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Assessment of Levels of Metabolic Hormones and Lipid Profile in Growth Hormone Deficient Patients

Ghada Kh. Al-hindawi [1*], Makarim Q. Al-Lami[1] and Abdilkarim Y. Al-Samarriae[2]
[1] Department of Biology, College of Science, University of Baghdad, Baghdad, Iraq
[2] National Diabetic Center for Treatment and Research /Al-Mustansiriya University, Baghdad, Iraq

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Abstract

The current study aims to evaluate levels of metabolic hormones and lipid profile in a sample of growth hormone (GH) deficient patients. Seventy-five GH deficient patients and twenty healthy subjects, used as a control group, participated in this study during their attendance to the National Diabetic Center for Treatment and Research/Al-Mustansiriya University. The studied subjects' ages were with a range of 3-15 years. Blood samples were collected from the studied subjects to determine basal levels of GH, GH2 and GH3 after 1 hr. and 1:30 hr. of provocation with clonidine. In addition, levels of insulin-like growth factor-1 (IGF-1), metabolic hormones [thyroid profile: triiodothyronine (T3), thyroxin (T4), and thyroid stimulating hormone (TSH) and cortisol], and lipid profile elements [cholesterol, triglyceride, high density lipoprotein (HDL), low density lipoprotein (LDL) and very low density lipoprotein (VLDL)] were studied.

The findings of the anthropometric measurements of the studied groups revealed that insignificant ($P>0.05$) difference was found in the weight between the patients (26.59 ± 1.35 kg) and the control (30.95 ± 2.09 kg), while the mean of height in the patients (123.35 ± 2.18 cm) was significantly ($P<0.05$) lower than its value in the control (132.72 ± 3.60 cm). Mean BMI values demonstrated insignificant ($P>0.05$) differences between the patients (16.71 ± 0.54 kg/m^2) and the control (17.12 ± 0.55 kg/m^2). However, mean BMI percentile and mean BMI Z-score revealed a significant ($P<0.05$) decrease in the patients (27.90 ± 3.16; -1.107 ± 0.21) compared to their values in the control (44.30 ± 5.75; -0.169 ± 0.16), respectively.

The results also showed insignificant ($P>0.05$) differences in the level of basal GH between the patients (0.39 ± 0.04 ng/ml) and the control (0.57 ± 0.18 ng/ml), while a highly significant ($P<0.01$) decrease was found in the levels of GH2 (2.97 ± 0.27 ng/ml) and GH3 (2.43 ± 0.23 ng/ml) in the patients group compared to the control group (12.70 ± 0.45 ng/ml; 8.10 ± 0.43 ng/ml), respectively. The Level of IGF-1 showed a significant ($P<0.05$) decrease in the patients (128.75 ± 10.69 ng/ml) compared to the control (176.85 ± 16.36 ng/ml).

The results of metabolic hormones revealed insignificant ($P>0.05$) differences in the level of serum T3 between the patients (2.39 ± 0.07 nmol/L) and control (2.19 ± 0.12 nmol/L), while a highly significant ($P<0.01$) decrease was found in serum T4 level in the patients (98.19 ± 1.76 nmol/L) compared to control (114.85 ± 4.42 nmol/L). An insignificant ($P>0.05$) difference was found in serum TSH between the patients (2.51 ± 0.14 µIU/ml) and control (2.45 ± 0.14 µIU/ml). The data of serum cortisol showed a significant ($P<0.05$) increase in the patients (138.72 ± 18.05 ng/mL) compared to control (112.61 ± 13.92 ng/mL).

The results of lipid profile showed insignificant ($P>0.05$) differences in serum cholesterol level between the patients (186.34 ± 5.56 mg/dL) and control (150.35 ± 4.31 mg/dL), while triglyceride showed a significant ($P<0.05$) increase in the patients (88.90 ± 6.19 mg/dL) compared to control (61.40 ± 4.15 mg/dL). An

*Email: ghadakhairy98@gmail.com

insignificant (P>0.05) difference was found in serum HDL between the patients (44.26 ± 1.72 mg/dL) and control (46.55 ± 1.75 mg/dL), while a significant (P<0.05) increase was found in levels of LDL and VLDL in the patients (110.55 ± 4.99 mg/dL; 17.88 ± 1.23 mg/ml) compared to the control (85.22 ± 5.01 mg/dL; 12.97 ± 0.88 mg/ml), respectively.

