Journal of Inflammation Research

Dovepress
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ORIGINAL RESEARCH

Periductal Mastitis, a Disease with Distinct Clinicopathological Features from Granulomatous Lobular Mastitis

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Purpose: Periductal mastitis (PDM) is a chronic inflammatory lesion of the breast with an unknown etiology, and it is difficult for clinicians to differentiate it from granulomatous lobular mastitis (GLM), although they have different treatment strategies and prognosis. This study aimed to investigate the differences in their clinicopathologic features to inform treatment strategies.

Patients and Methods: Between 2011 and 2020, 121 patients diagnosed with PDM and 57 patients with GLM were retrospective analysis. Patient data were extracted on demographics, clinical presentation, pathologic characteristics, treatments and clinical response. Histopathological evaluations were performed on core needle biopsy specimens. Immunohistochemical stains using antibodies against CD3, CD4, CD8, CD20, and CD138 was performed to define immune cell infiltration.

Results: PDM patients had a higher median age compared to GLM patients (38 vs 32, p<0.001). PDM was primarily located in the areolar area, while GLM predominantly affected the peripheral quadrant of the breast (56.20% vs 75.44%, p<0.001). Histopathologically, more ductal dilatation (90.08% vs 3.51%, p<0.001), ductal wall thickening (47.93% vs 1.75%, p<0.001), and ductal rupture (44.63% vs 5.26%, p<0.001) were observed in PDM. GLM presented with significantly more granuloma (94.74% vs 10.74%, p<0.001), microabscess (68.42% vs 28.93%, p<0.001), and lipid vacuole (40.35% vs 8.26%, p<0.001) formation than PDM. Immunohistochemical analysis revealed a significant presence of CD20+ B lymphocytes in PDM and a higher prevalence of CD8+ T lymphocytes in GLM, indicating differing immune responses. Treatment outcomes varied, with PDM patients responding well to surgery and anti-mycobacterial therapy, while GLM patients showed favorable responses to steroid therapy.

Conclusion: PDM is a specific entity with a similar clinical presentation but distinct histopathological features and immune profiles to GLM. Further research is needed to elucidate the pathogenesis and optimize therapeutic approaches for these breast inflammatory conditions.

Keywords: etiology, granulomatous lobular mastitis, immunology, pathology, periductal mastitis

Introduction

Periductal mastitis (PDM) is a benign, inflammatory disease that usually occurs in the major ducts of the breast and accounts for 1–2% of all breast diseases.[1] The main clinical manifestations include non-cyclical mastalgia, nipple retraction, nipple discharge, subareolar breast lumps, and even periareolar abscesses or breast fistulas.[2] The pathogenesis and etiology of PDM remain unclear, and possible mechanisms have been suggested, including hormonal effects, mammary duct obstruction, smoking and bacterial infection.

Granulomatous lobular mastitis (GLM), which was first described by Kessler and Wolloch in 1972, used to be an uncommon benign inflammatory disease mainly affecting women of childbearing age with recent lactation and pregnancy history.[3] However, the incidence of GLM has recently increased sharply in Mediterranean countries and Asian, such as

Received: 28 March 2024
Accepted: 30 May 2024
Published: 14 June 2024

China, Iran, and Turkey.[4,5] The etiology of GLM is not well established and is often thought to be idiopathic; however, several mechanisms, such as infection,[6–8] autoimmunity,[9] and hypersensitivity reactions,[10] have been proposed.

PDM and GLM present similar clinical presentations, commonly including breast pain, redness, nipple retraction, breast lumps, draining sinus, and ulcers.[11] Imaging features for both conditions are nonspecific, necessitating histopathological diagnosis. Core needle or incisional/excisional biopsy procedures are commonly used to obtain tissue samples, however PDM and GLM exhibit significant histopathological similarities, primarily characterized by chronic inflammation, fibrosis, and epithelial changes. Both conditions feature a dense infiltrate of lymphocytes, plasma cells, and macrophages, along with varying degrees of fibrosis. Additionally, both diseases can lead to abscess formation and contain multinucleated giant cells, either as part of the granulomatous process or in response to chronic inflammation. Going et al[12] admit an overlap between PDM and GLM, but still believe they are two different disorders, emphasizing the necessity of differential diagnosis "on clinical and histological grounds". To date, what constitutes these grounds is undefined. The aim of the present study was to compare and differentiate the clinicopathological features of PDM and GLM, elucidating their distinct characteristics to provide references for their pathogenesis and treatment options.

