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# Assessment of Trace Elements on Newborns with Neonatal Jaundice Attending Federal Teaching Hospital, Owerri

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## ABSTRACT

**Original Research Article**

Neonatal jaundice is a very prevalent problem in newborn babies and continues to be a major cause of morbidity and hospital admission of neonates especially in low- and middle-income nations. Although the importance of bilirubin metabolism in the pathophysiology is well known, there is accumulating evidence that abnormalities in trace elements homeostasis may influence oxidative stress, integrity of erythrocytes and bilirubin metabolism. This study determined the serum bilirubin concentrations and other selected trace elements in neonates with neonatal jaundice attending Federal Teaching Hospital, Owerri. This comparison investigation was done on 120 babies (60 neonates with neonatal jaundice and 60 seemingly healthy newborns of the same age as controls). Venous blood (5 ml) was aseptically obtained from each participant using sterile disposable syringes and needles by a competent laboratory scientist or qualified health care provider. Samples were placed in plain specimen tubes, allowed to clot and retract, and centrifuged at 3000 rpm for 5 minutes to collect serum. Serum total, direct and indirect bilirubin, zinc, copper, iron, selenium and magnesium concentrations were tested by the proper laboratory methods. The data were analysed using Statistical Package for Social Sciences (SPSS) version 25.0. Mean values between the groups were compared by using Student's t-test. The link between bilirubin and trace elements concentrations was determined by using Pearson's correlation analysis. Results are reported as mean ± standard deviation, and $p<0.05$ was considered statistically significant. The mean levels of total bilirubin (18.36 ± 5.40 mg/dL), direct bilirubin (2.87 ± 1.09 mg/dL), and indirect bilirubin (15.50 ± 4.32 mg/dL) were significantly higher in neonates with neonatal jaundice compared to controls (7.13 ± 2.93, 0.72 ± 0.26, and 6.41 ± 2.68 mg/dL, respectively; p=0.000 for all). Newborns with neonatal jaundice had significantly lower serum concentrations of zinc, selenium, and magnesium compared to controls (54.66 ± 7.96 µg/dL vs 87.95 ± 9.10 µg/dL; 37.18 ± 7.68 µg/L vs 67.22 ± 6.88 µg/L; and 1.60 ± 0.20 mg/dL vs 2.16 ± 0.13 mg/dL, respectively; p=0.000 for all). In contrast, babies with neonatal jaundice had significantly higher levels of copper and iron (153.31 ± 17.87 µg/dL and 146.63 ± 16.02 µg/dL, respectively) compared to controls (118.72 ± 10.75 µg/dL and 98.37 ± 8.81 µg/dL, respectively; p=0.000 for both). Total bilirubin in neonates with neonatal jaundice was significantly negatively correlated with zinc ($r = -0.896$, p=0.000), selenium ($r = -0.766$, p=0.000), and magnesium ($r = -0.910$, p=0.000) but significantly positively correlated with copper ($r = 0.958$, p=0.000) and iron ($r = 0.797$, p=0.000). Neonatal jaundice in newborns showed considerable perturbation in serum trace element levels with decreased zinc, selenium and magnesium and elevated copper and iron concentrations. The substantial relationships between bilirubin and certain trace elements suggest that altered trace element homeostasis may be connected with the metabolic pathways involved in newborn jaundice. Additional research is needed to explain the mechanisms behind these changes and to assess the possible therapeutic significance of these changes in the therapy of newborn jaundice.

**Keywords:** Neonatal jaundice, hyperbilirubinemia, trace elements, zinc, copper, iron, selenium, magnesium, bilirubin, infants.

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## INTRODUCTION

Neonatal jaundice is a frequent clinical illness in the neonatal period and continues to be a significant cause of infant morbidity and hospital admission globally. It is characterised by yellowing of the skin, sclera, and mucous membranes due to increased levels of bilirubin in the blood, a disease called hyperbilirubinemia. Neonatal jaundice results when the amount of bilirubin produced exceeds the ability of the immature neonatal liver to metabolise and excrete bilirubin efficiently. Jaundice is normal and self-limited in many babies but it can become pathological when high bilirubin accumulates and leads to severe neurological problems if not detected and treated quickly [1].

