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Male Breast Cancer Initially Misdiagnosed as Gynecomastia: A Case Report of Delayed Diagnosis in a 62-Year-Old Man

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Abstract

Male breast cancer is a rare malignancy, accounting for approximately 1% of all breast cancer cases; however, its rarity frequently contributes to delayed diagnosis. Gynecomastia, a benign proliferation of male breast glandular tissue, may mimic early-stage male breast cancer and represents a significant diagnostic challenge. This case report describes a 62-year-old man with a right subareolar breast mass that was initially diagnosed as gynecomastia based solely on clinical assessment. Conservative management was pursued until progressive enlargement and nipple retraction prompted surgical referral. Core needle biopsy confirmed invasive ductal carcinoma, not otherwise specified (NOS), Grade II, with pathological staging of pT2N1M0 (Stage IIB). The patient underwent modified radical mastectomy followed by adjuvant chemotherapy and hormonal therapy. This case highlights that male breast cancer predominantly presents as invasive ductal carcinoma (85–95%), with hormone receptor positivity observed in more than 90% of cases. Clinical red flags can help distinguish male breast cancer from gynecomastia when systematically assessed, while core needle biopsy remains the diagnostic standard. This case underscores the critical importance of tissue biopsy in male patients presenting with a unilateral, firm, or eccentric breast mass, regardless of initial clinical suspicion. Early histopathological evaluation is essential to minimize diagnostic errors and optimize patient outcomes.

INTRODUCTION

Male breast cancer is a rare malignancy, accounting for approximately 1% of all breast cancer cases (Mukherjee et al., 2023); however, its rarity frequently contributes to delayed diagnosis (Koseci et al., 2021). Gynecomastia, a benign proliferation of male breast glandular tissue, may mimic early-stage male breast cancer and represents a significant diagnostic challenge (Faridi et al., 2025). This case report describes a 62-year-old man with a right subareolar breast mass that was initially diagnosed as gynecomastia based solely on clinical assessment (Lee et al., 2026). Conservative management was pursued until progressive enlargement and nipple retraction prompted surgical referral (Almass et al., 2025). Core needle biopsy confirmed invasive ductal carcinoma, not otherwise specified (NOS), Grade II (Khattab et al., 2024), with pathological staging of pT2N1M0 (Stage IIB) (Luo et al., 2025). The patient underwent modified radical mastectomy followed by adjuvant chemotherapy and hormonal therapy (Straccia & Rossi, 2025). This case highlights that male breast cancer predominantly presents as invasive ductal carcinoma (85–95%) (El Fouhi et al., 2020), with hormone receptor positivity observed in more than 90% of cases (Khattab et al., 2024). Clinical red flags can help distinguish male breast cancer from gynecomastia when systematically assessed, while core needle biopsy remains the diagnostic standard (Faridi et al., 2025). This case underscores the

critical importance of tissue biopsy in male patients presenting with a unilateral, firm, or eccentric breast mass, regardless of initial clinical suspicion (Eric & Niba, 2025). Early histopathological evaluation is essential to minimize diagnostic errors and optimize patient outcomes (Aljehani et al. 2023; Bera et al. 2019; Farahani et al. 2022; Niriella et al. 2024; Rastogi 2018).

CASE PRESENTATION

Patient History

A 62-year-old Indonesian male presented to the surgical outpatient clinic with a chief complaint of a progressively enlarging right breast mass. The patient reported onset of swelling approximately six months prior, initially noted as a painless lump beneath the right nipple-areolar complex. He had sought evaluation at a primary healthcare center approximately four months before the current visit, where he was informed — without tissue sampling — that the mass was consistent with gynecomastia and was instructed to observe without intervention.

Over the subsequent two months, the mass grew in size, became mildly tender on palpation, and the patient observed progressive nipple retraction. No axillary masses were noted by the patient at that time. He denied skin changes, fever, or discharge. There was no prior history of breast or prostate malignancy. Family history revealed that a paternal uncle had undergone treatment for an unspecified malignancy. The patient denied use of hormonal therapy, anabolic steroids, or medications known to cause gynecomastia (e.g., spironolactone, cimetidine, digoxin). No occupational radiation or toxin exposure was documented.

Physical Examination

Table 1. Summary of physical examination findings on initial surgical consultation.

Parameter	Findings
Vital Signs	BP 140/88 mmHg, HR 82 bpm, RR 18/min, T 36.6°C
Right Breast	4.5 × 3.8 cm firm-to-hard, irregular, non-mobile subareolar mass; nipple retraction present; overlying skin intact (no peau d'orange)
Left Breast	Symmetrical, no palpable mass
Left Axilla	No palpable lymphadenopathy
Abdomen	No hepatomegaly, splenomegaly, or ascites

Source: Physical examination findings documented at initial surgical consultation, Faculty of Medicine, University of Nusa Cendana (2020).

