The Journal of Maternal-Fetal & Neonatal Medicine

ISSN: 1476-7058 (Print) 1476-4954 (Online) Journal homepage: http://www.tandfonline.com/loi/ijmf20

Management of mastitis and breast engorgement in breastfeeding women

Olga Pustotina

To cite this article: Olga Pustotina (2016) Management of mastitis and breast engorgement in breastfeeding women, The Journal of Maternal-Fetal & Neonatal Medicine, 29:19, 3121-3125, DOI: 10.3109/14767058.2015.1114092

To link to this article: http://dx.doi.org/10.3109/14767058.2015.1114092

Accepted author version posted online: 29 Oct 2015.
Published online: 30 Nov 2015.

THE JOURNAL OF
MATERNAL-FETAL
& NEONATAL
MEDICINE

http://informahealthcare.com/jmf
ISSN: 1476-7058 (print), 1476-4954 (electronic)

J Matern Fetal Neonatal Med, 2016; 29(19): 3121–3125
 DOI: 10.3109/14767058.2015.1114092

Taylor & Francis
Taylor & Francis Group

REVIEW ARTICLE

Management of mastitis and breast engorgement in breastfeeding women

Olga Pustotina

Department of Obstetrics, Gynecology and Perinatology, Peoples' Friendship University of Russia, Mikluho-Maklaya Str 6, Moscow 117198, Russian Federation

Abstract

Objective: To identify the best management approaches to mastitis management in breastfeeding women and heavy breast engorgement in the early postnatal period.
Methods: We compared various international guidelines and reviews on mastitis management in breastfeeding women and breast engorgement treatment.
Results: Effective milk removal is recommended as a first step in mastitis management. Active emptying of the breasts can prevent mastitis development in most cases. If it fails, antibiotics should be administered for 10–14 days with continuing breastfeeding. Russian guidelines recommend antibiotic therapy during 5–7 days with temporary bromocriptine-induced breastfeeding suppression. In case of heavy breast engorgement after lactation is initiated, Progesterone-containing gel can be administered. Application of the progesterone-containing gel on the breast skin improves swelling, and reduces engorgement and tenderness in 15–20 minutes.
Conclusions: Antibiotics with temporary suppression of breastfeeding are more effective than with continuing breastfeeding in mastitis management. The progesterone-containing gel is recommended on the 3rd–4th days after childbirth in heavy breast engorgement to prevent mastitis.

Keywords

Breast engorgement, breastfeeding, mastitis

History

Received 18 September 2015
Revised 15 October 2015
Accepted 25 October 2015
Published online 24 November 2015

Introduction

The incidence of mastitis among breastfeeding women in different countries varies from 2% to 33%, approximately 10% on average [1], and shows no trend towards decline. According to the official data, the incidence of mastitis in Russia is 2–3% [2], while according to the results of the Sample Survey of Reproductive Health of 10 000 Russian women conducted in 2011 by the Federal State Statistics Service together with the Ministry of Health and Social Development, the UN Population Fund (UNFPA), Centers for Disease Control and Prevention (CDC) (USA), mastitis occurs in 10% of breastfeeding women [3]. The HAI Epidemiological Service explains this data divergence by an incomplete record of postpartum diseases in hospitals.

To identify the best approaches to mastitis management in breastfeeding women, we reviewed available international guidelines [2,4–10]. We collected information about causative agents, their correlation with breast-fed infants and sensitivity to antibiotics, incidence of different types of mastitis and its main treatment principles. Usually, mastitis is predisposed by breast engorgement or blocked ducts. We analyzed the existing methods of breast engorgement management and provided pathogenic substantiation for a two-stage strategy in heavy breast engorgement in the early period after childbirth.

Etiology of mastitis

The main causative agent of mastitis is *Staphylococcus aureus*. It is isolated as a pure bacterial culture in milk and/or purulent discharge in most women (90%), and much less frequently (10%) the bacterial culture is represented in associations with Gram-negative bacteria such as *Proteus*, *Klebsiella*, *Escherichia*, *Streptococcus epidermidis* and/or *Enterococcus*. In the majority of cases mastitis pathogenic microorganisms can be isolated from affected and healthy breasts. At the same time, the presence of pathogenic bacteria in breast milk does not necessarily indicate breast infection. *Staphylococcus aureus* can be cultured from milk of some healthy women in amounts usually not exceeding 10^2 CFU/mL, which is not require specific antibacterial therapy or restrictions of breastfeeding [11–13].

