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Staphylococcus aureus Mastitis: Cause, Detection, and Control

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Summary

Staphylococcus aureus (S. aureus) mastitis is extremely difficult to control by treatment alone. To date, successful control is gained only through prevention of new infections and culling of infected animals. *S. aureus* organisms colonize teat ends and/or teat lesions. Spread of infection can occur through milkers' hands, washcloths, teat cup liners, and flies. During milking, irregular vacuum fluctuations can force bacteria up into the teat canal, leading to the potential for new infection. If not culled, infected cows must be segregated from the milking herd and milked last, or milked with separate milking units. A backflush system may help reduce bacterial numbers within the liners, but rinsing units by hand is certainly not recommended.

Introduction

Staphylococcus aureus causes one of the most common types of chronic mastitis. Though some cows may flare up with clinical mastitis (especially after calving) the infection is usually subclinical, causing elevated somatic cell counts (SCC) but no detectable changes in milk or the udder. The bacteria persist in mammary glands, teat canals, and teat lesions of infected cows and are contagious. The infection is spread at milking time when *S. aureus*-contaminated milk from an infected gland comes in contact with an uninfected gland, and the bacteria penetrate the teat canal. Once established, *S. aureus* infections do not respond well to antibiotic therapy and infected cows must be segregated or culled from the herd. In some herds with SCC below 200,000/mL, dairy managers have not been able to eradicate *S. aureus* despite the use of standard milking-time hygiene techniques (Roberson et al. 1994).

Recently published work has shown that 3 percent of all animals are infected with *S. aureus* (Schukken et al. 2009). However, *S. aureus* represents 10 to 12 percent of all clinical mastitis infections (Tenhagen et al. 2009). Interestingly, cows infected with *S. aureus* do not necessarily have elevated SCC. During 1978-1980, nearly 27,000 milk samples from 28 herds were aseptically collected. Culture results showed 10 percent of cows were infected with *S. aureus* (Jones et al. 1984). Only 60 percent of the infections were found in cows producing milk with SCC greater than 200,000/mL.

Heifers are also a reservoir for *S. aureus* infections. In several research trials, 12 to 15 percent of first-lactation cows were found infected with *S. aureus* at calving (Boddie et al. 1987; Trinidad, Nickerson, and Adkinson 1990; Trinidad, Nickerson, and Alley 1990). Furthermore, infected heifers left untreated produce 10 percent less milk in early lactation when compared with those who received dry cow antibiotic treatment prior to calving (Owens et al. 1991). Many animals remain infected throughout the first lactation and act as reservoirs for infecting other cows in the herd. Although as many as half of the cows with high SCC may be infected with *S. aureus*, SCC alone are not sensitive enough to positively diagnose *S. aureus* infections.

Damage Caused by *Staphylococcus aureus* Mastitis

S. aureus bacteria produce toxins that destroy cell membranes and can directly damage milk-producing tissue. White blood cells (leukocytes) are attracted to the area of inflammation, where they attempt to fight the infection. Initially, the bacteria damage the tissues lining the teats and gland cisterns within the quarter,

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which eventually leads to formation of scar tissue. The bacteria then move up into the duct system and establish deep-seated pockets of infection in the milk secreting cells (alveoli). This is followed by the formation of abscesses that wall-off the bacteria to prevent spread but allow the bacteria to avoid detection by the immune system. The abscesses prevent antibiotics from reaching the bacteria and are the primary reason why the response to treatment is poor.

However, bacteria can also escape the killing effects of some antibiotics by hiding within neutrophils (white blood cells) and other host cells. As the neutrophils attempt to remove bacteria, many organisms survive and become dormant within them, preventing contact with antibiotics. When the white blood cells die (usually in one to two days) the bacteria are released to resume the infection process.

During infection, destruction of alveolar and ductal cells reduces milk yield. These damaged cells may combine with leukocytes and clog the milk ducts that drain the alveolar areas, contributing to further scar tissue formation, occlusion of ducts, and decreased milk production. The ducts may reopen at a later time, but this usually results in a release of *S. aureus* organisms to other areas of the mammary gland. The spread of *S. aureus* within the gland results in the formation of additional abscesses that can become quite large and detectable as lumps within the udder.

