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## Methods of Dental Diagnosis of Hypertrophic Gingivitis

**Rajabov O.A., Ostonova A.Sh.**  
**Bukhara State Medical Institute**

**Relevance of research.** Periodont tissues are not fully formed and are in a state of physiological stress for a long time. The hormonal imbalance associated with puberty affects the epithelium of the gums, which in turn depends on the clinical and functional characteristics of the parodont. From these positions, the oral cavity is seen as an ecological system in which different biological processes interact together, causing multidirectional pathological processes. However, it does not allow you to completely solve the tasks set in front of the above, which leads to the need to look for the most rational, effective and pathogenetically based diagnostic methods. The literature places great emphasis on local risk factors that aggravate the course of chronic hypertrophic gingivitis [1.3.5].

It should be noted that the mechanism of the onset of puberty has not yet been sufficiently studied. During adolescence, there are significant changes in the functioning of various endocrine glands. Strengthening the function of the gonads during puberty has a significant effect on the functioning of other endocrine glands and can upset the balance of the child's childhood. During this period, most children have a clinical picture of endocrine disorders. It is known that estrogens and androgens, as well as other hormones, have the ability to stimulate or excite the synthesis of various RNAs that transmit information contained in genes. Currently, it is assumed that during puberty, the body's ability to isolate the amount of sex hormones is genetically determined. Especially important is the effect of estrogens on the anterior pituitary gland, indirectly through the hypothalamus, while the amount of growth hormone in the pituitary decreases when exposed to estrogen [2.4.6].

Milk has measured fluid interleukin-1beta (il-1B) and tumor necrosis factor alpha (TNF-A) levels and is based on changes in periodontal indices in young healthy women during the menstrual cycle. Suppression of estrogen levels during the reproductive period can also lead to an adequate formation of the highest level of bone mass, a pathological decrease in bone mineral density. Thus, a review of literature data has shown that impaired estrogen secretion contributes to the development of parodont pathologies, as parodont has a genomic effect on target cells that are hormone-sensitive and contain estrogen parodont tissues. When diseases with hypoestrogenism occur against the background of a decrease in bone density (osteoporosis, osteopenia), hyperplastic processes with hyperestrogenism are also observed.

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