RESEARCH PAPER

Microbial Spectrum and Antibiogram of Breast Abscesses: A Tertiary-Centre Comparison of Lactational and Non-Lactational Disease

Dr Smruti Paramjyoti[1], Dr Nanditha Gudi[2], Dr M S Dwarakesh[3], Dr Sudarshan P B[4]

[1]Post Graduate, Department of General Surgery, Saveetha Medical College and Hospital, Chennai, India.
[2]Associate Professor, Department of General Surgery, Saveetha Medical College and Hospital, Chennai, India.
[3]Post Graduate, Department of General Surgery, Saveetha Medical College and Hospital, Chennai, India.
[4]Professor, Department of General Surgery, Saveetha Medical College and Hospital, Chennai, India.

Abstract

Background: While lactational mastitis remains the classic precursor to breast abscess, an increasing share of cases now presents outside any lactational context. We set out to map the causative organisms of breast abscess in both lactating and non-lactating women treated at our institution, and to translate the resulting antibiogram into practical guidance for first-line antibiotic selection.

Methods: We conducted a one-year retrospective chart review (March 2023–March 2024) of every patient admitted with breast abscess at our tertiary centre, drawing age, presenting features, pus-culture outcomes, and sensitivity data from hospital electronic records and the microbiology laboratory register.

Results: The final cohort numbered 100 patients — 78 (78%) with lactational abscess (Group I) and 22 (22%) with non-lactational abscess (Group II) — with a mean age of 32.4 ± 8.7 years (range 18–56) and 83% falling between 18 and 34 years. Culture yield was positive in 92% of specimens. Staphylococcus aureus dominated the isolate profile in both arms (83.8%, 62/74 in Group I; 55.6%, 10/18 in Group II), while Group II additionally harboured Group B Streptococci (22.2%), Proteus spp. (11.1%), and Acinetobacter spp. (11.1%). Not a single MRSA isolate was recovered from either arm.

Conclusions: Pinpointing the causative organism and its susceptibility profile remains the cornerstone of rational breast-abscess therapy. Non-lactational disease carries a distinctly broader, more mixed bacterial signature than lactational disease, though S. aureus retains its position as the single most frequently recovered pathogen across both groups.

Keywords: Antibiotic susceptibility; Breast abscess; Lactational breast abscess; Microbial flora; Non-lactational breast abscess; Staphylococcus aureus

How to cite this article: Paramjyoti S, Gudi N, Dwarakesh MS, Sudarshan PB. Microbial spectrum and antibiogram of breast abscesses: a tertiary-centre comparison of lactational and non-lactational disease. Int J Drug Deliv Technol. 2026;16(74s): 766-772. DOI: 10.25258/ijddt.16.74s.91

Introduction

Breast abscess imposes a substantial burden on women's health, with disproportionate impact on younger patients, those from lower-income backgrounds, and individuals with obesity, active smoking, diabetes, or HIV infection [1,2]. Its pathogenesis is rarely attributable to a single cause; rather, it emerges from a convergence of factors undertreated antecedent mastitis, first pregnancy, older maternal age at childbirth, psychological stress, sleep disruption, suboptimal latching technique, immune compromise, and cigarette smoking all contribute [2,3]. Staphylococcus aureus is traditionally cited as the leading pathogen across both lactational and non-lactational presentations, though the underlying infection is often polymicrobial in character, encompassing coagulase-negative staphylococci such as S. epidermidis, streptococcal species including S. pyogenes and the viridans group, and anaerobic organisms [4,5,6].

In certain regions, notably parts of India, clinicians must also keep atypical organisms such as Mycobacterium tuberculosis on the differential list, especially when presentations are unusual or fail to respond to standard antimicrobial therapy [7]. The lactational form of the disease is most often traced to milk stasis following obstruction of the lactiferous ducts, with the causative organisms typically seeded from the breastfeeding infant's nasopharynx or the mother's own skin flora [3,8]. A parallel and growing concern is the expanding footprint of community-acquired methicillin-resistant Staphylococcus aureus (MRSA); agents conventionally reserved for such strains trimethoprim-sulfamethoxazole, fluoroquinolones, clindamycin display inconsistent and shifting resistance profiles, making local surveillance indispensable. Against

this backdrop, the present study set out to define the contemporary microbial landscape — MRSA prevalence included and the antibiotic susceptibility profile of lactational versus non-lactational breast abscess at our centre, with the aim of sharpening both empirical and definitive prescribing practice.