Methods

Clinical Information

This study was approved by the Ethics Committee of the Second Hospital of Shandong University and was conducted per the Declaration of Helsinki. Written informed consent was obtained from all participants. Data were collected retrospectively from electronic medical records between January 2011 and October 2020. PDM was diagnosed based on clinical findings and histological evidence of duct ectasia and periductal inflammatory changes.[13,14] The diagnosis of GLM was characterized by the presence of non-caseating granulomas centered on lobular, excluding out conditions such as tuberculosis, sarcoidosis, and Wegener's granulomatosis.[14,15] Electronic medical records were reviewed for pertinent clinical information, including patient age, body mass index (BMI), age of menarche, pregnancy and abortion history, relevant past medical history, clinical manifestations, treatment, and outcome. Follow-up information was obtained through telephone consultations and outpatient records.

Histopathological Evaluation

All included cases were diagnosed using a core needle biopsy. Two pathologists reviewed all specimens to confirm the diagnosis and completed a self-designed pathological feature description questionnaire. The questionnaire included the extent of the lesion, lesion location, duct dilation, duct wall thickening, periductal fibrosis, ductal rupture, squamous metaplasia, epithelial proliferative, intraductal secretion, microabscess, lipid vacuoles, fat necrosis, cholesterol crystals, vasculitis, and granuloma formation. The EnVision two-step method was used for the immunohistochemical analysis. Differential infiltrates of $CD3^+$ (T lymphocytes), $CD4^+$ (helper T lymphocytes), $CD8^+$ (cytotoxic T lymphocytes), $CD20^+$ (B lymphocytes), and $CD138^+$ (plasma cells) cells were compared between the two groups.

Statistical Methods

Demographics, disease variables, and treatment variables were analyzed using descriptive statistics. Continuous variables were expressed as mean ± standard deviation or mean (range) using the Kruskal–Wallis test, and categorical variables were expressed as frequencies (%) using the Pearson $\chi 2$ test. We obtained a copyright license for IBM SPSS Statistics ver. 24.0 (IBM Co., Armonk, NY, USA) and used it for statistical analysis; $P<0.05$ was considered statistically significant.

Results

Demographic Characteristics

In this study, a total of 178 female patients were included, of whom 121 were clinically diagnosed as PDM and 57 were diagnosed as GLM. Demographic information is shown in Table 1. The median age of the 121 patients with PDM was 38 years (range: 15–81 years). Of these, 49 patients were ≤34 years of age and 110 had a history of both pregnancy and

Table 1 The Demographic Characteristics of PDM and GLM

	PDM (n=121)	GLM (n=57)	P value
Age			<0.001**
Median (range)	38 (15–81)	32 (18–49)	
BMI			0.96
<18.5(%)	5 (4.13)	2 (3.51)	
18.5–23.9(%)	69 (57.02)	37 (64.91)	
24.0–27.9(%)	40 (33.06)	14 (24.56)	
>28(%)	7 (5.79)	4 (7.02)	
Pregnancy	110 (90.91)	55 (96.49)	0.23
Number of births			0.70
0(%)	11 (9.09)	2 (3.51)	
1(%)	59 (48.76)	32 (56.14)	
≥2(%)	51 (42.15)	23 (40.35)	
Abortion history	50 (41.32)	17 (29.82)	0.16
Lactation	108 (89.26)	52 (91.23)	0.45
Time postpartum (month)			<0.001**
Mean ± SD	125.1±119.7	44.4±29.4	
Mastitis (%)	18 (14.88)	6 (10.53)	0.48
Smoking (%)	1 (0.83)	1 (1.75)	0.54
Passive smoking (%)	45 (37.19)	15 (26.32)	0.23
Hyperprolactinemia (%)	1 (0.83)	1 (1.75)	0.54
Pituitary microadenoma (%)	2 (1.65)	1 (1.75)	1.00

