Lymphocytic Mastopathy - Entity with Diagnostic Dilemma: A Case Report

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Abstract: Lymphocytic mastopathy is a condition that affects the breasts of people who have had diabetes for a long time. It frequently manifests as huge hard lumps, raising cancer risk and necessitating unnecessary surgery. Histopathologically, it is characterized by sclerosing lymphocytic mastitis. A nulliparous woman appeared with a firm, palpable breast lump. Malignancy was the clinical impression. They conducted imaging tests, which confirmed a malignancy suspicion. Histopathology revealed sclerosing fibrosis with keloid-like characteristics and abundant per lobular and intralobular lymphocytic infiltrates. Most of these instances will be resolved by clinicopathological correlation, which will help to avoid invasive surgical operations. However, imaging studies may not be appropriate in many situations. Understanding this pathology’s proclivity is crucial because it shares many clinical and radiological features with breast cancer. Here we report a unique case study of Lymphocytic mastopathy in a 44-year-old female patient with complaints of pain in the right breast for 15 days. Ultrasonograms and histopathological studies have been conducted, which evidenced lymphocytic mastopathy.

Keywords: Sclerosing Lymphocytic Mastitis, Diabetic Mastopathy, Malignancy and Mimicker.
1. INTRODUCTION

Lymphocytic mastopathy, also known as fibrotic mastopathy, diabetic mastopathy, or sclerosing lymphocytic lobulitis, is a benign clinic pathological condition affecting young and middle-aged women (34–47 years). Its principal differential diagnosis is breast carcinoma. It is a set of clinical, radiographic, and histological characteristics identified in dense fibrous masses of the breast that was first characterized in 1984 by Soler and Khardori.1 Even though it is benign, its radiological features are notable since they intuitively look like those of malignant breast disease. Since this condition has historically been recorded on both type one and type two people with diabetes independent of their level of insulin dependency, long-term diabetic patients have generally been connected with it.2,4 The existence of a suspicious breast lesion in a postmenopausal woman practically demands surgical excision, even though core biopsy samples may be definitive for lymphocytic mastopathy. This is applicable daily when dealing with a newly diagnosed breast mass in a previously disease-free patient (from the perspective of the breast). Due to its propensity to recur, this disease should be carefully monitored by a breast surgeon because it has the potential to be deforming. It can have a single, multiple, unilateral, or bilateral focus.3,18. Due to its contradictory characteristics and lack of malignant pathological findings, imaging evaluation has forever been troublesome. Nevertheless, radiological highlights demonstrative of cancer is regularly noticed, particularly in ultrasoundography (US). The principal observed features are irregular, hypoechoic masses with enhanced acoustic shadow.10,13,16 With dense parenchymal mass not distorting the normal breast tissue and regularly revealed micro calcifications, mammograms commonly give no additional data.17 This demonstrates that, while being an uncommon and benign condition, lymphocytic mastopathy can occasionally bear a heavy burden of disease like in our case, giving the initial dilemmatic impression of suspicion of malignancy.

2. CASE REPORT

44 years’ nulliparous female presented with complaints of pain in the right breast with 15 days’ duration.

2.1 Medical History

History of swelling in the right breast since two weeks which is insidious in onset and gradually attained present size. No history of nipple discharge or breast trauma. No significant family history.

2.2 Observation and Examination

Examination showed swelling of size 7x5 cm in both lower quadrants between 3-7 o clock position of right breast, nipple retraction present, firm in consistency, skin is erythematous, anterior axillary lymph nodes palpable. The left breast examination is unremarkable.

2.3 Investigation

Ultra sonogram (Figure 1, 2) revealed a hypoechoic, lobulated, irregular lesion in the right breast extending from 6-9 o clock position, inflammatory changes present in the fibro glandular layer of the right breast; features suggestive of mass lesion of right breast BIRADS-IV.

Fig 1: Ultrasonogram
2.4 Diagnosis

Gross pathology demonstrated multiple grey-white soft tissue bits altogether, measuring 1 cc. All sections were sent to be processed and stained with hematoxylin and eosin. Histopathological analysis of the prepared slides was performed. Histologically, the section studied shows multiple fragments of breast parenchyma containing fibrofatty stroma with few acini and ducts. Collections of chronic inflammatory cells consisting of lymphocytes and plasma cells are seen in a few places. Blood vessels are thick-walled and congested. The occasional duct shows epithelial proliferation and fibrous tissue scarring. The patient is doing well and will be followed up on.

