REVIEW

European Journal of BREAST HEALTH

DOI: 10.4274/ejbh.galenos.2025.2025-10-7
Eur J Breast Health 2026;22(2):126-138

Idiopathic Granulomatous Mastitis: A Comprehensive Review of Etiology, Diagnosis, and Management

Parisa Hashemi Moghanjoughi[1], Sina Neshat[2], Afshin Heidari[3], Matin Bidares[4], Mahshid Sadat Chenarani Moghadam[5], Dorsa Shekouh[6], Keyvan Heydari[7], Fatemeh Mohammad Alizadeh[8], Hazhir Moradi[9], Roya Modaresi[3]

[1]Department of Obstetrics and Gynecology, Preventative Gynecology Research Center, Shahid Beheshti University of Medical Sciences, Tehran, Iran
[2]Adjunct Professor of Anatomy and Physiology, Department of Health Science, South College, Atlanta, United States of America
[3]School of Medicine, Isfahan University of Medical Sciences, Isfahan, Iran
[4]Health Policy Research Center, Shiraz University of Medical Sciences, Shiraz, Iran
[5]Department of Dermatology, Radboud University Medical Center, Nijmegen, Netherlands
[6]Student Research Committee, School of Medicine, Shiraz University of Medical Sciences, Shiraz, Iran
[7]Gastrointestinal Cancer Research Center, Non-communicable Diseases Institute, Mazandaran University of Medical Sciences, Sari, Iran
[8]Department of Neurology, Boston Children's Hospital, Harvard Medical School, Boston, United States of America
[9]Nosocomial Infection Research Center, Isfahan University of Medical Sciences, Isfahan, Iran

ABSTRACT

Idiopathic granulomatous mastitis (IGM) is a rare, benign, and chronic inflammatory breast disease of uncertain etiology. It often mimics infectious mastitis and inflammatory breast cancer in both clinical and radiologic presentations, leading to diagnostic and therapeutic challenges. This review aims to provide a comprehensive summary of the current literature regarding the etiology, pathogenesis, clinical manifestations, diagnostic strategies, and treatment options for IGM. A narrative review was conducted using an extensive search of the PubMed database, focusing on articles that discuss various aspects of IGM, including its potential autoimmune, hormonal, and infectious origins, as well as current diagnostic and management approaches. IGM most commonly affects women of reproductive age, often within a few years postpartum. Histologically, it is characterized by non-caseating granulomatous inflammation centered on breast lobules. Although corticosteroids are widely used as the first-line therapy, treatment regimens vary significantly across centers, and relapse is not uncommon. Immunosuppressive agents, such as methotrexate, have shown promising results in steroid-resistant cases. Surgical interventions are generally reserved for refractory cases because of the risk of recurrence and unfavourable cosmetic outcomes. The role of infectious agents, particularly *Corynebacterium kroppenstedtii*, remains controversial, and distinguishing between idiopathic and infectious GM is crucial for management. IGM is a multifactorial and clinically heterogeneous condition requiring individualized, multidisciplinary management. There remains a need for further prospective studies and consensus guidelines to optimize diagnosis and treatment, especially in recurrent or refractory cases.

Keywords: Idiopathic granulomatous mastitis; inflammatory breast disease; breast diseases; inflammatory lesions; chronic mastitis

Corresponding Author: Sina Neshat, MD
E-mail: sinaneshat@gmail.com **ORCID:** orcid.org/0000-0001-6522-1865
Received: 30.10.2025 **Accepted:** 21.12.2025 **Available Online Date:** 24.03.2026
Cite this article as: Hashemi Moghanjoughi P, Neshat S, Heidari A, Bidares M, Chenarani Moghadam MS, Shekouh D, et al. Idiopathic granulomatous mastitis: a comprehensive review of etiology, diagnosis, and management. Eur J Breast Health. 2026;22(2):126-138

KEY POINTS

- Idiopathic granulomatous mastitis (IGM) is a rare, benign breast condition that can closely mimic infection or cancer; diagnosis usually requires a tissue biopsy and careful exclusion of other causes.
- Most patients improve with medical therapy—typically corticosteroids—and methotrexate can help when steroids fail or relapse occurs, while surgery is now reserved for complicated or persistent cases due to recurrence risk and cosmetic concerns.
- Because IGM likely has multiple causes and behaves differently across patients, care should be individualized and ideally coordinated by a multidisciplinary team; more prospective studies are needed to guide standardized treatment.

Introduction

Idiopathic granulomatous mastitis (IGM), also called idiopathic granulomatous lobular mastitis, is a benign, chronic inflammatory breast disease of unknown etiology.

GM is a broad clinical entity divided into two categories: specific GM and IGM. Kessler and Wolloch (1) first described IGM in 1972. The etiology of IGM is assumed to involve infectious, autoimmune, and possibly chemical exposure-related mechanisms (2).

The disease typically manifests as an inflammatory mass in the outer part of the breast and may occasionally present as multiple abscesses with ulceration and inflammation of the overlying skin (3). The formation of sinuses, nipple retraction, axillary adenopathy, and peau d'orange-like skin changes represent clinical features that can resemble malignancyare clinical features that may mimic malignancy. Additionally, the imaging features of IGM closely resemble those of mastitis and breast cancer. This similarity in characteristics raises the possibility of misdiagnosis, resulting in delayed and inappropriate medical interventions (4).

There is no single pathognomonic clinical or imaging feature for IGM. However, histopathologic confirmation, most often via core needle biopsy, remains the cornerstone of diagnosis. Treatment options vary widely, ranging from observation and antibiotics to corticosteroids, immunosuppressive agents, and in selected cases, surgery.

This article reviews the current literature on IGM and aims to provide clinicians with an updated and structured overview of its etiology, diagnosis, and management.

Epidemiology

IGM is a rare disease, constituting only 0.44–1.6% of breast biopsies based on cytologic and pathologic diagnosis (5). IGM exhibits a higher incidence in developing countries, possibly owing to underdeveloped public health systems and misdiagnosis as other granulomatous inflammatory diseases, including tuberculosis. IGM is more commonly observed in women of Hispanic descent, particularly among Spanish and Asian women of childbearing age, suggesting a certain degree of genetic predisposition (6). The most prevalent age at which a patient develops this disease is during the childbearing years, occurring mostly five years after breastfeeding. The youngest patient diagnosed with IGM was 11 years old, and the oldest was in her 80s (7).

Etiology and Pathogenesis

The etiology and pathogenesis of IGM remain obscure (8). The pathogenesis of IGM is not yet precisely understood, but different steps may contribute to this disease's pathological process. One of these stages entails a non-specific inflammatory response within lobules, affecting multiple lobules simultaneously, known as lobulitis, which may cause reactive lymphoplasmacytic infiltration. At times, the deformation of a lobule results in granulomas characterized by central suppurative necrosis, leading to abscess formation due to proliferation of these foci (9).

As presented in Figure 1, three main hypotheses have been suggested for the pathogenesis of IGM: infection, autoimmunity, and hormonal disorder (10). Among these three main reasons, certain predisposing factors facilitate the procedure. Risk factors for IGM include pregnancy, breastfeeding, smoking, use of oral contraceptives (OCs),and antitrypsin deficiency (7, 11). The proposed pathogenic mechanisms are not conclusively independent and typically involve multiple contributing factors and mechanisms (12, 13).

α1-Antitrypsin Deficiency

α1-antitrypsin (AAT), a glycoprotein synthesized in the liver by hepatocytes, is a member of the serine protease inhibitor family. It primarily inhibits proteases, including cathepsin G, elastase, and proteinase 3, which are secreted by activated neutrophils. AAT is considered an acute-phase reactant because of its increased levels during inflammation. AAT is considered an acute-phase reactant because it is elevated during inflammation. AAT deficiency primarily contributes to liver and lung pathology.

In 2001, Schelfout and colleagues identified AAT deficiency in a 37-year-old female patient who was diagnosed with IGM. However, the authors did not identify any additional causative factors in this study and proposed that AAT deficiency might be the primary and sole etiological factor. Nonetheless, they recommended further investigation to validate this hypothesis (14).

