

## RISK FACTORS FOR DELAYED SEXUAL DEVELOPMENT IN ADOLESCENT GIRLS

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**Annotation.** In the available literature, there are isolated and isolated references to the influence of extragenital pathology and heredity on the development of delayed sexual development. Social and medical factors that provoke this pathology have not been studied, and therefore there are no recommendations for preclinical identification of children at risk of late puberty.

Studying the influence of unfavorable factors and content on the main prognostic risk and complex, timely prevention of delayed sexual development is an urgent task. Modern methods of treating this pathology are often ineffective. This is due, on the one hand, to a late visit to the doctor, and on the other hand, to the availability of sufficiently effective diagnostic and treatment methods that make it possible to form reproductive health and normalize the functions of the second system: psychoemotional, endocrine, and immune.

Delayed sexual development in adolescent girls requires further study and development of recommendations for the prognosis, diagnosis and treatment of this disease.

**Keywords:** gender, development, girls, adolescents, immunity, endocrine system

**Relevance.** Delayed sexual development in the structure of gynecological diseases occurs in 15-18% of girls. When conducting a targeted medical examination, this indicator can increase to 23%. Over the past decade, interest in pediatric gynecology has increased, but many issues of diagnosis, treatment and prevention of sexual development disorders are insufficiently studied. Considering that over the past 10 years the number of adolescent girls with chronic somatic diseases and disorders in the formation of the reproductive system, 7 has increased to 76.7%, this problem is gaining not only medical, but also social significance. The features of delayed sexual development at the present stage are changes not only in the

reproductive system, but also in the endocrine, immune, and nervous systems. Under these conditions, studying the premorbid state of the girl's body, identifying the main pathogenetic mechanisms, and timely diagnosis and treatment of detected disorders will not make a significant contribution to solving this problem. In the available literature, there are isolated and isolated references to the influence of extragenital pathology and heredity on the development of delayed sexual development. Social and medical factors that provoke this pathology have not been studied, and therefore there are no recommendations for preclinical identification of children at risk of late puberty.

Studying the influence of unfavorable factors and content on the main prognostic risk and complex, timely prevention of delayed sexual development is an urgent task. Modern methods of treating this pathology are often ineffective. This is due, on the one hand, to a late visit to the doctor, and on the other-to the presence of sufficiently effective diagnostic and treatment methods that allow you to form reproductive health and normalize the functions of the second system: psychoemotional, endocrine, and immune.

Thus, delayed sexual development in adolescent girls with requires further study and development of recommendations for the prognosis, diagnosis, and treatment of this disease.

**Purpose of the study.** Identification of risk factors that provoke delayed sexual development in adolescent girls.

**The materials and methods of research are investigated.**

We examined 30 adolescent girls aged 12-18 years who applied to the Samarkand Regional Multidisciplinary Children's Medical Center for 2020-2023 using a retrograde analysis of their medical history and outpatient records, and divided them into the following groups, respectively: group 1 consisted of 15 girls with a diagnosis of grade I sexual development delay, group 2 15 girls with a diagnosis 33.3% were girls with a diagnosis of delayed sexual development of II-III degrees, and the control group consisted of girls from groups 1 and 2 of the health level and 10 girls of the same age with normal sexual development.

The risk factors for MDG of central origin in adolescent girls were studied, systematized in the form of 4 complexes: features of genealogical history, pathology of pregnancy and

childbirth, features of the neonatal period, morbidity and development of the child in other age periods.

From the anamnesis of the offspring, the following were distinguished: gynecological diseases of the mother, late menstruation in the mother (after 15 years), as well as vadelay in sexual development in relatives of the 1st and 2nd degrees of kinship on the part of the father and mother.

When studying the obstetric history, attention was paid to the mother's bad habits during pregnancy (smoking, alcoholism), the risk of miscarriage, gestosis, viral and bacterial diseases of the mother, taking medications during pregnancy.

Features of the process of childbirth in the intrauterine period are noted: preterm labor, rapid and difficult labor, weakness of labor forces.

In the neonatal period, the health status of children was studied: large fetus (weighing 4000 g or more); small children (less than 3000 g); preterm birth.

In addition, fontook into account the general background, such as an early transition to artificial feeding (up to 4 months), the formation of motor skills at an early age and slow rates of physical development; diseases of other age groups (chronic pathology of the gastrointestinal tract, nephrourological pathology, chronic pathology of ENT organs, bronchopancerous diseases, tubinfection, neurological pathology etc.), the presence of increased physical activity.

#### **Research results and discussion.**

As a result of the research, out of the total number of parameters that characterize the health status of the surveyed adolescent girls, 26 signs were selected that had significant differences in the compared groups and were characterized by the greatest informative value.

Depending on the value of the AR coefficient, 3 degrees of risk were identified. Level I (minimal risk) -  $AR < 30\%$ , level II (medium risk) -  $AR = 30-60\%$ , level III (high risk) -  $AR > 60\%$ . The probability of predicting this pathology increased from grade I to grade III.

A comparative analysis of AR values showed that the highest percentage of development of ASD in the study group I was expected in adolescent girls with a severe hereditary history. II Group II girls, however, had a higher risk at an early age (AR-69.9%), as well as a lower body weight of less than 3000 g (AR - 61.4%), slower motor skills and physical development.