Materials and Methods

Study Design and Setting

Retrospective cohort study at Saveetha Medical College and Hospital, a tertiary care centre .

Data were retrieved from hospital electronic medical records and the microbiology database for all women diagnosed with breast abscess between March 1, 2023 and March 31, 2024. 320 records with a diagnosis of breast abscess (ICD-10 code N61) were initially identified and screened. After applying inclusion/exclusion criteria, the final analytical sample comprised 100 patients.

Inclusion Criteria

- Female patients aged 18 years and above.
- Clinical and/or ultrasound-confirmed diagnosis of breast abscess.
- Availability of a complete electronic medical record.
- Availability of a pus sample sent for culture and sensitivity testing.

Exclusion Criteria

- Incomplete medical records or missing laboratory reports.
- Underlying active malignancy of the affected breast.
- Abscess resulting from direct trauma or post-surgical infection.

Data Collection

Data were extracted using a standardised, pre-piloted form by two independent researchers, with discrepancies resolved by a third senior investigator. Variables recorded:

- Demographic data: age, parity, lactational status.
- Clinical presentation: symptom duration, pain, swelling, erythema, fever, axillary lymphadenopathy.
- Risk factors: diabetes mellitus, smoking, obesity (BMI ≥30 kg/m²), nipple piercing, HIV status.
- Microbiological data: pus culture result and identified organism(s).
- Antibiotic susceptibility: Kirby–Bauer disk diffusion per CLSI guidelines.
- Treatment data: empirical/definitive antibiotics and drainage procedure performed.

Definitions

- Lactational Breast Abscess: diagnosed in women breastfeeding at abscess onset or within 6 weeks of cessation.
- Non-lactational Breast Abscess: diagnosed in women not breastfeeding and beyond 6 weeks post-partum.
- Culture Positivity: growth of a pathogenic organism on standard aerobic culture media after 48 hours of incubation.

Statistical Analysis

Analyses were performed in SPSS version 28.0. Categorical variables are reported as frequency and percentage; continuous variables as mean ± standard deviation. Between-group comparisons used the Chi-square or Fisher exact test as appropriate; significance was set at $p<0.05$. Microbial flora and resistance profiles are summarised in tables and figures.

Results

Of 100 patients meeting inclusion criteria, 78 (78%) had lactational breast abscesses (Group I) and 22 (22%) had non-lactational abscesses (Group II). Mean age of the cohort was 32.4 ± 8.7 years (range 18–56); 83% were young adults aged 18–34 years. Group I mean age was 28.5 years versus 43.1 years in Group II, reflecting an older, peri-menopausal profile in the non-lactational group. Breast pain (98%) and localised swelling (96%) were the most common presenting symptoms in both groups. Fever occurred in 65.4% of Group I versus 40.9% of Group II. Axillary lymphadenopathy was present in 58% overall, more frequent in Group I (64.1%) than Group II (36.4%). Pus culture was positive in 92% (92/100) of samples overall — 94.9% (74/78) in Group I and 81.8% (18/22) in Group II.

Staphylococcus aureus was the predominant organism in both groups: 83.8% (62/74) of Group I isolates versus 55.6% (10/18) of Group II isolates. Group II showed a markedly more polymicrobial flora, additionally including Group B Streptococci (22.2%), Proteus spp. (11.1%), and Acinetobacter spp. (11.1%). No MRSA isolates were identified in either group. Antibiotic sensitivity patterns differed markedly between groups: Group I S. aureus isolates were highly sensitive to ciprofloxacin (98%), cephalexin (95%), cloxacillin (93%), and cefotaxime (90%), with notable resistance to ampicillin (88%) and gentamicin (35%). Group II gram-negative organisms (Proteus and Acinetobacter spp.) showed substantial resistance to first-line agents (ampicillin, ciprofloxacin, cephalexin) but retained

sensitivity to amikacin, tobramycin, meropenem, and piperacillin.

