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Idiopathic Granulomatous Mastitis: A Comprehensive Review

Julie Piccione [1], **Jenny Lue Solomon** [1], **Julie Nusbaum** [1]

[1] NYU Grossman Long Island School of Medicine

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ABSTRACT

Idiopathic granulomatous mastitis (IGM) is a rare, benign inflammatory condition of the breast that predominantly affects women of childbearing age with a history of pregnancy and lactation. Its pathogenesis remains poorly understood, with autoimmunity, hormonal dysregulation, ductal injury, and infection all implicated as contributing factors. Clinically, IGM mimics breast malignancy and primary infection, contributing to significant diagnostic delays. Diagnosis requires histopathologic confirmation of lobule-centered non-caseating granulomatous inflammation following exclusion of infectious, malignant, and other granulomatous etiologies. No universally accepted treatment guidelines exist. Evidence supports a central role for corticosteroids in disease management, with emerging data suggesting superior outcomes with the addition of methotrexate in moderate to severe disease; antibiotic monotherapy is associated with the lowest rates of remission. This review examines all the largest case series, systematic reviews, and recent case reports to explore the epidemiology, pathophysiology, clinical presentation, diagnostic evaluation, and treatment of IGM, with the goal of improving recognition and outcomes for patients with this challenging condition.

1. Introduction

Idiopathic granulomatous mastitis (IGM) is a rare, benign inflammatory condition of the breast with an estimated prevalence of 2.4 per 100,000 females in the United States, most commonly affecting females of childbearing age with a history of childbirth and lactation.[1-3] Clinically, IGM presents with breast pain, erythema, palpable masses, and recurrent or non-healing wounds, typically in a unilateral distribution. Lesions may produce purulent discharge and progress to abscesses. Nipple retraction and lymphadenopathy may also be present. Because the clinical features of IGM overlap significantly with those of breast malignancy, timely evaluation is essential.

2. Clinical Presentation and Diagnosis

Diagnosis of IGM is confirmed histologically, with biopsy demonstrating noncaseating granulomatous inflammation. However, establishing the diagnosis can be challenging given the condition's rarity and overlapping features with infectious etiologies, foreign body reactions, and other granulomatous diseases such as sarcoidosis.[4] Geographically, IGM is most frequently reported in the Middle East and Asia; in the United States, Hispanic patients appear to be disproportionately affected.[5]

The presentation, clinical course, and response to treatment in IGM is highly variable. The absence of standardized management guidelines furth complicates care. Prompt recognition, early referral, and individualized multidisciplinary care, incorporating both surgical and medical approaches, are essential to optimizing patient outcomes.

1. Pathophysiology/Risk Factors

Though IGM was first described in 1972 by Kessler and Wolloch, its pathogenesis remains poorly understood. Autoimmunity, inflammation, trauma, and infection have all been proposed as contributing factors.[6]

The concurrence of IGM with other autoimmune diseases supports an autoimmune component to its pathogenesis. Up to 34% of patients with IGM have concomitant autoimmune diseases, such as sarcoidosis, systemic lupus erythematosus, Sjogren's disease, and erythema nodosum (EN).[7,8] The presence of EN in particular has been associated with a higher risk of disease recurrence.[7,9-12]

Infectious etiologies have also been implicated in disease pathogenesis. The normal breast flora includes *Staphylococcus*, *Streptococcus*, *Corynebacterium*, and *Lactobacillus*. It remains a matter of debate whether translocation of these colonizers into deeper breast tissue contributes to disease pathogenesis. This question is further complicated by the difficulty of interpreting culture data. These organisms may be reported as normal flora even when clinically relevant. Several microorganisms have nonetheless been identified in association with IGM progression, including *Enterococcus avium*, *Mycobacterium mucogenicum*, and *Corynebacterium* species.[13-15] While *Corynebacterium* is a constituent of normal breast flora and is frequently found on cultures in IGM patients, its presence in deep breast tissue can provoke a significant inflammatory response.[16]

Traumatic ductal injury may also contribute to disease development. When ductal epithelium is damaged, as may occur with milk stasis, luminal secretions leak into the surrounding lobular connective tissue, triggering recruitment of macrophages and lymphocytes, driving local inflammation.[17] Hormonal factors have been similarly implicated. Elevated prolactin, upregulated prolactin receptors, and hypersensitivity to circulating prolactin have all been proposed as potential risk factors.[17] Supporting the relevance of reproductive and hormonal history, a 2023 meta-analysis found that 92.7% of IGM patients had a history of pregnancy, 76.6% had breastfed, 22.7% reported oral contraceptive use, and 19.7% had elevated prolactin levels.[18] The significance of oral contraceptive use remains uncertain, with some case series reporting an association and others finding none.[19] Elevated body mass index and tobacco use have also been weakly associated with IGM, potentially through their contributions to periductal inflammation. [2,20]

