Aetiology of idiopathic granulomatous mastitis

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Abstract

Idiopathic granulomatous mastitis is a rare chronic inflammatory lesion of the breast that can clinically and radiographically mimic breast carcinoma. The most common clinical presentation is an unilateral, discrete breast mass, nipple retraction and even a sinus formation often associated with an inflammation of the overlying skin. The etiology of idiopathic granulomatous mastitis is still obscure. Its treatment remains controversial. The cause may be the autoimmune process, infection, a chemical reaction associated with oral contraceptive pills, or even lactation. Various factors, including hormonal imbalance, autoimmunity, unknown microbiological agents, smoking and α 1-antitrypsin deficiency have been suggested to play a role in disease aetiology. In this review, causing factors in the aetiology of idiopathic granulomatous mastitis are reviewed in detail.

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Key words: Mastitis; Granulomatous mastitis; Idiopathic granulomatous mastitis; Granulomatous lobular mastitis; Inflammation
parasitic and fungal infections, Wegener’s granulomatosis, giant-cell arthritis, polyartheritis nodosum, sarcoidosis, foreign body reaction, etc.) will support the diagnosis of IGM.

In this article, we discuss factors that may play a role in the aetiology of IGM.

DEFINITION

Idiopathic granulomatous mastitis (GM) is a rare, benign, chronic, inflammatory lesion of the breast, and its aetiology has not been fully elucidated. It was defined for the first time in 1972 by Kessler and Woollock and was described in detail in 1977 with a five-case series by Cohen.[1,2]

GM is generally divided into two main groups of specific and non-specific. The term “specific GM” refers to conditions for which the aetiologic factor can be identified, whether an isolated inflammatory event only applies to the breast, or the breast is involved in a systemic inflammatory event. Nonspecific GM is also known as idiopathic granulomatous mastitis or granulomatous lobular mastitis, which generally refers to conditions that can lead to a granulomatous reaction in the breast or conditions for which the aetiological factors cannot be determined.

Chronic granulomatous inflammation constitutes 24% of all inflammatory events of the breast that are histopathologically defined.[3] All factors that lead to granulomatous inflammation in the breasts are presented in Table 1.[8]

GENERAL REMINDERS

While IGM mostly emerges in young-middle age women (third and fourth decades), the age range that has been reported in the literature (11-83 years) is considerably wider.[8,10,11] IGM is usually seen within a couple of years after giving birth, and the majority of patients have a history of at least one live birth and breast feeding.[8] In contrast, specific GM is frequently seen in Asian and African countries and can be detected at any age.[9]

IGM may present with clinical findings that mimic the two endpoints of breast diseases such as breast abscess and breast cancer.[9] A palpable mass in the breast is the most common complaint, but nipple retraction, hyperaemia in breast skin, oedema, ulceration and fistule development during the chronic period are also potential complaints[9]. Systemic symptoms such as fever are generally not present[9]. While the incidence is the same in both breasts, the lesion is usually unilateral and cases with bilateral involvement have been reported only rarely[8,10,11].

PATHOGENESIS

The pathogenesis of IGM is not exactly known, but different steps occur in the disease pathogenesis. One of these steps is nonspecific lobulitis, which involves multiple lobules, and causes reactive lymphoplasmocytic infiltration. A granulomatous formation with central suppurative necrosis occasionally occurs because of lobule deformation. Abscesses develop because of an increase in the number of these foci.[12]

Some studies have indicated the similarity between IGM and granulomatous inflammation of the testicles or the thyroid gland when IGM was defined for the first time[9]. Considering that mechanical factors are responsible for the formation of granulomas of the thyroid gland in multifocal granulomatous thyroiditis (palpation thyroiditis), the possibility that trauma represents another stage in IGM pathogenesis should not be disregarded.[9]

A process starting with non-puerperal secretion has been proposed as the most rational theory for the pathogenesis of IGM. A hormonal imbalance due to a deviation in the oestrogen-progesterone ratio or hyperprolactinemia is believed to cause this secretion and inflammation. Ductal ectasia occurs due to the intra-ductal accumulation of a protein-rich secretion. Permanent inflammation occurs following perforation of the duct and contact between the secretion and stromal cells. The accumulation of secretion, ductal ectasia, galactoporisitis (intraductal inflammation) and chronic GM are steps in the pathophysiological process. Autoimmunity against a secretion that is extravasated from the lobules is also considered to cause this event.[8,10,11].

