Serial MR Findings and Comprehensive Review of Bilateral Lupus Mastitis with an Additional Case Report

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ABSTRACT

Lupus Mastitis (LM) is a rare presentation of lupus panniculitis involving the breast. Because it often presents as a tender palpable mass, a workup for malignancy usually ensues. It is well documented that surgery may worsen the condition; therefore, it is important to consider LM in the differential of a palpable breast mass in patients with systemic lupus erythematosus (SLE). Currently, management of LM remains primarily medical. We discuss the multi-disciplinary work-up of LM, and further describe its appearance on serial Magnetic Resonance (MR) exams.

CASE REPORT

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Our patient is a 40 year-old African-American female with a 20 year history of discoid-variant SLE (DLE). At the time her symptoms were being managed by steroid-sparing therapy (hydroxychloroquine 200 mg twice daily); however, when she began to experience alopecia as a side effect, she became noncompliant with her medication.

She was first referred to the mammography section of our radiology department with a painful palpable lesion in her left breast. Both her comparison and current mammogram were reviewed. The comparison mammogram of her left breast from 3.25 years earlier demonstrated coarse central calcifications and typical changes of a prior surgical excisional biopsy performed at another institution in her remote past. No calcifications or masses were visualized in her right breast (Figure 1).

The initial bilateral mammograms (figure 2) at our institution and subsequent ultrasound (figure 3) of the left breast demonstrated no significant change in the appearance of the coarse, dystrophic calcifications in her left breast. However, the right breast demonstrated development of suspicious fine linear-branching calcifications in a ductal distribution at the 10 o'clock location (figures 2 and 3 [compression magnification]).

Because of their suspicious appearance, biopsy versus short term follow up was discussed with the patient who elected to proceed with a core-needle biopsy despite her known history DLE. Prior to the biopsy, however, the patient underwent a Breast MRI (results discussed later). The final pathology (figures 5 and 6) returned as "hyalinization … lymphocytic inflammation and necrosis … [most consistent with] lupus mastitis."

The patient was restarted on hydroxychloroquine, and she experienced mild improvement of her breast symptoms. When her alopecia returned with associated cutaneous lesions, she again became noncompliant with her medication. 10 months later, the patient returned with a new painful palpable mass in her left breast. Given her history of DLE, and prior negative bilateral breast biopsies, it was decided to forgo the biopsy in favor of a second MRI to compare to the initial MRI from 10 months earlier (both results discussed later).

The patient's hydroxychloroquine was restarted and a topical steroid (clobetasol 0.05%) cream was added. She tolerated the therapy well and her painful, palpable breast masses slowly improved, this time without an excisional biopsy which might have exacerbated her condition. Over a 26 month period, she received 4 serial MR exams at approximately a 6 month interval because her second MRI was assigned a BI-RADS 3 (figures 8, 10, 11, 12). The latter 3 MRI exams demonstrated interval decrease in the thickness of the peripheral inflammatory enhancement which mirrored the patients clinical improvement.

Our Experience with Lupus Mastitis and Breast MRI

For Breast MRI, we use the following protocol. IV Contrast enhanced MRI of both breasts is performed on a Siemens TrioTrim 3.0 Tesla Magnet using a 7 channel; InVivo Breast Array MR coil with the following pulse sequences: 3 Plane Localizer, T2 TIRM (STIR) Axial BLADE (FOV 340; Time to Repetition [TR] 11070; Time to Echo [TE] 137; 3 mm @ .8 mm Time = 4:49), Axial T1 FL3D (FOV 340; TR 6.7; TE 2.63; 1.5mm @ 20% Time = 1:02), Axial T1 FL3D (Q-fat saturation technique; FOV 340; TR 4.0; TE 1.4; 1.00 mm @ 20% Time = 6:31 - 1 run without and the remaining 5 runs after contrast administration), and Sagittal T1 (FOV 240; TR 4.35; TE 1.75; 2.0 mm @ 20% Time = 2:04). Images are reviewed on a dedicated DynaCad workstation (Invivo Corp).

Prior to the biopsy of suspicious calcifications in her right breast, the initial MRI in August 2009 (figure 7) demonstrated hypointense, coarse calcifications in the left breast on T1W. No other significant findings were visualized on T1WI, secondary to the dense parenchymal tissue not uncommon in younger women (the age range which SLE is most prevalent). Axial STIR (BLADE) demonstrated scattered fibrocystic changes adjacent to the subtle peripheral high intensity signal surrounding the LM. The delayed post-contrast GRE sequence demonstrated a continuous, irregular, thick rim of enhancement. Because the enhancement was sub-threshold (initial peak enhancement of < 60%), no dynamic enhancement pattern was assigned.

From her second MRI to her fourth (a 16 month interval in which the patient was compliant with her medications), the peripheral enhancement on contrast-enhanced MR sequences thinned and became discontinuous. Initially, the thickest, uninterrupted area of enhancement (figure 7a arrow) was 13 mm in thickness. On her second MRI, this thickness was similar at 12 mm. However, as the patient complied with her prescribed medications, this thickness decreased to 5 mm over the following 16 months. This corresponded clinically with improvement of her painful breast symptoms and resolution of her palpable left breast mass. Additionally the final mammogram (figure 9) demonstrated only benign-appearing course dystrophic calcifications, replacing the previously suspicious fine linear-branching calcifications.

DISCUSSION

Panniculitis is a nonspecific inflammatory condition involving the subcutaneous fat. It is referred to as lupus erythematosus profundus when it is occurs in patients who have systemic or discoid lupus. Breast involvement is termed lupus mastitis, and the first documented case was reported in 1971 by Tuffanelli [1-2].

Lupus panniculitis is also known as Irgang-Kaposi syndrome after Kaposi who first described the findings of "lupus panniculitis" in 1883, and Irgang who introduced the term "lupus erythematous profundus" in 1940 [3-5]. Skin involvement occurs in up to 70% of lupus patients with a predilection for the arms, shoulder, face and buttocks [6]. It is estimated that only 2-3% of patients with SLE develop lupus paniculitis - and of those, only a few develop lupus mastitis. [1,3,7,8]. Only 36 cases of LM were identified on a September 2012 PubMed search.

To our knowledge, there are no cases in the literature describing the multi-stage findings of LM on serial MR examinations; with specific regard to the response of LM during treatment. Our literature search revealed only one case describing LM findings from a single MR exam [3]. Since this singular report, there have been advancements in post-processing technology and improved surface coils for use in Breast MRI. These advancements have chiefly led to increased use of breast MRI in the workup of suspected malignancy, including inflammatory conditions such as LM which can mimic a malignancy.