Ultrasound-guided needle aspiration was the predominant drainage modality in Group I (85%), whereas incision and drainage was more frequently required in Group II (59%) owing to thicker pus and loculation. Empirical cloxacillin was the most common initial antibiotic (68% Group I, 45% Group II); the regimen was subsequently modified based on culture and sensitivity results in 78% of cases.

Table 1. Demographic and Clinical Profile of Study Participants

Characteristic	Total (n=100)	Group I: Lactational (n=78)	Group II: Non-lactational (n=22)
Mean age, years ± SD	32.4 ± 8.7	28.5 ± 5.2	43.1 ± 7.9
Age 18–34 years, n (%)	83 (83%)	72 (92.3%)	11 (50.0%)
Breast pain, n (%)	98 (98%)	77 (98.7%)	21 (95.5%)
Localised swelling, n (%)	96 (96%)	75 (96.2%)	21 (95.5%)
Fever, n (%)	60 (60%)	51 (65.4%)	9 (40.9%)
Axillary lymphadenopathy, n (%)	58 (58%)	50 (64.1%)	8 (36.4%)

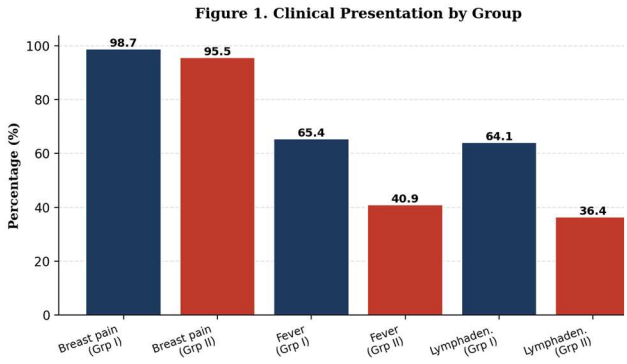

Figure 1: Comparison of key presenting clinical features between lactational (Group I) and non-lactational (Group II) breast abscess patients.

Table 2. Distribution of Isolated Microorganisms (Culture-Positive Cases)

Organism	Group I: Lactational (n=74)	Group II: Non-lactational (n=18)
Staphylococcus aureus	62 (83.8%)	10 (55.6%)
Staphylococcus epidermidis	12 (16.2%)	0 (0.0%)
Group B Streptococci	0 (0.0%)	4 (22.2%)
Proteus spp.	0 (0.0%)	2 (11.1%)
Acinetobacter spp.	0 (0.0%)	2 (11.1%)

No case of methicillin-resistant Staphylococcus aureus (MRSA) was identified in either group.

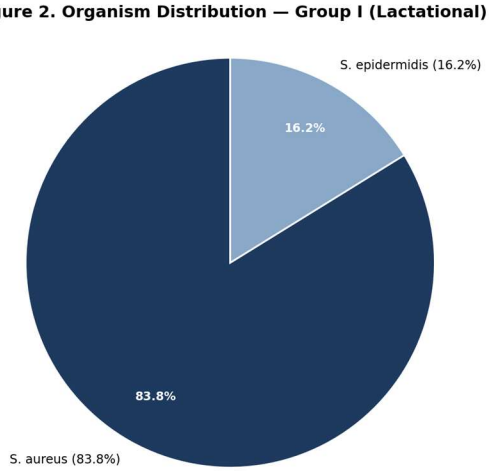

Figure 2: Distribution of culture-positive organisms among lactational (Group I) breast abscess patients (n=74).

