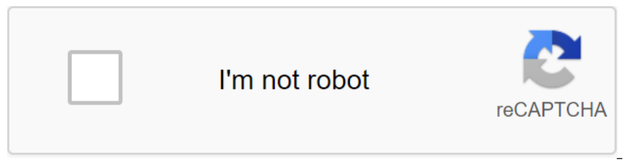
Ginecomastia masculina tratamento pdf



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Estradiol and progesterone stimulate breast growth in adult women. While estradiol promotes the growth of ducts, progesterone acts on alveolar development. For estrogenic and progestogenic effects to manifest themselves completely, the actions of other hormones such as prolactin, GH and IGF-1 are necessary. In contrast, androgens inhibit breast development. This scenario becomes increasingly complex as estrogens may be the result of peripheral conversion of androgens such as androstendeyone and testosterone enzyme aromatase. Figure 1: Breast Tissue Development Figure 2: Synthesis of androgens and estrogens 17'HSD: 17 -hydroxysteroid-dehydrogenase; 3'-HSD: 3 hydrosteroid dehydrogenase. Gynecomastia is the result of an imbalance between estrogenic and androgenic effects on male breast tissue. In normal development there are situations in which this imbalance is considered physiological, as it happens in the neonatal period, puberty and old age. Distinguishing these physiological situations from pathological conditions (some severe, such as neoplasma) is the main problem of the doctor in the face of the case of Gynecomastia. Table 1 lists the main causes of Gynecomastia.Table 1: Causes of Gynecomastia About 25% of patients with Gynecomastia have no identifiable cause for pathology, and Gynecomastia is therefore considered idiopathic. Another 25% have puberty of gynecomastia; Between 10% and 20% of cases are related to drug use. Cirrhosis of the liver, malnutrition and primary hypogonadism are 8%; testicular tumors, 3%; Hypogonadism, 2%; hyperthyroidism, 1.5%; and chronic renal failure, 1%. DIFFERENTIAL DIAGNOSIS OF GYNeCOMASTIA Every patient who presents with Gynecomastia must undergo a history and physical examination with special attention to the use of drugs, drugs and alcohol. Signs and symptoms of systemic diseases such as hyperthyroidism, hypogonadism, kidney or liver failure should be actively investigated. The age of installation and the rate of progression of the condition can help in differential diagnosis. Rapid progression of gynecomastia is usually associated with excess hormone production. Physical examination should be done with the patient lying in a position on the back and palpation from the periphery to the center. Breast tissue should be measured in cross-sectional diameter. Evaluation of the testicles is necessary for testicular atrophy (suggests hypogonadism) or testicular mass (suggests neoplasia). Physiological gynecomastia As mentioned, gynecomastia is considered physiological during the neonatal period, during puberty old age. Neonatal gynecomastia as a result of high levels of estradiol and progesterone is transmitted from the mother to the newborn placenta. Some newborns have a discreet papalar discharge, the so-called witch's milk. Neonatal gynecomastia is bilateral, can affect more than 90% of newborns, and does not imply specific treatments, as it is self-governing and should regress for the sixth month of life. Pubertal gynecomastia is the second period of development in which gynecomastia occurs physiologically. Breast tissue measuring more than 0.5 cm can be palped in 50% of boys between the ages of 10 and 16. Less than 10 per cent of boys have more pronounced gynecomastia detected in the general survey. Puberty gynecomastia has a peak incidence of about 14 years, and in most cases it is bilateral. However, glandular involvement can be asynchronous or asymmetrical. An unusual variant of sexual gynecomastia is macrogynecomatia. In this case, the breast tissue is more than 5 cm in diameter, and hardly has spontaneous regression. The pathophysiology of puberty Gynecomastia seems to lie in reducing the ratio of androgens/estrogens during puberty, as evidenced by some studies. Senil gynecomastia Gynecomastia that occurs with aging is also considered physiological. Although the exact mechanism responsible for Gynecomastia in adult men is not fully clarified, some elements seem to be involved, such as lowering serum testosterone levels and increasing peripheral secondary flavoring that fatty tissue and aromatase activity, resulting in higher estrogen production. These processes are accompanied by elevated levels of the sex hormone binding protein (SHBG), which has a greater affinity for testosterone than estradiol, ultimately leading to a lower testosterone/estradiol ratio. It is difficult to assess the true prevalence of gynecomastia in adults, because its difference from lipomastia is difficult, especially in obese people. Some studies indicate a prevalence of up to 40% in healthy men, reaching up to 65% in hospitalized men. Pathological gynecomastia are numerous causes of pathological gynecomastia. Thus, a clinical trait common to several diseases, some of which have the potential for fatality, should be considered. The doctor should be aware of this fact, and should look for other clinical or laboratory features that offer some systemic diseases that curse with Gynecomastia. The main causes of pathological gynecomastia will be discussed below. Testicular tumors can cause gynecomastia