Onset of Puberty in a Sample of Iraqi Adolescents

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Abstract

Background
The age of onset of puberty was estimated in a sample of Iraqi adolescent depending upon the date of the first menstrual cycle (menarche),LH,FSH,E2 in girls, and ejaculation (or wet dreams),FSH,LH,Testo.,in boys,B.M.I was also evaluated for all of them.

Objective
To shade a light on the age of onset of puberty in a sample of Iraqi adolescent.

Setting
The central public health laboratory (C.P.H.L),Clinical chemistry.

Individual methods
130 girls and 100 boys was enrolled in this study for determing their age of onset of puberty, they were all apparently adolescent , FSH,LH,E2,B.M.I for girls, while the parameters for boys adolescent was LH,FSH, Testo., B.M.I

Results
The age mean±SD of onset of puberty was 14.0±0.07 , 15.37±0.03 for girls and boys respectively.

Conclusion
There is an increased precent of late onset of puberty associated with low B.M.I and low sex steroid values, in addition to 6.1% of girls adolescent had elevated LH/FSH ratio.

INTRODUCTION

1.1 Puberty

Normal physiological process during which there is a gradual transition to the more sexual maturity when the children begin to mature biologically, psychologically, socially and cognitively, this process can take 1-6 years during which a child's body takes on those characteristics that define it sexually. Puberty leads to adolescence, girls start to grow into women , boys into men [(1)].

1.1.1 Adolescence

It is a transitional period between childhood and adulthood , its changes don't occur on a strict timeline ; instead these changes occur at different times , it can be a very difficult time. The body are no longer a child , but he is not an adult yet either [(2)].

1.1.2 When dose puberty happen ?

Puberty is an important period of biological changes that our bodies go through it. For girls it can begin between the age of nine to fourteen , while for boys it is around the age of ten to seventeen, and the whole process can last from only one to as long as six years [(3)]. Some time these physical and emotional changes don’t happen at the same time, A girl or boy can possibly mature emotionally before they do physically and the opposite thing could happen , this can be a very difficult time, sometimes kids who start to develop early are not prepared for the changes going on in their bodies, it can make them very conscious and even feel very socially isolated [(4)]. Puberty is considered early(precocious) if it occurs before the age of eight years in girls, and nine years in boys. Puberty is considered late or delayed if it has not begun prior to the age of thirteen for girls and fourteen years for boys [(5)]. The recent studies have shown that puberty is occurring at an increasingly earlier age in developed countries and late in poorer ones [(6)].

1.1.3 What causes all these changes ?

Hormones cause these changes as the brain knows when the body is nearing sexual maturity, there will be different Gonadotropines which are responsible for different changes in the body during puberty [7].

As the children get nearer to puberty, the brain and pituitary gland release Gonadotrpin hormones due to diminished sensitivity of the pituitary gland or hypothalamous or both to the negative feed back effect of the sex steroid (with unclear mechanism) and as puberty approaches, nocturnal secretion of gonadal steriods rise gradually over several years before stabilizing into adult level when full sexual maturity is reached. So at the onset of puberty gonadotropines secretion increases, as it does in male, ovarian estrogen secretion rises and stimulate the development of female secondary sex characteristics and the onset of the menstruation (menarche) [8]. In male as puberty approaches the pulse amplitude and frequency of LH secretion in particular increase, initially , this occurs during the sleep but later continues throughout the day, leydig cell function and testo. secretion increase so stimulates the development of secondary male characteristics, Gonadotropin secretion also stimulates meiosis of previously dormant germ cells in the seminiferous tubules and so the production of sperm [9].

1.2 Endocrine function

Reproductive endocrinology encompasses the hormones of the hypothalamic -pituitary - adrenal axis as well as the adrenal gland , these hormones are crucial for proper reproductive, function; Gn RH, LH, FSH, androgens, estrogens which are synthesized by ovaries , testes, adrenal gland and are responsible for manifestation of primary and secondary sex characteristics . Steriods that feminize are classified as estrogens ; those that musculinize are known as androgens [10].

