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REVIEW

Breast tuberculosis – a review and diagnostic pathway

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Breast tuberculosis (TB) is a rare form of extrapulmonary tuberculosis, accounting for 0.1% of all breast conditions globally, with a higher incidence in endemic regions such as India, East Asia, and sub-Saharan Africa. It can mimic breast carcinoma, complicating timely diagnosis. Early recognition and proper treatment are crucial for favourable outcomes in breast TB. This review offers a detailed examination of breast TB, and presents a diagnostic pathway designed to improve the diagnosis and management of the disease. This literature review considers the epidemiology, pathophysiology, clinical features, and diagnostic approaches, highlighting the need for a high level of clinical suspicion, particularly in TB-endemic areas. By providing a structured diagnostic framework, this paper aims to improve prompt and accurate diagnosis of breast TB, enhancing patient care and outcomes.
Keywords: extrapulmonary tuberculosis, granulomatous mastitis, breast infection, breast tuberculosis

Introduction

Tuberculosis (TB) is a contagious, infectious disease caused by *Mycobacterium tuberculosis (MTB)*.[1] It is estimated that the common ancestor of modern strains of MTB might have appeared for the first time 20 000–15 000 years ago.[2] The first case of breast TB was documented in 1829 by Sir Astley Cooper who described it as a "scrofulous swelling in the bosom of a young woman."[3] TB currently infects nearly 2 billion people worldwide, with around 7–10 million new cases of TB each year.[2]

Incidence

Pulmonary TB accounts for most of the worldwide TB cases, with breast TB only making up a small percentage of the total TB burden. Breast TB has an incidence of around 0.1% of all breast cases, rising to 3% in endemic countries like India, East Asia and sub-Saharan Africa.[4,5] There is currently a re-emergence of TB in the West, which can be attributed to the increased prevalence of human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS), the emergence of multi-medication-resistant strains of TB and the increased movement of people.[6]

Pathophysiology

The reason breast TB is such a rare disease, even in endemic TB areas, is thought to be due to breast tissue being resistant to the development of TB because it provides an infertile environment for the survival and multiplication of the TB bacilli.[7] Breast TB can be described as either primary breast TB, where no other focus of TB can be detected, or secondary TB, from the spread of a TB focus elsewhere in the body. The proposed pathways that TB bacilli can reach the breast are – lymphatic spread, hematogenous spread, neighbourhood spread, transcutaneous spread and direct spread.

Lymphatic spread

Centripetal lymphatic spread is thought to be the most common route of spread of the organism to the breast. Spread can be traced from the lungs to the breast via tracheobronchial, paratracheal, mediastinal, internal mammary and axillary nodes. According to Cooper's theory, communication between axillary lymph nodes and the breast results in secondary breast involvement by retrograde lymphatic extension. Axillary lymphadenopathy is present in 35–80% of cases with breast TB, which supports this theory. Whether the concomitant lymphadenopathy is a primary source of the tuberculous breast infection or is secondary to breast involvement is unknown. Still, axillary lymphadenopathy occasionally precedes the appearance of a breast mass, thus implicating retrograde spread.[6,8,9]

Hematogenous spread

Hematogenous spread to the breast can potentially occur in miliary TB. Spreading via this route is thought to be extremely rare because breast tissue appears to be resistant to the hematogenous spread of TB. In an autopsy series of 34 patients who had died of miliary TB, TB was demonstrated in almost all organs except the breast.[7,10]

Neighbourhood spread

Direct spread can occur from a tuberculous infection near the breast. Examples would be a costal or sternal bone lesion (tuberculous osteitis), the shoulder joint (tuberculous arthritis), or the pleural space (tuberculous pleurisy).[8,10]

Transcutaneous spread

Rarely, transcutaneous penetration can occur through a cutaneous abrasion resulting from trauma to the breast.[11]

Direct spread

Penetration from the nipple by the milk ducts – dilated ducts during pregnancy and lactation are particularly susceptible to tuberculous infection.[6]

Risk factors

Several risk factors have been identified, leading to the increased likelihood of developing breast TB. These are multiparity, pregnancy and breastfeeding, immunosuppression, previous suppurative mastitis and trauma.

