ASSESSMENT OF GROWTH HORMONE LEVEL IN THE LOW BIRTH WEIGHT NEWBORNS

Mohammad M. El Mazahi*, Magdy M.A. Sakr*, Magdy Z. Al Ghannam; Hussein M. Abd El Maksoud*; Hanan Bakry***

*Pediatric; and **Clinical pathology Departments; Al-Azhar Faculty of Medicine

ABSTRACT

Background: Low birth weight is a significant medical problem especially in regions with high prevalence. Growth hormone may have a pathophysiological role in development of low birth weight.

Aim of the study: to investigate the relationship of growth hormone level at birth and birth weight to detect effect of growth hormone on intrauterine growth

Subjects and methods: This study was conducted on 66 neonates with Low birth weight. They were all full term, 35 of them were males and 31 of them were females. Meanwhile, 16 healthy neonates, 5 were males and 11 were females, served as control group. All neonates were subjected to full maternal history taking, thorough neonatal clinical examination and determination of serum growth hormone (GH) of neonates using ELISA technique, and blood glucose level.

Results of this study showed that there was a statistically significant increase in serum level of growth hormone (GH) in low birth weight neonates when compared to control neonates ($P<0.001$). Correlation studies revealed that among the studied neonates, there was significant negative correlation between growth hormone level and birth weight, birth length, gestational age, head circumference and abdominal circumference; there was significant positive correlation between growth hormone level and ponderal index and positive correlation between growth hormone level and blood glucose level.

Conclusion: serum levels of growth hormone are increased in low birth weight neonates reflecting its protecting effect to the fetal brain from hypoglycemia through inducing insulin resistance and providing alternative fuels for metabolism through their lipolytic effects, thus suggesting increased lipolysis in low birth weight neonates

Keywords: neonate; low birth weight, growth hormone**.**

INTRODUCTION

In regions with high prevalence of low birth weight, LBW had an enormous impact on health and reproduction of both individual and society **(Siega-Riz et al., 2004).** In addition, preterm delivery is a major cause of perinatal mortality and morbidity. The premature child represents one of the most important medical problems. The problems of prematurity are related to the difficulty

in extrauterine adaptation due to immaturity of organ and systems **(Vohr et al, 2005)**. In developing countries, approximately 70% of low birth weight infants have intrauterine growth restriction **(Adams-Chapman et al., 2008).** Children with intrauterine growth restriction (IUGR) are at higher risk for intellectual deficit and permanent debilitating short stature **(Holeman et al., 2003)**.

Growth hormone (GH) has been detected in the fetal circulation by 10 weeks' gestation and appears to originate exclusively from the fetal pituitary, independently of growth hormone releasing hormone (GHRH), pituitary GH or placental GH in the maternal circulation **(Setia et al., 2007).** The mitogenic actions of growth hormone are mediated through increases in the synthesis of insulin like growth Factor-I (IGF-I) which is mostly secreted by the liver and its secretion is regulated mainly by GH and nutritional status **(Parks an Felner, 2008)**. IUGR newborns were found to have low insulin levels **(Setia et al., 2007)**, which were found to correlate more strongly with ponderal index **(Setia et al., 2006)**.

AIM OF THE WORK

The aim of this work was to study the relationship of growth hormone level at birth and birth weight to detect effect of growth hormone on intrauterine growth.

SUBJECTS AND METHODS

Study Population: The present study comprised 82 neonates taken by random sampling from the Obstetric Department of Damietta Faculty of Medicine, Alazher University and Damietta General Hospital from January 2009 to June 2010.

These neonates were classified into four groups: **Group 1:** They were 22 preterm neonates, 12 were males and 10 were females, their gestational age ranged between 28 weeks and 36 wks, and their birth weight ranged between 800 -2400 gm. Gestational age was determined according to last menstrual period and new Ballard scoring system **(Ballard et al., 1991). Group 2:** They were 22 full term neonates, who were IUGR; 12 were males and 10 were females. Their gestational ages ranges between 37 weeks – 40 weeks. Their birth weight ranged from 1600 - 2460gm. "Birth weight at or below the 10^{th} percentile for gestational age and sex, according to weight centiles". **Group 3:** They were 22 preterm IUGR neonates; 11 were males and 11 were females. Their gestational age ranged between 28 weeks – 36 wks. Their birth weight ranged

between 650 – 1900gm. **Group 4:** They were 16 full term normal neonates as a control group; five were males and 11 were females. Their gestational age ranged between 38 - 40 wks. Their weight ranged between 3000 – 3500gm.

