CASE REPORT

Association between Hyperprolactinemia and Granulomatous Mastitis

Anatoly Nikolaev, PhD,* Cassann N. Blake, MD,† and Diane L. Carlson, MD†

*Charles E. Schmidt College of Medicine, Florida Atlantic University, Boca Raton, Florida; †Cleveland Clinic Florida, Weston, Florida

Abstract: Granulomatous mastitis (GM) is a relatively uncommon inflammatory breast lesion with multiple suggested etiologies. Although most GM cases show association with lactation and pregnancy, a minority of cases have been linked to hyperprolactinemia caused by either dopamine antagonist medications or with intracranial lesions, such as pituitary adenoma. The goal of this study is to review the GM cases reported in the literature with a specific emphasis on those cases associated with hyperprolactinemia and prolactinomas and to identify cases of GM seen at the Cleveland Clinic Florida which demonstrate co-occurrences of GM and intracranial lesions. CoPath and Epic data bases at Cleveland Clinic Florida were searched for cases describing inflammatory breast lesions in patients with pituitary pathology. Chart reviews were conducted and pertinent medical history was extracted for case reports. H&E-stained paraffin-embedded sections retrieved from Cleveland Clinic Florida pathology storage were evaluated by light microscopy. Four cases showing a co-occurrence of GM and hyperprolactinemia were consequently identified. A prolactin-secreting pituitary adenoma was present in two of the three GM cases. The third case demonstrated a concomitant craniopharyngioma, which was also associated with a rise in serum prolactin. This phenomenon was presumably attributable to compression, resulting in compromised transport of dopamine to the adenohypophysis and subsequent disinhibition of prolactin secretion by lactotrophs. The fourth patient with GM had a similar history of elevated prolactin. Classical histopathological features of GM were found in all four cases, including noncaseating granulomas, multinucleated giant cells, epithelioid histiocytes, and chronic inflammation. Intriguingly, complete resolution of inflammatory breast lesions along with normalization of prolactin levels occurred following the surgical excision of the craniopharyngioma, suggesting that intracranial lesion–induced hyperprolactinemia might be directly causal in GM. Therefore, the authors would suggest screening for pituitary tumors and evaluate prolactin levels in the workup of GM patients without a recent history of lactation and pregnancy and no other identified etiology.

Key Words: breast cancer, granulomatous mastitis, hyperprolactinemia, pituitary adenoma, prolactinoma

Granulomatous mastitis (GM) is a relatively rare chronic inflammatory breast condition with multiple potential etiologies. The first cases of GM were originally described by Kessler and Wolloch in 1972 (1). They reported five cases of inflammatory breast lesions with clinical features ascribed to malignancy, such as a palpable mass and nipple retraction. Multiple cases of GM have been subsequently described in the literature since this first report (1–10). Most patients are women of child-bearing age, parous, with a history of lactation. GM usually present as a one or more unilateral nodules in the breast that are often hard and tender (1–10). Cardinal features of inflammation, including erythema, swelling, and pain, are usually present (1–5,7–9). Fever has been reported in only a minority of GM cases (7,23). Additional clinical symptoms include cutaneous fistulas and draining sinuses (5–10,23). A clinically important feature of GM is that it may mimic malignancy. These lesions may present with nipple retraction, axillary lymphadenopathy, and peau d’orange, in association with a breast mass (1–10). Radiographically, these lesions are usually breast imaging-reporting and data system (BI-RADS) category 4 (2–5,9). Herein, we will present a review of the literature as it relates to GM, with a specific emphasis on the cases associated with hyperprolactinemia and prolactinomas. Additionally, we will present the Cleveland Clinic Florida (CCF) experience, including GM cases demonstrating an association between GM and prolactin-affecting intracranial lesions, including pituitary adenomas.