Neonatal jaundice is among the most common diseases of the newborn infant affecting approximately 60–80 % of term and preterm newborns in the first week of life [2]. Jaundice resolves without major sequelae in

most of the affected neonates. However, severe neonatal hyperbilirubinemia remains a major public health issue due to the risk of acute bilirubin encephalopathy, kernicterus, permanent brain damage and death. The cost is particularly heavy in low- and middle-income countries, where delayed detection, poor access to bilirubin testing, insufficient phototherapy services and difficulties in accessing prompt healthcare contribute to preventable problems [3].

In developing nations, the burden of neonatal jaundice is further aggravated by the presence of factors that predispose neonates to high bilirubin production. These include glucose-6-phosphate dehydrogenase (G6PD) deficiency, haemolytic illness of the newborn due to ABO or Rhesus incompatibility, neonatal sepsis, preterm, birth trauma and inadequate nutrition. Limited access to prenatal and postnatal healthcare services, poor carer awareness, delayed presentation to health facilities and lack of access to efficient treatment may also contribute to the progression of uncomplicated jaundice to severe hyperbilirubinemia [4]. Neonatal jaundice is a significant burden in Nigeria since the ailment is frequently recognised among the leading reasons of neonatal admission and readmission in tertiary health care facilities. Prematurity, newborn infection, blood type incompatibilities, delivery trauma, G6PD deficiency, poor maternal awareness and delay in obtaining medical care have been implicated in the persistence of neonatal jaundice and associated consequences [5]. Severe hyperbilirubinemia therefore continues to be a substantial clinical, emotional and financial burden to the affected families and healthcare systems.

The pathophysiology of newborn jaundice is mostly related to the synthesis, metabolism and removal of bilirubin. Bilirubin is mostly derived from the degradation of haemoglobin after the destruction of senescent erythrocytes by the reticuloendothelial system. Heme oxygenase changes the heme to biliverdin. Biliverdin reductase changes biliverdin to unconjugated bilirubin. Unconjugated bilirubin is lipid soluble and is carried in the blood largely bound to albumin before being delivered to the liver. In hepatocytes, bilirubin conjugation is catalysed by uridine diphosphate glucuronosyltransferase (UGT1A1) to form water-soluble conjugated bilirubin that may be expelled in the bile and removed thru the gastrointestinal tract [6]

Newborns are physiologically prone to elevated bilirubin concentrations, due to their comparatively large red blood cell bulk, shorter erythrocyte life span, immature hepatic conjugation processes, and enhanced enterohepatic circulation. These features may lead to bilirubin production exceeding hepatic clearance in the early newborn period, leading to physiologic jaundice. However, pathological circumstances such haemolytic illness, sepsis, preterm, hereditary enzyme deficiencies, metabolic abnormalities and hepatic dysfunction can lead to increased bilirubin production or reduced clearance, resulting in severe hyperbilirubinemia.

Several maternal and neonatal variables have been implicated in the development and severity of newborn jaundice. Maternal factors are diabetes mellitus, prolonged labour, some drugs taken during pregnancy and blood group incompatibility. Neonatal factors include prematurity, low birth weight, birth asphyxia, cephalohaemotoma, bruising at birth, infection, dehydration, delayed feeding, genetic conditions such as G6PD deficiency. Important causes of severe newborn jaundice in resource limited settings are haemolytic disorders due to ABO or Rhesus incompatibility [7]. Poor intake of breast milk in early newborn period may also increase enterohepatic circulation and lead to hyperbilirubinemia.

