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REVIEW ARTICLE

Breast Tuberculosis

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Abstract Tuberculosis affects over a billion people worldwide. There is a raise in incidence of extrapulmonary tuberculosis in recent years. Mammary tuberculosis has been estimated to be 0.1 % of breast lesions examined histologically, and it constitutes about 3–4.5 % of surgically treated breast diseases in developing countries. Breast tuberculosis is paucibacillary and routine diagnostic tests such as microscopy, culture, and nucleic acid amplification tests such as polymerase chain reaction techniques do not have the same diagnostic utility as they do in pulmonary tuberculosis. Also, the histology resembles various other granulomatous mastitis. The coexistence of carcinoma and breast tuberculosis adds challenge to diagnosis. Correct diagnosis of tuberculous mastitis is important as the treatment of differential disease varies from steroid to surgery which can have devastating consequences in patients suffering from breast tuberculosis.

Keywords Tuberculosis breast · Mastitis · Granulomatous mastitis · Tuberculosis in pregnancy

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Introduction

Although over one billion people suffer from tuberculosis worldwide, mammary tuberculosis (TM) is an extremely rare condition [1]. Its prevalence has been estimated to be 0.1 % of breast lesions examined histologically, and it constitutes about 3–4.5 % of surgically treated breast diseases in developing countries [2].

The incidence of tuberculosis is sharply rising in developing and developed countries, and rare extrapulmonary manifestations of the past can pose challenges in clinical practice. Hani-Bani K et al. [3] believed that immigration from endemic areas, and the increasing prevalence of immunosuppressive disorders, including HIV infection, development of drug resistant strains of *Mycobacterium tuberculosis* might be responsible for increasing the incidence of TM in Western countries in the future. Moreover, the disease is not diagnosed easily because of its physical similarity to carcinoma and bacterial abscesses and other granulomatous diseases like idiopathic granulomatous mastitis which are also on a raise. TM is paucibacillary routine diagnostic tests such as microscopy, culture, and nucleic acid amplification tests such as polymerase chain reaction techniques do not have the same diagnostic utility as they do in pulmonary tuberculosis [4]. Thus, it is not uncommon for breast TB to be misdiagnosed either as nonspecific abscess or carcinoma [5]. A misdiagnosis can have devastating consequences as treatment of the differential diagnosis varies from steroid to surgery.

Epidemiology

The first case of mammary tuberculosis was recorded by Sir Astley Cooper in 1829 who called it “scrofulous swelling of the bosom” [6]. Breast tuberculosis is rare in the western

countries, incidence being <0.1 % of breast lesions examined histologically [7]. Incidence of this disease is higher in countries endemic for tuberculosis, like the Indian subcontinent, where it may be as high as 4 % [8]. In the Arabian Gulf, the frequency of the disease is reported to be between 0.4 and 0.5 % [9]. The most common causative organism is *M. tuberculosis* though there have been cases reported of atypical mycobacterium causing tubercular mastitis [10].

Although mammary tuberculosis is much more common in females, it has been previously reported to also occur in males. Lilleng et al. [11] in a study of 809 cases of male breast mass did not find a single case of mammary tuberculosis. Khanna et al. [1] reported two cases of male mammary tuberculosis within a series of 52 patients; Shinde et al. [12] reported three cases of male mammary tuberculosis within a series of 100 patients; and Harris et al. [13] reported one case of male mammary tuberculosis within a series of 38 patients. Gupta et al. [14] reported that comprising of 160 patients, only 6 were males. Incidence of male to female breast tuberculosis being around 1:30. The risk factors associated with TM include multiparity, lactation, trauma, past history of suppurative mastitis, and AIDS [15]. The increased susceptibility to the tubercle bacilli by lactation has been reported to be 7 % by Shinde et al. [12]. While Khanna et al. [1] noted a higher susceptibility of 30 %, the explanation includes the stress of childbearing and increased vascularity of the breast which could raise the chance of infection.

Routes of Infection There is a hypothesis that mammary gland tissue, like spleen and skeletal muscle, is resistant to and unsuitable for the survival and multiplication of *M. tuberculosis* [16]. Mckeown and Wilkinson [17] classified breast tuberculosis as primary when the breast lesion was the only manifestation of tuberculosis and secondary when there was a demonstrable focus of tuberculosis elsewhere in the body. Vassilakos [18] debated that primary breast tuberculosis was probably quite rare and breast infection is usually secondary to a tuberculous focus somewhere else such as pulmonary or lymph nodes, which may not be clinically or radiologically noticeable. Primary form may rarely result from infection of the breast through abrasions or through openings of the ducts in the nipple [18].

