

A FOLLOW-UP STUDY OF PERIPUBERTAL OBESE GIRLS

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Abstract: *between 1977 and 1984 5236 new gynecological patients were examined for different medical problems at the Pediatric Gynecological Ward of the Postgraduate Medical University, Budapest, Hungary. The medical examination is always recorded by a so-called "Functional Somatometry" which contains 20 body measurements including 8 skinfolds and 10 calculated indices or equations. Among these patients there were 378 girls (7.21%) as obese and 132 girls (2.52%) as overweight. These former patients were requested for control, but the follow-up was conducted on 136 adults only (26.66%).*

Analysing their actual gynecological state together with the actual weight and final height, it is documented that 29 women gained the ideal body mass for height (21.3%), the number of actual overweight is 33 persons (24.2%). Among those, who were or became obese 63 women (46.32%) had an extreme obesity, around 80–100 kgs. We found that 54% of former obese girls remained obese, while 36% of former overweight girls changed to obese adults. This is a bad prognosis for pubertal girls being obese or overweight regarding their adult gynecological life.

Key words: *Puberty; Obesity; Menstrual disturbances.*

Introduction

The nature of the basic abnormality in the development of obesity is not yet known. This condition may be defined as: (1) an increase in fat cell number (hyperplastic obesity); (2) an abnormal growth of the adipose tissue due to an enlargement in fat cell size (hypertrophic obesity), and (3) a combination of both.

Obesity means a disturbed control of energy in the storage and in the expenditure of it. Many children grow fat as a result of genetic influence, the parents of the affected children are in most of the cases obese as well. In most recent studies (Price et al. 1990a, 1990b, Eckel 1989, Rajput-Williams et al. 1988, Rosen et al. 1989, Sorensen et al. 1989, Zonta et al. 1987) a concept has been documented for the mode of inheritance of obesity, including moderate polygenic inheritance (34% of variance resulting from many genes with small effects) and common regressively expressed major genes (21% of frequency for a few gene with large effects). The mechanism is strongly related to a lower metabolic rate, which has been proven to be strictly familial (Roberts et al. 1988, Ravussin et al. 1988). The individual genetic background with the possible role of enzyme genetic polymorphism – besides cultural, nutritional and geographic factors – can influence the clinical variability of obesity. Therefore it is extremely difficult to separate the genetic element from environmental influences such as eating habits in the families that lack of physical activity. Our interest is to prevent obesity – if it is possible – from early childhood and the pediatric gynecology is an excellent field of studying this problem. The obese patients generally suffer from genital inflammations or menstrual disturbances and we have the opportunity to carry our prophylactic care of obesity together with the given medical problem. The aim of this follow-up study was to recognize the results of our former efforts.

Material and Methods

The pediatric gynecologist must be complemented with some methods of somatometry, given that the growth and development are incorporated significantly in these periods of a girl's life, especially during the prepubertal–pubertal process, when a completely new hormonal system starts to work. It is inevitable to determine first the biological age of an 11 year old girls, who can be sexually immature (in the hormonally quiescent period) but also in the postmenarche, after the first menstruation. The practising pediatric gynecologists are facing the actual problems of genital diseases at the same time with those of the sexual maturation. Therefore we use a so-called "Functional Somatometry" (Örley 1984) which is an applied form of somatometry with a special aim of a diagnostic tool. It contains 20 body measurements, including 8 skinfolds. This measuring method has been a constant part of the author's medical work since 1977. Every girl at every medical examination is being measured by this method. All measurements are made on the left side of the body, except the skinfolds, which are recommended to be bilaterally symmetrically measured at the trunk, since 1986. In the same year we do parallel measurements on the skinfolds both by the Lange skinfold caliper and by ultrasonographic technique, using SCANNER 700 real-time ultrasound imager. During the observation period (1986–1987) a correction scale was prepared (by Z. Zachár, mathematician for those who can work with the caliper only to recombine their results to the skinfold amount measured by ultrasonographic technique (*Table 1* Zachár Z. 1987). The functional somatometry deals with calculated indices which are on the *Table 2*. These simple calculations serve to the interpretation of somatometric data.