Neonatal jaundice is of clinical concern mainly because of the neurotoxicity of unconjugated bilirubin. Unconjugated bilirubin can pass the blood brain barrier in large doses and deposit in brain tissues causing acute bilirubin encephalopathy. Lethargy, poor eating, hypotonia, high-pitched wailing, convulsions and altered awareness are all signs and symptoms. Kernicterus is an irreversible neurological illness linked with cerebral palsy, hearing loss, intellectual disabilities, speech difficulties, gaze abnormalities and dental enamel hypoplasia. It results from persistent or severe bilirubin toxicity. Such potentially fatal consequences signify the necessity for early detection, proper lab evaluation and prompt treatment [8]. Over the past several years, much emphasis has been given to the role of micronutrients and trace elements in the health and sickness of the newborn. Trace elements are minerals required in relatively small quantities, but are vital for a variety of physiological and biochemical functions, including growth and development, immunological control, enzymatic activity, antioxidant defence, and the maintenance of cellular homeostasis. Perturbations in the levels of these elements may produce detrimental health effects, especially during the newborn period when rapid growth and development are associated with significant metabolic demands [9].

Newborns are thot to be particularly vulnerable to the repercussions of trace element imbalance because of their inadequate nutritional stores, underdeveloped physiological systems and greater susceptibility to oxidative stress. Zinc, copper, iron, selenium, magnesium are all essential elements for normal haematopoiesis, immunological competence, neurological development, antioxidant protection, energy metabolism and cellular integrity. Changes in their concentration might consequently be of considerable importance for the health of the neonate [10]. Zinc is an essential micronutrient involved in over 300 enzymatic activities and is vital for DNA and protein synthesis, cell division, immunological control, tissue growth and antioxidant defence. It stabilises cell

membranes and protects against oxidative damage. It is a cofactor of antioxidant enzymes such as superoxide dismutase. Therefore, low availability of zinc may compromise immune function and antioxidant defence and increase susceptibility to oxidative injury [11].

Copper is a vital trace element, and a co-factor for enzymes involved in energy production, iron metabolism, connective tissue creation, neurotransmitter synthesis and antioxidant defence. Copper is a component of the proteins ceruloplasmin and copper-zinc superoxide dismutase, which play crucial roles in maintaining the redox balance of cells. Thus, abnormal copper concentrations may disrupt oxidative homeostasis and the function of erythrocytes. Iron is needed to make haemoglobin, to carry oxygen, for cell respiration and for a number of enzyme functions. Proper iron homeostasis is especially critical during the newborn period since both iron deficiency and excess iron availability may impair cellular function. Excess iron can generate reactive oxygen species thru Fenton chemistry, which can lead to oxidative stress and tissue damage.

Selenium is a component of various selenoproteins, such as glutathione peroxidase, an important antioxidant enzyme. It has a role in antioxidant defence, immunological modulation, thyroid hormone metabolism and protection from oxidative-stress induced tissue harm. Reduced selenium availability may decrease antioxidant capacity and increase susceptibility to oxidative and inflammatory processes. Magnesium is often studied along with trace elements, although it is normally considered a significant mineral and not a trace element, because it takes part in a number of enzymatic and cellular activities. It is needed for energy metabolism, nucleic acid synthesis, neuromuscular function, membrane stability, and cellular homeostasis. Therefore, adequate amounts of magnesium are required for appropriate physiological function in the newborn period.

Involvement in oxidative stress pathways and bilirubin metabolism may contribute to a possible association between trace element status and newborn jaundice. Oxidative stress is an imbalance between the production of reactive oxygen species and the ability of the biological system to detoxify reactive intermediates. Newborns are especially vulnerable because their antioxidant systems are still growing, and they face considerable metabolic and oxidative hurdles throughout the transition from intrauterine to extrauterine life. Several trace elements are cofactors or components of antioxidant enzymes and can therefore alter the ability of cells to resist oxidative injury [12].

Alterations in trace element levels may potentially impact bilirubin metabolism thru oxidative stress and erythrocyte integrity. Changes in zinc, selenium and other antioxidant-related micronutrients may weaken cellular antioxidant defence and increase oxidative damage which may be involved in erythrocyte injury and haemolysis and hence increased bilirubin production. In contrast, an excess of iron availability may cause oxidative stress by free-radical production. Bilirubin itself is an antioxidant so that interactions between bilirubin metabolism and trace element dependent antioxidant systems might be biologically significant [13]. Previous research have yielded contradictory outcomes despite a growing interest in the association between trace elements and newborn jaundice. Differences in reported findings may be due to changes in research populations, sample size, nutritional status, genetic background, environmental exposures, analytical methodologies and geographical location. The exact link of trace element homeostasis with newborn jaundice needs more clarification.

