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BODY MASS INDEX–ASSOCIATED PATTERNS OF REPRODUCTIVE DYSFUNCTION IN PUBERTAL GIRLS

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Background

Reproductive dysfunction in pubertal girls remains one of the most relevant problems in pediatric gynecology and endocrinology, as it develops during the critical period of maturation of the hypothalamic-pituitary-ovarian axis and may substantially influence future reproductive potential. Menstrual disorders associated with abnormalities in body mass index (BMI) are of particular importance, since both obesity and underweight can significantly affect the tempo of pubertal development, the establishment of ovulatory function, hormonal homeostasis, and the morphofunctional characteristics of the reproductive organs. During puberty, alterations in metabolic status often become one of the key factors contributing to the development of amenorrhea, opsomenorrhea, acyclic uterine bleeding, and other menstrual disturbances. Therefore, the study of clinical, hormonal, and instrumental features of reproductive dysfunction in adolescent girls according to BMI is of clear scientific and practical relevance.

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Aim

To investigate the clinical, hormonal, and instrumental features of reproductive dysfunction in pubertal girls according to body mass index.

Materials and Methods

The study included 118 adolescent girls aged 12–18 years who presented with menstrual disorders and 30 apparently healthy age-matched girls with normal BMI and regular menstrual cycles. The main cohort was divided into two groups: 71 patients with high BMI and 47 patients with low BMI; the control group consisted of 30 girls. The study design included clinical-anamnestic, anthropometric, gynecological, hormonal, ultrasonographic, and statistical assessment. Height and body weight were measured, and BMI was calculated. The age at menarche, types of menstrual disorders, Tanner stages of secondary sexual characteristics, and severity of hirsutism according to the Ferriman-Gallwey score were evaluated. Hormonal assessment included serum levels of follicle-stimulating hormone, luteinizing hormone, estradiol, prolactin, testosterone, thyroid-stimulating hormone, and free triiodothyronine and thyroxine. All participants underwent pelvic ultrasonography and Doppler evaluation of uterine and ovarian blood flow. Differences were considered statistically significant at $p<0.05$.

Results

Comparative analysis revealed significant differences in the clinical phenotype of reproductive dysfunction depending on BMI ($p<0.05$). In the high-BMI group, the mean height was 158 cm, the mean body weight was 80 kg, and the mean BMI was 27.6. In the low-BMI group, the corresponding values were 162 cm, 45 kg, and 17.1, whereas in the control group they were 160 cm, 55 kg, and 21.5,

respectively. These differences reflected not only distinct patterns of physical development but also different endocrine and reproductive profiles.

Assessment of pubertal development showed that girls with high BMI more often exhibited advanced stages of sexual maturation: thelarche stage M3–M4 was observed in 74.6%, pubarche stage P3–P4 in 78.8%, and axillary hair development stage Ax2–Ax4 in 84.5%. In contrast, girls with low BMI predominantly demonstrated less mature stages, with thelarche stage M2–M3 in 68.1%, pubarche stage P2–P3 in 74.5%, and axillary hair development stage Ax1–Ax2 in 78.7%, indicating slower pubertal progression in the setting of body weight deficiency.

The age at menarche also varied according to BMI. Early menarche at 11–12 years was more common in girls with high BMI, accounting for 46.5%, compared with only 12.8% in the low-BMI group. Conversely, late menarche at 15–16 years was observed in 40.4% of girls with low BMI versus 12.7% in the high-BMI group, while very late menarche after 17 years was identified exclusively in patients with body weight deficiency (10.6%). These findings indicate that excess body weight is associated with accelerated pubertal maturation, whereas underweight is linked to delayed sexual development.

Analysis of menstrual disorder patterns made it possible to identify two distinct clinical profiles. In girls with low BMI, amenorrhea and opsomenorrhea predominated: amenorrhea was observed in 42.6% and opsomenorrhea in 34.0%. In girls with high BMI, along with opsomenorrhea (26.8%), acyclic uterine bleeding (21.1%) and polymenorrhea (11.3%) were recorded more frequently. Thus, body weight deficiency was more commonly associated with a hypoestrogenic pattern of menstrual dysfunction, whereas high BMI was associated with dysfunctional bleeding and signs of an endometrial hyperplastic response.