Lupus paniculitis is most often seen in patients with a known diagnosis of Lupus, usually the discoid variant (70%), and there are very few reports of LM as the initial presentation of the disease [1,3,8]. While cases of lupus panniculitis have been reported in males, 90% occur in women of childbearing age [1,3,5,9]. Even though the exact etiology remains unclear, there seems to be an auto-immune component given that LM tends to improve with immunosuppressants [1].

LM often presents both clinically and mammographically as a mass. Suspicious calcifications are also frequently visualized on mammography [10]. These findings usually prompt a workup for malignancy. Acutely, LM is usually painful, and often associated with a mass, or occasionally masses which can be bilateral. Conversely, deep lesions, secondary to the lymphocytic vasculitis, may not be palpable. Palpable superficial lesions tend to involve the skin, especially with the discoid variant. On clinical examination, these superficial lesions may appear normal, atrophic, violaceous, erythematous, or discoid. As the lesions progress, some may ulcerate, and others resolve without visible sequelae. Because the course of the disease is chronic, LM may recur at the same place, or present de novo in either breast. Additionally multiple lesions at different points of acuity may be present simultaneously [1-3,11,12]. In rare cases, LM may mimic a Paget's Carcinoma when atrophy, ulceration, and erythematous changes are severe enough [13].

In addition to primary breast malignancy, there are a few other differential considerations which may mimic LM. Diabetic mastopathy may present with similar painful masses but can usually be differentiated by clinical history and labs [1]. TB and other granulomatous causes of mastitis do not present with a lymphocytic vasculitis. In addition, both diabetic mastopathy and TB/granulomatous mastitis tend to present as non-masslike enhancement on MRI as opposed to an area of well-defined peripheral enhancement seen in LM. Lymphoma, most often Non-Hodgkin's lymphoma, can be excluded with immunohistological chemical staining (IHC) [1,4,6]. Finally in contrast to LM, lymphoma tends to be bilateral without calcifications on mammogram.

Various inciting or exacerbating factors have been reported and include trauma, surgery, iodinated-contrast agents, ultra-violet light, and occasionally biopsy [3,4,8]. However, when clinical history is not obvious or available, a tissue sample may be required for diagnosis to exclude malignancy. Although surgery has been known to exacerbate LM, core needle biopsy is usually adequate for diagnosis and is also significantly less likely (than surgery) to cause a flare-up [14].

Hyaline fat necrosis is key to the diagnosis, and characterized by anucleated adipocytes on a background of glassy collagenous stroma. The other predominate feature is a lymphocytic lobular panniculitis with small mature lymphocytes interspersed among plasma cells which typically involve the fat lobule. This lymphocytic process is also known to cause a vasculitis of small- and medium-sized vessels [1,3]. Identification of germinal centers and IHC, specifically linear deposition of IgM and C3 at the dermo-epidermal junction, help to distinguish LM from a low-grade lymphoma [1,6,8].

Major and minor pathology criteria have been defined and when present, are highly specific for the diagnosis. However, not all criteria need be present to make the diagnosis. Major criteria include hyaline fat necrosis, lymphocytic infiltration with lymphoid nodules surrounding the necrosis, and panniculitis. Minor criteria include discoid lesions in overlying skin, lymphocytic vasculitis, mucin deposition, and hyalinization of subepidermal papillary lesions [6,8].

Although the breast masses caused by LM are often seen on mammography, the confounding feature tends to be the calcifications. Paralleling the course of fat necrosis, calcifications progress from suspicious (fine linear branching or amorphous/indistinct) to benign (dystrophic and coarse) in appearance [11]. Early calcifications frequently simulate malignancy and present in a ductal distribution, or appear fine linear-branching in nature. Prominent central calcifications may also simulate the appearance of contrast retained from a ductogram [5]. As calcifications increase in size and become increasingly coarse, they are more easily visualized on ultrasound and MRI [8,11,14].

On ultrasound, LM often appears as a heterogeneous and hyperechoic mass with irregular and ill-defined margins. Dermal thickening, prominent glandular tissue, and hypervascularity are also frequently visualized. Coarse shadowing calcifications become visible with disease progression [5,8].

MRI is very useful in showing the extent of LM and the presence of skin involvement. However, in general the MR features of LM are non-specific. On MR, LM tends to mimic the appearance of fat necrosis. However, as with most inflammatory breast processes, MR cannot differentiate LM from malignancy. Although not confirmed on our MR exams, Sabate et al. reported that LM paralleled the signal intensity of fat as hyperintense on both T1 and T2 non-contrast sequences. Post-contrast images demonstrated an irregular-shaped mass with rim enhancement and a type 3 (washout) kinetic curve [3].

In our case, neither T1 or T2/STIR sequences demonstrated specific information due to the dense parenchyma and fibrocystic change, respectively. However, dynamic contrast-enhanced and delayed post-contrast sequences showed a 5.6 cm area of peripheral enhancement. The enhancement was thick and continuous when first discovered, and this corresponded to the interval development of a palpable mass in the left breast. As the patient's medications were optimized, the enhancement thinned, became discontinuous, and the clinically palpable mass resolved. Therefore in our experience, the contrast enhanced sequences proved most diagnostically useful. Our findings suggest that post-contrast MR sequences of lupus mastitis can be used to follow and help optimize treatment. As a patients' medical therapy is optimized (assuming patient compliance), the thick, continuous, inflammatory peripheral enhancement (of LM on MRI) thins, becomes discontinues and resolves in certain areas. This also mirrored clinical and subjective resoluction of the palpable mass.

Oral therapy beginning with hydroxychloroquine +/- oral steroids is the mainstay of treatment for LM. Treatment may also require the addition of topical corticosteroids in cases unresponsive to oral therapy. Because many masses persist and may not fully resolve with the addition of steroids, steroid-sparing immunosuppression drugs may be attempted to avoid long-term steroid use. Cyclophosphamide has proved less efficacious, but may be attempted in those who don't achieve remission within the first 3-6 months. Diffuse, uncontrolled cases may rarely require mastectomy [1,2,8].