FIG 2: Ultrasonogram

Fig 3: Lymphocytic infiltration surrounding breast ducts. H AND E (100X)

FIG 4: Lymphocytic infiltration and congested blood vessels. H AND E (100X)
3. DISCUSSION

Lymphocytic mastitis, or diabetic mastopathy or sclerosing lymphocytic lobulitis, is a fibrotic inflammation of the breast created by long-haul insulin-dependent diabetes. It can likewise be connected to the immune systems and endocrine ailments. Soler and Khardori depicted it without precedent for 1984. However, Byrd et al. didn’t give it a name until 1987. The reason for this disease is questionable. A few thoughts have been introduced, including that it is an incendiary or immunological reaction to exogenous insulin treatment. Hyperglycemia changes the extracellular framework, as per another view. High degrees of glycemia increment development factors, which thus instigate the making of collagen impervious to obliteration on account of an interaction called glycosylation. This collagen acts as an antigen, causing B-lymphocyte expansion and counteracting agent development as an optional immunological reaction. The presence of macrophages would expand the number of development factors, bringing about expanded collagen arrangement. This makes sense of the pathology’s histologic highlights, which incorporate different levels of Type B lymphocytic invasion at the per lobular (lobulitis), periductal (ducts), and perivascular (vasculitis) levels, as well as a lot of keloid fibrous tissue. Epithelioid histiocytes are likewise present, principally in diabetes patients. Diabetes, thyroid disorders, and connective tissue abnormalities are entirely connected, as per Soler and Khardori. HLADR3, HLADR4, and HLADR5 were found in their DMP occasions, as per Soler and Khardori. HLADR3, HLADR4, and HLADR5 were found in their DMP occasions, which are indicated in autoimmune disorders. The inflammatory lobules in diabetic mastopathy (DMP) are like lymphoepithelial lesions seen in Hashimoto thyroiditis and Sjögren condition. Consequently, this could be the etiology. In their examination, Sternberg et al. found that in diabetics, glycosylation and expanded intermolecular cross-linkages render collagen impervious to degradation. This outcome in the development of connective tissue, which is a component of different connective tissue diseases in diabetics, for example, mastopathy. Tomaszewski guessed that advanced glycosylated end products created in hyperglycemia are neo-antigens, producing an immune system reaction portrayed by B-cell expansion and autoantibody amalgamation. Because of the cytokine discharge, the framework would expand. Whenever Hunfeld and Bassler assessed the stromal and lymphocytic qualities of DMP and non-diabetic lymphocytic mastitis, they found that the latter has more noteworthy T cell extents and less apparent fibrosis. More prominent stromal fibrosis, lobular atrophy, and the presence of epithelioid myofibroblasts recognize DMP. Because of the keloid-like fibrosis, patients present with an enormous firm to hard lump. This is as often as possible confused with malignant growth. Patients with well-established DM are bound to have a drawn-out long-term history. Mammography and ultrasonography are regularly insufficient in recognizing definitive lesions. The most widely recognized imaging discoveries in ultrasonography are irregular hypoechoic masses with noticeable posterior acoustic shadowing. The attributes of attractive reverberation imaging discoveries range from diminishing diffuse contrast material upgrade to fast, substantial enhancement that is indistinguishable from breast cancer. As per Camuto, 60% of DMP repeat after careful surgical excision and will often be in a similar region, including more breast tissue than the past lesion; consequently, thorough biopsy should stay away. FNACs are noncontributory because keloid-like fibrosis creates few cells. Patients with recurrent lesions can be observed utilizing FNAC. In interesting cases, Core needle biopsy can be used as an analytic tool. Excisions and other superfluous surgical procedures need to be considered. This is huge since specific authors accept that removing the lumps expands the gamble of repeat.

4. CONCLUSION

In patients with long-term diabetes, lymphocytic mastopathy should be considered in the differential diagnosis of a breast lump. While it is commendable to be aware of this condition, it should be remembered that malignancy can lurk in the shadows of lymphocytic mastopathy. Because breast cancer is the most common differential diagnosis for lymphocytic mastopathy, understanding its clinical, pathological, and radiological presentations is paramount.

5. AUTHORS CONTRIBUTION STATEMENT

Dr. M. Pavithra - Significant conception and design contributions, data gathering, data study, and explanation while writing the article. By properly examining it for effective
intellectual content for the final version. DR. J. Thanka - Data analysis and interpretation, critical revision for effective

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7. CONFLICTS OF INTEREST

There are no conflicts of interest.

8. REFERENCES