The breast may become infected in a variety of ways, e.g., (i) hematogenous, (ii) lymphatic, (iii) spread from contiguous structures, (iv) direct inoculation, and (v) ductal infection. Of these, the most accepted view for spread of infection is centripetal lymphatic spread [19].

The path of spread of the disease from lungs to breast tissue was traced via tracheobronchial, paratracheal, mediastinal lymph trunk, and internal mammary nodes. According to the Cooper's theory, communication between the axillary glands and the breast results in secondary involvement of the breast by retrograde lymphatic extension [20].

Supporting this hypothesis was the fact that axillary node involvement was shown to occur in 50 to 75 % of cases of tubercular mastitis [21]. Breast is resistant to tuberculous infection by blood stream, even in debilitated patients of tuberculosis. Occasionally, direct extension from contiguous structures such as infected rib, costochondral cartilage, sternum, shoulder joint, and even through the chest wall from a tuberculous pleurisy or via abrasions in the skin can occur [22].

Coincidental tuberculosis of the faucial tonsils of suckling infants has been suggested as one of the common routes of spread of breast tuberculosis from the suckling infant to the nipple, and in turn, to the lactating breast via lacticiferous ducts [22].

Classification of Breast Tuberculosis Breast tuberculosis was first classified into five different types by Mckeown and Wilkinson [17]: (i) nodular tubercular mastitis, (ii) disseminated or confluent tubercular mastitis, (iii) sclerosing tubercular mastitis, (iv) tuberculous mastitis obliterans, and (v) acute miliary tubercular mastitis.

The nodulocaseous form of breast tuberculosis presents as a well circumscribed, slowly growing painless mass the overlying skin may later get involved with formation of sinuses. In early stage, it is difficult to differentiate from a fibroadenoma, while at later stages, it mimics a carcinoma [23].

The diffuse or disseminated form is the second most common variety and involves the entire breast with multiple intercommunicating foci of tubercles within the breast. The overlying skin is thickened with multiple ulcers and discharging sinuses. Ipsilateral axillary lymph nodes are usually enlarged and matted. This form is more common in older females. The third type described by Mckeown is the sclerosing form. There is extensive fibrosis rather than caseation, in which the entire breast is hard and the nipple is retracted. This form is often seen in involuting breasts of older females and may be also mistaken for carcinoma breast.

The last two forms described by Mckeown are tuberculous mastitis obliterans and acute miliary tuberculous mastitis. Tuberculous mastitis obliterans is characterized by duct infection producing proliferation of lining epithelium and marked epithelial and periductal fibrosis. The ducts are occluded and cystic spaces are produced resembling "cystic mastitis." Acute miliary tuberculous mastitis occurs as a part of generalized miliary tuberculosis. Both forms are rarely encountered in recent literature and may be of historical importance only.

Tewari [16] has recently suggested reclassifying breast tuberculosis into three categories, namely, nodular, disseminated, and abscess varieties. The new classification takes into consideration the changes seen in clinical presentation of tuberculosis over the last two decades. Sclerosing tubercular mastitis, tuberculous mastitis obliterans, and acute miliary tubercular mastitis are all very rare today, while tuberculous breast abscess is more frequent. The latter is common among

young females and represents up to 30 % of cases in recent publications.

Clinical Features

Constitutional symptoms of tuberculosis (fever, weight loss, night sweats, or failing of general health) are infrequently encountered in the series by Khanna et al. [1], Shinde et al. [12], and Harris et al. [13], 21, 20, and 16 % of patients. Although both breasts are susceptible to infection, only 3 % of patients with tuberculosis of the breast are affected bilaterally [2].

Breast tuberculosis most commonly presents as a lump. The commonest location of the lumps is the central or upper outer quadrant of the breast [24]. It is probably due to frequent extension of tuberculosis from axillary nodes to the breast. Multiple lumps are less frequent. The lump is often indistinguishable from carcinoma breast being irregular, hard, and at times, fixed to either skin or muscle or even chest wall. But the lump is usually painful. Breast remains mobile unless involvement is secondary to tuberculosis of the underlying chest wall. Tuberculosis of the chest wall constitutes 1 to 5 % of all cases of musculoskeletal tuberculosis and can involve the sternum, costochondral junctions, rib shafts, costovertebral joints, and the vertebrae [25, 26].

Tuberculosis involving ribs and presenting with breast mass is a very rare entity, and only a few cases have been to date reported in the literature. A direct fistulous tract with the pleura or a destroyed rib fragment in the abscess can be seen due to disease.