In summary, lupus mastitis may present in patients with SLE as a painful palpable mass. Serial mammograms may show initial suspicious fine linear branching calcifications, in one or both breasts, which progress to benign coarse calcifications over time. Ultrasound may demonstrate a mass and/or calcifications. MRI findings include an irregular rim enhancing mass. Serial MR may be useful in evaluating response to therapy as there appears to be a correlation between the thinning of peripheral enhancement and clinical resolution of symptoms. Accurate patient history and knowledge of the typical imaging appearance of LM may help prevent or minimize surgical intervention, which carries the potential to exacerbate the condition.

TEACHING POINT

Lupus mastitis is important to include as a differential consideration in Lupus patients with a breast mass, because treatment and prognosis are markedly different than with malignancy. In cases where clinical history is ambiguous and tissue diagnosis is necessary, core needle biopsy is preferred over surgical excision because it is less likely to exacerbate lupus mastitis. In the setting of suspected lupus mastitis, serial MRI exams may be useful in evaluating response to therapy as there appears to be a correlation between the decreasing thickness of peripheral enhancement and clinical resolution of symptoms.

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FIGURES

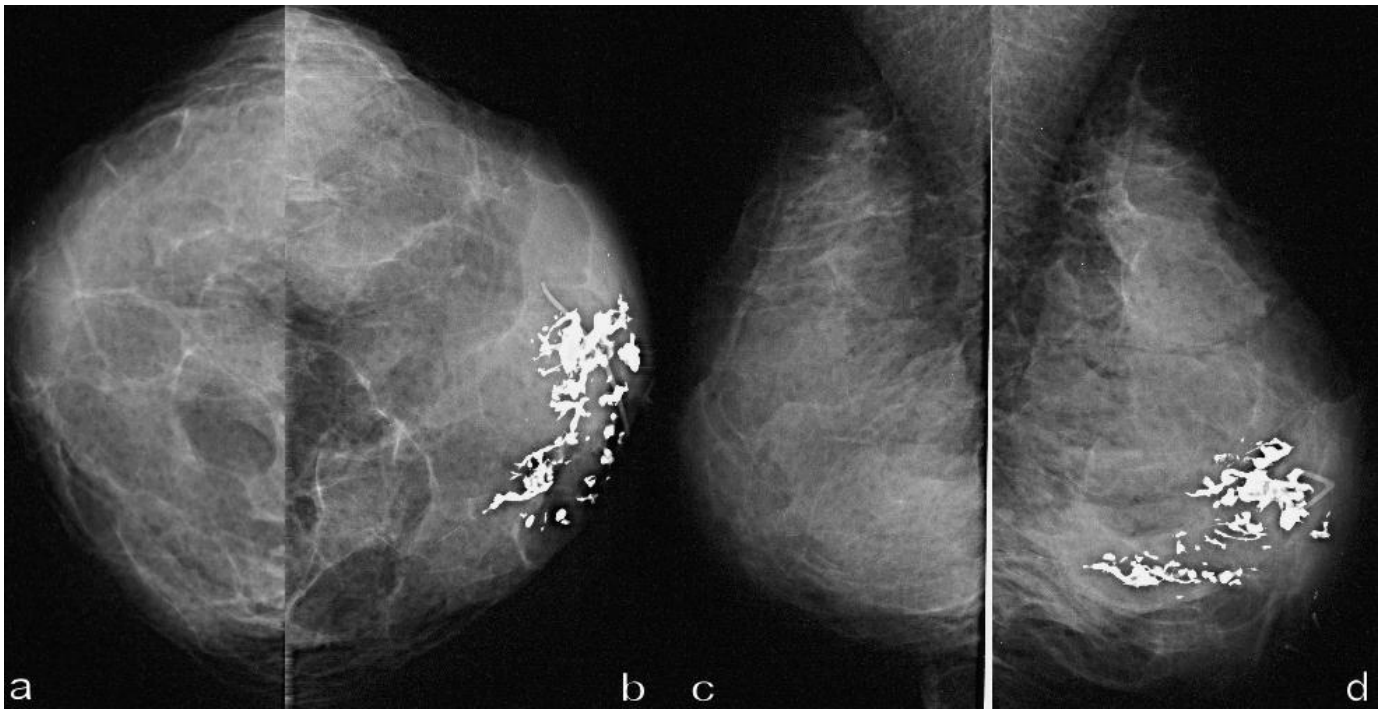

Figure 1: This is a 40 year-old, black female with the discoid variant of SLE who would later develop the rare manifestation of lupus mastitis. CC (a - right; b - left) and MLO (c - right; d - left) views 3.25 years before her initial presentation to our mammography section demonstrate symmetric, extremely dense fibroglandular tissue with coarse dystrophic calcifications in the left breast, but no calcifications in the right breast. A scar maker indicates the site of a prior left excisional biopsy.

