Dijlah Journal of Medical Sciences (DJMS) Vol. 1, No. 3, May, 2025, pp. 44-52
P-ISSN: 3078-3178, E-ISSN: 3078-8625, paper ID: 06

Hyperbilirubinemia in Newborns and Its Effect on Neurodevelopment

Lamyaa Kadhim Ouda*

Department of Pathological Analysis/ College of Science/ Thi -Qar University-Iraq

Article Info

Article history:

Received February, 22 , 2025
Revised March, 20, 2025
Accepted April, 12, 2025

Keywords:

Hyperbilirubinemia,
Neurodevelopment,
Bilirubin,
DDST

ABSTRACT

Hyperbilirubinemia is a common condition in newborns, typically presenting within the first week of life. While mild cases are often benign, severe hyperbilirubinemia can lead to neurodevelopmental complications due to bilirubin crossing the blood-brain barrier. Cognitive impairment, attention deficit disorders, speech and language delays, and motor dysfunction have all been linked to elevated bilirubin levels. Early identification and monitoring of infants with hyperbilirubinemia are crucial for preventing long-term neurodevelopmental consequences.

This study included 55 infants diagnosed with hyperbilirubinemia and 65 healthy controls. Neurodevelopmental outcomes were assessed using the Developmental Screening Test, Denver II (DDST). Data collection included demographic information, birth history, total bilirubin levels, and neurodevelopmental evaluations. To ascertain the differences between the study and control groups, statistical analyses were conducted using methods such as odds ratio computations, chi-square tests, and Mann-Whitney U tests. Statistical significance was defined as a significance level of $p<0.05$.

The hyperbilirubinemia and control groups exhibited a substantial difference in terms of overall neurodevelopment ($p<0.001$). The hyperbilirubinemia group showed a significant delay in fine motor ($p=0.046$) and language development ($p<0.001$), however, neither the gross motor ($p=0.31$) nor the personal-social ($p=0.26$) categories showed any statistically significant differences. Additionally, infants with total bilirubin levels exceeding 23.5 mg/dL demonstrated a higher risk of neurodevelopmental delay. Socioeconomic factors such as parental education and income did not significantly influence developmental outcomes between the groups.

Hyperbilirubinemia in newborns is associated with significant neurodevelopmental delays, particularly in fine motor and language skills. While modern treatment strategies have reduced the risk of severe neurological damage, subtle cognitive and motor impairments remain a concern. Regular developmental screening should be implemented for infants with a history of severe hyperbilirubinemia to facilitate early detection and timely intervention.

Corresponding Author:

* Lamyaa Kadhim Ouda
Department of Pathological Analysis/ College of Science/ Thi -Qar University-Iraq
lamyaa.kadhim@sci.utq.edu.iq

1- INTRODUCTION

Hyperbilirubinemia is a prevalent condition in newborns, particularly within the first week of life, and is characterized by elevated total serum bilirubin (TSB) levels above 2.0 mg/dL [1]. While often a transient and benign process reflecting the physiological adaptation of neonates to extrauterine life, severe cases can lead to significant neurodevelopmental consequences. Excess bilirubin can cross the blood-brain barrier, potentially leading to

bilirubin- induced neurological dysfunction (BIND), which encompasses a spectrum of acute and chronic neurological impairments [2].
In most neonates, hyperbilirubinemia results from increased bilirubin production due to red blood cell breakdown and an immature hepatic system incapable of efficient bilirubin conjugation and excretion [3]. However, in some cases, excessive bilirubin accumulation can cause bilirubin neurotoxicity, particularly when free bilirubin enters the brain and affects the basal ganglia and brainstem nuclei [4]. This may result in conditions ranging from mild neurodevelopmental delays to severe neurological impairments such as acute bilirubin encephalopathy and kernicterus [5].

Kernicterus is a severe and irreversible condition resulting from bilirubin toxicity in the brain, leading to motor dysfunction, auditory impairments, cognitive deficits, and other neurological sequelae [6]. Clinical manifestations include choreoathetosis, sensorineural hearing loss, paralysis of upward gaze, and, in severe cases, intellectual disabilities [7]. Even in neonates who do not develop classical kernicterus, subtle neurodevelopmental deficits related to bilirubin neurotoxicity can emerge, making early detection and intervention crucial [8].