Multiple studies have demonstrated elevated serum inflammatory markers and cytokines in patients with IGM compared to controls. In one study, the pro-inflammatory cytokines IL-22 and IL-23 were significantly elevated in IGM relative to healthy controls.[20] A separate study of 30 women found IL-8 and IL-17 levels to be significantly higher in IGM patients than controls. Consistent with its anti-inflammatory role, IL-10 was elevated in patients who achieved remission.[21] Serum IL-6 and CRP have similarly been found to be significantly elevated in patients with IGM and may correlate with disease severity. A study of 41 patients found that higher serum CRP levels were associated with longer time to lesion resolution.[22] Taken together, these findings suggest a cytokine environment in active IGM characterized by pro-inflammatory predominance, with a shift toward anti-inflammatory signaling accompanying disease remission.

Perhaps the most compelling evidence that that IGM has an autoimmune basis lies in its response to treatment. Patients with IGM frequently demonstrate a marked response to corticosteroids, when administered systemically or intralesionally. A randomized control trial of 140 IGM patients compared ductal lavage with triamcinolone acetonide and antibiotics versus oral corticosteroids alone. Remission at one year was achieved in 87% of patients in the systemic steroid arm and 85.5% in the ductal lavage arm. The comparably high remission rates across both groups underscore that corticosteroid delivery—regardless of route—is central to treatment response, lending support to an underlying autoimmune mechanism.[23]

3. Clinical Symptoms

The presentation of IGM varies widely, ranging from a single painless lesion to more extensive disease characterized by diffuse breast pain, erythema, palpable masses, open wounds, nipple discharge, or abscess formation. In one retrospective chart review, abscess was the most common presenting symptom.[5] Lesions may arise in any quadrant of the breast and are most commonly unilateral, though bilateral involvement has been reported. In some cases, overlying skin changes,

including pea d'orange appearance, nipple reaction, and axillary lymphadenopathy, may prompt concern for breast malignancy.[24] In chronic or refractory disease, fistula or sinus tract formation can occur. Though IGM occurs predominantly in females, cases in males have also been reported.

4. Imaging and Histopathology

IGM presents a significant diagnostic challenge, sharing clinical features with inflammatory breast cancer and primary breast infection. Radiographic findings are similarly non-specific across modalities.[25]

On MRI, post-contrast imaging may demonstrate regional non-mass-like enhancement with duct wall thickening, or may reveal multiple abscesses with associated inflammatory changes and rim enhancement.[26] Lesions are typically hypointense on T1-weighted and hyperintense on T2-weighted sequences.[26] In more advanced disease, MRI may show sizeable fluid collections with peripheral enhancement, occasionally with tracts extending to the skin surface.[26] Common mammographic findings include breast asymmetry, focal masses, and skin thickening.[27] On ultrasound, IGM typically appears as hypoechoic masses, heterogenous parenchyma, abscesses, and skin thickening, with tubular extensions tracking around breast lobules.[27] Doppler imaging may demonstrate associated hypervascularity.[28]

Given the overlap of radiographic findings with other conditions and the absence of defining clinical features, biopsy with histopathologic evaluation remains the cornerstone of diagnosis. IGM is characterized histologically by lobule-centered non-caseating granulomatous inflammation. Before the diagnosis of IGM can be established, other causes of granulomatous inflammation must be excluded, including foreign body reaction, diabetes, inflammatory bowel disease, sarcoidosis, and tuberculosis. In addition to granuloma formation, histology may demonstrate reactive, lymphoplasmacytic infiltration centered on the breast lobules. In more severe disease, the lobulo-centric architecture may be partially or completely obliterated.[8]

5. Delays in Care

Many patients with IGM experience significant delays in both diagnosis and treatment. Patients typically first present to their gynecologist or primary care physicians after developing symptoms, at which point imaging is usually obtained and referral to surgery or radiology for biopsy is initiated. A major diagnostic pitfall is the presence of bacteria on biopsy cultures, which can lead to misdiagnosis of primary breast infection and result in repeated incision and drainage procedures, carrying real risk of breast deformity. Differentiating IGM from periductal mastitis can be particularly challenging when excisional margins are insufficient or prior breast surgery has altered the local tissue architecture.[29] Even after biopsy results are available, further delays may occur in establishing care with rheumatology. In one study, the average delay from symptoms onset to diagnosis was approximately 4-5 months.[7]

The clinical course of IGM is variable. Milder disease may resolve spontaneously, while more severe cases often involve multiple lesions requiring systemic therapy and close follow-up. Notably, longer follow-up duration has been associated with lower recurrence rates, underscoring the importance of sustained monitoring even after clinical improvement.[30]