Distribution of the studied groups according to gender revealed that most of the patients with GHD (60 %) were boys while (40 %) of them were girls, while the control was divided into two subgroups (60 % boys and 40 % girls). Distribution of the studied groups according to BMI values showed that the percentage of underweight was higher in the patients (36%) compared to the control (5%), while the percentage of normal weight was higher in the control (95%) compared to the patients (46.7%).

It can be concluded from the present study that the diagnosis of GHD cannot be done at the basal serum of GH. A high level of GH was detected after 1 hr. provocation with clonidine compared with its value after 1.30 hr. provocation. The IGF-1 is an appropriate parameter to expect GHD in children and adolescences whom GHD was detected by GH stimulation testing. Low GH secretion is associated with a high level of cortisol resulting in GHD. Patients with GHD displayed a tendency towards lipids disturbances. Growth hormone deficiency appear to be prevalent in males and predominant in underweight GH deficient patients.

Keywords: Growth hormone, metabolic hormones, lipid profile

تقدير مستويات الهرمونات الايضية واشكال الدهون في مرضى نقص هرمون النمو

غادة خيري مالك[1]، مكارم قاسم اللامي[1] و عبد الكريم يحيى السامرائي[2]

[1]قسم علوم الحياة، كلية العلوم، جامعة بغداد، بغداد، العراق

[2]المركز الوطني لعلاج وبحوث السكري، الجامعة المستنصرية، بغداد، العراق

الخلاصة

تهدف الدراسة الحالية الى تقدير مستويات الهرمونات الايضية واشكال الدهون في عينة من مرضى نقص هرمون النمو. تمت مشاركة 75 مريضا بنقص النمو و 20 من الاشخاص الاصحاء، كمجموعة سيطرة، اثناء مراجعتهم للمركز الوطني لعلاج وبحوث السكري/الجامعة المستنصرية، اعمارهم بين (3–15 سنة). جمعت عينات الدم من المشاركين بالدراسة لتقدير مستويات هرمون النمو الاساسي (GH)، هرمون النمو بعد ساعة (GH_2) وساعة ونصف (GH_3) من الحث باستخدام الكلوندين، على التوالي؛ عامل النمو شبيه الانسولين (IGF–1)؛ مستويات الهرمونات الايضية [صورة الغدة الدرقية والتي تشمل ثلاثي يودات الثايرونين (T3)، الثايروكسين (T4)، والهرمون المحفز للغدة الدرقية (TSH)، وهرمون الكورتيزول؛ واشكال الدهون [الكولستيرول، الكليسيريدات الثلاثية، الكوليستيرول عالي الكثافة (HDL) ,الكوليستيرول واطىءالكثافة (LDL), والكوليستيرول واطىء الكثافة جد(VLDL)].

اظهرت نتائج المؤشرات البشرية للمجاميع المدروسة فرق غير معنوي (P>0.05) في الوزن بين المرضى والسيطرة، بينما كان معدل الطول اوطأ معنويا (P<0.05) في المرضى مقارنة مع السيطرة. اظهر معدل مؤشر كتلة الجسم (BMI) فرق غير معنوي (P>0.05) بين المرضى والسيطرة، بينما اظهرت معدلات النسبة المئوية لل BMI (BMI percentile) و(BMI Z–score) نقصانا معنويا (P<0.05) في المرضى مقارنة مع السيطرة.

اظهرت النتائج وجود فرق غير معنوي (P>0.05) في مستوى GH الاساسي بين المرضى والسيطرة، بينما وجد نقصان معنوي عالي (P<0.01) في مستويات GH_2 GH_3 في المرضى مقارنة مع السيطرة. اظهر مستوى (IGF–1) نقصان معنوي (P<0.05) في المرضى مقارنة مع السيطرة.

