

## Tuberculous Mastitis in Systemic Lupus Erythematosus : A New Case Report

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### Abstract

*We report a 45-year-old woman with a systemic lupus erythematosus complicated with a tuberculous mastitis. The patient developed bilateral breast swelling. Neoplasia was ruled out by radiological investigations. Biopsy and histological examination were consistent with the diagnosis of breast tuberculosis. It's a rare infectious complication in patients with systemic lupus erythematosus. A breast neoplasia or a lupus mastitis should be ruled out by appropriate investigations and deep biopsies. The first line treatment is based on anti tubercular drugs.*

**Keywords:** *Systemic lupus erythematosus, mastitis, tuberculosis.*

### INTRODUCTION

Systemic lupus erythematosus (SLE) is a non-specific autoimmune disease characterized by a clinical and a biological polymorphism. Its diagnosis is based on a combination of criteria defined by the American College of Rheumatology (ACR). If the different visceral manifestations are a part of the diagnostic criteria, they also have a prognostic and therapeutic value since the treatment depends on them. Like most dysimmune disease, corticosteroid therapy is the cornerstone of treatment. It is sometimes associated with immunosuppressive therapy that is mainly used in patients with renal or neurological impairment. The complications of this treatment are numerous and sometimes serious potentially life-threatening requiring vigilant monitoring. They are dominated by infectious complications which were previously the main cause of death in lupus patients. Tuberculosis is a very common opportunistic infection in this area. It can affect all organs. Tuberculous mastitis is exceptionally described in SLE. It poses a diagnostic problem since it requires eliminating underlying neoplasm and can be confused with lupus mastitis. We report a new case of tuberculous mastitis in SLE.

### CASE REPORT

A 45-year-old woman was admitted to our service for exploration of generalized edema. She had no particular history. The anamnesis revealed the occurrence since one month of edema of both lower limbs which was white, soft, pitting associated with painless swelling of the face and both hands and exertional dyspnea. These symptoms were associated with fever. There was no concept of impaired general condition or recent streptococcal infection. The general examination revealed a febrile patient at 38.5°C, mucocutaneous pallor, proteinuria three cross in urinary strips without hematuria, and confirmed edema of both lower limbs. Blood pressure was 16/9 cm Hg in both arms and cardiac auscultation was normal. Pleuropulmonary examination revealed a right pleural syndrome. Abdominal examination showed ascites of average abundance. Clinical examination didn't show other abnormalities. The electrocardiogram was normal. Biology showed microcytic hypochromic anemia (Hb=7.2 g / dL), leukopenia with lymphopenia (lymphocytes= 900 élé/mm<sup>3</sup>) and a normal platelet count. The inflammatory syndrome was evidenced by the rise of inflammatory markers such as C-reactive protein

(CRP), fibrinogen, and erythrocyte sedimentation rate (ESR). An impure nephrotic syndrome was retained in our patient since he had hypoprotidemia to 55g / l, hypoalbuminemia to 21.8g / l and proteinuria to 17.5g/24h with renal failure (creatinine clearance =53 ml / min). The serum complement was consumed and direct Coombs test was positive. On imaging, chest radiography showed pleural effusion of the right lung base and abdominal ultrasound showed an intraabdominal effusion associated with pleural and pericardial effusion confirmed by echocardiography. Immunological tests revealed a positivity of antinuclear antibody (ANA), anti DNA and anti Sm. Our patient had been diagnosed with SLE according to the ACR criteria namely: pleuropericarditis, lymphopenia, renal involvement with proteinuria > 0.5g/24h, positive ANA, positive anti DNA and anti Sm antibody. Anti phospholipid antibodies were negative. Ophthalmologic examination as well as salivary gland biopsy were without abnormalities. On the evaluation of dissemination of the SLE, the renal biopsy finds lupus nephritis stage 4. The therapeutic management was based on corticosteroids initiated by three boli of methylprednisolone 1 g / day, relayed by prednisone at a dose of 1 mg / kg / day for one month followed by a gradual degression. An immunosuppressive treatment based on cyclophosphamide at a dose of 750 mg every month for six months was also prescribed. The outcome was favorable with normalisation of renal function and disappearance of effusions. Antimalarials initially prescribed in our patient were then arrested because of ocular toxicity. Ten years later, a bilateral swelling of both breasts was discovered by the patient during self-examination without fever or poor general condition. There was no personal or familial history of breast pathology. At the mammary exam, she had normally configured breasts with a visible bilateral swelling about 7 cm in size on the right upper outer quadrant and 4 cm in size on the left upper outer quadrant. The swelling was painless, hard, limited and movable. There were no inflammatory signs. There were no nipple discharge or palpable lymph nodes in axillary or supra-clavicular areas. Biology showed no inflammatory syndrome. Breast ultrasound showed confluent, heterogeneous, hypoechoic lesions at both breasts. Mammography showed two irregular hypodense lesions without associated microcalcifications. Histological examination of the biopsy of the left breast revealed fibroinflammatory

alterations, steatonecrosis without signs of malignancy. However, histological examination of the biopsy of the right breast showed granulomatous mastitis with caseous necrosis. The chest radiography was normal. The tuberculin skin test was positive. The diagnosis of tuberculous mastitis in a patient with SLE was retained. There were no other location of *Mycobacterium tuberculosis*. TB treatment was prescribed for six months with a dual clinical and laboratory follow-up. The outcome was favorable with disappearance of breast lesions.