Gross examination and Histopathological Findings

Gross examination of the right breast mass was performed. Gross examination revealed mass with pale-gray firm tissue, approximately 3.8 cm in length.

Figure 1. Gross Examination of Right Breast Mass
Source: Gross examination findings documented at the time of core needle biopsy, Faculty of Medicine, University of Nusa Cendana (2020)

Microscopic examination: Sections revealed infiltrating nests and cords of atypical epithelial cells arranged in tubular patttern 60% and solid patterns within a desmoplastic stroma.

Tumor cells exhibited moderate nuclear pleomorphism, conspicuous nucleoli, and frequent mitotic figures (20/10 HPF). No ductal carcinoma in situ (DCIS) component was identified in the biopsy cores. No evidence of angiolymphatic invasion was noted in the sampled tissue.

Histopathological diagnosis: Invasive Ductal Carcinoma (IDC), Not Otherwise Specified (NOS), Nottingham Grade II (Tubule score 2, Nuclear score 2, Mitosis score 2; Total score 6/9).

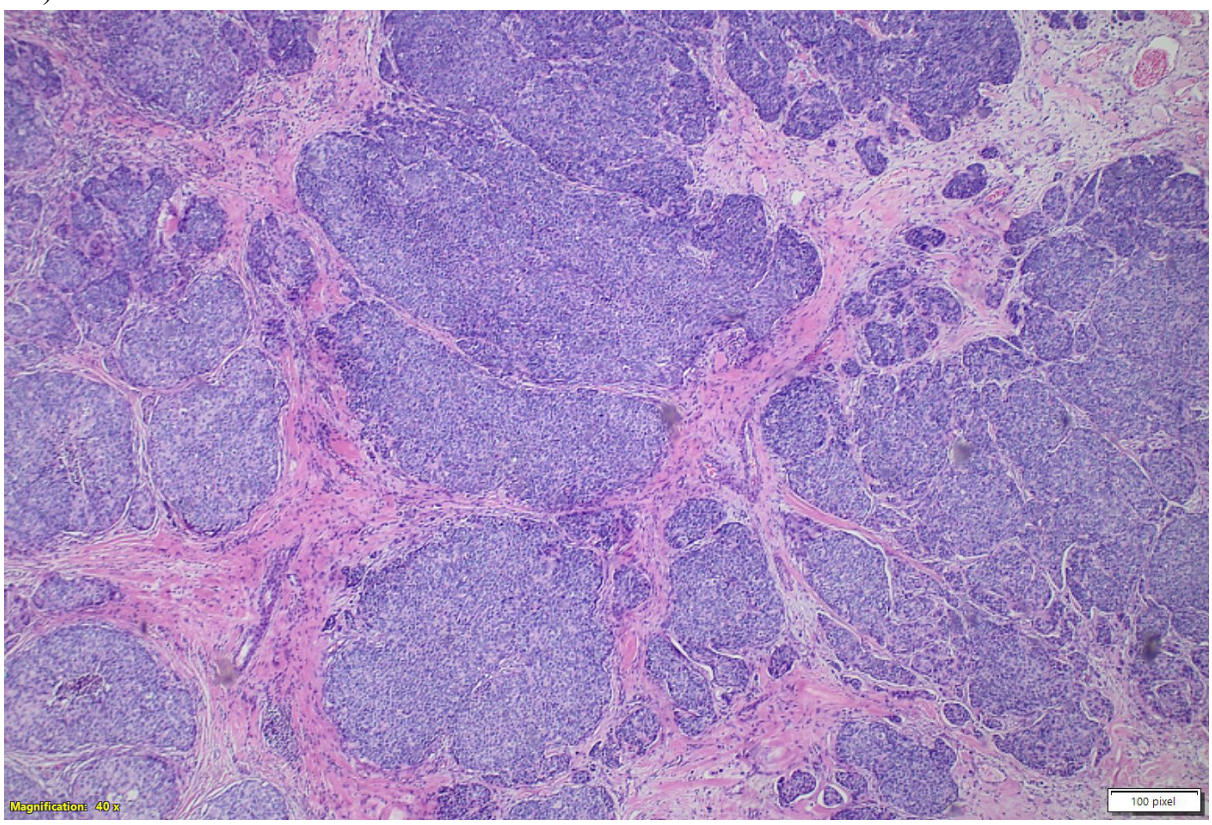

Figure 2. Histopathological Examination at 40x magnification

Source: Histopathological image from core needle biopsy specimen, Faculty of Medicine, University of Nusa Cendana (2020)