1.2.1 LH

A glycoprotein which is formed in the anterior lobe of the pituitary , its biological half life in the circulation is 20-25 minutes, it consist of two polypeptide chains (Alpha and Beta subunits) the latter determines the specific biological action and the immunological behavior of the hormone , the Alpha sub units is identical to the Alpha subunits of other hormones glycoprotien. The primary action of LH is the regulation of the gonadal function, it controls the synthesis of the sex steroids; in women the synthesis of the progesterone in the ovaries and in men that of testo. In the testes together with FSH it is involved in those mechanisms which trigger ovulation, its synthesis and release from pituitary gland are stimulated by (LH-RH) formed in the hypothalamous, sex steroid regulate its secretion by mean of a multiple feed back mechanism (Negative-inhibition and positive stimulation) [11].

1.2.2 FSH.

A glycoprotien formed in the anterior lobe of the pituitary. Its biological half life in the circulation is 10-15 minutes. It consist of two polypeptide chains Alpha, Beta subunits , the latter determines the specific biological action and the immunological behavior of the hormone, the Alpha subunite is identical to the Alpha subunits of the human glycoprotien hormones such as LH. The primary action of FSH is regulation of gonadal function, together with LH they controls the synthesis of the ovum in women, its synthesis in the pituitary and their releases are stimulated by LH-RH formed in the hypothalamous, sex steroids regulate its secretion by mean of multiple feedback mechanism (negative -inhibition and positive stimulation), sertoli cells secrete inhibin, aglyco protein that inhibits the pituitary secretion of FSH [(12)].

1.2.3 Testosterone.

It is a steriod hormon mainly synthesized by the leydig cells of the testes in male and by ovaries and adrenal cortex in female, its production in both sexes is regulated by LH, in men testo. circulates bound 44-65% to SHBG with very high affinity , 33-50 % bound to albumin, only 2-3% circulates freely in plasma. In women testo is bound 66-78% to SHBG, 20-30% to albumin , and 1% free. At the time of puberty, pituitary gonadotropines increase stimulating testicular maturation, LH acts directly on the interstitial mesenchimal elements , causing them to secret testo. and estrogen , as they are developing into mature leydig cells. FSH acts on the seminiferous tubules to induce and maintaine normal spermatogenesis, at the onset of puberty testo. stimulate the development of male sex characteristics (external genetalia, accessory sex organ, hair growth, linear growth, voice timbre, psyche, muscle and bone tissue mass) which is maintained throughout life. The age at which maximum testo. levels are observed varies from 15-20 to 25 years. Testo. measurement is not a lone sufficient in the assessment of gonadal function, in male it must be supplemented at least with careful clinical examination of the patient, evaluation of the ejaculate as well as determination of gonadotropins [(13)].

1.2.4 Estrogens

They are sex hormone responsible for development and maintenance of the female sex organs and secondary sex characteristics. In conjunction with progesterone they participate in the regulation of the menstrual cycle, breast , uterine growth and in the maintenance of pregnancy, they affect calcium homeostasis and have a beneficial effect on bone mass, decreasing bone resorption, and in prepubertal girls they accelerate linear bone growth and result in epiphyseal closure , they increase the level of sex hormone binding globulin, cortisol binding globulin, thyroid binding globulin. Plasma protiens that are bound to copper and iron also are elevated in response to estrogen as those of high density and very high density lipoproteins. In normal women most of estrogens secreted by ovarian follicles and the corpus luteum, and during pregnancy by the placenta, adrenals and testes are also believed to secrete minute quantities of estrogens . The most potent estrogen secreted by the ovary is 17-estradiol (E2) and because it is derived almost exclusively from the ovaries, its measurement is often considered sufficient to evaluate ovarian function, liver is the primary site for the inactivation or metabolism of the estrogen [(14)].

1.3. Stages of puberty

Puberty normally occurs in a series of five stages , according to the Tanner stages for growth and development that typically begin within the ages of 8 and 13 years in girls and 9 and 14 years for boys [(15)].

1.3.1 Girls (Tab 1.1).

The first sign of puberty in girls, which occurs at an average age 10.5 years is breast development (thelarche), this begins with breast budding, or the formation of small lump or nodules under one or both nipples, these lumps may be tender and they may be of different sizes at first, this is usually also the begining of their growth purt, next in about six months, pubic hair develops (adrenarche), although in some children, pubic hair is the first sign of puberty, and then axillary hair begins to grow . Over the next few years, breast size will continue to increase and there will be a progressive increase in development of pubic hair and the external genitalia, leading to the first period of menstruation (Menarche) which occurs at average age of 12 .5 - 13 year, and usually occurs about two years after puberty begins and coincides with their peak in height velocity . Development continues and the whole process is completed in 3-4 years, eventually reaching adult breast and areolar size and an adult pattern of pubic hair. A child will have also reached the final adult height about two years after menarche [(16)].

