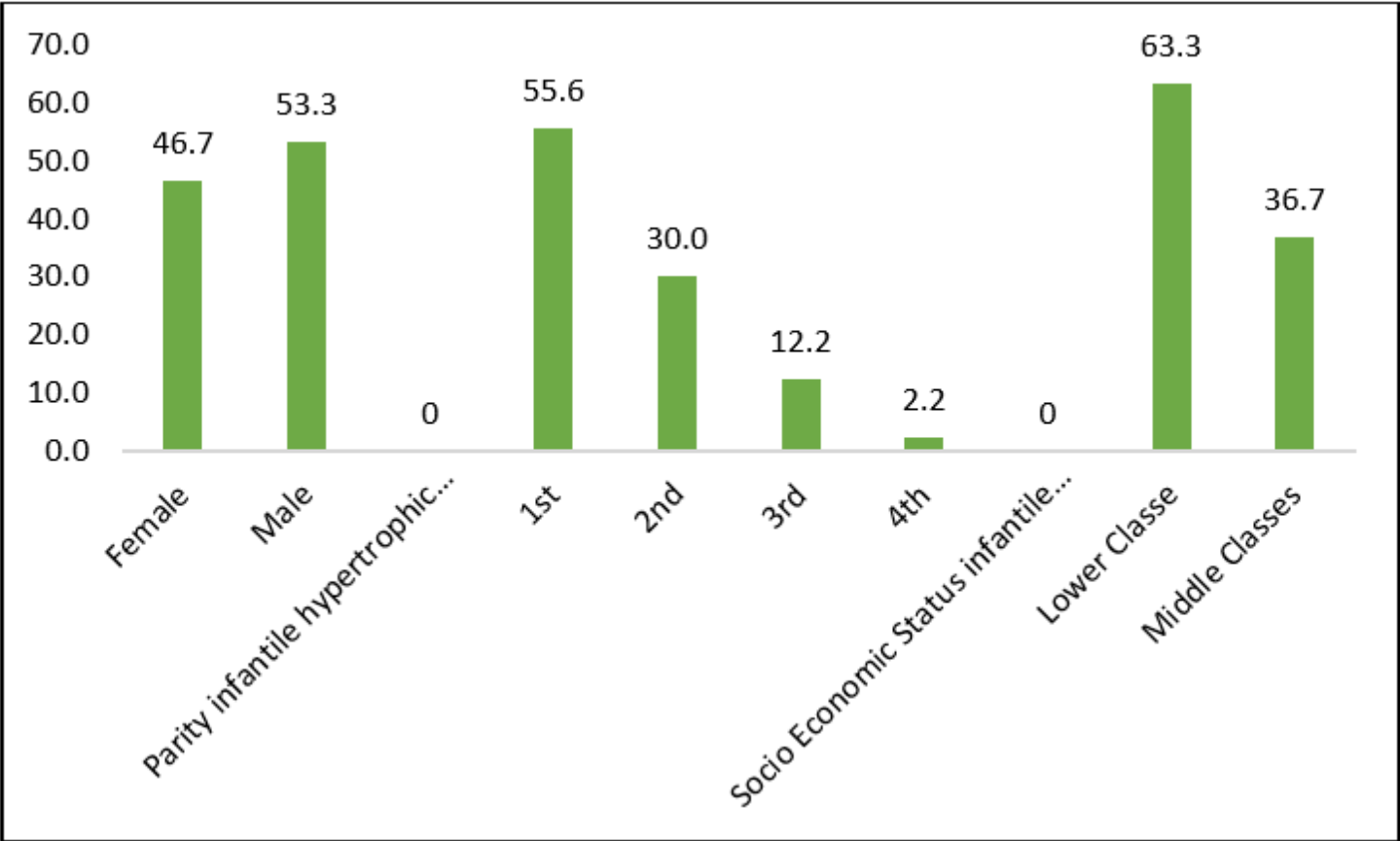


Figure 4

Gender, Parity, socio economic status of infantile hypertrophic pyloric stenosis