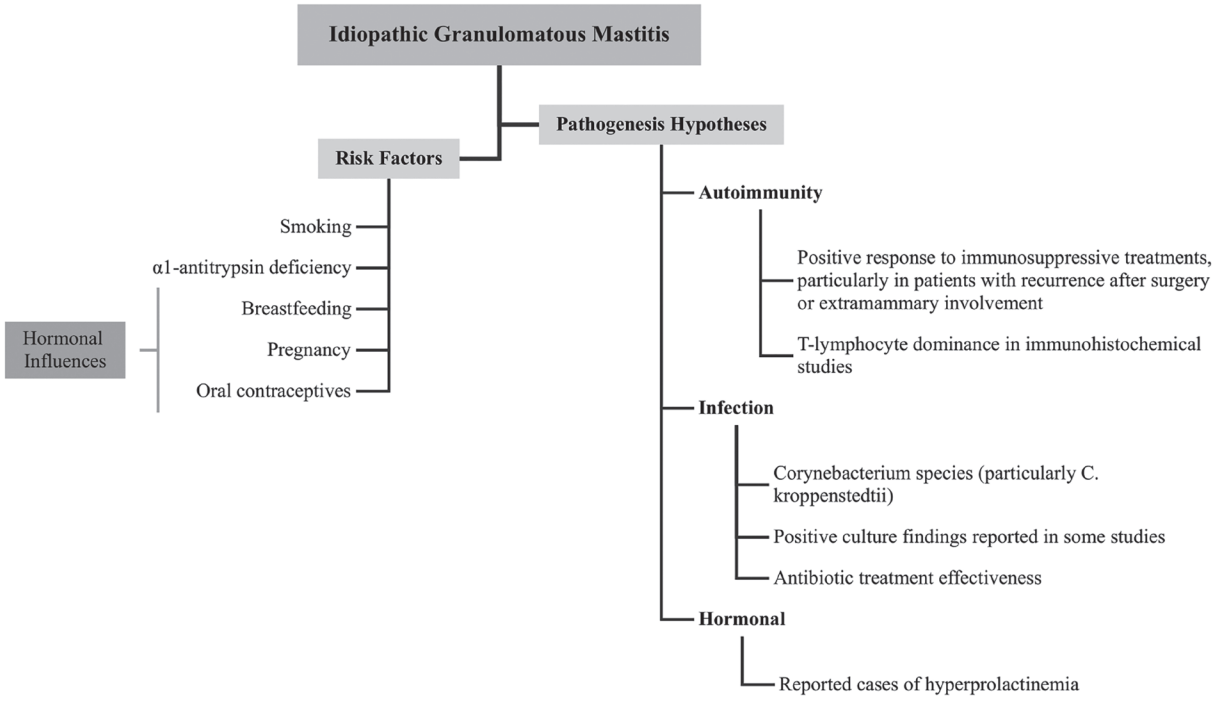

Figure 1. Risk factors and pathogenesis of idiopathic granulomatous mastitis. A tree chart is presented, offering a structured breakdown of IGM's etiology and pathogenesis. The chart branches from the central topic of IGM into primary divisions of "risk factors" and "IGM pathogenesis hypotheses"

C: Corynebacterium; IGM: Idiopathic granulomatous mastitis

Oral Contraceptives

The evidence supporting the relationship between OCs and IGM is controversial. OCs may predispose individuals to IGM by increasing breast secretions (9).

Although no significant association has been found between IGM and OCs, many studies indicate that OCs are potential risk factors. Al-Khaffaf et al. (15), Asoglu et al. (16), Gurleyik et al. (17), Oran et al. (18) and Bani-Hani et al. (7) reported 10 cases (10/46, 21.7%), 8 cases (8/19, 42.1%), 5 cases (5/18, 27.7%), 24 cases (8.3%), and 18 cases (11.1%), respectively. Data indicate the rejection of OCs as a risk factor for IGM, as demonstrated by Baslaim et al. (3), who reported that none of the 20 patients included in their study had a history of OC use. While reported frequencies of OCs use among IGM patients range widely across studies (0–42%), these values primarily derive from small case series and should not be interpreted as reflecting population-level risk. Instead, they suggest that OCP use may act as a potential—but unproven—cofactor in susceptible individuals.

Gestation, Birth, and Breastfeeding

The epidemiology of IGM, characterized by a peak incidence under the age of 50 and a frequent history of recent childbirth and breastfeeding, suggests that these factors are involved in the etiology of this disease. Changes in hormonal levels during this time and their effects on inflammation and secretions may play a significant role in disease pathology (3, 15, 17). In Bani-Hani et al.'s (7) study of 24 cases, only two individuals lacked a history of pregnancy; four were currently pregnant, and another four had given birth and breastfed within the past six months. In a case series of 11 individuals, the authors reported that 10 women had given birth and breastfed within the preceding five years (19). Further, Baslaim et al. (3) all reported cases had a history of pregnancy and breastfeeding; two cases were actively breastfeeding, and one was pregnant. Oran et al. (18) reported that only three of 46 cases were nulliparous. Additionally, Gurleyik et al. (17) reported that among 19 cases, four were actively breastfeeding, while the remaining 15 had previously breastfed.

Individuals diagnosed with IGM, a condition typically occurring during the childbearing years, are likely to have a history of pregnancy and breastfeeding, given that gestation primarily takes place between the ages of 20 and 40. However, the presence in the literature of male cases (20) and individuals spanning a broad age range complicates attributing IGM etiology solely to gestation, childbirth, and breastfeeding.

Hyperprolactinemia

According to the secretion theory, hyperprolactinemia, similar to other hormonal disorders, could also be considered responsible for the pathogenesis of IGM (21, 22). Rowe (21), in his 1984 case presentation, reported that prolactinoma was present as a comorbidity in the IGM case. Nonetheless, further studies did not report detailed prolactin levels. Erhan et al. (23) demonstrated that, in an examination of 18 women, recurrence occurred in three patients (16%), and hyperprolactinemia was identified in two of these patients. Bani-Hani et al. (7) measured prolactin levels in the blood in 7 cases and found increased levels in only one case among 24 patients (4.1%).

Smoking

Although smoking is considered one of the risk factors in the etiology of this disease, a relationship between IGM and smoking has not yet been established. As indicated in the study by Asoglu (16), 14 cases out of 18 (77.8%) had a smoking experience before, meanwhile, Baslaim et al. (3) declaimed that none of their cases was a smoker. Although a causal association between smoking and the development of IGM has not been established, smoking is a well-known inhibitor of wound healing and is associated with delayed resolution of abscesses in inflammatory breast conditions. Several authors therefore caution against surgical intervention in active smokers, as recurrence and poor cosmetic outcomes appear more common in this subgroup. Smoking cessation should be emphasized as part of the therapeutic plan.

Autoimmunity

Among hypotheses about IGM, one theory posits an immunological basis for IGM and has garnered significant attention. Literature reviews show an excellent response to immunosuppressive treatment and steroids, especially in patients with recurrence after surgery, in patients with confirmed T-lymphocyte dominance based on immunohistochemical studies, and in patients who have extramammary involvement (such as arthritis or *erythema nodosum*), which confirms the autoimmunity hypothesis (1, 9, 19, 24-26).

Ozel et al. (27) in a study on 8 cases, reported that 25% of cases were positive for anti-double-stranded DNA (anti-dsDNA), and antinuclear antibody (ANA), and 75% of cases were positive for rheumatoid factor (RF). In the latter study, in which surgery was the chosen treatment option, they demonstrated that two patients with recurrence were anti-dsDNA-positive, ANA-negative, and RF-negative. However, the disease was resolved after steroid treatment. In another study conducted by Erhan et al. (23), they performed an immunohistochemical experiment and determined T-cell dominance in 14 out of 18 cases. This discovery was interpreted as an autoimmune pathophysiological consequence, characterized by the development of centrilobular granulomas in response to ductal damage and reactive T-cell-mediated inflammation. Additionally, literature reports have documented one case of IGM with Sjögren's syndrome, one with *erythema nodosum* and arthritis, two with *erythema nodosum*, and one with Weber-Christian disease (28-31). Although studies have indicated that a coexisting autoimmune disorder represents only a minority of all cases, some research supports the autoimmune hypothesis. Moreover, classical serological tests commonly employed for autoimmune disorders, such as RF and ANA, yielded variable results in IGM cases. Asoglu et al. (16), in their case series of 18 cases, reported that none tested positive for RF or ANA. In the study conducted by Altintoprak et al. (32), they investigated the autoimmunity hypothesis for IGM etiology; in 26 cases, they examined extractable nuclear antibody levels and ANA. However, we did not obtain results sufficient to evaluate the autoimmunity hypothesis (32).

Because of the suspected autoimmune component and frequent coexistence of systemic inflammatory manifestations such as *erythema nodosum* and arthritis, many patients are referred to rheumatology for evaluation and management. This has contributed to the widespread use of systemic immunosuppression and corticosteroids in IGM care. Recent studies have also explored intralesional steroid administration—often performed by breast surgeons or rheumatologists—as a targeted modality to suppress localized inflammation while reducing systemic toxicity.

