**Obat: Jurnal Riset Ilmu Farmasi dan Kesehatan**
**Volume. 2 No. 6 November 2024**
e-ISSN : 3031-0148, dan p-ISSN : 3031-013X, Hal. 288-296
DOI: https://doi.org/10.61132/obat.v2i6.879
*Available online at:* https://journal.arikesi.or.id/index.php/OBAT

# Effect Of Hyperbilirubinemia On Some Physiological Parameters In Infantile In Karbala City

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**Abstract**. Given that these substances' levels are essential for informing medical personnel about the body's reaction to the different treatments being administered, this study was planned to assessment some physiological variations to infants infected with hyperbilirubinemia. This will help the medical staff manage the condition properly with lower morbidity and mortality. In this study, 60 patients and 40 healthy controls (clinically evaluated by a specialist physician) are selected from males who are only 1 year old. This study conducted in Al-hussein medical city Hospital in Karbala city. This study showed that significant decrease in weight, hemoglobin and packed cell volume in patients in comparison with healthy, while noted significant increase in total white blood cells in patients comparison with healthy, while showed there are insignificant change in red blood cells and platelet.

**Keyboards:** *hyperbilirubinemia, hemoglobin and packed cell volume*

## Introduction

Jaundice is also known as ictrus, and it is characterized by hyperbilirubinemia-induced yellowish staining of the mucous tissues sclera and skin (1). For the color to be apparent, the blood's bilirubin level must be higher than 2-3 mg/dl. The French word jaune yellow is where the word jaundice originates (2), Jaundice that occurs during the first few months of life without accompanying symptoms is referred to as physiological jaundice(3). The infant is at high risk for jaundice due to poor calorie intake and/or dehydration brought on by inadequate breastfeeding, which results in weight loss, delayed stool passage, and postpartum bruising(4) (5) (6). Non-physiological jaundice in newborns has been linked to risk factors such as jaundice during the first 24 hours of life, jaundice observed prior to hospital discharge, a sibling who received phototherapy for jaundice, 35–36 weeks is the near-term gestational age, or cephalhematoma(7)(8). The most prevalent enzyme deficiency in humans is G6PD deficiency, which is an enzymatic disorder(9). Neonatals who experience hyperbilirubinemia inside the primary 24 hours of life, Asian males, levels of bilirubin higher than the 95th percentile, or a sibling's history of jaundice should all be evaluated for the G6PD deficiency (10). ß-globin chain production can either be completely absent in thalassemia (ß0-thalassemia) or partially reduced (ß+thalassemia), whereas α-thalassemia results in either absent or partially reduced α globin gene production (11). A surplus of α-globin in beta thalassemia causes α-globin tetramers to form and accumulate in the erythroblast, whereas an excess of ß-globin in α-thalassemia causes ß-globin tetramers, known as Hb-H, to form. These tetramers can

*Received: Agustus 12,2024; Revised: September 26,2024; Accepted: November 05, 2024; Published : November 22, 2024*

precipitate, damaging the RBCs membrane and causing RBCs rupture(12). Clinically known as ß-thalassemia major, beta homozygous thalassemia is a transfusion-dependent condition that manifests as varying degrees of anemia starting in early childhood. ß-heterozygous individuals with thalassemia minor, on the other hand, have normal or slightly lower hemoglobin levels and are essentially asymptomatic. Though, middle disorder that can have either a homozygous or heterozygous design of tradition, usually appears after the age of two, is severe enough to be compared to thalassemia minor, but its clinical course is milder than that of thalassemia major, and requires little to no blood transfusions(13). RH incompatibility arises when the fetus has Rh-positive blood and the pregnant woman has Rh-negative blood. Another name for Rh-induced newborn hemolytic illness (14). Unlike Rh disease, approximately 50% of ABO HDN cases involve a first-born child, and the condition does not worsen with subsequent pregnancies. Through increased hemolysis, the RH and ABO raise the production of bilirubin (15) Only a very small percentage of pregnancies in Caucasian populations result in symptomatic ABO HDN, but the mother and the fetus are incompatible with ABO in about one-fifth of pregnancies(16). Only mothers with blood group O can experience the latter due to their capacity to generate enough IgG antibodies to induce hemolysis(17).