MATERIALS AND METHODS

Following IRB approval, CoPath and Epic data bases were searched for cases describing inflammatory breast lesions in patients with pituitary pathology. Chart reviews were conducted and pertinent medical history was extracted for case reports. H&E-stained paraffin-embedded sections retrieved from Cleveland Clinic Florida pathology storage were evaluated by light microscopy. Four cases showing a co-occurrence of GM and hyperprolactinemia were consequently identified. A prolactin-secreting pituitary adenoma was present in two of the three GM cases. The third case demonstrated a concomitant craniopharyngioma, which was also associated with a rise in serum prolactin. This phenomenon was presumably attributable to compression, resulting in compromised transport of dopamine to the adenohypophysis and subsequent disinhibition of prolactin secretion by lactotrophs. The fourth patient with GM had a similar history of elevated prolactin. Classical histopathological features of GM were found in all four cases, including noncaseating granulomas, multinucleated giant cells, epithelioid histiocytes, and chronic inflammation. Intriguingly, complete resolution of inflammatory breast lesions along with normalization of prolactin levels occurred following the surgical excision of the craniopharyngioma, suggesting that intracranial lesion–induced hyperprolactinemia might be directly causal in GM. Therefore, the authors would suggest screening for pituitary tumors and evaluate prolactin levels in the workup of GM patients without a recent history of lactation and pregnancy and no other identified etiology.
breast lesions in patients with intracranial lesions. Chart reviews were conducted. Known history of disease progression, the effects of steroid treatment, and prolactin levels were collected. Cases identified to have a co-occurrence of pituitary tumors and inflammatory breast lesions were included in the study. H&E-stained paraffin-embedded sections obtained from CCF pathology storage were evaluated by light microscopy. Results of the serum prolactin tests were obtained from patient records and correlated with the presence of granulomatous inflammatory lesions.

**RESULTS**

Following review of the literature, 260 GM cases were identified, with four cases of GM associated with pituitary prolactinoma, and five cases associated with dopamine antagonist drug-induced hyperprolactinemia (Table 1).

| Authors          | Number of cases | Median age, years | Presentation                                | Associated medical history                                      | Treatment types                          | % of relapses          |
|------------------|-----------------|-------------------|--------------------------------------------|-----------------------------------------------------------------|-----------------------------------------|------------------------|
| Neel et al., 2013| n = 23          | 39                | Unilateral extra-areolar lump of varying size (78%); abscess (26%); peau d’orange | Autoimmune or inflammatory disease (21%); Delivery (74%) and breast-feeding (35%) before presentation; Antipsychotic drug (17%, 4/23); Prolactinoma (1/23) | Antibiotics initially (18/23, 78%); Steroids (11/13, 85%); Surgical excision (39%) | 78% overall; 46% for steroid treatment; 78% for surgical excision; Prolactinoma: 1/1; Antipsychotic drug: 2/4 |
| Gautier et al., 2012 | n = 11         | 38.7              | Unilateral breast mass with erythema (73%, 8/11) | Breast-feeding (5/11) before/at presentation; Prolactinoma (1/11); Oral contraceptives (1/11) | Antibiotics initially (4/11), surgery (2/11) | 18% overall (2/11) Prolactinoma: 1/1 on the opposite breast |
| Al-Khaffaf et al., 2008 | n = 18         | 36                | Painless breast lump (4/18); inflammatory breast abscess (14/18) | Smokers (3/18, 17%); delivery in the last 5 years (56%, 10/18); parity (94%); oral contraceptives (5/18, 28%); hyperprolactinemia (1/18) | Steroids (4/18); antibiotics (13/18); surgical excision (4/18); incision and drainage (7/18) | 3/18; patient with hyperprolactinemia had longest episode of GM |
| Katz et al., 2007 | n = 4           | 36                | Nipple retraction; painful breast mass; redness of skin | Risperidone treatment and elevated prolactin levels (1/4) | Steroids (4/4); methotrexate (2/4); abscess drainage (2/4) | 1/4, case with elevated prolactin |
| Erhan et al., 2000 | n = 18         | 34.55             | Unilateral, firm, discrete breast mass | Delivery (100%); hyperprolactinemia (2/18) | Excisional biopsy (100%); re-excision with antiprolactinemic therapy for recurrent cases with high prolactin; re-excision plus steroids for recurrent case with normal prolactin | 3/18, including 2 patients with hyperprolactinemia |
| Going et al., 1987 | n = 9           | 28.4              | Not reported                                 | Phenothiazine induced hyperprolactinaemia 1/9; oral contraceptives 7/9 Delivery 100% | Antibiotics, steroids, surgical procedures | Several cases |
| Rowe, 1984       | n = 1           | 38                | Recurrent unilateral breast abscesses       | Delivery; hyperprolactinemia; pituitary prolactinoma | Bromocriptine; Incisional drainage of abscesses | Multiple recurrent abscesses before treatment; resolved after bromocriptine therapy |