Microbiological Agents

The indigenous bacterial flora of the normal breast is comparable to that of the skin, with predominant organisms comprising *Corynebacterium* species, coagulase-negative streptococci, and *Propionibacterium* species. These findings have been identified in breast tissue obtained during mammoplasty in nipple discharge cultures (33). These flora may migrate into deeper layers of the breast via ductal pathways (34). Indeed, *Corynebacterium* may cause mastitis in livestock, although these bacteria are not assumed to be pathogenic in humans. Taylor et al. (35), who reported finding *Corynebacterium* in 34 IGM cases, made these bacteria the focus of attention in 2003. *Corynebacteria*, Gram-positive bacteria found in the skin flora, pose a challenge in determining whether they contribute to contamination, infection, or colonization (36). However, because it is hard to distinguish the outcomes, it is essential to confirm >104 colony-forming unit/mL of dominant *Corynebacterium* species or the presence of purulent matter in an abscess (37).

Funke et al. (38) demonstrated that these bacteria might be a potent risk factor if a *Corynebacterium* species is detected in tissue expected to be sterile under normal conditions, or if a Gram-positive bacillus accompanying polymorphonuclear leukocytes is present. In IGM cases, four distinct *Corynebacterium*

species have been identified. *Corynebacterium kroppenstedtii (C. kroppenstedtii)* is the most commonly detected species and, due to its positive esculin test and lipophilic nature, differs from other corynebacteria. In Taylor et al. (35) study, 62 patients were diagnosed with IGM, and 38 of these patients (about 54.8%) had a positive culture for *Corynebacterium*. Comparison of the remaining 28 cases demonstrated that fistulas, fever, and neutrophilia were more frequent in patients with positive bacterial cultures. The most frequently observed species was *C. kroppenstedtii* (14 patients; 41.1%) in the latter study. Paviour and colleagues obtained *Corynebacterium* samples from breast tissue in 24 cases, conducted histopathological examinations in 12 cases, and diagnosed IGM in nine cases. Notably, *C. kroppenstedtii* emerged as the most frequently identified species, with *Corynebacterium tuberculostearicum (C. tuberculostearicum)* and *Corynebacterium amycolatum (C. amycolatum)* being other commonly isolated specieswhile *C. tuberculostearicum* and *C. amycolatum* were also commonly isolated. In a subsequent investigation, a three-week intravenous penicillin treatment was administered to one patient. However, because anticipated benefits were lacking, the treatment was switched to oral doxycycline (100 mg) (36). According to the authors, surgery was not necessary following this treatment. Extensive literature supports the idea that a microbiological etiology is essential (37, 39, 40). Two of these studies did not specify the particular species, but Ang and Brown (37) reported the isolation of *Corynebacterium accolens*. All three studies affirmed the effectiveness of antibiotic therapy.

Diagnosis

Identifying IGM relies on a comprehensive differential diagnosis involving histopathologic and radiologic assessments. To accurately diagnose IGM, it is crucial to exclude potential histopathologic mimics such as fat necrosis, duct ectasia, foreign-body reactions, and granulomatous inflammation (e.g., sarcoidosis, tuberculosis, Wegner's granulomatosis). Concurrently, radiologic evaluations should be conducted to distinguish IGM from other conditions that present with similar imaging findings, including malignancies, abscesses, and necrotic lesions. By employing a precise diagnostic approach, clinicians can significantly impact treatment strategies. A combination of diagnostic modalities is utilized for IGM assessment, including mammography, sonography, magnetic resonance imaging (MRI), and histopathologic examination. Each of these methods is explained below and summarized in Figure 2.

Clinical Presentation

The most typical manifestation of IGM is a mass with indistinct borders in the upper outer quadrants of the breasts of females aged 30–45. This mass could be in any quadrant, but is predominantly located in the upper quadrant (41). The mass exhibits soft borders and may be accompanied by skin abnormalities, such as erythema, peau d'orange, nipple inversion, and skin retraction; it may resemble inflammatory breast cancer. The most common complications of the disease are abscesses, fistulas, ulcerations, infections, and sinus formation. In Aziza et al. (42) study, of 474 cases reviewed, 19.4% had skin changes and 17.7% had nipple inversion. Moreover, patients exhibiting skin changes experienced a higher recurrence rate. In IGM, the presence of both skin changes and a breast mass can make differentiation from inflammatory breast cancer challenging, even with imaging modalities such as ultrasound, mammography, and MRI. Furthermore, it is crucial to consider the rare occurrence of mammary tuberculosis, particularly because it tends to affect the lymph nodes and lungs. Additionally, the presence of caseous necrosis and breast duct involvement should be assessed by histological examination.

Imaging

Mammography

IGM lacks distinctive features on mammography, and the visibility of small lesions is frequently obscured by the dense breast tissue commonly found in women of reproductive age (43, 44). Typically, it appears on mammograms as a mass with asymmetric density and uneven borders, attributed to nipple inversion, local skin thickening, and axillary lymphadenopathy (45). Barreto et al. (46) described the common mammographic manifestation of IGM as structurally distorted, asymmetric masses or nodules. Dursun et al. (45) noted that approximately one-quarter of IGM cases exhibited masses with irregular shapes or obscured borders, with single masses being the most prevalent. However, caution is warranted, as mammograms may appear normal in individuals with dense breast tissue or in those with mild disease (47). Notably, calcifications are seldom observed in IGM and have been reported only in isolated cases (48). Thus, mammographic findings may raise suspicion of IGM, but a definitive diagnosis relies on histopathologic examination rather than imaging alone.

Sonography

Because of numerous sensitivity issues and the lack of diagnostic capability of MRI for IGM, sonography seems valuable for screening breast masses (47). More than 80% of masses detected by sonography are heterogeneous and hypoechoic (43). Because the structure of masses in IGM is highly variable, the specificity is very low due to structural distortion, irregular masses, parenchymal edema, skin thickening, effusion, and axillary lymph node enlargement (49). The most frequent finding on ultrasonography is either a mixed hypoechoic or a hyperechoic mass. However, hypoechoic masses are much more common, exhibiting indeterminate, angular shapes and may have