Notes: **P<0.05 indicates a statistically significant difference.
Abbreviations: PDM, Periductal mastitis; GLM, Granulomatous lobular mastitis; BMI, body mass index; SD, standard deviation.

parity. Among them, 50 patients (41.32%) had a history of abortion, 108 (89.27%) had a history of lactation, and 18 (14.88%) had a history of mastitis during lactation, and the mean time between onset and the last birth was 125.1±119.7 months. Of the 57 patients diagnosed with GLM, the median age was 32 years (range: 18–49 years), 55 (96.49%) had a history of childbirth, 17 (29.82%) had a history of abortion, and 52 (91.23%) had a history of lactation; of these, 6 (10.53%) had a history of lactational mastitis, and the mean time from onset to last childbirth was 44.4±29.4 months. The medical histories of 178 patients were thoroughly reviewed. Two patients had a history of hyperprolactinemia and three had a history of pituitary microadenomas. The median age of PDM patients was statistically higher than that of GLM patients (38 vs 32, p<0.001). In addition, more patients in the GLM group had a delivery within 4 years. (64.91% vs 34.71%, p<0.001). However, regarding BMI, age at menarche, pregnancy, number of births, mastitis, smoking, passive smoking, and oral contraceptive use, no statistically significant difference was found between the groups.

Local Manifestations

Detailed local manifestations are presented in Table 2. Most patients were unilateral, although 5 cases of PDM and 2 cases of GLM had bilateral onset, and there was no tendency for one side to be affected more often than the other. PDM occurred mostly in the areolar area, while lesions with GLM were in the peripheral quadrant of the breast, with a statistically significant difference (56.20% vs 75.44%, p<0.001). The majority of the 101 patients with PDM (83.47%) and the 54 patients with GLM (94.74%) presented with lumps. The mean size of GLM was often greater than that of PDM (4.83 cm vs 2.91 cm, p<0.001), and the PDM patients were more likely to experience skin redness and swelling. Nipple discharge occurred in 16 patients in the PDM group and 5 in the GLM group. Abscesses developed in 15 patients with PDM and 6 patients with GLM, and a total of 10 patients developed skin ulcers and sinus subsequently. In both groups, ipsilateral axillary lymph node enlargement was occasionally observed. The main clinical manifestations of PDM and GLM are shown in Figure 1.

Table 2 The Clinical Manifestations of PDM and GLM

	PDM (n=121)	GLM (n=57)	P value
Side (n, %)			0.51
Right	53 (43.80)	31 (54.39)	
Left	63 (52.07)	24 (42.11)	
Bilateral	5 (4.13)	2 (3.51)	
Location (n, %)			<0.001**
Around areola	68 (56.20)	11 (19.30)	
Peripheral part	49 (40.50)	43 (75.44)	
Overlap	4 (3.31)	3 (5.26)	
Mass (n, %)	101 (83.47)	54 (94.74)	0.05
Diameter of mass (cm)			
Mean (range)	2.91 (0–15)	4.83 (1–11)	<0.001**
Pain (n, %)	79 (65.29)	44 (77.19)	0.12
Redness and swelling (n, %)	91 (75.21)	20 (35.09)	<0.001**
Nipple retraction (n, %)	17 (14.05)	12 (21.05)	0.28
Nipple discharge (n, %)	16 (13.22)	5 (8.77)	0.46
Abscess (n, %)	15 (12.40)	6 (10.53)	0.80
Sinus (n, %)	4 (3.31)	6 (10.53)	0.08
Axillary lymph node enlargement (n, %)	44 (36.36)	16 (28.07)	0.31

Notes: **P<0.05 indicates a statistically significant difference.
Abbreviations: PDM, Periductal mastitis; GLM, Granulomatous lobular mastitis.