NB: All neonates had normal chest, cardiac, abdominal and neurological examination, with good neonatal reflexes. APGAR score at 5 min was 9 -10.

Exclusion criteria: newborn with congenital malformations or genetic disorders was excluded from the study.

All subjects were subjected to complete obstetric history with special emphasis on complications during pregnancy and medical history of the mother (diabetes, hypertension, preeclampsia, mal-nutrition, antepartum hemorrhage, anemia, smoking, drug intake and radiation exposure). Clinical examination included APGAR Scoring at one & five minutes, birth weight, length, head circum-ference, abdominal circumference and ponderal index. Systemic examinations for detection of complications including: cardiac, respiratory and abdominal exami-nations were done. Then, cord blood sample was taken after birth, for detection of growth Hormone by ELISA technique, and measurement of blood glucose level by quantitative enzymatic-colorimetric of glucose in blood by procedure No.1075

Statistical Methods: data were coded, entered and processed on computer using SPSS (Statistical Package for Social Science) (version 15). The level $P<0.05$ was considered the cut-off value for significance. Description of quantitative variables in the form of mean, standard deviation (SD) and range were performed. Student's t-test: was used to assess the statistical significance of the difference between two population means in a study involving independent samples. Correlation analysis: assessing the strength of association between two variables. The correlation coefficient denoted symbolically r, defines the strength and direction of the linear relationship between two variables. Chi-Square test χ^2: was used to test the association variables for categorical data. Fisher exact test: was performed in table containing value less than 5.

RESULTS

This study included 22 appropriate for gestational age (AGA) preterm neonates (26.8%), 22 full term IUGR neonates (26.8%) and 22 preterm IUGR neonates (26.8%) and 16 AGA full term neonates (19.5%) as a control group. Thus,

the total number of neonates in studied groups was 82 neonates: 40 males (48.8%) and 42 females (52.2%). The birth weight of studied neonates ranged between 0.65 and 3.50kg. Their gestational age ranged between 28 and 40 wks.

Table (1a): Comparison between study and control groups as regard to anthropometric measurements.

	Preterm AGA	Full term IUGR	Preterm IUGR	Control	P1	P2	P3
Wight (kg)	1.42±0.53	2.09±0.27	1.18±0.45	3.19±0.17	<0.001*	<0.001*	0.002*
Length (cm)	39.09±6.97	48.41±4.4	37.1±4.96	47.97±2.1	<0.001*	0.70(NS)	0.01*
Ponderal index	2.37±0.41	1.87±0.31	3.97±5.63	2.91±0.29	0.024*	<0.001*	0.46(NS)
Head C.(cm)	28.14±4.8	33.93±2.58	26.77±3.45	33.63±1.44	0.02*	0.67(NS)	0.003*
Abdominal C. (cm)	26.55±4.05	31.77±3.94	23.95±2.76	33.87±1.34	<0.001*	0.51(NS)	<0.001*

* = significant; NS = non-signficant; P1= test of significant between preterm AGA group and control group; P2: test of significance between full term IUGR and control group; P3: test of of significance between preterm IUGR and control group.

Table (1b): Comparison between study and control groups as regard to growth hormone and blood glucose levels in

	Preterm AGA	Full term IUGR	Preterm IUGR	Control	P1	P2	P3
Serum GH(ng/ml)	33.24±21.27	13.98±10.8	37.84±21.33	13.53±4.31	0.01*	0.87(NS)	0.014*
Bl. Gl (mg/dl)	113.36±55.23	61.68±0.13	113.55±1.26	68.81±14.99	0.03*	0.39(NS)	0.02*

* = significant; NS = non-significant

In the present work, there is significant decrease in AGA preterm group when compared to control group as regard to weight, length and abdominal circumference, ponderal index and head circumference. However, there is significant increase in AGA preterm group when compared to control group as regard to serum

GH level and serum glucose level. In addition, there is significant decrease in full-term IUGR group when compared to control group as regard to weight and ponderal index. On the other hand, there was no significant difference between full term IUGR group when compared to control group regarding length, head circumference, abdominal circumference, serum growth hormone level and serum glucose levels. Finally, there was significant decrease in preterm IUGR group when compared to control group as regard to abdominal circumference, weight, head circumference, length, and significant increase in serum growth hormone and blood glucose levels. But, there was no sigficant difference between both groups as regard to ponderal index (table 1a,b).