Paths of developing breast infection

The study of mother-child dyads showed that newborns are of key importance in epidemiology of mastitis. The bacterial

Address for correspondence: Olga Pustotina, Department of Obstetrics, Gynecology and Perinatology, Peoples' Friendship University of Russia, Mikluho-Maklaya Str 6, Moscow, 117198, Russian Federation. Tel: +7(916)9267652. E-mail: pustotina@gmail.com

culture of swabs demonstrated pathogenic Staphylococci in 10–15% of newborns on their first day of life, in 70–75% on the 3rd–4th days of life and in over 90% on the 7th day of life. The bacterial flora cultured from mothers' breasts and newborns' nasopharynx is usually identical. The outbreak of Staphylococcal infection among newborns often occurs simultaneously with the increase in clinical mastitis frequency in obstetric hospitals, which strongly suggests that mastitis is caused by nosocomial infection. This fact allows one to consider mastitis as a nosocomial infection. Conversely, breastfeeding of a newborn with *Staphylococcus aureus* is the dominant path of developing breast infection. Postpartum women as well as hospital staff carrying Staphylococcus are much more seldom source of infection [12–15].

Symptoms of mastitis

Most cases of mastitis are shown to develop between the 2nd and 4th weeks after childbirth and hospital discharge, which in some cases means late diagnosis and treatment along with incorrect self-treatment.

Most often, mastitis is predisposed by breast engorgement or blocked ducts and develops from non-infective to infective mastitis leading to breast abscess. A typical feature of mastitis is rapid progression of inflammation when breast abscess develops within 4–5 days from the onset, i.e. sudden high-grade fever (38–39 °C) and breast tenderness. Along with the typical course of the disease, there can be subclinical forms of mastitis. They are characterized by vague clinical symptoms or even their total absence and discrepancy between clinical symptoms and the real course of the disease.

Treatment of mastitis

In order to prevent the occurrence of severe purulent mastitis, which require surgical intervention and lead to serious breastfeeding problems, treatment should start as soon as the first signs and symptoms of mastitis appear. Lactostasis always precedes mastitis. Hence, its rapid elimination can prevent the majority of incipient mastitis cases only by using active expression of breast milk to get rid of milk stagnation even before administering antibiotics. All international guidelines are based on the principle ''Effective milk removal'', which means more frequent breastfeeding and additional expression of milk after breastfeeding by hand or a pump. Warm breast compresses before breastfeeding and cold compresses after are recommended for pain relief along with anti-inflammatory agents such as ibuprofen or paracetamol. Good rest between breastfeeding and drinking plenty of fluids are also advised. Of note, fluid intake of up to 2.5–3 L per day does not affect the amount of milk produced. A higher volume of fluid intake (up to 4–5 L per day) may even suppress prolactin secretion by the pituitary, leading to reduced milk production. In a vast majority of cases this strategy proved to be effective as it helps to eliminate lactostasis and breast inflammation and improve mother's condition [4,16].

Persistent fever and a tender palpable breast lump 24 h after starting an active mastitis management strategy are an absolute indication to administration of antibiotics. The treatment of choice includes synthetic penicillins and cephalosporins, resistant to bacterial b-lactamases. In case of methicillin-resistant *Staphylococcus aureus* (MRSA), vancomycin should be considered. The duration of treatment is at least 10–14 days. It is recommended to continue breastfeeding, which is proved to be safe for an infant even in breast abscess [4–10]. According to the 2013 systematic review [17], antimicrobial therapy at early stages of mastitis is not feasible. The use of antibiotics within 24 h of mastitis onset is as effective in preventing breast abscess as active expression of breast milk along. This can help to avoid antibiotics in the majority of breastfeeding women. The Russian Guidelines of Mastitis Management [2] are somewhat different from the international recognized approaches to mastitis management.

Comparative analysis of Russian and international guidelines of mastitis management

A prominent Russian scientist Boris Gurtovoy, who dedicated many years of his research and clinical activities to obstetric infections, who dedicated many years of his research and clinical activities to obstetric infections and was one of the pioneers in mastitis management school in former USSR. He managed 642 breastfeeding women with mastitis in the time of a massive outbreak of Staphylococcus infection in Moscow in 1973–1977 [18]. He implemented clinical guidelines, which were followed by practitioners. Later his recommendations formed the basis for the Clinical Guidelines for mastitis management of the Russian Society of obstetricians and gynecologists [2].