Histopathological Data

Histopathologically, PDM showed mainly ductal dilatation, ductal luminal secretion formation, and periductal inflammatory reaction with fibrosis. GLM was characterized by granuloma formation centered on lobules with or without microabscesses, surrounded by inflammatory cell infiltration. Pathological features typical of PDM and GLM are shown in Figure 2. PDM lesions were mainly periductal (n = 102, 84.30%), while GLM lesions were centered on the lobules (n = 47, 82.46%), with a statistically significant difference (p <0.001). Ductal dilatation (90.08% vs 3.51%, p<0.001), ductal rupture (44.63% vs 5.26%, p<0.001), periductal fibrosis (45.45% vs 0%, p<0.001), and wall thickening (47.93% vs 1.75%, p<0.001) were more frequent in PDM than in GLM. While Both PDM and GLM formed granulomas, GLM had significantly more granulomas than PDM (94.74% vs 10.74%, p<0.001), and granulomas were mainly composed of epithelioid cells, multinucleated giant cells, and fibroblasts. Microabscesses (68.42% vs 28.93%, p<0.001) and lipid vacuoles (40.35% vs 8.26%, p<0.001) were more common in GLM. No statistical differences were found between PDM and GLM in terms of squamous epithelial chemosis, epithelial cell proliferation, luminal necrotic material, fat necrosis, cholesterol crystals, and small vasculitis

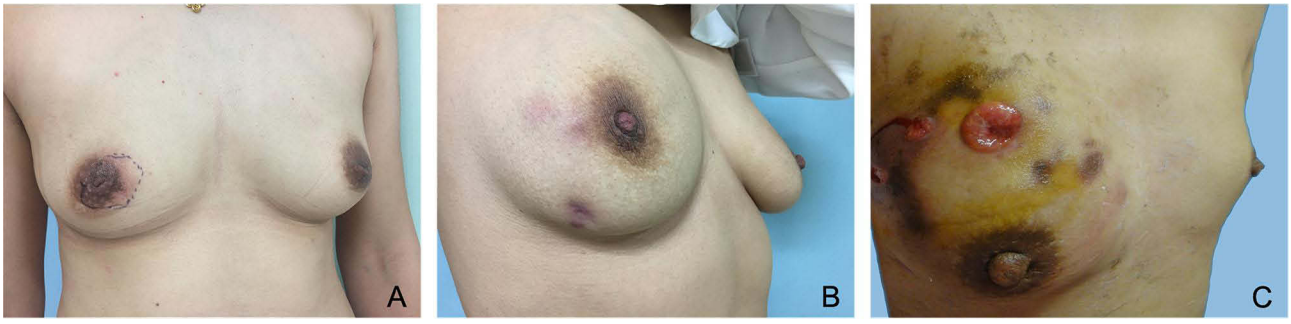

Figure 1 Typical clinical manifestations of PDM and GLM. (**A**) Patients with periductal mastitis present with a periareolar mass with surface skin redness and nipple retraction. (**B**) Patients with granulomatous lobular mastitis present with a peripheral breast mass with skin redness and nipple retraction and skin ulceration. (**C**) Patients with severe periductal mastitis suffer from extensive abscess formation, skin ulceration, and fistula formation.
Abbreviations: PDM, Periductal mastitis; GLM, Granulomatous lobular mastitis.