The mass can be fluctuated and is usually covered with indurated tissue. It is usually fixed to the skin and fistulization is not uncommon. Nipple and skin retraction can also occur, but breast discharge and pain are uncommon [27]. Peau d' orange is often seen in patients with extensive axillary nodal tuberculosis. Purulent nipple discharge or persistent discharging sinus may be the rare presenting feature. Rare cases may present with erythema nodosum [28].

The clinical situations that arise are the presence of carcinoma and tuberculous mastitis, carcinoma in the breast with axillary tuberculous adenitis or both [29]. There does not appear to be a casual link between mammary tuberculosis and breast cancer, and there is no evidence that TB is carcinogenic at any site [30]. The simultaneous occurrence of carcinoma and tuberculosis can lead to many problems regarding diagnosis and treatment as there are no pathognomonic symptoms or signs to distinguish breast tuberculosis from breast cancer, especially if the upper outer quadrant is involved.

Idiopathic granulomatous mastitis (IGM) and TM have similar clinical symptoms. A breast mass is the most common presentation in IGM. The mass will sometimes penetrate the breast skin or the underlying pectoralis muscle. Nipple retraction, sinus formation, and axillary lymphadenopathy may be seen in IGM patients. These finding were also observed in TM and breast cancer.

Diagnosis

Breast tuberculosis diagnosis warrants a high index of suspicion on clinical examination and pathological or microbiological confirmation of all suspected lesions.

Imaging

The chest X-ray may show evidence of active or healed tuberculous lesion in the lungs and may also reveal clustered calcifications in the axilla suggesting the possibility of lymph node tuberculosis in suspected patients.

The common mammographic findings are coarse stromal texture with or without an ill-defined breast mass and skin thickening, which all are nonspecific for making a diagnosis. The mammographic picture of nodular tuberculosis is usually of a dense round area with indistinct margins seen without the classic halo sign found in fibroadenoma [31]. Disseminated variety mimics inflammatory carcinoma, and the radiographs show dense breast with thickened skin [32]. Sclerosing tubercular mastitis reveals a homogenous dense mass with fibrous septa and nipple retraction. A unique finding strongly suggestive of TM is the presence of a dense sinus tract connecting an ill-defined breast mass to localized skin thickening and bulge [33].

Lesions due to tuberculosis have no specific ultrasonographic findings. They are observed as heterogeneous, hypoechoic, irregular bordered masses with internal echoes, or sometimes as thick-walled cystic lesions that show internal septa and posterior acoustic enhancement [34]. In some cases, fistulas and thickening of Cooper ligaments and subcutaneous tissue were reported [35]. Ultrasound helps evaluate the relation between the lesion and the chest wall. Determination of more than one lesion that were connected to each other and fistula formation is consistent with disseminated type of mammography pattern. Color Doppler US findings of breast tuberculosis are not mentioned in the literature. Increased circumferential vascularization in avascular centered lesions might be interpreted as a sign of continuity of the infective process. The mammographic and sonographic features of tubercular mastitis in a study by Sakar and group found mass lesion mimicking malignant tumors (30 %), smooth bordered masses (40 %), axillary or intramammary adenopathy (40 %), asymmetric density and duct ectasia (30 %), skin thickening and nipple retraction, macrocalcification (20 % each), and skin sinus (10 %). On ultrasound, 60 % had hypoechoic masses, 40 % focal or sectorial duct ectasia, and 50 % axillary adenopathy [36].

CT scan is rarely performed other than in defining the involvement of thoracic wall in patients presenting with deeply adhered breast lump. Tubercular breast abscess may be seen as smoothly marginated, nonhomogeneous, hypodense lesion with surrounding rim on contrast CT [37]. A direct fistulous tract with the pleura or a destroyed rib fragment in the abscess can also be seen [38]. Breast abscess may be drained using CT guidance and also guided biopsy can be performed. CT can show areas of lung destruction beneath the pleural disease and is a valuable tool in demonstrating the extent of disease, in planning of surgery, and also in assessment of response to treatment [39].

MRI of the breast may reveal a smooth or irregular bright signal intensity lesion on T2-weighted images suggesting a breast abscess. The findings are nonspecific, and reports on MRI of the breast may help only in demonstrating the extramammary extent of the lesion [40].

Tissue Diagnosis

Fine needle aspiration (FNAC) is the most widely used initial invasive method for diagnosis of breast tuberculosis. Approximately 73 % of the cases of TM can be diagnosed on FNAC when both epithelioid cell granulomas and necrosis are present [41]. Failure to demonstrate necrosis on FNAC does not exclude tuberculosis in view of small quantity of the sample harvested and examined. Granulomatous inflammation of the breast is an inflammatory process with multiple etiologies (Table 1). It can be caused by breast cancer, tuberculosis, granulomatous mastitis (GM), sarcoidosis, fungal infections such as actinomycosis, parasites such as filariasis, Wegener's granulomatosis, duct ectasia, brucellosis, and traumatic fat necrosis [42, 43].