Table 1. Correction to ultrasonographically measured skinfolds (elaborated by Z. Zachár, 1987)

| | | | |
|-------------------|---|--------------------------|---------|
| <i>Right side</i> | | | |
| U–sc | = | 1.72143 + 0.312509 xC–sc | C: 0.89 |
| U–sc | = | 0.42165 xC–sc | |
| U–si | = | 0.34792 + 0.47382 xC–si | C: 0.94 |
| U–si | = | 0.49557 xC–si | |
| U–sp | = | 0.33836 + 0.45368 xC–sp | C: 0.96 |
| U–sp | = | 0.37695 xC–sp | |
| U–u | = | 0.67484 + 0.42908 xC–u | C: 0.90 |
| U–u | = | 0.460294 xC–c | |
| <i>Left side</i> | | | |
| U–sc | = | 1.83371 + 0.305449 xC–sc | C: 0.91 |
| U–sc | = | 0.42116 xC–sc | |
| U–si | = | 0.36388 + 0.4569 xC–si | C: 0.93 |
| U–si | = | 0.48028 xC–si | |
| U–sp | = | 0.1338 + 0.46407 xC–sp | C: 0.95 |
| U–sp | = | 0.473199 xC–sp | |
| U–u | = | 0.20906 + 0.39277 xC–u | C: 0.88 |
| U–u | = | 0.44779 xC–c | |

Table 2. The calculated indices to the functional somatometry

| | |
|---|---|
| <p>1. index of Kaup = $\frac{\text{weight (g)}}{\text{height (cm)}^2}$</p> | <p>Index of Kaup = $\frac{\text{height (cm)}}{\sqrt[3]{\text{weight (kg)}}}$</p> |
| <p>2. Fullness index of Rohrer = $\frac{\text{weight (g)} \times 100}{\text{height (cm)}^3}$</p> | |
| <p>3. Robusticity index of Pignet-Vervach = $\frac{\text{weight (kg)} + \text{chest circumference (cm)}}{\text{height (cm)}}$</p> | |
| <p>4. Density index of Dumin-Rahaman (1967): $D = 1.1369 - 0.0598 \times \text{Log (BI + TR + SC + SI skinfolds)}$</p> | |
| <p>5. Body Fat Percentage of Siri (1956): $\text{BF\%} = \left(\frac{4.95}{D} - 4.5 \right) \times 100$</p> | |
| <p>6. Lean Body Mass = (weight - body fat percentage)</p> | |
| <p>7. Body Surface Area according to Body's equation $\text{BSAm}^2 = \text{weight (g)}^{0.7285} - 0.0118 \log \text{Wt} \times \text{height (cm)}^{0.03} \times 3.207 \times 10^{-4}$</p> | |
| <p>8. Total Body Water according to Mellits and Cheek's equation $\text{TBW} = -10.313 + 0.154 \text{ height (cm)} + 0.252 \text{ weight (kg)}$</p> | |
| <p>9. Fat Body Mass according to Dugdale and Griffith's equation $\text{FBM} = 6.629 + 0.645 \text{ Wt} - 0.144 \text{ Ht} - 0.118 \text{ T} + 0.035 \text{ Sc} + 0.206 \text{ B} + 0.059 \text{ Si}$</p> | |
| <p>10. Fat Body Mass calculated to the body surface area after Dugdale and Griffith's equation, multiplying the skinfolds with the body surface area</p> | |

Since 1977 we possess an always growing cataster for "Functional Somatometry" because we based our medical practice on this method. We meet yearly approximately 1000 new patients but get about 3000 repeated measurements and calculated indices, equations. Our data are linked to medical examinations. Between 1977 and 1991 we had measured 11 049 new patients and repeated measurements and calculations were made on 34 706 times. The data are recorded with computer system.

The basic reference data is prepared for each age from healthy 50 girls' data, who were all our patients and therefore their complete medical analysis is at hand. Their measurements data are correctly depicted by graphical technique according to the mean values at each measurement, with equal diminution. The sexual development in girls starts around the age of 10 year in Hungary, therefore the sample of healthy girls should be divided into 5 different groups, according to the Tanner definition. The different body shapes show us clearly, who had their first menses and who have not had as yet.

We also selected our patients' data by weight in each developmental phase (according to Tanner's score). Thus a developmental Atlas for lean, average size and obese girls has been prepared for own use, by the simultaneous use of indices (Kaup + Hirata) and calculations e.g. Body Fat percentage, Total Body Water, Lean Body Mass etc. It represents for us the validity of the criteria of the indices used, interpreting overweight or obesity in prepubertal-pubertal girls (Table 3).

