PLASMA IONIZED MAGNESIUM AND CALCIUM LEVELS IN FULL-TERM SAUDI NEONATES WITH UNCONJUGATED HYPERBILIRUBINEMIA. A CROSS SECTIONAL STUDY

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ABSTRACT

BACKGROUND: Hyperbilirubinemia is one of the most common problems encountered in early neonatal period. Mg ions seem to act against or compensate for the neurotoxic effects of bilirubin molecules. We aimed to investigate and correlate the plasma levels of ionized Mg and Ca in full-term non hemolytic hyperbilirubinemia.

METHODS: It is a cross-sectional case control study included 100 Saudi full-term, neonates having unconjugated non hemolytic hyperbilirubinemia as the study group and another 50 healthy newborns as the control group during their follow up visit in the 1st 48 hr of postnatal life. Two blood samples were taken from each neonate; first at initial visit and the second 48 hours later to determine plasma ionized Mg and Ca and serum bilirubin levels.

RESULTS: The mean total, indirect & direct bilirubin levels, ionized Mg level and retics were significantly higher among cases ($P<0.001$). Significant positive correlations were found between the mean total, direct & indirect bilirubin levels and ionized Mg levels at admission. After 48 hours, significance positive correlations were found between the mean total & indirect bilirubin levels and ionized Mg levels ($p= 0.040$ and 0.038) respectively. No significant correlations were detected between ionized Ca and bilirubin levels.

CONCLUSION: Increased ionized Mg levels possibly has a neuroprotective role or a compensatory mechanism to reduce bilirubin toxicity. Further studies are needed to evaluate its predictive value in developing significant hyperbilirubinemia and its role in treatment of neonatal hyperbilirubinemia.

KEYWORDS: Ionized Mg, ionized Ca, full-term neonate, hyperbilirubinemia.

INTRODUCTION

Neonatal jaundice or neonatal hyperbilirubinemia, or neonatal icterus is a yellowing of the skin and other tissues of a newborn. A bilirubin level of more than 85 μmol/l (5 mg/dL) leads to a jaundice in neonates whereas in

adults a level of 34 μmol/l (2 mg/dL) is needed for this to occur. In newborns, jaundice is detected by blanching the skin with pressure applied by a finger so that it reveals underlying skin and subcutaneous tissue. **(1)** Severe unconjugated hyper-bilirubinemia can result in chronic bilirubin encephalopathy (kernicterus) which has mortality rate at least 10 percent and long-term sequelae at least 70 percent. **(2)** In neonates, jaundice tends to develop because of two factors - the breakdown of fetal hemoglobin as it is replaced with adult hemoglobin and the relatively immature metabolic pathways of the liver, which are unable to conjugate and so excrete bilirubin as quickly as an adult. This causes an accumulation of bilirubin in the blood (hyperbilirubinemia), leading to the symptoms of jaundice. **(1)**

Deposition of unbound bilirubin or its acid form in the neuron membrane causes permanent neuronal injury with a distinctive regional topography throughout the CNS considering the affinity of bilirubin molecule to phospho-lipids of the plasma membrane **(3)**, the sequence of membrane events initiated by bilirubin molecules damages all adjacent membrane-bound enzymes and receptors. However distant plasma membrane structures such as N-methyl-D-aspartate (NMDA) receptor/ion channel complex located within neuronal membranes on the synaptic surface of neurons are disrupted as well. **(4)**

NMDA receptor Ion\channel complex is one of the Iontropic glutamate receptors and has an important role during brain development. **(5)** It is a legand gated channel that contains multiple recognition sites responsible for modulation of its function. **(6, 7)** These include specific sites for glutamate, glycine and polyamines as spermine, magnesium and zinc, it also contains a selective ion channel for calcium, sodium and potassium. **(8)**

NMDA receptors are important for brain plasticity, neuronal growth, synaptogenesis, and the development of learning, memory and vision. Despite the physio-logical role of (NMDA) receptor in normal development of brain, increased activation of the receptor associated with brain cell injury. The immature brain is more sensitive to over stimulation than the adult brain. **(9)**

Magnesium ion is one of the most important antagonistic regulators of the NMDA recepto-r/ion channel complex. **(10, 11)** Many physiologic functions of Mg

ions seem to act against or compensate for the neurotoxic effects of bilirubin molecules. **(12)** It protects the CNS against hypoxia and exerts its neuroprotective effects by blocking excitotoxic and NMDA receptor–mediated neuronal injury mechanisms. **(13)** Plasma levels of ionized Mg, which is thought to reflect the metabolic status of the physiologically active fraction of Mg truly and accurately. **(14)** We aimed to investigate and correlate the plasma levels of ionized Mg and Ca in neonatal non hemolytic hyperbilirubinemia by comparing the newborns with and without significant hyperbilirubinemia.