by producing production of androgens with subsequent aromatization or secretion of gonadotropins, which stimulate normal Leydig cells. Estrogen-secreting tumors of the testicles of leidig-cell tumors, Sertoli cell tumors, and cell tumor granulosis. Tumors of germ cells, which include seminomas and non-seminomas, are capable of producing K-HCG, which has an action similar to the luteinizing hormone (LH) on Leydig cells, causing increased estrogen secretion by these cells. Leidig cell tumors account for between 1% and 3% of testicular tumors. They occur in men between the ages of 20 and 60 years and are characterized by a palpable mass of testicles. Gynecomastia occurs in about 30% of patients, and as a result of increased estrogen synthesis of tumors. Certoly cell tumors account for 1% of testicular tumors occurring in all age groups, and a third in children under the age of 13, usually in children under 6 months of age. Gynecomastia occurs in a third of cases. Extra-testicle tumors of bronchogenic, liver tumors and stomach carcinomas can also produce K-HCG, causing Leidig cells to be stimulated in the same way as what occurs in the tumors of the germ cells of the testicles. Adrenal tumors can secrete DHEA, DHEAS and androstenedione, which are peripherally flavored with estradiol. In rare cases, adrenal adenocarcinoma secretes estradiol and eston. These tumors are rare and very malignant, with a reserved prognosis. Hepatocellular carcinomas can cause gynecomastia due to increased aromatization of androgens. Hypogonadism When it occurs in childhood, it presents with delayed puberty. When this occurs in adulthood, clinical manifestations decrease libido, erectile dysfunction, weakness, fatigue, infertility, loss of hair on the body and muscle mass. Reduced androgen secretion and higher estrogen production (due to greater aromatization as a result of increased adipose tissue characteristic of hypogonadism) are causes of gynecomastia. The main causes of primary hypogonadism (testicular lesions) are Klinefelter syndrome (cariotype XXY), congenital anomaly, viral or bacterial orchid, trauma, radiation of the testicles and spinal cord injury. Gynecomastia is observed in 50% of patients with dialysis renal failure stemming from the hypogonadism they represent. Excessive apolization There are cases of family gynecomastia, whose cause is associated with increased activity of extragonadic aromatase. Obesity and hyperthyroidism also increased aromatase activity. Ginecomastia is known to occur in feedback after periods of innoonation. The cause is not well understood, but it is probably due to a decrease in hepatic clearance of androgens, followed by an increase in peripheral aromatization. The same mechanism is involved in gynecomastia associated with hepatopathy. Congenital adrenal hyperplasia is characterized by a deficiency of the synthesis of adrenal corticosteroids. The most common cause is a deficiency of the enzyme 21-hydroxylase. Lack of cortisol production leads to an increase in ACTH production, followed by overproduction of androgens, which can subsequently be flavored, causing gynecomastia. Drugs Several drugs are involved in the pathogenesis of Gynecomastia on the following mechanisms: an action similar to estrogens; increase in endogenous estrogen production, as well as estrogen precursors, which are subsequently flavored; production or action of androgens or the release of estrogen from its carrying protein. Gynecomastia can occur when there is occasional contact with estrogen-containing foods such as skin cream, vaginal creams, intercourse, taking milk or meat from animals treated with estrogen (tables 2 and 3). Table 2: Drugs That Cause Gynecomastia by the Known Mechanism of Drug Binding To Estrogen ReceptorCreames or Substances, Estrogen-containing DigitalClonilicS Clomiphene Cannabis Isoniazid Stimulating estrogen synthesis Gonadotropins Growth Hormone Precursors for The Aromatization of Androgenexogens Test Defeat Busulfan Nitrous Vincristina Ethanol Blocking Synthesis Of Testosterone Ke totoconazole Spironolactone Metronidazole Etomidate Leuprolide Unit androgenic Action Finasteride Ciproteridine Spironolactone Estrogen Release from ShBG Spironolactone Ethanol Table 3 : Drugs that cause Gynecomastia unknown mechanism Class drug antihypertensive drugs or heart-acting Calcium blockers channel ACE inhibitors beta-blockers Amiodarone Methyldopa Nitrates Psychotropic drugs Neuroleptic Diazepam phenytoin Tricyclic Antidepressants Haloperidol Infectious Diseases Drugs Anti-HIV Therapy Medications Other amphetamines Theophylline Amperazole Domperidon Heparin COPEMENTION TESTS After a detailed history and physical examination, each patient must undergo a dosage of testosterone, estradiol, LH and HCG. Further testing should be ordered on the basis of history and physical examination. The presence of high levels of HCV involves a tumor of the testicles, which should be investigated initially by ultrasound. In the absence of testicular tumors, secretory extratesticular neoplasm should be excluded. Low testosterone levels with elevated LH and normal or high estradiol indicate hypergonadotrophic hypogonadism (primary). Low levels of testosterone