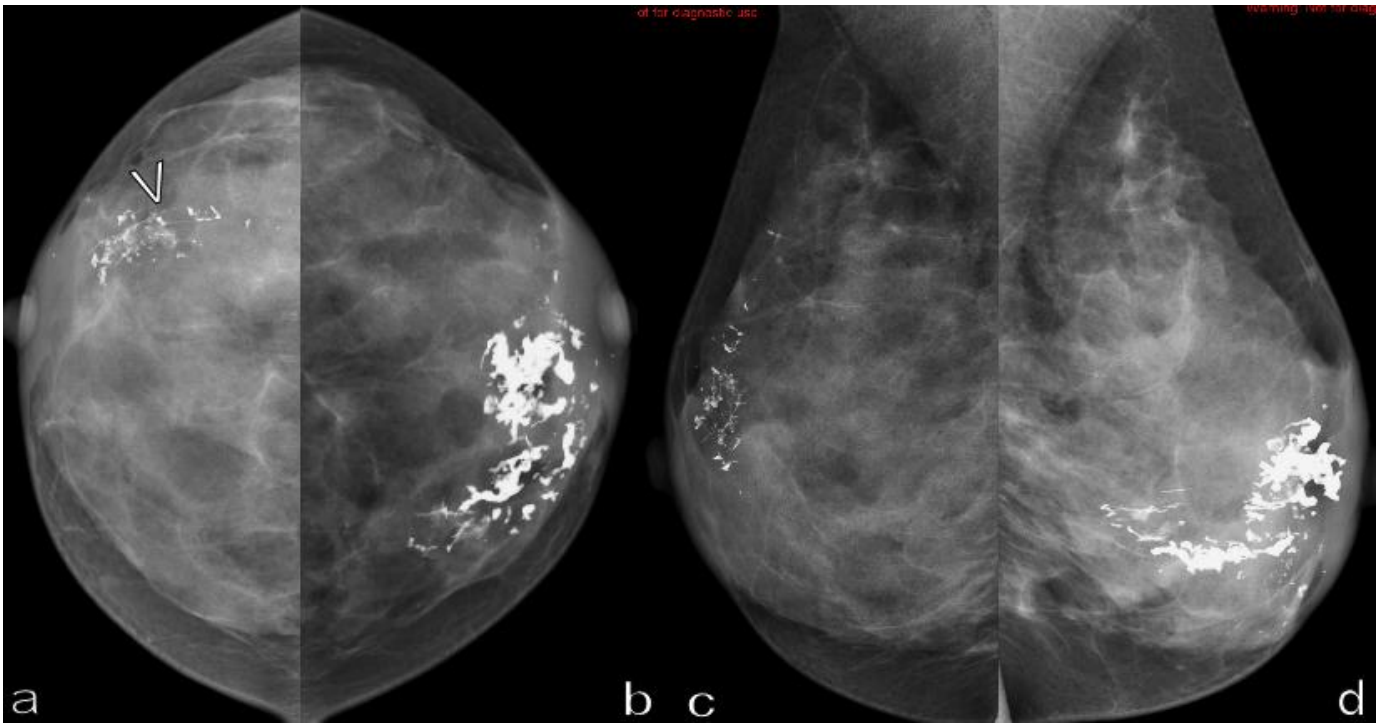

Figure 2: This is a [now] 43 year-old, black female with the discoid variant of SLE who would soon be diagnosed with the rare manifestation of lupus mastitis. CC (a - right; b - left) and MLO (c - right; d - left) views from her initial presentation demonstrate interval devlopment of suspicious fine linear, branching calcifications in the right breast (arrowhead). There is progression of the coarse left sided calcifications.

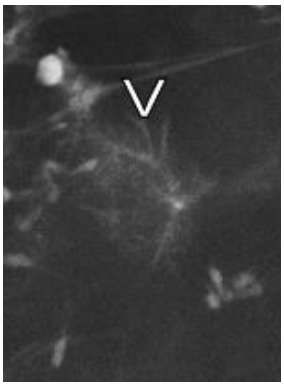

Figure 3: This is a 43 year-old, black female with the discoid variant of SLE who would soon be diagnosed with the rare manifestation of lupus mastitis. The magnified, spot compression magnification ML view (a) of the right breast from a diagnostic mammogram upon her initial presentation demonstrates interval development of worrisome, .

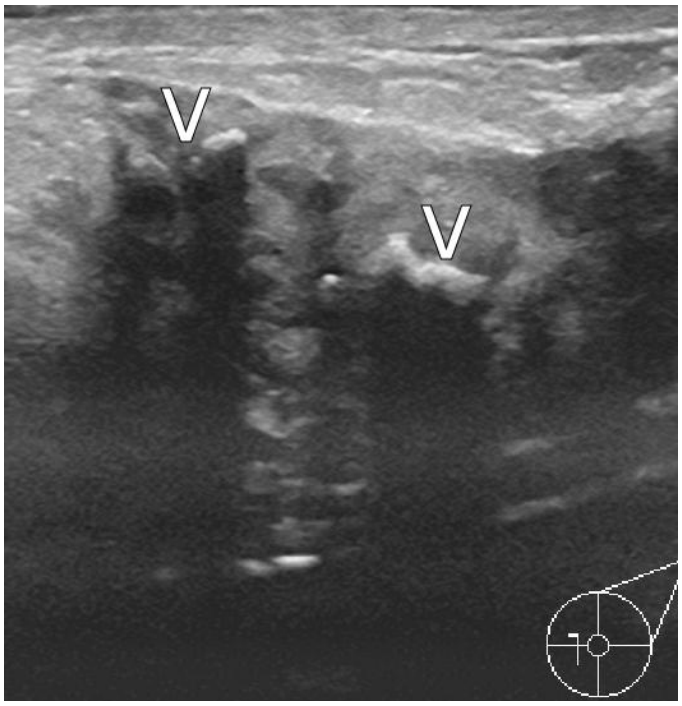

Figure 4: This is a 43 year-old, black female with the discoid variant of SLE who would soon be diagnosed with the rare manifestation of lupus mastitis. Ultrasound of the left breast with a 9 mHz linear transducer in the area of palpable concern upon her initial presentation demonstrates benign coarse, dystrophic calcifications (arrowhead) and no focal mass.

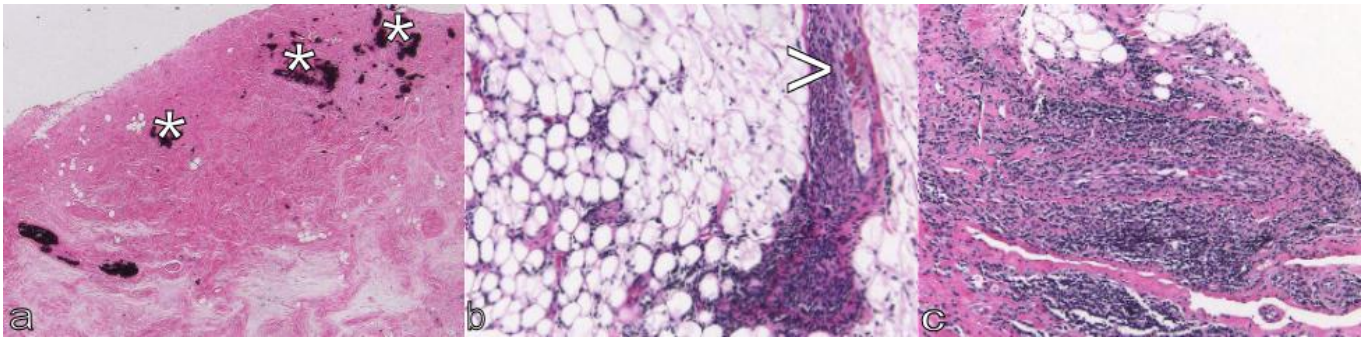

Figure 5: This is a 43 year-old, black female with the discoid variant of SLE who would be diagnosed with the rare manifestation of lupus mastitis upon interpretation of these biopsy specimens each stained with hematoxylin and eosin. a. A 2x magnification slide demonstrates calcifications (star) surrounded by hyalinized fat necrosis. b. A 10x magnification slide shows lymphoplasmacytic inflammation infiltrating between the fat and surrounding a blood vessel in a perivascular distribution (arrowhead). c. A 10x magnification shows dense perivascular lymphoplasmacytic inflammation similar to the arrowhead in b.

