

The Role of *Staphylococcus aureus* in Mastitis: A Multidisciplinary Working Group Experience

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ABSTRACT

Background: Breastfeeding women are at risk of developing mastitis during the lactation period. *Staphylococcus aureus* has emerged as the community-acquired pathogen responsible for virulence (methicillin resistance and Panton-Valentine leukocidin toxin producing).

Research aim: The aim was to compare the microorganisms responsible for mastitis and breast abscesses during breastfeeding.

Methods: This observational study was conducted with a sample of women ($N = 60$) admitted to our hospital between 2016 and 2018. Participants affected by mastitis and breast abscess were studied and cared for by a multidisciplinary working group. A diagnostic breast ultrasound identified the pathology.

Results: Twenty-six participants (43.3%) were affected by mastitis and 34 (56.7%) by breast abscess. The most common microorganism identified was *Staphylococcus aureus* (*S. aureus*; mastitis, $n = 13$; abscesses, $n = 24$). Methicillin resistance was identified in 21 (44.7%) *S. aureus* strains: 17 (80.9%) cases of abscess and four (19.1%) cases of mastitis. The median number of months of breastfeeding was smaller in the methicillin-resistant *S. aureus* (MRSA) cases (median = 3, range = 1–20 months) than in the methicillin-sensitive *S. aureus* (MSSA) cases (median = 6.5, range = 3–21 months). The Panton-Valentine leukocidin toxin gene was detected in 12 (25.5%) cases (MRSA, $n = 8$, 66.7%; MSSA, $n = 4$, 33.3%). Hospitalization was required more frequently in

MRSA ($n = 8$, 38%; five Pantone-Valentine leukocidin positive) than in MSSA cases ($n = 5$, 19%; one Pantone-Valentine leukocidin positive). Four women out of the eight MRSA cases (50%) that were Pantone-Valentine leukocidin positive stopped breastfeeding during mammary pathologies, three (37.5%) participants continued breastfeeding until the follow-up recall, and one case was lost at follow-up.

Conclusion: Clinical severity was probably complicated by the presence of the Pantone-Valentine leukocidin toxin, which required hospitalization more frequently.

KEYWORDS

anatomy, breast, breastfeeding, human milk

BACKGROUND

Breastfeeding represents a unique opportunity for both infant and maternal health, providing the physiological nutrition for healthy growth and development (Patel et al., 2017). Theoretically, every mother is able to breastfeed her child once she has received the correct education and assistance. Exclusive breastfeeding is recommended following birth up to 6 months of age, as it is considered the best source of nourishment for infants and the best protection against infectious and chronic infant diseases (Mediano, Fernández, Rodríguez, & Marín, 2014; World Health Organization, 2009). Furthermore, breastfeeding contributes to the health and well-being of mothers, reducing the risk of ovarian and breast cancer (Unar-Munguía, Torres-Mejía, Colchero, & Gonzalez de Cosio, 2017).

Globally, researchers have reported an approximately 20% rate of developing mastitis during the lactation period (Cullinane et al., 2015; Yu, Sun, & Zhang, 2018). Breast abscess, a localized collection of pus within the breast, is often a complication of mastitis but may also occur without an apparent preceding mastitis. The incidence of breast abscess also varies widely, with most estimates coming from retrospective studies with participants who have had mastitis. Even though mastitis is a common and distressing condition among lactating women, little is known regarding the bacteria involved.

