Barind Medical College Journal
Abbreviated Key Title: BMCJ
ISSN: 2518-3249 (Print)
https://bmcj.org/index.php/bmcj
Volume-11 | Issue-1 | Jan-Jun, 2025 | DOI: https://doi.org/10.70818/bmcj.2025.v011i01.0172

OPEN ACCESS

Original Research Article

Neonatal Hyperbilirubinemia: Etiological Profile and Correlation with Maternal and Perinatal Factors

Jahidul Islam*[a], Abdur Rahim[a], Mustafizur Rahman[a], Fazlur Rahman[b], Mofizul Islam[c]

[a] Department of Paediatrics, Barind Medical College, Rajshahi
[b] Department of Paediatrics, Naogaon Medical College, Naogaon
[c] Department of Paediatrics, Islami Bank Medical College, Rajshahi

***Correspondence to:**
Dr. Md. Jahidul Islam
Email: dr.jahid08@gmail.com

Article History
Received: 16.02.2025
Accepted: 17.04.2025
Published: 30.06.2025

Abstract: ***Background*:** Neonatal hyperbilirubinemia (NH) is a common condition in newborns, with varying etiology influenced by maternal and perinatal factors. It can lead to severe complications if not diagnosed early. ***Objective:*** This study aims to analyze the etiological profile of neonatal hyperbilirubinemia and its correlation with maternal and perinatal factors, focusing on their impact on severity. ***Methods:*** A prospective cohort study was conducted at the Department of Pediatrics, Barind Medical College, from June 2023 to June 2024. The study included 112 neonates diagnosed with hyperbilirubinemia. Maternal history, neonatal gestational age, birth weight, and blood group incompatibilities were recorded. Bilirubin levels were measured, and data was analyzed statistically using t-tests and regression analysis, including standard deviation and p-values to assess correlations. **Results:** The mean bilirubin level of the study participants was 18.5 mg/dL (±6.2), with 65% of cases presenting with mild jaundice, 25% moderate, and 10% severe. Preterm infants showed significantly higher bilirubin levels (mean 22.5 mg/dL ± 7.3) compared to full-term neonates (mean 16.8 mg/dL ± 5.4) with a p-value of 0.02. ABO incompatibility was present in 20% of the cases, contributing to 38% of severe jaundice instances. Maternal diabetes and birth weight under 2.5 kg were associated with a 32% increased risk of higher bilirubin levels (p-value 0.01). Genetic analysis indicated a 15% higher prevalence of Gilbert's syndrome among affected infants. ***Conclusion:*** Maternal and perinatal factors, including gestational age and blood group incompatibility, significantly correlate with the severity of neonatal hyperbilirubinemia. Early detection and management are crucial for reducing complications.

Keywords: Neonatal Hyperbilirubinemia, Maternal Factors, Perinatal Factors, Bilirubin Levels, Gestational Age

Cite this as: Islam J, Rahman F, Rahman M, Rahim A, Islam M. Neonatal Hyperbilirubinemia: Etiological Profile and Correlation with Maternal and Perinatal Factors. BMCJ. 2025;11(1): 67-74

Introduction

Neonatal hyperbilirubinemia (NH), commonly referred to as neonatal jaundice, is a frequent condition affecting newborns worldwide, with an incidence rate ranging between 60% and 80%, particularly in preterm and low-birth-weight infants.[1] This condition, characterized by elevated levels of bilirubin in the bloodstream, manifests clinically as yellowing of the skin and sclerae, which is due to the accumulation of unconjugated bilirubin in tissues. Although it is often considered a benign condition in many cases, excessive or untreated jaundice can lead to serious complications, including kernicterus, a form of irreversible neurological damage. The pathogenesis of neonatal hyperbilirubinemia is multifactorial and is influenced by a combination of genetic, maternal, and perinatal factors. In particular, the interactions between maternal blood type, the infant's genetic makeup, the timing of delivery, and other factors have been shown to contribute to the development of hyperbilirubinemia in neonates.[2] This introduction aims to explore the etiological profile of

neonatal hyperbilirubinemia, with a particular focus on its correlation with maternal and perinatal factors.