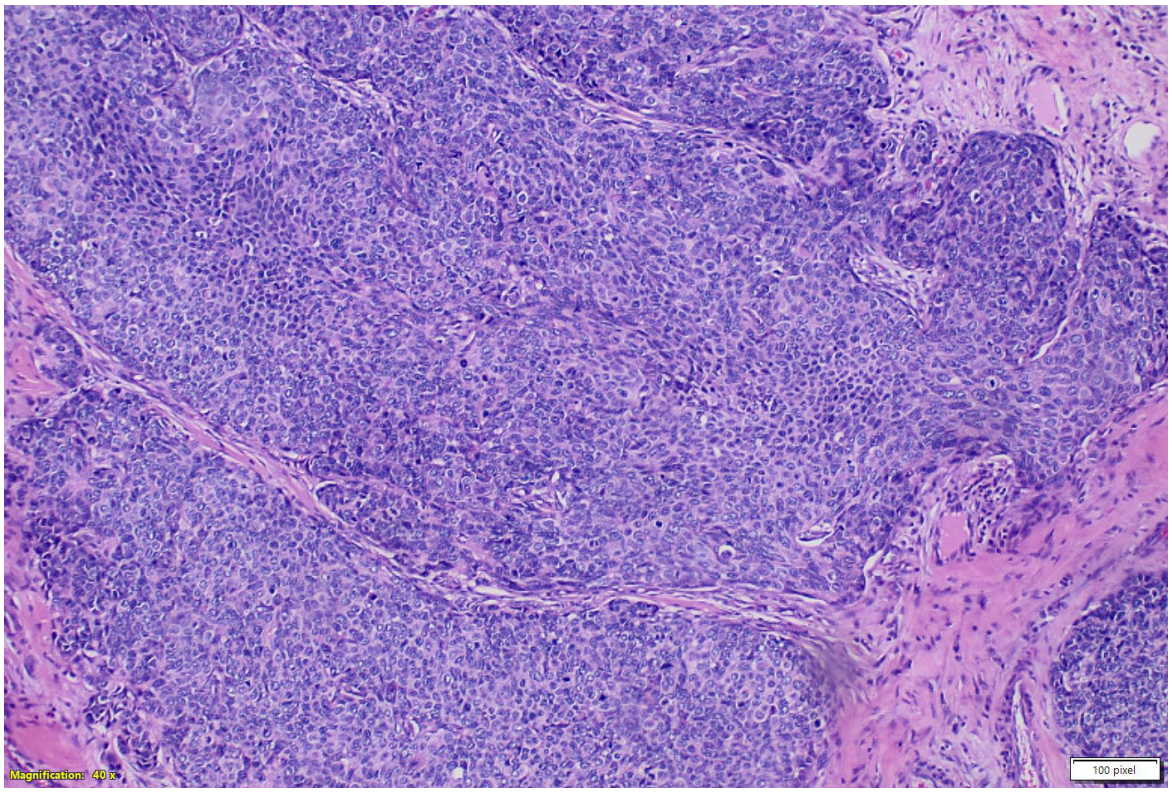

Figure 3. Histopathological Examination at100x magnification

Source: Histopathological image from core needle biopsy specimen, Faculty of Medicine, University of Nusa Cendana (2020).