Multiparity

Multiparity is often linked with socioeconomic factors such as poor nutrition, overcrowding, and limited healthcare access, which can weaken systemic immunity and heighten susceptibility to TB, especially in endemic regions.[8,12,13]

Pregnancy and breastfeeding

Lactation, thought to protect the breast from carcinoma, increases susceptibility to TB, especially in the presence of poor general health and the stress of childbearing. This period leads to increased vascularisation of the mammary gland, which explains its susceptibility to TB.[14] Interestingly, increased breast vascularity during lactation facilitates the dissemination of the mycobacterium. During lactation, the mammary ducts are ectatic, which encourages canalicular contamination.[15]

Immunosuppression

The current rate of breast TB and HIV coinfection is not known, but there is a high rate of TB and HIV coinfection in general. John et al.[16] reported a 95% HIV and TB coinfection rate in a South African hospital.

Previous suppurative mastitis

Suppurative mastitis is thought to increase the risk of developing breast TB by creating a favourable environment for the colonisation or activation of *MTB*. Recurrent abscesses and chronic sinuses can be a nidus for the hematogenous, lymphatic, or direct spread of TB bacilli. Moreover, chronic or poorly managed mastitis may weaken local immune responses, potentially reactivating latent TB infection in individuals exposed to the bacterium, particularly in TB-endemic regions.[7,17,18]

Trauma

Trauma has been reported as a risk factor for developing breast TB, although the exact mechanism remains unclear. It is hypothesised that trauma creates a localised area of tissue damage, which compromises the immune defences of the breast and makes it more susceptible to infection. Additionally, trauma may serve as a potential trigger for the reactivation of latent *MTB* infection in predisposed individuals.[14]

Classification of breast TB

Breast TB was first classified into five subtypes by McKeown and Wilkinson.[18] Hamit and Ragsdale, in 1982, proposed using only three different types to classify the disease clinically – (i) the nodular form, (ii) the sclerosing form, and (iii) disseminated TB mastitis.[19] This latter classification is most used in recent publications and will be described in more detail.

Nodular tuberculous mastitis

The nodular form of breast TB presents as a well-circumscribed, slowly growing, painless mass. As the disease progresses, the overlying skin may become involved, ultimately causing the formation of sinuses that open on the skin surface. In the early stages, it might be challenging to differentiate from other breast lumps like a fibroadenoma, while in later stages, it may resemble a carcinoma.

Sclerosing tuberculous mastitis

This type is characterised by extensive fibrosis rather than caseation. The clinical features are a hard, painless lump that grows slowly and may cause nipple retraction. Often, the whole breast is involved in the fibrotic process. Sclerosing tuberculous mastitis is associated with involuting breasts in older females and may also be mistaken for breast carcinoma.

Disseminated TB mastitis

The disseminated form is characterised by multiple lesions associated with sinus formation. This form mimics inflammatory breast cancer.[15]

Clinical presentation

The most common clinical presentation is a solitary, painless, well-defined mass in the central or upper outer quadrant of the breast.[11,15] Axillary lymphadenopathy is the most common associated finding in 35–80% of cases.[6,11,20,21] Constitutional symptoms of TB (fever, weight loss and night sweats) are not common and only occur in 16–21% of cases.[8,15,17] Breast TB can rarely present as multiple or bilateral lumps.[6,15] In the later stages, it may progress to skin induration, skin ulceration, a fluctuant mass representing a breast abscess and multiple draining sinuses.[11,15] Peau d'orange, chronic discharging sinuses, and erythema nodosum can be present in advanced disease stages.[6,22]