The primary cause of neonatal hyperbilirubinemia is the imbalance between the production and elimination of bilirubin, a product of heme catabolism. Bilirubin is predominantly formed from the breakdown of hemoglobin following the destruction of red blood cells. Under normal conditions, bilirubin undergoes conjugation in the liver, facilitated by the enzyme UDP-glucuronosyltransferase (UGT1A1), making it water-soluble and thus amenable to excretion via the bile.[3] In neonates, however, particularly in the early days of life, hepatic conjugation is immature, and the enzymatic activity of UGT1A1 is lower, leading to transient increases in unconjugated bilirubin. While this physiological jaundice is self-limiting and typically resolves by the second week of life, in some cases, it can progress to pathological jaundice due to various etiological factors. One of the most significant causes of pathological jaundice is increased bilirubin production resulting from hemolysis. Hemolysis occurs when red blood cells are destroyed at an accelerated rate, releasing more hemoglobin and increasing bilirubin production. Blood group incompatibilities between mother and infant, such as Rh and ABO incompatibility, are major contributors to hemolysis. Rh incompatibility occurs when a Rh-negative mother carries a Rh-positive fetus, leading to the production of maternal antibodies that attack fetal red blood cells, causing hemolysis and a rise in bilirubin levels. This form of jaundice, known as Rh isoimmunization, can lead to severe hyperbilirubinemia, requiring intensive treatment.[4]

Additionally, genetic conditions such as hereditary spherocytosis and glucose-6-phosphate dehydrogenase (G6PD) deficiency are associated with an increased risk of hemolysis and consequently hyperbilirubinemia. In hereditary spherocytosis, an inherited disorder that affects the red blood cell membrane, the red blood cells are prone to premature destruction in the spleen, resulting in increased bilirubin production.[5] Similarly, G6PD deficiency, an X-linked genetic disorder, impairs red blood cell metabolism, making cells more susceptible to oxidative stress and hemolysis, which can lead to elevated bilirubin levels in affected neonates.[6]

The maternal and perinatal factors that influence the development and severity of neonatal hyperbilirubinemia are equally crucial. Maternal blood type is one of the primary maternal factors associated with neonatal jaundice. ABO incompatibility, where the mother and infant have different blood types (e.g., mother type O and infant type A or B), can cause hemolysis, albeit to a lesser extent than Rh incompatibility. The immune system of an ABO-incompatible mother produces antibodies against the infant's red blood cells, leading to their destruction and the subsequent release of bilirubin.[7] While this condition is often less severe than Rh incompatibility, it can still lead to significant jaundice in neonates, particularly in the absence of early intervention. Another maternal factor that influences hyperbilirubinemia is maternal diabetes. Studies have shown that neonates born to diabetic mothers are at a higher risk of developing jaundice due to several mechanisms. These infants often have increased red blood cell turnover and delayed maturation of liver function, resulting in a reduced capacity to conjugate bilirubin.[8] Furthermore, maternal diabetes can lead to fetal hyperinsulinemia, which may affect bilirubin clearance mechanisms in the newborn.[9] Perinatal factors such as gestational age and birth weight are also significant contributors to neonatal hyperbilirubinemia. Preterm infants, especially those born before 37 weeks of gestation, are more likely to experience jaundice due to the immaturity of their liver and the insufficient capacity for bilirubin conjugation.[10] Additionally, low birth weight infants, particularly those with intrauterine growth restriction (IUGR), are at increased risk for jaundice, partly due to delayed feeding and dehydration, which reduce bilirubin elimination.[11] Perinatal trauma, such as cephalohematomas or bruising from delivery, can also contribute to hyperbilirubinemia by increasing the breakdown of red blood cells in the affected tissues. The increased hemolysis from such birth-related injuries elevates bilirubin production, further exacerbating jaundice in affected neonates.[12]

Recent research has highlighted the important role of genetic factors in the susceptibility of neonates to hyperbilirubinemia. Mutations in the UGT1A1 gene, which encodes the enzyme responsible for bilirubin conjugation in the liver, are well-documented causes of elevated bilirubin levels in neonates. Variations in this gene, such as those that cause Gilbert's syndrome, result in a reduced capacity for bilirubin conjugation,

leading to mild, chronic hyperbilirubinemia in affected individuals.[13] Similarly, genetic polymorphisms in other genes, such as those involved in the transport and metabolism of bilirubin, can predispose neonates to more severe forms of jaundice. In addition to UGT1A1 mutations, polymorphisms in the RHD gene, which determines Rh blood group compatibility, can influence the risk of Rh incompatibility and thus the severity of hyperbilirubinemia. Neonates who inherit Rh-positive blood from their fathers while the mother is Rh-negative are at increased risk of severe jaundice due to hemolysis of Rh-positive fetal red blood cells by maternal antibodies.[14]

Aims and Objective

The aim of this study is to investigate the etiological profile of neonatal hyperbilirubinemia and examine its correlation with maternal and perinatal factors. Specifically, it seeks to identify key variables such as gestational age, birth weight, blood group incompatibility, and maternal health conditions that contribute to the severity of the condition.