Herein, we report three additional cases of GM associated with intracranial lesions, one of which is a craniopharyngioma. As far as we know, this is the first case of GM associated with craniopharyngioma to be reported in literature.

**Case 1**

The patient was a 42-year-old Asian woman with two prior pregnancies and one delivery. The patient had a long-standing history of a pituitary tumor, hyperprolactinemia, and bilateral milky nipple discharge from the age of 21. An intrasellar mass lesion was confirmed via magnetic resonance imaging (MRI). The patient did not wish to have surgery. Her prolactin levels were above 200 ng/mL (normal 4.8–23.3 ng/mL). Initially, she was treated with the antiprolactinemic agent, bromocriptine for a few years, primarily to manage her nipple discharge. She...
was switched to a newer antiprolactinemic drug cabergoline (Pfizer, New York, NY, USA) due to the side effects of nausea and vomiting secondary to the bromocriptine. The patient was taking cabergoline for over 10 years. She discontinued taking cabergoline because of a pregnancy. Concomitantly, the patient identified lumps in her left breast by palpation and noticed a milky discharge. She presented to CCF with a painful palpable mass in the upper central quadrant of her left breast. Her prolactin levels were elevated. Mammographic studies of the left breast showed multiple BI-RADS category 4 suspicious abnormalities: a 6-cm mass located at the 12 o’clock position and a 3.6-cm mass located in the upper outer quadrant of the left breast. There was also a nodule in the upper outer quadrant of the left breast measuring 6 mm (Fig. 1A). No dominant masses were detected by mammography in the right breast.

Ultrasound demonstrated multiple abnormal findings in the left breast (Fig. 1B): two solid masses, one measuring 6 cm in the largest diameter at the 12 o’clock position and another measuring 4.6 cm in the largest diameter at the 2 o’clock position, both corresponding to the masses seen by mammography. In addition, a solid hypoechoic nodule was also noted at the 2 o’clock position measuring 9 mm and corresponding to mammographic findings.

Ultrasound-guided core needle biopsy of the left breast lesions showed histological features consisting of benign breast parenchyma with nonnecrotizing granulomas (Fig. 2A) containing epithelioid histiocytes (Fig. 2B–D) and multinucleated giant cells of Langhans type, in a background of foam cell macrophages with abundant chronic inflammatory cells (Fig. 2, Table 2). In addition, periductal inflammation was present (Fig. 2E). There was no ductal epithelial atypia. Multinucleated giant cells with centrally located nuclei were present. Polarization microscopy was negative for embedded foreign material. Special stains for acid fast bacilli (AFB) and periodic acid-Schiff (PAS) for fungi were negative for microorganisms.

She was treated with antibiotics (Bactrim) with some initial improvement. She was subsequently placed back on cabergoline, which successfully reduced her prolactin levels to normal; however, the painful breast lump re-occurred half a year later, despite treatment.