Table 1.1: The stages of development of puberty in girls [16]

Sexual maturity rating	Average age (years)	Features	What happens
1	9.5	Growth, breasts and pubic hair	Height spurt begins, body fat at 15-17%, Breasts are prepubertal; no glandular tissue, no pubic hair
2	11.5	Breasts	The areola enlarges and becomes darker it become amound (raises) with a small amount of breast tissue underneath (bud).
2	11 .75	Pubic hair and growth	A few long, downy, slightly darkened hairs appear along the labia magora, at the end of this stage , the body fat has increased to 18.9%.
3	11 .75	Growth	Peak height velocity (maximum growth rate) is reached. Body fat is now21.6%
3	12	Breast	Development of breast tissue past the edge of the areola.
3	12 .5	Pubic hair	Moderate amount of more curly, pigmented, and coarser hair on the mons pubis (the raised fatty area around labix magora). Hairs begin to spread more laterally. Menarche occurs in 20% of girls during this stage.
4	12 .75	Pubic hair	Hair is close to adult pubic hair in curliness and coarseness. Area of pubis covered is smaller than adult , and there are no hair on the middle surfaces of the thigh. Menarche occurs in 50% of girls.
4	13	Breast	Continued development of breast tissue; inside view, areeole and nipple protrude.
4	13.5	Growth	End of growth spurt, body fat reaches mature proportion 26%. Girls grow at most 3-4 inches
5	14 .5	Pubic hair body fat	Body fat 26.7%, longe pigmented hairs to grow on the inner thigh (addult)
5	15.25	Breasts	Adult breasts

1.3.2 Boys (Table 1.2)

puberty generally begins later in boys ; at an average age of 11.5-12 years. The first sign of puberty in boys is an increase in size of testicles. This is followed a few months later by the growth of pubic hair. Puberty continue with an increase in size of the testicles and penis and continued growth of pubic and axillary hair . Boys undergo their peak growth spurt about 2-3 years than girls. Also, this usually begins with an enlargement of the hand and feet and is latter followed by growth in the arms, legs , trunk and chest. Other changes include a deepening of the voice , an increase in muscle mass, the ability to get erection and ejaculate (especially spontanous nocturnal emissions or wet dreams), and in some boys, breast development Ggynecomastia. Development continues and the whole process is completed in 3-4 years, eventually reaching adult testicle and penis size and an adult pattern of pubic hair, this is followed by the development of chest and facial hair. Puberty is also associated with adolescents beginning to have axillary perspiration and body odor and acne [17].

Table : 1.2 The stages of development of puberty in Boys [17]

Age year	Features	What Happens
11-14	Growth of Hair and pubic hair	Hair usually begins to grow on various parts of the body when a boy is between 11-14 years. Hair can continue to spread to other parts of the body until a young man reaches about age of 20 years. Sparse of slightly pigmented pubic hair at the base of the penis around age 12.4 adult type with spread to the inside of the thighs but not up the abdomen (15-3 years) hair is typically seens on a young mans face, axillary area pubic area, abdomen ,chest , arms,leg and buttocks. The amount and distribution of hairs can vary considerably from one man to the next, this is entirly normal and may have genetic tendencies.
11-15	Voice changes	As a result of increased testo. Vocal cords become longer and thicker and the voice become lower, while these changes occurring it is not unusual for the voice to change pitch abruptly or crack at times (this can often be very embarrassing). Average age around 13.5 years with voice changing around 14.5 years.
13-16	External Genital development	Increased growth of the peins and scrotum often start at about age of 13 years and continues until adult size reached about 2 years later. Keep in mind there is a faire degree of variation in the age for genital development from one boy to the next.
11-20	Oil gland	It become active or over active
10-17	Growth	Growth spurt and the appearance of a more muscular and angular shape
12-18	Start in infancy related to puberty	Penile erection , but ejaculation is only for puberty on ward.