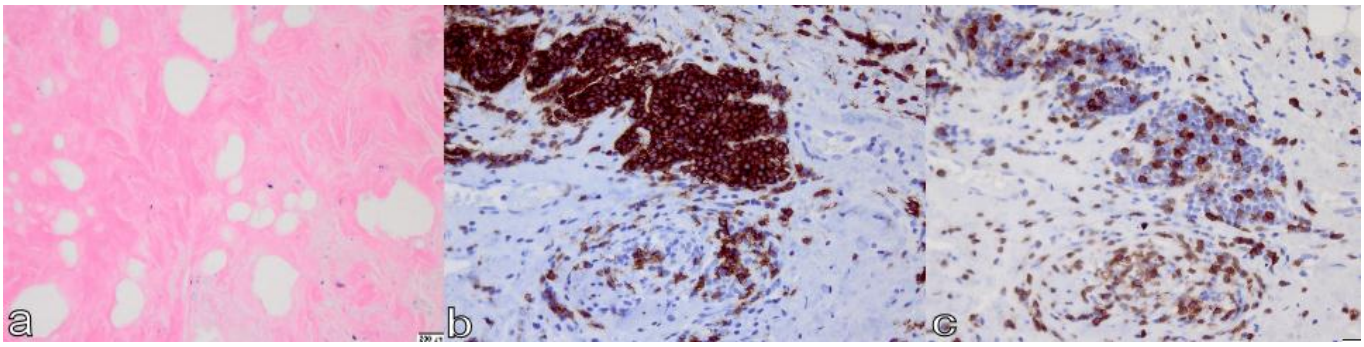

Figure 6: This is a 43 year-old, black female with the discoid variant of SLE who would be diagnosed with the rare manifestation of lupus mastitis upon interpretation of these biopsy specimens. A. 40x magnification image stained with hematoxylin and eosin demonstrates hyalinized fat necrosis. The adipocytes have no nuclei and the intervening stroma is dense and sclerotic. The middle image stained for CD20 (b) and right image stained for CD3 (c): IHC images both at 40x magnification show polyclonality (which excludes lymphoma), with a majority of lymphocytes staining CD20 positive (middle), and scattered CD3 positive lymphocytes (c). Cells positive for CD20 stain brown (b), and those positive for CD3 stain brownish red (c).

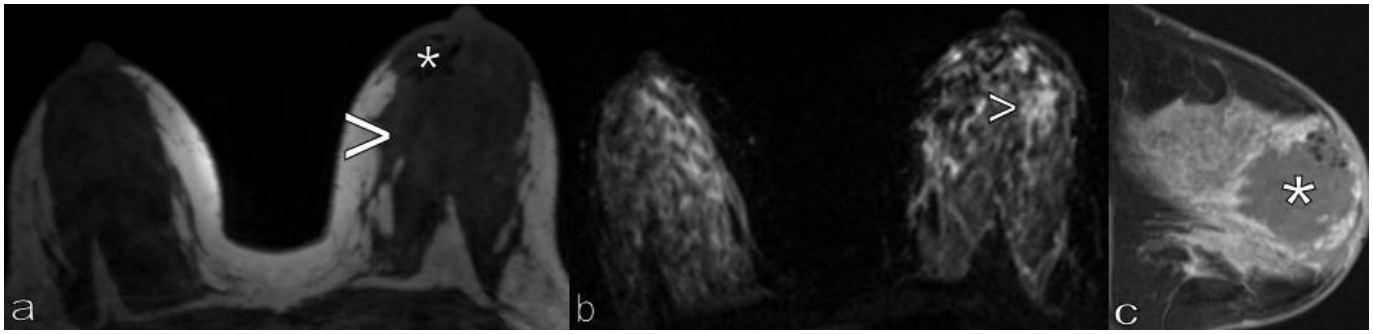

Figure 7: This is a 43 year-old, black female with the discoid variant of SLE who would soon be diagnosed with the rare manifestation of lupus mastitis following interpretation of the biopsy specimens in the pathology images above. Initial MRI: The axial T1 pre-contrast non-fat-saturated (a; TR 6.7; TE 2.63; ST 1.5 mm) sequence demonstrates coarse calcifications (star) as signal voids which are visible on the previously shown mammograms; however the dense bilateral parenchymal tissue (arrowhead) hides the LM. The axial BLADE (STIR, b; TR 11070, TE 137, ST 3 mm, no contrast) sequence demonstrates scattered fibrocystic changes adjacent to the subtle, peripheral, high intensity signal surrounding the LM in the left breast (arrowhead). Sagittal delayed post-contrast GRE (c; TR 4.35; TE 1.75; 2.0 mm, 1 cc/10kg IV Gadolinium contrast up to 20 cc) sequences of the left breast demonstrate a 5.6 cm, heterogeneous mass with a peripheral, irregular, thick, rim of enhancement (star).

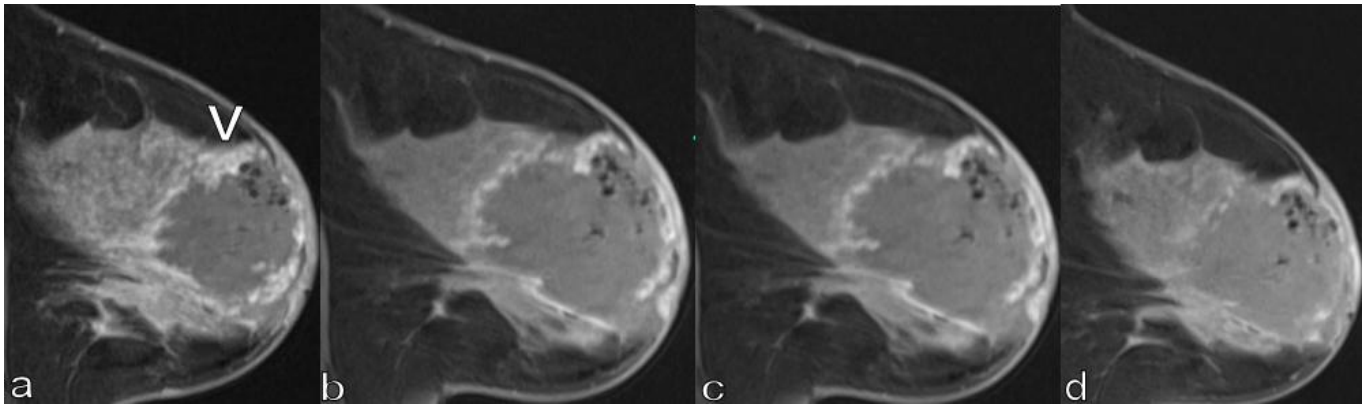

Figure 8: This is a 43 year-old, black female with the discoid variant of SLE who developed the rare manifestation of lupus mastitis. Sagittal delayed post-contrast GRE sequences of the left breast [TR 4.35; TE 1.75; 2.0 mm, 1 cc/10kg IV Gadolinium contrast up to 20 cc] in sequential order: (a) Initial MRI, (b) 10 months after the initial MRI, (c) 16 months after the initial MRI, and (d) 26 months after the initial MRI demonstrate interval decrease in the thickness of irregular peripheral enhancement. The superior-most margin of the uninterrupted peripheral enhancement which appears triangular in shape (arrowhead) decreases in thickness from 13 mm, to 12 mm, to 8 mm, and finally to 5 mm; respectively.