MATERIALS AND METHODS

Study design

It is a cross-sectional case control study conducted in Neonatology Department at Heraa General hospital, Makkah, Saudi Arabia during the period from January 2014 to December 2014.

Subjects and inclusion Criteria

The study included 100 Saudi full-term, appropriate for gestational age neonates having unconjugated non hemolytic hyperbilirubinemia as the study group and another 50 healthy newborns as the control group during their follow up visit in the 1st 48 hr of postnatal life.

Exclusion criteria:

1. Low birth weight neonate,
2. Newborns with cephalohematoma, any congenital malformation, inborn error of metabolism, proven sepsis or infection,
3. Newborn of mother received Mg sulfate at any time during pregnancy, hypoxic ischemic and infant of diabetic mother,
4. Newborn with aneamia, sign of hemolysis or with hemolytic hyperbilirubinemia.

Procedures

Two blood samples were taken from each neonate having unconjugated hyperbilirubinemia; first at initial visit and the second 48 hours later. Birth weight, mode of delivery, sex, gestational age, Apgar score and postnatal age of cases and controls were recorded.

The following investigations were done for all study group and controls:

1. Serum ionized Mg **(15, 16)** and plasma ionized Ca levels. **(17)**
2. Serum bilirubin level (total, direct, indirect**). (18)**
3. To detect hemolytic jaundice, the following investigations were done:

- Hemoglobin level and hematocrit value, blood film, reticulocytic count. **(19)**
- Blood group and Rh-typing in newborn–mother pairs. **(19)**
- Glucose-6-phosphate dehydrogenase assessment (when indicated). **(18)**
- Direct coomb's test. **(19)**

4. C–reactive protein, complete blood count and ESR, to exclude sepsis. **(18, 19)**

Ethical consideration

Ethical Approval was obtained from the Medical Research Advisory Committee at Heraa General hospital, Makkah. Sampling was performed after obtaining a signed written informed consent from the legal guardians.

Statistical analysis

Statistical analysis was carried out using the SPSS computer package version 21.0 (SPSS Inc., Chicago, IL, USA). For descriptive statistics: the mean, SD and median were used for quantitative variables while the number and frequencies were used for qualitative variables. Fischer's exact test (FET) was used to assess the differences in frequency of qualitative variables while independent samples t-test was applied in order to assess the differences in means of quantitative variables. Pearson Correlation Coefficient was used to correlate the study variables. The statistical methods were verified, assuming a significant level of $p< 0.05$.

RESULTS

The study included 100 full-tern Saudi neonate and another 50 healthy controls. The mean age of hyperbilirubinemia group was 38.5 ± 0.78 weeks ranged from 37.0 – 40.0 weeks, half of them were males and the mean birth weight was 3.0 ± 0.66 Kg ranged from 2.7 – 4.3 Kg. The mean Apgar score at 1, 5 and 10 minutes was 7.04 ± 0.19, 8.1 ± 0.3 and 9.83 ± 0.38 respectively. The mean post natal age PNA (h) at the first visit was 76.08 ± 20.77 h and was 124.08 ± 20.77 h after 48 h from the first visit. Statistical significant differences were observed between cases and controls regarding gestational age (P=0.013) and Apgar at 5 minutes (P=0.021). The general characteristics of both groups were shown in (table 1).

Table (1): General characteristics of the studied groups.