اظهرت نتائج الهرمونات الايضية فرق غير معنوي (P>0.05) في T3 المصل بين المرضى والسيطرة، بينما وجد نقصان معنوي عالي (P<0.01) في T4 المصل في المرضى مقارنة مع السيطرة. وجد فرق غير

معنوي (P>0.05) في TSH المصل بين المرضى والسيطرة. اظهرت معطيات كورتيزول المصل زيادة معنوية (P<0.05) في المرضى مقارنة مع السيطرة.

اظهرت نتائج اشكال الدهون فرق غير معنوي (P>0.05) في كولستيرول المصل بين المرضى والسيطرة، بينما اظهرت الكليسيريدات الثلاثية زيادة معنوية (P<0.05) في المرضى مقارنة مع السيطرة. وجد فرق غير معنوي (P>0.05) في HDL المصل بين المرضى والسيطرة، بينما وجدت زيادة معنوية (P<0.05) في مستويات LDL وVLDL في المرضى مقارنة مع السيطرة. اظهر توزيع المجاميع المدروسة حسب الجنس ان معظم مرضى نقص هورمون النمو (60%) من الاولاد بينما (40%)منهم هم من البنات وكذلك في مجموعة السيطرة(60%)هم من الاولاد و (40%) هم من البنات.اظهر توزيع المجاميع المدروسة حسب قيم مؤشر كتلة الجسم ان فئة الوزن القليل في المرضى كانت اعلى معنويا(36%)مقارنة مع مجموعة السيطرة(5%)،بينما كانت فئة الوزن الطبيعي اعلى معنويا في مجموعة السيطرة (95%)مقارنة مع المرضى(46.7%).

من الدراسة الحالية بالامكان الاستنتاج انه لا يمكن تشخيص نقص هرمون النمو اعتمادا على مستوى هرمون النمو الاساسي. ان العامل الشبيه بالانسولين يعد دلالة موثوقة لعمل هرمون النمو. ترتبط قلة افراز نقص هورمون النمو بمستوى عال من الكورتيزول مما يؤدي الى نقص هورمون النمو. اظهر المرضى الذين يعانون من نقص هورمون النمو الميل نحو اضطرابات الدهون. ان نقص هرمون النمو يكون اكثر سيادة في الذكور، وكذلك سيادة قلة الوزن في مرضى نقص هرمون النمو.

Introduction

Growth Hormone (GH) is a polypeptide hormone secreted by the anterior pituitary gland and is the principal driver of statutory growth throughout childhood [1]. Secretion of GH is stronger in the childhood and adolescent age range than in adulthood [2]**.** GH stimulates statutory growth directly by its action at the growth plate and indirectly through the production of insulin like growth factor-I (IGF-I) [3]. It also has an effect in most tissues, including lipolysis, protein synthesis, and reduced glucose utilization [4].

Insulin-like growth factor-I is a single chain polypeptide sharing great structural homology with proinsulin [5]. IGF-1 plays essential roles in the growth and development of the human body, as well as in cell proliferation, migration, differentiation, and survival at the cellular level [6].

Growth hormone deficiency (GHD) is defined as the lack of or inadequate GH secretion that results in a decrease in the creation of GH-dependent hormones and growth factors, such as IGF-1, IGF-2 and their binding proteins [7]**.** GHD may arise in childhood or be adult-onset in nature; childhood causes may be subdivided into those which are congenital or acquired [8].

Thyroid hormones are vital to steady body growth in infancy and childhood; they regulate the rate of body's metabolism and temperature [9]. Thyroid hormones apply a permissive effect upon the anabolic and metabolic effects of GH and increase the pituitary synthesis of this hormone. GH reduces the secretion of thyrotropin and the thyroid hormones and increases the peripheral conversion of T4 to T3 [10]**.**

Cortisol is one of the steroid hormones synthesized from cholesterol in the adrenal cortex; it has a role in the accumulation of GH granules in the cytosol of the cells in the anterior pituitary [11]. High levels of cortisol in children can restrict growth due to its capability to down-regulate GH [12]. In addition, the growth plate is directly targeted by increased cortisol levels in children under stress [13].