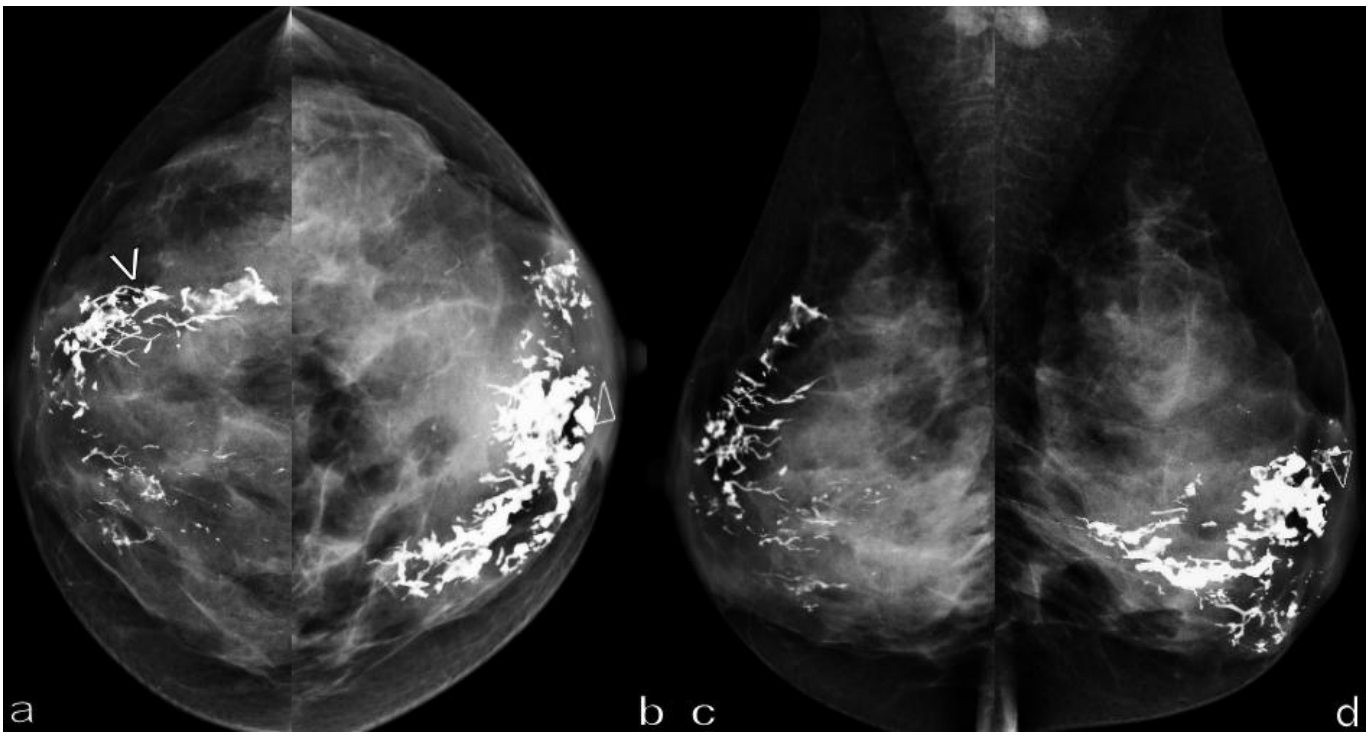

Figure 9: This is a 43 year-old, black female with the discoid variant of SLE who developed the rare manifestation of lupus mastitis. CC (a -right; b - left) and MLO (c - right; d - left) views 26 months after initial presentation demonstrate further development of benign calcifications in both breasts, with initially suspicious right sided calcifications maturing into typically benign coarse calcifications (arrowhead).