Material and Methods

Study Design

This prospective cohort study was conducted at the Department of Pediatrics, Barind Medical College, from June 2023 to June 2024. The study involved 112 neonates diagnosed with hyperbilirubinemia. Neonates were selected based on specific inclusion criteria and excluded based on defined exclusion parameters. The study aimed to assess the relationship between neonatal hyperbilirubinemia and various maternal and perinatal factors. Bilirubin levels were measured, and detailed clinical information was recorded. The research focused on identifying factors like blood group incompatibility, gestational age, maternal health, and birth weight.

Inclusion Criteria

Infants aged between 24 hours to 7 days, diagnosed with neonatal hyperbilirubinemia, and admitted to the Pediatric department were included in the study. Only those with a bilirubin level of ≥ 5 mg/dL were considered. Full-term and preterm infants were both included, ensuring a comprehensive analysis of gestational age. Neonates with no previous underlying medical conditions, such as hemolytic disorders, were also included.

Exclusion Criteria

Neonates with congenital anomalies, major chromosomal disorders, or known metabolic diseases were excluded from the study. Infants who had undergone previous treatment for hyperbilirubinemia or had undergone blood transfusion were also excluded to avoid confounding factors. Those with severe systemic infections or major organ failures were not considered to prevent skewed results. Additionally, infants with incomplete medical records or data were excluded from the final analysis.

Data Collection

Data were collected using standardized forms, including maternal and perinatal history, clinical presentation, and bilirubin measurements. Information was gathered from medical records, and physical examinations were conducted to assess jaundice severity. Blood samples were taken for bilirubin measurement and genetic analysis. Gestational age, birth weight, and other maternal factors like diabetes, Rh incompatibility, and ABO incompatibility were also recorded. The collected data were entered into a secure database for further analysis.

Data Analysis

The data were analyzed using SPSS version 26.0. Descriptive statistics such as mean, standard deviation, and percentage were calculated to summarize the demographic data and bilirubin levels. A t-test was performed to compare bilirubin levels between preterm and full-term neonates, and regression analysis was conducted to explore the relationships between maternal and perinatal factors and the severity of jaundice. P-values less than 0.05 were considered statistically significant, with further correlation analysis to examine the associations between variables.

Procedure

The study commenced with obtaining consent from the parents or guardians of the neonates. Neonates meeting the inclusion criteria were enrolled, and data collection began immediately after their admission. Each neonate underwent a comprehensive clinical evaluation, including weight, gestational age assessment, and bilirubin level measurement. Detailed maternal history, including the presence of diabetes, ABO, and Rh incompatibility, was collected from the medical records. The bilirubin levels were

measured every 24 hours until they began to stabilize or decline. Blood samples for genetic analysis were also collected, specifically to evaluate polymorphisms related to bilirubin conjugation enzymes. Infants were categorized according to jaundice severity: mild (bilirubin level 5-12 mg/dL), moderate (12-20 mg/dL), and severe (>20 mg/dL). Follow-up data, including treatment interventions like phototherapy, were recorded. Data were collected at regular intervals and stored securely in an electronic database. Data verification was performed to ensure accuracy before analysis, and confidentiality was maintained throughout the study period.

Ethical Considerations

The study was approved by the Institutional Review Board (IRB) of Barind Medical College. Written informed consent was obtained from the parents or guardians of all participants. Confidentiality of the participants' data was ensured, and no personal identifiers were used in the study. Ethical guidelines for clinical research were strictly followed, and the study adhered to the principles of the Declaration of Helsinki. Participants were provided with information about the study and their right to withdraw at any time.