Variables			Hyperbilirubinemia (n= 100)	Normal (Controls) (n= 50)	t/FET	P-value
GA (wks)		Mean ± SD Min-Max Median	38.5 ± 0.78 37.0 – 40.0 38.5	38.16 ± 0.77 37.0 – 40.0 38.0	2.52	0.013*
Gender	**Male**	No. (%)	50 (50.0)	26 (52.0)	0.05	0.864
	Female	No. (%)	50 (50.0)	24 (48.0)		
B. Wt (Kg)		Mean ± SD Min-Max Median	3.0 ± 0.66 2.7 – 4.3 3.0	3.18 ± 0.44 2.2 – 4.2 3.2	1.76	0.080
Apgar at 1 min		Mean ± SD Min-Max Median	7.04 ± 0.19 7.0 – 8.0 7.0	7.0 7.0 7.0	1.43	0.154
Apgar at 5 min		Mean ± SD Min-Max Median	8.1 ± 0.3 8.0 – 9.0 8.0	8.0 8.0 8.0	2.34	0.021*
Apgar at 10 min		Mean ± SD Min-Max Median	9.83 ± 0.38 9.0 – 10 10.0	9.76 ± 0.43 9.0 – 10 10.0	1.02	0.309
PNA (days) at 1st visit		Mean ± SD Min-Max Median	3.17 ± 0.86 1.0 – 5.0 3.0	3.0 3.0 3.0	1.39	0.168
PNA (days) after 48 h		Mean ± SD Min-Max Median	5.17 ± 0.86 3.0 – 7.0 5.0			
PNA (h) at 1st visit		Mean ± SD Min-Max Median	76.08 ± 20.77 24.0 – 120.0 72.0	3.0 3.0 3.0		
PNA (h) after 48 h		Mean ± SD Min-Max Median	124.08 ± 20.77 72.0 – 168.0 120.0			
PNA (h) at 1st visit		24 h	4 (4.0)			
		48 h	15 (15.0)			
		72 h	44 (44.0)	50 (100.0)		
		96 h	34 (34.0)			
		120 h	3 (3.0)			
PNA (h) after 48 h		72 h	4 (4.0)			
		96 h	15 (15.0)			
		120 h	44 (44.0)			
		144 h	34 (34.0)			
		168 h	3 (3.0)			

Values present as mean ± SD & analyzed by Independent Samples t-test.
Values present as number and % & analyzed by Fisher's Exact test.
*: Significant. PNA: Post natal age.

The mean total, indirect and direct serum bilirubin levels at 1st visit were 15.05 ± 2.02, 14.34 ± 1.97 and 0.71 ± 0.13 mg\dL respectively that decreased after 48 h to 8.63 ± 0.92 and 7.87 ± 0.93 mg/dL in both total and indirect levels respectively. The

mean ionized Ca and ionized Mg levels at 1st visit were 4.25 ± 0.47 and 2.23 ± 0.19 mg\dL respectively and were 4.52 ± 0.29 and 4.52 ± 0.29 mg/dL after 48 h respectively. The mean total, indirect & direct bilirubin levels, ionized Mg level and retics were higher among cases with statistical significant differences (P<0.001). Other laboratory findings of both groups were shown in (table 2 and figure 1).

Table (2) Laboratory findings of the studied groups.