There is insufficient local evidence on the trace element profile of neonates with neonatal jaundice in Nigeria notably in the southeast of Nigeria. The clinical assessment of newborn jaundice has traditionally been based mostly on the measurements of bilirubin concentrations and known clinical risk factors, with relatively limited consideration of the potential significance of trace element abnormalities. In addition, trace element concentrations may differ according to the maternal nutritional status, socio-economic conditions, environmental exposures and population characteristics. This is thus a crucial knowledge gap, because of the scarcity of local data. Federal Teaching Hospital, Owerri, attends to neonates with neonatal jaundice from Owerri and environs. However, there is very little published information on serum concentrations of zinc, copper, iron, selenium and magnesium in the neonates with neonatal jaundice within this setting. The trend of these trace elements and their correlation with bilirubin concentrations may give important biochemical information to understand the pathogenesis of newborn jaundice in this population. Hence, this study evaluated serum concentrations of several selected trace elements viz. zinc, copper, iron, selenium and magnesium in babies with neonatal jaundice attending Federal Teaching Hospital, Owerri and compared with the values of apparently healthy age-matched neonates. The study also investigated the correlation between the bilirubin levels and some trace elements. The findings are intended to give local evidence of trace element homeostasis in neonatal jaundice, contribute to an understanding of the biochemical processes associated with the illness and provide a platform for further research into potential diagnostic, prognostic and therapeutic implications.

## MATERIALS AND METHODS

### Study Design and Setting

A hospital-based case-control study was conducted among newborns attending Federal Teaching Hospital, Owerri, Imo State, Nigeria. Federal Teaching Hospital, Owerri, is a tertiary healthcare facility located

in Owerri Municipal Local Government Area and serves as a major referral centre for Imo State and neighbouring states in southeastern Nigeria. The hospital provides specialized neonatal and paediatric services through its Special Care Baby Unit (SCBU), neonatal ward, paediatric outpatient clinic, and medical laboratory department.

Owerri, the capital of Imo State, is located in southeastern Nigeria at approximately 5.485°N latitude and 7.035°E longitude. The city is an important healthcare, educational, and commercial centre within the South-East geopolitical zone of Nigeria.

**Ethical Considerations and Participant Recruitment**

Ethical approval for the study was obtained from the Health Research Ethics Committee of Federal Teaching Hospital, Owerri, following submission of an introductory letter and the research proposal from the Department of Medical Laboratory Science, Imo State University, Owerri. Permission to conduct the study and recruit participants was also obtained from the hospital management and the Department of Paediatrics. Parents or legal guardians of eligible newborns were informed about the objectives, procedures, potential benefits, and nature of the study.

**Study Population and Sample Size**

The study comprised newborns aged 0–28 days diagnosed with neonatal jaundice and apparently healthy age-matched newborns without clinical evidence of jaundice who served as controls. A total of 120 newborns were recruited, comprising 60 newborns with neonatal jaundice and 60 apparently healthy controls.

**Blood Sample Collection and Serum Preparation**

Five millilitres of venous blood was collected aseptically from each participant by a trained laboratory scientist or qualified healthcare professional using a sterile disposable syringe and needle. Blood samples were transferred into labelled plain sterile specimen tubes and allowed to clot and retract at room temperature.

The clotted samples were centrifuged at 3,000 rpm for 5 min, after which the separated serum was transferred into appropriately labelled cryovials. Serum aliquots were used for the determination of total and direct bilirubin, while the remaining samples were stored at −20°C until analysis of zinc, copper, iron, selenium, and magnesium. Laboratory analyses were performed according to the respective standard operating procedures and manufacturers' instructions.

**Statistical Analysis**

Data were coded and analysed using the Statistical Package for the Social Sciences (SPSS), version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were summarized as mean ± standard deviation (SD). The independent-samples Student's *t*-test was used to compare mean serum concentrations of bilirubin and selected mineral elements between newborns with neonatal jaundice and controls. Pearson's correlation coefficient was used to assess the relationships between bilirubin concentrations and selected mineral elements among the study participants. Statistical significance was set at $p<0.05$.