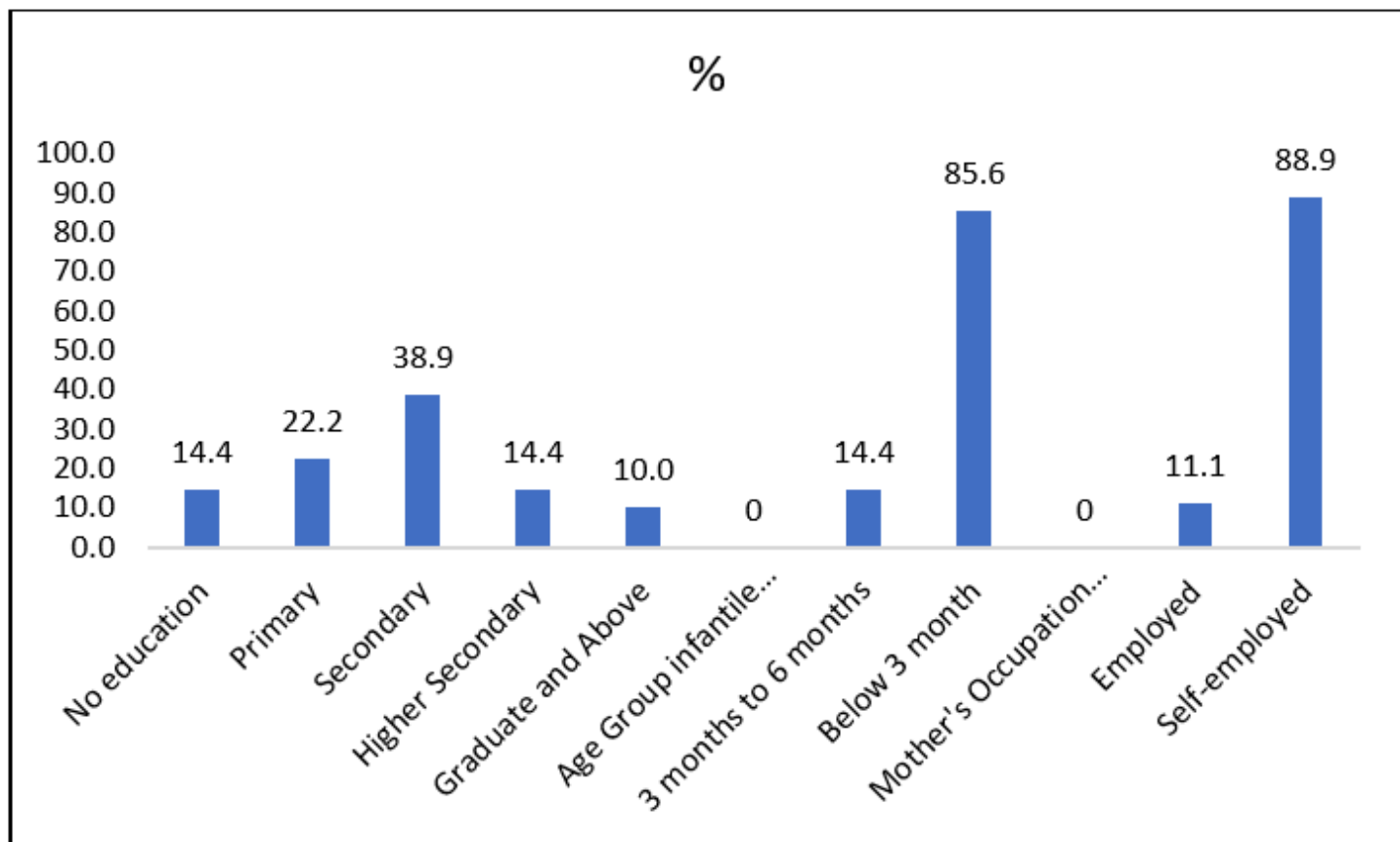


Figure 5

Mother education, Child age, and Mother's Occupation infantile hypertrophic pyloric stenosis pyloric stenosis