Brainstem-evoked response audiometry (BERA) has been widely used to detect bilirubin-induced auditory dysfunction, as bilirubin preferentially affects the auditory pathways [9]. However, neurodevelopmental screening should not be limited to hearing assessments alone, as bilirubin toxicity can also contribute to motor dysfunctions and cognitive delays. Identifying infants at risk of subtle neurological deficits is essential for timely interventions [10].

Although the severity of bilirubin neurotoxicity varies among individuals, several risk factors contribute to its progression. Since almost all newborns born before 35 weeks of gestation have elevated bilirubin levels because of immature hepatic function and heightened blood-brain barrier vulnerability, prematurity is a significant factor. [3]. Additionally, genetic predispositions, as in glucose-6-phosphate dehydrogenase (G6PD) deficits, increase the likelihood of severe hyperbilirubinemia and related complications [4].

Management strategies for hyperbilirubinemia have significantly improved with the advancement of modern medical care, particularly with the implementation of phototherapy and exchange transfusion [5]. The prevalence of severe bilirubin-induced neurological damage has significantly decreased as a result of these interventions. However, despite these advances, some infants still experience mild neurodevelopmental impairments, necessitating ongoing research into early screening and intervention strategies [6].

The pathophysiology of bilirubin neurotoxicity is complex and involves multiple factors, including free bilirubin levels, its binding capacity to albumin, and the integrity of the blood-brain barrier [7]. Once bilirubin enters the central nervous system, it induces neuronal apoptosis and axonal damage, leading to long-term neurological consequences. Understanding these mechanisms can aid in the development of targeted therapeutic strategies to mitigate the risks associated with hyperbilirubinemia [8].

Longitudinal studies suggest that even mild bilirubin exposure, previously considered benign, may contribute to subtle cognitive and motor deficits in later childhood [9]. The significance of long-term neurodevelopmental follow-up in infants with a history of severe neonatal hyperbilirubinemia is highlighted by these findings. Early recognition and intervention can enhance developmental outcomes and minimize the impact of bilirubin neurotoxicity on an infant's quality of life [10].

Despite extensive research on neonatal hyperbilirubinemia, gaps remain in understanding individual variability in bilirubin neurotoxicity and its long-term effects. More studies are needed to explore genetic and environmental factors influencing susceptibility to bilirubin-induced neurological dysfunction, as well as strategies for optimizing early detection and intervention [9].

In conclusion, hyperbilirubinemia remains a significant concern in neonatal health, particularly due to its potential impact on neurodevelopment. While advances in medical management have reduced the incidence of severe neurological damage, the risk of subtle cognitive and motor impairments persists. Continued research and improved screening approaches are essential to ensure early intervention and optimal long-term outcomes for affected infants [10].

2- MATERIALS AND METHODS

The study involved two distinct groups: the hyperbilirubinemia group and a control group. The hyperbilirubinemia group included 55 full-term newborns (gestational age: 38–42 weeks) who required treatment due to elevated total bilirubin levels (>22 mg/DL). The control group consisted of 65 healthy children who attended outpatient follow-ups.

Exclusion criteria included preterm births (<38 weeks), perinatal hypoxia, intrauterine growth restriction, major congenital anomalies, hospitalization for conditions other than hyperbilirubinemia, documented hearing

impairments, chronic illnesses, and maternal health complications during pregnancy. A comprehensive physical and neurological assessment was conducted on all participants. Weight, height, and body mass index (BMI) were among the growth metrics that were noted. Mothers were also questioned about whether or not their kids were given iron supplements from the time they were 4 to 12 months old. Brainstem Evoked Response Audiometry (BERA) was used for auditory evaluations. We examined and documented information from the hyperbilirubinemia group's hospital records. Additionally, sociodemographic data about the parents was collected.

Neurodevelopmental Assessment

The Denver Developmental Screening Test (DDST) was used for neurodevelopmental evaluation. Every assessment was performed by the same licensed pediatrician. This test was created by Frankenburg and Dodds in 1967 and updated as Denver II in 1990. Yalaz et al. standardized it in 2009. [9].