1.4 Timing for puberty (Table 1.3).

In most Asian and African countries menarche is later than in the western world. The timing of puberty in south America, Asia , Africa differs from the timing in western Europe or united state. The avarage of menarche age in united state is 12.8 years [18]. Data on the onset of puberity in Europe showed a gradual decrease in age of menarche in the last few decades untile 1990 where in some Europian countries no further changes was observed, in the Netherlands, the 1997 growth study didn't show a further significant changes toward earlier occurrence of puberty , in the last decade the influence of environmental factors on the timing of puberty in healthy Dutch children seems minimal, as no further decrease in the age of onset of puberty [19].

Table : 1.3 Reported age of menarche from different counteries [18,19]

Economy classification	Country	Mean age of menarche (years)
Low income	India (urban)	13.4
	India (rural)	14
	India (high SES)	13.2
	India (low SES)	14.6
Low income	Srilanka (Tamil)	13.8
Low income	Thialand (urban)	12.4
Low income	Bangladesh	13
Low income	Nepal (high altitude)	16.2
Low income	Senegal	16.1
Low income	Ghana	14
Low income	China	13.7
Low income	Nigeria (urban)	13.5
	Nigeria (Mixed)	14
Middle income (lower part)	Bolivia (all SES)	12.7
Middle income (upper part)	Argentina (urban)	12.5
High income	Hong kong	12.4
High income	The Nether lands	
	1980	13.3
	1997	13.1

1999 data ; according to the world Bank Group

1.4.1 Factors affect timing of puberty

1. Genetic influence
2. Environmental condition

a. Socioeconomic state (SES)

b. Health care facilities

c. Nutrition

Although a genetic influence was assumed in children as a good indicator for timing of puberty, environmental conditions should be viewed as the principal cause of earlier maturation [(20)]. The inverse correlation between improvement of SES, health facilities, and nutrition and the age of menarche in female or ejaculation age in male respectively was shown in several European countries [(21)]. Data on adolescent with very low B.M.I and a menorrhea show the importance of body composition in the onset and progression of puberty. Some studies from Dutch sample showes that taller and heavier adolescent had earlear menarche or ejaculation compared to smaller and thinner one, thus children who suffer from malnutrition may have delayed onset of puberty [(22)]. In general it is a consistent finding within countries that adolescent from higher social classes have their age of onset earlear than those from lower classes [(23)].

Mothers from lower social classes are at risk of unfavorable circumstances in utero and the long term influence of intra uterine growth retardation on several endocrinale axes has been described, and this could altered the hypothalamic control of LH release, it can therefore be assumed that programming of the setting of the GnRH pulse generator is influenced by prenatal factors [(24)].

1.4 .2 Female Puberty

In girls puberty is associated with rises in estrogen secretion by the ovary in response to gonadotrpin levels that increase in response to GnRH, estrogen secretion by the ovary increases and causes enlargement of the uterous and breasts, in the breast it will enhances growth of ducts, progesterone augment this effect. Puberty appear to begin with a diminished sensitivity of the pituitary gland or hypothalamous or both to the negative feedback effect of the sex steroids, As puberty reaches nocturnal secretion of gonadotropins occurs . The value for LH, FSH and gonadal steroids rise gradually over several years before stabilizing at adult levels, when full sexual maturity reached [(25)].

1.4.3 Male Puberty

The concentration of androstrenedione , DHEA and DHEA-S begin to increase as early as six or seven year of age several years before maturation of the HPA-axis, the onset of puberty is associated with a nocturnal surges in LH and to a Lesser extent FSH secretion, the overall changes associated with puberty reflect the theory that the HP-system becomes less sensitive to feedback inhibition by circulating androgen resulting in higher androgen concentration [26].

1.5 Iraqi studies

As far as we know no Iraqi study was reported with such work but only one study was prepaired concentrated upon aneamia among school adolescents in the city of Baghdad by Dr. AL-Sharbatti in which she pointed indirectly to menarche which was to be around 13 years [27].

SUBJECTS AND METHOD

Subjects

A total of 230 adolescents, 130 girls age 13-15 years, 100 boys age 14-16 years, all are apparently healthy and was rundomly selected from Baghdad city, they weren't complained of any diseases and with no history of drug intake. Girls and boys were each subdivided into five age groups each with its own mean value ± SD. B.M.1, LH, FSH in addition to E_2, Testo for girls and boys respectively.