## RESULT

**Table 1: Mean ± SD Values of Serum Bilirubin Levels in Newborns with Neonatal Jaundice and Apparently Healthy Control Subjects**

| Parameters | Neonatal Jaundice (n=60) | Control Subjects (n=60) | t-value | p-value (0.05) |
|---|---|---|---|---|
| **TBIL (mg/dL)** | 18.36 ± 5.40 | 7.13 ± 2.93 | 10.01 | 0.000* |
| **DBIL (mg/dL)** | 2.87 ± 1.09 | 0.72 ± 0.26 | 10.53 | 0.000* |
| **IBIL (mg/dL)** | 15.50 ± 4.32 | 6.41 ± 2.68 | 9.79 | 0.000* |

**KEY:**
n: Population size
*: Statistically significant (P < 0.05)
TBIL: Total Bilirubin
DBIL: Direct Bilirubin
IBIL: Indirect Bilirubin

**Table 2: Mean ± SD Values of Trace Elements in Newborns with Neonatal Jaundice and Apparently Healthy Control Subjects**

| Parameters | Neonatal Jaundice (n=60) | Control Subjects (n=60) | t-value | p-value (0.05) |
|---|---|---|---|---|
| **Zinc (µg/dL)** | 54.66 ± 7.96 | 87.95 ± 9.10 | -15.08 | 0.000* |
| **Copper (µg/dL)** | 153.31 ± 17.87 | 118.72 ± 10.75 | 9.09 | 0.000* |

| Parameters | Neonatal Jaundice (n=60) | Control Subjects (n=60) | t-value | p-value (0.05) |
|---|---|---|---|---|
| **Iron (µg/dL)** | 146.63 ± 16.02 | 98.37 ± 8.81 | 14.46 | 0.000* |
| **Selenium (µg/L)** | 37.18 ± 7.68 | 67.22 ± 6.88 | -15.96 | 0.000* |
| **Magnesium (mg/dL)** | 1.60 ± 0.20 | 2.16 ± 0.13 | -12.78 | 0.000* |

**KEY:**
n: Population size
*: Statistically significant (P < 0.05)

**Table 3: Pearson Correlation of Total Bilirubin with Zinc, Copper, Iron, Selenium and Magnesium in Newborns with Neonatal Jaundice**

| Variable | N | r TBIL | p-value |
|---|---|---|---|
| **Zinc** | 60 | -0.896 | 0.000* |
| **Copper** | 60 | 0.958 | 0.000* |
| **Iron** | 60 | 0.797 | 0.000* |
| **Selenium** | 60 | -0.766 | 0.000* |
| **Magnesium** | 60 | -0.910 | 0.000* |

**KEY:**
n: Population size
r: Pearson correlation coefficient
*: Statistically significant (P < 0.05)
TBIL: Total Bilirubin

## DISCUSSION

Neonatal jaundice is one of the most frequent clinical disorders in infants and continues to be a major contributor to neonatal morbidity especially in poor and middle-income countries where appropriate diagnosis and management may be difficult. The illness is due to an imbalance between the generation of bilirubin and its absorption, conjugation and excretion by the liver. Although neonatal jaundice is mostly physiological and self-limiting, severe hyperbilirubinemia can lead to acute bilirubin encephalopathy, kernicterus and other neurological sequelae if not addressed properly [14] There is an increasing body of evidence suggesting that micronutrient and mineral homeostasis may play a role in the aetiology of newborn jaundice through their involvement in antioxidant defence, erythrocyte integrity, cellular metabolism and oxidative stress. Thus, the present study was aimed at evaluating the serum bilirubin concentrations and some selected mineral elements in babies with neonatal jaundice attending Federal Teaching Hospital, Owerri. The results showed that the total bilirubin levels were considerably elevated in the neonates with neonatal jaundice compared to the seemingly healthy controls. This observation is consistent with the biochemical signature of newborn jaundice and represents the buildup of bilirubin due to the imbalance between bilirubin production and elimination. The relatively high bilirubin concentration in affected newborns may be related to the physiologic immaturity of hepatic bilirubin conjugating mechanisms, particularly reduced activity of uridine diphosphate glucuronosyltransferase, and the relatively short lifespan and increased turnover of neonatal erythrocytes. Moreover, the increased breakdown of foetal haemoglobin may also be involved in bilirubin generation throughout the newborn period [15] This observation is in agreement with the reports by [16] who reported significantly increased total bilirubin concentrations in jaundiced infants compared to healthy neonates.