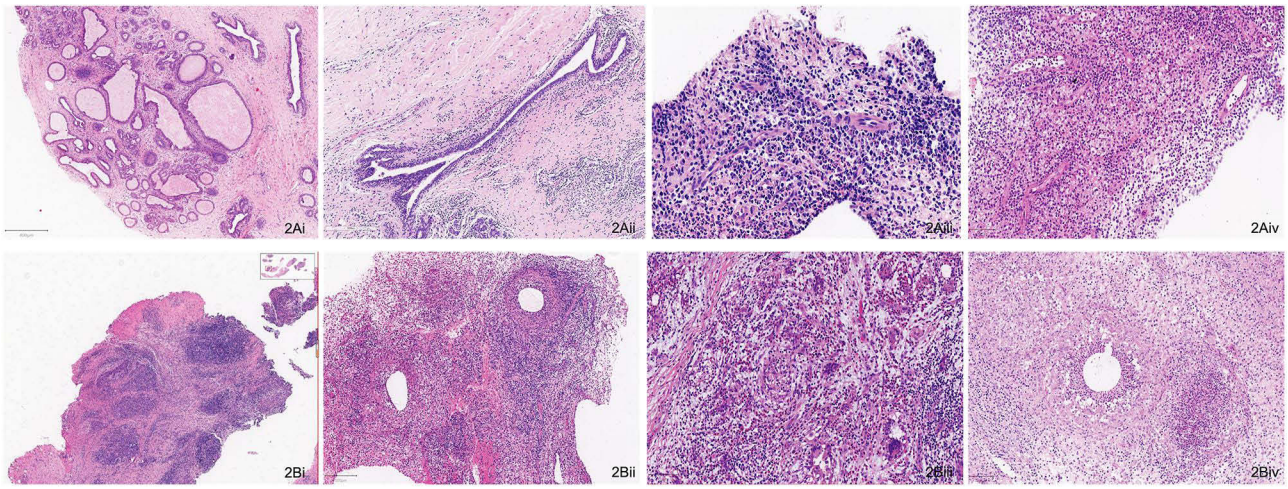

Figure 2 Pathological features of PDM and GLM. 2Ai: Disruption of the lobules of the breast with cystic dilatation of the ducts, which contain a large amount of secretions; 2Aii: Hyperplasia of fibrous tissue around dilated breast ducts with marked lymphocytic infiltration; 2Aiii: Large infiltration of foam cells in the interstitium of the breast; 2Aiv: Massive plasma cell infiltration around the dilated duct. 2Bi: Multifocal non-caseating granulomas centered on lobular units of the breast; 2Bii: The fusion of lesions has destroyed the lobular structure of the breast and microabscesses can be observed. 2Biii: Infiltration of eosinophils in the lobules of the breast; 2Biv: Lipid-dissolving vacuoles rimmed by neutrophils surrounded by lymphocytes, epithelioid cells, plasma cells.
Abbreviations: PDM, Periductal mastitis; GLM, Granulomatous lobular mastitis.

formation (Table 3). The types of inflammatory cells infiltrating PDM and GLM lesions were also different. The number of foam cells infiltrating the lumen and outside the lumen was significantly higher in the PDM than in the GLM, where the ductal contents leaked and tissue cells phagocytosed the necrotic material to form foam cells.

Table 3 The Pathological Features Between PDM and GLM

	PDM (n=121)	GLM (n=57)	P value
Extent of lesions (n, %)			<0.001**
Centered on lobules	4 (3.31)	47 (82.46)	
Periductal	102 (84.30)	2 (3.51)	
Undefined	15 (12.40)	8 (14.04)	
Histomorphology (n, %)			
Duct dilation	109 (90.08)	2 (3.51)	<0.001**
Duct wall thickening	58 (47.93)	1 (1.75)	<0.001**
Periductal fibrosis	55 (45.45)	0 (0.0)	<0.001**
Ductal rupture	54 (44.63)	3 (5.26)	<0.001**
Squamous metaplasia	3 (2.48)	0 (0.0)	0.55
Epithelial proliferative	9 (7.44)	0 (0.0)	0.06
Intraductal secretion	50 (41.32)	1 (1.75)	<0.001**
Microabscess	35 (28.93)	39 (68.42)	<0.001**
Lipid vacuoles	10 (8.26)	23 (40.35)	<0.001**
Fat necrosis	7 (5.79)	2 (3.51)	0.72
Cholesterol crystals	8 (6.61)	0 (0.0)	0.06
Vasculitis	4 (3.31)	2 (3.51)	1.00
Granulomas (n, %)	13 (10.74)	54 (94.74)	<0.001**
Epithelioid cell	13 (10.74)	52 (91.22)	<0.001**
Multinucleated giant cells	13 (10.74)	50 (87.72)	<0.001**
Foreign body giant cell	9 (7.44)	1 (1.75)	0.17
Foreign body	2 (1.65)	0 (0.0)	1
Fibroblasts	8 (6.61)	37 (64.91)	<0.001**

Notes: **P<0.05 indicates a statistically significant difference.
Abbreviations: PDM, Periductal mastitis; GLM, Granulomatous lobular mastitis.