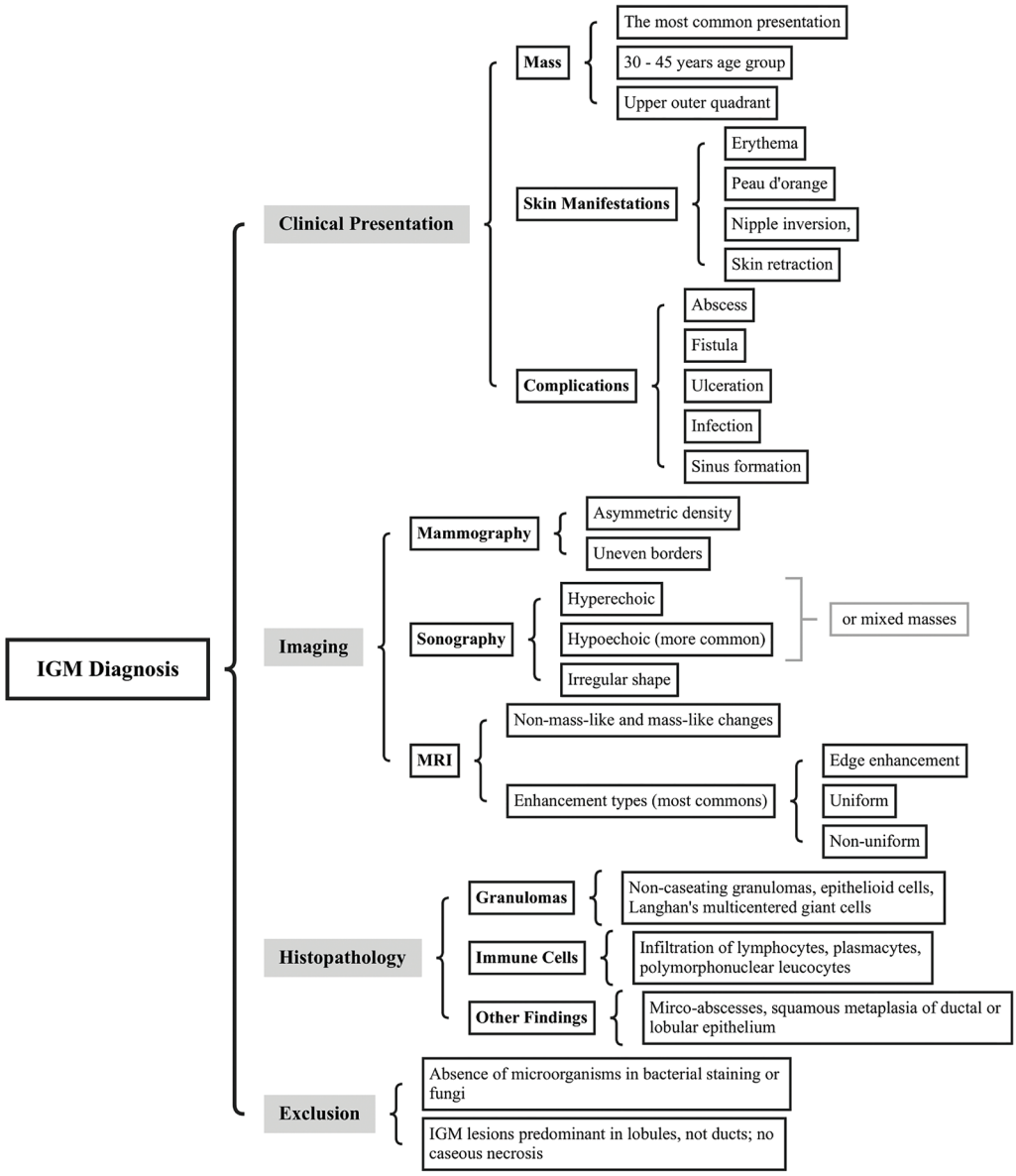

Figure 2. Overview of idiopathic granulomatous mastitis diagnosis. This figure presents a comprehensive, structured representation of the diagnostic approach to idiopathic granulomatous mastitis. It illustrates the four main diagnostic pillars using a brace map format: clinical presentation, imaging, histopathology, and exclusion criteria. Each category further bifurcates into specific components, offering a holistic view of the diagnostic features and methodologies

IGM: Idiopathic granulomatous mastitis; MRI: Magnetic resonance imaging

infiltrated the adjacent parenchyma. The detected masses may be accompanied by posterior shadowing, enhancement, or other posterior acoustic phenomena (50). Doppler imaging may show increased blood flow in masses and adjacent breast tissue, a sign of inflammation (51). Sonography offers the benefits of high sensitivity and non-invasiveness and has potential to effectively screen individuals with mild ailments. Moreover, it can assess the degree of inflammation, the presence of sinus tracts, and lymph node involvement.

Magnetic Resonance Imaging

The MRI features of IGM can differ based on the level of inflammation. Dursun et al. (45) employed the breast imaging reporting and data system, established by the American College of Radiology, to examine MRI images from 35 IGM cases. The results indicated non-mass-like changes in 77% (28 out of 36) of patients and mass-like alterations in 66% (24 out of 36) of patients (45). Morphologically, 23% of patients exhibited oval masses,

while 17% exhibited lobulated masses. Moreover, the affected parenchyma showed substantial enhancement on imaging; common enhancement types included edge enhancement (with or without interior separation), uniform enhancement, and non-uniform enhancement (52). Micro-abscess formation around the lesions may suggest edge enhancement on the T2 signal (19). The parenchymal apparent diffusion coefficient in IGM-affected areas was lower than in non-diseased regions. However, Yilmaz et al. (52) showed that diffusion-weighted-MRI is unreliable for diagnosing IGM and may be misleading because it can resemble breast malignancies. Furthermore, Dursun et al. (45) illustrated that, in 25% of IGM cases, the time-intensity curve exhibited signal changes resembling those observed in breast cancer. As a result, MRI shows limited diagnostic specificity for IGM and can occasionally be unreliable in distinguishing between IGM and breast cancer. Specifically, cases of IGM with non-mass-like changes may be misdiagnosed as invasive or non-invasive breast cancer.

Histopathology

Crucially, the identification of IGM relies on histopathologic results. In certain studies, open biopsy is employed for diagnostic purposes during procedures such as lesion resection or mastectomy. Needle biopsy, a widely endorsed sampling method, is performed by two techniques: percutaneous needle biopsy and fine-needle aspiration cytology (FNAC), the most commonly used method in suspected cases. Advantages of FNAC include its simplicity, rapidity, and minimally invasive nature. However, the primary limitation of FNAC is its low sensitivity (53, 54). Many studies have shown that only about one-fifth of IGM cases can be diagnosed by FNAC. However, for patients who underwent FNAC, further open biopsies may be needed for a definitive diagnosis (55, 56). In complex cases in which needle biopsy is insufficient, open biopsy is essential because the needle biopsy may fail to detect granulomas.

Histologically, IGM is identified by the presence of central non-caseating granulomas consisting of epithelioid cells and Langhans multinucleated giant cells. The chronic inflammation induced by IGM in the mammary stroma is characterized by the infiltration of lymphocytes, plasmacytes, and polymorphonuclear leukocytes. The formation of micro-abscesses is common, and squamous metaplasia of ductal or lobular epithelium may also be be observed.

Exclusion

An essential characteristic of IGM is that no microorganisms are detected on bacterial or fungal staining, which therefore helps to exclude infectious granuloma. Additionally, one difference between tuberculous granuloma and IGM is that tuberculous granuloma has caseous necrosis, whereas IGM does not, and IGM lesions mainly involve the lobules rather than the ducts. However, it is rarely difficult to distinguish culture-negative tuberculous granuloma from IGM.

When performing incision and drainage of a breast abscess in patients with suspected IGM, clinicians should obtain tissue for histopathologic examination. Failure to submit tissue may delay diagnosis and lead to inappropriate management, particularly when distinguishing IGM from infectious and malignant etiologies.

Emerging Classification Systems

Recent studies have attempted to classify IGM into clinically and radiologically meaningful subgroups to guide management. Radiologic classification based on MRI and ultrasound typically differentiates non-mass-like enhancement patterns from mass-like enhancement patterns, which have been associated with differing risks of abscess formation and treatment resistance (45, 46).

Clinically, several authors have proposed stratifying IGM into mild, moderate, and severe disease based on lesion size, presence of abscesses, sinus tracts, skin ulceration, and systemic inflammation. Such classification systems aim to individualize therapy and may help identify candidates for topical therapy, systemic immunosuppression, or combined surgical approaches.

These emerging frameworks highlight the heterogeneity of IGM and underscore the need for standardized, validated classification systems in prospective studies.

Treatment

The establishment of a gold-standard treatment for IGM remains controversial, as no definitive guidelines currently exist. Given the complex interplay of etiologies contributing to IGM's pathophysiology, a multifaceted approach is essential for its management. Primary treatment strategies include watchful waiting, surgical resection, steroid therapy, antibiotics, and immunosuppressive agents. These methods, explained below and summarized in Figure 3, provide a comprehensive framework for addressing IGM.

Expectant Management

Recently, the watch-and-wait approach has been one of the most important treatment modalities. Notably, recent studies report that 50% of IGM patients heal spontaneously. A study by Lai et al. (57) in 2005 showed that 50% of these patients healed after a two-year waiting period without medication. Two other studies by Bouton et al. (58) and Yaghan et al. (20), conducted in 2015 and 2020, reported that healing occurred within 6–12 months of close monitoring. Hur et al. (59) reported that lesion size affects whether expectant treatment is appropriate. The study revealed

that small lesions (1–2 cm in diameter) healed, whereas lesions greater than 5 cm formed breast abscesses. No criteria categorize lesions by size. Hur et al. (59), in their meta-analysis, concluded that, in mild cases, the best treatment is observation. Davis et al. (60) reported that the patient's age at her first childbirth is related to the time required for the wait-and-watch treatment; the older the patient is, the longer the treatment time should be. In another study, Bouton et al. (58) confirmed that the recurrence rate for IGM is about 11%, and recurrence may worsen the disease before spontaneous remission. We should note that expectant management of IGM has disadvantages, such as reliance on patient adherence. Moreover, this method is suitable only for mild cases and sometimes requires a long duration.

Antibiotics

Antibiotics are among the most commonly used treatments for IGM because skin lesions, seen in 20% of patients, and their similarity to bacterial mastitis result in misdiagnosis. If there is no improvement after antibiotic therapy, alternative diagnoses, such as IGM, should be considered. In this situation, a needle biopsy is usually performed, and treatment is determined by pathological findings.