The comparative analysis of the Russian guidelines of mastitis management with international ones showed differences in terminology of mastitis development stages. Serous, infiltrative and purulent forms of the disease correspond to non-infective, infective forms of mastitis and breast abscess.

The incidence of purulent mastitis (breast abscess) in USSR use to be very high because of late mastitis development between the 2nd and 4th weeks after childbirth, which in some cases means untimely diagnosis and late treatment along with incorrect self-treatment. The average time from the onset of the disease to hospital admission was 15 days, so more severe purulent forms developed in 64% of patients: infiltrative purulent – in 54%, phlegmonous – in 9%, and gangrenous – in 1% of patients, while breast abscess was diagnosed only in 36% of patients. Moreover, a prolonged hospital stay of post-operative patients resulted in recurrent infection of their surgical wounds because of contamination with different hospital-acquired strains. The number of coliform gram-negative bacteria, especially *Proteus*, in the wound discharge rapidly increased. It significantly complicated the course of the disease and often required repeated surgeries. Gentamycin and lincomycin are recommended in such cases as well as in a recurrent infection during the postoperative period, bacteria being taken into account (associations of pathogenic Staphylococcus and gram-negative microorganisms). Proteus and other gram-negative bacteria are resistant to other antibiotics [13,19,20].

According to recent Russian [2] and international studies [15,16], advanced forms of breast abscess, including those caused by reinfection of a postoperative wound, occur more

rarely than in USSR. The reason for this may be an earlier discharge of patients from hospital after mastitis surgery and their management in out-patient settings. Such approach contrary to a longer stay of maternity patients in hospital allows minimization of postoperative wound contamination risks with hospital infections and considerably improves their recovery prognosis.

Russian experts recommend temporary discontinuation of breastfeeding in order to prevent severe purulent forms of the disease and reinforce antibiotics effect. All therapeutic measures have to be focused on a rapid response to breast inflammation to prevent pus formation. Thus, temporary lactation suppression should be additionally considered. The prolactin inhibitor – bromocriptine is recommended as a suppressive agent [21]. Such strategy is based on the following facts: mastitis usually develops in breastfeeding women (''no lactation – no mastitis''), mastitis does not occur in postpartum women whose lactation is suppressed, and experimental data indicate high susceptibility to breast infection in breastfeeding animals.

Originally, a combination of estrogen and androgen was used to suppress lactation along with diuretics, osmotic laxatives and ointment compresses. The use of non-pharmacological measures of lactation suppression (e.g. tight breast compression, fluid restriction and avoidance of breast milk expression) in mastitis is unwarranted and ineffective.

Since the beginning of the 1970s bromocriptine, which inhibits prolactin secretion (the main hormone of galactpoiesis), has been used. To suppress postpartum lactation, bromocriptine is administered orally 2.5 mg 2–3 times a day for 3–5 days. Concurrent administration of antibiotics with prolactin inhibitor rapidly results in breast softening, re-absorption of accumulated milk and resolution of inflammation. Small doses of bromocriptine in short-course treatment enable to avoid subsequent cessation of milk production and resume breastfeeding [21,22].

To stop lactation completely, bromocriptine is used in treatment courses, each lasting 2–3 weeks [13,21]. It is important to note that no cases of thrombosis in postpartum women taking bromocriptine have ever been reported. Shorter periods are required for galactopoiesis suppression when another dopamine receptor agonist – cabergoline – is used at 250 μg twice a day for two days [23]. Our experience in administering cabergoline to suppress well-established lactation shows that a two-day therapy is not enough. In most cases galactopoiesis is restored. For this reason, we recommend to use cabergoline for four days.

The comparison of mastitis therapy effectiveness showed better results in mastitis management with reduced galactopoiesis than with continuing breastfeeding. The criteria of effectiveness such as improved general state of patients, reversal of mastitis symptoms, normalization of body temperature and hematological parameters, prevention of purulent infiltration in the breast, wound healing and absence of repetitive surgical interventions in breast abscess revealed a significantly higher treatment efficacy of galactopoiesis-suppressive medications versus antibiotics with continuous breastfeeding. The use of such approach in Moscow enabled to reduce the incidence of purulent mastitis 3.1-fold over a 5-year period (1973–1977) [24]. Moreover, this resulted in shorter disease duration and, accordingly, antibacterial therapy (5–7 days versus 10–14 days as set by the international standards [1,4–10]) and rapid breastfeeding resume.