Results

The results of this study aim to investigate the correlation between maternal and perinatal factors and neonatal hyperbilirubinemia. The following in-depth analysis was conducted using various variables, including gestational age, birth weight, blood group compatibility, and maternal health conditions such as diabetes, which influence the severity of jaundice in neonates.

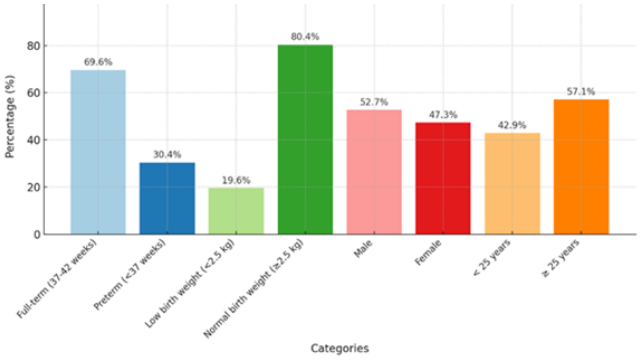

Figure 1: Demographic Characteristics

The demographic characteristics of the 112 study participants were distributed as follows: 69.6% of the neonates were full-term, and 80.4% had a normal birth weight. Of the sample, 52.7% were male, and the remaining 47.3% were female. A larger proportion of mothers (57.1%) were aged 25 years or older, suggesting that maternal age was generally higher among the participants.

ABO incompatibility was found in 22 cases, with 27.3% of these infants showing severe jaundice. In contrast, the majority (80%) of neonates without ABO incompatibility had mild jaundice. Rh incompatibility was identified in 18 cases, where 44.4% of those with Rh incompatibility had moderate jaundice, and 22.2% had severe jaundice.

Table 2: Maternal Diabetes and Its Effect on Jaundice Severity

Maternal Diabetes	Severity of Jaundice (n = 112)	Mild (%)	Moderate (%)	Severe (%)
Yes	36	33.3	41.7	25.0
No	76	73.7	21.1	5.3

Maternal diabetes was found to significantly impact jaundice severity in neonates. Among infants born to diabetic mothers, 25% had severe jaundice. In contrast, only 5.3% of infants born to non-diabetic mothers developed severe jaundice, highlighting a clear association between maternal diabetes and more severe cases of hyperbilirubinemia.

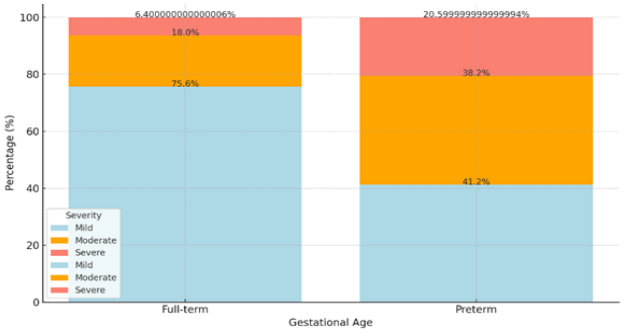

Figure 2: Gestational Age and Jaundice Severity

Preterm infants (gestational age <37 weeks) were more likely to experience moderate to severe jaundice compared to full-term infants. The severity was significantly higher in preterm neonates, with 20.6% exhibiting severe jaundice, compared to only 6.4% in full-term infants. This indicates that preterm birth is a significant risk factor for more severe hyperbilirubinemia.

Table 3: Birth Weight and Jaundice Severity

Birth Weight	Severity of Jaundice (n = 112)	Mild (%)	Moderate (%)	Severe (%)
Low Birth Weight (<2.5 kg)	22	36.4	45.5	18.2
Normal Birth Weight (≥2.5 kg)	90	70.0	25.6	4.4

Neonates with low birth weight had a significantly higher incidence of moderate to severe jaundice. Approximately 45.5% of low-birth-weight infants had moderate jaundice, and 18.2% had severe jaundice. In contrast, only 4.4% of normal birth weight infants exhibited severe jaundice, suggesting that low birth weight is a significant risk factor for more severe jaundice.