The Proportion of Lymphocyte Subsets Distribution

Lymphocyte subsets were tested in 51 cases of PDM and GLM. The results showed that both of them were mainly infiltrated by $CD20^+$ B lymphocytes. Figure 3 shows that the mean proportion of $CD20^+$ cells were higher in PDM than in GLM (45.4% vs 41.6%, p = 0.015), and the mean proportion of $CD3^+$ cells was 42.0% in PDM and 40.0% in GLM, with no significant difference. By contrast, the mean percentage of $CD138^+$ cells were less in PDM than in GLM (12.3% vs 18.5%, p<0.001). Among T lymphocytes, both PDM and GLM were dominated by $CD4^+$ T lymphocyte infiltration. The mean percentage of $CD4^+$ cells was higher in PDM patients than in GLM patients (67.0% vs 64.6%), while the mean percentage of $CD8^+$ cells was lower in PDM patients than in GLM patients (33.0% vs 35.4%), though these differences were not statistically significant (Figure 4).

Treatment and Outcome

Various treatment strategies were used in the study population. Of the 121 patients with PDM, 52 received triple anti-mycobacterial drugs with isoniazid (300 mg/d), rifampicin (450 mg/d), and ethambutol (15 mg/kg/d). Among these, 45 patients achieved remission, while 7 experienced recurrences. Fifty-five were treated surgically, 49 were cured and one recurrence was observed; nine patients received surgery in combination with anti-mycobacterial therapy and seven achieving remission. Extended excision was the main surgical procedure, and incision and drainage were performed in six patients. The other five patients were given expectant treatment and remitted completely. Of the 57 patients with

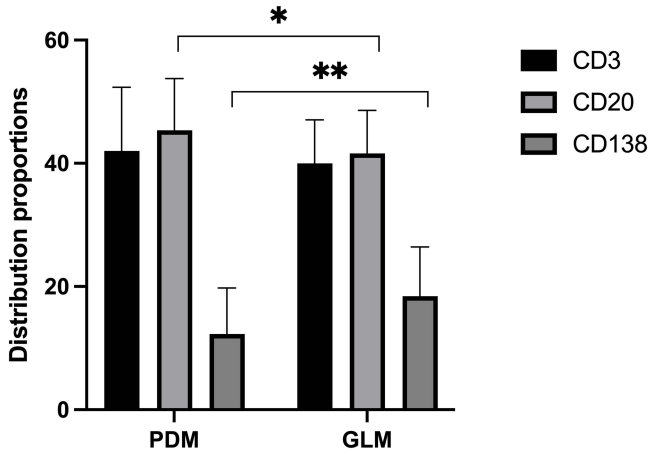

Figure 3 Differences in the distribution proportions of CD3, CD20, and CD138 positive cells in PDM and GLM. *p=0.015, **p<0.001.
Abbreviations: PDM, Periductal mastitis; GLM, Granulomatous lobular mastitis.

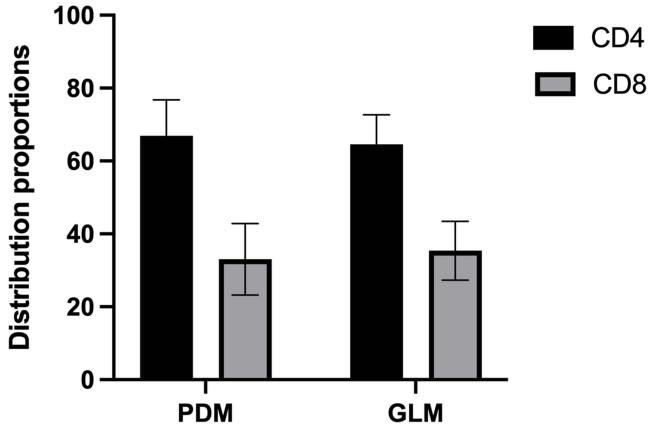

Figure 4 The difference in the distribution proportions of CD4 and CD8 positive cells in PDM and GLM.
Abbreviations: PDM, Periductal mastitis; GLM, Granulomatous lobular mastitis.

