



FACULTY OF NURSING

Chapter-07

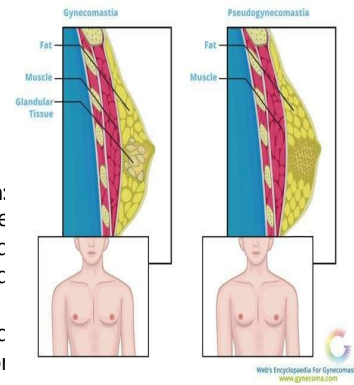


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Definition

► **Gynecomastia** is benign enlargement of the male breast caused by proliferation of glandular breast tissue.

► **Pseudogynecomastia:** Enlargement of the male breast, as a result of increased fat deposition is called Pseudogynecomastia. synonymous terms are used like **Adipomastia**, or **lipomastia**.

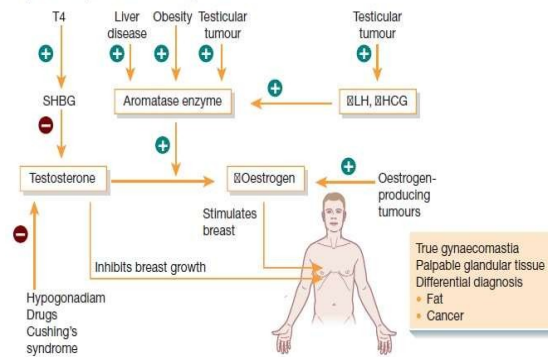


Pathophysiology

- ▶ Gynecomastia results from an imbalance between the stimulatory effect of estrogen on ductal proliferation and the inhibitory effect of androgen on breast development.
- ▶ The imbalance is most commonly caused by increased production of estrogens, decreased production of testosterone, or increased conversion of androgens to estrogens in peripheral tissue.
- ▶ Disorders of sex hormone-binding globulin or with androgen receptor binding and function can also result in gynecomastia.

Hormonal influences on Gynecomastia:

Figure 29.1 (a) Gynaecomastia. Source: Davey P (ed.) (2014) Medicine at a Glance, 4th edn. Reproduced with permission of John Wiley & Sons Ltd

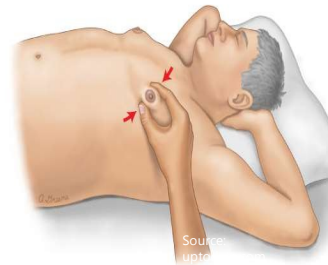
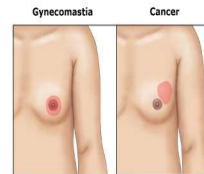


Clinical manifestation

- ▶ Gynecomastia usually manifests as a palpable, discrete button of tissue radiating from beneath the nipple and areola.
- ▶ Gynecomastia feels “gritty” when the breast is pinched between the thumb and forefinger.
- ▶ Fatty tissue (Pseudogynecomastia), unlike gynecomastia, will not cause resistance until the nipple is reached. (Difference in clinical examination).

Examination Findings:

- ▶ The examination is performed by having the patient lie on his back with his hands behind his head. The examiner then places his or her thumb and forefinger on each side of the breast and slowly brings them together
- ▶ Gynecomastia is appreciated as a concentric, rubbery-to-firm disk of tissue, often mobile, located directly beneath the areolar area.
- ▶ Pseudogynecomastia presents no discrete mass,
- ▶ Other masses due to disorders such as cancer tend to be eccentrically positioned (insert)



Prevalence

- ▶ **Asymptomatic palpable breast tissue is common in normal males, particularly in the neonate (60%–90%), at puberty (60%–70%), and with increasing age (20%–65%, > 50 years).**
- ▶ **Because of this high prevalence, gynecomastia is considered a relatively normal finding during these periods of life. Gynecomastia is often called physiologic at these ages.**

Physiological gynecomastia in different age group

- ▶ Neonatal gynecomastia results from placental transfer of estrogens.
- ▶ During early puberty, production of estrogens begins sooner than testosterone production, thus causing an imbalance in the ratio of estrogens to androgens.
- ▶ With aging, testosterone production decreases, and peripheral androgen to estrogen conversion often increases because of an age-related increase in adipose tissue. Drugs and medical conditions may also contribute.

Causes of Gynecomastia

- ▶ Persistent pubertal gynecomastia – 25 percent
 - ▶ Drugs – 10 to 25 percent, Common drugs includes *Spirolactone, Cimetidine, Ketoconazole, Estrogens and estrogen agonists, human chorionic gonadotropin (hCG), antiandrogens, gonadotropin-releasing hormone (GnRH) agonists, and 5-alpha-reductase inhibitors.*
 - ▶ **No detectable abnormality (idiopathic) – 25 percent.**
 - ▶ **Cirrhosis or malnutrition – 8 percent.**
 - ▶ **Hypogonadism – Primary (8 percent), secondary (2 percent).**
 - ▶ **Testicular tumors – 3 percent**
 - ▶ **Hyperthyroidism – 1.5 percent**
 - ▶ **Chronic renal insufficiency – 1 percent.**

Classification

Simon Classified gynaecomastia into the following groups:

- **Stage-1: A slight volume increase with no excess skin.**
- **Stage-2a: A moderate volume increase with no excess skin.**
- **Stage-2b: A moderate volume increase with excess skin.**
- **Stage-3: A marked volume increase with excess skin**

Histopathological Classification

- ▶ **Three histologic patterns of gynecomastia have been recognized.**
 - ▶ **Florid pattern:** consists of an increase in the number of budding ducts, proliferation of the ductal epithelium.
 - ▶ **Fibrous type:** has dilated ducts, minimal duct epithelial proliferation, ductal edema, and a virtually acellular fibrous stroma.
 - ▶ An **intermediate pattern** contains features of both types.
- ▶ The duration of gynecomastia is the most important factor in determining the pathologic picture.
- ▶ Florid pattern response well to medical treatment, while fibrous type needs surgical management.