The direct bilirubin level was also considerably greater in the babies with neonatal jaundice compared to the control group. Direct bilirubin is the conjugated part of bilirubin that has already been processed by the liver. Unconjugated hyperbilirubinemia is the most frequent biochemical pattern in the majority of neonatal jaundice cases, but higher direct bilirubin may be seen in combination with greater bilirubin turnover and the physiologic immaturity of hepatic and biliary excretory mechanisms. Therefore, hepatocellular immaturity and temporary constraints in bilirubin processing may contribute to the noted increase [17]. The present conclusion is in agreement with the report of [18], who reported considerably greater direct bilirubin concentrations in jaundiced neonates compared with the healthy babies. Neonates with neonatal jaundice also had considerably higher levels of indirect bilirubin compared to controls. This observation is consistent with the known pathophysiology of newborn jaundice in which unconjugated bilirubin often comprises the main fraction of circulating bilirubin. The immature hepatic conjugation system of infants, with comparatively low uridine diphosphate glucuronosyltransferase activity, leads to inefficient transformation of unconjugated bilirubin to its water-soluble conjugated form, resulting in its buildup in the circulation [19] The present finding is in agreement with previous observations by [20] who reported increased indirect bilirubin concentrations in infants with jaundice.

In addition, serum zinc levels in babies with neonatal jaundice were considerably lower than those in healthy controls. Zinc is an essential mineral that is involved in many enzymatic activities, antioxidant defence, membrane stability, immunological modulation, and cellular function. Lower zinc content in jaundiced group can suggest disturbed mineral homeostasis, which is related to metabolic and oxidative demands associated with hyperbilirubinemia. Zinc also has been linked in bilirubin metabolism and may alter

enterohepatic circulation by lowering intestinal reabsorption of unconjugated bilirubin [21]. Thus, the lesser availability of zinc may be potentially connected with greater retention of bilirubin. The current observation is in agreement with the reports of [22] who reported significantly decreased serum zinc concentrations in jaundiced infants compared with healthy controls.

When compared to zinc, the serum copper concentrations were considerably greater in neonates with neonatal jaundice. Copper is an essential mineral and acts as a cofactor for various enzymes involved in the antioxidant defence, iron metabolism and cellular redox control. Under some biological situations, however, increased levels of redox-active copper can induce oxidative stress. The elevated copper concentration identified in the present study may, then, be indicative of altered mineral metabolism related with newborn jaundice and the concomitant oxidative and inflammatory processes. Alterations in hepatic copper processing and transport may also account for fluctuations in circulating levels [23]. The present result is in agreement with the study of [24] who detected higher copper concentrations among jaundiced neonates.

Similarly, the serum iron values were considerably greater in the babies with neonatal jaundice than in the controls. Iron is a key component of haemoglobin formation, oxygen transport and cellular metabolism. Excess circulating iron may contribute to oxidative stress as iron can catalyse reactions that generate reactive oxygen species. The higher iron content seen in this study may be at least partially attributable to enhanced erythrocyte turnover and haemolysis, both of which are directly important to bilirubin production during the newborn period. This increase in serum iron after breakdown of haemoglobin may thus explain the observed change in serum iron concentrations [25]. Similar increases in iron concentrations have been documented among infants with hyperbilirubinemia [26].