The severity of clinical hyperandrogenism also differed substantially between the groups. In the high-BMI group, the absence of hirsutism was noted in only 29.5% of adolescents, while mild hirsutism was present in 43.6% and pronounced hirsutism in 23.9%. In the low-BMI group, the absence of hirsutism was documented in 74.4%, mild hirsutism in 19.1%, and pronounced hirsutism in only 4.2%. These data indicate a greater prevalence of the hyperandrogenic phenotype among girls with overweight and obesity.

The hormonal profile confirmed the observed clinical differences. In the high-BMI group, follicle-stimulating hormone levels were lower than in controls (5.2±0.31 vs 6.0±0.37 mIU/L, $p<0.05$), whereas luteinizing hormone, prolactin, and testosterone were increased to 10.4±0.52 mIU/L, 18±2.09 ng/mL, and 2.1±0.23 nmol/L, respectively; estradiol level was 45±3.11 pg/mL. In girls with low BMI, a relative increase in follicle-stimulating hormone to 7.0±0.43 mIU/L was observed ($p<0.05$), accompanied by lower luteinizing hormone (6.5±0.35 mIU/L), estradiol (35±2.07 pg/mL), prolactin (12±1.51 ng/mL), and testosterone (0.9±0.12 nmol/L). In the control group, the hormonal profile was more balanced: follicle-stimulating hormone 6.0±0.37 mIU/L, luteinizing hormone 7.5±0.44 mIU/L, estradiol 55±2.51 pg/mL, prolactin 15±1.82 ng/mL, and testosterone 1.2±0.17 nmol/L. Overall, the major intergroup hormonal differences were statistically significant ($p<0.05$*).* These findings suggest that high BMI is associated with a hyperandrogenic, hyperluteinizing, and dysmetabolic variant of reproductive dysfunction, whereas low BMI is linked to a hypoestrogenic state and relative suppression of ovarian activity.

Instrumental findings further supported the clinical and hormonal profile. In girls with low BMI, uterine hypoplasia was detected more frequently (34%), along with thin endometrium (40.5%) and persistent follicles (20.0%), which was consistent with insufficient estrogenic stimulation and anovulatory dysfunction.

In the high-BMI group, the most characteristic ultrasound finding was multifollicular ovaries, observed in 19.7% of cases, while Doppler assessment revealed increased vascular resistance in 50.7% of patients. In contrast, reduced ovarian arterial blood flow was found in 44.7% of girls with low BMI. In the control group, normal pelvic echostructure was observed in 86.7% of the participants.

Taken together, these findings indicate that two distinct clinical and pathogenetic variants of reproductive dysfunction are already formed during adolescence. The first is associated with high BMI and is characterized by accelerated pubertal development, early menarche, hyperandrogenic manifestations, elevated luteinizing hormone, prolactin, and testosterone levels, as well as a higher frequency of acyclic uterine bleeding. The second variant is associated with low BMI and is manifested by delayed menarche, immaturity of secondary sexual characteristics, predominance of amenorrhea and opsomenorrhea, hypoestrogenism, uterine hypoplasia, and thin endometrium.

Conclusion

Reproductive dysfunction in pubertal girls demonstrates a clear clinical, hormonal, and instrumental association with body mass index. High BMI is linked to a hyperandrogenic-dysmetabolic phenotype characterized by accelerated pubertal maturation, early menarche, hirsutism, elevated luteinizing hormone and testosterone levels, and a higher frequency of acyclic uterine bleeding. In contrast, low BMI is associated with a hypoestrogenic phenotype marked by delayed pubertal development, late menarche, amenorrhea, reduced estradiol levels, and signs of morphofunctional immaturity of the uterus and endometrium. These findings support a differentiated clinical and diagnostic approach to adolescents with menstrual disorders, with mandatory consideration of BMI, somatometric parameters, pubertal development pattern, hormonal profile, and ultrasound characteristics.