



## **Gynecomastia**

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## Introduction

Gynecomastia is a common clinical condition characterized by the benign proliferation of male breast glandular tissue. It is essential for healthcare providers to understand its etiology, pathophysiology, and management, particularly in patients with cancer, where it may complicate the clinical picture.

## Definition and Epidemiology

Gynecomastia is defined as the enlargement of male breast (PHOTO 1) tissue due to an increase in the glandular component. It is distinct from pseudogynecomastia, which is characterized by fat deposition without glandular proliferation. Gynecomastia affects 32-65% of men at some point in their lives, with peaks during neonatal, pubertal, and older adult periods [1].

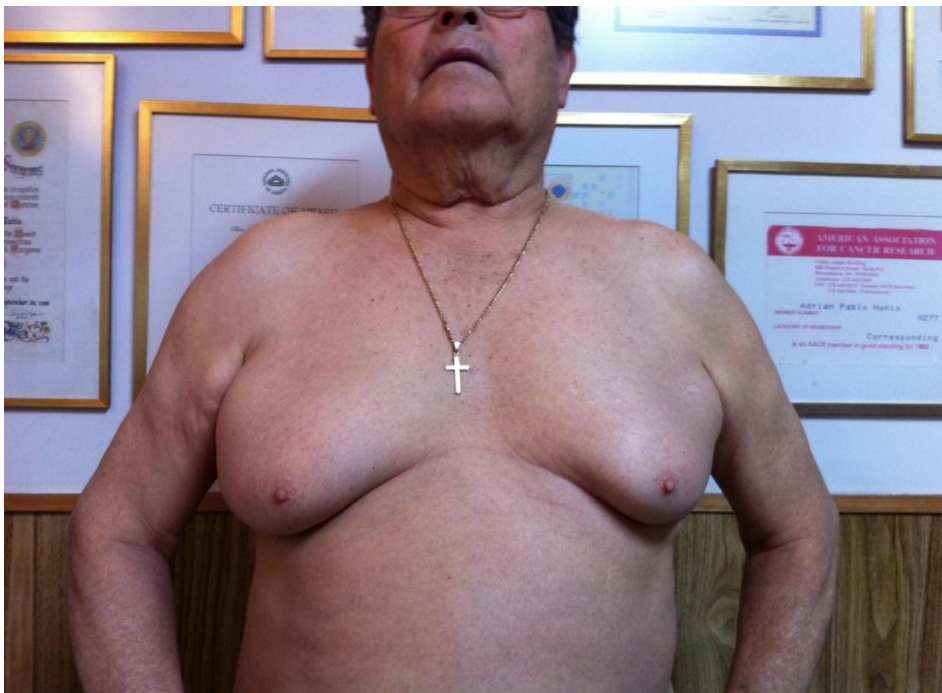


Photo 1

## Gynecomastia in Cancer Patients

In oncology, gynecomastia can be a significant concern due to:

1. **Hormonal Therapies:** Treatments such as androgen deprivation therapy (ADT) in prostate cancer can lead to hormonal imbalances, increasing estrogenic activity relative to androgens [2].
2. **Chemotherapy:** Some chemotherapeutic agents can disrupt endocrine function, leading to gynecomastia.
3. **Syndromes:** Rarely, tumors may secrete hormones or hormone-like substances that induce breast tissue proliferation.

## **Etiology**

The causes of gynecomastia are multifactorial:

1. **Physiological:** Occurs in newborns due to maternal estrogen, in adolescents due to hormonal changes, and in the elderly due to decreased testosterone production.
2. **Pharmacological:** Drugs such as spironolactone, cimetidine, and certain antipsychotics have been implicated in gynecomastia development [3].
3. **Pathological:** Conditions such as hyperthyroidism, chronic liver disease, and testicular tumors can alter the estrogen-androgen balance.

## **Pathophysiology**

The pathophysiological basis of gynecomastia involves an increase in the estrogen-to-androgen ratio, which stimulates ductal epithelial hyperplasia and stromal proliferation in breast tissue. This imbalance can result from increased aromatase activity, leading to higher estrogen levels, or from decreased androgen production or action [4].

## **Clinical Evaluation**

A thorough clinical evaluation is crucial:

1. **History:** Assess for drug use, systemic diseases, and duration of breast enlargement.
2. **Physical Examination:** Differentiate between true gynecomastia and pseudogynecomastia. Evaluate for signs of malignancy or systemic disease.
3. **Laboratory Tests:** Measure serum testosterone, estradiol, luteinizing hormone (LH), and follicle-

stimulating hormone (FSH) levels. Consider liver and renal function tests.

4. Imaging: Ultrasound or mammography may be used to evaluate breast tissue characteristics and exclude malignancy.

## **Management**

The approach to managing gynecomastia involves:

1. Reassurance and Observation: In cases where gynecomastia is mild and asymptomatic, especially in adolescents, reassurance and periodic monitoring may suffice [5].

2. Medical Treatment:

- Selective Estrogen Receptor Modulators (SERMs): Tamoxifen and raloxifene can be effective in reducing breast volume.

- Aromatase Inhibitors: Anastrozole may be used to decrease estrogen production.

3. Surgical Intervention: Indicated for persistent, painful, or psychologically distressing gynecomastia. Techniques include liposuction and subcutaneous mastectomy.

## **Conclusion**

Gynecomastia, while often benign, can have significant implications in cancer patients due to its potential impact on quality of life and treatment adherence. A comprehensive understanding of its causes, pathophysiology, and management options is essential for optimizing patient care.

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