**Literature review**

**2.1: Neonatal Jaundice:-**

Hemoglobin in red blood cells breaks down to produce bilirubin. Newborns' RBCs only live for 70 to 90 days, in contrast to older children whose cells last for 120 days(18). Newborn jaundice is caused by immature bilirubin breakdown systems, decreased hepatic blood flow, and increased RBC destruction(18).

**2.2.: Causes of indirect hyperbilirubinemia:**-(19).

A: Hemolysis disorders include drug-induced hemolysis (vitamin K), genetic hemolysis, and fetal-maternal blood incompatibility.

B: Hemostases, cerebral and pulmonary hemorrhages, and swallowed blood are examples of extravascular blood (petechie).

C: Placental transfusions (cord striping), maternal-fetal or feto-fetal transfusions, and chronic fetal hypoxia are examples of polycythemia.

D: Reduced perstalisis and mechanical obstruction are examples of excessive enterohepatic circulation.

E: Reduced bilirubin hepatic absorption includes ductusc venosus shunt persistence.

F: Gilbert disease, familial non-hemolytic jaundice, congenital reductoglucourenyl transferase, and enzyme inhibitor galactosemia are among the conditions that cause decreased bilirubin conjugation.

**2.3: Risk Elements for Elevated Bilirubinemia:-**

Total serum bilirubin levels in newborns without known danger reasons are hardly greater than 12 mg/dl. The likelihood of developing noticeably elevated bilirubin levels rises with the number of risk factors (20).

**2.3.1: Principal risk factors:-** (21).

•The TSB/TCB concentration in the area at great danger at discharge.

• During the initial twenty-four hours, jaundice was noted.

• Incompatibility of blood groups with other known hemolytic diseases (G6PD deficiency), as well as a positive direct antiglobulin test.

• 35–36 weeks gestational age.

• The former sibling underwent phototherapy.

• Serious bruises or a cerebral hemorrhage.

• Put off breastfeeding, particularly if you're losing a lot of weight and nursing isn't going well.

• The race of East Asia.

**2.3.2: Minor risk factors:-** (21).

• The discharge T/TCB concentration was in the zone of great intermediate danger.

• 37–38 weeks of pregnancy

• There was jaundice and discharge.

• An earlier sibling who had jaundice.

• A baby with macrosomia whose mother has diabetes.

• The mother is older than 25.

• The gender of men.

**2.4: Bilirubin Metabolism and Jaundice:-**

The following two phenomena occur simultaneously and cause neonatal physiological jaundice:-

1. A higher breakdown of fetal erythrocytes results in an increase in bilirubin production. This is because neonates have larger erythrocyte masses and fetal erythrocytes have a shorter lifespan.

2: Hepatocytes have low levels of The enzyme glucuronyl transferase, which changes bilirubin into glucuronic acid and renders it water soluble (conjugation), has low activity and the binding protein ligandin, both contribute to the low hepatic excretory capacity (20).

**2.5: Classification of neonatal jaundice:-**

**2.5.1: Physiological jaundice:-**

Jaundice is a common condition in newborns. In term neonates, the total serum bilirubin concentration usually peaks between days two and four at 5–6 mg/dl, whereas in preterm neonates, it peaks between days four and seven at 8–12 mg/dl. The jaundice reaches its maximum intensity on the fourth daytime in term neonates and the seventh daytime in preterm neonates, and it is clinically undetectable after 14 days. No treatment is necessary, but the infant should be closely monitored for any signs of worsening jaundice (22).

**2.5.2: Nonphysiological jaundice:-**

If direct-reacting bilirubin levels are higher than 2 mg/dl or if hyperbilirubinemia lasts longer than 10–14 days of life (10 days in full term and 21 days in preterm).However, for up to two weeks, breastfed infants may have mild jaundice, if jaundice appears within 24 hours of birth, if bilirubin levels increase at a rate greater than 0.5 mg/dl per hour or 5 mg/dl per day, if total bilirubin levels surpass 12 mg/dl in a full-term infant or 10–14 mg/dl in a preterm infant, or if acute hemolysis is visible, then jaundice must be classified as either non-physiological or pathological(22).