Table (2a): Relation between neonate gender and anthropometric measurements, GH and blood glucose in different groups.

Group	Gender	Weight (kg)	Length (cm)	Ponderal Index	Head C. (cm)	Abdominal C. (cm)
Control group	Male	3.05±0.07	46.8±2.05	2.99±0.32	33.5±1.67	32.9±0.55
	Female	3.25±0.17	48.5±0.98	2.87±0.28	33.68±1.42	34.32±1.36
	p	0.02*	0.14(NS)	0.44 (NS)	0.82 (NS)	0.041*
AGA Preterm Group	Male	1.04±0.20	33.8±2.3	2.66±0.21	24.67±1.58	23.5±2.02
	Female	1.87±0.42	45.4±5.04	2.01±0.29	32.3±2.08	30.2±2.38
	p	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*
Full-term IUGR	Male	1.93±0.22	46.3±3.9	1.97±0.29	32.58±2.52	29.75±4.24
	Female	2.28±0.19	50.9±2.89	1.75±0.28	35.55±1.57	34.20±1.49
	p	0.002*	0.002*	0.09(NS)	0.004*	0.005*
Preterm IUGR Group	Male	0.87±0.35	33.77±4.19	5.69±7.75	24.31±2.62	22.0±1.87
	Female	1.50±0.28	40.59±2.91	2.25±0.31	29.23±2.03	25.91±2.03
	p	0.002*	0.003*	0.16(NS)	0.004*	0.002*

* = significant; NS = non-signficant

Table (2b): Relation between neonate gender and GH & blood glucose in different groups

Group	Gender	Serum GH (ng/ml)	Bl. Gl (mg/dl)
Control group	Male	17.48±2.49	79.8±11.1
	Female	11.73±3.74	63.82±14.1
	p	0.01*	0.044*
AGA Preterm Group	Male	49.4±14.2	65.76±19.7
	Female	13.8±6.46	60.3±3.01
	p	<0.001*	<0.001*
Full-term IUGR	Male	19.06±12.07	77.17±31.95
	Female	7.90±4.31	43.10±12.93
	p	0.012*	0.005*
Preterm IUGR Group	Male	56.35±13.63	158.6±29.4
	Female	19.33±4.02	68.45±13.33
	p	<0.001*	<0.001*

* = significant; NS = non-significant

In control group, there was significant decrease of male weight and abdominal circumference when compared to corresponding female values. However, there was significant increase of both serum growth hormone and blood glucose in males when compared to females of control group. On the other hand, in AGA preterm group, there was significant decrease of weight, length, head circumference, abdominal circumference and significant increase of ponderal index, serum GH and blood glucose in males when compared to females. Furthermore, in full term IUGR group, there was significant decrease of weight, length, head circumference and abdominal circumference; while there was significant increase of serum GH and blood glucose in males when compared to females; however, the difference between males and females as regard to ponderal index was statistically non-significant. Finally, in preterm IUGR group, there was significant decrease of weight, height, head and abdominal circumferences and significant increase of serum GH and blood glucose levels in males when compared to females. However, the difference as regard ponderal index was statistically non-significant (table 2a,b).

Table (3a): Relation between mode of delivery and anthropometric measurements, GH and blood glucose in different groups

Group	Mode of delivery	Weight(kg)	Length (cm)	Ponderal Index	Head C. (cm)	Abdominal C. (cm)
AGA preterm group	NVD	1.82±0.59	43.30±8.27	2.30±0.65	30.50±4.37	29.20±2.79
	CS	1.30±0.46	37.85±6.28	2.38±0.34	27.44±4.12	25.76±4.06
	p	0.051(NS)	0.10(NS)	0.70(NS)	0.20(NS)	0.09(NS)
Full term IUGR	NVD	2.19±0.24	51.50±2.87	1.61±0.22	35.69±1.96	34.13±3.10
	CS	2.04±0.27	46.64±3.74	2.02±0.24	32.93±2.39	30.43±3.81
	p	0.20(NS)	0.005*	0.003*	0.01*	0.03*
Preterm IUGR	NVD	1.29±0.36	37.81±3.51	2.34±0.17	27.37±2.68	24.75±2.75
	CS	1.12±0.49	36.82±5.72	4.89±6.98	26.43±3.88	35.50±2.77
	p	0.50(NS)	0.60(NS)	0.30(NS)	0.50(NS)	0.30(NS)

* = significant; NS = non-significant.