Clinical staging

Routine evaluation of the participant included history taking to search for girls menarche and boys ejaculation (or wet dream). Next was physical examination of the secondary sexual characteristics for assessment of pubertal state of development and including only stage four and five in the study for simplicity [1].

B.M.I

The height in Cm (to the nearest 0.5 cm) and the weight in kg (to the nearest 0.5 kg) were measured and then the B.M.I was calculated for every adolescent from the following equation;
B.M.1 = body weight (Kg)**/**body height (m^2)

Methods

Blood sample.

A bout five ml of venous blood were collected from each adolescent which were centrifuged at 4000 rpm for 10 minutes, and serum was separated, then kept in a new sterile tube and stored at -20 ^oC until time of hormones estimation which were determined at the central public health laboratory (C.P.H.L).

For the girls blood sample was aspirated during early follicular phase of the cycle for LH, FSH estimation, and then another sample was aspirated at midluteal phase for E_2 estimation. Also LH/FSH ratio was estimated for girls adolescent, and those with a ratio above two were sent for checking to ultrasonography to asses polycystic changes of ovaries cases which were evaluated by another blood sample for testesteron estimation, in those with poly systic ovaries (PCO) the hormonal evaluation for boys adolescents included LH, FSH, Testo. hormones.

Hormones estimation

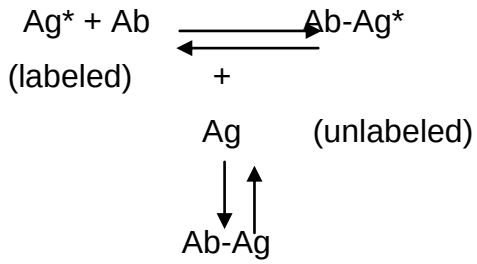
The main principle for hormonal assay used in this study is the one-step-sandwich method in which complex of hormones in the sample and (^{125}I) labelled antihormones antibodies is formed during this proccess, and at the end of the reaction the amount of the free tracer is removed by decantation and subsequent washing. The amount of the tracer specifically bound to the coated tubes is measured with a gammascintillation counter.

Preparation of the reagents

Kit components were brought up to room temperature, the standards and the control serum were dissolved in 500 µl twice distilled water , the washing buffer was prepared by dissolving three buffer tablets in 300 ml distilled water (supplied by CIS bio international company).

Principle of radioimmuno assay (RIA)

It is specific, sensitive , and simple method. The principle is



In the absence of unlabelled (Ag) a certain amount of labeled(Ag*)
Will bind to the bantibodies (Ab) according to the law of mass action.

In prescene of unlabeled Ag , the amount of Ab-Ag* will decrease because the Ag will compete with it for the binding site on Ab. The amount of labeled (Ag*) displaced from the Ab is proportional to amount of Ag present in system . This method can detect levels of nanogram or picogram. Compound to be measured by RIA are many as peptide hormones,steriods Drugs (digoxin,morphine),and miscellaneous (prosto glandin , CAMP , B12 , mRNA,T3 , T4) . The standard used must be immunologically identical to substance measured, this is checked by plotting dose-response curve for both standard and the unknown on semilog paper if the two curves are parallel to the standard and the compound measured are immunologically identical. Usually RIA used different concentration of standard to plot different curves [28].

RESULTS

Table 3.1 illustrate the main parameters related to the girls, it included the mean value ± SD for 130 adolescent girls. Their age of onset of puberty 14 ± 0.07 year, B.M.I, 18.48 ± 1.22 (kg/m^2). While the mean ± SD for midluteal E_2 , early follicular LH and FSH where found to be 173.78 ± 15.52 pg/ml, 7.35 ± 1.64 mlU/ml, 5.73 ± 1.25 mlU/ml respectively. In the same table girls were classified into five groups, each has its own mean value ± SD of the B.M.1, E2, LH, FSH.

Table 3.2 illustrate the main parameters related to the boys adolescents just like that for girls where boys was divided into five groups with 14,14.5,15,15.5 , and 16 years , each with its own B.M.I, and FSH , LH, and Testo hormones. This table also included the mean ± SD for age of onset of puberty, B.M.I , L.H, FSH, Testo.

