Duct Ectasia and Periductal Mastitis in Indian Women

Kirithiga Ramalingam · Anurag Srivastava ·
Seenu Vuthaluru · Anita Dhar · Rama Chaudhry

Received: 3 October 2013 / Accepted: 14 April 2014 / Published online: 8 May 2014
© Association of Surgeons of India 2014

Abstract There is very little awareness of the general physicians and surgeons about the benign breast conditions such as duct ectasia (DE) and periductal mastitis (PDM) causing nipple discharge. Not only that these benign breast diseases ring a false alarm of cancer, they are also the second most common cause of benign breast diseases. The objective was to study the clinical and microbiological profiles of duct ectasia and periductal mastitis in Indian women for better understanding of the disease process, in order to be able to treat them well. Forty-one consecutive patients presenting to the Surgical Out-Patient Department with non-bloody nipple discharge with clinical and radiological features suggestive of DE or PDM were included. Microbial culture and cytopathological study of the nipple discharge were done. Histopathological studies and culture of the ductal tissue taken intraoperatively were carried out. There is no significant difference in the age distribution among women with DE and PDM. Smoking is not associated with DE and PDM of Indian patients in contrast to the Western literature evidence. Infective etiology was present in nearly 46 % of the patients in the study population more so in the periductal mastitis cases. The most common isolated pathogens were *Staphylococcus aureus* and *Staphylococcus epidermidis*, unlike in Western population where nearly 50 % were anaerobes. Since the isolated organisms were resistant to the routinely

used antibiotics in high proportion of cases, culture and sensitivity should be done in all possible cases for appropriately treating the subareolar sepsis before proceeding with the definitive treatment in the form of duct excision.

Keywords Duct ectasia · Periductal mastitis · Nipple discharge · Culture of nipple discharge · Duct ectasia in Indian women · Mastitis in Indian women · Mammary duct excision · Benign breast disease · Management of periductal mastitis/duct ectasia

Introduction

Nipple discharge accounts for 5 to 10 % of all the breast problems [1–3]. Although 95 % of nipple discharge is related to benign condition [2, 4], it alarms the ladies who develop a fear of cancer. There is very little awareness of the general physicians and surgeons about the most common benign breast conditions causing non-sanguineous nipple discharge [4] such as duct ectasia (DE) and periductal mastitis (PDM). They are also the second most common cause of benign breast disorders next to breast cysts [5].

Often, these patients are investigated for prolactinoma by repeated estimations of serum prolactin levels and imaging of pituitary fossa which are unnecessary for the vast majority of cases. Most of them are treated inappropriately with anti-secretagogues. These women are also frequently misdiagnosed to have cancer as the clinical and radiological pictures can be confusing as is evident by the abundance of literature on periductal mastitis mimicking as cancer [6, 7]. Because of the lack of awareness of the treating physicians, these patients are subjected to undue stress and radical procedures which are unnecessary most of the times. Till date, the only definitive treatment for this condition is only surgical (microdochectomy, fistulectomy, and major mammary duct excision) [7–9]. Hence, it becomes surgically relevant to study the disease process, in

K. Ramalingam · A. Srivastava · S. Vuthaluru · A. Dhar
Department of Surgical Disciplines, All India Institute of Medical
Sciences, New Delhi, India

R. Chaudhry
Microbiology Department, All India Institute of Medical Sciences,
New Delhi, India

K. Ramalingam (✉)
Door No. D'3, Second Floor, Queens' Apartment, Banyan Beach
Resort Compound, Old pattinam Pathai, Kottakuppam,
Pondicherry 605104, India
e-mail: kirithigaramalingam@gmail.com

order to gain a good knowledge of these benign breast diseases, which is in turn necessary to alleviate the fear of cancer and appropriately treat the disease.

The objectives of the present study were to study the clinical and microbiological profiles of DE and PDM in North Indian patients.

Materials and Methods

Forty-one consecutive patients presenting to the Surgical Out-Patient Department of All India Institute of Medical Sciences between June, 2010 and June, 2012 with non-bloody nipple discharge with clinical and radiological features of DE/PDM were included in this study with due permission from the Institute Ethics Committee.

Case definition of DE: clinical—presence of thick, colored, or creamy nipple discharge, thickened subareolar ducts, nipple retraction, and absence of signs of inflammation; sonographic—dilated subareolar ducts; mammographic—prominent ducts appearing as conical opacities with apex of the cone towards the nipple and coarse calcifications (characteristic) which can be ring like, lying on the duct wall or circular or needle shaped when the duct contents are calcified.