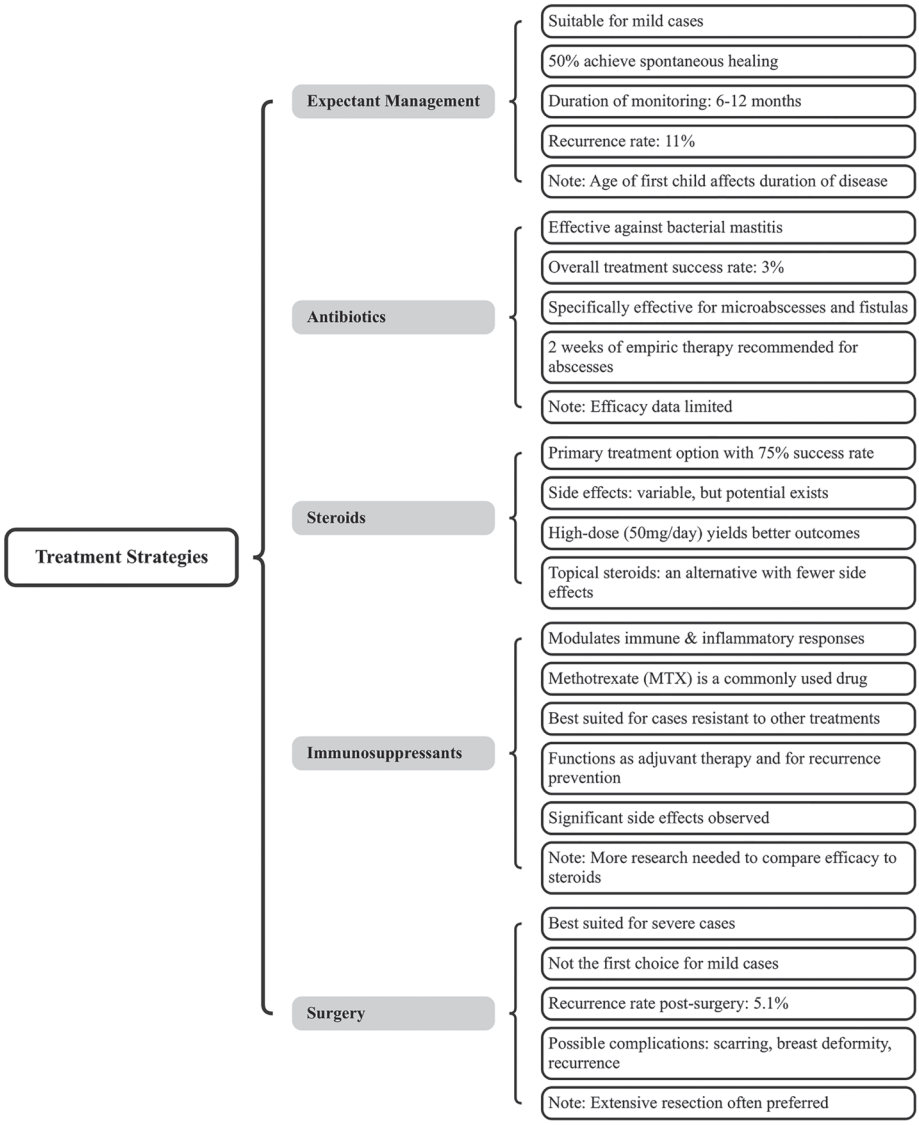

Figure 3. Treatment strategies for idiopathic granulomatous mastitis. This brace map delineates the pivotal treatment strategies for idiopathic granulomatous mastitis. It offers a structured approach, categorizing the primary treatment methods: expectant management, antibiotics, steroids, surgery, and immunosuppressants

MTX: Methotrexate

Aghajanzadeh et al. (61) reported that the treatment rate for IGM with antibiotic therapy was only 3%. The therapeutic effect of antibiotics is minimal. On the other hand, in the study performed by Li (62) in 2019, antibiotic therapy effectively treated microabscesses and fistulas. It can help in the preoperative period as an adjunctive therapy.

According to Benson and Dumitru (63), empiric antibiotic therapy for two weeks is used in IGM with abscess formation and, if not cured, is followed by surgical resection and drainage. Initial antibiotic therapy is indicated if *Corynebacterium* is isolated from the lesion. Although some articles have recommended anti-mycobacterial therapy, there is no solid evidence (64).

Based on the above, antibiotics are not an effective treatment for IGM, and more data are needed to support them as an initial treatment.

Steroids

Steroids are the primary treatment for IGM, with a success rate of 75%, and are preferred to surgery in most cases (43, 65). According to a meta-analysis by Martinez Ramos et al. (2), among 3060 patients, steroid therapy was the most frequent treatment in developing and developed countries (75% and 69%, respectively). Considering the adverse effects of steroids—including impaired glucose tolerance, weight gain, osteoporosis, Cushing's syndrome, peptic ulcer, and mental disorders— the duration and dosage of therapy should be minimized and tailored to the patient's response. Wolfrum et al. (66) and Sakurai et al. (67) suggested 3–6 months of treatment, rather than 3–4 weeks, to prevent disease recurrence. Azlina et al. (68) showed that 50% of patients experienced recurrence after short-course treatment (4 weeks, 60 mg/day). Montazer et al. (69) reported that the high-dose group (50 mg/day) had a significantly better treatment response (93.3% vs. 53.3%) and a lower recurrence rate (0% vs. 37.5%) than the low-dose group (5 mg/day). Steroid therapy is as reliable as initial therapy, and administration of an adequate dose for a sufficient duration is recommended.

In Gunduz et al. (70) study, only 2 of 11 patients experienced recurrence after three months of topical steroid pomade use. Supporting this study, Altintoprak et al. (71) reported that only 3 of 28 patients had a recurrence. Two patients were cured after repeating the same treatment regimen. In both of the latter studies, no adverse effects were reported. In 2019, Çetin et al. (72) conducted a study comparing the efficacy of steroid treatment across routes of administration. The study concluded that there was no significant difference among systemic, combined, and topical steroid administration routes in complete clinical regression, treatment success, or recurrence rate. The author states that topical steroid therapy should be considered the initial treatment route, considering the adverse effects (72). IGM patients should be evaluated for concurrent infection before initiating steroid treatment.

Intralesional steroid injection has recently gained attention as an effective and minimally invasive alternative to systemic steroids. Several prospective and retrospective studies have demonstrated that ultrasound-guided triamcinolone acetonide injection can rapidly reduce inflammation, shorten recovery time, and minimize systemic adverse effects.

Combination regimens involving intralesional and topical steroids have been reported to yield high remission rates with fewer relapses compared with systemic therapy alone (66, 72).

This modality is particularly useful for localized lesions or for patients who have contraindications to systemic corticosteroids.

Studies suggest that IGM may have an autoimmune component in its pathophysiology, e.g., *erythema nodosum*. Patients with both IGM and *erythema nodosum* exhibit an excellent response to systemic immunosuppressive therapy.

Immunosuppressants

The pathway of action of immunosuppressants on IGM is regulation of abnormal immune and inflammatory responses, similar to that of steroids. Methotrexate (MTX) is one of the most widely used immunosuppressants worldwide, acting by inhibiting dihydrofolate reductase and causing serious adverse effects such as pulmonary fibrosis, hepatic and renal damage, and bone marrow suppression. Indications for MTX use in IGM are as follows: first, the most common indication is failure to respond to steroid therapy and surgery (73, 74). The second is to be used as adjuvant therapy with steroids to reduce the duration of steroid use and side effects (75). The third is to eliminate recurrence during steroid dose reduction (76, 77). More data are needed to compare steroids and MTX. However, the first randomized clinical trial conducted by Haddad showed that MTX resulted in significantly higher rates of symptom remission (77). Akbulut revealed that the MTX is a good choice for treatment in her systematic review article, but in some cases still, surgery is neededreported in her systematic review that MTX is a good treatment choice; however, surgery is still required in some cases (73). MTX could be used as one of the initial treatment options for IGM patients.

Surgery

To date, the most effective approach for IGM has been surgical resection till now (59, 63, 78, 79). However, a meta-analysis investigated that surgical treatment had no more apparent profits in comparison with drug therapy and, observation in mild cases (59). In managing patients with IGM and extensive lesions, surgical resection has demonstrated notable advantages

when combined with steroid monotherapy. Wang et al. (80) demonstrated that the recurrence rate in the non-surgical group (22%) was higher than that in the surgical group (5.1%). The study included 200 IGM cases with lesions greater than 5 cm; all received intravenous steroids five days before surgery. Therefore, surgery is a reliable treatment for severe cases (80). According to Shin et al. (81), complications such as scarring, recurrence, and breast deformity are much more common among patients undergoing surgical treatment.

Smoking has been repeatedly associated with impaired wound healing, a higher risk of postoperative complications, and increased recurrence of inflammatory breast diseases. For this reason, several authors recommend avoiding surgical intervention for active smokers unless absolutely necessary and emphasize preoperative smoking cessation strategies.

Surgery should not be the first choice of treatment in IGM cases, but should be considered in the event of complications or recurrence.

Regarding the resection method, most of the literature supports extensive resection because margins or lymph nodes may serve as sources of recurrence or disease flare-up (42, 82, 83).