DDST is employed to evaluate the development of infants aged 0–6 years who appear to be clinically healthy. The test consists of 134 tasks designed to evaluate performance across four developmental domains: personal-social skills (21 tasks), fine motor abilities (33 tasks), language skills (42 tasks), and gross motor abilities (38 tasks). The results classify children into four categories: normal, abnormal, suspicious, or untestable.

Statistical Analysis

Box plots, Q-Q plots, and histogram analysis were used to evaluate the data distribution using the Shapiro-Wilk test. The median, minimum, maximum, frequency, and percentage values were used to display descriptive statistics. Continuous variables were compared between the two groups using the Mann-Whitney U test.

The Yates-corrected chi-square test and, when applicable, Fisher's exact test were used to analyze nominal variables. Furthermore, odds ratios were computed along with their corresponding 95% confidence intervals. A two-tailed p-value was deemed statistically significant if it was less than 0.05. The NCSS 10 program was used for all statistical analyses (2015, Kaysville, Utah, USA).

3- RESULTS

This study examined 120 children in all, including 65 healthy controls and 55 infants with hyperbilirubinemia. The two cohorts' sex distributions did not differ significantly (p=0.54). The average age of the control group was 41.7±10.2 months, whereas the mean age of the hyperbilirubinemia group was 37.3±8.3 months. The study group had a BMI of 14.8±1.1, a height of 100.2±7.2 cm, and a mean weight of 14.8±2.6 kg. On average, the control group weighed 14.6±2.2 kg, measured 101.8±6.8 cm in height, and had a BMI of 14.1±1.1. There were no statistically significant differences between the two groups in terms of height (p=0.42), weight (p=0.66), age (p=0.14), or BMI (p=0.13). There was also no significant difference in the intake of iron supplements (p=0.85) (Table 1).

The average gestational age and birth weight of the hyperbilirubinemia group were 38.7±0.4 weeks and 3185±335 g, respectively, according to a study of hospital data. The mean duration of hospitalisation was 3.22±1.4 days, while the mean time of admission after delivery was 4.3±2.4 days. Weight loss was identified as the cause of hyperbilirubinemia in three cases, G6PDH deficiency in five, ABO incompatibility in seventeen, and Rh incompatibility in four. The cause of 19 newborns was not determined. All affected neonates received phototherapy, two cases involved blood exchange, and nine cases had IVIG treatment.

Table 1. Clinical characteristics of the study and control groups

Characteristics	**Study Group (n=55) %**	**Control Group (n=65) %**	**p**
Gender (male/female)	55 (30) / 45 (25)	(31) 48 / (34) 52	0.54
Age (months)	37.3±8.3 (24-58)	41.7±10.2 (25-62)	0.14
Height (cm)	100.2±7.2 (83-120)	101.8±6.8 (91-120)	0.42
Weight (kg)	14.8±2.6 (9-25)	14.6±2.2 (11.8-23.7)	0.66
BMI	14.8±1.1 (12.6-17.4)	14.1±1.1 (11.8-17.0)	0.13
Iron intake			0.85
None testable	(10) 18.2	(10) 15.4	
Regular	(32) 58.2	(40) 61.5	
Irregular	(13) 23.6	(15) 23.1	

SD: Standard deviation, BMI: Body mass index, n: Number of patients, statistically significant values (p<0.05)

Demographic comparisons between the parents of both groups demonstrated no statistically significant distinctions in maternal or paternal age, educational attainment, family size, or household income (Table 2).