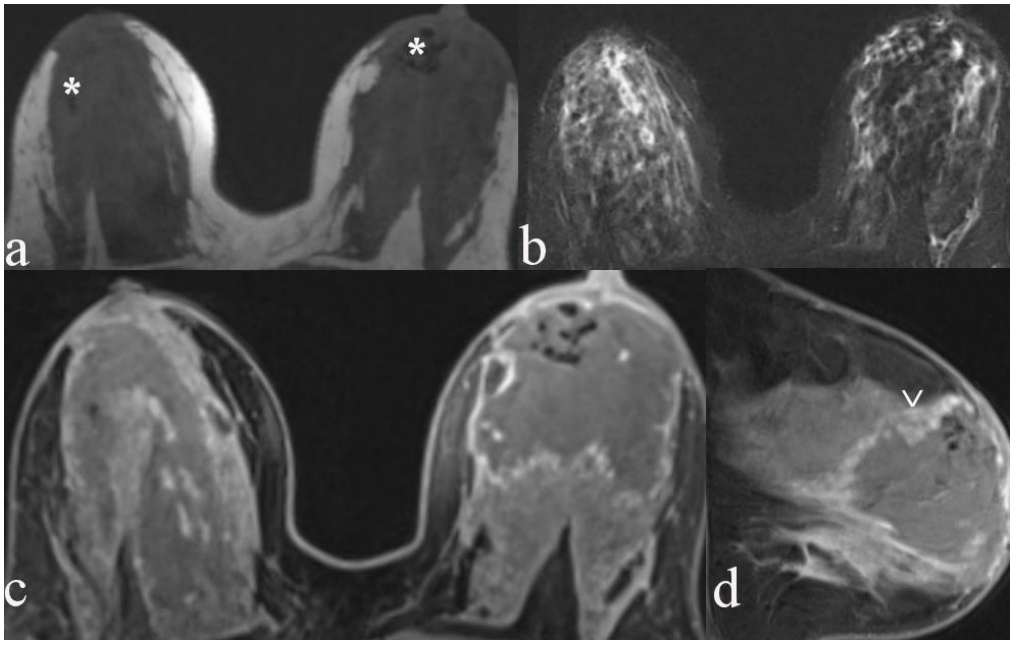

Figure 10: This is a 43 year-old, black female with the discoid variant of SLE who developed the rare manifestation of lupus mastitis. These are MR axial T1 pre-contrast non-fat-saturated (a; TR 6.7; TE 2.63; ST 1.5 mm, no contrast), axial BLADE (b; TR 11070, TE 137, ST 3 mm, no contrast), axial delayed post-contrast GRE (c; TR 4.0; TE 1.4; 1.00 mm, 1 cc/10kg IV Gadolinium contrast up to 20 cc), sagittal delayed post-contrast GRE (d; TR 4.35; TE 1.75; 2.0 mm, 1 cc/10kg IV Gadolinium contrast up to 20 cc) images 10 months after the initial MRI. Calcifications are now visualized in both breasts as flow voids (asterisks) on axial T1 pre-contrast non-fat-saturated image, fibrocystic changes persist on the BLADE image obscuring visualization of the mass, and the thickness of the irregular enhancement (arrowhead) surrounding the heterogeneous mass on axial (c) and sagittal (d) delayed- post-contrast sequences has decreased in maximum thickness to 12 mm (measured at the point of original maximum thickness)

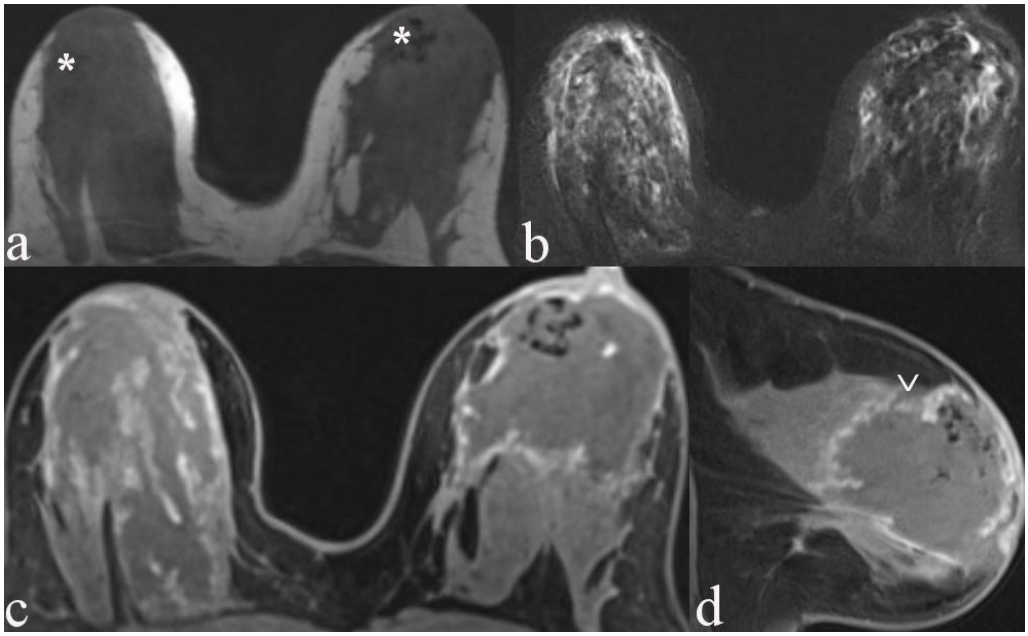

Figure 11: This is a 43 year-old, black female with the discoid variant of SLE who developed the rare manifestation of lupus mastitis. These are MR axial T1 pre-contrast non-fat-saturated (a; TR 6.7; TE 2.63; ST 1.5 mm, no contrast), axial BLADE (b; TR 11070, TE 137, ST 3 mm, no contrast), axial delayed post-contrast GRE (c; TR 4.0; TE 1.4; 1.00 mm, 1 cc/10kg IV Gadolinium contrast up to 20 cc), sagittal delayed post-contrast GRE (d; TR 4.35; TE 1.75; 2.0 mm, 1 cc/10kg IV Gadolinium contrast up to 20 cc) images 16 months after the initial MRI. Calcifications are now visualized in both breasts as flow voids (asterisks) on axial T1 pre-contrast non-fat-saturated image, fibrocystic changes persist on the BLADE image obscuring visualization of the mass, and the thickness of the irregular enhancement (arrowhead) surrounding the heterogeneous mass on axial (c) and sagittal (d) delayed- post-contrast sequences has decreased in maximum thickness to 12 mm (measured at the point of original maximum thickness)