Though most cases of *S. aureus* mastitis are subclinical, chronic cows usually have high SCC, abnormal mammary tissue, and recurrent cases of clinical mastitis. Clinically infected quarters often show moderate swelling and visible clots (chunks) in the milk, especially in forestrippings. Acute *S. aureus* infections generally develop late in the lactation. However, the clinical symptoms (udder swelling or hardness, changes in appearance of milk) do not show up until calving or early in the next lactation. It becomes difficult to successfully treat an infection because drugs are not able to penetrate to all infection sites and because the bacteria can avoid contact with antibiotics while residing inside leukocytes. Many strains of *S. aureus* have acquired antibiotic resistance – the ability to produce an enzyme that inactivates penicillin-based and other antibiotics – rendering the treatment ineffective. The development of antibiotic resistance during treatment with some beta-lactam antibiotics, e.g., penicillin, is an additional reason for therapy failures.

Transmission of *Staphylococcus aureus* Infections

The major reservoirs of *S. aureus* are infected udders, teat canals, and teat lesions, but these bacteria also have been found on teat skin, muzzles, and nostrils. The bacteria are spread to uninfected quarters by teat cup liners, milkers' hands, washcloths, and flies. Staphylococci do not persist on healthy teat skin but readily colonize damaged skin and teat lesions. The organisms multiply in infected lesions and result in increased chance of teat canal colonization and subsequent udder infection.

Heifers infected during gestation that carry infections through calving represent an important reservoir from which *S. aureus* can spread to uninfected herd mates. There is considerable debate surrounding the route of *S. aureus* infection in heifers prior to first calving, but calves fed colostrum from an *S. aureus*-infected dam is a likely source. Early work suggested *S. aureus*-infected colostrum was not a culprit for first-calf heifers calving with the infection (Barto et al. 1982). However, later work did show a positive correlation between feeding *S. aureus*-infected colostrum to a calf and that calf then calving with *S. aureus* mastitis (Roberson et al. 1998). Though the data is limited, if an *S. aureus* problem exists on a farm, careful colostrum selection, e.g., pasteurization, is certainly one area to consider. Clearly, good mastitis control programs will address the presence of this disease in heifers.

Detection of *Staphylococcus aureus* Mastitis

Culture of bulk-tank milk is easy, economical, and an important aid in monitoring bacterial counts in milk. However, this does not replace an individual cow culture. Bulk-tank cultures can be used to monitor the status within a herd. For example, in a herd with no history of contagious mastitis, a positive culture or series of cultures would warn the producer to examine individual cows. When troubleshooting a herd with a high SCC problem, we recommend culturing all high-SCC cows (more than 400,000 cells/mL). If that includes more than 20 percent of the herd, then it is best to culture the 20 to 30 cows with the highest SCC. These results will indicate the type of mastitis problem in a given herd, which allows for more appropriate recommendations based on the individual farm results.

Alternatively, the producer can use the California Mastitis Test (CMT) on cows with elevated Dairy Herd Improvement program (DHI) SCC to determine which quarters may be infected, then selectively culture positive quarters. Herds not on test can also use the CMT periodically on all cows to identify quarters for culturing. This is an excellent starting point for identifying positive cows and moving them to a separate group. It is important to identify infections early in order to prevent spread to other animals and increase chances of a successful treatment.

Control of *Staphylococcus aureus* Mastitis

The most effective ways to prevent new infections are to eliminate conditions that expose teat ends to bacteria and reduce the possibility of spread from cow to cow – many of which are discussed below.

1. Hygienic procedures

    a. Milkers should always wear gloves and change them frequently, especially when dirty or after stripping an infected animal. It is recommended to forestrip five squirts of milk from each quarter and check for abnormal milk or flakes.

    b. Dirt should be brushed off teats with the use of a dry, single-use towel. Water should not be used as part of any milking procedures, even if a sanitizing solution is added. Sanitizers do not maintain activity throughout a milking, and water can introduce pathogens that are very difficult to cure.

    c. A commercially available predip should be applied with a dipper or cup and given 30 seconds of contact time. Sprayers can be used, but proper coverage is difficult to attain, especially on the teats furthest from the milker. Foamers are also commonly used, but caution must be taken to ensure that adequate parts per million of the active ingredient reach the teat end and teat skin.