Table (3b): Relation between mode of delivery and GH & blood glucose in different groups

Group	Mode of delivery	Serum GH (ng/ml)	Bl. Gl (mg/dl)
AGA preterm group	NVD	22.44±17.34	79.40±63.12
	CS	36.42±21.7	123.4±1.62
	p	0.20(NS)	0.10(NS)
Full term IUGR	NVD	7.36±5.90	45.87±18.51
	CS	17.77±11.3	70.71±32.3
	p	0.02*	0.06(NS)
Preterm IUGR	NVD	28.24±17.3	89.87±42.9
	CS	43.33±22.1	127.1±52.1
	p	0.10(NS)	0.10(NS)

* = significant; NS = non-significant.

In both AGA preterm group and preterm IUGR, there was no significant difference between those delivered by NVD and CS as regard weight, length, ponderal index, head circumference, abdominal circumference, serum GH or blood glucose. However, in full term IUGR group, there was significant increase of length, head and abdominal circumferences and significant decrease of ponderal index, and serum growth hormone in cases delivered by NVD when compared to those deliveries by CS (table 3a, b).

Table (4a): Relation between preeclampsia and controls as regard to anthropometric measurements.

	Preeclamptic Preterm AGA	Preeclamptic Full term IUGR	Preeclamptic Preterm IUGR	Control	P1	P2	P3
Wight (kg)	1.46±0.53	1.99±0.29	1.37±0.41	3.19±0.17	0.001*	<0.001*	<0.001*
Length (cm)	39.90±7.16	46.50±4.37	40.10±5.38	47.97±2.1	0.004*	0.60(NS)	0.003*
Ponderal I.	2.29±0.39	2.0±0.27	2.13±0.41	2.91±0.29	0.002*	<0.001*	<0.001*
Head C. (cm)	28.80±4.28	32.42±2.69	28.20±3.51	33.63±1.44	0.001*	0.08(NS)	<0.001*
Abdominal C. (cm)	26.60±4.72	29.17±4.06	25.20±2.41	33.87±1.34	<0.001*	0.003*	0.003*

* = significant; NS = non-significant

Table (4b): Relation between preeclampsia and controls as regard to GH and blood glucose.

	Preeclamptic Preterm AGA	Preeclamptic Full term IUGR	Preeclamptic Preterm IUGR	Control	P1	P2	P3
Serum GH (ng/ml)	30.81±22.8	21.22±14.8	29.38±19.8	13.53±4.31	0.04*	0.50(NS)	0.003*
Bl. Gl (mg/dl)	113.0±52.6	71.0±40.5	92.20±39.8	68.81±14.99	0.002*	0.70(NS)	0.014*

* = significant; NS = non-significant

In neonates delivered for pre-eclamptic mothers in preterm AGA group, there was significant decrease of weight, length, ponderal index, head and abdominal circumferences; where there was significant increase of both serum growth hormone and blood glucose; when compared to control group. However, in full-term IUGR neonates delivered for preeclamptic mothers, there was significant decrease of weighty, ponderal index and abdominal circumference in full-term IUGR when compared to control group. Finally, in neonates delivered for preeclamptic mothers in preterm IUGR group, there was significant decrease of weight, length, ponderal index, head and abdominal circumferences; where there was significant increase of both serum growth hormone and blood glucose; when compared to control group (table 4a,b).

Table (5): Correlation between levels of serum GH and other variables.

Parameters correlated	Growth hormone level	
	r	P
Gestational age (weeks)	0.91	<0.001*
Weight (kg)	0.85	<0.001*
Length (cm)	0.88	<0.001*
Ponderal index	0.24	0.04*
Head circumference (cm)	0.87	<0.001*
Abdominal circumference(cm)	0.85	<0.001*
Serum glucose (mg/dl)	0.99	<0.001*

* = significant; NS = non-significant

In the present work, there is significant positive correlation between serum growth hormone level and serum glucose levels (P <0.001). However, there is significant negative correlation between serum growth hormone level and gestational age, weight, length, head circumference and abdominal circumference (P <0.001). In addition, there is significant positive correlation between serum growth hormone

level and ponderal index; (P <0.05) (table 5).