Questions to be solved

- ▶ **Gynecomastia / Pseudogynecomastia/Breast Cancer?**
- ▶ **Characteristics: Unilateral / Bilateral, Tenderness, Size, progression**
- ▶ **Physiological / Pathological**
- ▶ **Possible cause of gynecomastia, eg: Systemic cause, Drugs**
- ▶ **Any underlying hormonal derangement, eg: Hypogonadism**
- ▶ **Associated malignancy, eg: hCG-secreting testicular tumor.**
- ▶ **Management**

Evaluation:

HISTORY:

- > **Age**
- > **Duration** of the gynaecomastia
- > Presence of **pain**.
- > Use of **drugs** including recreation drugs e.g. Alcohol
- > Presence of symptoms suggestive of **pathological cause**.
 - Symptome of **hypogonadism** - reduced libido, erectile dysfunction.
 - **Systemic disease**: Hepatic, Renal, Endocrine disease. (Thyrotoxicosis, Cushing syndrome).

Evaluation: (Cont.)

Physical examination:

□ Breasts:

➤ Pinch breast tissue between thumb and forefinger—distinguish from

• fat. (Method Previously Described)

➤ Measure glandular tissue diameter. Look for galactorrhea.

□ Testicular palpation:

□ 2° sex characteristics.

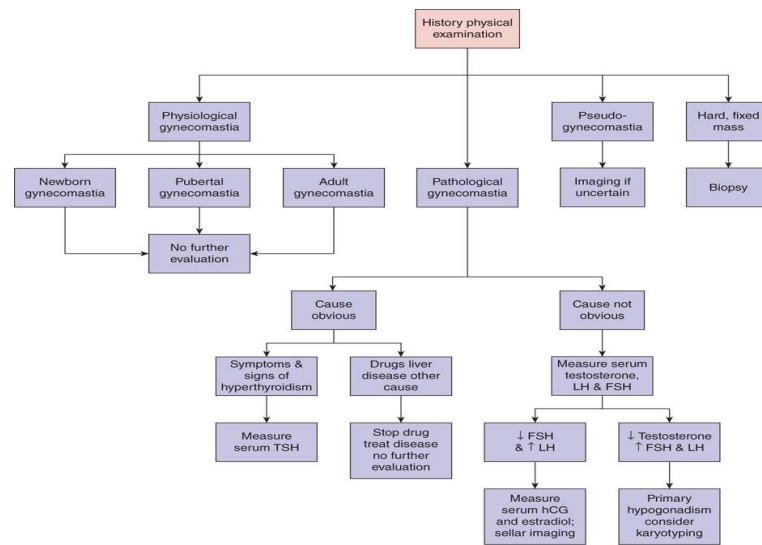
□ Look for evidence of **systemic disease** e.g. chronic liver or renal disease,

Baseline investigations

- ▶ Serum testosterone.
- ▶ Serum estradiol.
- ▶ LH and FSH.
- ▶ Prolactin.
- ▶ SHBG.
- ▶ hCG.
- ▶ Liver function tests.

Additional investigations

- ▶ If testicular tumour is suspected, e.g. raised estradiol/hCG: testicular USG.
- ▶ If adrenal tumour is suspected, e.g. markedly raised estradiol: abdominal CT or MRI scan.
- ▶ If breast malignancy is suspected: mammography; FNAC/tissue biopsy.
- ▶ If lung cancer is suspected, e.g. raised hCG: chest radiograph.
- ▶ Other investigations, depending on clinical suspicion, e.g. *renal* or *thyroid* function.



Source: David G. Gardner, Dolores Shoback: Greenspan's Basic & Clinical Endocrinology, Tenth Edition Copyright © McGraw-Hill Education. All rights reserved.

Management

Medical Treatment:

- ▶ The underlying disease should be corrected if possible, and offending drugs should be discontinued.
- ▶ ER antagonists (tamoxifen, 10 to 20 mg daily, or raloxifene, 60 mg daily) are effective in treating pubertal and adult gynecomastia and preventing gynecomastia induced by androgen deprivation therapy.
- ▶ Other drugs can be used, such as Clomifene (50–100mg/day), Danazol (300– 600mg/day), Testolactone (450mg/day), Anastrozole (1mg/day).
- ▶ Medical therapy is ineffective for chronic, fibrous gynecomastia.

Management (Cont.)

Surgery:

- **With long standing gynecomastia (more than 1 year): surgical reduction mammoplasty (i.e. removal of breast tissue with or without periareolar adipose tissue) is necessary if breast enlargement is severe, painful, socially embarrassing or disfiguring.**

Management (Cont.)

Radiation therapy

- ▶ Low-dosage external beam radiation therapy (900 cGy or less) is effective, but less effective than tamoxifen, for prevention of **gynecomastia** due to anti-androgen monotherapy for prostate cancer.

Course and Prognosis

- ▶ **Pubertal gynecomastia usually regresses spontaneously within 1 or 2 years.**
- ▶ **Patients who develop drug-induced gynecomastia generally have complete or near-complete regression of the breast changes if the offending drug is discontinued during the early, florid stage.**
- ▶ **Once gynecomastia from any cause has reached the fibrotic stage, little or no spontaneous regression occurs, and medical therapy is ineffective.**

Thank
YOU