Case definition of PDM: clinical—history suggestive of previous episodes of subareolar sepsis, presence of pus/serous nipple discharge, and signs of periductal inflammation, tender palpable subareolar ducts, nipple retraction, and mammary fistula; radiological—dilated ducts and subareolar abscess.

Women with features suggestive or suspicious of duct papilloma or malignancy on imaging and on nipple discharge cytology were excluded from the study. A detailed history was elicited and findings of a thorough clinical examination were recorded on all patients. Ultrasonography of the breasts and axillae were done in all 41 patients. Bilateral digital mammography was performed on those above 35 years of age (35 patients). In this study, oral erythromycin 500 mg at six hourly intervals and metronidazole 400 mg eight hourly were given for 3 weeks in patients (10 patients) who presented with acute inflammation or subareolar abscess prior to surgical removal of mammary duct cone. The bacterial aerobic cultures of nipple discharge were done before starting the antibiotic therapy.

Nipple discharges of 38 patients who presented with active nipple discharge were smeared on glass slides and allowed to dry followed by which staining was done with Pap stain and Giemsa stain and viewed for cytopathology. After due informed and written consent, the patients were subjected to major mammary duct excision. Hematoxylin and eosin staining of the major mammary duct cones of all the 41 patients was performed for a detailed histopathological study on light microscopy. A microbiological study of the duct content

extracted during operation was also carried out by aerobic and anaerobic culture and sensitivity.

Results

The age of the study population ranged from 26 to 60 years with mean of 41.98 years. Duration of symptoms in the patients ranged from 1 to 96 months. The mean duration was 20.22 months. All the patients were multiparous except for one lady who was nulliparous. None were smokers and were consuming hormonal drugs. One patient had family history of ovarian cancer in her maternal aunt. Seven patients (17.07 %) out of 41 had undergone previous breast surgery (six incision and drainage of non-lactational subareolar abscesses and one case had undergone major mammary duct excision).

There is a statistically significant difference present among the women diagnosed to have DE, DE+PDM, and PDM on final histopathology with respect to the spontaneity of nipple discharge as the Fisher's *p* value is 0.023. Women in the DE+PDM and PDM group had more of a spontaneous nipple discharge than women in DE group, who had nipple discharge on expression. In 30 patients (78.95 %) out of the 38 patients with active nipple discharge, the discharge was intermittent. In majority of the cases, the discharge was from multiple ducts (39 patients, 95.12 %). The nature of the nipple discharge was serous in 11 patients (28.95 %), pus in 10 patients (26.32 %), green in four patients (10.53 %), yellow in one patient (2.63 %) and black in three patients (7.89 %), and watery in two patients (5.26 %).

Retroareolar inflammatory masses were present in six patients (14.63 %). Retroareolar pain was complained by 30 women in the study (73.17 %). Axillary lymphadenopathy was present in 16 cases (39.02 %). The remaining important clinical findings namely the menopausal status, diabetes mellitus, active nipple discharge, spontaneous nipple discharge, pus discharge, presence of subareolar sepsis in the present or past, nipple retraction, and presence of thickened ducts in the study population are depicted in Table 1.

On sonography, 21 patients (51.22 %) had features of duct ectasia, seven (17.07 %) had features of duct ectasia with retroareolar inflammatory mass, six (14.63 %) had features of abscess/mastitis, and seven (17.07 %) had features of fibrocystic disease (FCD) with duct ectasia. Nipple discharge cytology done in all the 38 patients with active nipple discharge had foamy macrophages in proteinaceous background.

Histopathological study: nine patients (21.95 %) had only DE, six had only PDM, six had both DE and PDM, five had only fibrocystic disease (FCD), seven had fibrocystic disease with PDM or DE, three had only papilloma, one had papilloma with fibrocystic disease, two had features of papillomatosis,

Table 1 Clinical features of study population

Features	Yes (%)	No (%)
Menopause	20 (48.78)	21 (51.22)
Diabetes	2 (4.88)	39 (95.12)
Active nipple discharge	38 (92.68)	3 (7.32)
Spontaneous nipple discharge	20 (52.63)	18 (47.37)
Pus discharge	10 (26.32)	28 (68.29)
Active infection	10 (24.39)	31 (75.6)
Subareolar sepsis	20 (48.78)	21 (51.22)
Nipple retraction	23 (56.1)	18 (43.9)
Thickened milk ducts	35 (85.37)	6 (4.63)

DE, and PDM, and two had epithelioid granulomas consistent with tuberculous mastitis (TB mastitis).

