

## PEDIATRIC ASPECTS OF GYNECOMASTIA

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**Abstract:** The article presents theoretical information about gynecomastia – enlargement of the mammary glands in men. It also presents our own observation data of 22 teenage boys, including 20 with pubertal gynecomastia and two young men with pathological gynecomastia.

**Keywords:** pediatrics, adolescents, gynecomastia, mammary gland.

### INTRODUCTION

Gynecomastia (from Latin genes – woman, mastos – breast, mammary gland – female breast) is a visible or palpable enlargement of the mammary glands in males. It can be a physiological manifestation of puberty or indicate pathological disorders associated with impaired synthesis, metabolism and inactivation of estrogens [1, 2, 4]. In any case, examination and dispensary observation of patients with gynecomastia are indicated [1, 2, 4]. A pediatrician, as a primary health care specialist, should be able to diagnose and know the tactics of action in case of gynecomastia. According to various authors, enlargement of the mammary glands occurs in 40 to 75% of pubertal boys [1, 2, 4]. The size of the mammary gland in gynecomastia can range from 1 to 10 cm, on average, about 4 cm. There are several peaks in the incidence of gynecomastia. The first, neonatal, is associated with the action of maternal estrogens; the second, pubertal, in which the development of mammary glands is associated with a physiological decrease in the level of free testosterone, an increase in the level of estradiol and SHBG (sex hormone-binding globulin).

### MATERIALS AND METHODS

Gynecomastia occurs due to hormonal disorders - absolute or relative increase in estrogen levels. Absolute increase in estrogen levels is observed when taking medications containing estrogens; development of a Leydig cell tumor (a rare type of testicular tumor that synthesizes estradiol, in 90% of cases the tumor is benign); tumors synthesizing estrogens - rare, usually benign neoplasms; tumors producing human chorionic hormone (hCG) (testicular tumors and carcinomas of various localizations). Relative increase in estrogen levels is observed in age-related hypogonadism - decreased testosterone secretion by the testicles; primary hypogonadism - Klinefelter syndrome; hypogonadotropic hypogonadism - decreased testosterone synthesis while maintaining estrogen production by the adrenal glands (relative hyperestrogenemia develops). A relative increase in estrogen levels occurs with refeeding syndrome after prolonged malnutrition; renal failure and dialysis, in which the level of luteinizing hormone (LH), estradiol (E), prolactin (P) is increased and the level of testosterone (T) is decreased; chronic liver failure (when a decrease in androgen metabolism in the liver leads to an increase in the aromatization of androgens into estrogens, an increase in the level of SHBG, and a decrease in the level of free T); hyperthyroidism (gynecomastia is caused by an increase in SHBG and increased activity of aromatization of androgens into estrogens); hyperprolactinemia (stimulates the proliferation of epithelial cells of the mammary gland and suppresses the production of gonadotropins, which leads to the occurrence of secondary hypogonadism and the development of gynecomastia), as well as HIV infection, which leads to hyperprolactinemia and symmetrical gynecomastia,

iatrogenic gynecomastia and drug addiction. Iatrogenic gynecomastia is associated with the use of drugs (narcotic analgesics, spironolactone, cardiac glycosides, phenothiazines, cytostatics, cimetidine).

## RESULTS AND DISCUSSION

There are several stages of gynecomastia: developing (initial stage lasting 4 months, at which regression is possible), intermediate (maturation of breast tissue, lasting up to 4 months, reverse development or regression is possible, but difficult) and fibrous stage, characterized by the appearance of mature connective tissue in the mammary gland, deposition of fatty tissue around the glandular tissue and the impossibility of reverse development of the mammary gland (regression). When identifying a teenager with gynecomastia, a thorough anamnesis should be collected (duration of the existence of an enlarged mammary gland, the presence of complaints of pain, taking medications, changes in body weight). During the examination, attention is paid to the symptoms of thyrotoxicosis, palpation of the mammary glands, abdomen (to exclude splenomegaly, hepatomegaly, enlarged adrenal glands), examination of the external genitalia and assessment of sexual development. Patients with gynecomastia should undergo a full examination (biochemical blood test with determination of creatinine level, liver enzymes; studies of hormone levels - total testosterone, SHBG, E, LH, follicle-stimulating hormone (FSH), P, dehydroepiandrosterone sulfate (DHEA-S), thyroid-stimulating hormone (TSH), free thyroxine (T4), beta-hCG; if indicated, mammography or ultrasound of the mammary glands, FNAB - fine-needle aspiration biopsy are performed).

In 20 clinical cases (91%) pubertal (juvenile) gynecomastia was diagnosed (17 bilateral and 3 unilateral); in one patient it was pathological (iatrogenic) and associated with taking cytostatics for a blood disease, and in another young man it was due to microprolactinoma. Blood biochemistry and hormone levels in patients with juvenile gynecomastia were normal. The average hormone levels were: P -  $141.3 \pm 92.0$  (normal 53–360.0 mIU/L); LH -  $1.4 \pm 0.7$  (normal 0.8–7.6 IU/L); FSH -  $1.5 \pm 0.4$  (normal 0.7–11.1 IU/L); T -  $14.8 \pm 2.5$  (normal 3.5–21.4 nmol/L); TSH -  $1.9 \pm 0.2$  (normal 0.4–4 mU/L); free T4 -  $15.2 \pm 2.3$  (normal 10–25 pmol/L); DHEA-S -  $3519.2 \pm 1791.8$  (normal 1200–9600 nmol/L) and E -  $54.8 \pm 20.2$  (normal 20–130 pmol/L). The echo picture of the mammary glands in juvenile gynecomastia looked like this: the glandular tissue was represented by fat lobules (hypoechoic areas) and supporting (connective tissue) stroma (hyperechoic areas).

Patients with pathological gynecomastia were examined and received etiopathogenetic treatment in specialized clinics. Patients with juvenile gynecomastia were examined once every 6–12 months; there were no indications for surgical treatment; regression was noted at the age of 17–18 years.

## CONCLUSION

In most cases, young men are diagnosed with pubertal, i.e. physiological gynecomastia, which does not require special treatment, but requires dispensary observation. Knowledge of the problem of gynecomastia in pediatric practice is relevant due to the presence of various etiological factors for the enlargement of the mammary glands in males.

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