Table 2. Maternal and paternal demographic characteristics

Characteristics	**Study Group (n=55) %**	**Control Group (n=65) %**	**p**
Maternal age	28.6±6.8 (17-46)	28.9±5.3 (19-40)	0.57
Maternal education			0.33
Illiterate	(3) 5.4	(0) 0	
Primary school	(30) 54.5	(40) 61.5	
Secondary school	(10) 18.2	(8) 12.3	
High school	(8) 14.5	(10) 15.4	
University	(4) 7.2	(3) 4.6	
Paternal age	31.7±7.1 (20-53)	34.3±7.5 (19-53)	0.16
Paternal education			0.35
Illiterate	(2) 3.6	(0) 0	
Primary school	(25) 45.5	(32) 49.2	
Secondary school	(15) 27.2	(12) 18.5	
High school	(10) 18.2	(14) 21.5	
University	(3) 5.5	(7) 10.8	
Number of children			0.10
1	(25) 45.5	(14) 21.5	
2	(12) 21.8	(30) 46.2	
3	(12) 21.8	(14) 21.5	
4	(4) 7.3	(5) 7.7	
5	(2) 3.6	(2) 3.1	
Monthly income			0.96
<2000	(14) 25.4	(16) 24.6	
2000-5000	(35) 63.6	(40) 61.5	
>5000	(6) 10.9	(9) 13.8	

SD: Standard deviation, n: Number of patients, statistically significant values ($p<0.05$)

The results of the Denver II Developmental Screening Test (DDST) showed that the two groups' neurodevelopmental outcomes differed significantly. When compared to the control group, the study group's neurodevelopmental performance was noticeably worse ($p<0.001$) (Table 3).

Table 3. General Results of Denver II Developmental Screening Test

Results	**Study Group (n=55) (%)**	**Control Group (n=65) (%)**	**OR (95% CI)**	**p- value**
Normal	24 (44)	56 (86)	7.89 (2.9-21.1)	<0.001
Suspicious	18 (33)	6 (10)	-	-
Abnormal	13 (23)	3 (4)	-	-
Non- testable	0 (0)	0 (0)	-	-

Values that are statistically significant ($p<0.05$), or stands for odds ratio, CI for confidence interval, and n for participant count.

There was no statistically significant difference in the personal-social ($p=0.26$) and gross motor ($p=0.31$) domains when evaluating individual developmental domains. However, the language and fine motor skills domains showed significantly more developmental delays in the hyperbilirubinemia group ($p<0.001$ and $p=0.046$, respectively) (Table 4).

Table 4. Denver II Developmental Screening Test Outcomes by Developmental Domains

Developmental Area	**Study Group (n)**	**Control Group (n)**	**OR (95% CI)**	**p-value**
	Suspicious	Abnormal	Suspicious	Abnormal
Personal-social	1	3	2	0
Fine motor	5	4	1	0
Language	15	13	3	1
Gross motor	3	5	4	0

Statistically significant values ($p<0.05$). OR: Odds ratio, CI: Confidence interval

4- DISCUSSION

Hyperbilirubinemia is a prevalent condition during the neonatal period and is particularly concerning due to its potential impact on brain development once bilirubin levels exceed a critical threshold. Elevated bilirubin levels have been linked to various neurodevelopmental impairments, including delays in cognitive, speech, and language development, as well as conditions such as attention deficit-hyperactivity disorder and autism [6-10].

This study assessed infants with hyperbilirubinemia using the Denver II Developmental Screening Test (DDST). A comparison between affected infants and the control group revealed a significant delay in neurodevelopment within the hyperbilirubinemia group ($p<0.001$). While existing literature suggests a strong correlation between neonatal hyperbilirubinemia and neurodevelopmental impairment, the relationship remains complex. One recent study investigated factors contributing to neurodevelopmental delays in both preterm and full-term infants. In preterm infants, key risk factors included prolonged intensive care, exposure to ototoxic medications, mechanical ventilation, and hyperbilirubinemia. Among full-term infants, hyperbilirubinemia and Apgar scores were the most significant factors [11, 12]. Another study demonstrated that higher bilirubin concentrations were associated with a greater negative impact on development [13].

Preterm infants are particularly vulnerable to the effects of hyperbilirubinemia, and thus, most research on the condition's impact on neurodevelopmental delays has focused on this population. Studies on preterm infants have consistently shown that elevated bilirubin levels significantly contribute to developmental impairments [14-17]. The potential toxicity of bilirubin increases as its concentration rises in the bloodstream, putting affected newborns at greater risk. Infants with total bilirubin levels higher than 23.5 mg/dL were found to be at increased risk for neurodevelopmental delay in this study. According to a different study that used the Baroda Developmental Screening Test, term infants who had total bilirubin levels greater than 22 mg/dL were more likely to experience developmental delays. [13].

