**S25. Can we define lactational mastitis?**

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*Introduction* Lactational mastitis is a common and problematic complication of breastfeeding. It makes mothers feel very ill and may lead to premature weaning. The wide range of incidences reported in the scientific literature (2.6 % and 33 %) suggests that there is some dissension about the definition of the disease. In 2010, a concept analysis[1] of the usage of the term "lactational mastitis" in contemporary scientific literature confirmed the problem of defining the ailment.

Although the symptoms are generally agreed upon, we still have no clear consensus about the cause of these symptoms. Theories include permeation of the breast milk into breast tissue, caused either by simple fullness of the ducts or by blocked ducts, an active immune response and the effect of cytokines leaking through, what should be, tight cell junctions. Recent clinical and bacteriological research has revealed that healthy breastfeeding women have an abundance of bacteria in their breast milk, including both potential pathogens and probiotics[2-4]. Moreover, potential pathogens have been found in similar concentrations in the milk of breastfeeding women both with and without symptoms of mastitis[4]. These findings suggest a normal bacterial flora in the breast milk of lactating women, which might seem obvious, but is something that has only recently been clearly expressed in the literature. If milk containing a diversity of bacteria is pressed out into the connective tissue of the breast the subsequent immune response will give rise to all the symptoms of mastitis that we are familiar with; sudden onset, pyrexia (at times extreme), breast tension, erythema and pain. Though interesting, one more theory of the aetiology of symptoms does nothing to help us decide on an operational definition for mastitis.

*Do we need an operational definition for mastitis?* This question might well be posed – if awoman has all the symptoms, then she has mastitis and should be treated. But how should she be treated? Recent research has shown that 85 % of women with these symptoms recovered spontaneously without recourse to antibiotic therapy[4]. Instead, the women received care interventions to help relieve symptoms. The severity of the women's symptoms did not correlate with the amount of bacteria in their milk. Furthermore, the 15 % who received antibiotic treatment did not differ from the remaining 85 % either for the severity of their symptoms or the amount of potential pathogens in their breast milk. Neither was it possible to predict by type and amounts of bacteria in the breast milk which women would develop a breast abscess[4].

*Why are these findings important and how can we use them?* Of the utmost importance is the need to be constantly vigilant regarding over-use of antibiotics in order to avoid an ever-increasing bacterial resistance. Research reports from many high-income countries show that between 77 % and 97 % of women with symptoms of mastitis are prescribed antibiotics. These figures may be far too high; we do not know how many of the 15 % who were given antibiotics in the study reported above[4] might also have made a spontaneous recovery. The occurrence of residual symptoms requiring antibiotics within six weeks after the episode of mastitis was 4.5 %. When we treat breastfeeding women with antibiotics we treat even the child, with the inherent risk of disrupting the developing flora of the infant gut, another important reason why these results should give rise to careful consideration regarding antibiotic treatment.

*So, can we define mastitis?* If we base definition on signs and symptoms, it is obvious that we risk treating many women with antibiotics who might well recover spontaneously. If we base definition of mastitis on the type and amount of bacteria in the milk, the same risk occurs. Researchers have recently suggested that mastitis is a disbiotic process[5] and continuing research into that area may in future help us to define mastitis more clearly. However, until that time, I contend that although a universal definition might take mastitis

research forward at a quicker pace, it may be meaningless in a clinical situation. It is possible that the question of which women with mastitis require antibiotics cannot be answered by applying epidemiological methods. Instead, the answer may lie in the workings of the individual woman's immune response. Do women who develop breast abscess do so because of a strongly active immune response rather than mis-management of mastitis?

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**S26. Appropriate treatment for infectious mastitis during lactation**.

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Breast milk is a source of bacteria ($<10^3$ CFU/ml milk) to the infant gut. Staphylocci, streptococci and lactic acid bacteria are among the predominant cultivable bacteria. Therefore, this fluid plays a key role in the acquisition of the infant gut microbiota. However, there are a variety of factors that may lead to the development of an infectious mastitis, the main medical cause for early weaning. This process of mammary bacterial disbiosis is characterized by a high increase in the concentration of the etiological agent/s ($>10^4$ CFU/ml milk) and the disappearance of other bacteria that are usually present in human milk. Such microbial alteration is the main responsible for the inflammatory state and the obstruction of the mammary ducts.

The main etiological agents of mastitis belong to the genera *Staphylococcus* (*S. aureus*, *S. epidermidis*), *Streptococcus* (*S. mitis*, *S. salivarius*…) and, at a lower extent, *Corynebacterium*. *S. aureus* is the main etiological agent of acute mastitis, a process easily diagnosed and generally characterized by an intense local inflammation accompanied by systemic flu-like symptoms, and that, eventually, may lead to breast abscess. These are the prototypical cases of "mastitis" but, actually, they represent a small percentage of infectious mastitis. A higher number of cases can be classified as subacute mastitis, a process characterized by a pricking or needle-like pain or a burning breast pain. They are usually due to overgrowth of coagulase-negative staphylococci or *viridans* streptococci in the mammary ducts, and are widely underrated due to the absence of breast redness and systemic symptoms. Independently of the species, mastitis-causing strains generally share some properties, such as ability to form dense biofilms, resistance to methicillin and other clinically-relevant antibiotics, and mechanisms to evade the action of the immune system. On the other hand, despite the existence of unjustified beliefs, the incidence of yeasts as an agent of infectious mastitis (or even sore nipples) is extremely low.

Milk cultures and antibiograms would facilitate a rational treatment of the different types of mastitis but microbiological analyses of human milk are not performed routinely. In this context, the clinician should be able to associate each type of mastitis with its characteristic