*Table 3. Development of girls according to Tanner's score and body composition (lean, normal, obese)
(Örley, 1990)*

| | Tanner 2 | | | Tanner 3 | | | Tanner 4 | | | Tanner 5 | | |
|-------------------------|----------|--------|--------|----------|--------|-------|----------|--------|-------|----------|--------|-------|
| | Lean | Normal | Obese | Lean | Normal | Obese | Lean | Normal | Obese | Lean | Normal | Obese |
| <i>Age (year)</i> | 11.02 | 10.3 | 10.73 | 11.76 | 11.55 | 11.52 | 12.89 | 12.05 | 12.9 | 12.92 | 12.33 | 12.79 |
| <i>Weight (kg)</i> | 31.16 | 35.81 | 46.57 | 36.35 | 41.12 | 53.15 | 40.6 | 44.7 | 57.63 | 42.69 | 48.11 | 59.66 |
| <i>Height (cm)</i> | 144.2 | 144.7 | 148.09 | 152.4 | 150.1 | 163.8 | 155.5 | 154.5 | 158.8 | 161.1 | 155.7 | 158.1 |
| <i>BF%</i> | 23.82 | 28.58 | 31.26 | 24.57 | 28.27 | 31.8 | 25.25 | 28.78 | 32.39 | 25.86 | 29.2 | 31.24 |
| <i>LBM</i> | 23.73 | 25.75 | 32.01 | 27.36 | 29.49 | 36.24 | 30.34 | 34.39 | 38.96 | 31.22 | 34.06 | 41.02 |
| <i>Total Body Water</i> | 19.75 | 21.00 | 24.23 | 22.33 | 23.18 | 28.31 | 23.88 | 24.74 | 28.67 | 25.26 | 25.80 | 29.07 |
| <i>Kaup index</i> | 1.49 | 1.7 | 2.12 | 1.56 | 1.82 | 1.98 | 1.67 | 1.87 | 2.22 | 1.64 | 1.98 | 2.38 |
| <i>Hirata index</i> | 45.83 | 43.9 | 41.16 | 46.03 | 43.51 | 43.57 | 45.26 | 43.53 | 41.11 | 46.10 | 42.82 | 40.46 |
| | N=100 | N=96 | N=80 | N=100 | N=100 | N=82 | N=100 | N=100 | N=80 | N=100 | N=100 | N=90 |

For those patients who are endocrinologically not healthy and are suffering from different menstrual disturbances a similar classification and an atlas has been prepared, according to the age category for lean, normally weighted or obese girls. We possess an Atlas with 125 pages for all age and for all genetic or endocrine problems in pediatric gynecology.

Gynecological problems of obesity in peripubertal age. Former studies and observations

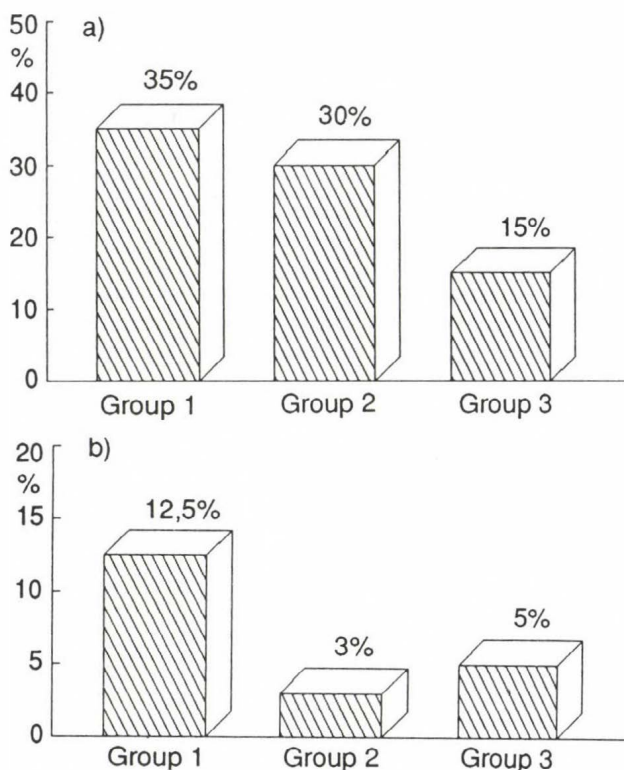
The obese girl is more frequently seen for genital inflammation than normally weighted healthy peer. Therefore, we have serially followed up 113 obese girls, who were quarterly controlled and measured by the functional somatometry. Cases of pathological obesity (e.g. Prader-Willi, Laurence Moon Biedl syndrome or Cushing disease) were excluded from the forthcoming studies. In this sample our heaviest patient weighted 124 kgs (1277.53 pounds) at the age of 14. Their hormone results pointed to an altered LH/FSH ratio in prepubertal-pubertal process (Örley et al. 1980) (Table 4).