AETIOLOGY

The aetiology of IGM remains unclear. Various factors, including hormonal imbalance, autoimmunity, unknown microbiological agents, smoking and α1-antitrypsin deficiency have been suggested to play a role in disease aetiology.

α1-antitrypsin deficiency

α1-antitrypsin (AAT) is a glycoprotein synthesised by hepatic cells. Similar to anti-thrombin 3, ovalbumin and thyroid-binding globulin, AAT is a member of the serine-protease inhibitor family. Its primary function is to

| Table 1  Causes for granulomatous inflammatory reaction in the breasts |
|-----------------|-------------------------------|
| Infectious      | Mycobacterium tuberculosis   |
|                 | Blastomycosis                 |
|                 | Cryptococcosis                |
|                 | Histoplasmosis                |
|                 | Actinomyces                   |
|                 | Filariar infection            |
|                 | Corynebacterium               |
| Autoimmune process | Wegener granulomatosis       |
| Duct ectasis    | Giant cell arteritis          |
|                 | Foreign body reaction         |
|                 | Plasma cell mastitis          |
|                 | Subareolar granuloma          |
|                 | Periductal mastitis           |
| Diabetes mellitus|                               |
| Sarcoïdosis     |                               |
| Fat necrosis    |                               |
| Idiopathic      |                               |
prevent the destructive effects of proteases secreted from activated neutrophils (proteinase 3, elastin and cathepsin G). Because AAT level is elevated during inflammation, it is also accepted as an acute phase reactant. Deficiency in AAT leads primarily to lung and liver pathologies. In their case presentation in 2001, Scheflout et al. demonstrated AAT deficiency in a 37-year-old female patient diagnosed with IGM. According to that study, the authors did not determine any other aetiologic factors, and suggested that AAT deficiency could be the aetiologic factor; however, further studies were not performed.

Oral contraceptives
The secretion theory has an important place in the pathophysiology of IGM. Oral contraceptives (OCS) have been considered a potential aetiological factor, as they increase breast secretion. However, a significant association between OCS and IGM has not been determined. Oran et al. found 10 cases (10/46; 21.7%) that had a history of OCS use; Gurleyik et al. found eight cases (8/19, 42.1%) that had a history of OCS use; and Al-Khaffaf et al. found five cases (5/18, 27.7%) that had a history of OCS use. In contrast, Baslaim et al. reported that none of 20 patients had a history of OCS use. Bani-Hani et al. found that only two of 24 cases (8.3%) had a history of OCS use, and Asoglu et al. found that only two of 18 cases (11.1%) had a history of OCS use. In conclusion, the association between IGM and OCS use has been reported to range between 0%-42%.

Gestation, birth, and breast-feeding
Given that IGM is usually detected in women < 50 years of age, and frequently involves a recent history of birth or breast-feeding, these factors have been considered in the disease aetiology. Hormonal alterations during these processes, secretion, and inflammation have an effect on disease pathophysiology. Bani-Hani et al. carried out a study on 24 cases, and found that four had an active gestation, four had a history of birth and breast-feeding within 6 months and only two cases did not have a history of gestation. According to a study by Baslaim et al., all cases had a history of gestation and breast-feeding, whereas two cases were actively breast-feeding, and one case had an active gestation. Similarly, Gurleyik et al. determined that four of 19 cases had a history of active breast-feeding, and the remaining 15 cases had a history of breast-feeding. Moreover, Oran et al. reported that only three of 46 cases were nulliparous. Gautier et al. conducted a case series study on 11 cases and emphasised that all cases except one male case had a history of birth and lactation within the past 5 years.

While almost all studies reported a history of parity, various studies have failed to explain the timing of the parity. It is expected that cases with IGM, which is a reproductive age disorder, have a history of gestation and breast feeding, as gestation occurs between the ages of 20-40 years. In addition to the male case, cases with a wide age range (11-83 years) in the literature make it difficult to hold only gestation, birth and breast feeding responsible for the aetiology of IGM.

Hyperprolactinemia
Considering the secretion theory, hyperprolactinemia has also been considered responsible for the pathogenesis of IGM, similar to other hormonal disorders. In a case presentation in 1984, Rowe determined co-morbid prolactinoma in an IGM case. However, future studies did not provide prolactin levels in detail. Bani-Hani et al. analysed prolactin levels in seven of 24 cases and found elevated prolactin levels in one patient (4.1%). Erhan et al. carried out a case-series study on 18 women and reported recurrence in three cases (16%), and identified hyperprolactinemia in two of these patients.