Breast engorgement management

Mastitis always develops from breast engorgement. Hence, its rapid resolution considerably improves the effectiveness of complex therapy. In most cases incipient mastitis can be treated by active emptying of the breast even before administration of antibiotics. At the same time, in heavy breast engorgement, which occurs on the 3rd–4th days after childbirth mostly in non-breastfeeding women, typical recommendations on intensive expression of breast milk and breastfeeding do not produce expected results and often make the situation even worse.

Breast engorgement in non-breastfeeding mothers in the first few days after childbirth is the sign of a breast dysfunction [25–27]. Lack of peripheral prolactin receptors stimulation in the breast provokes hormonal imbalance, i.e. increased level of prolactin in combination with decline of oxytocin secretion and concentration of placental steroids, primarily of progesterone (Figure 1). According to our data, serum progesterone concentration on the 3rd–4th days postpartum in women with normal galactopoiesis is on average 6.8 ± 1.8 nmol/L and prolactin concentration is 5182 ± 1117 mIU/L, whereas in breast engorgement the average progesterone level is significantly lower, while the average prolactin level is higher, i.e. 5.5 ± 1.4 nmol/L and 6632 ± 1074 mIU/L respectively ($p < 0.05$) [25].

Severe swelling, breast engorgement and tenderness occur in connection with the hormonal imbalance, which disrupts both expression of breast milk and breastfeeding. Besides, heavy expression of breast milk in such conditions worsens swelling and engorgement of the breasts and may also cause hemorrhages and alveolar damage.

The review of traditional methods of severe breast engorgement management shows their ineffectiveness. Thus, cabbage leaf compresses, massage, acupuncture and physiotherapeutic procedures are barely effective, drotaverine with oxytocin injections intended to cause contraction of alveolar myoepitheliocytes are not effective either, given that milk ducts are compressed by swollen breast milk glands and, conversely, warming alcohol compresses block the effect of oxytocin on the contractile activity of alveolar myoepitheliocytes [25,27].

B.L. Gurtovoy was the first to offer a two-stage therapy of severe breast engorgement [21]. On the first stage the inhibitor of prolactin synthesis – bromocriptine – is administered during one or two days, which enables to eliminate hormonal imbalance; expression of breast milk then follows. Treatment with 2.5 mg bromocriptine 2–3 times a day leads to diminished breast engorgement in 1–2 days, provided the breasts are given rest during this period.

Since 2005, we first started to use the progesterone-containing gel (Progestogel) for transdermal therapy in women with heavy breast engorgement after childbirth. Progesterone-containing gel is widely used in gynecology to treat mastalgia and mastodynia, which develop due to progesterone deficiency, occurring in breast engorgement.

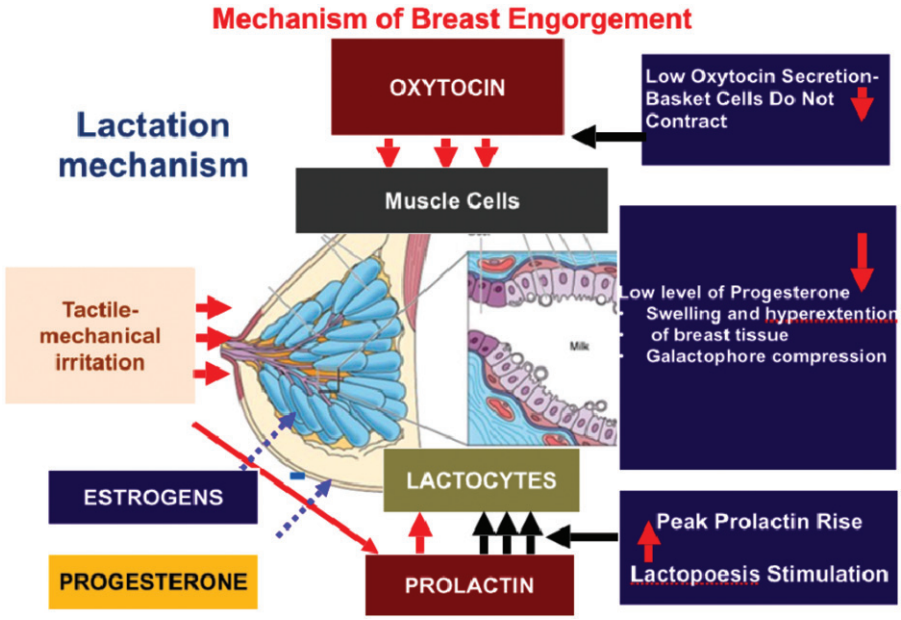

Figure 1. The mechanism of breast engorgement.