Because of the heterogeneous nature of IGM, treatment algorithms increasingly emphasize individualized therapy based on clinical severity, radiologic features, and the presence of abscesses or sinus tracts. Recent literature suggests a tiered approach incorporating expectant management for mild disease, topical or intralesional steroids for localized lesions, systemic immunosuppression for moderate-severe disease, and surgery only for refractory or complicated cases. These updates have now been incorporated into the relevant subsections.

Discussion and Conclusion

IGM remains a diagnostic and therapeutic challenge due to its unclear etiology, variable clinical course, and lack of standardized management protocols. While several mechanisms have been proposed—including autoimmune responses, hormonal influences, and infectious triggers—none provides a definitive explanation for all cases, reinforcing the concept that IGM is likely a heterogeneous disease entity.

The role of autoimmunity is supported by the predominance of lymphoplasmacytic infiltration in histopathological specimens and by the clinical response to immunosuppressive agents, particularly corticosteroids and MTX (3, 7, 9). However, a subset of patients fails to respond to these treatments, suggesting that alternative mechanisms may be operative. In this regard, the presence of *C. kroppenstedtii* in some histologic samples has led to the hypothesis that a subset of IGM cases may, in fact, represent chronic bacterial mastitis rather than true idiopathic disease (11, 12). Differentiating these subtypes is clinically relevant, as antibiotic therapy may benefit patients in the latter group, while immunosuppressive treatment may be inappropriate or even harmful.

Diagnosis remains a major hurdle, often requiring a tissue biopsy to rule out malignancy and infectious etiologies, such as tuberculosis and fungal mastitis. Imaging modalities, including mammography and ultrasound, are largely non-specific. Core needle biopsy is currently considered the gold standard for diagnosis (6, 8), but it does not always clarify the pathogenesis or guide management decisions.

Therapeutic strategies for IGM vary considerably. While corticosteroids remain the first-line treatment, the absence of consensus on dosing, tapering schedules, and duration of therapy leads to heterogeneous outcomes (9, 14). Immunosuppressants such as MTX and azathioprine have been employed with some success in refractory or relapsing cases, but long-term safety data are lacking (10, 13). Surgical intervention, once widely used, is now generally reserved for persistent or complicated cases due to the risk of recurrence and disfigurement (15, 16).

The lack of large-scale prospective studies and randomized controlled trials hampers our ability to draw firm conclusions about optimal management. Moreover, most available data derive from single-center studies or small case series with heterogeneous methodologies. There is a pressing need for collaborative multicenter research efforts to stratify patients based on underlying pathophysiology, evaluate the efficacy and safety of treatment regimens—including biologic agents—and develop evidence-based guidelines.

IGM should be considered a complex, multifactorial condition that requires individualized management. Advancing our understanding of its pathogenesis and refining diagnostic criteria are essential steps toward establishing a rational and effective therapeutic framework.

Given the heterogeneity of IGM and the limitations of retrospective, single-center studies, there is an urgent need for well-designed prospective trials and multicenter registries. Standardized diagnostic criteria, validated classification systems, and comparative effectiveness studies of medical versus minimally invasive treatments would greatly improve clinical decision-making and help define evidence-based management pathways.

IGM is a complex disease that continues to pose diagnostic and therapeutic challenges despite decades of investigation. Its resemblance to breast cancer and other granulomatous diseases of the breast and its varied pathophysiology make it challenging to diagnose. However, based on the current literature,

histopathological findings remain the most reliable diagnostic method.

Topical steroid therapy is often used for mild cases of IGM, whereas surgical intervention is typically reserved for severe cases. However, the classification of IGM into mild, moderate, and severe groups remains a topic of debate, and there is still much to learn about prognostic factors for this disease.

A significant limitation in the field of IGM is the need for more guidelines and a global database for the disease. While a wealth of information is available, more research is necessary to understand this illness comprehensively. Overall, there is still much to be learned about IGM, and continued efforts are needed to develop effective diagnostic and treatment strategies for patients.

Footnotes

Authorship Contributions

Concept: P.H.M., S.N.; Design: P.H.M., S.N., A.H., D.S., K.H.; Data Collection or Processing: P.H.M., M.S.C.M., K.H., F.M.A., H.M., R.M.; Analysis or Interpretation: S.N., A.H., M.B., D.S., K.H.; Literature Search: A.H., M.B., M.S.C.M., H.M., R.M.; Writing: P.H.M., S.N., M.B., F.M.A., H.M., R.M.

Conflict of Interest: No conflict of interest declared by the authors.

Financial Disclosure: The authors declare that this study received no financial disclosure.

References

1. Kessler E, Wolloch Y. Granulomatous mastitis: a lesion clinically simulating carcinoma. Am J Clin Pathol. 1972; 58: 642-646. (PMID: 4674439) [Crossref]
2. Martinez-Ramos D, Simon-Monterde L, Suelves-Piqueres C, Queralt-Martin R, Granel-Villach L, Laguna-Sastre JM, et al. Idiopathic granulomatous mastitis: a systematic review of 3060 patients. Breast J. 2019; 25: 1245-1250. (PMID: 31273861) [Crossref]
3. Baslaim MM, Khayat HA, Al-Amoudi SA. Idiopathic granulomatous mastitis: a heterogeneous disease with variable clinical presentation. World J Surg. 2007; 31: 1677-1681. (PMID: 17541683) [Crossref]
4. Haitz K, Ly A, Smith G. Idiopathic granulomatous mastitis. Cutis. 2019; 103: 38-42. (PMID: 30758334)
5. Chandanwale S, Naragude P, Shetty A, Sawadkar M, Raj A, Bhide A, et al. Cytomorphological spectrum of granulomatous mastitis: a study of 33 cases. Eur J Breast Health. 2020; 16: 146-151. (PMID: 32285037) [Crossref]
6. Brown KL, Tang PH. Postlactational tumoral granulomatous mastitis: a localized immune phenomenon. Am J Surg. 1979; 138: 326-329. (PMID: 464240) [Crossref]
7. Bani-Hani KE, Yaghan RJ, Matalka II, Shatnawi NJ. Idiopathic granulomatous mastitis: time to avoid unnecessary mastectomies. Breast J. 2004; 10: 318-322. (PMID: 15239790) [Crossref]
8. Akbari M, Negahi A, Dabbagh N, Salimi Kordasiabi AH, Zarean Shahraki S, Akbari ME. Idiopathic granulomatous mastitis (IGM): clinical features and non-surgical management. Int J Cancer Manag. 2023; 16: e119945. [Crossref]
9. Cserni G, Szajki K. Granulomatous lobular mastitis following drug-induced galactorrhea and blunt trauma. Breast J. 1999; 5: 398-403. (PMID: 11348321) [Crossref]
10. Sheybani F, Naderi HR, Gharib M, Sarvghad M, Mirfeizi Z. Idiopathic granulomatous mastitis: Long-discussed but yet-to-be-known. Autoimmunity. 2016; 49: 236-239. (PMID: 26829298) [Crossref]
11. Uysal E, Soran A, Sezgin E; Granulomatous Mastitis Study Group. Factors related to recurrence of idiopathic granulomatous mastitis: what do we learn from a multicentre study? ANZ J Surg. 2018; 88: 635-639. (PMID: 28749045) [Crossref]
12. Ciftci AB, Bük ÖF, Yemez K, Polat S, Yazıcıoğlu İM. Risk factors and the role of the albumin-to-globulin ratio in predicting recurrence among patients with idiopathic granulomatous mastitis. J Inflamm Res. 2022; 15: 5401-5412. (PMID: 36158516) [Crossref]
13. Rajkumar SV, Kyle RA, Therneau TM, Melton LJ 3rd, Bradwell AR, Clark RJ, et al. Serum free light chain ratio is an independent risk factor for progression in monoclonal gammopathy of undetermined significance. Blood. 2005; 106: 812-817. (PMID: 15855274) [Crossref]
14. Schelfout K, Tjalma WA, Cooremans ID, Coeman DC, Colpaert CG, Buytaert PM. Observations of an idiopathic granulomatous mastitis. Eur J Obstet Gynecol Reprod Biol. 2001; 97: 260-262. (PMID: 11451563) [Crossref]
15. Al-Khaffaf B, Knox F, Bundred NJ. Idiopathic granulomatous mastitis: a 25-year experience. J Am Coll Surg. 2008; 206: 269-273. (PMID: 18222379) [Crossref]
16. Asoglu O, Ozmen V, Karanlik H, Tunaci M, Cabioglu N, Igci A, et al. Feasibility of surgical management in patients with granulomatous mastitis. Breast J. 2005; 11: 108-114. (PMID: 15730456) [Crossref]
17. Gurleyik G, Aktekin A, Aker F, Karagulle H, Saglamc A. Medical and surgical treatment of idiopathic granulomatous lobular mastitis: a benign inflammatory disease mimicking invasive carcinoma. J Breast Cancer. 2012; 15: 119-123. (PMID: 22493638) [Crossref]
18. Oran EŞ, Gürdal SÖ, Yankol Y, Öznur M, Calay Z, Tunacı M, et al. Management of idiopathic granulomatous mastitis diagnosed by core biopsy: a retrospective multicenter study. Breast J. 2013; 19: 411-418. (PMID: 23663101) [Crossref]
19. Gautier N, Lalonde L, Tran-Thanh D, El Khoury M, David J, Labelle M, et al. Chronic granulomatous mastitis: imaging, pathology and management. Eur J Radiol. 2013; 82: e165-e175. (PMID: 23200627) [Crossref]
20. Yaghan RJ, Ayoub NM, Shenawi HM, Yaghan LR. Idiopathic granulomatous mastitis in the male population: a clinical analysis of 13 reported cases. Breast J. 2020; 26: 1481-1482. (PMID: 32034843) [Crossref]
21. Rowe PH. Granulomatous mastitis associated with a pituitary prolactinoma. Br J Clin Pract. 1984; 38: 32-34. (PMID: 6538433) [Crossref]
22. Going JJ, Anderson TJ, Wilkinson S, Chetty U. Granulomatous lobular mastitis. J Clin Pathol. 1987; 40: 535-540. (PMID: 3584506) [Crossref]
23. Erhan Y, Veral A, Kara E, Ozdemir N, Kapkac M, Ozdedeli E, et al. A clinicopthologic study of a rare clinical entity mimicking breast carcinoma: idiopathic granulomatous mastitis. Breast. 2000; 9: 52-56. (PMID: 14731585) [Crossref]
24. Cohen C. Granulomatous mastitis. A review of 5 cases. S Afr Med J. 1977; 52: 14-16. (PMID: 560723) [Crossref]
25. Tavassoli FA. Pathology of the breast: McGraw Hill Professional; 1999. [Crossref]
26. Coşkun T, Kara E, Kaya Y, Güler Y, Kandiloğlu AR, Göktan C. Granülomatöz mastit: cerrahi tedavi-rekürrens ilişkisi. Meme Sağlığı Dergisi. 2006; 2: 26-30. [Crossref]