DISCUSSION

Low Birth Weight is a major cause of infant morbidity and mortality **(Mandruzzato et al., 2008).** Growth hormone (GH), insulin and growth factors (insulin-like growth factors [IGFs] and their binding proteins [IGFBPs]) are known to influence fetal growth (**Chiesa et al., 2008).** The function of growth hormone (GH) in the fetus has not been conclusively established. The concentration of circulating GH-binding protein is low in fetal blood, probably reflecting the lower levels of expression of GH receptor in fetal tissue **(Setia and Sridhar, 2009**). Human placental lactogen acts as a fetal growth hormone and is a member of the same gene family, which consists of placental lactogen, growth hormone, and prolactin gene family. The growth-promoting actions of placental lactogen are mediated by stimulation of IGF production in the fetus and by increasing the availability of nutrients to fetal tissues **(Mittal et al., 2007).**

Evidently, growth hormone (GH) and insulin growth factor I (IGF-I) can function independently in the fetus **(Waters and Kaye, 2002).** However, the mean birth length of infants, with congenital growth hormone deficiency or growth hormone resistance is reduced by about 1 SD or 1 inch, indicating that growth hormone action accounts for some linear growth before birth. In contrast, mean birth weight is normal suggesting that these newborns have a relative excess of weight. The clinical impression that this excess consists mainly of fat is in line with experimental evidence attributing lipolytic and insulin-antagonizing properties to growth hormone before birth **(Setia et al., 2007).** Furthermore, this lipolytic effect of growth hormone depends on the fat localization. The highest lipolytic effect is exerted at the abdominal fat tissue. This explains the relative excess of weight and the increased amount of abdominal fat deposits in patients with growth hormone deficiency **(Delemarre et al., 2007).** The influence of GH on fetal size does not exceed 20% at birth, since GH targets may be concerned more with differentiation rather than growth and the ability of non-pituitary GH, placental growth hormone or placental lactogens to substitute for pituitary GH may obscure growth hormone actions **(Waters and Kaye, 2002).**

In this study, it was hypothesized that growth hormone levels would differ between AGA preterm, full term with intrauterine growth restriction (IUGR), preterm with IUGR and the full term AGA neonates (control group) because preterm and IUGR neonates represent abnormalities of growth and development.

In the current study gender had an effect on growth hormone levels in all studied groups (patients and controls) showing a statistically significant difference between males and females (males > females) as regards GH levels ($P<0.05$). This result is in agreement with **Geary et al. (2003)** who found higher GH concentrations in males than in females and they suggested that there are sexually dimorphic patterns of fetal growth regulation.

In the present study gender had a significant effect on birth weight, length**,** head circumference, and abdominal circumference measurement ($P<0.05$), which is in agreement with **Geary et al. (2003)** who found that birth weight, length, and head circum-ference measurements were different between males and females.

In studying the frequency of risk factors of low birth weight in patient groups, 32% of cases (patients) in the present study were due to maternal pre-eclampsia. This result is in agreement with **Xiao et al. (2003)** who suggested that women with pre-eclampsia were 36.6 times more likely to deliver a small for gestational age (SGA) newborn as compared to normotensive women, so there was a strong relationship between pre-eclampsia and restricted fetal growth.

In the current study 13% of cases, neonates were due to maternal hypertension. **Zetterstrom et al. (2006)** proved that chronic hypertension is an independent risk factor for birth of small for gestational age offspring. The results of this study revealed a statistically highly significant increase in serum levels of GH in LBW neonates compared to control group ($P<0.01$). Correlation results in our study revealed a very highly significant negative correlation between GH and birth weight ($P<0.001$). This result is in agreement with **Rajesh et al. (2000)** who demonstrated higher growth hormone levels in low birth weight (LBW) neonates and concluded that growth hormone has an important role to play in intrauterine growth.

In this study there was significant increase of serum levels of GH in preterm IUGR neonates

compared to control group (P<0.05). Higher growth hormone levels in intrauterine growth restricted neonates have been implicated in inducing insulin resistance and protecting the fetal brain from hypoglycemia. Growth hormone is involved in providing alternative fuels for metabolism through their lipolytic effects **(Setia et al., 2007).**

GH hypersecretion may be the direct or indirect result of a reduced negative feedback exerted by IGF-I that circulates in lower concentrations in growth-restricted fetuses **(Rajesh et al., 2000).** These results correlate with the results of ***Setia*** **et al. (2007)** who reported that at birth, intrauterine growth restricted infants display increased growth hormone levels which correlate negatively with birth weight.