Case 2

Patient was a 35-year-old black woman with three prior pregnancies (including two live births and one miscarriage). Five years prior to presentation at CCF, she had a history of elevated prolactin levels, amenorrhea, and galactorrhea. While she had been prescribed cabergoline, she was noncompliant. An MRI done 4 months prior to presentation at our institution demonstrated an intrasellar and suprasellar mass, posterior to the pituitary stalk. Prolactin levels were elevated at that time. The patient was not taking any drugs known to cause hyperprolactinemia (e.g., antipsychotics).

This patient presented to CCF with a right breast mass in the lower outer quadrant. The breast appeared enlarged and edematous. Three and a half months prior to presentation at CCF, she failed treatment with antibiotics (Augmentin and Bactrim) and underwent an incision and drainage of the right breast.
mass. At CCF, a diagnostic digital mammogram with computer-aided detection revealed a 3–4 cm mass of the right breast with adjacent architectural distortion, possibly representing an abscess. Multiple benign-appearing nodules were detected in the posterior right breast most likely representing enlarged lymph nodes. Concurrently, a right breast ultrasound showed a cluster of complex cysts 3 cm from the nipple the largest of which measured 8 mm, similarly with a differential diagnosis including abscess. A core needle biopsy showed benign breast parenchyma with nonnecrotizing granulomas, multinucleated giant cells, and lymphocytic infiltration (Fig. 3 and Table 2), consistent with the diagnosis of GM. PAS stain for fungi and AFB stain for bacilli were negative.

Four months following her presentation with the aforementioned inflammatory breast lesion, the patient underwent a craniotomy. The final pathology demon-

---

Table 2. The Histopathological Characteristics of the GM Cases

| Case no. | Noncaseating granulomas | Multinucleated giant cells | Lymphocyte infiltration | PMN infiltration | Microabscesses |
|----------|-------------------------|---------------------------|------------------------|-----------------|---------------|
| Case 1   | Present                 | Present, of Langhans type, with horseshoe pattern of nuclei | Present, with plasma cells | Diffuse infiltration of PMNs; Periductal inflammation with PMNs | Clusters of PMNs present |
| Case 2   | Present                 | Present, of Langhans type, with horseshoe pattern of nuclei | Present, with plasma cells | Diffuse infiltration of PMNs; Periductal inflammation with PMNs | Clusters of PMNs present |
| Case 3   | Present                 | Present, of Langhans type, with horseshoe pattern of nuclei | Present, with plasma cells | Diffuse infiltration of PMNs; Periductal inflammation with PMNs | Clusters of PMNs present |
| Case 4   | Present                 | Present, of Langhans type, with horseshoe pattern of nuclei | Present, with plasma cells | Diffuse infiltration of PMNs; Periductal inflammation with PMNs | Clusters of PMNs present |
strated the brain tumor resected by left craniorbital zygomatic craniotomy to be a craniopharyngioma. Four months after neurosurgical resection, the right breast mass decreased significantly in size. Upon physical examination at follow-up, no dominant mass could be identified. No solid or cystic lesions could be identified in the breast by ultrasound 4 months after the craniotomy. The patient was treated with daily low dose of corticosteroids (30 mg) initiated by the neurosurgery team postoperatively. Upon follow-up one and one half years thereafter, the patient’s breast examination was normal; the previously biopsied lesion in the right breast completely resolved.