Clinically, breast TB can resemble pyogenic and fungal infections, duct ectasia, idiopathic lobular granulomatous mastitis (ILGM), granulomatous reaction to autoimmune diseases, diabetic mastopathy and breast cancer. ILGM is the most common differential of breast TB and can present clinically with identical features to breast TB. Therefore, it is essential to exclude breast TB as a cause since the treatment of ILGM is with immunosuppression, which will aggravate breast TB. Breast TB mimicking breast carcinoma is uncommon, as evidenced by the limited number of cases reported in the literature.[23,24]

Diagnostic strategies

Current literature reports a delay to diagnosis of three to eight months, which could be attributed to the very low incidence and, therefore, low index of suspicion of breast TB.[9,21,25] To prevent a delayed diagnosis, a high index of suspicion is warranted when performing a clinical examination or dealing with an atypical breast infection.

Imaging

Chest X-ray

A chest X-ray (CXR) is indicated to assess for evidence of an active or healed tuberculous lesion in the lungs. An extramammary source is identified in less than 15% of cases, but if features of TB are seen on CXR, it may expedite the diagnosis.[26]

Ultrasound of the breast and axilla

In nodular TB, an ultrasound scan often reveals a well-defined oval hypoechoic mass with posterior acoustic enhancement (Figure 1). The ultrasound picture may resemble a fibroadenoma. These hypoechoic lesions demonstrate no vascularity but rather a fluid collection containing debris. Ultrasound-guided aspiration of purulent fluid distinguishes these lesions from solid breast masses. In the sclerosing form, textural change with no visible fluid may mimic inflammatory carcinoma. The disseminated form of TB is associated with multiple anechoic collections, with and without debris, scattered throughout the breast with or without associated fistulation to the skin (Figure 2). Axillary lymphadenopathy is a common finding in breast TB; lymph nodes can show a spectrum of cortical thickening, solid or necrotic lymph nodes (Figure 3).[20,25]

Mammogram

In the nodular type of breast TB, the mammogram may show increased density consistent with a mass lesion, which can be single or multiple. Mass lesions can vary from having a regular or irregular border, which can resemble breast cancer lesions (Figure 4). The sclerosing and disseminated types of the disease have non-specific mammographic findings, showing increased density and stromal thickening. Skin thickening and axillary lymphadenopathy are other common findings.[27,28]

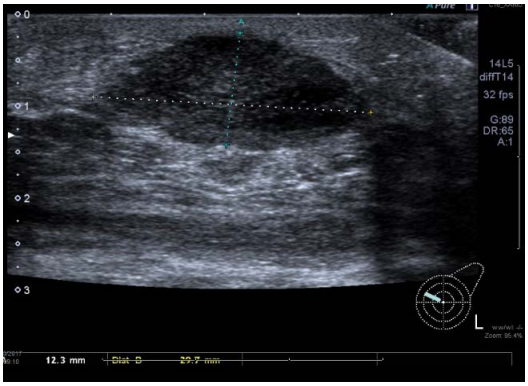

Figure 1: Well-defined oval hypoechoic mass with posterior acoustic enhancement

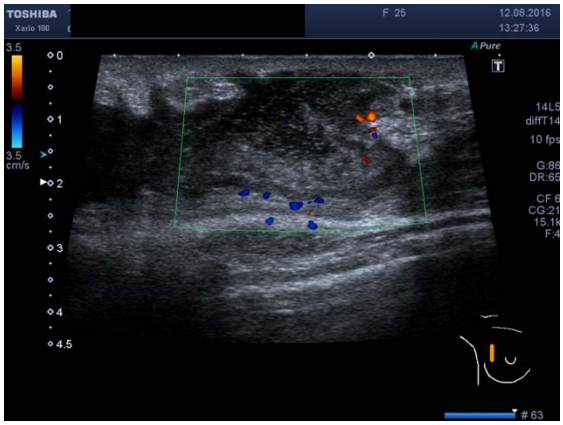

Figure 2: Large complex collection with non-dependent echogenic debris, skin thickening and increased vascularity peripherally