Together with *Streptococcus* spp., *Corynebacterium* spp., and *Enterococcus* spp., *Staphylococcus aureus* (*S. aureus*) is a colonizer of skin and mucosa (Fetsch, Roesler, Kraushaar, & Friese, 2016) responsible for methicillin resistance and the production of Pantone-Valentine leukocidin (PVL) toxin, a cytotoxin capable of inducing the inflammatory response and the local necrotic process (Li,

Zhou, Zhan, Huang, & Wang, 2018). *S. aureus* possesses many virulence factors, some of which enable it to manipulate the innate and adaptive immune responses of the host (Koymans et al., 2017). In particular, the development of skin abscesses in healthy individuals is associated with certain strains of community acquired methicillin-resistant *S. aureus* (CA-MRSA) and community-acquired methicillin-sensitive *S. aureus* (CA-MSSA; Aung et al., 2016), which encode the PVL gene (Changchien, Chen, Chen, & Chu, 2016). Unfortunately, infections occur in settings that generally include close physical contact (e.g., breastfeeding), the sharing of clothes, and contact with other people (Changchien et al., 2016). The incidence of community-acquired skin and soft-tissue infection (SSTIs) due to *S. aureus* has increased worldwide during the past few decades (Alabi et al., 2018); it is the major cause of bovine mastitis (Hoekstra et al., 2018).

PVL and other virulence factors (e.g., phenol-soluble modulins, alpha toxin, arginine catabolic mobile element, and Protein A) have an important role in the severity, persistence, and increased transmission of the disease (Kale & Dhawan, 2016). PVL, the cytotoxin produced by some clones of *S. aureus*, causes leukocyte destruction and tissue necrosis. It is associated with infections ranging from uncomplicated skin and soft-tissue infections to life-threatening necrotizing pneumonia. The gene encoding for PVL is a virulence factor recognized in CA-MRSA and also associated with CA-MSSA (Chiu, Lo, & Wang, 2012). Data about *S. aureus* infections during the puerperium are lacking. The aim of this study was to compare the microorganisms responsible for mastitis and breast abscesses during breastfeeding.

METHODS

Design

This is a longitudinal, prospective, observational design. It was the most appropriate design because little is known on this topic. All data used in the study previously had been anonymized, according to the requirements set by Italian Data Protection Code (Leg. Decree 196/2003) and by the general authorizations issued by the Data Protection Authority. Therefore, further approval of an institutional review board was not required.

Setting

The city of Milan is the main urbanized area in Lombardy region, in the North of Italy, with a population of 1,395,274. Out of 724,000 women, about 80,000 live in the L. Sacco Hospital area, and 20% are foreigners. The L. Sacco Hospital is located in an urban area characterized by a low

socioeconomic level. This hospital has a Breastfeeding Unit dedicated to treating breastfeeding pathology with women referred from all over Milan, not only the areas neighboring the hospitals. Although no UNICEF Baby-Friendly hospitals have been recognized since 2018 in Milan, L. Sacco Hospital follows UNICEF 10 steps to successful breastfeeding and has succeeded during the first phase evaluation for the Baby-Friendly hospital certificate process (the second phase is currently in process). In 2017, 1,215 children were born. The rate of breastfeeding at discharge in healthy children (according to UNICEF–World Health Organization [WHO] definition) in 2016 to 2018 was 81%.

L. Sacco Hospital has a shared breastfeeding policy accessible to everyone, and the Maternal-Neonatal Department staff are trained according to UNICEF-WHO to support breastfeeding. A space dedicated to welcoming new parents and children (“Moms, Dads, and Babies Space”) and a multidisciplinary Breastfeeding Unit group was established to care for breastfeeding women in 2016

Sample

Women ($N = 60$) affected by mastitis and breast abscess, referred to the Breastfeeding Unit, were the target population. Inclusion criteria were mastitis and/or breast abscess; exclusion criterion was breast engorgement. Mastitis was defined as a tender, hot, swollen, wedge-shaped area of breast associated with temperature of 38.5°C or higher, chills, flulike aching, and systemic illness (Amir & Academy of Breastfeeding Medicine Protocol Committee, 2014). Breast abscess was suspected if a well-defined area of the breast remained hard, red, and tender. All participants had a breast ultrasound to rule in or rule out abscess. Due to the observational design, no formal sample size calculation was previously performed. All 60 consecutive women with a diagnosis of mastitis and/or abscess were enrolled.