Case 3

This patient was a 45-year-old white Hispanic woman. She had three pregnancies and one delivery. The patient had a 10-year history of hyperprolactinemia prior to presentation and a more recent history of a right breast infection. The initial symptoms included bilateral breast tenderness, a palpable mass in the right breast, and fevers, for approximately 3 months. An attempt had been made to aspirate the mass, but no fluid was aspirated. The patient was treated (at an outside institution) with IV antibiotics, including ceftriaxone and ciprofloxacin. Following antibiotic therapy, she presented to CCF with a “walnut size” palpable mass in the right breast, near the nipple in the lower outer quadrant. On physical examination at that time, the area was not tender and there was no erythema. An ultrasound of the right breast demonstrated a retroareolar 2.1 cm hypoechoic mass-like area and extensive edema throughout the breast, without a localized fluid collection. Ultrasound-guided core needle biopsy revealed pathological findings consistent with GM (Table 2). Concurrently, the patient was complaining of headache, lightheadedness, vertigo, and nausea/vomiting for 3 months. She reported episodes of diplopia and altered consciousness. Her serum prolactin was found to be significantly elevated at 52.2 ng/mL (normal range 4.8–23.3 ng/mL). An MRI of the brain and prolactin management therapy were recommended; however, the patient was non-compliant. Her prolactin levels continued to rise to 100.7 ng/mL, in conjunction with increased pain and tenderness associated with the right breast lump. Patient was lost to the follow-up.

Case 4

This patient was a 38-year-old woman presenting with a painful breast lump, 3–4 inches in diameter. She had a history of chronic bilateral clear milky breast discharge and elevated prolactin levels. On physical examination, a nontender mass was identified in the outer lower quadrant of the patient’s left breast, 1.5 inches in diameter. There were no overlying skin changes and nothing could be discharged from the nipple. Neurological evaluation failed to reveal any deficits. However, serum prolactin levels were found to be elevated at 65.0 ng/mL. Left breast biopsy
demonstrated GM (Table 2). An MRI of the brain was requested; however, the patient did not return for follow-up.

**DISCUSSION**

Herein, we describe several patients with inflammatory breast lesions demonstrating the histological features of GM, with associated hyperprolactinemia and intracranial pathology. Core biopsy or incisional biopsy is required for the accurate diagnosis of GM (1–10). Histologically, GM is characterized by inflammatory infiltrates of lymphocytes, plasma cells, and non-necrotizing granulomas. In GM, the inflammation demonstrates a lobular spread with granulomata found in the fibrous tissue surrounding the ductal and lobular acini (7,8,16,23). Microabscesses are often present, containing neutrophils (1–4,7,8,23). T-cell-dominated lymphocytic infiltrate and loss of acinar structure have been described (16,23). Some cases reported in the literature have been described to be devoid of granulomas, only demonstrating acute inflammatory infiltrates and epithelioid histiocytes (2,7). In addition to the lobular granulomatous inflammation, our cases also demonstrate a periductal inflammation with polymorphonuclears and lymphocytes, as well as diffuse infiltration with plasma cells. This latter histological feature is characteristic of the “plasma cell mastitis,” a benign inflammatory condition of breast originally described by Cheatle and Cutler in 1931 (30).

Granulomatous mastitis is considered to be a diagnosis by exclusion. Bacterial infections, such as tuberculosis, and fungal infections, such as histoplasmosis, have been reported to cause GM (26,27). Additional conditions associated with breast granulomas include systemic granulomatous diseases (sarcoidosis, Wegener disease), periductal mastitis, mammary ductal ectasia, and foreign body reaction (e.g., to breast implants) (28–30). All of these potential causes need to be ruled out before making the diagnosis of GM. While PAS stain for fungi and AFB stains for TB should be negative, the presence of certain bacteria on Gram stains has been reported, including *Staphylococcus aureus*, *Corynebacterium* spp., and *Streptococcus* spp. (2,3,7,8). We did not observe any PAS-positive or AFB-positive microorganisms in any of our cases of GM. Autoimmune markers such as anti-nuclear antibody (anti-ANA) have been found to be positive in some cases, including a case of presumed systemic lupus erythematosus with GM (11).