Computer tomography and magnetic resonance imaging

Computer tomography (CT) and magnetic resonance imaging (MRI) are not universally available in developing countries, and there have been few reports on CT and MRI findings in individuals with breast TB. The role of these imaging modalities is complementary to mammography and ultrasound scanning, especially in documenting the extramammary extent of the disease.[6,29]

Tissue diagnosis

The diagnosis of breast TB could be a challenge. Therefore, various tests are used in the diagnosis and further evaluation of patients with breast TB. The available tests are (i) Mantoux testing, (ii) Ziehl–Neelsen (ZN) stain, (iii) TB tissue culture and sensitivity, (iv) histopathology, (v) polymerase chain reaction (TB-PCR) tests, e.g. Xpert® MTB/RIF.[13]

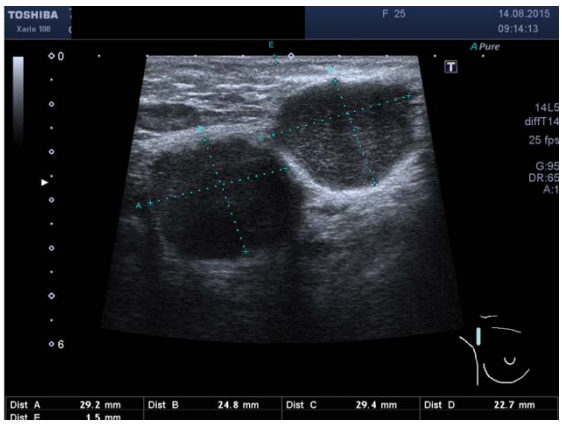

Figure 3: Large necrotic axillary nodes

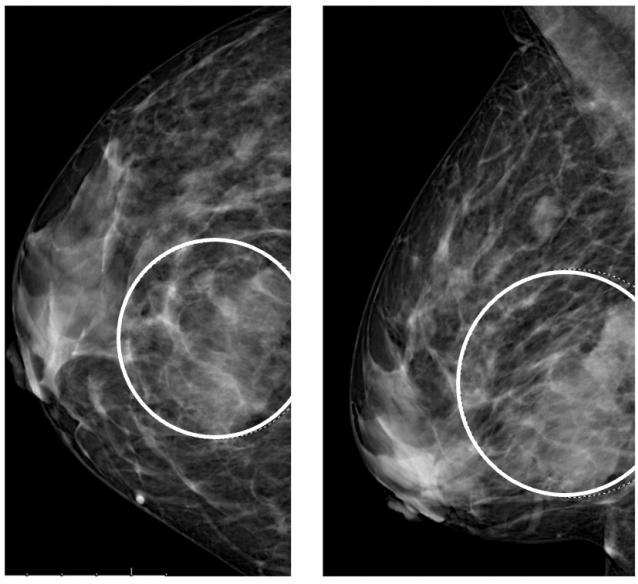

Figure 4: CC and MLO view of the right breast showing an irregular increased density in the lower inner quadrant

Mantoux testing does not offer a definitive diagnosis but confirms the patient's exposure to tuberculous bacilli. In endemic TB areas, the Mantoux test is of questionable value and likely to be positive because of previous exposure, and neither confirms nor rules out the diagnosis.[30]

Tissue samples can be obtained using fine-needle aspiration cytology (FNAC), core needle biopsy (CNB) and open biopsy. Tissue biopsy is an essential procedure in assessing breast lumps, providing a definitive diagnostic method for excluding malignancy during the early stages of evaluation. While uncommon, there have been documented cases of the simultaneous coexistence of breast TB and breast cancer in the same patient.[31,32] The most extensive systematic review on breast TB conducted by Quaglio et al.[33] found that histological findings consistent with TB were apparent in 64.1% of cases who underwent FNAC and 92.8% of cases who underwent a biopsy. These tests can be used in isolation or combination to improve sensitivity and specificity. FNAC samples should be fixed onto slides and sent to the laboratory for microscopy and ZN staining for the detection of acid-fast bacilli (AFB). CNB specimens should be divided and sent as two separate samples – one portion should be placed in 10% formalin for histological analysis. At the same time, the remaining tissue should be forwarded to the laboratory for molecular testing and MTB culturing.[34]