Table 3.3 illustrated the strongest negative Correlation (r) between the menarche and E_2 (-0.964) with a significant P. value 0.008, then next comes BMI, FSH, LH in order of frequency. For boys Table 3.4 does not show such significant correlation between the onset and other parameters.

Although Table 3.1 showed high E2, BMI, LH, FSH at an earlier onset yet ANOVA test did not find any significant difference between these groups which should be at the 0.05 level as in Table 3.5. And also the same things for boys as in Table 3.6.

T. test was done to find if there is any significant difference between boys and girls, Table 3.7 showed that there was a significant difference in sex steroids , BMI and the onset of puberty between both sexes i.e. the higher the BMI the earlier age of onset.

Table : 3.1 Hormone Profile , BMI and Onset of puberty in Girls.

Girls			B.M.I Kg/m^2 ± SD	E_2 (pg/ml) ± SD	LH (mIU/ml) ± SD	FSH (mIU/ml) ± SD
Onset (year)	Number	Percent (%)				
13.0	31	23.846	19.65 ± 1.02	191.1 ± 16.1	10.4 ± 1.5	8 ± 1.21
13.5	20	15.384	19.77 ± 1.09	188.4 ± 14.3	7.27± 1.7	5.62±1.23
14.0	36	27.692	18.72 ± 1.1	172.6 ± 15.4	6.95 ± 1.4	5.77 ± 1.31
14.5	18	13.846	17.8 ± 1.4	168.8 ± 15.1	6.7 ± 1.09	5 ± 1.45
15.0	25	19.23	16.49 ± 1.3	148 ± 14.22	5.44 ± 1.62	4.27 ± 0.99
Mean age of onset ± SD year			Mean B.M.I ± SD	Mean E_2 ± SD	Mean LH ± SD	Mean FSH ± SD
14.0 ± 0.07			18.48 ± 1.22	173.78 ±15.52	7.35 ± 1.64	5.73 ± 1.25

Table : 3.2 Hormone Profile , BMI and Onset of puberty in Boys.

Boys			B.M.I (kg/m^2) ± SD	Testo. (nmo/L) ± SD	LH (mIU/ml) ± SD	FSH (mIU/ml) ± SD
Onset (year)	Number	Percent (%)				
14	12	12%	22.42 ± 1.09	21 ± 2.1	5.65 ± 0.9	4.7 ± 0.5
14.5	15	15%	19.37 ± 1.12	18.8 ± 2.6	6.2 ± 0.78	4.2 ± 0.39
15	26	26%	21.64 ± 1.03	21.68 ± 2.4	4.15 ± 0.81	3.45 ± 0.61
15.5	18	18%	21.83 ± 1.08	18.93 ± 2.5	4.32 ± 0.82	3.99 ± 0.4
16	29	29%	20.55 ± 1.2	14.42 ± 2.5	4.17 ± 0.84	3.72 ± 0.43
Mean age of onset ± SD year			Mean B.M.I ± SD	Mean Testo. ± SD	Mean LH ± SD	Mean FSH ± SD