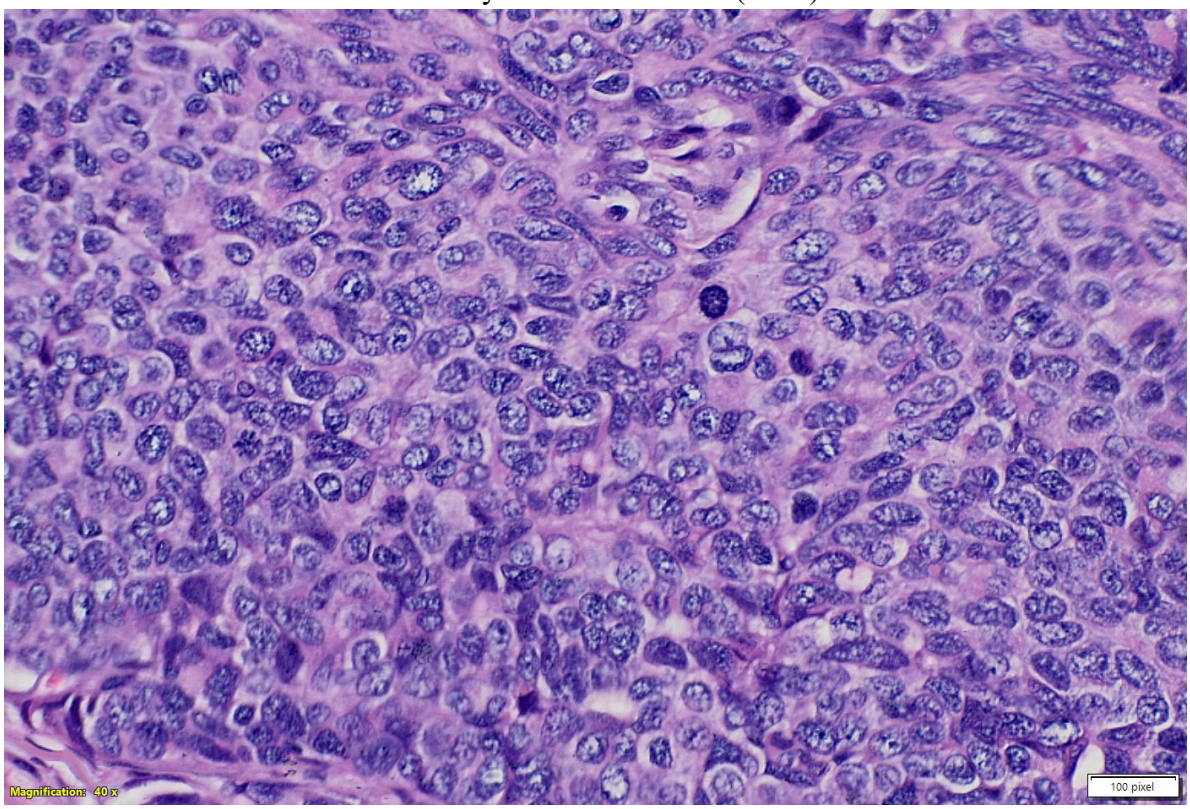

Figure 4. Histopathological Examination at 400x magnification

Source: Histopathological image from core needle biopsy specimen, Faculty of Medicine, University of Nusa Cendana (2020).

Staging and Final Diagnosis

Chest X-ray and abdominal ultrasound revealed no distant metastasis. TNM Clinical Staging (AJCC 8th edition): cT2N1M0, Stage IIB.

Final Pathological Diagnosis
Invasive Ductal Carcinoma (IDC), NOS — Right Breast
Nottingham Grade II, pT2N1M0 (Stage IIB)

Management

Following multidisciplinary team (MDT) discussion, the patient underwent modified radical mastectomy (MRM) of the right breast

The patient planned to received 6 cycles of adjuvant chemotherapy (Cyclophosphamide, Doxorubicin, Fluorouracil — CAF regimen), followed by tamoxifen for 5 years given the premenopausal-equivalent hormonal milieu. Planned to get radiation therapy to the chest wall and regional lymphatics was administered (50 Gy in 25 fractions). The patient is currently under 6-monthly follow-up with no evidence of locoregional recurrence at 12 months post-surgery.

RESULTS AND DISCUSSION

Epidemiology: Incidence, Age Distribution, and Survival

Global MBC incidence data from GLOBOCAN 2020 estimated approximately 3,990 new MBC cases in the United States and 41,900 worldwide in 2020, representing 0.5% of global breast cancer and 0.6% of all male malignancies (Sung et al., 2021). Age-adjusted incidence in high-income countries is approximately 1.0–1.5 per 100,000 males per year, with a rising trend over the past three decades attributed to increasing male obesity rates, prolonged male life expectancy, and improved case ascertainment in national registries.

The median age at MBC diagnosis is 67 years — 5–10 years older than in female breast cancer patients — with the 60–80-year age group accounting for approximately 60% of all cases. This older age at presentation, combined with the higher prevalence of cardiovascular comorbidities in this age group, compounds the clinical complexity of MBC management and increases the likelihood that breast symptoms will be misattributed to age-related benign conditions, including gynecomastia.

Survival data from SEER demonstrate 5-year relative survival rates of approximately 95% (Stage I), 72% (Stage II), 53% (Stage III), and 25% (Stage IV). Stage-for-stage comparison with female breast cancer reveals broadly similar survival, refuting the historically held belief that MBC is intrinsically more aggressive. The inferior overall MBC survival in population-level data is therefore predominantly a product of advanced stage at diagnosis — itself the direct consequence of diagnostic delay — rather than inherent biological aggressiveness (Giordano, 2018). This epidemiological reality underscores the paramount importance of reducing the diagnostic delay interval.

Risk Factors and Etiopathogenesis

The pathogenesis of MBC is fundamentally driven by a relative estrogenic excess within the male breast microenvironment, arising from conditions that either increase estrogen availability, decrease androgen levels, or enhance sensitivity of breast epithelium to estrogenic stimulation. This hormonal imbalance also underlies the pathogenesis of gynecomastia,

creating a shared hormonal substrate between the two conditions — a fact that further complicates clinical differentiation but does not imply a shared oncological risk trajectory.

Table 3. Established risk factors for male breast cancer with relative risk estimates and pathogenic mechanisms (Cardoso et al., 2018; Gucalp et al., 2019).