Growth hormone acts on the adipose tissue to increase lipolysis, leading to the release of free fatty acids into the blood [14]. Children with GHD reveal a tendency towards a lipid disturbance. Also, GH levels are inversely correlated with abdominal fat mass [15]. Therefore, GHD causes increased abdominal fat, unusual carbohydrate and lipid metabolism [16].

Materials and Methods

Studied subjects

Seventy-five GH deficient patients (45 boys and 30 girls), along with twenty healthy subjects (12 boys and 8 girls) in terms of none-GHD who were used as a control group, participated in this study during their attendance to the National Diabetic Center for Treatment and Research/Al-Mustansiriya

University. The subjects' ages were within a range from 3 to 15 years. The anthropometric measurements of the studied subjects were recorded in a form of questionnaires.

Collection of blood samples

Venous blood samples (5 ml) were collected from the studied subjects after overnight fasting. The blood was transferred to a clean dry gel tube and allowed to clot at 37^{o}C for 30 minutes. The tubes were centrifuged at 5000 rpm for 5 minutes, and then the serum was collected and kept at -20^{o}C until used.

Assessment of BMI, BMI percentile and BMI Z-score

Body mass index (BMI) of the studied subjects was calculated by the following equation [17]:

BMI =Weight (kilograms) /Height (meters^{2})

Percentiles assort the position of an individual by indicating what percent of the reference population the individual would meet or override. While, Z-score is a numerical measurement used in statistics of a value's relationship to the mean of a group of values divided by the standard deviation for the reference population [18].

Estimation of GH and IGF

Serum GH and IGF-I levels were estimated by a sandwich chemiluminescence immunoassay [19].

Determination of metabolic hormones (thyroid profile assay and cortisol)

Minividas device was used to carry out the thyroid profile assay (T3, T4 and TSH) and cortisol according to the manufacturer's recommended method by using a specific kit for each hormone [20].

Lipid profile assay

Levels of cholesterol, triglyceride, and HDL were estimated spectrophotometrically using commercial kits [21]. Levels of LDL and VLDL were calculated according to Friedewald equation [22].

Statistical analysis

The statistical analysis was performed using the statistical analysis system (SAS, 2012) program and computer software. All data were expressed as mean ± standard error (SE). Student's t-test was used to compare between the studied groups and Chi-square test was used for the significance comparison between the percentages. The level of significance was determined at P<0.05 [23].

Results and Discussion

Distribution of the studied subjects according to sex

Table-1 shows the distribution of the studied subjects according to sex. It is clear that most of the patients (60%) were males, while (40%) of them were females. These findings are statistically considered as highly significant (P<0.01). Also, the control group showed the same distribution as the patients (60% were males and 40% were females), indicating the age matching between the patients and control groups.

Table 1-Distribution of the studied subjects according to sex

Study group	Sex				P-value
	Male		Female		
	No.	%	No.	%	
GH deficient patients	45	60	30	40	0.0094 **
Control	12	60	8	40	0.0094 **
** (P<0.01)					

These results are similar to those reported by other authors [24] who demonstrated that GHD was more prevalence in males than females; the reasons why most cases of GHD were among males are possibly that the function of the pituitary and the particular secretion of GH might be more vulnerable in males than female [25]. The explanation behind the results of the control group which are similar to those of the patient's group is to match between the selected samples.

Distribution of the studied subjects according to the BMI values

Distribution of the studied groups according to BMI is shown in Table-2. The results indicated that the percentage of underweight was significantly (P<0.01) higher in the GH deficient patients (36%)

compared to the control (5%). While, the percentage of normal weight was significantly (P<0.01) higher in the control (95%) compared to the GH deficient patients (46.7%). In the same time, the percentage of overweight was significantly (P<0.05) higher in the GH deficient patients (10.7%) compared to the control (0%).