Table 4. Hormone results in 113 obese girls (1979, Budapest, Hungary)

| Hormonally quiet period (age 6 to 10 years) | | | Prepubertal-pubertal maturation process (age 11 to 15 years) | | |
|--|--------------------|-------------------|--|--------------------|--------------------|
| FSH 5.6 mU/ml | LH 7.5 mU/ml | PRL 7.54 ng/ml | FSH 9.1 mU/ml | LH 30.04 mU/ml | PRL 16.51 ng/ml |
| LH/FSH = 1.33 | | | LH/FSH = 3.30 ↑ | | |
| T3 1.12 nmol/l | T4 114.4 nmol/l | TSH 4.43 mU/ml | T3 1.02 nmol/l | T4 107.6 nmol/l | TSH 2.78 mU/ml |
| urinary 17 ketosteroids: | | 4.24 mg/die | urinary 17 ketosteroids: | | 5.01 mg/die |
| urinary oestrogens: | | 14.00 mg/die | urinary oestrogens: | | 22.86 mg/die |

From 1979-1982 we found among 3024 new patients 188 cases of obesity (6.21%). Vulvovaginal infections or amenorrhoea secundaria were the most frequent problems. Analysing their hormonal state we also had a high LH/FSH ratio, which is typical for chronic anovulation syndrome. Therefore, we have learned that obesity in *itself* has to be seen critically in young girls in respect to a later evolved ovarian decompensation. The study of this problem became urgent, that we composed 3 different groups from our *new* obese, already menstruating patients. Between 1983-1988 we had 5605 new patients (Örley 1992).

In the first group, we selected 40 healthy, normally menstruating obese girls. The second group contained 60 obese raromenorrhoeic patients with freshly developed striae. The third group involved 40 obese raromenorrhoeic patients with evident hyperandrogenic features (beard, mustache, hirzute piles, deep voice, etc.) The comparison was again made by hormone results. As it had been expected from former



Group 1: 40 healthy obese girls
 Group 2: 60 obese raronorrhoeic patients
 Group 3: 40 obese raronorrhoeic hirzute patients

Fig. 1: Distribution of the obese relatives in the 3 groups (a), and percentage of patients with more than 8 obese relatives in the 3 groups (b)

studies, the LH/FSH ratio was always disturbed, especially in those girls who had menstrual problems. In the 3 group, the testosterone was elevated too and in most cases, polycystic ovaris were diagnosed by ultrasonographic method (Table 5).

Table 5. Comparison among the groups examined between 1983 and 1988, Budapest, Hungary

| | 1 40 healthy obese girls | 2 60 obese raro- menorrhoeos patients | 3 40 obese hirzute raro- menorrhoeos patients | | |
|---------------------------------|--------------------------------|---|---|---------------------------------|--|
| Age | \bar{x} 14.14 years | \bar{x} 15.48 years | \bar{x} 15.76 years | | |
| Menarche | \bar{x} 12.21 years | \bar{x} 12.75 years | \bar{x} 12.48 years | | |
| Kaup index: | \bar{x} 2.90 | \bar{x} 2.88 | \bar{x} 2.68 | | |
| Hirata inedx: | \bar{x} 38.32 | \bar{x} 38.16 | \bar{x} 39.09 | | |
| $\text{LH/FSH} = 3.68 \uparrow$ | | $\text{LH/FSH} = 4.75 \uparrow$ | | $\text{LH/FSH} = 3.09 \uparrow$ | |