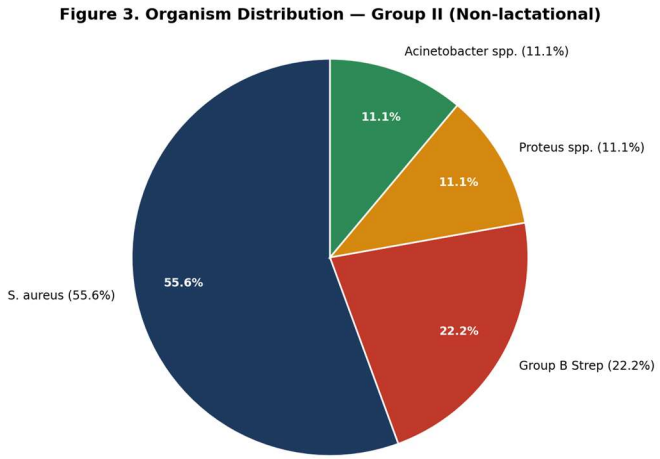

Figure 3: Distribution of culture-positive organisms among non-lactational (Group II) breast abscess

patients (n=18), illustrating a markedly more polymicrobial pattern than Group I.

Table 3. Antibiotic Sensitivity and Resistance Profiles

Organism	Highly Sensitive Antibiotics	Resistant Antibiotics
Group I: S. aureus	Ciprofloxacin, Cephalexin, Cloxacillin, Cefotaxime	Ampicillin, Gentamicin
Group I: S. epidermidis	Ciprofloxacin, Cefoxitin, Cephalexin, Cloxacillin, Erythromycin	Ampicillin, Gentamicin
Group II: S. aureus	Erythromycin, Cefoxitin, Methicillin, Ciprofloxacin, Cloxacillin	Ampicillin, Gentamicin
Group II: Group B Strep	Penicillin, Ampicillin, Erythromycin	Gentamicin, Cloxacillin
Group II: Proteus spp.	Amikacin, Tobramycin, Cefepime, Netilmicin	Ampicillin
Group II: Acinetobacter spp.	Amikacin, Tobramycin, Polymyxin, Meropenem, Cefotaxime, Ceftazidime, Piperacillin	Ampicillin, Ciprofloxacin, Ofloxacin, Cephalexin

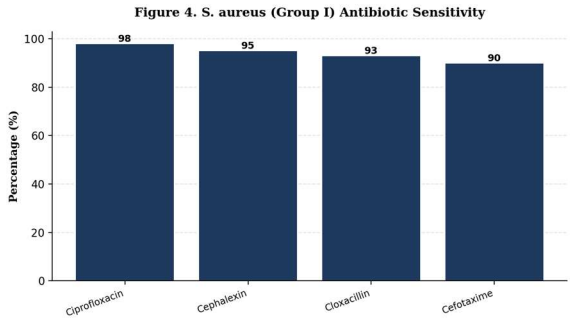

Figure 4: Percentage sensitivity of Group I Staphylococcus aureus isolates to key first-line antibiotics.

Drainage and empirical therapy: In Group I, ultrasound-guided needle aspiration was the predominant procedure (85%), followed by continuous catheter drainage (10%) and incision and drainage (5%). In Group II — owing to thicker pus and loculation — incision and drainage was performed more frequently (59%), followed by needle aspiration (36%) and catheter drainage (5%). Empirical cloxacillin was the most common initial antibiotic (68% Group I, 45% Group II), typically combined with amoxicillin-clavulanic acid or metronidazole for presumed polymicrobial non-lactational infection. Therapy was subsequently modified based on culture and sensitivity in 78% of cases.

Discussion

Two broad clinical categories describe this disease — lactational and non-lactational. Puerperal mastitis, reported across a wide range of 2.5–33% of breastfeeding women, is the usual antecedent to lactational abscess [1], with most cases emerging within the first three post-partum months or during the weaning period [2]; the driving mechanism is milk stasis behind an obstructed lactiferous duct [3]. By contrast, the non-lactational form is seen chiefly in peri-menopausal women and is linked to congenital ductal anomalies, ductal ectasia, and, in a smaller subset, an underlying malignancy [4]. Diabetes, smoking, obesity, and nipple piercing are recognised additional risk contributors [5], and the typical presentation is an acute, well-circumscribed fluctuant mass accompanied by systemic inflammatory signs [6,7].