All the anaerobic cultures of the ductal tissue taken intraoperatively were negative (cultured up to 5 days). Out of 10 patients who had acute attack of subareolar sepsis at the time of presentation with active pus discharge from the nipple, two of them had grown *Staphylococcus aureus*, one had grown *Staphylococcus epidermidis*, and two had grown *Klebsiella pneumoniae*, one *Streptococcus pyogenes*, and two *Citrobacter freundii*. Two of the nipple discharge cultures were sterile. The aerobic culture results of 41 ductal tissues taken intraoperatively are correlated with the preoperative nipple discharge culture and final histopathological diagnosis in the Table 2.

Out of the 35 aerobic cultures of the ductal tissue of patients who were histopathologically proven to be DE/PDM, 19 were sterile and three were showing growth of more than three types of bacteria. Four patients had grown *S. aureus* in their culture, three had grown *S. epidermidis*, one *S. pyogenes*, one *K. pneumoniae*, one *Pseudomonas aeruginosa*, and two *C. freundii* and *Micrococcus* sp.

There is statistically significant difference in the culture positivity among the three groups (DE, DE+PDM, and PDM) as the Fischer's *p* value is 0.046. Only one culture was positive

in DE group (*Micrococcus* sp.) (one out of nine) unlike the DE+PDM (three out of six were positive) and PDM groups (four out of six were positive) (Table 3).

The organisms cultured showed resistance in most of the times for the following commonly used antibiotics namely penicillin, co-trimoxazole, erythromycin, and ciprofloxacin (Table 4).

In 16 patients, the indication of major mammary duct excision was constant retroareolar pain associated with the persistent nipple discharge. In 14 patients, the indication was presence of previous attacks of subareolar sepsis. In six patients, surgery was done for correction of nipple retraction and relief from nipple discharge. In three patients, surgery was performed for persistent retroareolar inflammatory mass. Two patients underwent surgery for copious nipple discharge which caused social embarrassment.

Discussion

The largest case control study (85 patients) on the risk factors for chronic mastitis including duct ectasia and periductal mastitis showed higher association with increased oral contraceptive pill usage which is not seen in our study [10]. Various studies from the Western literature have shown increased association with smoking and periductal mastitis [11–13]. However, in the present study, none of the women were smokers. On the contrary to Dixon et al. [14] study in 88 patients and Frantz et al study [15] which showed PDM is common in the younger age group and DE in older age group, there is no significant difference in the age distribution among Indian women with DE, DE+PDM, and PDM. The lack of difference in age distribution could be due to small sample size (41 patients). Nipple discharge was more spontaneous in women with PDM compared to DE, the difference being statistically significant. This suggests the active

Table 2 Aerobic culture of nipple discharge and ductal tissue

Organism	Nipple discharge (%)	Duct excision tissue (%)	Duct excision tissue in histopathologically proven element of DE/PDM (%)
Sterile	2 (20)	22 (53.66)	19 (54.29)
<i>Staphylococcus aureus</i>	2 (20)	5 (12.2)	4 (11.43)
<i>Staphylococcus epidermidis</i>	1 (10)	3 (7.32)	3 (8.57)
<i>Klebsiella pneumoniae</i>	2 (20)	1	1
<i>Pseudomonas aeruginosa</i>	–	1	1
<i>Streptococcus pyogenes</i>	1 (10)	1	1
<i>Micrococcus</i> sp.	–	1	1
<i>Citrobacter freundii</i>	1 (10)	2	2
>3 bacteria	–	4 (9.76)	3 (8.57)
Total	10	41	35

Table 3 Positive cultures of the ducts in patients with DE, DE+PDM, and PDM

	Sterile	<i>Staphylococcus aureus</i>	<i>Staphylococcus epidermidis</i>	<i>Streptococcus pneumoniae</i>	<i>Citrobacter freundii</i>	<i>Micrococcus</i> sp.	>3 types of bacteriae	Total
DE	8	0	0	0	0	1	0	9
DE+PDM	2	2	0	0	0	1	0	6
PDM	1	1	1	1	2	0	1	6
Total	12	3	1	1	2	1	1	21

Fisher's *p* value comparing the proportion of sterile culture versus positive bacterial culture in different groups=0.046

secretion of nipple discharge in patients with PDM. The comparison of clinical features of DE and PDM among the various studies is presented in the following Table 5.