Histology remains one of the most important diagnostic modalities for diagnosing MTB. The main histological findings in breast TB are caseous necrosis epithelioid granulomas and Langhans giant cells with lymphohistiocytic aggregates (necrotising granulomatous inflammation).[35] The main question regarding the diagnosis of breast TB is whether it requires the detection of AFB on ZN stain or culture or whether morphologic features of necrotising granulomatous inflammation on cytology or histopathology are adequate for diagnosis.

The gold standard for diagnosing breast TB is the detection of AFB on ZN staining or MTB tissue culture. However, both tests have low diagnostic yields due to the paucibacillary nature of breast TB.[27] As reported, the diagnostic yield ranges from 2–26.6% for ZN staining and 25% for tissue culture.[13,22,33] In a study by Bailey et al.,[36] 12 of 15 cases with cytologic features of necrosis and granulomas were culture-positive for *M. tuberculosis*, but AFB was only demonstrated in four cases.Another challenge with tissue culture is the lengthy time required to obtain results, which can take up to eight weeks.[37] Fluorescent microscopy for AFB detection has demonstrated higher sensitivity than ZN staining in extrapulmonary sites and paucibacillary tissues, with reported sensitivities reaching up to 40%. Although its application has been described in individual case reports, no systematic reviews or large-scale studies have been conducted to evaluate its diagnostic accuracy, specifically in breast TB.[38,39]

TB-PCR is infrequently used in low- to middle-income countries in the diagnosis of breast TB, mainly because of availability and cost. It was used in only 2% of cases, with a positive diagnosis in 58% of cases, as reported by Quaglio et al.[33] Xpert MTB/RIF Ultra® (Xpert Mycobacterium Tuberculosis / Rifampicin Ultra) is an automated molecular (nucleic acid amplification) test for diagnosing TB with comparable accuracy to culture.[40] Results are available after two hours with minimal hands-on technical time. When