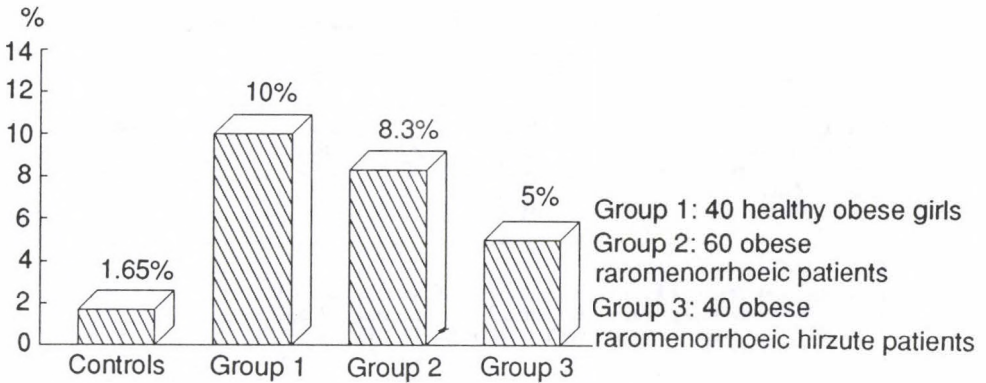


Fig. 2: Occurrence of diabetes mellitus among the first-grade relatives of the examined obese patients (mother—father—sibs)

We controlled our patients for genetic predisposition. A very high percentage of obese relatives was proven in the group 1, and in the group 2 (Fig. 1). Diabetes mellitus first-grade relatives were also high in these two groups (Fig. 2). Therefore, we have concluded that obesity, itself, promotes menstrual disturbances by altered gonadotropin values and later concomittant ovarian dysfunction.

All these studies and facts have prompted us to continue our work in this respect and a questionnaire was circulated for those former patients who were examined and treated at our pediatric gynecological Ward between 1977 and 1984. In these years we had 5236 new patients. Among them 378 girls were obese (7.21%). Their mean age was $\bar{x} = 11.979$ yrs. The remained 132 girls were overweight (2.52%) with a mean age $\bar{x} = 11.415$ yrs. The distinction of obesity based on the calculation of ideal weight related to actual height (IW/AH). We ranged overweight between 100–120% of it, thus the obesity could have been ranged into moderate above 140% or excessive form, above 180%. Generally, the use of the indices Kaup and Hirata together with the BF%, helped us in diagnosing obesity.

Our former patients were requested to reply or seek medical help from us.

Results

Among the 510 former patients, 136 adults (26.6%) replied or requested medical help from us. Their actual physical characteristics are enumerated at the Table 6. We recorded the normal gynaecological state of these follow-up women (Table 7). Among them 61 women became normally menstruating (44.85%), 31 women delivered (22.79%) and 33 were on oral contraception (24.26%). The Table 8 shows the gynecological pathology of these adults. The most frequent menstrual problem remained: raromenorrhoea in 34 cases (25%). Sterility occurred 3 times more in obese women than in ideally weighted ones (10 to 3 respectively). Miscarriage was in equal number in all re-examined patients present (3–4–3). We found 5 obese hyperprolactinaemics on bromocriptine regimen, in 6 cases surgical intervention was made on the ovaries because of cystic deformations. One women suffered from adrenal adenoma, she was obese with 110 kgs. Another epileptic obese patient's weight augmented to 140 kgs at her age of 14. Hypertension was diagnosed in one overweight woman and in two obese patients (Table 9).

Table 6. Physical characteristics of adult women

| Characteristics | Overweight | Obese | Normal |
|------------------|------------|-------|--------|
| Mean age (y) | 19.70 | 22.30 | 19.40 |
| Mean height (cm) | 167 | 165 | 166 |
| Mean weight (kg) | 66.50 | 83.90 | 57.60 |
| Index of Kaup | 2.38 | 3.08 | 2.09 |
| Index of Hirata | 41.22 | 37.69 | 42.98 |

Differentiation was made by the use of the indices of Kaup and of Hirata:
Index of Kaup: overweight means as Kaup i: 2.2—2.4; Hirata i: 40.5—42.0.
Index of Hirata: obese means as Kaup i: > 2.4; Hirata i > 40.5

Table 7. Normal Gynecological state of adult women

| | Overweight | Obese | Normal |
|----------------------------------|------------|-------|--------|
| Delivery | 4 | 20 | 7 |
| Oral contraception | 8 | 17 | 8 |
| Medical termination of pregnancy | — | 4 | 2 |
| Normal menses | 17 | 30 | 14 |