Milk specimens were collected prior to starting antibiotic therapy. In the case of mastitis, milk was collected through milk expression, and in the case of breast abscess, fluid or pus was collected by needle aspiration under ultrasound guidance. Human milk collection was obtained by a handexpressed midstream sample into a sterile container (a small quantity of the initially expressed milk was discarded to avoid contamination). The patient or provider washed hands prior to milk expression and wore gloves. The nipple was cleaned prior to collection to reduce skin contamination and minimize false-positive culture results.

Measurement

Milk samples were introduced into blood culture bottles (BacT/ALERT, BioMérieux, Marcy L'Etoile, France) and cultured on selective agar plates if positive. The isolates identification was performed with MALDI-TOF (BioMérieux, Marcy L'Etoile, France) and antimicrobial susceptibility with the automated analyzer Vitek.2 (BioMérieux, Marcy L'Etoile, France), according to the European Committee on Antimicrobial Susceptibility Testing (2018) breakpoints. Genomic DNA extraction was performed using the UltraClean microbial DNA isolation kit (Mo Bio Laboratories, Carlsbad, CA), as described in the manufacturer's protocol. To confirm the presence of the *mecA* gene and to assess the presence of the PVL gene, DNA extracted from bacterial isolates was tested with the RealCycler SAMAPV Real-Time PCR (Progenie Molecular, Spain).

Data Collection

Maternal demographic characteristics, body mass index, smoking status, marital status, employment status, pregnancy variables, delivery characteristics, peripartum variables, neonatal outcomes, breastfeeding features in the hospital, time of onset of mammary pathologies, coexistence of mammary pathologies, antibiotic therapy, and surgical treatment were collected into a database. Participants were admitted to the Breastfeeding Unit of the Obstetrics and Gynecology Unit of L. Sacco Hospital in Milan between January 2016 and January 2018. Participants were cared for by a multidisciplinary working group composed of a gynecologist, a microbiologist, an infectious disease physician, a neonatologist, and a radiologist trained in the clinical and therapeutic management of mastitis, according to a predefined clinical protocol. In our clinical protocol, the participants with a diagnosis of mastitis or breast abscess were empirically treated with amoxicillin-clavulanic acid, penicillin, clindamycin, or cephalosporin. Therapy was eventually shifted according to antibiogram results and/or after 24 to 48 hr of empiric therapy without improvement of the clinical symptoms. A weekly follow-up was performed until the end of the antibiotic therapy and the disappearance of symptoms. Written informed consent for routine diagnostic and medical procedures was obtained for each patient. Human milk samples were collected by the nurses and physicians by needle aspiration or under ultrasound guidance. All examined isolates were cultured as part of the routine diagnostics (standard care) and preserved according to local epidemiological surveillance regulations. Colonization by a PVL producing *S. aureus* was evaluated by nasal swab sampling.

Data Analysis

Descriptive statistics were performed on all variables. Breastfeeding continuation rates were calculated for median months and range. All descriptive variables were statistically analysed without significant differences. Categorical variables were displayed as frequencies. For convenience, participants were divided into an *S. aureus* group (SAG; $n = 47$, 78.3%) and a non-*S. aureus* group (NSAG; $n = 13$, 21.7%). Means (standard deviations) if normally distributed (as determined by the Shapiro-Wilk test) or medians (range) were calculated for microorganism variables. Stata Version 15.1 software (StataCorp, Chicago, USA) was used to analyze data.

RESULTS

Characteristics of the Sample

Demographic, anthropometric, socioeconomic, smoking, and obstetric variables were similar between the SAG and NSAG (Tables 1 and 2). Rates of breastfeeding at delivery were the same for the SAG and NSAG (Table 3). The median number of months of breastfeeding was smaller in the MRSA-positive group (median = 3 months, range = 1–20) than in the MSSA-positive group (median = 6.5 months, range = 3–21) and the NSAG (median = 11 months, range = 0.1–15). Nipple excoriations and the use of nipple shields were more frequent in the SAG than in the NSAG (Table 3).