Table 1 Etiologic differential diagnosis in granulomatous lesions of the breast

Infectious
Mycobacterium tuberculosis
Blastomycosis
Cryptococcosis
Histoplasmosis
Actinomycosis
Filarial infection
Corynebacterium
Autoimmune process
Wegener granulomatosis
Giant cell arteritis
Foreign body reaction
Duct ectasia
Plasma cell mastitis
Subareolar granuloma
Periductal mastitis
Diabetes mellitus
Sarcoidosis
Fat necrosis
Idiopathic

Core needle biopsy yields a good sample often yielding a positive diagnosis. Open biopsy of breast lump, ulcer, sinus, or from the wall of a suspected tubercular breast abscess cavity almost always confirms breast tuberculosis. Mammary duct ectasia could be considered in the differential but epithelioid granulomas are not a usual feature of this disease: the macrophages tend to be foamy in appearance and multinucleate giant cells tend to be associated with cholesterol crystals. Wegener's granulomatosis can be excluded in the absence of a necrotising vasculitis. The granulomas in tuberculosis granulomas are seen to be centered around ducts rather than lobules, but in the larger more destructive granulomas, it was difficult to work out any spatial relationship with any structures [44, 45].

One of major differential of tuberculous mastitis is idiopathic granulomatous mastitis (IGM). Bani-Hani et al. [3] stated that the largest reported series of IGM came from developing countries. They suggested that IGM might reflect TM under diagnosis. The cytomorphologic pattern seen in TM is similar to that seen in IGM. The pathological criteria for the diagnosis of IGM include granulomatous inflammation with multinucleated giant cells, epithelioid histiocytes, and occasional features of fat necrosis, abscesses, sinus tract, and eosinophils. It is centered on lobules, but extensive inflammation may obliterate this feature. Minor ductal and periductal inflammation is usually seen. A predominantly neutrophilic background and the absence of necrosis favor a diagnosis of IGM. It should be considered when high numbers of epithelioid histiocytes are seen [46].

Culture of *M. tuberculosis* is the gold standard for the diagnosis of TB; it is often negative due to the paucibacillary nature of breast TB. Detection of AFB in a smear requires more than 10,000 organisms/mL; nucleic acid amplification test could be very helpful in establishing the diagnosis of TB in smear-negative samples [47]. Nucleic acid amplification tests (NAAT) such as polymerase chain reaction (PCR) are rapid and specific but suffer from low sensitivity especially in AFB smear-negative cases. Sensitivity as low as 50 % have been reported in some series [46]. This low sensitivity is due to the presence of polymerase enzyme inhibitors in approximately 20 % of extrapulmonary specimens. If formalin-fixed tissue is the only available material, sensitivity of NAAT is further compromised. Thus, a negative NAAT result does not exclude TB disease with certainty. Interferon gamma release assays and serology are of limited diagnostic value given that adults from TB endemic areas are expected to have high rates of positivity for these tests [48].

The role of polymerase chain reaction (PCR) in the diagnosis of breast tuberculosis, however, is less often reported. Khurram et al. reported a case series of 22 patients diagnosed

with breast carcinoma with an associated granulomatous reaction in axillary lymph nodes with or without necrosis [49]. All samples were examined using ZN stain for AFB and nested PCR assays for *M. tuberculosis* DNA. In all the cases, ZN stains for AFB were negative. *M. tuberculosis* DNA was detected in 11 (50 %) out of the 22 cases. Six of 12 cases which had granulomas in association with necrosis were positive for MTB-DNA, while 5 of 10 cases without necrosis were also positive for MTB-DNA. Given the paucibacillary nature and culture negativity in breast tissue and the overlap of clinical presentation, NAAT may serve as a valuable tool for diagnosis of breast tuberculosis. As treatment of other conditions that may be confused with tuberculous mastitis can potentially lead to dissemination of disease (steroids and methotrexate for idiopathic granulomatous mastitis), relying on procedures like FNAC and histopathology alone is not adequate. This is particularly true in countries endemic with TB or for patients belonging to any high-risk group, like immigrants from endemic areas. Tuberculous mastitis should seriously be considered in such clinical settings, and MTB-PCR should be part of the investigation requested in clinical samples from breast tissues [50].