Neonatal jaundice new borns showed a considerably decreased serum selenium levels when compared to controls. Selenium is a key component of various selenoproteins such as glutathione peroxidase that plays a crucial part in antioxidant defence and protection against oxidative cellular injury Reduced circulating selenium in jaundiced neonates may reflect impaired antioxidant equilibrium in the presence of hyperbilirubinemia. However, since the present study was observational, the drop cannot be considered as evidence that hyperbilirubinemia causes selenium deficiency. The observed variation may also be due to a different dietary state, metabolic requirements or other clinical reasons. The data however is consistent with observations by [27] who observed reduced selenium concentrations among jaundiced infants. Neonates with neonatal jaundice had likewise considerably lower serum magnesium concentrations compared with healthy controls. Magnesium has an essential role in several metabolic reactions such as energy metabolism, membrane stability, enzyme activity, and cellular homeostasis. The decrease noted may be due to changes in mineral metabolism or greater metabolic demands from newborn sickness and oxidative stress. Magnesium deficiency may also damage cellular and erythrocyte stability, and increase susceptibility to oxidative stress. The current conclusion is in agreement with the data published by [20], who observed low magnesium concentrations in neonates with hyperbilirubinemia.

Connection research indicated a substantial negative connection between total bilirubin and serum zinc in babies with neonatal jaundice. So the higher the amounts of bilirubin the lower the concentrations of zinc. The association might be indicative of interplay between bilirubin metabolism and oxidative stress and zinc-dependent cellular defence mechanisms. Zinc may potentially have an effect on bilirubin management via effects on enterohepatic circulation [8]. In [10] similar inverse correlations between bilirubin and zinc were identified. However, the association does not demonstrate whether changes in zinc status cause hyperbilirubinaemia or whether the two parameters are affected by other underlying causes.

There found a highly positive connection between total bilirubin and copper concentrations. Jaundiced neonates had greater serum bilirubin related with higher serum copper. This association may be a reflection of parallel changes in mineral metabolism and oxidative balance in newborn hyperbilirubinemia. As copper is involved in redox reactions, alterations in its circulating content may be related to oxidative processes associated with newborn disease. The result is in line with the favourable association described in [27]. However, further mechanistic studies are required to elucidate the molecular basis of this connection.

There was also a high positive connection between serum iron concentrations and total bilirubin. This link may represent the intimate biological relationship between degradation of haemoglobin and generation of bilirubin and iron metabolism. Haemoglobin is degraded and iron recycled as a consequence of the breakdown of erythrocytes, which also contributes to the formation of bilirubin. Thus, the positive connection between bilirubin and iron may be partly explained by common pathways of erythrocyte turnover and haem metabolism. [28] have documented similar association between iron status and hyperbilirubinemia.

A strong unfavourable association was also found between the total bilirubin and selenium concentrations in the study. This research demonstrates a negative association between serum selenium and

bilirubin concentrations in jaundiced neonates. Selenium-dependent antioxidant systems may be particularly important at times of increased oxidative stress, although the present cross-sectional correlation cannot determine the direction of the link. The observed connection is in agreement with [29] who showed an inverse association between selenium level and newborn jaundice.

Finally, serum magnesium content was significantly negatively correlated with total bilirubin. Thus, there was a decrease in magnesium concentrations with an increase in bilirubin concentrations among the jaundiced neonates. This association could represent coordinated abnormalities in mineral balance, oxidative stress and cellular metabolism during neonatal sickness. Similar observations have been reported in [30]. However, the processes behind this relationship are still unclear and need further studies.

## CONCLUSION

The study showed considerably higher serum total, direct and indirect bilirubin concentrations in neonates with Jaundice compared to apparently healthy controls. Significant changes in selected mineral elements were also found with decreased blood concentrations of zinc, selenium and magnesium, and higher serum concentrations of copper and iron in jaundiced neonates. Moreover, total bilirubin had substantial inverse associations with zinc, selenium and magnesium and significant positive correlations with copper and iron.

The results demonstrate that newborn jaundice is associated with significant changes in bilirubin metabolism and selective mineral homeostasis. Observed relationships may reflect interactions of oxidative stress, erythrocyte turnover, haem metabolism and bilirubin management. However, these data should be interpreted as connections, not causality. Additional longitudinal and mechanistic research are needed to determine if these mineral abnormalities contribute to the development or severity of newborn jaundice or are subsequent to the illness process.

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