and LH indicate hypogonadotrophic hypogonadism (secondary). The concomitantity of elevated testosterone, L.H. and estradiol suggests resistance to androgens. Hyperestrogenism with LH block is present in estradiol-secretor neoplasms (leidig or sertolia cell tumors and some adrenal tumors). In the case of adrenal tumors, levels of DHEA and DHEAS help diagnose. Patients with chronic renal failure have high levels of LN, FFG and prolactin, in addition to low testosterone. Breast neoplasia should be suspected in the presence of a solid, irregular, immobile, one-sided and eccentricly localized mass. We can also find a refutation of nipples, the selection of papillas and the conceivable lymph nodes of megamile. Mammography is the best test for assessing malignancies, while ultrasound distinguishes between breast and adipose tissue. Both tests should be considered complementary in the diagnosis of gynecomastia. Since adult gynecomastia is usually bilateral, patients over 35 years of age with unilateral gynecomastia must undergo a mammogram to rule out a breast cancer hypothesis. TREATMENT GYNECOMASTIA As mentioned, Gynecomastia's greatest value is the possibility that it is a sign of a serious illness. Gynecomastia as such does not require treatment unless it is a cause of embarrassment for the patient. Therapy in patients with gynecomastia is divided into etiological, clinical and surgical treatment. Etiological treatment of gynecomastia When gynecomastia has been present for more than a year, it practically does not regress spontaneously, even after treatment or control of the underlying disease, which is the result of the development of breast tissue fibrosis. Medications that cause Gynecomastia should, when possible, be suspended or exchanged for others that do not have this effect. Myalcies tumors, such as leidig and cell sertoly, and adrenal tumors should be treated surgically. In cases of germ cell tumors, chemotherapy is needed. Clinical treatment of gynecomastia There are three groups of drugs that can be used to treat gynecomastia: Androgens: make up testosterone, dihydrotestosterone and danazol. Treatment of Gynecomastia in hypogonad males with testosterone does not have good results when the condition is established, and may even aggravate Gynecomastia's height of estradiol secondary testosterone flavoring. Dihydrotestosterone may be an alternative because it is not flavored. Danazole is a weak androgen capable of suppressing the secretion of gonadotrophons, causing gynecomastia regression in some cases. Antiestrogens: Clomiphene citrate, tamoxifen and raloxifen are used. Some studies have shown a good improvement in Gynecomastia using Clomifene citrate, with a regression rate of up to 95%. The use of tamoxifen is associated with an improvement in the condition, however, with a low rate of complete regression. Despite little chance of full resolution and high relapse rates, tamoxifen is widely used for its low cost and several side effects. The dose should be 10 mg twice a day for at least three months. Aromatase inhibitors are letrozole, anastrozole, fadrosol and asemesan. There are several studies with this class of drugs in the treatment of Gynecomastia. Surgical treatment of adolescents Gynecomastia Pubescent, who did not respond to clinical treatment, in which Gynecomastia causes physical, psychological or social discomfort, should undergo surgical treatment. Surgery should also be considered in patients with macro-gynaessy as a lack of response to pharmacological treatment. When Gynecomastia has been present for more than two years, clinical treatment is also ineffective and surgical treatment is the only option. In patients with prostate carcinoma, who will undergo a deficiency of androgens as part of treatment, the radiation of the breast in low doses prevents the onset of gynecomastia. IMPORTANT TOPICS AND RECOMMENDATIONS Gynecomastia stems from the downtime between estrogenic and androgenic effects on male breast tissue. About 25% of patients with gynecomastia have no identifiable cause. Gynecomastia can be physiological in neonatal, puberty and senile periods. Estrogen-producing testicles can be trained with Gynecomastia. Increased aromatase activity, which can occur in obesity and hyperthyroidism, often curses Gynecomastia. Hypogonadism is a common cause of gynecomastia. Patients with gynecomastia without obvious cause with history and physical examination should undergo testosterone, estradiol, LH and HCG dosage. If the level of HCV is elevated, it is necessary to suspect a tumor of the testicles. Low testosterone levels with elevated LH suggest primary hypogonadism; if LH has diminished, suggest hypergonadotrophic hypogonadism. Breast neoplasia should be suspected in the presence of a solid, irregular, immobile, one-sided and eccentricly localized mass. Treatment of gynecomastia should ideally be directed to etiology. Medications for the treatment of gynecomastia include androgens, antistrogens and aromatase inhibitors. Consider surgical treatment for patients without an answer to clinical therapy, with great psychological or physical discomfort with Gynecomastia. 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