Smoking
While smoking is among the factors considered in the disease aetiology, a definitive association between smoking and IGM has not yet been established. According to a study by Asoglu et al., 14 of 18 cases (77.8%) had a history of smoking, whereas Baslaim et al. reported that none of their 20 cases had a history of smoking. In addition, the smoking rate was 34.8% according to Oran et al., 16.7% according to Al-Khaffaf et al., and 50% according to Ozel et al.

Autoimmunity
A hypothesis that suggests an immunological basis for IGM has received considerable attention. Literature findings, including a good response to steroid and immunosuppressive treatment, patients who had recurrence after surgery showing a good response to steroid treatment, patients with extramammary involvement (such as erythema nodosum, or arthritis) and the demonstration of T-lymphocyte dominance in immunohistochemical studies support the autoimmunity hypothesis. Ozel et al. conducted a study on eight cases and found that six were positive for rheumatoid factor (RF), and two were positive for anti-nuclear antibody (ANA) and anti-double stranded DNA (anti-dsDNA). In that study, surgery was the preferred treatment option for all patients, and the authors reported recurrence in two patients who were RF-, ANA- and anti-dsDNA-positive, but obtained a positive response after steroid treatment. Erhan et al. conducted an immunohistochemical evaluation, and determined that 14 of 18 cases had T-cell dominance, and this finding was interpreted as an autoimmune pathophysiological outcome that progressed with reactive T-cell-mediated inflammation and centriflobular granulomas against ductal damage. Furthermore, two IGM cases with erythema nodosum, one IGM case with erythema nodosum and arthritis, one IGM case with Weber-Christian disease and one IGM case with Sjögren’s syndrome have been reported in the literature. However, cases with a co-morbid autoimmune disorder constitute only a minor fraction of all cases. In contrast to these studies that support the autoimmune hypothesis, classical serological tests, which are used for au-
immune disorders such as ANA and RF, reveal different results in patients with IGM. Asoglu et al\cite{21} conducted a case-series study on 18 cases and determined that all cases were negative for ANA and RF. We conducted a study in our clinic to investigate the autoimmunity hypothesis for IGM aetiology, and evaluated ANA and extractable nuclear antibody levels in 26 cases, but we did not obtain results to support the autoimmunity hypothesis\cite{37}.

**Microbiological agents**

The normal endogenous bacteria flora of the breast is similar to the skin flora. Dominant organisms include coagulase-negative streptococci, Propionibacterium sp. and Corynebacterium sp. These findings have been proven through nipple discharge and breast tissue cultures that were collected during mammoplasty\cite{38}. These bacteria are considered to go deeper into the breast tissue via the ductal system\cite{13}.

*Corynebacteria* cause mastitis in livestock. However, these bacteria are not expected pathogens in humans\cite{13}. These bacteria became the centre of attention in 2003, with detection of corynebacteria in 34 IGM cases by Taylor et al\cite{39}.

*Corynebacteria* are Gram-positive bacteria and members of the skin flora. It is hard to distinguish whether these organisms cause infection, colonisation or contamination\cite{40}. Despite the difficulty in distinguishing outcome, it is significant to detect purulent matter in an abscess or > 10^7 CFU/mL dominant *Corynebacterium sp.*\cite{41}. According to a study by Funke et al\cite{42}, these bacteria could be a possible factor if: (1) a Gram-positive bacillus accompanying polymorphonuclear leukocytes is present; or (2) a *Corynebacterium sp.* is detected in a tissue that is expected to be sterile under normal conditions.

Four different Corynebacterium species have been detected in IGM cases. *Corynebacterium kroppenstedtii* (*C. kroppenstedtii*) is the most frequently observed species, and is different from other corynebacteria due to its lipophilic nature and positive esculin test\cite{39,40}.

Taylor et al\cite{39} conducted a study of 62 patients who were histologically diagnosed with GM, and detected *Corynebacterium* in 34 patients (54.8%). A comparison among the remaining 28 cases showed that fever and neutrophilia were more frequently observed in cases that were bacteria-positive, and they had more frequent fistule formation. *C. kroppenstedtii* was the most frequently observed species (14 patients; 41.1%) in that study.