Variables		Hyperbilirubinemia (n= 100)	Normal (Controls) (n= 50)	t	P-value
S. T. Bil (mg/dL) at 1st visit	Mean ± SD Min-Max Median	15.05 ± 2.02 12.0 – 19.2 14.8	5.25 ± 0.75 3.1 – 6.7 5.2	33.05	< 0.001*
S. T. Bil (mg/dL) after 48 h	Mean ± SD Min-Max Median	8.63 ± 0.92 6.7 – 11.0 8.5			
S. D. Bil (mg/dL) at 1st visit	Mean ± SD Min-Max Median	0.71 ± 0.13 0.3 – 0.9 0.7	0.41 ± 0.09 0.3 – 0.7 0.4	14.26	< 0.001*
S. D. Bil (mg/dL) after 48 h	Mean ± SD Min-Max Median	0.76 ± 0.1 0.6 – 0.9 0.8			
S. Ind. Bil (mg/dL) at 1st visit	Mean ± SD Min-Max Median	14.34 ± 1.97 11.2 – 18.3 14.1	4.83 ± 0.71 2.8 – 6.1 4.8	33.04	< 0.001*
S. Ind. Bil (mg/dL) after 48 h	Mean ± SD Min-Max Median	7.87 ± 0.93 5.8 – 10.2 7.8			
S. Ionized Ca (mg/dL) at 1st visit	Mean ± SD Min-Max Median	4.25 ± 0.47 3.0 – 5.4 4.1	4.32 ± 0.46 3.5 – 5.3 4.3	0.89	0.371
S. Ionized Ca (mg/dL) after 48 h	Mean ± SD Min-Max Median	4.52 ± 0.29 3.9 – 5.3 4.6			
S. Ionized Mg (mg/dL) at 1st visit	Mean ± SD Min-Max Median	2.23 ± 0.19 1.8 – 2.6 2.3	1.81 ± 0.16 1.6 – 2.3 1.8	13.12	< 0.001*
S. Ionized Mg (mg/dL) after 48 h	Mean ± SD Min-Max Median	2.05 ± 0.09 1.9 – 2.3 2.0			
Retics (%)	Mean ± SD Min-Max Median	1.37 ± 0.24 1.1 – 2.0 1.3	1.01 ± 0.08 0.85 – 1.2 1.0	10.23	< 0.001*
Hb (gm/dL)	Mean ± SD Min-Max Median	14.77 ± 0.84 12.9 – 17.0 14.7	14.73 ± 0.71 13.6 – 16.1 14.7	0.33	0.742

Values present as mean ± SD & analyzed by Independent Samples t-test.
*: Significant.

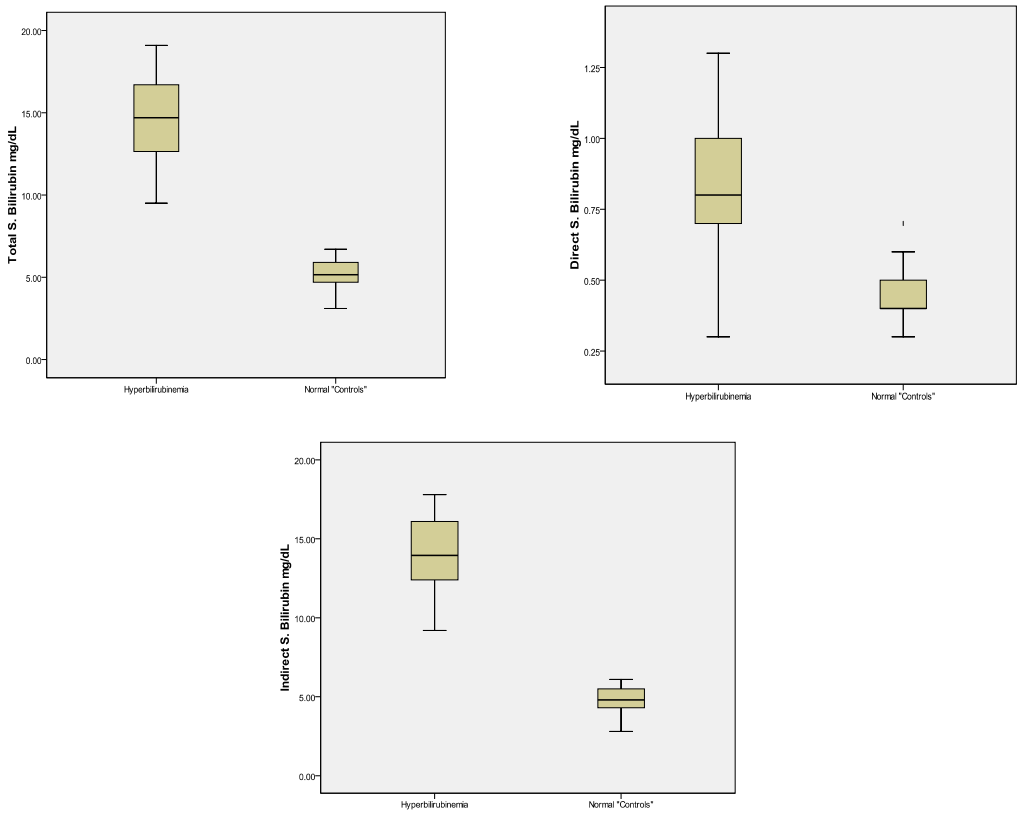

Figure (1): The mean total, direct and indirect S. bilirubin levels among hyperbilirubinemia cases and normal "control".