GLM, 33 were administered steroid therapy, of which 28 were in remission, but 4 relapsed during follow-up. Ten patients were cured with antimycobacterial agents, with one relapse. Seven patients underwent extended resection, and one patient relapsed two months after surgery. The other seven cases received expectant treatment, with six achieving remission. All patients with abscesses underwent ultrasound-guided puncture drainage or abscess incision, and broad-spectrum antibiotics were administered in the acute phase of inflammation.

Discussion

PDM is characterized by frequent periductal inflammation associated with duct ectasia. The pathogenesis of this disorder remains unclear, though duct ectasia and bacterial presence in nipple secretions are major predisposing factors.[16] Recent studies have also confirmed smoking as a significant etiology, with heavy smokers showing increased incidence of anaerobic bacteria in abscesses and fistula formation.[17]

GLM is a chronic, noninfectious inflammatory disease typically occurring in parous females, accounts for 1.8% of benign breast diseases.[18] The etiology of GLM is poorly understood, but autoimmune reactions, bacterial infections, and hormonal disorders are thought to trigger this disease.[6–10] Clinically, GLM and PDM present similarly with breast pain, redness, nipple retraction, and lumps,[4] but diagnosis often requires histopathological confirmation due to nonspecific imaging features. Despite some studies have demonstrated the pathological characteristics of GLM, current studies have paid limited attention to PDM, and few systematic studies on their differences have been conducted. The aim of this study was to further investigate the differences in clinicopathologic features between PDM and GLM.

Our findings indicate PDM patients were generally older than those with GLM. The median age of PDM was 38 years (range: 15–81), while GLM typically affects women aged 30–45 years, with a median age of 32 in our study. A strong association between GLM and recent pregnancies was also confirmed,[15] with the time from last delivery to disease onset ranging from 3 to 157 months (mean: 44.4 months) for GLM, and 2 to 604 months (mean: 125.1 months) for PDM, suggesting that pregnancy, labor and delivery, and breastfeeding may be causative factors for GLM. Previous studies have supported an association between the onset of PDM and GLM with factors such as smoking, history of oral contraceptive use, hyperprolactinemia, and pituitary microadenoma,[4,13] while no difference was observed in this study because of the limited number of patients.

Local manifestations were also evaluated. Initial diagnoses of PDM included nipple discharge, breast lumps, and later infection, which may cause breast pain or abscess. According to Gurleyik et al, the main clinical manifestations of GLM are large and irregular painful masses.[19] In our study, both PDM and GLM presented primarily with breast masses, but PDM lesions were mostly in the areolar area, while GLM lesions are predominantly in the peripheral breast quadrant (56.20% vs 75.44%, $p<0.001$). GLM lesions were significantly larger than that of PDM (4.83 cm vs 2.91 cm, $p<0.001$). However, PDM patients exhibited more acute-phase symptoms, including skin redness and swelling (75.21% vs 35.09%, $p<0.001$). GLM was associated with nipple retraction (12 cases, 21.05%) and nipple discharge (5 cases, 8.77%). While the clinical presentations of PDM and GLM are similar, key differences remain.