**Etiology of GM and Hyperprolactinemia**

While multiple etiologies have been proposed for GM, the majority of cases have been demonstrated to have a direct association with pregnancy, delivery, and breast-feeding (2–5,7,12). Intriguingly, several reports had shown a link between GM and hyperprolactinemia, either drug-induced (2–8,14) or caused by a pituitary prolactinoma (2–8,15). Furthermore, some patients with high prolactin levels were reported to have recurrent GM disease (2–8,16) and/or longer episodes of GM (16). So far, four cases of co-occurrence of GM and pituitary prolactinoma had been reported in literature (2,3,15). We report three additional cases of such an unusual association. Serum prolactin levels were abnormally high in all of these cases. Some of the GM patients with pituitary prolactinoma had been successfully treated with antiprolactinemic agents (15,16), suggesting that the high prolactin levels might be driving GM disease in patients with pituitary adenomas (15). In accord with this hypothesis, one of our GM patients with prolactinoma (Case 1) developed lumps.

**Treatment**

Surgical excision of the inflammatory breast mass and/or steroid treatments are the therapeutic approaches most commonly used to manage GM (2–8). Both are often preceded by or combined with a course of antibiotics (2–13). Steroid therapy appears to be highly efficacious in resolving inflammatory breast lesions in the majority of cases; steroids achieved curative effects in 11 of 13 (85%) cases described in the study by Neel et al. (2). Recurrences of GM have been reported when the steroids have been discontinued (46% in (2)). Surgical excision of the affected breast tissue appears to be somewhat less effective in the long-term management of GM (2,4). Neel et al. reported as many as 78% of GM patients had relapses of GM episodes after having breast surgery (2). While the reported GM recurrence rates after surgery were similar or higher compared with the ones after steroid therapy, the relapsed disease had been subsequently cured by steroids in several studies (3,10). For instance, Ocal et al. describe recurrent GM lesions in the 6 of 12 cases after the surgical resection (10). Treatment of these six cases of the relapsed GM disease with oral prednisone resulted in complete remission of inflammatory breast lesions, without any further recurrence of GM (10).
in her breast and milky discharge after discontinuation of cabergoline treatment. This observation appears to be consistent with the role of high prolactin levels in the induction of GM. Our second case, describing an association between GM and hyperprolactinemic craniopharyngioma, appears to be the first such association to be reported. Prolactin abnormalities identified were most likely due to pituitary stalk compression by the craniopharyngioma. Interestingly, GM was essentially cured in this patient after the surgical resection of the hyperprolactinemic craniopharyngioma combined with low dose of steroid treatment. This evidence further supports a notion that a high serum prolactin and an inflammatory response might be involved in the maintenance of GM pathology. Five cases of GM associated with antipsychotic drug-induced hyperprolactinemia have been reported in patient with concomitant pituitary adenomas (2,6,8,14,17–19,24). Treatment with potent D2 dopamine receptor antagonists, such as risperidone, has been linked to the development of pituitary adenomas, hyperprolactinemia, and galactorrhea (17–19,24). It has also been hypothesized that the treatment of depression with selective serotonin reuptake inhibitors may alter prolactin levels leading to GM (25). As hyperprolactinemia appears to be a common theme in many GM cases, it has therefore been proposed to be one of the causative factors for this disease (2,3,5,6,15,16). High prolactin levels may be part of a common pathway in the development of GM associated with pituitary adenomas, as well as GM associated with antipsychotic drug use. Prolactin may have several distinct roles in promoting inflammatory response in the breast (20,24). High levels of prolactin may lead to excessive production of milk secretions and possibly to accumulations of the static secretion products within the mammary lobules. These static secretions may either become infected or escape into perilobular stroma, leading to T-cell-mediated immune response and the formation of granulomas (7). Indeed, several infectious agents have been identified in GM patients, most notably Corynebacterium spp. (7). Nonetheless, no infectious agent is identified in the majority of GM cases (1–10). This may be either due to a noninfectious etiology of those GM cases, or perhaps inability to visualize and/or culture the infectious agent. In addition to causing the accumulation of static secretion products in the breast, prolactin had been shown to induce the nuclear factor kappa-light-chain-enhancer of activated B cells (NF-kB) signaling pathway in mammary epithelial cells, resulting in the production of the proinflammatory cytokines such as IL-1, IL-6, TNF-α, and importantly, INF-γ and granulocyte-macrophage colony-stimulating factor (GM-CSF), which in turn may trigger an inflammatory response and the formation of granulomas in the breast (20). Furthermore, significant response of GM to steroid therapy may also be related to the proposed involvement of prolactin in GM. It has been well established that the anti-inflammatory effects of steroids are in part due to the inhibition of pro-inflammatory NF-kB signaling (21,22). Therefore, it is conceivable that steroids are efficacious in resolving the inflammatory lesions of the breast because they block proinflammatory NF-kB signaling pathway downstream of prolactin in mammary epithelium. It had been also hypothesized that damage to the ductal or lobular epithelium, for instance due to trauma, may cause milky secretions escape into adjacent lobular stroma leading to granulomatous inflammation around the lobules (7,24). However, the exact triggers of this epithelial destruction in the absence of physical trauma remain unknown.