Table 8. Gynecological pathology of adult women

| | Overweight | Obese | Normal |
|------------------------------|------------|-------|--------|
| Sterility | — | 10 | 3 |
| Miscariage | 3 | 4 | 3 |
| Metrorrhagia | — | 6 | 1 |
| Raromenorrhoea | 8 | 19 | 7 |
| Amenorrhoea sec | — | 2 | — |
| Dysmenorrhoea | 3 | 5 | 3 |
| Polymenorrhoea | 1 | 2 | 2 |
| Prolactinoma | 1 | 5 | — |
| Endometriosis | — | 1 | 1 |
| Adnex-operation | 1 | 4 | 1 |
| Glandular cystic hyperplasia | — | 1 | — |
| Cong. genito-urin. anomalies | — | 3 | — |

Table 9. Complications in adults

| | Overweight | Obese | Normal |
|-------------------|------------|-------|--------|
| Adrenal adenoma | — | 1 | — |
| Epilepsy | — | 2 | — |
| Hypertonia | 1 | 2 | — |
| Schizophrenia | — | — | — |
| Mental deficiency | — | 1 | — |
| ITP + splenectomy | 1 | — | — |

At least 29 women reached the normal weight for height (21.3%), 33 persons are overweight (24.2%). Those who were or became obese had an extreme obesity (63 wo-

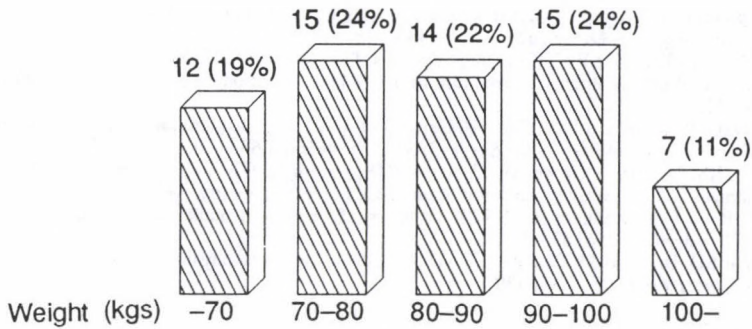


Fig. 3: Weight distribution of obese adults. Number of patients: 510 girls from 5236 new patients. Number of the reexamined patients: 136 girls. Total number of obese adults: 63

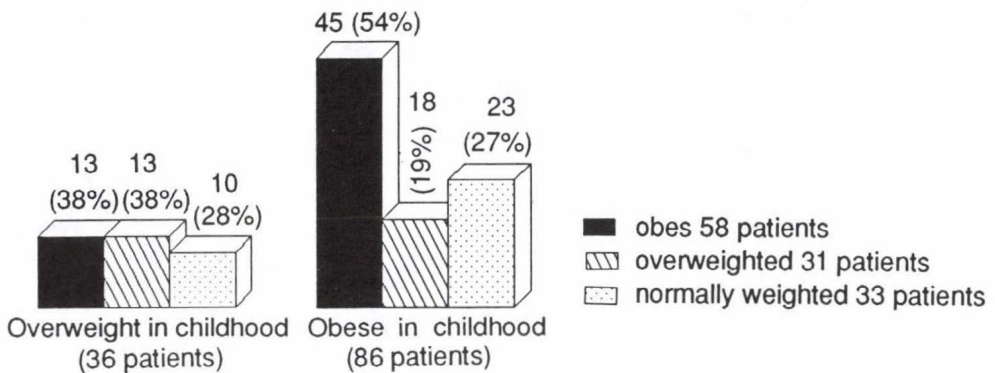


Fig. 4: Weight distribution in adults. Number of patients: 510 girls from 5236 new patients. Number of the reexamined patients: 136 girls

men, 46.32% whose weight distribution is depicted at the picture (Fig. 3). Figure 4 demonstrates the changes in weight of the follow-up patients. It means that this is a bad prognosis in peripubertal ages to be overweight or obese. 54% of obese girls remained obese (but in serious form) in adulthood, while 36% of overweight girls changed to obese adults. The pubertal obesity should be considered as a serious warning for future obesity together with all secondary complications of it, even in young women.

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