Table 2-Distribution of the studied subjects according to BMI values

BMI Categories	Patients		Control		Chi-Square (X^2)
	No.	%	No.	%	
Underweight	**27**	**36**	**1**	**5**	**8.93 ****
Normal Weight	**35**	**46.7**	**19**	**95**	**6.62 ****
Overweight	**8**	**10.7**	**0**	**0**	**4.52 ***
Obese	**5**	**6.6**	**0**	**0**	**2.17 NS**
Total	**75**	**100**	**20**	**100**	**-------**
*** (P<0.05), ** (P<0.01), NS: Non significant**					

The current results are in agreement with those of a previous study [26] which stated that some of the GH deficient patients were underweight. This finding may be due to some factors such as the malnutrition, socioeconomic status, region, and the sex of the patients. The higher number of individuals within the normal weight range in the control subjects as compared to the GH deficient patients confirms the previous notion that BMI cannot be considered as a specific indicator in the diagnosis of GHD [27]. On the other hand, the higher number of overweight cases in the patients may be due to the fact that the deficiency of GH causes an increased visceral fat [16]. A previous study [28] reported that spontaneous and stimulated peak GH levels are lower in overweight and obese GH deficient patients and these studies suggested an inverse correlation between stimulated peak GH and BMI value in obese cases.

Anthropometric measurements of GH deficient patients and control

Table-3 shows the anthropometric measurements of GH deficient patients and control. A non-significant difference was found in the mean age between the patients (10.76±0.37 year) and the control (10.70±0.92 year). The results of weight also revealed a non-significant difference between the patients (26.59±1.35 kg) and the control (30.95±2.09 kg), while the mean of height in the patients (123.35±2.18 cm) was significantly (P<0.05) lower than its value in the control (132.72±3.60 cm). The results of BMI detected a non-significant difference between the patients (16.71±0.54 kg/m^2) and the control (17.12±0.55 kg/m^2). The mean values of BMI percentile (27.90±3.16%) and BMI Z-score (-1.107±0.21) revealed significant (P<0.05) decreases in the patients compared to their values in the control (44.30±5.75 % and -0.169±0.16, respectively).

Table 3-Anthropometric measurements of GH deficient patients and control group.

Anthropometric measurements	Mean ± SE		P -value
	Patients	Control	
Age	10.76^{a} ±0.37	10.70^{a} ± 0.92	0.945 NS
Weight (kg)	26.59^{a} ± 1.35	30.95^{a} ± 2.09	0.128 NS
Height (cm)	123.35^{b} ± 2.18	132.72^{a} ± 3.60	0.0457*
BMI (kg/m^2)	16.71^{a} ± 0.54	17.12^{a} ± 0.55	0.711NS
BMI percentile (%)	27.90^{b} ± 3.16	44.30^{a} ± 5.75	0.05*
BMI Z-score	-1.107^{b} ± 0.21	-0.169^{a} ± 0.16	0.031*
*** (P<0.05), NS: Non-Significant**			

- Means with different superscripts within each row are significantly different (P<0.05).
- Means with similar superscripts within each row are non-significantly different (P>0.05).

A non-significant difference in the mean of age between the patients and the control group can be due to the matching in the range of the age between the subjects of the two groups. The recent results of the weight are similar to those reported in a previous study [28], which revealed a non-significant difference in weight between GH deficient patients and control. However, the present results disagree with those published by another group [29]. The discrepancies between the studies may be due to the

differences in sample size, nutritional status and the lifestyle of the studied subjects. Similar to the present finding, a previous study [30] reported a significant decrease in the height in the GH deficient patients compared to the control group. This finding may be due to the fact that the cause of short stature is the GHD which severely limits bones maturation [31].

The present results of BMI are in agreement with those of an earlier work [28], which reported non-significant differences in BMI between patients and control. This finding could be due to the fact that BMI measurement incorporates weight and height [32]. Also, in the current study, most of the studied subjects were within the normal weight range. The statistical analysis of BMI percentile revealed a significant decrease in the patients compared with the control group. Z-scores and percentiles are interchangeable, and the choice to use either one is based primarily on convention or preference [18]. The current results are in agreement with the data published by other authors [33], who reported significant differences between patients and control in BMI Z-score. BMI Z-score and percentile were calculated by a BMI calculator or by a growth chart for boys and girls presented by the Centers for Disease Control and Prevention in 2015). In this study, the studied subjects were within the normal range, which is in agreement with Lee *et al*., 2013 [28], based on a BMI range between the 5^{th} and the 84^{th} percentile [18].