Breastfeeding was the exclusive feeding method in all NSAG participants (Table 3). The percentage of participants who stopped breastfeeding during therapy was higher, although not significantly, in the PVL-positive (50%) than in the PVL-negative MRSA cases (23%). Four out of eight PVL-positive MRSA participants stopped breastfeeding during mammary pathologies, three participants continued breastfeeding until follow-up recall (median = 3 months, range = 1–13), and one case was lost at follow-up. None of the women with PVL-positive MSSA stopped breastfeeding during treatment, and all breastfed until their children were 5 months old.

Pathology Distribution

The milk cultures collected from the 60 participants enrolled identified the presence of 69 different microorganisms: 10 (16.7%) were polymicrobial cultures, and 49 (81.6%) identified a single pathogen. Only 1 (1.7%) case showed a negative culture. As reported in Table 1, the most common microorganism identified was *S. aureus*. Distribution of SAG and NSAG bacterium according to mammary pathology is described in Table 4.

In NSAG cases, mastitis was the pathology most frequently reported, whereas breast abscesses were more frequent in SAG cases. *S. aureus* was methicillin resistant in 44.6% of cases. MRSA was responsible of 17 cases (50%) of breast abscess and four cases (15%) of mastitis.

MSSA was detected in 14 cases (41%) of breast abscess and 11 cases (43%) of mastitis. The presence of the PVL gene was reported in 12 SAG cases (eight MRSA and four MSSA) out of 47 cases (25.5%) investigated. The clinical features of the 12 participants with PVL-positive *S. aureus* strains were as follows: MSSA PVL-positive strains were reported in mastitis ($n = 2$), purulent mastitis ($n = 1$), and abscess ($n = 1$); MRSA PVL-positive strains were reported in abscess ($n = 5$), bilateral abscess ($n = 2$), and purulent mastitis ($n = 1$).

All cases were clinically resolved with antibiotics and needle aspiration when required; despite the high percentage of benzyl-penicillin resistance identified in the microorganisms isolated (33 out of 47 *S. aureus* strains, 70%; nine out 12 PVL-positive *S. aureus* strains, 75%), a clinical resolution was obtained using clindamycin or ceftriaxone.

Among the SAG, MRSA cases (eight cases; five PVL positive, 62.5%) required hospitalization more frequently than MSSA cases (five cases; 1 PVL positive, 20%), although not significantly (Table 4). The presence of PVL toxin was detected in two cases of mastitis, six cases of abscess, three cases of bilateral abscess, and one case of purulent mastitis. Recurrent mammary pathologies were observed in four cases of MSSA, in three cases of MRSA, and in two cases in the NSAG. PVL-positive MRSA was detected in one case of recurrent mastitis.

No maternal or neonatal complications during therapy were reported in the sample. An allergic reaction occurred in two mothers without any influence on breastfeeding because they had already stopped breastfeeding. A periumbilical pustule was reported in the neonate of an abscess-affected mother who had PVL-positive MRSA, which developed prior to the onset of the mammary pathology. The neonatal cutaneous infection resolved with local antibiotic therapy. The participant chose to stop breastfeeding after the onset of the mammary pathology. One participant with a PVL positive culture had inguinal pyodermitis prior to the onset of breast abscess. The 12 PVL-positive participants in the SAG screened for molecular detection by nasal swab were negative.

DISCUSSION

Pérez, Orta, Padilla, and Mesquida (2013) have pointed out the rising frequency of CA-MRSA in puerperal infection (e.g., mastitis, abscesses, and wound infections). The incidence of *S. aureus* in puerperal mastitis has been reported around 40% to 50% (Contreras & Rodríguez, 2011), rising to 67% to 84% in the presence of breast abscesses (Ramakrishnan, Trichur, Murugesan, &

Cattamanchi, 2017). However, data on MRSA incidence have been mostly reported in abscess cases, with great variability in different geographical areas (less than 5% in United Kingdom, 60% in United States). In our sample, rates of *S. aureus* were higher than previously reported, confirming an MRSA rate similar to the MRSA incidence reported in Italy and in the Lombardy region (Bellino et al., 2018). Our study was the first to evaluate the diffusion of CA-MRSA in puerperal mammary pathologies in Italy. The high rate of *S. aureus* infection was probably due to the selection of the sample, because particularly critical cases were referred to our attention.