### DISCUSSION

We report a case of tuberculous mastitis in a patient with SLE. Although TB is a common opportunistic infection in patients with SLE, its breast localization remains an infectious complication rarely featured on this field since two cases have been reported in the literature [1]. Despite high prevalence of tuberculosis, mammary cells offer great resistance to the survival and multiplication of *mycobacterium tuberculosis* [2]. Sir Astley Cooper was the one who reported first case of mammary tuberculosis in 1829 and named it "scrofulous swelling of bosom", since then there are off and on few cases reported in the literature [3]. The incidence of breast tuberculosis varies from 0.1%–3% of all breast diseases [4]. Several factors contribute to its development in SLE including the immunosuppression due to the disease itself and favored by the prescribed treatments but also the emergence of several diseases such as HIV infection inducing anergy. Mammary tuberculosis is said to be primary if no other focus of tuberculosis exists and secondary in the presence of a demonstrable focus. This primary focus could be in lungs or lymph nodes including axillary, paratracheal and internal mammary lymph nodes [5]. Primary forms result from infection of breast tissue through abrasions or through infection of lactiferous ducts in the nipple [6]. Secondary spread could be through hematogenous route, lymphatic route or via spread from contiguous structures. Most widely accepted hypothesis is retrograde lymphatic extension from axillary lymph nodes [7, 8]. Studies report axillary lymph node enlargement in 50%–70% cases of mammary tuberculosis which supports this hypothesis [9]. The clinical manifestations are not specific. Indeed, it may be a simple breast swelling without inflammatory signs or local breast lump with signs of malignancy that may lead to misdiagnosis. Moreover, many complications may be seen such

as ulceration, abscess formation, fistulization or an atrophic scar in the chronic forms [10]. Biological investigations may show an inflammatory syndrome, a rise of ANA titer or a consumption of serum complement fractions. Radiological investigations are still needed and are represented mainly by breast ultrasonography and mammography. They are not very helpful in making the diagnosis, these may however help in defining the extent of lesion but are unreliable in distinguishing tubercular mastitis from carcinoma [6, 11]. Mammographic abnormalities can be an asymmetrical density, irregular masses, an increased parenchymal density in the affected breast, suspicious microcalcifications, a skin thickening and a nipple retraction. Normal mammograms are reported with varying frequency, depending on the size and location of the lesion at the time of diagnosis and the density of the surrounding breast parenchyma [12, 13]. In the absence of distinctive clinical biological and radiological criteria, breast tuberculosis is often misdiagnosed and patients are subjected to extensive investigations before a definite diagnosis is made. Differential diagnoses can be discussed and are represented mainly by breast carcinoma and lupus mastitis. Therefore, biopsy lesions and histopathological examination remain necessary for diagnosis by showing granulomatous mastitis with caseous necrosis. In case of tumor diseases, signs of malignancy can be observed. Lupus mastitis is a rare benign inflammatory condition characterized by inflammation of the deep subcutaneous adipose tissues of the breast. It can be seen in patients with a known history of systemic lupus erythematosus or discoid lupus or rarely can be the initial presentation of these diseases. Histologically, the most common findings are the presence of a dense lymphoplasmacytic infiltration of lobules and hyaline fat necrosis. Radiologic characteristics of LM can mimic a malignant lesion. However, because histologic features of this lesion have been well defined, correlation with clinical history is important to arrive at an accurate diagnosis and, therefore, deliver appropriate patient management [14]. The first line of treatment is with antimalarial drugs such as hydroxychloroquine. Other drugs such as systemic steroids and cyclophosphamide have also been used [15]. Some authors have successfully treated LM with dapsone and thalidomide [10, 16]. Lupus mastitis does not require surgical treatment. In fact, it has been reported that trauma can initiate or exacerbate the condition [17].

Management of tuberculous mastitis remains based on anti tubercular therapy for a recommended period of nine months with two months of quadruple therapy HRZE and seven months of bitherapy HR. The results are often favorable with only medical treatment. However, excision of lump, aspiration or drainage of abscess or surgical management may be required. In refractory cases with destruction of the breast, simple mastectomy may be performed.

### CONCLUSION

Tuberculous mastitis remains a rare complication in SLE that one should know where to suspect. Its diagnosis requires the elimination of differential diagnoses dominated by tumour pathology. The positive diagnosis aided by radiological investigations is essentially based on histological study. Early diagnosis will optimize the therapeutic management and limit the tuberculous infection whose incidence is increasing due to several infectious, inflammatory, neoplastic or dysimmune pathologies as is the case in our patient. Antitubercular therapy remains the cornerstone of treatment with a favorable prognosis.

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## Tuberculous Mastitis in Systemic Lupus Erythematosus: A New Case Report

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