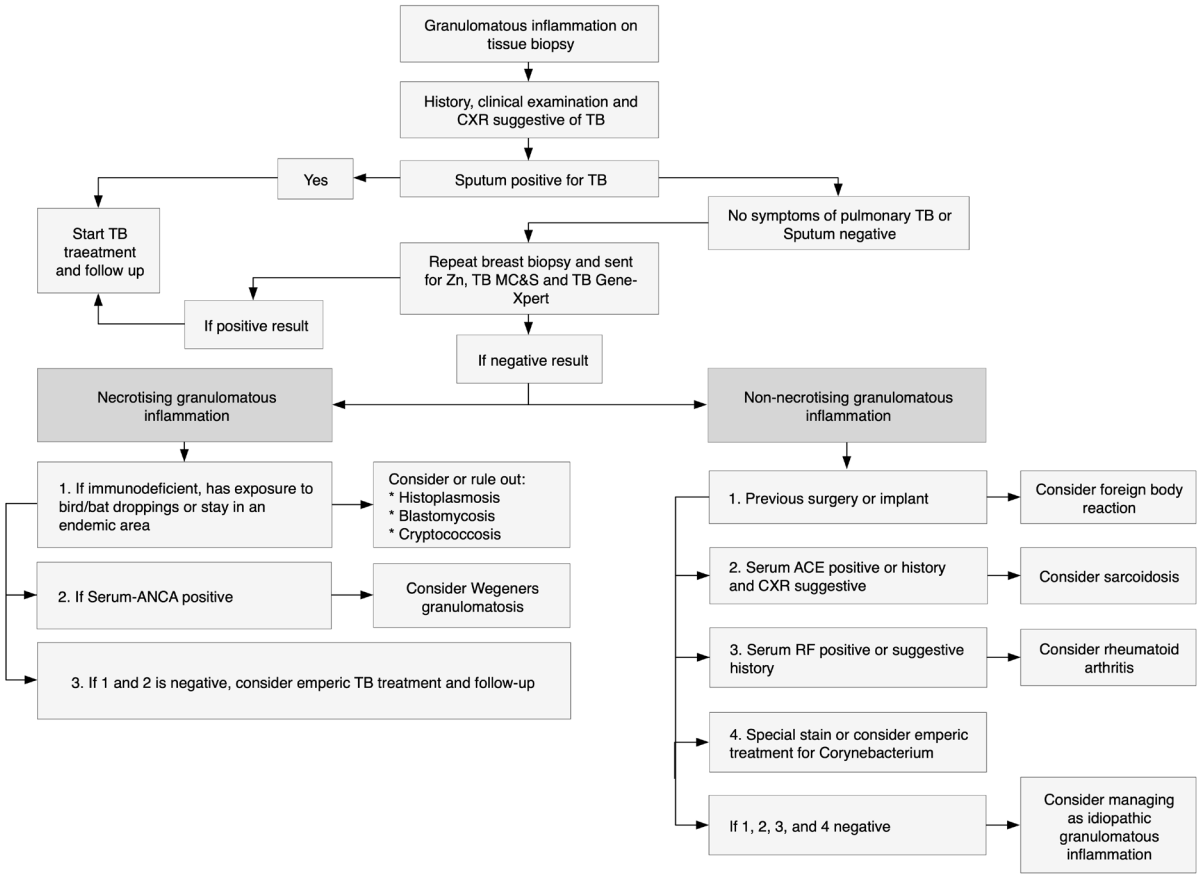

Figure 5: Diagnostic pathway for diagnosing breast TB

available, it offers an attractive option for both producing a quick diagnostic turnaround time and the presence of multidrug-resistant (MDR) TB. It has not been evaluated on breast TB but is effective on lymph node specimens.[41] South Africa was the first country to migrate from smear-based to molecular TB diagnostics, implementing Xpert in 2011, and the test is readily available across the country. It is routinely performed in the diagnosis of TB from pulmonary as well as extrapulmonary sites and should be incorporated in the diagnostic pathway of breast TB.[42]

One needs to be mindful that TB is not the only condition that can lead to granulomatous inflammation, as represented in Table I.[43] The definitive histological hallmark for diagnosing TB is the presence of necrotising granulomas. However, the absence of necrotising granulomas does not preclude the diagnosis of breast TB, and TB often presents with a combination of necrotising and non-necrotising granulomas influenced by the inflammatory process. Additionally, a small biopsy sample size can result in the non-visualisation of necrotising granulomas.[44] Other pathologies, such as infections with non-tuberculous mycobacteria, Wegener's granulomatosis, and granulomatous fungal infections, can also produce necrotising granulomas in breast tissue, as represented in Table II.[45] Despite the relatively lower prevalence of granulomatous breast fungal infections compared to breast TB, it is imperative to exclude these conditions, especially in patients with relevant histories, immunodeficiencies, or those residing in endemic regions.[45,46] In non-endemic TB areas, the most common causes of granulomatous inflammation of the breast include silicone granuloma, fat necrosis, and idiopathic granulomatous mastitis.[46] Tailoring investigative approaches based on patient history, physical exam, and geographical region is crucial, as certain diagnostic tests may be unnecessary in specific contexts. This approach ensures a more efficient and accurate diagnostic process, avoiding redundant or irrelevant investigations. Figure 5 illustrates a proposed diagnostic pathway for breast TB, emphasising the inclusion of serological tests, such as serum ANCA, ACE, and rheumatoid factor, to exclude alternative diagnoses before initiating TB treatment.