Bundred et al. [20] isolated organisms from the spontaneous nipple discharge and pus from abscesses or fistula in 62 % of the patients (total of 51 patients). In the present study, we were able to isolate pathogens in nearly 46 % of patients in the

aerobic culture of the excised ductal tissue. This finding supports the role of infection in the pathogenesis of PDM group in our study. However, only one out of nine samples in patients with DE grew bacteria (*Micrococcus*? skin contaminant), indicating that the DE is probably non-infective in pathogenesis supporting the findings of previous studies [13, 20]. Some of the previous studies have mentioned that

Table 4 Antibiotic sensitivity pattern of the study population

	Penicillin	Rifampicin	Linezolid	Amoxicillin+ sulbactam	Cefoxitin	Cefoperazone+ sulbactam	Cefotaxime	Ceftazidime	Erythromycin	Amikacin
<i>Staphylococcus aureus</i>	S	S	S	—	S	—	—	—	R	S
	R	S	S	—	S	—	—	—	S	S
	R	S	S	—	S	—	—	—	R	S
	S	S	S	—	S	—	—	—	R	S
<i>Staphylococcus epidermidis</i>	R	S	S	—	R	—	—	—	R	R
	R	S	S	—	S	—	—	—	S	S
	R	S	S	—	S	—	—	—	R	S
<i>Klebsiella pneumoniae</i>	R	R	S	R	R	—	—	—	S	S
<i>Pseudomonas aeruginosa</i>	—	—	—	R	—	S	—	R	—	R
<i>Streptococcus pyogenes</i>	R	S	S	—	S	—	—	—	S	S
<i>Citrobacter freundii</i>	—	—	—	S	—	S	R	R	—	S
	—	—	—	S	—	S	S	S	—	S

	Netilmicin	Co-trimoxazole	Ciprofloxacin	Vancomycin/ teicoplanin	Piperacillin+ tazobactam	Ertapenem	Imipenem	Meropenem
<i>Staphylococcus aureus</i>	S	R	R	S	—	—	—	—
	S	S	S	S	—	—	—	—
	S	R	S	S	—	—	—	—
	S	R	R	S	—	—	—	—
<i>Staphylococcus epidermidis</i>	R	R	R	S	—	—	—	—
	S	S	S	S	—	—	—	—
	S	R	R	S	—	—	—	—
<i>Klebsiella pneumoniae</i>	S	R	R	—	—	—	—	—
<i>Pseudomonas aeruginosa</i>	R	—	S	—	R	—	R	R
<i>Streptococcus pyogenes</i>	S	R	S	S	—	—	—	—
<i>Citrobacter freundii</i>	S	—	S	—	S	S	R	R
	S	—	S	—	S	S	S	S

Table 5 Comparison of clinical features of DE/PDM

	Haagensen [16]	Cromar et al. [17]	Walker et al. [18]	Thomas et al. [19]	Dixon et al. [14]	Present study
No. of patients	110	24	34	78	108	41
Mean age (years)	54	40	—	45	46	41
Nipple retraction (%)	25	42	8	32	53	56
Mass (%)	90	100	8	32	43	15
Subareolar sepsis (%)	18	—	9	9	22	49

periductal mastitis and duct ectasia are just the two stages of the same disease process [21]. However, this statement has been contradicted by the finding of an entity of patients with only pure DE without the evidence of preceding periductal mastitis (sterile cultures).

The most commonly isolated organisms were *S. aureus* (four out of 21), *S. epidermidis* (three out of 21), and *C. freundii* (two out of 21). Similar predominance of *Staphylococcus* sp. in aerobic cultures was observed by Walker et al. [18]. In their study, the common aerobes isolated from the culture of the fluid aspirated from the retroareolar regions with PDM were *S. epidermidis*, *S. aureus*, and *Streptococci* sp. This observation suggests that infection could be due to poor hygiene of the nipple and areola. The possible explanation could be that Poor hygiene leads to colonization of the skin of nipple and areola in turn leading to colonization of the ductal tissue. In contrast to the Walker et al. study [22], we did not observe much of a mixed flora grown in the cultures of the ductal tissue.