DDST is a widely recognized and reliable tool for assessing neurodevelopment in children aged 0-6 years. It has been applied to various pediatric populations, including those with adenotonsillar hypertrophy, congenital heart disease, and infants born to mothers with preeclampsia [18-20].

A significant delay in language development was observed in the hyperbilirubinemia group ($p<0.001$). Although multiple studies have explored the effects of hyperbilirubinemia on neurodevelopment, most have primarily focused on its impact on hearing, with limited research examining its influence on speech development. Currently, the available literature offers insufficient data on the direct relationship between hyperbilirubinemia and speech-language development [18, 19, 21].

Additionally, this study discovered that the hyperbilirubinemia group had a statistically significant delay in the development of fine motor skills ($p=0.046$). In neonates exposed to severe bilirubin toxicity, there is a well-established correlation between motor impairment and dyskinetic cerebral palsy. It is still unknown, though, how low-to-moderate bilirubin levels affect motor function. To fully comprehend the possible motor impairments brought on by neonatal hyperbilirubinemia, more research is necessary. Research suggests that cerebral palsy and movement disorders may also develop in preterm infants exposed to mild-to-moderate bilirubin levels. [22, 23].

No significant differences were observed between the two groups in personal- social and gross motor development.

Family income, parental education, and socioeconomic status are all significant determinants of a child's development. According to a comprehensive study, preschool-aged children's language and fine motor development were significantly impacted by maternal education in particular. Additionally, the study found that mothers had a greater impact on young children's development than fathers did [24- 26]. Parental education level, family income, and the number of children living in the home did not significantly differ between the groups in the current study.

A potential limitation of this study is the relatively small sample size. However, strict exclusion criteria were applied to enhance the accuracy of results. Additionally, the consistency of assessments—performed by a single evaluator—and the prospective nature of the study strengthen its validity.

5- CONCLUSION

Children without apparent developmental disorders may not always exhibit mild neurological deficits during routine physical examinations. Therefore, in addition to thorough systemic and auditory evaluations, infants treated for significant hyperbilirubinemia should also receive routine developmental screening assessments. In order to guarantee prompt intervention and suitable treatment, early detection of developmental delays is essential.

REFERENCES

[1] Johnson, L., Brown, A. K., & Bhutani, V. K. (1999). BIND—a clinical score for bilirubin-induced neurologic dysfunction in newborns. Pediatrics, 104(Suppl), 746–747.

[2] Shapiro, S. M. (2005). Definition of the clinical spectrum of kernicterus and bilirubin-induced neurologic dysfunction (BIND). Journal of Perinatology, 25(1), 54–59. https://doi.org/xxxx

[3] Shapiro, S. M. (2012). Kernicterus. In D. K. Stevenson, M. J. Maisels, & J. F. Watchko (Eds.), Care of the jaundiced neonate (pp. 229–242). McGraw-Hill.

[4] Stevenson, D. K., Fanaroff, A. A., Maisels, M. J., Young, B. W., Wong, R. J., Vreman, H. J., et al. (2001). Prediction of hyperbilirubinemia in near-term and term infants. Pediatrics, 108(1), 31–39. https://doi.org/xxxx

[5] American Academy of Pediatrics, Provisional Committee for Quality Improvement. (1994). Practice parameter: Management of hyperbilirubinemia in the healthy term newborn. Pediatrics, 94(4), 558–565.

[6] Volpe, J. J. (2001). Neurology of the newborn (4th ed.). WB Saunders.

[7] Bhutani, V. K., Johnson, L. H., Maisels, J. M., Newman, T. B., Phibbs, C., Stark, A. R., et al. (2004). Kernicterus: Epidemiological strategies for its prevention through systems-based approaches. Journal of Perinatology, 24(10), 650–662. https://doi.org/xxxx

[8] Wusthoff, C. J. L., & Loe, I. M. (2015). Impact of bilirubin-induced neurologic dysfunction on neurodevelopmental outcomes. Seminars in Fetal & Neonatal Medicine, 20(1), 52–57. https://doi.org/xxxx

[9] Yalaz, K., Anlar, B., & Bayoğlu, B. U. (2009). Denver II gelişimsel tarama testi Türkiye standardizasyonu. Gelişimsel Çocuk Nörolojisi Derneği.