Table I: Causes of granulomatous lesions of the breast

Infection • Mycobacterium tuberculosis • Nontuberculous mycobacteria • Blastomycosis • Cryptococcosis • Histoplasmosis • Actinomycosis • Filarial infection • Corynebacterium
Foreign body reaction • Suture granuloma • Silicone granuloma
Autoimmune disease • Wegener's granulomatosis • Sjogren syndrome • Rheumatoid nodules
Miscellaneous • Idiopathic granulomatous mastitis • Sarcoidosis

Table II: Causes of necrotising granulomatous infection

Mycobacterium tuberculosis
Nontuberculous mycobacterial disease / atypical mycobacterial infection
Granulomatous fungal infections: • Histoplasmosis • Blastomycosis • Cryptococcosis
Wegener's granulomatosis

Treatment

Breast TB has a good prognosis, with anti-tuberculous therapy (ATT) forming the cornerstone of treatment. No specific guidelines are available for medical therapy of breast TB, and the most common approach is the standard anti-tuberculous regimes used for pulmonary TB with two months of isoniazid, rifampicin, pyrazinamide, and ethambutol, followed by four months of isoniazid and rifampicin. Extension of treatment to a nine-month regimen, consisting of two, months of isoniazid, rifampicin, pyrazinamide, and ethambutol, and seven months of isoniazid and rifampicin might be needed if the breast is not healed after six months of treatment.[15,47] In cases with slow clinical response, the continuation phase may extend to 12–18 months. In general, complete resolution is obtained in most patients.[8,15] Primary infection of the breast with MDR TB has been reported but is extremely rare.[48,49] The possibility of MDR TB needs to be considered if there was no initial culture done to confirm sensitivity and the infection fails to respond to standard or extended treatment. The treatment of MDR TB is with a combination of first-line and second-line drugs that include kanamycin, ofloxacin, ethionamide, para-aminosalicylic acid, pyrazinamide, and isoniazid.[48] Due to challenges in microbiological confirmation of breast TB, empiric TB treatment should be considered in TB-endemic areas when the clinical picture strongly suggests TB, particularly in the presence of necrotising granulomatous inflammation on histopathology or fine-needle aspiration biopsy. Early initiation of empiric treatment with standard ATT can prevent complications and progression of the disease.[13] Patients initiated on ATT, especially empiric treatment, should have regular follow-ups, with visits scheduled at least three, six, and twelve months after starting treatment to assess their response.

Surgery might be necessary in combination with anti-tuberculous medication in the treatment of breast TB. Less than 5% of the cases require radical surgical treatment in the form of subtotal or total mastectomy. Radical surgery might be an option in individuals not responding to medical treatment who have large, painful, ulcerative lesions involving the entire breast.[15] Other surgical interventions that might be needed include excision of a lump (to exclude malignancy), incision and drainage, cold abscess aspiration and resection of sinus formation or necrotic tissue. In the review done by Quaglio et al.,[33] excision was performed in 39% of cases, incision and drainage in 23% and 11% had cold abscess aspiration and resection of sinus formation or necrotic tissue.

Breast TB, if not promptly treated, can lead to long-term complications such as chronic sinus formation, residual scarring, and cosmetic deformity, which may cause psychological distress. Persistent fibrosis can result in chronic pain, and inadequate treatment increases the risk of

relapse. These outcomes underscore the importance of early diagnosis and complete adherence to ATT.[7,15,17]

Conclusion

TB of the breast is a rare condition, even in regions where TB is endemic, and it often mimics other breast diseases such as carcinoma or pyogenic infections. A high index of suspicion is essential in cases of atypical breast infections, particularly in patients from endemic areas. The presence of necrotising granulomatous inflammation in a tissue biopsy should prompt further evaluation for breast TB. Microbiological confirmation, such as culture or PCR for MTB, is ideal but may not always be feasible and does not always yield a positive result. Treatment involves standard ATT. Surgery is rarely necessary and is usually limited to diagnostic excision to rule out malignancy or to manage complications. Proper investigation and management are crucial to avoid complications and misdiagnosis.

Conflict of interest

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