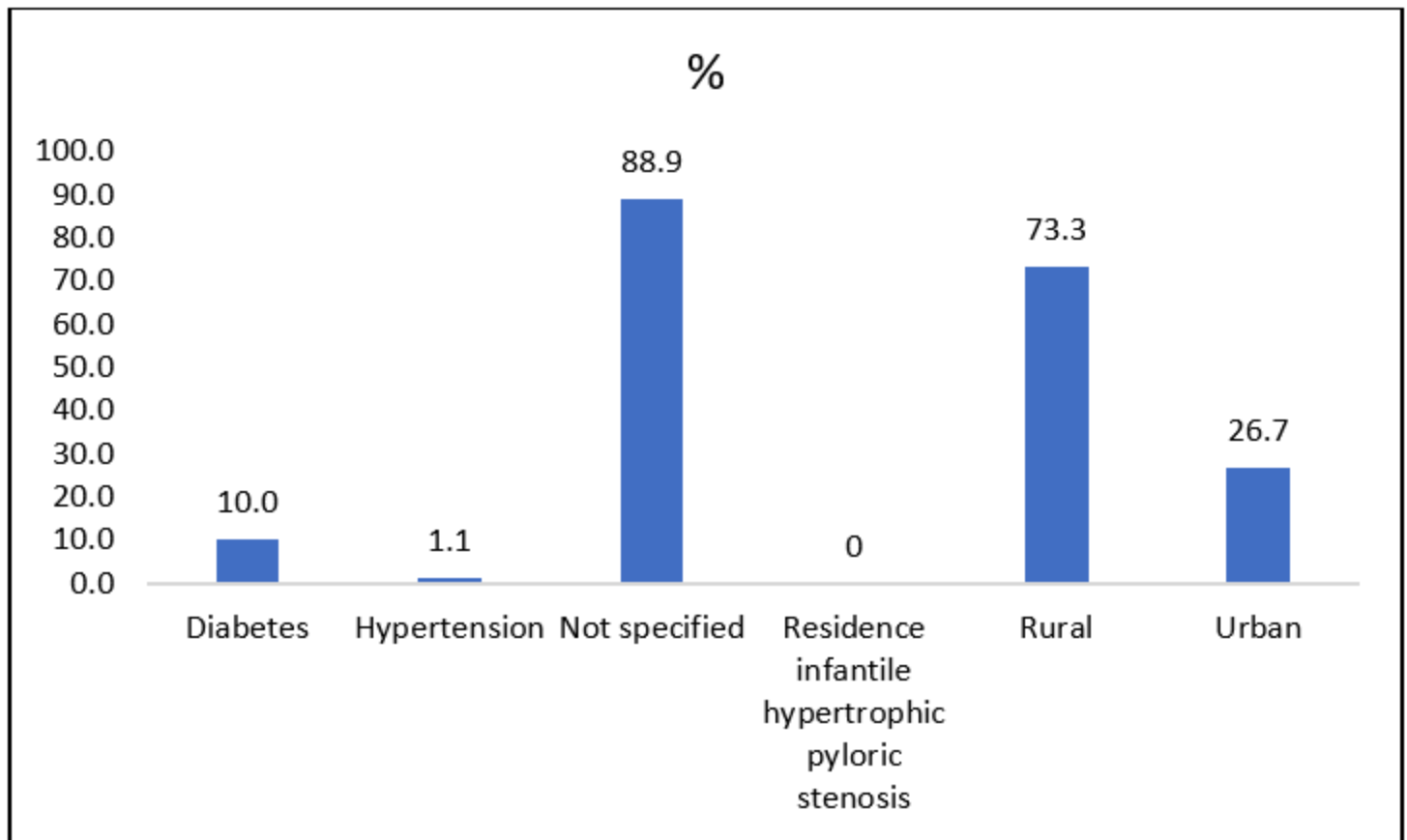


Figure 6

Mother education, Child age, and Mother's Occupation infantile hypertrophic pyloric stenosis pyloric stenosis

Supplementary Files

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