**CONCLUSIONS**

Granulomatous mastitis is a benign inflammatory lesion of the breast with a low incidence of occurrence. The causative factors that trigger this condition remain variable. A review of GM cases in the literature and of the cases identified at CCF further demonstrates an association between prolactin-secreting intracranial lesions and GM. Since antiprolactinemic agents and surgical resections of prolactin-secreting intracranial lesions appear to have therapeutic effects in GM patients, we propose that high prolactin levels may be a causal factor in the pathogenesis of GM. In addition, given the association of hyperprolactinemia with GM, one should consider screening for elevated prolactin levels in GM patients. Hyperprolactinemic patients with GM should be further evaluated for the presence of intracranial lesions, such as pituitary adenoma and craniopharyngioma.

**REFERENCES**

1. Kessler E, Wolloch Y. Granulomatous mastitis: a lesion clinically simulating carcinoma. *Am J Clin Pathol* 1972;58:642–6.
2. Neel A, Hello M, Cottereau A, *et al.* Long-term outcome in idiopathic granulomatous mastitis: a western multicentre study. *QJM* 2013;106:433–41.
3. Gautier N, Lalonde L, Tran-Thanh D, et al. Chronic granulomatous mastitis: imaging, pathology and management. Eur J Radiol 2013;82:165–75.

4. Yau FM, Macadam SA, Kuusk U, Nimmo M, Van Laeken N. The surgical management of granulomatous mastitis. Ann Plast Surg 2010;64:9–16.

5. Al-Khaffaf B, Knox F, Bundred NJ. Idiopathic granulomatous mastitis: a 25-year experience. J Am Coll Surg 2008;206:269–73.

6. Katz U, Molad Y, Ablin J, et al. Chronic idiopathic granulomatous mastitis. Arch Pathol Lab Med 2007;131:603–8.

7. Al-Khaffaf B, Knox F, Bundred NJ. Idiopathic granulomatous mastitis: a 25-year experience. J Am Coll Surg 2008;206:269–73.

8. Katz U, Molad Y, Ablin J, et al. Chronic idiopathic granulomatous mastitis. Arch Pathol Lab Med 2007;131:603–8.

9. Al-Khaffaf B, Knox F, Bundred NJ. Idiopathic granulomatous mastitis: a 25-year experience. J Am Coll Surg 2008;206:269–73.

10. Al-Khaffaf B, Knox F, Bundred NJ. Idiopathic granulomatous mastitis: a 25-year experience. J Am Coll Surg 2008;206:269–73.

11. Al-Khaffaf B, Knox F, Bundred NJ. Idiopathic granulomatous mastitis: a 25-year experience. J Am Coll Surg 2008;206:269–73.

12. Al-Khaffaf B, Knox F, Bundred NJ. Idiopathic granulomatous mastitis: a 25-year experience. J Am Coll Surg 2008;206:269–73.