Levels of GH and IGF-1 in GH deficient patients and control

The data presented in the Table-4 shows levels of GH and IGF-1 in the studied groups. A non-significant difference was found in basal GH level between the patients (0.39±0.04 ng/ml) and the control (0.57±0.18 ng/ml). However, a highly significant (P<0.01) decrease was found in levels of GH_2 after 1hr. of provocation with clonidine (2.97±0.27 ng/ml) and in levels of GH_3 after 1:30 hr. of provocation with clonidine (2.43±0.23 ng/ml) in the patients compared to control (12.70±0.45 ng/ml and 8.10±0.43 ng/ml, respectively). According to the present findings, it is obvious that the peak GH was recorded after 1hr. of provocation with clonidine (GH_2) in the studied groups. Regarding the level of IGF-1, the results showed a significant (P<0.05) decrease in the patients (128.75±10.69 ng/ml) compared to the control (176.85±16.36 ng/ml).

Table 4-Levels of GH and IGF-1 in GH deficient patients and control

Parameters	Mean ± SE		P -value
	Patients	Control	
Basal GH (ng/ml)	0.39^{a} ± 0.04	0.57^{a} ± 0.18	0.161 NS
Peak GH_2 (ng/ml)	2.97^{b} ± 0.27	12.70^{a} ± 0.45	0.01**
GH_3 (ng/ml)	2.43^{b} ± 0.23	8.10^{a} ± 0.43	0. 01**
IGF-1 (ng/ml)	128.75^{b} ± 10.69	176.85^{a} ± 16.36	0.0343*
*** (P<0.05), ** (P<0.01), NS: Non-Significant.**			

- Means with different superscripts within each row are significantly different (P<0.05), (P<0.01**)**
- Means with similar superscripts within each row are non-significantly different (P>0.05), (P>0.01**)**

The present findings are in agreement with a previous investigation [34], which indicated no significant difference at basal GH between GH deficient patients and control. This may be due to the pulsatile manner of GH secretion, being less in the day and peaks at the night time. Also, there are several factors such as stress, heavy exercises, aging, fasting and obesity that modulate the production of GH [35]. The current results of GH_2 and GH_3 levels are in agreement with the data from another study [34], which showed similar results when a comparison was investigated between GH deficient patients and control, on one hand, and between their levels after 1 hr. and after 1:30 hr. of provocation with clonidine, on another hand. This may be due to the pulsatile way of GH secretion and its increased level after the stimulation test.

Regarding the results of serum IGF-1 levels, they are in agreement with those of Abdul Rahem and Al-Samarraie, 2012 [35], who reported a significant decrease in serum IGF-1 levels in GH deficient patients when compared to control. The explanation behind these results could be that IGF-1 is a dependable indicator of GH function and is affected by many factors such as age, sex, fasting state and liver disorders [36].

Metabolic hormones' levels in GH deficient patients and control

The results of metabolic hormones' levels in the studied groups are shown in Table-5. A non-significant difference was found in serum T3 level between the patients (2.39±0.07 nmol/L) and the control (2.19±0.12 nmol/L), while a high significant (P<0.05) decrease was found in serum T4 level in the patients (98.19±1.76 nmol/L) compared to the control (114.85±4.42 nmol/L). The results revealed a non-significant difference in serum TSH level between the patients (2.51±0.14 µIU/ml) and the control (2.45±0.14 µIU/ml). The data of serum cortisol revealed that a significant (P<0.05) increase was found in the patients (138.72±18.05 ng/mL) compared to the control (112.61±13.92 ng/mL).

Table 5-Metabolic hormones levels in GH deficient patients and control

Metabolic hormones	Mean ± SE		P-value
	Patients	Control	
T3 (nmol/L)	2.39^{a} ± 0.07	2.19^{a} ± 0.12	0.0186 NS
T4 (nmol/L)	98.19^{b} ± 1.76	114.85^{a} ± 4.42	0.01**
TSH (µIU/ml)	2.51^{a} ± 0.14	2.45^{a} ± 0.14	0.825 NS
Cortisol (ng/mL)	138.72^{a} ± 18.05	112.61^{b} ± 13.92	0.0432*
* (P<0.05), ** (P<0.01), NS: Non-Significant.			