Paviour et al\cite{40} isolated *Corynebacterium* from breast tissue in 24 cases, carried out a histopathological evaluation in 12 of these cases and diagnosed nine cases with IGM. Similarly, *C. kroppenstedtii* was the most frequently isolated species in that study; *C. amycolatum* and *C. tuberculostearicum* were other identified species. In that study, a 3-week intravenous penicillin treatment was tested on one patient; however, when the expected benefit was not observed, the treatment was switched to doxycycline (100 mg, oral), which has better fat solubility. The authors reported that there was no need for surgery after this treatment.

Case presentations in which *Corynebacterium sp.* have been detected are also present in the literature\cite{41,43,44}. A specific species was not reported in two of these studies, whereas Ang et al\cite{41} reported that they isolated *C. accolens*. All three studies stated that antibiotherapy was effective for treatment.

In our clinic, we carried out a study on 45 patients with IGM and 34 bacteria using a universal DNA primer, but we did not detect positivity for any microbiological agent (unpublished data).

**Ethnicity**

During our search of GM in the PubMed database (1995-2014), we searched the terms “idiopathic granulomatous mastitis”, “granulomatous lobular mastitis” and...
“granulomatous mastitis” and found approximately 200 articles. We hypothesised that an evaluation based on the location of the centres in which the authors worked would provide a rough estimate of the distribution of the cases. While most of these studies were case presentations, we found that larger case series frequently originated from the Mediterranean region and the developing countries in Asia. Some authors have considered that undiagnosed tuberculosis cases might lead to GM. According to our search on the PubMed database, the highest number of cases has been reported in Turkey (> 200 cases). This is followed by China (129 cases) and South Korea (128 cases). France had the highest number of cases (55 cases) among European countries, and no other country exceeded 50 cases. In contrast, we found 126 cases in the United States. The total number of cases recorded per country is presented in Table 2 and Figure 1.

According to a Centers for Disease Control and Prevention report, which was published in Morbidity and Mortality Weekly Report in 2009, seven cases were detected in Indiana between 2006 and 2008, and six of these cases were born in Mexico and had a Hispanic background. According to the report, this series was the most comprehensive case series reported in the United States49. However, Larsen et al. published a study on 54 cases in the same year, but the authors did not evaluate ethnicity46. Gautier et al.50 carried out a study on 11 cases in Canada and reported that three cases were French, two cases were Canadian of French origin, two cases were British origin, two cases were Latin American and one case was Russian. Furthermore, Omaninpour et al.50 reported a series of 43 cases in Iran, Bani-Hani et al.51 reported a series of 24 cases in Jordan and Baslaim et al.52 reported a series of 20 cases in Saudi Arabia. In Turkey, different IGM series have been reported by Asoglu et al.21 (18 cases), Ozel et al.41 (8 cases), Gurleyik et al.53 (46 cases) and Altintoprak et al.57 (26 cases). These findings indicate that a previous comprehensive evaluation of ethnicity does not exist, and that more elaborate studies on this topic are required.

Table 2 The distribution of idiopathic granulomatous mastitis cases that were reported in PubMed since 1995 according to country

| > 100 cases | 20-100 cases | 5-20 cases | < 5 cases |
|-------------|-------------|-----------|---------|
| Turkey: > 200 | Saudi Arabia: 96 | Spain: 17 | Netherlands: 4 |
| China: 129 | France: 55 | Canada: 15 | Israel: 4 |
| South Korea: 128 | United Kingdom: 48 | Pakistan: 14 | Austria: 3 |
| United States: 126 | Iran: 46 | Sri Lanka: 12 | Belgium: 3 |
| | Brunet: 43 | Tunis: 12 | Taiwan: 2 |
| Malaysia: 42 | Saudi: 11 | Caribbean: 2 |
| India: 36 | Australia: 9 | Peru: 1 |
| Japan: 33 | Italy: 7 | Nigeria: 1 |
| Morocco: 30 | | |
| Jordan: 25 | | |
| New Zealand: 24 | | |
| Mexico: 21 | | |
| Oman: 20 | | |

CONCLUSION

In conclusion, while several factors have been considered as potential aetiologic factors, these factors are not the primary aetiological factors, but rather “secondary factors” that can accompany the process once the primary factor triggers the event, or contribute to the acceleration of the ongoing process. Given that: (1) a higher number of cases are being reported from certain geographical locations; and (2) patients respond positively to steroid treatment, we believe that the “ethnicity and autoimmune hypothesis” are the major subjects to focus on. It is possible that our failure in searching for a single aetiological factor will become more evident as details are elucidated; however, the disease is likely to continue to carry the “idiopathic” prefix for a long time.