This is in disagreement with **Chiesa et al. (2008)** who demonstrated that intrauterine growth restriction involved alteration in the fetal GH-IGF axis in the form of increased GH and decreased IGF-1 levels and found negative correlation between birth weight and cord blood growth hormone level. In addition, **Leger et al. (1996)** documented increased serum growth hormone levels in intrauterine growth restricted neonates as compared with normal neonates. On his prospective study for these neonates during the first 2 years of life all parameters (GH, IGF-I, and IGF binding protein (BP-3) levels were within normal limits concluding that none of the biologic parameters at birth were predictive of later growth. **Diaz et al. (1996)** reported higher serum levels of growth hormone in intrauterine growth restricted neonates at birth and demonstrated an endocrine profile of IUGR fetus to be: hypo-insulinemic, hypothyrotropinemic, hypoglycemic, hypoalbuminemic, hypocholesterolemic and hyper-magnesemic with lower IGF-I levels, yet higher than normal group growth hormone levels at birth**.**

In our study, there was statistically significant increase of growth hormone and level in both preterm and preterm IUGR neonates. This result is in agreement with **Zegher et al. (1990)** who found that cord serum growth hormone levels were significantly elevated in small for gestational age (SGA) infants, both at term and preterm birth and reported that these results further support a homeostatic function for growth hormone (by its insulin-antagonizing action) in the late-gestational human fetus. Furthermore, some authors like **Furuhashi et al.**

(1983) found a negative correlation between birth weight and serum growth hormone level at birth.

In our study, there was no significant increase in serum levels of GH in full term IUGR neonates compared to control group, these results are in agreement with **Yang et al. (2000)** who found no correlation between serum growth hormone level in the full term IUGR neonates and their birth weight and suggested that fetal growth depends on fetal levels of IGF-I, insulin-like growth factor binding protein (IGFBP)-3 and maternal factors, but not on insulin or growth hormone. Furthermore, **Kim et al. (1998)** found no correlation between serum growth hormone levels in the neonates and their birth weight**. Wiznitzer et al. (2001)** in his study on concordant (intertwin birth weight difference less than 20%) and discordant (intertwin birth weight difference greater than 20%) twin gestations found that growth hormone levels did not correlate with intertwin birth weight differences and stated that precise role of growth hormone in fetal growth restriction remains uncertain.

In the current study there was a statistically significant increase in levels of GH in AGA preterm and IUGR preterm neonates born to preeclamptic mothers when compared to control neonates ($P<0.05$). **Halhali et al. (2003)** found that IGF-I is significantly lower in the umbilical cord blood of small for gestational age (SGA) neonates both term and preterm born to preeclamptic mothers. This may explain our results. IGF-I circulates in lower concentrations in preterm and preterm IUGR neonates born to preeclamptic mothers. This leads to reduced negative feedback exerted by IGF-I on GH, thus leading directly or indirectly to this significant increase in levels of growth hormone in preterm and preterm IUGR neonates born to preeclamptic mothers compared to control neonates.

In this study there was significant increase in GH in AGA preterm neonates compared to control neonates ($P<0.05$). This is in agreement with **Rajesh et al. (2000)** who found that Growth hormone levels were higher in AGA preterm neonates as compared to term neonates.

In the current study, there was a highly significant negative correlation between GH level and gestational age ($P<0.001$).

This agreed with **Rajesh et al. (2000)** who found a negative correlation between cord blood growth hormone level and gestational age.

However, this is in contradiction with **Kim et al. (1998)** who found no correlation between growth hormone level and gestational age. Neonatal weight, length, head circumference, abdominal circumference in our study had a very highly significant negative correlation with GH level (P<0.001). This result is in agreement with **Chiesa et al. (2008)** who found that birth weight, birth length and head circumference negatively correlated to GH level.