**Methods and Materials**

**blood samples collection and study groups**

This study aimed to quantify certain hematological alterations in hyperbilirubinemia patients. In this study, a 5-ml blood sample from a male patient who is only 1 year old is taken, and it is placed in a tube containing EDTA, an anticoagulant that prevents blood clotting, in order to be used for hematological investigations. There are 60 patients and 40 healthy controls (clinically evaluated by a specialist doctor). This study was carried out at Karbala's Al-Hussein Medical City Hospital.

**Hematological tests:**

Hematological tests were measured by (Sysmex xp300 B1269) an automatic hematoanalyzer. Among the hematological tests that were involved were platelet count, PCV, WBCs count, RBCs count and Hb.

**Examination of ESR:**

A Westergren tube that was open at both ends and had a diameter of 2.5 mm and a length of 300 mm was used. We mixed four parts blood (2 ml of blood and 0.5 ml of anticoagulant)

with one part anticoagulant (3.8% trisodium citrate solution). After drawing the mixture into a Westergren tube until it reached the zero mark, the tube was placed upright in a stand with rubber at the bottom and a coil clip on top. An hour later, the red cell column's top level was measured.

**Statistical analysis**

The data underwent one-way analysis of variance and was presented as mean ±SE. ANOVA Data analysis was conducted using IBM SPSS Program version 20, to ascertain whether there was a important change among means using LSD, the post hoc test was employed (23).

## Results

**Table (1) effect of hyperblirubinemia on weight (kg)**

| **weight** | | **Mean ± S. D.** | **No.** |
|---|---|---|---|
| **Groups** | patients | A **2.37 ± 0.46** | 60 |
| | Control | B **3.59± 0.13** | 40 |
| **Total** | | | 100 |

**At p≤0.05, distinct letters indicate a significant change.**

**Table (2) effect of hyperblirubinemia on some of hematological parameters (mean ± SE)**

| **WBC 10^3/mm** | | **PCV L/L** | | **Hb g/dl** | |
|---|---|---|---|---|---|
| **Patients** | **Controls** | **Patients** | **Controls** | **Patients** | **Controls** |
| **A** | **B** | **A** | **B** | **A** | **B** |
| **15.18± 1.09** | **6.73 ± 0.34** | **0.38± 0.027** | **0.49 ±0.023** | **10.13 ±0.74** | **15.74± 0.78** |

**At p≤0.05, distinct letters indicate a significant change**

**Table (3) effect of hyperblirubinemia on some of hematological parameters (mean ± SE)**

| RBCs count millions/mm$^3$ | |
|---|---|
| **Patients** | **Controls** |
| **A** | **A** |
| **4.49± 0.09** | **4.52±0.28** |

| Platelet count 10^3/mm | |
|---|---|
| **Patients** | **Controls** |
| **A** | **A** |
| **288.34± 10.303** | **288.74± 10.657** |

**At p≤0.05,**

**distinct letters indicate a significant change**

**Discussion**

This study shown that there are significant rise in total white blood cells in patients with **hyperblirubinemia** compared with control, and The current study's findings concur with those acquired by (24) and (25), who propose that a significant hemolysis is caused by a G6PD deficiency because the G6PD enzyme is crucial for shielding Hb and RBCs from oxidative stress. Regarding thalassemia, our findings concurred with (26) who described that no rise in WBC count in patients and disagreed with (27) who showed neutropenia might result from hypersplenism. while noted there are no significant difference in red blood cells. These findings supported (28) assertion that hemolysis was confirmed in a neonate with G6PD deficiency, however, hematological indices were unchanged from the controls. Severe hemolysis can happen in neonates lacking G6PD even when there are no hematological changes that would indicate a hemolytic process. This study showed that significant decrease in weight, hemoglobin and packed cell volume in patients compared with control. Results in this study in agreement with (25). (29) who showed significant decrease in Hb level in B-thalassemia patients. while noted there are no significant difference in platelet and red blood cells, the results agreed with (30) the cause of jaundice is hemolysis that affect the RBCs but not the bone marrow.(31) who found higher platelet counts in thalassemia patients.

**Conclusions**

This study suggests that ABO incompatibility-induced hemolytic disease in newborns is a major cause of neonatal hyperbilirubinemia, especially in the first three days of life, but it also results in mild hemolysis and anemia.

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