15.37 ± 0.03	21.16 ± 1.08	18.96 ± 2.53	4.89 ± 0.85	4.0 ± 0.42

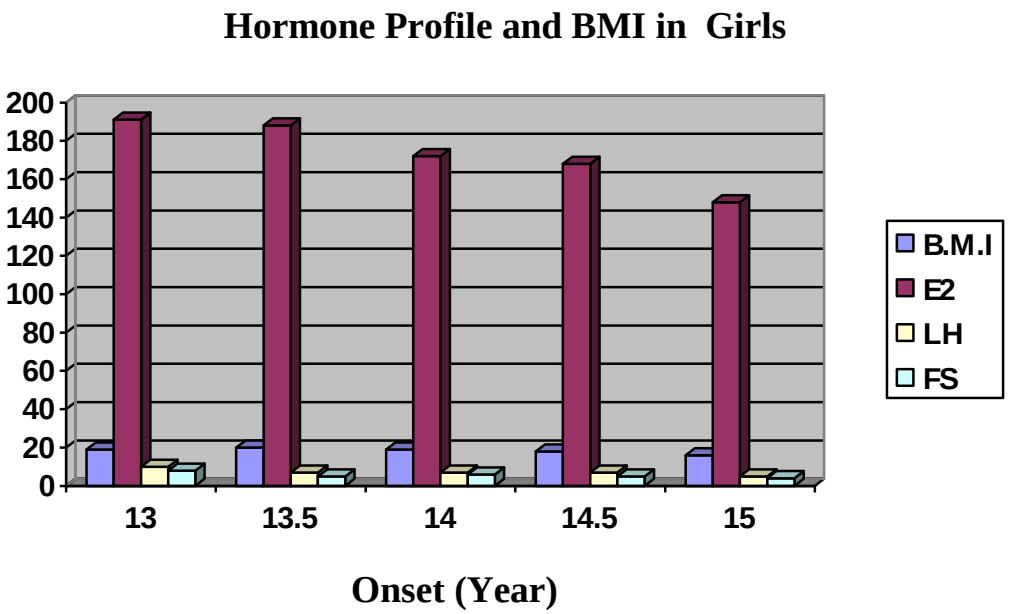


Fig (1): Histogram view of girls results

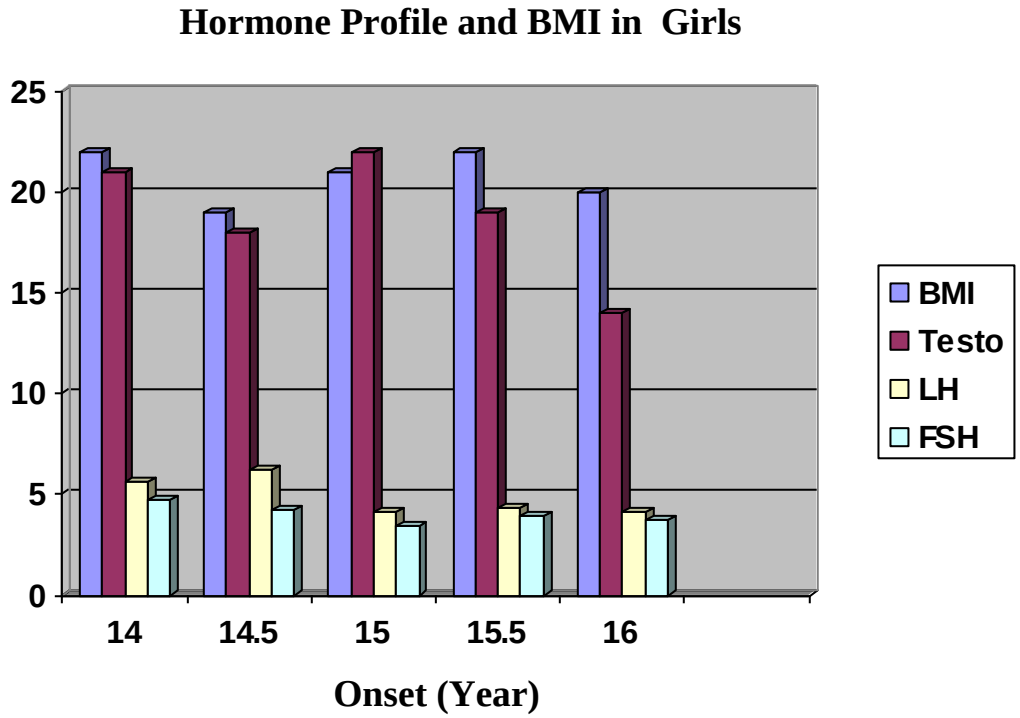


Fig (2): Histogram view of boys results

Table 3.3: Correlations between BMI, E_2, LH, FSH and Menarche.

Parameters		Onset
BMI	r	- 0.956 **
	P. value	0.011
E2	r	- 0.964**
	P. value	0.008
LH	r	- 0.902*
	P. value	0.037
FSH	r	- 0.913*
	P. value	0.030

* Correlation is significant at the 0.05 level.
** Correlation is significant at the 0.01 level.

Table 3.4: Correlation between BMI, Testo., LH., FSH and onest of puberty.

Parameters		Onset
BMI	r	- 0.167
	P. value	0.788
E2	r	- 0.726
	P. value	0.165
LH	r	- 0.797
	P. value	0.106
FSH	r	- 0.719
	P. value	0.171

* correlation is significant at the 0.05 level .