Separating these two aetiologies matters clinically, since it shapes an individualised treatment pathway and may curb overall morbidity [8]. A demographic parallel is worth noting: Pachani and colleagues found 70% of their breast-abscess patients fell within the 20–39-year bracket [9], and our own cohort was similarly youthful — 83% aged 18–34 years — pointing to a consistent skew toward younger women. Sandhu et al. and Ekland et al., however, described an older peak (36–45 years) in their respective populations [10,11], hinting that geography or referral patterns may shift this demographic picture. Efem's series of 299 women likewise found lactational disease to be the dominant subtype [12], echoing our own split of 78% lactational to 22% non-lactational cases. This runs counter to Sandhu et al. and Bundred et al., who instead found non-lactational abscess more prevalent [10] — a divergence best explained by differing case-mix, catchment population, and referral behaviour between centres rather than any single unifying cause. S. aureus topped the isolate list among our lactational cases, mirroring earlier reports [1].

Notably, we recovered zero MRSA isolates, a finding that stands in contrast to the appreciable MRSA burden documented by Berens et al. and Al Benwan et al. [16,17]; while reassuring, this should be read cautiously given our single-centre scope, and may reflect local stewardship practice as much as any broader epidemiological trend. The non-lactational arm of our cohort displayed a markedly richer polymicrobial signature than the lactational arm — Group B Streptococci, Proteus spp., and Acinetobacter spp. all featured alongside S. aureus — underscoring the more heterogeneous gram-positive/gram-negative mix characteristic of this subgroup. Staying current with local antibiogram data is not optional if empirical prescribing is to succeed [19]. We would advocate a combined approach: antibiotics paired with prompt decompression, whether by needle aspiration or ultrasound-guided catheter drainage, reserving open incision and drainage for large or densely loculated collections [20].

Our own susceptibility findings support cloxacillin as a sound empirical starting point, supplemented with metronidazole or amoxicillin-clavulanic acid where a polymicrobial, typically non-lactational, picture is suspected [21,22]. Beyond antibiotic selection, our data carry a wider operational message for units managing breast sepsis: the striking divergence in organism profile between Group I and Group II argues against a "one-size-fits-all" empirical protocol. A unit-level policy that defaults every new breast abscess to a narrow-spectrum, staphylococcus-directed regimen risks under-treating the sizeable minority of non-lactational patients harbouring gram-negative or streptococcal co-pathogens; conversely, blanket broad-spectrum cover for all-comers would needlessly expose the majority lactational group to unnecessary antimicrobial exposure and its attendant resistance-selection pressure. It is also worth situating our zero-MRSA finding within a broader clinical-governance context. Regular, prospective local antibiogram surveillance — rather than reliance on historical or externally-published resistance data — should underpin empirical policy in any unit managing breast sepsis, since resistance patterns can drift over a relatively short interval and are known to vary substantially even between neighbouring institutions within the same city.

Strengths: dual-researcher data abstraction with third-party adjudication of discrepancies minimised transcription error; culture and susceptibility testing followed standardised CLSI methodology throughout, permitting direct inter-group comparison; and the one-year, single-season-spanning study window reduced the likelihood of the results being skewed by a short-lived local outbreak strain.

Limitations: the retrospective design cannot exclude residual documentation bias in symptom recording; the single-centre setting may limit generalisability to hospitals with differing catchment demographics or antibiotic stewardship practices; culture positivity of 92% still leaves a minority of abscesses microbiologically undefined, and these culture-negative cases could not be characterised further; and formal molecular typing (e.g., strain-level MRSA/MSSA genotyping) was not performed, which would have added mechanistic depth to the observed resistance patterns.

Future work would benefit from a prospective, multicentre design incorporating molecular characterisation of isolates and longitudinal antibiogram tracking, to determine whether the favourable susceptibility profile observed here is stable over time or represents a point-in-time snapshot.