Treatment

Medical therapy is the mainstay of therapy with antituberculous therapy (ATT). No specific guidelines are available for chemotherapy of breast tuberculosis, and therapy generally follows guidelines used for pulmonary tuberculosis. Success rate of medical therapy approaches 95 % in most series with 6 months of antituberculous therapy (2 months of isoniazid, rifampicin, pyrazinamide, and ethambutol/4 months of isoniazid and rifampicin). Some authors prefer the 9-month regimen (2 months of isoniazid, rifampicin, pyrazinamide, and ethambutol/7 months of isoniazid and rifampicin) due to lower relapse rate in general.

However, in some series, this therapy was administered in a different manner. For example, Khanna et al. [1] treated 52 patients with breast TB for a mean duration of 9 months, and they extended this period to 12–18 months in 18 patients. In a study by Harris et al. [13], one third of the patients were given a 9-month therapy. But in a study by Silva et al. [51], 6-month therapy was sufficient for all patients to recover. The standard infection with multidrug-resistant tuberculosis (MDR) has been reported. Therapy with combination of first-line and second-line drugs that include kanamycin, ofloxacin, ethionamide, para-amino salicylic acid (PAS), pyrazinamide, and isoniazid has to be used [52].

Minimal surgical intervention is required for drainage of breast abscess or biopsy from the abscess wall, scraping of sinuses in the breast, incisional or excisional biopsy. Small lesions are eminently treatable by an excision biopsy followed by a full course of ATT. Residual lump following ATT may require surgical removal. Simple mastectomy with or without axillary clearance is rarely required for extensive disease comprising large, painful ulcerated mass involving the entire breast and draining axillary lymph nodes rendering organ preservation impossible. For concomitant breast cancer, the form of surgery is dependant upon the stage of breast cancer.

Tuberculosis in Pregnancy

Tuberculosis not only accounts for a significant proportion of the global burden of disease; it is also a significant contributor to maternal mortality, with the disease being among the three leading causes of death among women aged 15–45 years [53].

A study by Schaefer reported a new case rate of 18–29/100,000 in pregnancy [54]. Also, untreated tuberculosis in pregnant women would be a definite risk for transmission of disease to the newborn. Patients co-infected with HIV have a greater incidence of extrapulmonary tuberculosis and multidrug-resistant tuberculosis. Since there is an increased blood flow to breast during pregnancy and lactation, there may be increased incidence of breast tuberculosis. Adequate data assessing the safety, tolerability, and long-term treatment outcomes of pregnant and postpartum women with tuberculosis are lacking, particularly for HIV-positive women on ART. The benefits of treatment during pregnancy, however, outweigh the risks [55]. INH, rifampin (RIF), and ethambutol (EMB) are all Food and Drug Administration (FDA) pregnancy category C, but available data do not suggest any significant adverse maternal–fetal effects or need for dose adjustment in pregnancy. INH rarely causes peripheral neuropathy in well-nourished adults; pregnancy may increase risk. Some experts recommend vitamin K for infants born to mothers taking rifampicin because of its potential association with hemorrhagic disease in newborns. Supplemental pyridoxine should be administered to an infant on INH or if the breast-feeding mother is taking INH because pyridoxine deficiency may cause seizures in the newborn. Streptomycin has been proved to be potentially teratogenic throughout pregnancy causing fetal malformations and eighth nerve paralysis with deficits ranging from mild hearing loss to bilateral deafness. Other aminoglycosides including kanamycin, amikacin, and capreomycin are also contraindicated during pregnancy. The safety of breast-feeding is an important issue. Several studies have measured the concentration of ATT drugs in breast milk [56, 57].

There is a consensus that breast-feeding should not be discouraged. Women with tuberculosis mastitis should breastfeed from the unaffected breast. ATT drugs should be taken preferably after breast-feeding, and the next feed could be a bottle feed. Drug concentration in breast milk is low and has no therapeutic value [58].

Conclusion

Extrapulmonary tuberculosis occurring in the breast is extremely rare. Breast tuberculosis is uncommon even in countries where the incidence of pulmonary and extrapulmonary tuberculosis is high. Tuberculous mastitis is paucibacillary, and routine diagnostic tests such as microscopy, culture, and nucleic acid amplification tests such as polymerase chain reaction techniques do not have the same diagnostic utility as they do in pulmonary tuberculosis. Presence of epithelial granulomas with caseous necrosis on histology is confirmatory. Newer diagnostic tests like PCR can be used in atypical cases with smear and culture negativity. Treatment of breast tuberculosis consists of antitubercular drugs with surgery in persistent lesions following treatment.

Conflict of Interest There is nothing to disclose.

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