The studies done by Beigelman and Rantz [23], Strum et al. [24], Scholefield et al. [25], and Leach et al. [26] showed significant predominance of anaerobes. In contrast, the present study has not shown any positive growth in anaerobic culture. This could be explained by delayed inoculation of the tissue in the RCM broth in some cases and the use of preoperative metronidazole for 3 weeks in those patients who had pus discharge preoperatively to which the anaerobes are susceptible. It could also reflect the difference in the infective pattern among the Western and Indian patients. Similar to our study, predominance of aerobic gram-positive (staphylococcal) infection in periductal mastitis has also been [8, 12, 27] documented in a Spanish article by Paredes López A and Moreno G [27].

The organisms that were grown in the ductal tissue of women with DE/PDM were shown to have resistance to the commonly used antibiotics namely penicillin, co-trimoxazole, and erythromycin. This necessitates preoperative culture sensitivity of the nipple discharge and use of preoperative antibiotics according to the sensitivity pattern for the eradication of infection. As the resistance to erythromycin was found to be high (~60 %), we started using oral linezolid or ciprofloxacin as empirical treatment instead of erythromycin till the culture and sensitivity report became available. If necessary, based on the culture and sensitivity report, an appropriate full course of

sensitive antibiotic was administered. It is recommended that a prolonged course of antibiotics to cover both gram-positive cocci and anaerobes (2–3 weeks) is given for patients who present with recurrent subareolar sepsis [28] before taking them for surgery, as they tend to recur very often and fail to resolve with the short course of empirical antibiotics alone [8, 12, 29].

Nipple discharge, retraction, and subareolar abscess are also few of the versatile manifestations of tuberculosis of breast. With the incidence of breast tuberculosis being up to 3 % of breast diseases in our country [30], presence of recurrent nipple discharge, and subareolar sepsis despite of complete course of antibiotics and satisfactory duct excision should raise the suspicion of tuberculosis. This is especially so when the final histopathology is not characteristic of duct ectasia/periductal mastitis and suggestive of granulomatous/plasma cell mastitis (which are the differential diagnoses of tuberculosis on histopathology) [31]. In a Chinese study, they have even documented response to antimycobacterial drug therapy in patients of periductal mastitis with fistula having related this condition with possible infection by non-tuberculous mycobacteria [32].

Conclusions

There is no statistically significant difference in the age distribution among women with DE, DE+PDM, and PDM, and smoking is not associated with DE and PDM of Indian patients in contrast to the Western literature evidence. In patients presenting with non-sanguineous nipple discharge, DE is commoner than PDM alone or PDM+DE. Infective etiology was present in nearly 46 % of the patients in the study population more so in the periductal mastitis cases. The most commonly isolated pathogens were *S. aureus* and *S. epidermidis*, unlike in Western population where nearly 50 % of the growths isolated were anaerobes. This finding suggests that proper local hygiene could prevent subareolar sepsis among Indian women. Since the isolated organisms were resistant to the routinely used antibiotics (especially erythromycin in our practice) in high proportion of cases, culture and sensitivity should be done in all possible cases for appropriately treating subareolar sepsis. In India, as

tuberculosis is rampant, tuberculous mastitis should be considered as a differential diagnosis if the disease is not manageable by conventional measures.