Although recognizing the primary functions of mammary glands as immunologic support and protection of the infant during milk production (Cetin et al., 2014), in our study, half of the participants with this infection stopped breastfeeding. This finding suggests that in our population, breastfeeding did not have a protective role against CA PVL-positive *S. aureus* neonatal infections; however, additional research is needed to further explore this situation. In our sample, a mother–child transmission of soft-tissue infection due to PVL-positive MRSA, which has been described by Franck et al. (2017), was observed in only one case. Our findings require further microbiological investigation into the vertical transmission of *S. aureus* colonization.

We also studied for the first time the PVL toxin in mammary pathologies, which was elevated in contrast to the low rate (5%) previously reported (Bakthavatchalam, Nabarro, Ralph, & Veeraraghavan, 2017).

Clinical severity noted earlier was probably complicated by the presence of PVL toxin (Rimoldi et al., 2018). In our experience, all the women (PVL positive or PVL negative) affected by mastitis showed a clinical resolution following antibiotic treatment; therefore, we can affirm that the presence of PVL does not represent a limit in the clinical resolution. The role of this toxin in the clinical severity and in the therapeutic approach also needs further investigation.

Limitations

The small sample size was a limitation; therefore, larger studies are needed. The findings may have been influenced by the severity of the disease referred to our Breastfeeding Unit. Another potential limitation of this study is lack of data about the screening of *S. aureus* using nasal swab in the maternal and neonatal groups, limiting our ability to assess whether *S. aureus* at the site of infection was associated with a nasal colonization.

CONCLUSION

This is the first observational study reporting microbiological data of women affected by postpartum mastitis. The high percentage of participants with PVL-positive mammary pathologies during breastfeeding in absence of an outbreak should be considered when developing clinical protocols and starting proper therapy. Moreover, given the high recurrence and the difficulty in eradicating PVL-positive infections, further epidemiologic study and molecular analysis of *S. aureus* are required in recurrent infections in order to better understand the incidence and the spread of these *S. aureus* strains.

Authors' Note

Rimoldi and Pileri equally contributed to the work.

Declaration of Conflicting Interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Table 1. Demographic Characteristics of the Sample ($N = 60$).

Characteristic	<u>SAG ($n = 47$)</u>		
	MSSA ($n = 26$) n (%)	MRSA ($n = 21$) n (%)	NSAG ($n = 12$) n (%)
Ex-smoker	4 (15)	2 (9)	2 (16)
Pregnancy complications ^a	3 (11)	4 (19)	3 (25)
Primiparous	20 (77)	13 (62)	11 (92)
Cesarean section	3 (11)	3 (14)	3 (25)
Vacuum extractions	3 (11)	1 (4)	0
Inducing labor	9 (34)	4 (19)	3 (25)
Peridural analgesia	8 (30)	5 (24)	4 (16)
Postpartum complications ^b	0	1 (4)	0
Neonatal complications ^c	3 (11)	1 (4)	2 (16)

Note. SAG = *Staphylococcus aureus* (*S. aureus*) group; NSAG = non-*S. aureus* group; MRSA = *S. aureus* methicillin resistance; MSSA = *S. aureus* methicillin sensitive.

^aPregnancy complications included gestational diabetes ($n = 5$), preeclampsia ($n = 1$), HELLP syndrome ($n = 1$), cholestasis ($n = 1$), and intrauterine growth restriction ($n = 2$). ^bPostpartum complications included postpartum hemorrhage ($n = 1$). ^cNeonatal complications included jaundice ($n = 4$), frenulotomy lingual ($n = 1$), and moderately preterm birth ($n = 1$).

Table 2. Demographic Characteristics of the Sample ($N = 60$).