Risk Factor	Relative Risk	Mechanism and Clinical Notes
BRCA2 germline mutation	~80×	Lifetime MBC risk 6–8%; most penetrant genetic risk factor; 10–15% of MBC cases
Klinefelter syndrome (47,XXY)	~50×	Reduced testosterone + relative hyperestrogenism; also strong gynecomastia association
BRCA1 mutation	~20×	Less penetrant than BRCA2 for MBC; germline testing indicated for all MBC patients
Hepatic cirrhosis	~5×	Impaired estrogen inactivation; major contributor in Sub-Saharan Africa where MBC rates are highest
Obesity (BMI >30 kg/m²)	~2–3×	Peripheral aromatization of androgens to estradiol in adipose tissue; index case BMI 29.4 kg/m²
Chest/thoracic irradiation	~2.5–5×	Dose-dependent; Hodgkin lymphoma survivors at particular risk
Exogenous estrogen / anti-androgens	~2–5×	Transgender females (MtF); prostate cancer androgen deprivation therapy
Family history (first-degree)	~2–3×	Amplified risk in BRCA2-positive families; warrant genetic counselling referral
PALB2 / CHEK2 mutations	~3–5×	Emerging evidence; comprehensive panel testing increasingly recommended

Source: Compiled from systematic clinical and radiological differentiation guideline (Giordano, 2018; Fentiman, 2018; Cardoso et al., 2018; Johnson & Murad, 2009).

Among genetic risk factors, BRCA2 pathogenic variants command particular clinical attention. Approximately 10–15% of all MBC cases carry germline BRCA2 mutations — a prevalence threefold to fivefold higher than in female breast cancer (approximately 3–5%). The penetrance of BRCA2 mutations for male breast cancer is substantially higher than for BRCA1, making BRCA2 testing the highest-yield genetic investigation in any male breast cancer patient. BRCA2-associated MBC tends to present at a younger age than sporadic MBC, with higher histological grade and a propensity for greater genomic instability. Consistent with the near-universal hormone receptor-positive phenotype of MBC, BRCA2-associated tumours are predominantly ER-positive — a finding with direct implications for endocrine therapy eligibility (Cardoso et al., 2018).

Pathophysiology: Gynecomastia and Male Breast Cancer Compared

1. Normal Male Breast Anatomy

The adult male breast consists predominantly of adipose tissue with rudimentary ductal structures — elongated, simple tubules lined by one to two layers of cuboidal epithelial cells, embedded in a fibrous stroma. Under normal androgenic conditions, the male breast does not develop terminal duct-lobular units (TDLUs), acini, or secretory lobules — the epithelial compartments that constitute the functional unit of the female breast and the site of origin of lobular carcinoma and most ductal carcinomas in women. This fundamental anatomical difference explains both the rarity of lobular carcinoma in males (representing <2% of MBC

versus 10–15% in females) and the near-exclusive dominance of IDC NOS among MBC histological subtypes (WHO, 2022).

2. Pathophysiology of Gynecomastia

Gynecomastia arises from an imbalance between estrogenic stimulation and androgenic inhibition of the ductal epithelium, leading to ductal elongation, branching, and periductal fibrovascular proliferation without lobular formation. The histological progression is temporally dependent: (1) the florid phase (<4 months of onset) is characterised by active ductal epithelial hyperplasia, micropapillary projections, and cellular periductal stroma with increased vascularity — features that can superficially resemble neoplastic proliferation on cursory examination; and (2) the fibrous phase (>12 months) demonstrates dilated ducts lined by flat, atrophic epithelium within dense, paucicellular fibrous stroma with no active proliferation. Neither phase exhibits cytological atypia, invasion, necrosis, or angiolymphatic permeation — the features that define malignancy.

Three physiological age peaks for gynecomastia are recognised: neonatal (transplacental estrogen exposure), pubertal (transient testosterone:estradiol imbalance), and older adult (declining testosterone with preserved peripheral aromatization in adipose tissue). Pathological gynecomastia in middle-aged and older males is commonly drug-induced (spironolactone, cimetidine, digoxin, finasteride, proton pump inhibitors, anabolic steroids, anti-androgens) or secondary to systemic disease (cirrhosis, renal failure, hypogonadism, adrenal or testicular tumours producing estrogen) (Johnson & Murad, 2009).

3. Molecular Pathogenesis of MBC

MBC is predominantly a Luminal B-type cancer by TCGA molecular classification, characterised by ER/PR positivity, HER2 negativity, and elevated Ki-67. Key oncogenic alterations include: activating mutations in PIK3CA (20–30%), GATA3 (12–18%), and MAP3K1 (~10%); cyclin D1 (CCND1) amplification driving G1/S cell cycle progression; and PTEN loss with consequent hyperactivation of the PI3K/AKT/mTOR pathway. TP53 mutations, identified in approximately 15–20% of MBC cases, correlate with higher histological grade and poorer prognosis. BRCA2-related MBC demonstrates a distinct genomic landscape featuring large-scale chromosomal instability, higher mutational burden, and HR-deficiency signatures that may eventually inform PARP inhibitor eligibility (Cardoso et al., 2018; Shaaban et al., 2012).