The average risk factors for girls I in group I include delayed sexual development in relatives of the 1st and 2nd degrees of maternal kinship (AR-32,0,0%).

The average risk factors for girls II in group II were: severe obstetric history (preterm birth-38.5.5%, risk of miscarriage-32.6%, gestosis-31.0%); long-term use of the drug during pregnancy-31.6%. Moderate risk factors in both groups surveyed: early artificial feeding (AR-30.9.9%) - I group I and AR-31.0% II- group II), chronic gastrointestinal pathology (AR-48.4% and AR). -58.7%, chronic nephrourological pathology (AR-42.8% and AR-46.39%), chronic pathology of ENT organs (ar-50.7% and ar-50.8%), frequent respiratory diseases (ar-40.0% and ar-42.6%).).

Minimal risk factors played a smaller role in the formation of the studied pathology. In both groups examined, there were: single-parent families (13.8% I of group I and 8.7% II of group II), a severe hereditary history of gynecological diseases of the mother (16.8% and 19.5%), sexual disorders in relatives of the 1st and 2nd degrees of kinship on the part of the father (8.7% and 7.6%), the mother's age (5.7%). father's age under 20 (1.2% and 1.4%), father's age over 35 (1.5% and 3.0%), mother's age over 35 (2.5% and 20.8%), mother's bad habits during pregnancy (2.5% and 20.8%), viral and bacterial diseases (17.5% and 27.5%), rapid and severe labor (1.6% and 0.3%), labor weakness (3.4% and 10.1%), birth weight 4000 g or more (1.0%, 1.1%), perinatal encephalopathy (28.4% and 23.0%), neurological pathologies detected (26.6% and 26.6%), tuberculosis (7.7% and 1.6%), physical activity (16.8% and 2.3%).

Among girls with ASD I in Group I, physical development was assessed as harmonious on average in 5 people (33.3%). 2 children (13.3 %) showed a decrease in physical development indicators, 2 (13.3%) - low indicators, 1 child (0.66%) - high indicators, and 2 children (13.3%) - low indicators (Table 1).3.1).

The physical development of the control group was significantly different from the physical development of the girls in the main group. Harmonious physical development is typical for all 15 girls with normal sexual development, in 10 girls (66.6 %) it was assessed as moderately harmonious, in 2 girls (13.3- 3%) - as harmonious above average. In this group, the mesomorphic somatotype prevailed in 11 people (73.3%). A small proportion: leptomorph-2 in girls (13.3%), dolichomorph-2 (13.3%) and brachymorph-1 (0.66%).

When assessing the physical development of patients in group II, low indicators prevailed - 11 people (73.3 %) and a decrease in physical development indicators - 4 people (26.6 %).

Выявлен The somatotype of all examined girls was revealed-anthropometric (morphological) features of the body (Y..E..Veltishchev., 1998). According классификации to Bunak's classification, the following somatotype variants were obtained соматотипов: in the group with grade I ASD, the mesomorphic type prevailed-45.5.5%, a significant part was leptomorphic type-28.4%. The indeterminate type was 13.6.6%, the smaller part was made up of somatotypes: dolichomorph-78.0%, brachymorph-3.4% and 1.1 % andromorph.

In II group II, indeterminate somatotype prevailed in the studied girls соматотип- 76.9.9%, leptomorph-18.0% and mesomorph-5.1%.

In the control group, the mesomorphic somatotype had a large proportion-82.1.1%, a small proportion fell on leptomorphic-5.4%, dolichomorphic-3.6% and brachymorphic-1.8% somatotypes.

The sexual development of adolescent girls in all groups was evaluated on the Tanner scale and expressed by the formula: Ma, topor, p, I, where Ma is the mammary glands; Ax is the hair of the armpits; p is the hair of the chow-chow area; i is the menstrual age.

The majority of girls with ASD in group 1 had violations of the sexual formula: secondary sexual characteristics are weakly expressed-52.3% Mai AXL Pi; secondary sexual characteristics do not lag significantly behind the age - related ones-in 29.5% of cases > Ma2 ax2 P2; complete absence of secondary sexual characteristics - in 10.2% of cases, Mao Axo P0; secondary sexual characteristics are traditionally developed - Ma3 Ax 3 R 3 was detected in 7.9% of cases3 R.

In all patients of group II, the sex formula to grade did not correspond to age: 8 people (53.3%) had no secondary sexual characteristics  $Ma0 Ax0 P0$ ; secondary sexual characteristics in 3 people (20%)  $Ma1 Ax1 P1$  developed poorly.

When assessing sexual development in the control group, the development of secondary sexual characteristics varied depending on age: 12-13 years  $Ma2 Ax2 P2$ ; 14 or more years  $Ma3 Ax3 P3$  and corresponds to age standards.

**Outputs.** Thus, for the degree 1 central genesis of ASD in adolescent girls, a severe hereditary history-delayed menstruation in the mother, premature birth, low birth weight is a high risk factor. In girls with central ASD, hormonal physical development prevails over disharmonious development. Girls with ovarian-specific ASD are characterized by disharmonious physical development.

Thus, analyzing sexual development in the group with central origin, it was noted that secondary sexual characteristics are poorly expressed, in rare cases they are absent. Most girls in group II do not have secondary sexual characteristics.

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