Conclusion

Correct antibiotic selection is central to effective management of breast abscess in both lactational and non-lactational patients. Non-lactational breast abscesses are characteristically polymicrobial, whereas Staphylococcus aureus remains the single most prevalent organism across both groups. Treatment should be initiated promptly — most commonly in the emergency department — with early drainage via ultrasound-guided needle aspiration or continuous catheter placement to hasten resolution; incision and drainage should be reserved for extensive or refractory disease. Empirical first-line therapy with cloxacillin (or flucloxacillin), with addition of metronidazole or amoxicillin-clavulanic acid guided by clinical suspicion of polymicrobial infection and subsequently refined by culture and sensitivity results, provides an effective and evidence-based treatment strategy.

References

1. Foxman B, D'Arcy H, Gillespie B, Bobo JK, Schwartz K. Lactation mastitis: occurrence and medical management among 946 breastfeeding women in the United States. Am J Epidemiol. 2002;155(2):103-14.
2. Dener C, Inan A. Breast abscesses in lactating women. World J Surg. 2003;27(2):130-3.

3. Kwist LJ, Larsson BW, Hall-Lord ML, Steen A, Schalén C. The role of bacteria in lactational mastitis and some considerations of the use of antibiotic treatment. Int Breastfeed J. 2008;3:6.
4. Cunningham RM. Abscess in the non-lactating breast. Am Surg. 1967;33(4):339-43.
5. Rizzo M, Gabram S, Staley C, Peng L, Frisch A, Jurado M, et al. Management of breast abscesses in nonlactating women. Am Surg. 2010;76(3):292-5.
6. Ekland DA, Zeigler MG. Abscess in the nonlactating breast. Arch Surg. 1973;107(3):398-401.
7. Silverman JF, Lannin DR, Unverferth M, Norris HT. Fine needle aspiration cytology of subareolar abscess of the breast: spectrum of cytomorphologic findings and potential diagnostic pitfalls. Acta Cytol. 1985;30(4):413-9.
8. Cusack L, Brennan M. Lactational mastitis and breast abscess: diagnosis and management in general practice. Aust Fam Physician. 2011;40(12):976-9.
9. Pachani AB, Shah JK, Pachani AB, Shojai AR. Breast diseases managed at a tertiary care center with a rural set up in rural area of India: critical analysis. Int J Med Res Professionals. 2016;2(3):213-22.
10. Sandhu GS, Gill HS, Sandhu GK, Gill GP, Gill AK. Bacteriology in breast abscesses. Scholars J Applied Med Sci. 2014;2(4E):1469-72.
11. Efem SE. Breast abscesses in Nigeria: lactational versus non-lactational. J R Coll Surg Edinb. 1995;40(1):25-7.
12. Benson EA. Management of breast abscesses. World J Surg. 1989;13(6):753-6.
13. Kataria K, Srivastava A, Dhar A. Management of lactational mastitis and breast abscesses: review of current knowledge and practice. Indian J Surg. 2013;75(6):430-5.
14. Berens P, Swaim L, Peterson B. Incidence of methicillin-resistant Staphylococcus aureus in postpartum breast abscesses. Breastfeed Med. 2010;5(3):113-5.
15. Al Benwan K, Al Mulla A, Rotimi VO. A study of the microbiology of breast abscess in a teaching hospital in Kuwait. Med Princ Pract. 2011;20(5):422-6.
16. Krishnapriya J, Gopal R, Shaw P. Breast abscess due to Escherichia coli — a case report. Ind J Res Reports Med Sci. 2014;4(4):16-8.
17. Trop I, Dugas A, David J, El Khoury M, Boileau J, Larouche N, et al. Breast abscesses: evidence-based algorithms for diagnosis, management, and follow-up. Radiographics. 2011;31(6):1683-99.
18. Ramakrishnan R. Breast emergencies. In: David SS, editor. Clinical Pathways in Emergency Medicine, Volume II. 1st ed. India: Springer; 2016. p. 393-402.
19. Ferrara JJ, Leveque J, Dyes DL, Lorino CO. Nonsurgical management of breast infections in nonlactating women: a word of caution. Am Surg. 1990;56(11):668-71.