REFERENCES

1. Kessler E, Wolloch Y. Granulomatous mastitis: a lesion clinically simulating carcinoma. Am J Clin Pathol 1972; 88: 642-646 [PMID: 4674439]
2. Cohen C. Granulomatous mastitis. A review of 5 cases. S Afr Med J 1977; 52: 14-16 [PMID: 560723]
3. Ozmen V, Cantürk Z, Celik V, Güler N, Kapkaç M, Koyuncu A, Müslümanoğlu M, Utkan Z. Breast Disease. Federation of Breast Diseases Society, Ankara: Güneş Medical Publishing, 2012: 55-65
4. Bakaris S, Yakuel M, Ciragil P, Guven MA, Ezberci F, Bulbuloglu E. Granulomatous mastitis including breast tuberculosis and idiopathic lobular granulomatous mastitis. Can J Surg 2006; 49: 427-430 [PMID: 17324073]
5. 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 2011; 17: 661-668 [PMID: 21951547 DOI: 10.1016/j.breast.2011.01.016.x]
6. Tulı R, O’Hara BJ, Hines J, Rosenberg AL. Idiopathic granulomatous mastitis masquerading as carcinoma of the breast: a case report and review of the literature. Int Semin Surg Oncol 2007; 42: [PMID: 17662130]
7. Bani-Hani KE, Yaghian R, Matalik IA, Shatnawi N. Idiopathic granulomatous mastitis: time to avoid unnecessary mastectomies. Breast 2004; 10: 318-322 [PMID: 15239790 DOI: 10.1016/j.breast.2003.10.021.x]
8. Azlina AF, Ariza Z, Arni T, Hisham AN. Chronic granulomatous mastitis: diagnostic and therapeutic considerations. World J Surg 2003; 27: 515-518 [PMID: 12751214 DOI: 10.1007/s00268-003-6806-1]
9. Désing D, Axt-Fliedner R, Hornung D, Weiss JM, Diedrich K, Friedrich M. Granulomatous mastitis. Arch Gynecol Obstet 2004; 269: 233-236 [PMID: 15205978 DOI: 10.1007/s00404-003-0561-2]
10. Erhan Y, Veren A, Kara E, Ozdemir N, Kapkac M, Ozdedeli E, Yilmaz K, Koyuncu A, Erhan Y, Ozbal O. A clinicopathologic study of a rare clinical entity mimicking breast carcinoma: idiopathic granulomatous mastitis. Breast 2000; 9: 52-56 [PMID: 14731585 DOI: 10.1016/j.breast.2001.03.031]
11. Tavassoli FA. Pathology of the Breast, 2nd ed. New York: McGraw-Hill, 1999: 1099-1116
12. Cserni G, Szajki K. Granulomatous Lobular Mastitis Following Drug-Induced Galactorrhea and Blunt Trauma. Breast J 1999; 5: 398-403 [PMID: 11348321 DOI: 10.1046/j.1524-5747.1999.79040.x]
13. Pereira FA, Mudgil AV, Macias ES, Karsif K. Idiopathic granulomatous lobular mastitis. Int J Dermatol 2012; 51: 142-151 [PMID: 22250621 DOI: 10.1111/j.1365-4632.2011.05168.x]
14. Gautier N, Lalonde L, Tran-Thanh D, El Khouiry M, David
J. Labelle M, Patocskai E, Trop I. Chronic granulomatous mastitis: Imaging, pathology and management. Eur J Radiol 2013; 82: e165-e175 [PMID: 23206627 DOI: 10.1016/j.ejrad.2012.12.012]

Altunbas A, Ekiz F, Coban S, Yüksel O. Alfa 1 Antitripsin Eksikliği. Yeni Tıp Dergisi 2012; 29: 138-141

Schelfoot K, Tjmal 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 DOI: 10.1016/S0301-2115(00)00546-7]

Oran ES, Gürdal SO, Yankol Y, Özмир C, Calay Z, Tunaci M, Soybir GR. Management of idiopathic granulomatous mastitis diagnosed by core biopsy: a retrospective multicenter study. Surg J 2013; 19: 411-418 [PMID: 23663101 DOI: 10.1111/bij.12123]

Gurleyik G, Aktekin A, Aker F, Karagülle H, Saglamc A. Eksikliği. Yeni Tıp Dergisi 2012; 2: 645-650 [PMID: 23062578 DOI: 10.1007/s00595-011-0496-z]