Significant positive correlations were found between the mean total, direct & indirect bilirubin levels and ionized Mg levels at admission ($p<0.001$). (table 3 and figure 2).

Table (3): Correlations between mean total, direct & indirect bilirubin levels and ionized Mg & ionized Ca levels among hyperbilirubinemia cases at admission.

Variables		r	P-value
Mean total bilirubin	**Ionized Mg**	0.48	**<0.001 ***
	Ionized Ca	- 0.02	0.907
Mean direct bilirubin	**Ionized Mg**	0.45	**<0.001 ***
	Ionized Ca	0.12	0.245
Mean indirect bilirubin	**Ionized Mg**	0.47	**<0.001 ***
	Ionized Ca	- 0.02	0.844

r: Pearson Correlation Coefficient. *: Significant.

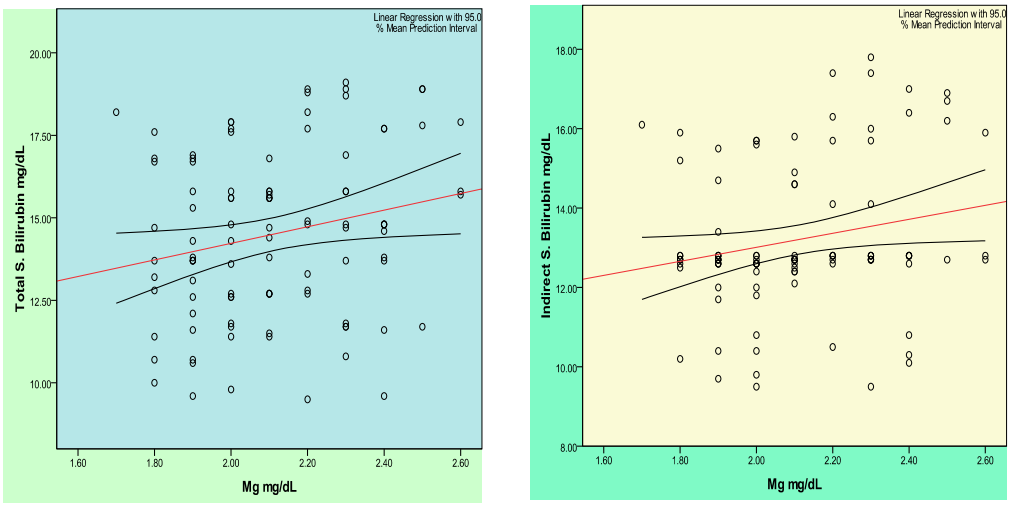

Figure (2): Significant positive correlations between the mean total & indirect bilirubin levels and Mg levels at admission.

Positive correlations were found between the mean total, direct & indirect bilirubin levels and both ionized Mg and Ca levels after 48 h with significance between total bilirubin and ionized Mg levels (p= 0.040). (table 4 and figure 3)

Table (4): Correlations between mean total, direct & indirect bilirubin levels and ionized Mg & ionized Ca levels among hyperbilirubinemia cases after 48 h.

Variables		r	P-value
Mean total bilirubin	**Ionized Mg**	0.21	**0.040 ***
	Ionized Ca	0.19	0.055
Mean direct bilirubin	**Ionized Mg**	0.13	0.197
	Ionized Ca	0.01	0.989
Mean indirect bilirubin	**Ionized Mg**	0.22	**0.038 ***
	Ionized Ca	0.19	0.059

r: Pearson Correlation Coefficient. *: Significant.