In the current study there was a statistically very highly negative correlation between growth hormone level and abdominal circumference ((P<0.001). This is due to the lipolytic effect of growth hormone depends on the fat localization. The highest lipolytic effect is exerted at the abdominal fat tissue. This explains the relative excess of weight and the increased amount of abdominal fat deposits in patients with growth hormone deficiency **(Delemarre et al., 2007).**

In the current study a statistically significant positive correlation was found between ponderal index and GH level (P<0.05) which is in contradiction with **Setia et al. (2007) and Chiesa et al. (2008)** who found that ponderal index is negatively correlated to GH level. Ponderal index in intrauterine growth restricted infants with symmetrical growth restriction is normal, whereas those with asymmetric growth restriction have a reduced ponderal index due to a normal length but low weight **(Murphy et al., 2006).** This may explain our results as different affections of the growth restriction on birth length and the type of the growth restriction determine the ponderal index and thus may be normal in IUGR patients and low in others. Thus, a straight correlation may not be obvious.

In the present study there was a statistically very highly significant negative correlation between head circumference and GH level (P<0.001). This is in agreement with **Chiesa et al. (2008)** who found that head circumference is negatively correlated to growth hormone level.

In studying the correlations between GH and studied parameters, there was a very highly significant negative correlation between GH and both birth weight and birth length (P<0.001) as shown in table (5). This is in agreement with **Setia et al. (2007)** who found a negative correlation of growth hormone level in cord blood and birth weight reflecting the increased secretion of growth hormone in cases of LBW neonates.

In our study there was a very highly significant positive correlation between growth hormone level and blood glucose level ($P<0.001$), This result in agreement with **Hall (2006)** who found that GH is a diabetogenic hormone because it decreases glucose uptake in tissues and stimulates glucose production in the liver. GH thus acts to raise the blood glucose concentration and therefore antagonizes the actions of insulin.

In our study, in IUGR group there was significant difference between GH and mode of delivery, here GH is more with CS, this is in disagreement with **Hendler et al. (2005)** who found that GH concentrations were lowest in CS than NVD, but **Gardner et al. (2001)** who said that mode of delivery varies according to country, size of mother and size of fetus at birth.

In one word, this study indicates that GH levels were significantly increased in serum of low birth weight neonates as compared to AGA full term control neonates, which may reflect increased lipolysis in low birth weight neonates to provide alternative fuels for metabolism.

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تقييم مستوي هرمون النمو لدي ناقصي الوزن من حديثي الولادة

محمد محمد المزاحي*، مجدي محمد العشماوي صقر*، مجدي زكي الغنام، حسين متولي عبد المقصود*، حنان بكري***

قسمي طب الأطفال*، والباثولوجيا الإكلينيكية **(كلية طب الأزهر)

يمثل نقص الوزن لحديث الولادة مشكلة طبية هامة وخصوصا بالمناطق التي يرتفع فيها معدل حدوث نقص الوزن. وربما يلعب هرمون النمو دورا في آلية حدوث المشكلة (نقص الوزن).

صممت الدراسة الحالية بهدف تقييم مستوي هرمون النمو عند الولادة وعلاقته بوزن الوليد.

واشتملت الدراسة على 66 من حديثي الولادة ممن يعانون من نقص الوزن. وقد كان العمر الجنيني تام لدي جميع حديثي الولادة، و35 كانوا من الذكور، 31 من الإناث. كما اشتملت الدراسة على 16 من حديثي الولادة ممن لا يعانون من نقص الوزن، 5 من الذكور، 11 من الإناث، كمجموعة ضابطة. وقد تم أخذ التاريخ المتعلق بالحمل من كل السيدات المشاركات في الدراسة، كما تم فحص جميع حديثي الولادة إكلينيكيا، وأخيرا قياس مستوي هرمون النمو باستخدام الاليزا.

وقد أسفرت نتائج الدراسة عن وجود زيادة يعتد بها إحصائيا في مستوي هرمون النمو لدي مجموعة ناقصي الوزن عند مقارنتها بالمجموعة الضابطة. كما وجدت علاقة عكسية يعتد بها إحصائيا بين مستوي هرمون النمو ووزن الجسم عند الولادة، وطول الجسم، والعمر الجنيني، ومحيط الرأس والبطن. بينما وجدت علاقة طردية يعتد بها إحصائيا بين مستوي النمو ومعامل بوندرال وأيضا مع مستوي السكر بالدم.

وباختصار فقد أسفرت نتائج الدراسة عن عن زيادة هرمون النمو لدي ناقصي الوزن من حديثي الولادة، وهو ما يعكس الدور الوقائي للهرمون على مخ الجنين من نقص السكر، وذلك بإحداث مقاومة للأنسولين، وتقديم بديل أيضي عن طريق زيادة الدهون بالدم.