Coskun T, Kara E, Kayay Y, Guler Y, Kandiloglu AR, Goktan C. Granulomatöz Mastit: Cerrahi Tedavi-Reküterren İlişkisi. Menė Sąlgūs Dergisi 2012; 2: 26-30

Bec S, Soy M, Vandi S, Sengul N, Yilmaz F. Erythema nodosum associated with granulomatous mastitis: report of two cases. Rheumatol Int 2010; 30: 1523-1525 [PMID: 19701754 DOI: 10.1007/s00296-009-1109-y]

Salesi M, Karimifar M, Salimi F, Mahzouni P. A case of granulomatous mastitis with erythema nodosum and arthritis. Rheumatol Int 2011; 31: 1095-1095 [PMID: 20012050 DOI: 10.1007/s00296-009-1273-0]

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 DOI: 10.1007/s10166-010-0363-x]

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 DOI: 10.1186/1477-7819-11-268]

Altintoprak F, Karakece E, Kivilcim T, Dikicier E, Cakmak G, Celebi F, Ciltci IH. Idiopathic granulomatous mastitis: an autoimmune disease? ScientificWorldJournal 2013; 2013: 148727 [PMID: 24082849 DOI: 10.1155/2013/148727]

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 DOI: 10.1097/00000637-1988100000-00008]

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: 12745475 DOI: 10.1111/j.1524-4741.2003.03265.x]

Bolay A, Baute L, Storey L, Park P. Granulomatous mastitis: treatment of granulomatous mastitis. Clin J Hosp Infection 2015; 101: 125-159 [PMID: 2439861 DOI: 10.1016/j.cji.2015.05.010]

Goldberg J, Baute L, Storey LP, Park P. Granulomatous mastitis diagnosed by core biopsy: a retrospective multicenter study. Surg J 2013; 19: 108-114 [PMID: 10750465 DOI: 10.1111/1075-122X.2005.21576.x]

Han BK, Choe YH, Park JM, Moon WK, Ko YH, Yang JH, Jang SY, Nam SJ. Granulomatous mastitis: mammographic and sonographic appearances. AJR Am J Roentgenol 1999; 173: 317-320 [PMID: 10430126 DOI: 10.2214/ajr.173.2.10430126]

Jorgensen MB, Nielsen DM. Diagnosis and treatment of granulomatous mastitis. Am J Med 1992; 93: 97-101 [PMID: 16265872 DOI: 10.1016/0002-9345(92)90688-8]

Salam IM, Alhomsi MF, Daniel MF, Sim AJ. Diagnosis and treatment of granulomatous mastitis. Br J Surg 1995; 82: 214 [PMID: 7746695 DOI: 10.1002/bs.180082227]

Sato N, Lamyschlita H, Kozaki K, Watanabe Y, Ohtsuka T, Kuroki S, Nakafusa Y, Ota M, Chijiiwa K, Tanaka M. Granulomatous mastitis diagnosed and followed up by fine-needle aspiration cytology, and successfully treated by corticosteroid therapy: report of a case. Surg Today 1996; 26: 730-733 [PMID: 8883249 DOI: 10.1007/BF0312005]

Rowe PH. Granulomatous mastitis associated with a pituitary prolactinoma. Br J Clin Pract 1984; 38: 32-34 [PMID: 6383433]

Going JJ. Anderson TJ, Wilkinson S, Chetty U. Granulomatous lobular mastitis. J Clin Pathol 1987; 40: 535-540 [PMID: 3584506 DOI: 10.1136/jcp.40.5.535]

Ozel L, Unal A, Unal E, Kara M, Erdogdu E, Krand O, Gunes P, Karagul H, Demiral S, titiz MI. Granulomatous mastitis: is it an autoimmune disease? Diagnostic and therapeutic dilemmas. Surg Today 2012; 42: 729-733 [PMID: 22068681 DOI: 10.1007/s00595-011-0496-z]
Altintoprak F et al. Granulomatous mastitis

Iyengar G. Granulomatous lobular mastitis: imaging, diagnosis, and treatment. AJR Am J Roentgenol 2009; 193: 574-581 [PMID: 19620458 DOI: 10.2214/AJR.08.1528]

Omranipour R, Mohammadi SF, Samimi P. Idiopathic granulomatous lobular mastitis - report of 43 cases from Iran; introducing a preliminary clinical practice guideline. Breast Care (Basel) 2013; 8: 439-443 [PMID: 24550752 DOI: 10.1159/000357320]

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