Characteristic	<u>SAG ($n = 47$)</u>		
	MSSA ($n = 26$) M (SD)	MRSA ($n = 21$) M (SD)	NSAG ($n = 12$) M (SD)
Maternal age, years	31.4 (4.5)	35.3 (4.8)	32.9 (5.4)
BMI, kg/m ²	21.3 (4.6)	19.8 (3.7)	20.9 (2.9)
Weight gain in pregnancy, kg	12.9 (3.9)	11.8 (3.9)	12.8 (5.3)
Gestational age at delivery, weeks	39.3 (1.0)	39.1 (1.8)	39.3 (1.5)
Birth weight, grams	3221.6 (347.5)	3231.5 (545.0)	3353.0 (534.0)
Hospital admission, postpartum days	3.2 (1.4)	3.5 (1.6)	3.7 (2.6)

Note. SAG = *Staphylococcus aureus* (*S. aureus*) group; NSAG = non-*S. aureus* group; MRSA = *S. aureus* methicillin resistance; MSSA = *S. aureus* methicillin sensitive; BMI = body mass index.

Table 3. Breastfeeding Patterns Grouped by Pathogen ($N = 59$).

Breastfeeding Pattern	SAG ($n = 47$)			Total n (%)
	NSAG ($n = 12$) n (%)	MSSA ($n = 26$) n (%)	MRSA ($n = 21$) n (%)	
Breastfeeding in hospital^a				
Exclusive breastfeeding	19 (73)	15 (71)	11 (92)	45 (76)
Breastfeeding	1 (4)	2 (9)	1 (8)	4 (7)
Nipple excoriations	13 (50)	9 (42)	3 (25)	25 (42)
Nipple shields	6 (23)	4 (19)	1 (8)	11(18)
Breast pump	3 (11)	5 (23)	1 (8)	9 (15)
Breastfeeding at onset of mammary pathologies^b				
Exclusive breastfeeding	15 (58)	13 (62)	12 (100)	40 (68)
Breastfeeding	4 (15)	3 (14)	0	7 (12)
Recently stop breastfeeding	1 (4)	2 (9)	0	3 (5)
Nipple shields	1 (4)	4 (19)	3 (25)	8 (13)
Breast pump	10 (38)	8 (38)	7 (58)	25 (42)
Ongoing breastfeeding ^c	7 (27)	7 (33)	3 (25)	17 (29)
Neonatal complications ^d	0	0	0	0
Lost at follow-up ^e	7 (27)	5 (24)	0	12 (20)
Hospitalization ^f	5 (19)	8 (38)	3 (25)	16 (27)
Recurrent mastitis/abscess	4 (15)	3 (14)	2 (16)	9 (15)
Fistulization/ulceration	0	4 (19)	0	4 (7)

Note. SAG = *Staphylococcus aureus* (*S. aureus*) group; NSAG = non-*S. aureus* group; MRSA = *S. aureus* methicillin resistance; MSSA = *S. aureus* methicillin sensitive.

^aMissing values on Breastfeeding in hospital: MSSA ($n = 6$); MRSA ($n = 4$). ^bMissing values on Breastfeeding at onset of mammary pathologies: MSSA ($n = 5$); MRSA ($n = 4$). ^cBreastfeeding continued beyond the end of the study. ^dJaundice, diarrhea, cutaneous or subcutaneous infections, sepsis. ^eCases lost at weekly follow up and at the recall. ^fCases required hospitalization by the severity of the mammary pathologies.

Table 4. Distribution of Mammary Pathologies in the Sample ($N = 60$).

Mammary Pathology	SAG ($n = 47$) n (%)	NSAG ($n = 12$) n (%)
Mastitis	14 (29.8)	8 (66.7)
Abscess	28 (59.6)	3 (25.0)
Bilateral mastitis	1 (2.1)	1 (8.3)
Bilateral abscess	3 (6.4)	0 (0)
Purulent mastitis	1 (2.1)	0 (0)

Note. SAG = *Staphylococcus aureus* (*S. aureus*) group; NSAG = non-*S. aureus* group.