Clinical and Radiological Differentiation: Gynecomastia vs. MBC

The clinical distinction between gynecomastia and MBC is achievable through a structured, systematic examination incorporating the features outlined in Table 4. No single feature is pathognomonic; clinical certainty requires the composite assessment of multiple parameters, and any suspicious composite mandates tissue biopsy regardless of the pre-test probability assigned.

Table 4. Systematic clinical and radiological differentiation between gynecomastia and male breast cancer.

Clinical Parameter	Gynecomastia	Male Breast Cancer
Laterality	Bilateral 70–80% of cases	Unilateral in >95% of cases
Location	Concentric, central subareolar; symmetric	May be eccentric; retroareolar or quadrant
Consistency	Rubbery-soft to firm; disc-like	Firm-to-hard; nodular
Margins	Smooth, well-defined, circumscribed	Irregular, poorly defined, spiculated
Skin adherence / mobility	Fully mobile; no skin tethering	May be adherent to skin or pectoralis
Nipple-areolar complex	Normal projection; no retraction	Nipple retraction, inversion, bloody discharge
Skin changes	Absent	Peau d'orange, dimpling, ulceration (advanced)
Axillary lymph nodes	Not palpable	Firm, enlarged; absent fatty hilum on USS
Tenderness	Common in florid phase	Variable; often painless at onset
Clinical course	May fluctuate; often bilateral and symmetric	Progressive, unrelenting, unilateral growth
Mammography	Flame-shaped density; BI-RADS 1–2	Spiculated mass; microcalcifications; BI-RADS 4–5
Ultrasonography	Fan-shaped periareolar hypoechoic tissue; regular margins	Irregular hypoechoic mass; angular margins; posterior shadowing; abnormal axillary node

Source: Adapted from WHO Classification of Tumours: Breast Tumours, 5th Edition (2022) and Shaaban et al. (2012).

Mammography in male patients achieves sensitivity of approximately 92% and specificity of 90% for malignancy detection, outperforming clinical examination alone. The lower background parenchymal density in the male breast compared to the female renders small masses conspicuous on standard two-view mammography. Ultrasound complements mammography by characterising mass vascularity, interrogating axillary lymph nodes, and guiding percutaneous biopsy. Any BI-RADS ≥4 finding in a male patient mandates tissue sampling without exception.

Histopathological Diagnosis of MBC

1. Histological Subtypes and Frequencies

The histological classification of MBC follows the WHO Classification of Tumours: Breast Tumours, 5th Edition (2022), applied identically to male and female breast carcinoma. The predominance of IDC NOS in males is a direct consequence of the absence of TDLUs, from which lobular carcinoma originates. Table 5 summarises the histological subtype distribution in MBC.

Table 5. Histological subtypes of male breast cancer and approximate frequencies (WHO, 2022).

Histological Subtype	MBC Frequency	Key Histological Features
IDC NOS (Invasive Ductal Carcinoma, Not Otherwise Specified)	**85–95%**	Infiltrating cords/nests/sheets; desmoplastic stroma; absent lobular architecture; dominant subtype due to absence of TDLUs
Invasive Papillary Carcinoma (incl. Encapsulated Papillary Ca.)	2–5%	Papillary fronds with fibrovascular cores; well-circumscribed; more common in elderly males
Mucinous (Colloid) Carcinoma	1–3%	Tumour cells floating in extracellular mucin pools; favourable prognosis
Invasive Lobular Carcinoma	<2%	Rare; E-cadherin negative; paucity of TDLUs in male breast explains rarity
DCIS (pure or as IDC component)	5–10% as pure; common as IDC component	Papillary and cribriform subtypes predominant in males

Source: Adapted from WHO Classification of Tumours: Breast Tumours, 5th Edition (2022) and Shaaban et al. (2012)

2. Nottingham Histological Grading System

The Nottingham Grading System — a standardised, internationally validated framework for breast carcinoma grade assessment — applies identically to MBC as to female breast carcinoma. Its three parameters (tubule formation, nuclear pleomorphism, mitotic count) are scored 1–3 each, yielding a composite score of 3–9 and a corresponding histological grade (Grade I: 3–5; Grade II: 6–7; Grade III: 8–9). The clinical relevance of the Nottingham Grade in MBC is substantial: it provides an independent prognostic signal, informs chemotherapy decision-making, and correlates with molecular subtype.

Table 6. Nottingham Grading System: scoring criteria, grade thresholds, and clinical significance (HPF = high-power field at ×400, FN22 field area = 0.55 mm²).