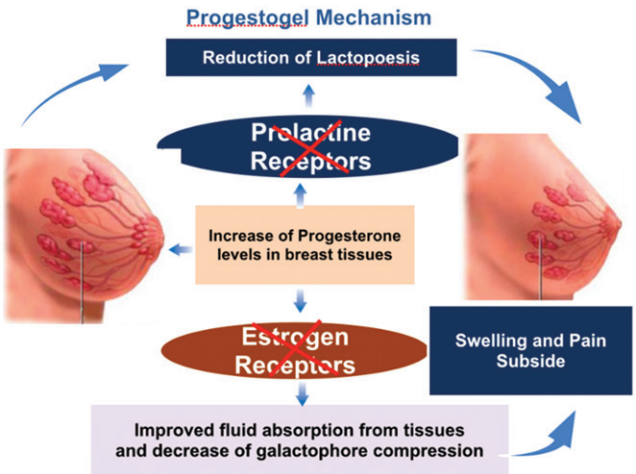

Figure 2 The mechanism of progestogel.

According to our observations, application of 2.5 g of the 0.025 g-progesterone-containing gel on the breast skin is leading to rapid reduction of breast swelling, engorgement and tenderness, which allows starting expression of milk in 15–20 minutes. Breast engorgement is eliminated in a vast majority of patients following a single application of the transdermal progesterone-containing gel. Only few of them (3% in our study) required repeated gel application to achieve clinical effect [25].

The skin application of the gel (Figure 2) compensates for progesterone deficiency caused by lactostasis in breast tissue. This stimulates loss of fluid from breast tissue and, hence, reduction of breast swelling and engorgement. In addition, progesterone at increased concentrations blocks prolactin receptors in breast tissue, resulting in decreased alveolar galactopoiesis, while exerting no systemic effect [25]. Progesterone is absorbed from tissues into bloodstream after one hour, allowing the mother to start breastfeeding. Rapid breast engorgement relief that can be completely achieved by the two-stage therapy (hormonal dysfunction correction followed by expression of breast milk) is the key element to prevent breast infection and mastitis development.

Conclusion

Having compared different guidelines, the most efficient type of mastitis management in breastfeeding women is ‘‘Effective milk removal’’ on the first stage, which leads to recovery in most women. If it fails, antibiotics with temporary breast-feeding suppression are administered. It is more effective than therapy with continuous breastfeeding.

In heavy breast engorgement on the 3rd–4th days after childbirth, the two-stage management is recommended; progesterone-containing gel should be considered. To prevent breast engorgement and consequently mastitis, it is necessary to comply with the basic principles of breastfeeding – the early start of breastfeeding and adherence to the breastfeeding technique and personal hygiene rules, breastfeeding on demand, rooming-in practice, hand hygiene and early discharge from the obstetric hospital.

To conclude, one can quote a saying of V.F. Voyno-Yasnetsky from his book ‘‘Purulent Surgery Essays’’ published in 1956: ‘‘Mammitis is as old as the hills. Millions of women suffer from it. Doctors have been trying to treat it since the dawn of time. However, even nowadays when surgery is highly developed, we cannot boast of our perfect skills to treat mastitis...’’ [28]. The answer to it can be a quote of B.L. Gurtovoy: ‘‘Treatment of mastitis should begin as early as possible, when there are the first signs of it. A timely complex therapy almost always allows to prevent suppuration development’’ [18].

Acknowledgements

I appreciate my colleague help Ylya Trusov for preparation of this publication.

Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of this article.