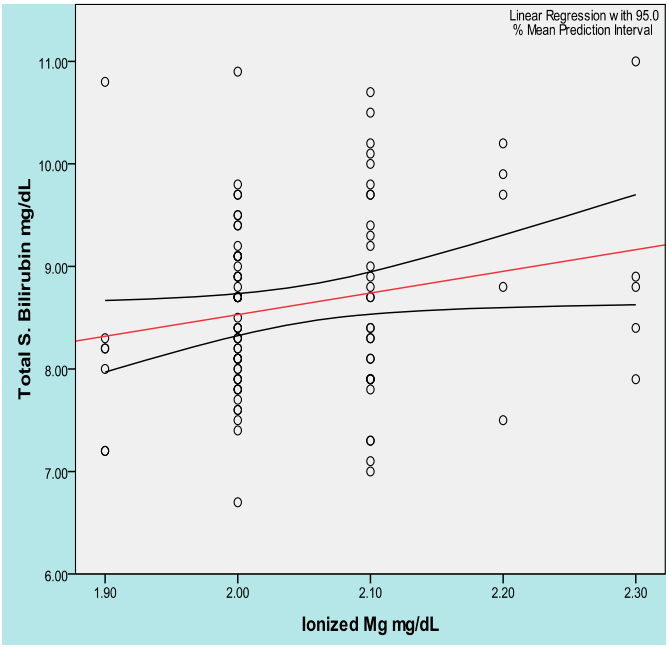

Figure (3): Significant positive correlation between the mean total S. bilirubin and ionized Mg levels after 48 h.

DISCUSSION

According to our result there is significant positive correlation between mean total and indirect bilirubin and ionized Mg level. These results suggest that increase in plasma ionized Mg may be due to extracellular movement of intracellular Mg resulting from cellular injury of neurons and erythrocytes. Also, bilirubin toxicity after the increase of serum bilirubin values to toxic levels not only is limited to neurons but also may cause generalized cellular injury. **(20)** Accordingly, in a study performed on pediatric intensive care patients, hypermagnesemia has been proposed to be a poor prognostic criterion being associated with critical cellular injury. **(21)** Increased levels of plasma Mg have been demonstrated in a few other situations in which generalized cellular injury occurs as a result of perinatal asphyxia and HIE **(22, 23)** and to neonatal hypoxemia and acidosis **(24, 25)** This finding also suggests the possibility of a neuroprotective role or a compensatory mechanism in ionized Mg increase against emerging toxicity risk of

increasing serum bilirubin values. **(15)**

Our result in agreement with El Masry et al. who found that there is positive significant correlation between plasma ionized Mg value and the mean serum indirect bilirubin in all studied cases. **(26)**

Also our result in agreement with Sarici et al. who investigated the level of ionized Mg in neonatal hyperbilirubinemia by comparing the newborn with and without significant unconjugated hyper-bilirubinemia and they found a significant positive correlation between plasma ionized Mg level in the group of significant unconjugated hyperbilirubinemia when compared with group of non significant unconjugated hyper-bilirubinemia. **(15)**

Our result disagreed with Tuncer et al. who investigated the serum level of zinc, copper and total Mg in umbilical cord blood of newborn with unconjugated hyperbilirubinemia and they reported lower zinc and total Mg concentration in newborns with unconjugated hyperbilirubinemia in comparison with newborn without unconjugated hyperbilirubinemia **(27)**.

The differences in Mg++ levels (decreased versus increased) between this study and our study may be due to type of Mg which measured as total Mg doesn't correctly reflect the free or ionized intracellular form of Mg++ that is physiologically active. Also may be due to differences in sampling (umbilical cord versus peripheral venous blood).

In the present study there is no significant correlation between mean total, direct and indirect bilirubin and ionized Ca this is in agreement with El Masry et al. who stated that there are no significant difference in the mean level of plasma ionized Ca between non hemolytic cases and control **(26)** and disagreed with Sarici et al., who stated that plasma ionized Ca level was significantly lower in a group of severe hyperbilirubinemia when compared with the group of moderate hyperbilirubinemia. **(15)**

Broner et al. reported that hyper-magnesemia and hypocalcaemia were found in 43.3% and 30% of the pediatric patients admitted to ICU respectively. **(28)** In another study, Ilves et al. investigate ionized Mg, ionized Ca and Na concentrations in asphyxiated term infant at age of 33 h (24–48h). At this age hypermagnesemia was discovered in 36% and hypocal-caemia in 33% of asphyxiated cases. **(29)** These finding suggest that hypocalcaemia and hyper-

magnesemia may be critical factors in the development of tissue injury.