** correlation is significant at the 0.01 level.

Table 3.5: Girls ANOVA test

	Sum of squares	df	Mean square	F	Sig.
Between Groups	458.746	4	14.687	.017	.999
within Groups	101204.5	15	6746.969		
total	101663.27	19			

Table 3.6: Boys ANOVA test

	Sum of squares	df	Mean square	F	Sig.
Between Groups	136.400	4	34.100	.445	.774
within Groups	1148.276	15	76.552		
total	1284.676	19			

Table 3.7: t-test for equality of means

	t	df	Sig. (2-taild)
AGEGB	-2.000	8.000	.081
BMIGB	-3.275	7.877	.012
LHGB	2.644	6.027	.038
FSHGB	2.602	4.918	.049

G. Girls
B. Boys

4. DISCUSSION

The Present study represents the timing for age of onset of puberty in an apparantly healthy 130 girls, and 100 boys whom are rundomly selected from Baghdad city. In this study girls are categorized as Tanner stage four and five with exclusion of stage one, two and three from enrolling in this study for purpous of simplicity and to minimize misclassification , which will presents great problem. The result were compared to other data from adolescents of different other counteries. B.M.I was included in this study which was potentially important parameter.

Depending upon the first menstrual cycle (Menarche) for girls and ejaculation (or wet dream's) for boys Beside clinical examination and hormonal estimation, Iraqi adolescents in this study shows some whate a later onset of puberty with a mean age of , 14 ± 0.07 , 15.37 for girls and boys respectively , while European data on the onset of puberty showed a gradual decreases in the age of menarche in the last few decades until 1990 where in some of these countries no further decrease was observed, and the reasons for these changes are not apparent but nutritional improvement, increasing obesity, physical activity could play a role far and even could be more than the genetic factor that is to say that environmental factor can change in some way or another the genetic factor in this condition [16]. Table 1.3 explained the relationship between the SES of the countries and the age of onset of their adolescents where high SES means decreasing the onset and vice versa. The results in this study showed that more than 60% of girls had menarche above 14 year and that 90% of Boys were ejaculated first at 15 year and above and this could explained the low SES that Iraqi population were used to live in especially in the last wars and the previous embargo. In general it is a consisten finding within countries that girls from higher social classes have their menarche earlier than their mates from lower classes, and that those adolescents with very low B.M.I and later menarche showed the importance of body composition in the onset and progression of the puberty. [17,18]. And in this study the adolescents in general had low B.M.I , and even with low mean heamoglobin value 11.2 ± 1.1 12.93 ± 0.7 , for boys and girls respectively , that will reflect the malnutrition , low SES, bad health care they live in , which might explained the late onset of puberty in them. In Table 3.1 girls with low B.M.I have a later onset of puberty than those with a higher B.M.I. The hormonal study for adolescent girls illustrated that those with a higher values had a chance to get earlear age of onset of puberty than others, more over those with an earlear age of onset had also a higher B.M.I this could explained the better SES the earlear age of onset and that

hormone is a protien substance where good nutrition is very important here and also during intrauterine life for improvement of the HPG-Axis [3]. On the other hand Engelbergt et al [35] studied the role of early undernutrition pre or posnatally on pubertal development; early malnutrition resulted in delayed onset of puberty in intrauterine -growth - retarded male and female rats, as well as in male food restricted rats, in female food restricted rats the onset of puberty was normal. This study and other data suggest that there may be an environmental factors in the pre or postnatal period having long-term effect on the HP-action.

4.1 LH/FSH Ratio and the Pcos probability

Another important point had been noticed during this study that there was an increased levels of LH for some adolescent girls and the LH/FSH ratio was more than two. Eight girls which constitute 6.15% from all girls have had a ratio more than two , those are 2.8,2.1,2.3,2.4 ,2.3, 2.1 , 2.2. such cases were examined by abdominal sonar to proved that their ovaries had polycystic changes.

Conclusion

There is an increased percent of late onset puberty in our adolescents in comparison to other countries. Most of the adolescents in this study have a low B.M.I which were found to be associated with the onset, that is to say low B.M.I mean a late puberty and vice versa. also it seems that those adolescents with a later onset had a low sex steroid level. in comparison between groups of girls it was found that those with early onset had highest sex steroid and BMI, while in boys such finding was not so much clear. an important 6.1 % of adolescents girls had elevated LH/FSH ratio which should attract attention for further study.

SUGGESTION

Further study to evaluate the effect of Nutritional state on the onset .

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