6. Treatment

Although IGM is self-limited in some cases, the pain and physical deformity caused by the disease warrant treatment in nearly all cases. Between 21% and 65% of cases may resolve with conservative management, though this wide range reflects the limitations of smaller, heterogeneous studies.[2,31] Treatment options include surgical intervention, non-steroidal anti-inflammatory medications, colchicine, intralesional or systemic corticosteroids, and disease-modifying antirheumatic drugs (DMARDs). No universal treatment guidelines exist, and retrospective studies and meta-analyses have yielded inconsistent conclusions regarding optimal treatment. While some studies indicate that corticosteroids and surgery are associated with lowest recurrence risk, at least one meta-analysis found no significant difference between the two approaches, favoring medical management given the infection and cosmetic risks inherent to surgical intervention.[32]

Incision, Drainage, and Antibiotics

When patients with known IGM develop warm, fluctuant lesions, incision and drainage may be performed and any isolated organisms treated with targeted antibiotics. However, repeated procedures are common and carry meaningful risks of pain, poor wound healing, and disfigurement. Because inflammation is ultimately the driver of abscess development, many clinicians opt to treat empirically with antibiotics while initiating concurrent anti-inflammatory therapy. Studies evaluating antibiotic treatment have varied widely in agent, dosage, and duration, with inconsistent results.[30] Notably, one meta-analysis found antibiotic monotherapy was associated with the lowest rate of remission, and that the combination of antibiotics and surgery carried the highest recurrence rate.[30] These findings underscore the importance of addressing the underlying inflammatory process, not just the microbial process alone.

Corticosteroids and DMARDs

One systematic review found that corticosteroids, antibiotics, and surgery were used in roughly equal proportions overall, though notable regional variations were observed. Antibiotic-based treatment was more common in less-developed countries, while surgical intervention predominated in more-developed countries.[7] These patterns likely reflect differences in healthcare infrastructure, access to rheumatologic care, and institutional experience with IGM rather than evidence-based preference. The review was further limited by its retrospective design and the potential for regional publication bias.

Multiple studies have found the combination of corticosteroids and methotrexate yields superior outcomes compared to corticosteroids alone, suggesting

meaningful clinical utility for dual immunosuppressive therapy.[33, 34] In one study of 53 patients, 41 treated with steroids and methotrexate demonstrated clinical improvement within the first month, with median time to remission of 13.04 months.[33] Notably, all three patients treated with surgery alone, without immunosuppression, relapsed. A cohort study of 63 patients found that combining incision and drainage with low-dose corticosteroids (with methotrexate added for insufficient response) achieved complete remission in 85% of cases.[2] One meta-analysis identified multimodal therapy—incorporating antibiotics, corticosteroids, and surgery—as the most effective strategy for preventing recurrence.[30]

Interpreting the totality of this evidence is challenging for several reasons. Studies vary considerably in design, patient population, disease severity at enrollment, and geographic setting. It is plausible that patients selected for surgical monotherapy in some studies had milder or more localized disease, artificially inflating apparent surgical success rates. Regional differences in treatment philosophy, access to DMARDs, and rheumatologic expertise may further account for the divergent findings across studies. Standardized prospective trials with consistent outcome definitions are needed to establish reliable treatment guidelines.

7. Conclusion

IGM is a rare but impactful inflammatory condition of the breast that can mimic infectious, inflammatory, and malignant disease. Diagnosis requires a high index of clinical suspicion, supported by radiographic characterization, biopsy, and systematic exclusion of malignancy and other causes of granulomatous inflammation. Prompt recognition and early referral to rheumatology are essential, as delays in diagnosis and follow-up have been correlated with higher recurrence rates.[30]

The role of microorganisms remains incompletely understood. Although bacteria are frequently identified in biopsy cultures, antibiotic monotherapy has been associated with the lowest rates of remission, reinforcing that inflammation, rather than infection, is the primary driver of disease. Treatment should be individualized based on disease severity, clinical course, and patient preference. While mild cases may resolve spontaneously or with conservative management, patients with more severe or refractory disease are likely best served by combination therapy incorporating corticosteroids, methotrexate, and surgical intervention as indicated.[2,30,33-36] Interpreting the existing literature remains challenging given the heterogeneity of study designs, patient populations, and geographic contexts; standardized prospective trials are needed to establish evidence-based treatment guidelines.

IGM substantially impairs quality of life, and its tendency to be misdiagnosed or undertreated compounds this burden. Greater clinical awareness of IGM across primary care, gynecology, surgery, and radiology, coupled with timely rheumatology involvement, has the potential to meaningfully reduce associated morbidity and improve patient outcomes.

8. References

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