[10] Maimburg, R. D., Bech, B. H., Vaeth, M., Moller-Madsen, B., & Olsen, J. (2010). Neonatal jaundice, autism, and other disorders of psychological development. Pediatrics, 126(5), 872–878. https://doi.org/xxxx

[11] Watchko, J. F., & Tribelli, C. (2013). Bilirubin-induced neurologic damage—mechanisms and management approaches. New England Journal of Medicine, 369(21), 2021–2030. https://doi.org/xxxx

[12] Nascimento, G. B., Kessler, T. M., Ramos de Souza, A. P., Costa, I., & Moraes, A. B. (2020). Risk indicators for hearing loss and language acquisition and their relationship with socioeconomic, demographic, and obstetric variables in preterm and term babies. Codas, 32(1), e20180278. https://doi.org/xxxx

[13] Thirunavukkarasu, A. B., Vishnu, B. B., & Noyal, M. J. (2012). Association between peak serum bilirubin and neurodevelopmental outcomes in term babies with hyperbilirubinemia. Indian Journal of Pediatrics, 79(2), 202–206. https://doi.org/xxxx

[14] Amin, S. B. (2004). Clinical assessment of bilirubin-induced neurotoxicity in premature infants. Seminars in Perinatology, 28(5), 340–347. https://doi.org/xxxx

[15] Oh, W., Tyson, J. E., Fanaroff, A. A., Vohr, B. R., Perritt, R., Stoll, B. J., et al. (2003). Association between peak serum bilirubin and neurodevelopmental outcomes in extremely low birth weight infants. Pediatrics, 112(4), 773–779. https://doi.org/xxxx

[16] Graziani, L. J., Mitchell, D. G., Kornhauser, M., Pidcock, F. S., Merton, D. A., Stanley, C., et al. (1992). Neurodevelopment of preterm infants: Neonatal neurosonographic and serum bilirubin studies. Pediatrics, 89(2), 229–234.

[17] Hack, M., Wilson-Costello, D., Friedman, H., Taylor, G. H., Schluchter, M., & Fanaroff, A. A. (2000). Neurodevelopment and predictors of outcomes of children with birth weights of less than 1000 g: 1992–1995. Archives of Pediatrics & Adolescent Medicine, 154(7), 725–731. https://doi.org/xxxx

[18] Ozmen, A., Terlemez, S., Tunaoglu, F. S., Soysal, S., Pektas, A., Cilsal, E., et al. (2016). Evaluation of neurodevelopment and factors affecting it in children with acyanotic congenital cardiac disease. Iranian Journal of Pediatrics, 26(6), e3278. https://doi.org/xxxx

[19] Soylu, E., Soylu, N., Polat, C., Sakallıoğlu, O., Uçur, O., & Bozdoğan, G. (2016). Developmental delays in preschool children with adenotonsillar hypertrophy. Kulak Burun Boğaz İhtisas Dergisi, 26(3), 129–134. https://doi.org/xxxx

[20] Ertekin, A. A., Kapudere, B., Eken, M. K., Ilhan, G., Dırman, S., Sargın, M. A., et al. (2015). Does aggressive and expectant management of severe preeclampsia affect the neurologic development of the infant? International Journal of Clinical and Experimental Medicine, 8(11), 19325–19331.

[21] Amin, S. B., Prinzing, D., & Myers, G. (2009). Hyperbilirubinemia and language delay in premature infants. Pediatrics, 123(1), 327–331. https://doi.org/xxxx

[22] Rose, J., & Vassar, R. (2015). Movement disorders due to bilirubin toxicity. Seminars in Fetal & Neonatal Medicine, 20(1), 20–25. https://doi.org/xxxx

[23] Przekop, A., & Sanger, T. D. (2011). Birth-related syndromes of athetosis and kernicterus. Handbook of Clinical Neurology, 100, 387–395. https://doi.org/xxxx

[24] Durmazlar, N., Ozturk, C., Ural, B., Karaagaoglu, E., & Anlar, B. (1998). Turkish children's performance on Denver II: Effect of sex and mother's education. Developmental Medicine & Child Neurology, 40(6), 411–416. https://doi.org/xxxx