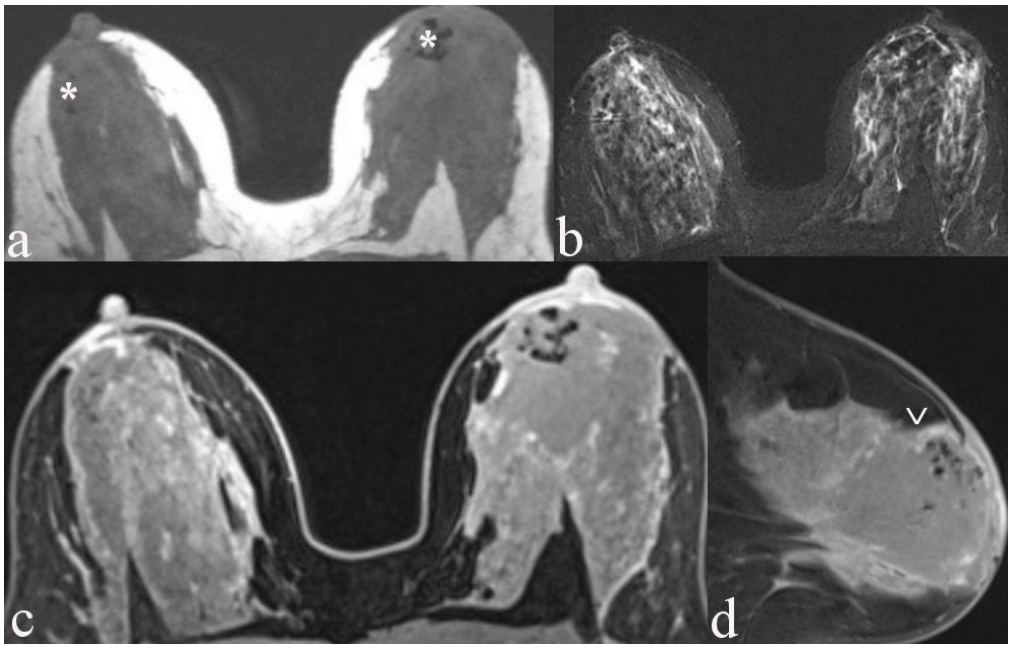

Figure 12: This is a 43 year-old, black female with the discoid variant of SLE who developed the rare manifestation of lupus mastitis. These are MR axial T1 pre-contrast non-fat-saturated (a; TR 6.7; TE 2.63; ST 1.5 mm, no contrast), axial BLADE (b; TR 11070, TE 137, ST 3 mm, no contrast), axial delayed post-contrast GRE (c; TR 4.0; TE 1.4; 1.00 mm, 1 cc/10kg IV Gadolinium contrast up to 20 cc), sagittal delayed post-contrast GRE (d; TR 4.35; TE 1.75; 2.0 mm, 1 cc/10kg IV Gadolinium contrast up to 20 cc) images 26 months after the initial MRI. Calcifications are now visualized in both breasts as flow voids (asterisks) on axial T1 pre-contrast non-fat-saturated image, fibrocystic changes persist on the BLADE image obscuring visualization of the mass, and the thickness of the irregular enhancement (arrowhead) surrounding the heterogeneous mass on axial (c) and sagittal (d) delayed- post-contrast sequences has decreased in maximum thickness to 12 mm (measured at the point of original maximum thickness).

Differential	Mammographic findings	Ultrasound findings	MRI findings
Lupus Mastitis	Mass with suspicious curvilinear microcalcifications, often in a ductal distribution. These calcifications become diffusely coarse and dystrophic over months to years.	Heterogeneous and hyperechoic mass with irregular and ill-defined margins. Dermal thickening, prominent glandular tissue, and hypervascularity occasionally visualized. Coarse shadowing calcifications become visible with disease progression	On MR, LM tends mimic the appearance of fat necrosis. Acutely there is a continuous, relatively thicker rim of enhancement. Paralleling subjective clinical improvement, this rim enhancement became thinner and discontinuous with treatment. Sabete et al. reported that LM paralleled the signal intensity of fat as bright on both T1 and T2 non-contrast sequences. In their report, post-contrast images demonstrated an irregular-shaped mass with rim enhancement and a type 3 (washout) kinetic curve
Primary Breast Cancer	Mass, calcifications, or both	Hypoechoic mass with dense posterior shadowing.	Enhancing mass with a type 3 (washout) kinetic curves.
Diabetic Mastopathy	Diffuse, bilateral, thick glandular tissue and/or masses. Given that this is caused by a systemic disease, DM often shows vascular calcifications.	Irregular mass, hypoechoic to fat with dense posterior shadowing	Persistent, slow, heterogeneous, non-mass like enhancement in both breasts.
Lymphoma (Usually Non-Hodgkin type; NHL)	The diffuse form of NHL demonstrates diffuse bilateral trabecular thickening, skin thickening, and often bilateral axillary adenopathy. More focal forms may also demonstrate ill-defined masses without calcifications or spiculations	Ill-defined, heterogeneous masses +/- mixed posterior features	Areas of lymphomatous involvement demonstrate a rapid enhancement which is usually hypo to isointense on T1WI. The focal form of NHL is usually well-defined.
Tuberculous and other forms of granulomatous mastitis	Poorly-defined, focal asymmetry often located centrally behind the nipple +/- trabecular thickening (edema)	Large, irregular hypoechoic masses with surrounding hypervascularity and edema	Slowly enhancing, non-mass like regional enhancement hypo- to isointense on T1WI, with +/. Small areas of T2WI/STIR hyperintensity from microabscesses

Table 1: Differential Table [1,4-6,8,11]: The differential for lupus mastitis includes primary breast cancer, lymphoma, diabetic mastopathy, and TB/granulomatous mastitis.

Etiology	The exact etiology remains unclear, although there seems to be an auto-immune component given that LM tends to improve with immunosuppressants.
Incidence	Develops in 2-3% of patients with Systemic lupus erythematosus, usually the discoid variant (70%)
Gender Ratio	At least 9:1 female
Age	Predominately in women of childbearing age; 20-40 years old
Risk Factors	Trauma, surgery, iodinated-contrast agents, ultra-violet light, and occasionally biopsy
Treatment	Hydroxychloroquine +/- oral and topical steroids is generally first-line therapy. Steroid-sparing immunosuppression drugs may be attempted to avoid long-term steroid use. Cyclophosphamide has proved less efficacious, but may be attempted in those who don't achieve remission within the first 3-6 months. Diffuse, uncontrolled cases may rarely require mastectomy
Prognosis	Although lesions usually respond clinically to immunosupressants, the mammographic course of the disease is chronic. Most lesions eventually develop calcifications, often mistaken for malignant calcifications at first, which then become course and dystrophic over time. On MR, the lesions typically present as a mass with irregular and often thick rim enhancement acutely. This rim enhancement may become thinner and possibly even resolve with chronicity and/or treatment.
Imaging Findings	Mammographically, LM usually presents as a mass with suspicious curvilinear microcalcifications, often in a ductal distribution. These calcifications become diffusely coarse and dystrophic over months to years On ultrasound, LM often appears as a heterogeneous and hyperechoic mass with irregular and ill-defined margins. Dermal thickening, prominent glandular tissue, and hypervascularity are not infrequently visualized. Coarse shadowing calcifications become visible with disease progression On MR, LM tends mimic the appearance of fat necrosis. Acutely there is a continuous, relatively thicker rim of enhancement. Paralleling subjective clinical improvement, this rim enhancement became thinner and discontinuous with treatment.

Table 2: Summary Table [1-5,9,11]: This table summarizes the key aspects of and imaging findings associated with lupus mastitis.

ABBREVIATIONS

CNB - Core needle biopsy
IHC - Immunohistochemistry
LM: Lupus Mastitis
MRI - Magnetic resonance imaging
T1WI - T1 weighted imaging
T2WI - T2 weighted imaging
TB - Tuberculosis

KEYWORDS

lupus mastitis; mastitis; lupus; SLE

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The views expressed are those of the author(s) and do not reflect the official policy of the Department of the Army, the Department of Defense or the U.S. Government.