Parameter	Score	Criterion and Histological Description
Tubule Formation	1	>75% of tumour area shows open tubular/glandular structures with distinct lumina
	2	10–75% tubular/glandular structures; mixed solid and glandular growth
	3	<10% tubules; predominantly solid sheets, cords, or trabeculae — no glandular differentiation
Nuclear Pleomorphism	1	Small, uniform nuclei; minimal variation in size; inconspicuous nucleoli
	2	Moderate nuclear enlargement; irregular contours; conspicuous nucleoli; moderate internuclear variation
	3	Marked nuclear enlargement; prominent pleomorphism; large nucleoli; vesicular chromatin; atypical mitoses
Mitotic Count (×400, FN22)	1	0–5 mitoses per 10 HPF
	2	6–10 mitoses per 10 HPF
	3	>10 mitoses per 10 HPF; atypical (multipolar) mitoses may be present

GRADE	**I**	Score 3–5: well-differentiated; low proliferative activity; best prognosis
	II	Score 6–7: moderately differentiated; intermediate behaviour; responsive to treatment
	III	Score 8–9: poorly differentiated; high proliferative activity; worst prognosis

Source: Adapted from established diagnostic guidelines for male breast cancer

In published MBC cohorts, Grade II tumours predominate (approximately 45–55%), followed by Grade III (30–35%) and Grade I (~15–20%). This grade distribution is consistent with the near-universal Luminal B molecular phenotype of MBC, which is histologically associated with higher proliferative activity than Luminal A tumours and carries an intermediate-to-high risk of recurrence without adjuvant systemic therapy. The mitotic count component of the Nottingham grade correlates most strongly with Ki-67 proliferative index and directly informs the decision to administer chemotherapy in borderline cases (Shaaban et al., 2012).

3. Comparative Immunohistochemical Receptor Profile

Complete MBC characterisation includes immunohistochemical receptor profiling — ER, PR, HER2, and Ki-67 — which defines the molecular subtype and guides systemic therapy selection. The receptor profile of MBC differs quantitatively but not qualitatively from female breast cancer: hormone receptor positivity is substantially higher in males, while triple-negative phenotype is correspondingly rarer.

Table 7. Comparative immunohistochemical receptor profile of male versus female breast cancer

Marker	MBC	Female Breast Ca.	Implication
ER (Estrogen Receptor)	88–95%	70–75%	Endocrine therapy eligibility
PR (Progesterone Receptor)	78–90%	65–70%	Luminal phenotype
HER2 (IHC/FISH amplified)	10–15%	15–20%	Trastuzumab eligibility
Triple Negative (TNBC)	<5%	15–20%	Poor prognosis; chemo-based
AR (Androgen Receptor)	50–95%	70–90%	Emerging therapeutic target
Ki-67 proliferation index (median)	25–35%	20–30%	Grade/subtype; chemo decision

Source: Compiled from immunohistochemical profiling studies (Cardoso et al., 2018; Shaaban et al., 2012).

Androgen receptor (AR) positivity, present in 50–95% of MBC cases, deserves specific mention as an emerging therapeutic target. Unlike in female breast cancer where AR is primarily a favourable prognostic marker, AR in MBC has dual potential: as a predictive biomarker for enzalutamide and other AR antagonists currently in clinical trials, and as a marker of hormone sensitivity in patients who cannot tolerate tamoxifen. AR testing is not yet universal practice but is increasingly included in comprehensive MBC molecular profiling panels.

Tissue Biopsy: Approach and Diagnostic Algorithm

The diagnostic pathway for any male patient presenting with a suspicious breast mass should be systematic, anchored by the principle that gynecomastia is a diagnosis of exclusion and that exclusion requires tissue. The proposed diagnostic algorithm is presented in Table 8.

Table 8. Evidence-based stepwise diagnostic algorithm for male patients presenting with a breast mass.

1	Structured history: mass duration, bilaterality, skin/nipple changes; drug review; systemic disease; family history of breast/ovarian/prostate cancer
2	Bilateral mammography (two views) as first-line imaging; ultrasound of breast + axilla as complementary; BI-RADS classification mandatory
3	Core needle biopsy (14–16 gauge, USS-guided, ≥3 cores) for BI-RADS ≥4; FNAC only if CNB technically unavailable; excisional biopsy if percutaneous sampling inconclusive
4	Histopathological examination: H&E morphology + Nottingham grade; WHO 2022 subtype classification; assess DCIS component, LVI, and perineural invasion
5	IHC receptor profiling (where available): ER, PR, HER2 (IHC ± FISH), Ki-67; E-cadherin if ILC suspected; AR optional; BRCA1/2 germline counselling for all MBC patients
6	Staging: CT thorax-abdomen-pelvis; bone scintigraphy; TNM staging per AJCC 8th edition
7	MDT Tumour Board: breast surgeon, medical oncologist, radiation oncologist, pathologist, genetic counsellor — for definitive treatment planning