[25] Rowe, M. L., Raudenbush, S. W., & Goldin-Meadow, S. (2012). The pace of vocabulary growth helps predict later vocabulary skill. Child Development, 83(2), 508–525. https://doi.org/xxxx

[26] Gonzalez, J. E., Acosta, S., Davis, H., Pollard-Durodola, S., Saenz, L., Soares, D., et al. (2017). Latino maternal literacy beliefs and practices mediating socioeconomic status and maternal education effects in predicting child receptive vocabulary. Early Education & Development, 28(1), 78–95. https://doi.org/xxxx

Dijlah Journal of Medical Sciences (DJMS) Vol. 1, No. 3, May, 2025, pp. 44-52
P-ISSN: 3078-3178, E-ISSN: 3078-8625, paper ID: 06

فرط البيليروبين في الدم عند الأطفال حديثي الولادة وتأثيره على التطور العصبي

الخلاصة

يعد فرط بيليروبين الدم حالة شائعة عند الأطفال حديثي الولادة، وعادة ما تظهر في غضون الأسبوع الأول من الحياة. في حين أن الحالات الخفيفة غالباً ما تكون حميدة، فإن فرط بيليروبين الدم الشديد يمكن أن يؤدي إلى مضاعفات عصبية نمائية بسبب عبور البيليروبين للحاجز الدموي الدماغي. ارتبطت مستويات البيليروبين المرتفعة بحالات مثل ضعف الإدراك، وتأخر الكلام واللغة، واضطرابات نقص الانتباه، وخلل الحركة.

يعد التعرف المبكر على الرضع المصابين بفرط بيليروبين الدم ومراقبتهم أمراً بالغ الأهمية لمنع العواقب العصبية التنموية طويلة المدى.

شملت هذه الدراسة ٥٥ رضيعاً تم تشخيص إصابتهم بفرط بيليروبين الدم و ٦٥ من الضوابط الأصحاء. تم تقييم نتائج النمو العصبي باستخدام اختبار الفحص التنموي دنفر الثاني .(DDST) تضمن جمع البيانات المعلومات الديموغرافية، وتاريخ الميلاد، ومستويات البيليروبين الكلية، وتقييمات النمو العصبي.

تم إجراء التحليلات الإحصائية، بما في ذلك اختبارات Mann-Whitney U test، واختبارات مربع كاي، وحسابات نسبة الأرجحية، لتحديد الاختلافات بين مجموعات الدراسة والتحكم. مستوى الدلالة $p<0.05$ يعتبر ذا دلالة إحصائية.

أظهرت النتائج وجود فرق كبير بين مجموعات فرط بيليروبين الدم ومجموعات الضبط من حيث النمو العصبي العام .($p<0.001$) في حين لم تُلاحظ أي فروق ذات دلالة إحصائية في المجالات الشخصية والاجتماعية ($p=0.26$) والحركية الإجمالية($p=0.31$) ، أظهرت مجموعة فرط بيليروبين الدم تأخيراً ملحوظاً في المهارات الحركية الدقيقة ($p=0.046$)وتطور اللغة .($p<0.001$) بالإضافة إلى ذلك، أظهر الرضع الذين تجاوزت مستويات البيليروبين الكلية لديهم ٢٣,٥ مجم / ديسيلتر خطراً أعلى للتأخر في النمو العصبي. لم تؤثر العوامل الاجتماعية والاقتصادية مثل تعليم الوالدين والدخل بشكل كبير على النتائج التنموية بين المجموعتين.

نستنتج من هذه الدراسة إرتباط الأطفال حديثي الولادة المصابون بفرط بيليروبين الدم بتأخيرات كبيرة في النمو العصبي، وخاصة في المهارات الحركية الدقيقة واللغوية. في حين أن استراتيجيات العلاج الحديثة قللت من خطر الإصابة بأضرار عصبية شديدة، إلا أن الإعاقات المعرفية والحركية الدقيقة لا تزال تشكل مصدر قلق. ينبغي تنفيذ فحص النمو المنتظم للأطفال الذين لديهم تاريخ من ارتفاع بيليروبين الدم الشديد لتسهيل الكشف المبكر والتدخل في الوقت المناسب.