References

1. Seltzer MH (2004) Breast complaints, biopsies, and cancer correlated with age in 10,000 consecutive new surgical referrals. *Breast J* 10(2): 111–117
2. Devitt JE (1985) Management of nipple discharge by clinical findings. *Am J Surg* 149(6):789–792
3. Leis HP Jr (1989) Management of nipple discharge. *World J Surg* 13(6):736–742
4. Dolan RT, Butler JS, Kell MR, Gorey TF, Stokes MA (2010) Nipple discharge and the efficacy of duct cytology in evaluating breast cancer risk. *Surgeon* 8(5):252–258
5. Guray M, Sahin AA (2006) Benign breast diseases: classification, diagnosis, and management. *Oncologist* 11(5):435–449
6. Kim BS, Lee JH, Kim WJ, Kim DC, Shin S, Kwon HJ et al (2013) Periductal mastitis mimicking breast cancer in a male breast. *Clin Imaging* 37(3):574–576
7. Richards T, Hunt A, Courtney S, Umeh H (2007) Nipple discharge: a sign of breast cancer? *Ann R Coll Surg Engl* 89(2):124–126
8. Lannin DR (2004) Twenty-two year experience with recurring subareolar abscess and lactiferous duct fistula treated by a single breast surgeon. *Am J Surg* 188(4):407–410
9. Hanavadi S, Pereira G, Mansel RE (2005) How mammary fistulas should be managed. *Breast J* 11(4):254–256
10. Oltean HN, Soliman AS, Omar OS, Youssef TF, Karkouri M, Abdel-Aziz A et al. (2013) Risk factors for chronic mastitis in Morocco and Egypt. *Int J Inflam [Internet]*. 2013 [cited 2014 Mar 19]. Available from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3847959/>
11. Furlong AJ, al-Nakib L, Knox WF, Parry A, Bundred NJ (1994) Periductal inflammation and cigarette smoke. *J Am Coll Surg* 179(4): 417–420
12. Ammari FF, Yaghan RJ, Omari A-KH (2002) Periductal mastitis. Clinical characteristics and outcome. *Saudi Med J* 23(7):819–822
13. Dixon JM (1998) Periductal mastitis and duct ectasia: an update. *Breast* 7(3):128–130
14. Dixon JM, Anderson TJ, Lumsden AB, Elton RA, Roberts MM, Forrest APM (1983) Mammary duct ectasia. *Br J Surg* 70(10):601–603
15. Frantz VK, Pickren JW, Melcher GW, Auchincloss H (1951) Incidence of chronic cystic disease in so-called “normal breasts”. A study based on 225 postmortem examinations. *Cancer* 4(4):762–783
16. Haagensen CD (1986) Diseases of the breast, 3rd edn. Saunders, Philadelphia, 1050 p
17. Cromar CDL, Docherty MB (1941) Plasma cell mastitis. *Proc Staff Meet Mayo Clinic* 16:775–782
18. Walker JC, Sandison AT (1964) Mammary-duct ectasia. A clinical study. *Br J Surg* 51(5):350–355
19. Thomas WG, Williamson RCN, Davies JD, Webb AJ (1982) The clinical syndrome of mammary duct ectasia. *Br J Surg* 69(7):423–425
20. Bundred NJ, Dixon JM, Lumsden AB, Radford D, Hood J, Miles RS et al (1985) Are the lesions of duct ectasia sterile? *Br J Surg* 72(10): 844–845
21. Diaconu C, Socolov D, Dragomir C, Costachescu G, Florea N (1996) 91 P - Periductal mastitis and mammary duct ectasia - two stages of the same disease? *Eur J Cancer* 32(Supplement 1):S19
22. Walker AP, Edmiston CE Jr, Krepel CJ, Condon RE (1988) A prospective study of the microflora of nonpuerperal breast abscess. *Arch Surg* 123(7):908–911
23. Beigelman PM, Rantz LA (1949) Clinical significance of bacteroides. *Arch Int Med (Chicago)* 84(4):605–631
24. Sturm AW (1989) Mobiluncus species and other anaerobic bacteria in non-puerperal breast abscesses. *Eur J Clin Microbiol Infect Dis* 8(9): 789–792
25. Scholefield JH, Duncan JL, Rogers K (1987) Review of a hospital experience of breast abscesses. *Br J Surg* 74(6):469–470
26. Leach RD, Eykyn SJ, Phillips I (1981) Vaginal manipulation and anaerobic breast abscesses. *Br Med J (Clin Res Ed)* 282(6264):610–611
27. Paredes López A, Moreno G (1995) Non-puerperal mastitis. Clinical study of 30 patients. *Ginecol Obstet Mex* 63:226–230
28. Mansel RE, Webster DJT, Sweetland HM (2009) Hughes, Mansel and Webster's Benign disorders and diseases of the breast, 3rd edn. Saunders, China, 163–192 p
29. Komenaka IK, Pennington RE Jr, Bowling MW, Clare SE, Goulet RJ Jr (2006) A technique to prevent recurrence of lactiferous duct fistula. *J Am Coll Surg* 203(2):253–256
30. Tewari M, Shukla HS (2005) Breast tuberculosis: diagnosis, clinical features & management. *Indian J Med Res* 122(2):103–110
31. Alagaratnam TT, Ong GB (1980) Tuberculosis of the breast. *Br J Surg* 67(2):125–126
32. Yu H, Wang Q, Yang J, Lian Z, Zhang A, Li W et al (2012) Antimycobacteria drugs therapy for periductal mastitis with fistula. *Zhonghua Wai Ke Za Zhi* 50(11):971–974