- Means with different superscripts within each row are significantly different (P<0.05), (P<0.01**)**
- Means with similar superscripts within each row are non-significantly different (P>0.05), (P>0.01**)**

Numerous data demonstrated that thyroid hormones are strongly involved in the regulation of body growth [37]. Regarding the thyroid profile findings, the recent results are in agreement with those from other authors [37, 38]. It was found that all the GH deficient patients were euthyroid, while the use of L-T4 drug from the start of GH replacement therapy in euthyroid patients was not recommended due to a slight evidence for the rise of clinically significant hypothyroidism in most of formerly euthyroid patients [38]. Overall, all these previous studies strongly suggest that it would be difficult to study GH regulation and physiological effects without taking thyroid function into account. The results of serum cortisol are similar to those t reported by other authors [39] who stated that low GH secretion is associated with a high level of cortisol and that this disorder results in GHD. Also, hypercortisolemia predisposes to unfavorable lipid profiles and abdominal fat accumulation, which are strongly associated with low GH levels [40].

Lipid profile in GH deficient patients and control

The results of the lipid profile are presented in Table-6. Cholesterol levels showed a non-significant difference between the patients (186.34±5.56 mg/dL) and the control (150.35±4.31 mg/dL), while TGs level showed a significant (P<0.05) increase in the patients (88.90±6.19 mg/dL) compared to the control (61.40±4.15 mg/dL). Concerning the HDL level, a non- significant difference was found between the patients (44.26±1.72 mg/dL) and the control (46.55±1.75 mg/dL), while there was a significant (P<0.05) increase in levels of LDL and VLDL in the patients [(110.55±4.99 mg/dL and 17.88 ±1.23 mg/dL, respectively) when compared to their values in the control [(85.22±5.01 mg/dL and (12.97±0.88 mg/dL), respectively).

Table 6-Lipid profile in GH deficient patients and control

Lipid profile	Mean ± SE		P -value
	Patients	Control	
Cholesterol (mg/dL)	186.34^{a}± 5.56	150.35^{a}± 4.31	0.105 NS
Triglyceride (mg/dL)	88.90^{a}± 6.19	61.40^{b}± 4.15	0.0268*
HDL (mg/dL)	44.26^{a}±1.72	46.55^{a}± 1.75	0.513 NS
LDL (mg/dL)	110.55^{a}±4.99	85.22^{b} ± 5.01	0.0478*
VLDL (mg/dL)	17.88^{a}± 1.23	12.97^{b}± 0.88	0.0428*
* (P<0.05), NS: Non-Significant			

- Means with different superscripts within each row are significantly different (P<0.05).
- Means with similar superscripts within each row are non-significantly different (P>0.05).

The current results are in agreement with a previous study [41] which showed non-significant differences in levels of cholesterol and HDL and a significant increase in levels of triglyceride, LDL and VLDL in GH deficient patients as compared to control. While in other study, a slight decrease in cholesterol level and an increase in HDL level were found in the GH deficient patients as compared to control [42]. A previous investigation [43] reported that children with GHD reveal a tendency towards lipid disturbance. In contrast, another study [44] reported that GHD in childhood is not associated with alterations in lipid profiles compared with healthy controls.

Conclusions

The following can be concluded from the present study:

It can be concluded from the present study that the diagnosis of GHD cannot be done at the basal serum of GH. A high level of GH was detected after 1 hr. provocation with clonidine compared with its value after 1.30 hr. provocation. The IGF-1 is an appropriate parameter to expect GHD in children and adolescences whom GHD was detected by GH stimulation testing. Low GH secretion is associated with a high level of cortisol resulting in GHD. Patients with GHD displayed a tendency towards lipids disturbances. Growth hormone deficiency appear to be prevalent in males and predominant in underweight GH deficient patients.

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