It is important to interpret the results in the context of certain study limitations. First, as a cross-sectional study, it describes the relationship between variables as general association and not to be taken as cause-effect relationship. Second, the study did not include neonates born in other hospitals whether governmental or private which may affect the representativeness of samples in addition to the relatively small sample size.

CONCLUSION

In conclusion, both the positive correlation between plasma ionized Mg levels and severity of hyperbilirubinemia in newborns who had a wide range of serum indirect bilirubin levels (8.5–607 μM) and the presence of significantly higher plasma ionized Mg levels in newborns suggest that increase in plasma ionized Mg may be due to extracellular movement of Mg, a principally intracellular ion, resulting from generalized cellular injury including neurons and erythrocytes. Considering neuroprotective functions and beneficial effects of Mg ion in improving neurologic outcome, we also may speculate the possibility of a neuroprotective role or a compensatory mechanism of increased ionized Mg levels to reduce bilirubin toxicity.

Determination of the exact pathophysiologic process responsible for elevation of ionized Mg levels and demonstration of the relationship and interactions between ionized Mg and hyperbilirubinemia will make it possible to use cord blood or early postnatal ionized Mg measurements in predicting the development of significant hyperbilirubinemia and to question the value of Mg treatment in the therapy of neonatal hyperbilirubinemia.

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مستويات البلازما للماغنيسوم المتأين والكالسيوم فى حديثى الولادة السعوديين مكتملى النمو المصابين بارتفاع نسبة البيليروبين الغير مباشر فى الدم دراسة مقطع عرضى

أحمد السيد حمور * و بدوى أبوبكر يوسف **

***مدرس طب الأطفال بكلية الطب جامعة الأزهر ، ** أستاذ مساعد الباثولوجيا الإكلينيكية بكلية الطب جامعة الأزهر – القاهرة**

فرط بيليروبين الدم هي واحدة من المشاكل الأكثر شيوعا التي تتواجد في فترات العمر الأولى للأطفال حديثى الولادة. يبدو أن أيونات الماغنيسيوم تعمل ضد أو تعوض الآثار العصبية لجزيئات البيليروبين. ونحن تهدف إلى التحقيق وربط مستويات البلازما من الماغنيسيوم والكالسيوم المتأين في الأطفال حديثى الولادة مكتملى النمو المصابين بارتفاع نسبة البيليروبين الغير مباشر غير الانحلالى

الطرق المستخدمة :

شملت الدراسة المستعرضة مراقبة حالة 100 طفل سعودي حديثي الولادة مكتملى النمو من الذين يعانون من فرط بيليروبين الدم الغيرمباشر غير الانحلالى مقارنة بمجموعة الدراسة التى شملت 50 مولود حديثى الولادة مكتملى النمو لا يعانون من ارتفاع نسبة البيليروبين كمجموعة السيطرة خلال زيارتهم للمتابعة في ال 48 ساعة الأولى من حياتهم . تم أخذ عينات دم من كل الأطفال. أولا في الزيارة الأولى والثانية بعد 48 ساعة لتحديد مستوى البلازما للماغنسيوم المتآين والكالسيوم ومستويات البيليروبين في الدم.

النتائج :

كان متوسط مستويات البيليروبين الكلي وغير المباشر والمباشر، ومستوى الماغنيسيوم المتأين والريتيكس أعلى بكثير بين الحالات (P <0.001). تم العثور على ارتباطات إيجابية كبيرة بين متوسط مستويات البيليروبين الكلية، المباشرة وغير المباشرة، ومستويات الماغنيسيوم المتأين فى الزيارة الاولى. بعد 48 ساعة، تم العثور على ارتباطات إيجابية معنوية بين متوسط مستويات البيليروبين الكلية وغير المباشرة ومستويات الماغنيسيوم المتأين (p = 0.040 و0.038) على التوالي. لم يتم الكشف عن ارتباطات كبيرة بين الكالسيوم المتأين ومستويات البيليروبين

الاستنتاجات :

زيادة مستويات الماغنيسيوم المتآينة ربما يكون لها دور وقائى للأعصاب أو آلية تعويضية للحد من سمية البيليروبين. هناك حاجة إلى مزيد من الدراسات لتقييم قيمته التنبؤية في تطوير فرط بيليروبين الدم الكبير ودوره في علاج فرط بيليروبين الدم الوليدي.