27. Ozel L, Unal A, Unal E, Kara M, Erdoğdu E, Krand O, et al. Granulomatous mastitis: is it an autoimmune disease? Diagnostic and therapeutic dilemmas. Surg Today. 2012; 42: 729-733. (PMID: 22068681) [Crossref]

28. Bes C, Soy M, Vardi S, Sengul N, Yilmaz F. Erythema nodosum associated with granulomatous mastitis: report of two cases. Rheumatol Int. 2010; 30: 1523-1525. (PMID: 19701754) [Crossref]

29. Salesi M, Karimifar M, Salimi F, Mahzouni P. A case of granulomatous mastitis with erythema nodosum and arthritis. Rheumatol Int. 2011; 31: 1093-1095. (PMID: 20012050) [Crossref]

30. Taniguchi Y, Kagawa T, Ishibashi A, Horino T, Kumon Y, Terada Y. Weber-Christian disease associated with granulomatous mastitis: a variant type of Weber-Christian disease? Mod Rheumatol. 2011; 21: 228-231. (PMID: 20922452) [Crossref]

31. Letourneux C, Diemunsch P, Korganow AS, Akladios CY, Bellocq JP, Mathelin C. First report of granulomatous mastitis associated with Sjögren's syndrome. World J Surg Oncol. 2013; 11: 268. (PMID: 24112140) [Crossref]

32. Altintoprak F, Karakece E, Kivilcim T, Dikicier E, Cakmak G, Celebi F, et al. Idiopathic granulomatous mastitis: an autoimmune disease? ScientificWorldJournal. 2013; 2013: 148727. (PMID: 24082849) [Crossref]

33. Thornton JW, Argenta LC, McClatchey KD, Marks MW. Studies on the endogenous flora of the human breast. Ann Plast Surg. 1988; 20: 39-42. (PMID: 3341714) [Crossref]

34. Pereira FA, Mudgil AV, Macias ES, Karsif K. Idiopathic granulomatous lobular mastitis. Int J Dermatol. 2012; 51: 142-151. (PMID: 22250621) [Crossref]

35. Taylor GB, Paviour SD, Musaad S, Jones WO, Holland DJ. A clinicopathological review of 34 cases of inflammatory breast disease showing an association between corynebacteria infection and granulomatous mastitis. Pathology. 2003; 35: 109-119. (PMID: 12745457) [Crossref]

36. Paviour S, Musaad S, Roberts S, Taylor G, Taylor S, Shore K, et al. Corynebacterium species isolated from patients with mastitis. Clin Infect Dis. 2002; 35: 1434-1440. (PMID: 12439810) [Crossref]

37. Ang LM, Brown H. Corynebacterium accolens isolated from breast abscess: possible association with granulomatous mastitis. J Clin Microbiol. 2007; 45: 1666-1668. (PMID: 17344355) [Crossref]

38. Funke G, von Graevenitz A, Clarridge JE 3rd, Bernard KA. Clinical microbiology of coryneform bacteria. Clin Microbiol Rev. 1997; 10: 125-159. (PMID: 8993861) [Crossref]

39. Stary CM, Lee YS, Balfour J. Idiopathic granulomatous mastitis associated with corynebacterium sp. Infection. Hawaii Med J. 2011; 70: 99-101. (PMID: 21857740) [Crossref]

40. Mathelin C, Riegel P, Chenard MP, Tomasetto C, Brettes JP. Granulomatous mastitis and corynebacteria: clinical and pathologic correlations. Breast J. 2005; 11: 357. (PMID: 16174160) [Crossref]

41. Naraynsingh V, Hariharan S, Dan D, Harnarayan P, Teelucksingh S. Conservative management for idiopathic granulomatous mastitis mimicking carcinoma: case reports and literature review. Breast Dis. 2010; 31: 57-60. (PMID: 21109722) [Crossref]

42. Azizi A, Prasath V, Canner J, Gharib M, Sadat Fattahi A, Naser Forghani M, et al. Idiopathic granulomatous mastitis: management and predictors of recurrence in 474 patients. Breast J. 2020; 26: 1358-1362. (PMID: 32249491) [Crossref]

43. Hovanessian Larsen LJ, Peyvandi B, Klipfel N, Grant E, Iyengar G. Granulomatous lobular mastitis: imaging, diagnosis, and treatment. AJR Am J Roentgenol. 2009; 193: 574-581. (PMID: 19620458) [Crossref]

44. Oztekin PS, Durhan G, Nercis Kosar P, Erel S, Hucumenoglu S. Imaging findings in patients with granulomatous mastitis. Iran J Radiol. 2016; 13: e33900. (PMID: 27853497) [Crossref]

45. Dursun M, Yilmaz S, Yahyayev A, Salmaslioglu A, Yavuz E, Igci A, et al. Multimodality imaging features of idiopathic granulomatous mastitis: outcome of 12 years of experience. Radiol Med. 2012; 117: 529-538. (PMID: 22020426) [Crossref]

46. Barreto DS, Sedgwick EL, Nagi CS, Benveniste AP. Granulomatous mastitis: etiology, imaging, pathology, treatment, and clinical findings. Breast Cancer Res Treat. 2018; 171: 527-534. (PMID: 29971624) [Crossref]

47. Fazzio RT, Shah SS, Sandhu NP, Glazebrook KN. Idiopathic granulomatous mastitis: imaging update and review. Insights Imaging. 2016; 7: 531-539. (PMID: 27221974) [Crossref]

48. Lapegue F, Faruch-Bilfeld M, Demondion X, Apredoaei C, Bayol MA, Artico H, et al. Ultrasonography of the brachial plexus, normal appearance and practical applications. Diagn Interv Imaging. 2014; 95: 259-275. (PMID: 24603038) [Crossref]

49. Yilmaz E, Lebe B, Usal C, Balci P. Mammographic and sonographic findings in the diagnosis of idiopathic granulomatous mastitis. Eur Radiol. 2001; 11: 2236-2240. (PMID: 11702165) [Crossref]

50. Han BK, Choe YH, Park JM, Moon WK, Ko YH, Yang JH, et al. Granulomatous mastitis: mammographic and sonographic appearances. AJR Am J Roentgenol. 1999; 173: 317-320. (PMID: 10430126) [Crossref]

51. Al-Khawari HA, Al-Manfouhi HA, Madda JP, Kovacs A, Sheikh M, Roberts O. Radiologic features of granulomatous mastitis. Breast J. 2011; 17:645-650. (PMID: 21929558) [Crossref]