In the present study, significant histopathologic differences were observed between PDM and GLM. The most prominent histological feature of GLM was non-caseating granulomas consisting of epithelioid histiocytes and Langhans' giant cells, accompanied by infiltration of plasma cells, lymphocytes, and minimal amounts of eosinophils within and around lobules. GLM lesions are centered on the lobules, while PDM lesions are primarily periductal, characterized by ductal dilatation accompanied by an inflammatory response in the ductal wall or peripheral tissues. Foam cells are the most commonly infiltrated inflammatory cells surrounding the breast duct. Ductal dilatation, increased secretion in the lumen, ductal rupture, and thickening of the duct wall were more frequent in PDM than in GLM ($p<0.001$). Granulomas can be observed in PDM, but microabscesses are rare. Most GLM granulomatous structures are accompanied by microabscesses. More lipid vacuoles were observed in the GLM than in the PDM, surrounded by a large number of neutrophils in the periphery, which may be interspersed with lymphocyte aggregates, infiltrating epithelioid cells, multinucleated giant cells, and fibroblasts outside of the neutrophil aggregation zone.

Previous studies have demonstrated that both PDM and GLM are related to immune reactions.[16,20] Our study revealed that both humoral and cellular immunity are involved in the pathogenesis of PDM and GLM, with B cell-mediated humoral immune responses dominating. Th2 and Tfh cells can promote B cell differentiation, and more B cell infiltration was observed in PDM than in GLM, suggesting that Th2- and Tfh-mediated immunity plays a key role in PDM. In contrast, GLM showed more granuloma structures with multinucleated giant cells and neutrophil infiltration, and greater

CD8+ T lymphocyte infiltration, suggesting a dominant role for Th1-mediated cellular immunity. Future studies should investigate the role of various cytokines in these immune processes.

Currently, there is no standard treatment for PDM. Surgical treatment is still the main treatment option, and studies revealed that surgery was effective in 79–91.7% of cases, with a recurrence rate of 1–50%.[21,22] In this study, 55 of 121 PDM patients underwent surgical treatment, 49 were cured, and 1 had recurrence, with a recurrence rate of 2.04%. Yu et al pointed out that anti-mycobacterial therapy can also be used for patients with recurrent sinus formation and pathologically confirmed PDM.[23] Fifty-two patients with PDM in the current study were treated with anti-mycobacterial therapy, of whom 45 achieved remission and 7 relapsed.

Steroids used to treat GLM were first proposed by Dehertogh et al in 1980, and have previously shown efficacy in GLM.[24] The full recovery rate with steroid treatment is 42–93.5%, the partial recovery rate is 6.4–58%, and the non-response is 6.5%.[21,25,26] In our study, 33 patients with GLM were administered steroid therapy, of which 28 were in remission, the recovery rate was 84.84%, and 4 patients relapsed during follow-up with a recurrence rate of 14.29%. Several studies have shown that anti-mycobacterial treatment is also effective for GLM.[27,28] Some studies found that GLM resolves spontaneously,[29,30] regardless of medical intervention, over a period of approximately 5 months or up to 20 months; therefore, intensive observation, reassurance, and patient education may also be an alternative approach to treating GLM. Six cases of GLM and five cases of PDM in our study were cured by expectant therapy, and the true influence of autoimmunity on GLM and PDM should be determined in future studies.

This study had several limitations. First, the retrospective design inherently limits the study due to potential biases in data collection and documentation. Second, although the study included 178 patients, the sample sizes for each group are relatively small, potentially limiting the statistical power to detect significant differences. Additionally, data were collected from a single center, which may limit the generalizability of the findings. Therefore, a randomized controlled study is needed in the future for broader applicability.

Conclusion

Our study demonstrates that PDM and GLM, while clinically similar, have distinct histopathological features and immune profiles that should be carefully considered by clinicians for accurate diagnosis and effective treatment. Recognizing these differences is crucial. Future research should focus on better understanding the pathogenesis and therapeutic strategies for these specific categories of breast inflammation.

Consent for Publication

Written informed consent was obtained from the patients for publication of this study and any accompanying images.

Acknowledgment

We thank Xiaohui Yan for reviewing the report contextually and grammatically, and the pathology department of Second Hospital of Shandong University for their support in the database search.

Funding

This study was supported by The Shandong Provincial Nature Science Foundation, China (Grant No. ZR2020QH254).

Disclosure

The authors report no conflicts of interest in this work.

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