References

1. Department of Child and Adolescent Health and Development. Mastitis: Causes and Management. Geneva: WHO; 2008.
2. Gurtovoy BL, Emelianova AI. Postpartum mastitis. In: Serov VN, Sukhikh GT, eds. Clinical recommendations. Obstetrics and Gynecology. 4th Issue (Russian). GEOTAR-Media, 2014:546–51.
3. Reproductive Health of the Population in Russia in 2011, Summary of the report. Available from: http://www.gks.ru/free_doc/new_site/population/zdrav/zdravo-2011.pdf [last accessed 14 Nov 2015].
4. World Health Organization: Mastitis: Causes and Management, Publication Number WHO/FCH/CAH/00.13. Geneva: World Health Organization; 2000.
5. Cusack L, Brennan M. Lactational mastitis and breast abscess – diagnosis and management in general practice. Austral Fam Physician 2011;40:976–9.
6. Kataria K, Srivastava A, Dhar A. Management of lactational mastitis and breast abscesses: review of current knowledge and practice. Indian J Surg 2013;75:430–5.
7. The Academy of Breastfeeding Medicine Protocol Committee (ABM) Clinical Protocol #4: Mastitis. Breastfeed Med 2008;3:177–80.
8. Jacobs A, Abou-Dakn M, Becker K, et al. Association of Scientific Medical Societies in Germany (AWMF) Guidelines. Geburtshilfe Frauenheilkd 2013;73:1202–8.
9. ACOG Committee Opinion N 361: Breastfeeding: maternal and infant aspects. Obstet Gynecol 2007;109:479–80.
10. Clinical Knowledge Summary. Mastitis and breast abscess; 2010. Available from: www.cks.nhs.uk.417660
11. Delgado S, Arroyo R, Rodriguez JM, Rodriguez RM. PCR-DGGE assessment of the bacterial diversity of breast milk in women with lactational infectious mastitis. BMC Infect Dis 2008;8:51–6.
12. Voropaeva SD, Gurtovoy BL, Emelyanova AI, Mirinova TG. Quantitative features of milk microflora in diagnosing lactation mastitis. Obstet Gynecol (Russ) 1983;8:61–3.
13. Gurtovoy BL. Postpartum mastitis. In: Gurtovoy BL, Kulakov VI, Voropaeva SD, eds. Antibiotics in obstetrics and gynecology. (Russian). Moscow: Triada-X, 2004:96–104.
14. Amir LH, Garland SM, Lumley J. A Case-control Study of mastitis: nasal carriage of *Staphylococcus aureus*. BMC Fam Pract 2006;7:57–61.
15. Crepinsek MA, Crowe L, Michener K, Smart NA. Interventions for preventing mastitis after childbirth. Cochrane Database Syst Rev 2012;CD007239.
16. Dixon JM, Khan LR. Treatment of breast infection. BMJ 2011;342:d396.
17. Jahanfar S, Ng CJ, Teng CL. Antibiotics for mastitis in breastfeeding women. Cochrane Database Syst Rev 2013;CD005458.
18. Gurtovoy BL, Grashchenkova ZP. Clinical symptoms and management of lactation mastitis. Obstet Gynecol (Russ) 1973;8:51–4.
19. Chadaev AP, Zverev AA. Acute purulent lactation mastitis (Russian). Moscow: Medicina, 2003:15–21
20. Kulakov AA, Shkoda SM, Astashov PY, et al. Lactation mastitis: problems and prospects. Surgery NI Pirogov J (Russ) 2004;6:36–8.
21. Gurtovoy BL, Emelyanova AI, Ryabenko LV, Mironova TS. Administration of parlodel in lactation mastitis. Obstet Gynecol (Russ) 1984;5:22–5.
22. Petersen EE. Infections in obstetrics and gynecology. New York: Thiem; 2006.
23. Rains CP, Bryson HM, Fitton A. Cabergoline. A review of its pharmacological properties and therapeutic potential in the treatment of hyperprolactinaemia and inhibition of lactation. Drugs 1995;49:255–60.
24. Akhmedyanova GU, Gurtovoy BL, Voropayeva SD. Justification of rational antibiotic therapy for lactational mastitis. Obstet Gynecol (Russian) 1977;5:49–53.
25. Pustotina OA. Lactation mastitis and breast engorgement. Russian Bulletin. Obstet Gynecol (Russ) 2007;2:55–7.
26. Salomon CW, Wegnelius G, Holmgren-Lie A. Incorrect breastfeeding technique and milk stasis are the most common problems. Lakartidningen 2000;97:4838–42.
27. Mangesi L, Dowswell T. Treatments for breast engorgement during lactation. Cochrane Database Syst Rev 2010;9:CD006946.
28. Voyno-Yasnetsky VF. Purulent surgery essays. (Russian). Leningrad: Medgiz, 1956:260–7.