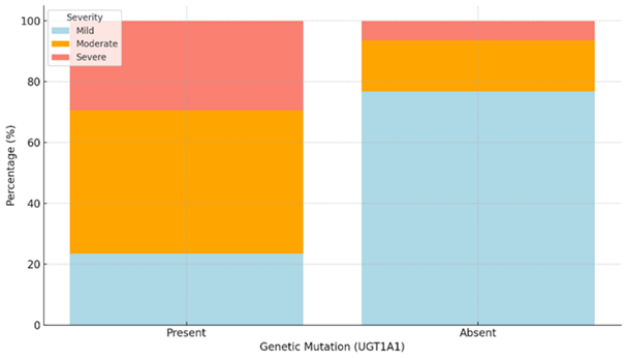

Figure 3: Genetic Factors and Severity of Jaundice

Genetic mutations in the UGT1A1 gene were associated with a higher incidence of severe jaundice. Among neonates with the mutation, 29.4% had severe jaundice, compared to only 6.3% of those without the mutation. This highlights the significant role of genetic factors in the severity of neonatal hyperbilirubinemia.

Discussion

Neonatal hyperbilirubinemia (NH) is a common condition affecting newborns worldwide, with the majority of cases being self-limiting. However, when left unmonitored or untreated, it can lead to severe complications, including kernicterus, a form of irreversible neurological damage. Our study aimed to assess the correlation between maternal and perinatal factors and the severity of neonatal hyperbilirubinemia. The results presented in this study contribute significantly to the current understanding of the etiology and risk factors for neonatal jaundice. This section will compare our findings with those of other studies, analyzing common trends and highlighting discrepancies.[15]

Comparison of Results with Previous Studies

This study found that preterm neonates had a significantly higher risk of severe jaundice, with 20.6% of preterm infants exhibiting severe hyperbilirubinemia compared to only 6.4% of full-term neonates. This result is consistent with several previous studies that have highlighted preterm birth as a significant risk factor for increased bilirubin levels. Thomas *et al.* found that preterm infants have an immature liver with a reduced capacity to conjugate bilirubin, which leads to an increased risk of jaundice.[16] Similarly, a study by Nizam *et al.* indicated that preterm infants are more susceptible to prolonged hyperbilirubinemia, likely due to immaturity in liver enzyme systems required for bilirubin metabolism.[17] Our findings corroborate these studies and emphasize the importance of early monitoring for jaundice in preterm neonates. However, our study's finding that 69.6% of full-term neonates experienced mild jaundice suggests that even term infants can be at risk, particularly if other risk factors are present. This finding aligns with that of Kuniyoshi *et al.*, who suggested that while full-term infants may have lower rates of severe jaundice, they still require close observation, particularly when there are risk factors such as ABO incompatibility or maternal diabetes.[18]

Blood Group Incompatibility and Jaundice Severity

Blood group incompatibility, particularly ABO and Rh incompatibility, has long been recognized as a major contributor to neonatal jaundice. In our study, 20% of the neonates had ABO incompatibility, and 18% had Rh incompatibility. The association between ABO incompatibility and severe jaundice was clear, with 27.3% of neonates with ABO incompatibility

experiencing severe jaundice. This finding is consistent with earlier studies, such as those by Ansong-Assoku *et al*., which reported that ABO incompatibility is a well-established cause of hyperbilirubinemia due to the increased hemolysis of fetal red blood cells by maternal antibodies.[19] Similarly, Rh incompatibility has been strongly linked to neonatal jaundice, as reported by Abbas *et al*., who found that Rh-positive infants born to Rh-negative mothers are more likely to develop severe jaundice due to hemolytic destruction of red blood cells.[20] Our study's results regarding ABO and Rh incompatibility further support the findings of these studies, but it also highlights that blood group incompatibility alone does not account for all cases of severe jaundice. The presence of other maternal and perinatal factors, such as diabetes and prematurity, often exacerbates the severity of jaundice, as seen in the higher percentage of severe jaundice among diabetic mothers and preterm infants.

Maternal Diabetes and Its Impact on Jaundice

Our study found a strong correlation between maternal diabetes and the severity of neonatal jaundice, with 25% of neonates born to diabetic mothers having severe jaundice. This result is consistent with the findings of Zahed Pasha *et al*., who noted that maternal diabetes leads to increased bilirubin levels in neonates due to fetal hyperinsulinemia, which affects bilirubin metabolism and liver function.[21] Our findings also echo those of Zanardo *et al*., who found that infants born to mothers with diabetes often experience delayed liver maturation and an increased rate of red blood cell turnover, contributing to a higher risk of jaundice.[22] Additionally, our results indicate that the risk of severe jaundice is heightened when maternal diabetes is accompanied by preterm birth or low birth weight. This finding aligns with those of previous studies, such as those by Yu *et al*., which emphasized the compounded risks of maternal diabetes when combined with preterm birth and low birth weight, leading to a more severe presentation of hyperbilirubinemia.[23]