Source: Adapted from established diagnostic guidelines for male breast cancer

Treatment of Male Breast Cancer

1. Surgical Management

Modified radical mastectomy (MRM) with ipsilateral axillary lymph node dissection (ALND) remains the standard surgical approach for node-positive MBC, given the limited breast volume that renders breast-conserving surgery technically challenging. Sentinel lymph node biopsy (SLNB), now validated in prospective series of clinically node-negative MBC patients, offers equivalent locoregional control to ALND with substantially reduced morbidity (arm lymphoedema rate 5–8% versus 15–20% for ALND). Intraoperative frozen section of sentinel nodes guides the extent of axillary surgery. Nipple-sparing or skin-sparing mastectomy may be considered in select cases with tumour-to-nipple distance >2 cm (Gucalp et al., 2019).

2. Systemic Therapy

Adjuvant chemotherapy protocols follow female breast cancer evidence, with anthracycline-based (AC: doxorubicin + cyclophosphamide) and/or taxane-based regimens (AC-T) most commonly employed in hormone receptor-positive MBC with high-risk pathological features (node-positive, Grade III, high Ki-67). Neoadjuvant chemotherapy is increasingly applied in locally advanced MBC to enable downstaging prior to surgery.

Endocrine therapy is the cornerstone of systemic management for the predominant ER+/PR+ MBC. Tamoxifen (20 mg/day, 5–10 years) is the preferred agent: it competitively inhibits estrogen binding to ER, is effective regardless of the source of estrogen (gonadal or peripheral aromatization), and is supported by the largest evidence base for male endocrine therapy. Aromatase inhibitors (anastrozole, letrozole, exemestane), while first-line in postmenopausal women, are less effective as monotherapy in males because they do not suppress the gonadotrophin-driven androgen synthesis that maintains estradiol levels via aromatization; their use in males requires concurrent GnRH agonist administration to achieve

adequate estrogen suppression. CDK4/6 inhibitors (palbociclib, ribociclib) combined with endocrine therapy are now increasingly offered in HR+/HER2- metastatic MBC based on expanding male patient inclusion in landmark phase III trials (Cardoso et al., 2018).

3. Radiation Therapy and Follow-up

Post-mastectomy radiotherapy (PMRT) is indicated in MBC patients with: tumour ≥5 cm (pT3/T4); positive or close surgical margins; ≥4 positive axillary nodes; or 1–3 positive nodes with additional adverse features. Standard PMRT delivers 50 Gy in 25 fractions to the chest wall and regional lymphatics. Locoregional recurrence risk is reduced by 60–70% with PMRT in high-risk patients (Fentiman, 2018). Surveillance following definitive treatment includes clinical examination every 3–6 months for the first 3 years, then annually; annual mammography of the contralateral breast; and ongoing endocrine therapy toxicity monitoring.

Prognosis and Outcome Modifiers

As established in Section 3.1, stage-for-stage survival in MBC is broadly comparable to female breast cancer. The modifiable determinant of poor MBC outcome is diagnostic delay, and the non-modifiable determinant — within a given stage — is the molecular subtype defined by histopathological and immunohistochemical characterisation.

Prognostic Factor	Favourable	Unfavourable
TNM Stage (AJCC 8th ed.)	Stage I (OS ~95% at 5 yr)	Stage III–IV (OS <55% at 5 yr)
Lymph node status	Node-negative (pN0)	≥4 positive nodes (pN2–N3)
Nottingham grade	Grade I (Score 3–5)	Grade III (Score 8–9)
Molecular subtype	Luminal A (ER+/PR+/HER2−/Ki-67 low)	Triple-negative; HER2-enriched
BRCA2 status	BRCA2 wild-type (sporadic MBC)	Germline BRCA2 mutation carrier

CONCLUSION

This case illustrates the diagnostic challenges arising from the clinical overlap between gynecomastia and male breast cancer in older male patients. A 62-year-old man with a unilateral, firm, subareolar breast mass accompanied by nipple retraction and axillary lymphadenopathy was initially misdiagnosed with gynecomastia for four months, resulting in delayed definitive diagnosis and presentation at a more advanced disease stage.

Clinicians, including primary care physicians, internists, and surgeons, should recognize that male breast masses with atypical features, such as unilaterality, firmness, eccentric location, nipple retraction, skin changes, or axillary lymphadenopathy, require prompt diagnostic evaluation through imaging modalities (mammography with or without ultrasound) and tissue biopsy. The anatomical pathologist plays a central role in the diagnostic pathway, as immunohistochemical profiling provides essential information for prognostic classification and treatment selection. A structured multidisciplinary approach integrating clinical, radiological, and histopathological expertise is essential to minimize diagnostic delays and improve survival outcomes in male breast cancer.

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