52. Yilmaz R, Demir AA, Kaplan A, Sahin D, Ozkurt E, Dursun M, et al. Magnetic resonance imaging features of idiopathic granulomatous mastitis: is there any contribution of diffusion-weighted imaging in the differential diagnosis? Radiol Med. 2016; 121: 857-866. (PMID: 27406630) [Crossref]

53. Mathew M, Siwawa P, Misra S. Idiopathic granulomatous mastitis: an inflammatory breast condition with review of the literature. BMJ Case Rep. 2015; 2015: bcr2014208086. (PMID: 25739797) [Crossref]

54. Martínez-Parra D, Nevado-Santos M, Meléndez-Guerrero B, García-Solano J, Hierro-Guilmain CC, Pérez-Guillermo M. Utility of fine-needle aspiration in the diagnosis of granulomatous lesions of the breast. Diagn Cytopathol. 1997; 17: 108-114. (PMID: 9258617) [Crossref]

55. Yip CH, Jayaram G, Swain M. The value of cytology in granulomatous mastitis: a report of 16 cases from Malaysia. Aust N Z J Surg. 2000; 70: 103-105. (PMID: 10711470) [Crossref]

56. Doughty JC, Wilson CR, Mallon EA. Granulomatous mastitis can mimic breast cancer on clinical, radiological or cytological examination: a cautionary tale. Breast. 2004; 13: 261; author reply 262. (PMID: 15177435) [Crossref]

57. Lai EC, Chan WC, Ma TK, Tang AP, Poon CS, Leong HT. The role of conservative treatment in idiopathic granulomatous mastitis. Breast J. 2005; 11: 454-456. (PMID: 16297091) [Crossref]

58. Bouton ME, Jayaram L, O'Neill PJ, Hsu CH, Komenaka IK. Management of idiopathic granulomatous mastitis with observation. Am J Surg. 2015; 210: 258-262. (PMID: 25746911) [Crossref]

59. Hur SM, Cho DH, Lee SK, Choi MY, Bae SY, Koo MY, et al. Experience of treatment of patients with granulomatous lobular mastitis. J Korean Surg Soc. 2013; 85: 1-6. (PMID: 23833753) [Crossref]

60. Davis J, Cocco D, Matz S, Hsu CH, Brown MJ, Lee J, et al. Re-evaluating if observation continues to be the best management of idiopathic granulomatous mastitis. Surgery. 2019; 166: 1176-1180. (PMID: 31400951) [Crossref]

61. Aghajanzadeh M, Hassanzadeh R, Alizadeh Sefat S, Alavi A, Hemmati H, Esmaeili Delshad MS, et al. Granulomatous mastitis: presentations, diagnosis, treatment and outcome in 206 patients from the north of Iran. Breast. 2015; 24: 456-460. (PMID: 25935828) [Crossref]

62. Li J. Diagnosis and treatment of 75 patients with idiopathic lobular granulomatous mastitis. J Invest Surg. 2019; 32: 414-420. (PMID: 29381437) [Crossref]

63. Benson JR, Dumitru D. Idiopathic granulomatous mastitis: presentation, investigation and management. Future Oncol. 2016; 12: 1381-1394. (PMID: 27067146) [Crossref]

64. Yin Y, Liu X, Meng Q, Han X, Zhang H, Lv Y. Idiopathic granulomatous mastitis: etiology, clinical manifestation, diagnosis and treatment. J Invest Surg. 2022; 35: 709-720. (PMID: 33691563) [Crossref]

65. Akahane K, Tsunoda N, Kato M, Noda S, Shimoyama Y, Ishigakis S, et al. Therapeutic strategy for granulomatous lobular mastitis: a clinicopathological study of 12 patients. Nagoya J Med Sci. 2013; 75: 193-200. (PMID: 24640175) [Crossref]

66. Wolfrum A, Kümmel S, Theuerkauf I, Pelz E, Reinisch M. Granulomatous mastitis: a therapeutic and diagnostic challenge. Breast Care (Basel). 2018; 13: 413-418. (PMID: 30800035) [Crossref]

67. Sakurai K, Fujisaki S, Enomoto K, Amano S, Sugitani M. Evaluation of follow-up strategies for corticosteroid therapy of idiopathic granulomatous mastitis. Surg Today. 2011; 41: 333-337. (PMID: 21365412) [Crossref]

68. Azlina AF, Ariza Z, Arni T, Hisham AN. Chronic granulomatous mastitis: diagnostic and therapeutic considerations. World J Surg. 2003; 27: 515-518. (PMID: 12715214) [Crossref]

69. Montazer M, Dadashzadeh M, Moosavi Toomatari SE. Comparison of the outcome of low dose and high-dose corticosteroid in the treatment of idiopathic granulomatous mastitis. Asian Pac J Cancer Prev. 2020; 21: 993-996. (PMID: 32334460) [Crossref]

70. Gunduz Y, Altintoprak F, Tatli Ayhan L, Kivilcim T, Celebi F. Effect of topical steroid treatment on idiopathic granulomatous mastitis: clinical and radiologic evaluation. Breast J. 2014; 20: 586-591. (PMID: 25228089) [Crossref]

71. Altintoprak F, Kivilcim T, Yalkin O, Uzunoglu Y, Kahyaoglu Z, Dilek ON. Topical steroids are effective in the treatment of idiopathic granulomatous mastitis. World J Surg. 2015; 39: 2718-2723. (PMID: 26148520) [Crossref]

72. Çetin K, Sıkar HE, Göret NE, Rona G, Barışık NÖ, Küçük HF, et al. Comparison of topical, systemic, and combined therapy with steroids on idiopathic granulomatous mastitis: a prospective randomized study. World J Surg. 2019; 43: 2865-2873. (PMID: 31297582) [Crossref]

73. Akbulut S, Yilmaz D, Bakir S. Methotrexate in the management of idiopathic granulomatous mastitis: review of 108 published cases and report of four cases. Breast J. 2011; 17: 661-668. (PMID: 21951547) [Crossref]

74. Çetin K, Sıkar HE, Güllüoğlu BM. Idiopathic granulomatous mastitis with erythema nodosum: Is it a variant of clinical presentation indicating treatment resistance? A retrospective cohort study. Breast J. 2020; 26: 1645-1651. (PMID: 32562354) [Crossref]

75. Raj N, Macmillan RD, Ellis IO, Deighton CM. Rheumatologists and breasts: immunosuppressive therapy for granulomatous mastitis. Rheumatology (Oxford). 2004; 43: 1055-1056. (PMID: 15258294) [Crossref]

76. Peña-Santos G, Ruiz-Moreno JL. Mastitis granulomatosa idiopática tratada con esteroides y metotrexato [Idiopathic granulomatous mastitis treated with steroids and methotrexate]. Ginecol Obstet Mex. 2011; 79: 373-376. Spanish. (PMID: 21966829) [Crossref]

77. Haddad M, Sheybani F, Arian M, Gharib M. Methotrexate-based regimen as initial treatment of patients with idiopathic granulomatous mastitis. Breast J. 2020; 26: 325-327. (PMID: 31495030) [Crossref]

78. Azizi A, Prasath V, Canner J, Gharib M, Sadat Fattahi A, Naser Forghani M, et al. Idiopathic granulomatous mastitis: management and predictors of recurrence in 474 patients. Breast J. 2020; 26: 1358-1362. (PMID: 32249491) [Crossref]

79. Lei X, Chen K, Zhu L, Song E, Su F, Li S. Treatments for idiopathic granulomatous mastitis: systematic review and meta-analysis. Breastfeed Med. 2017; 12: 415-421. (PMID: 28731822) [Crossref]

80. Wang J, Zhang Y, Lu X, Xi C, Yu K, Gao R, et al. Idiopathic granulomatous mastitis with skin rupture: a retrospective cohort study of 200 patients who underwent surgical and nonsurgical treatment. J Invest Surg. 2021; 34: 810-815. (PMID: 31818161) [Crossref]

81. Shin YD, Park SS, Song YJ, Son SM, Choi YJ. Is surgical excision necessary for the treatment of Granulomatous lobular mastitis? BMC Womens Health. 2017; 17: 49. (PMID: 28738795) [Crossref]

82. Yabanoğlu H, Çolakoğlu T, Belli S, Aytac HO, Bolat FA, Pourbagher A, et al. A comparative study of conservative versus surgical treatment protocols for 77 patients with idiopathic granulomatous mastitis. Breast J. 2015; 21: 363-369. (PMID: 25858348) [Crossref]

83. Alrayes A, Almarzooq R, Abdulla HA. Surgical treatment of granulomatous mastitis: our experience in Bahrain. Breast J. 2019; 25: 958-962. (PMID: 31187555) [Crossref]