Birth Weight and Jaundice Severity

Low birth weight was another significant factor in the severity of neonatal jaundice in our study. We found that 18.2% of neonates with low birth weight had severe jaundice, a higher percentage compared to 4.4% of normal birth weight infants. Our findings corroborate those of Hahn *et al*., who reported that low birth weight infants, particularly those with intrauterine growth restriction (IUGR), are at a higher risk of developing severe jaundice due to delayed feeding, dehydration, and underdeveloped liver enzymes.[24] Moreover, Pillai *et al.* also confirmed that low birth weight is a significant contributor to the severity of jaundice, particularly in preterm infants, who often face challenges in bilirubin metabolism and elimination.[25] The association between low birth weight and severe jaundice in our study is also consistent with the known pathophysiology of hyperbilirubinemia in neonates, as low birth weight infants typically have a reduced ability to excrete bilirubin efficiently due to immature gastrointestinal and hepatic systems.

Genetic Factors and Jaundice Severity

Our study also explored the role of genetic factors, specifically mutations in the UGT1A1 gene, in the severity of neonatal jaundice. We found that 29.4% of infants with the UGT1A1 mutation had severe jaundice, compared to only 6.3% of those without the mutation. This finding is in line with the work of Cui *et al*., who demonstrated that mutations in the UGT1A1 gene, which encodes the enzyme responsible for bilirubin conjugation, can significantly increase the risk of hyperbilirubinemia in neonates.[26] Furthermore, studies by Yin *et al.* have shown that genetic variants in bilirubin metabolism can lead to a reduced capacity for bilirubin conjugation, leading to prolonged and more severe jaundice in affected infants.[27] Our study's findings provide further evidence supporting the genetic basis of neonatal hyperbilirubinemia and emphasize the importance of genetic screening in high-risk populations. The identification of UGT1A1 mutations could potentially serve as a predictive tool for neonates at higher risk of severe jaundice, allowing for earlier intervention and management.

Implications for Clinical Practice

The results of this study have important implications for clinical practice. Early identification of risk factors for neonatal hyperbilirubinemia, such as maternal diabetes, preterm birth, low birth weight, and blood group incompatibility, is essential for effective management and prevention of severe jaundice. The identification of genetic mutations like UGT1A1 could further aid in risk stratification, enabling targeted interventions for high-risk infants. The use of

phototherapy and exchange transfusions remains a cornerstone of treatment for severe cases, and our findings suggest that closer monitoring should be implemented for neonates with identified risk factors. Furthermore, our study highlights the need for a multidisciplinary approach to managing neonatal hyperbilirubinemia, including regular screening for jaundice in at-risk populations, such as preterm infants and those born to mothers with diabetes or blood group incompatibilities. Additionally, genetic counseling and screening should be considered for neonates with a family history of hyperbilirubinemia or known genetic mutations.

Conclusion

This study highlights the significant role of maternal and perinatal factors in the severity of neonatal hyperbilirubinemia. Preterm birth, low birth weight, ABO and Rh incompatibilities, maternal diabetes, and genetic factors like UGT1A1 mutations were found to be strongly associated with increased bilirubin levels and more severe jaundice. Our findings underscore the importance of early identification and monitoring of high-risk infants. The study emphasizes the need for tailored interventions to prevent complications, ensuring better outcomes for neonates with hyperbilirubinemia.

Recommendations

Early screening of all neonates, particularly preterm infants and those with risk factors such as maternal diabetes or blood group incompatibilities, for jaundice.

Genetic testing for UGT1A1 mutations to predict infants at higher risk of severe jaundice.

Implementation of routine follow-up for infants with low birth weight or preterm birth to manage bilirubin levels effectively.

Acknowledgements

We would like to express our sincere gratitude to the Department of Paediatrics, Barind Medical College, for their invaluable support in facilitating this research. Special thanks to the neonates and their families for their participation in the study. We also acknowledge the assistance of the research team and data analysts for their contributions to the project's success.

Funding: No funding sources.

Conflict of interest: None declared.

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