    d. A separate paper or cloth towel should be used to dry teats and scrub teats five times or for 20 seconds (Rasmussen et al. 1992). Towels must not be used on more than one cow.

    e. Milking units should be attached 90 seconds after first tactile stimulation (stripping or wiping, whichever comes first).

    f. Teat ends should be examined for evidence of chapping, cracks, or lesions, which may harbor mastitis-causing bacteria.

    g. An effective postmilking teat dip should be applied after milking, ensuring that the entire barrel of each teat is covered. At the end of each milking any teat dip left in the dip cup should be discarded and cups should be rinsed with water and allowed to dry.

2. Milk *S. aureus*-infected cows separately and last

    a. Cows with clinical mastitis, *S. aureus* infections, or those that have been treated with antibiotics should be milked last, or milked with separate milkers. Segregation of *S. aureus*-infected cows has proven to significantly reduce the prevalence of *S. aureus* mastitis and bulk-tank SCC (Wilson et al. 1995). In many instances, if the animals cannot be culled, they must be separated to prevent the spread of contagious pathogens and a rapid and negative impact on bulk-tank SCC. In the odd case in which a separate group absolutely cannot be made, it is best to identify these animals with a leg band to ensure proper care in the parlor, such as changing gloves after prepping.

    b. Uninfected, first-lactation cows should be milked before older cows carrying subclinical mastitis infections. Since heifers may be infected at calving and because cure rates are highest in younger animals, aseptic milk samples should be collected and cultured shortly after calving.

3. Milking equipment

    a. *Staphylococcus aureus* infections can occur during milking when organisms penetrate the teat canal. Irregular vacuum fluctuations caused by liner slips, flooded lines, etc., may cause a backflow of milk against the teat end. With sufficient force, bacteria can be propelled up into the teat canal and teat cistern. Therefore, properly functioning equipment is essential in preventing new infections.

    b. Conditions that are associated with high-impact force against the teat end – including liner slips, excessive temporary vacuum losses, low vacuum reserve, inefficient vacuum regulation, and

abrupt milking unit removal – should be minimized. Teat cups should not be removed from the cow until the vacuum has been shut off. Research has shown that slipping teat cup liners may cause 10 to 15 percent of new mastitis infections. Liner slippage early in milking often results from a low vacuum level, blocked air vents, or restrictions in the short milk tube. Liner slippage late in milking is commonly caused by poor cluster alignment, uneven weight distribution in the cluster, or poor liner condition. Incomplete milking can be caused by poor type or condition of liner, a mismatch between claw inlet and short milk tube, clusters that are too light, clusters that do not hang evenly under the udder, or high milking-vacuum levels (Halleron 1997).

c. Regular, preventive maintenance is essential for milk quality and mastitis prevention. Vacuum controllers (regulators), pulsators, and air filters need to be cleaned monthly. All rubber components must be changed according to the manufacturer's instructions. Rubber that is cracked, flattened, or otherwise deteriorated should be replaced even if the recommended life of the product has not been reached. The milking system should be evaluated every three months or 500 hours of operation, to include the following tests: vacuum reserve, vacuum level, vacuum recovery time, vacuum regulator response, pulsator graphs, and stray voltage. Many of these tests need to be conducted during milking time and not between milkings.

4. Antibiotic treatment of *S. aureus* cows

Antibiotic treatment will not control this disease but it may, in certain cases, shorten the duration of the infection. Treatment effectiveness decreases as the cow becomes older and even as the first lactation progresses. Cure rates were 34 percent when 89 cows in 10 Dutch herds were treated for subclinical *S. aureus* mastitis (Sol et al. 1997). The results showed that the probability of cure was lower in older cows with high SCC and in cows infected in hindquarters during early and midlactation. *S. aureus* infections were found in 36 percent of clinical mastitis cases in Finnish herds (Pyorala and Pyorala 1997). Of these, 39 percent responded to treatment. Cows with an SCC of less than 1 million were more likely to cure an infection compared with those over the cut-off point. Successful treatment during lactation is greater if detected and treated early, whereas the response is lower when treating chronic infections. Use of a strip cup or similar device is strongly recommended for detecting abnormal milk. New clinical infections should be treated promptly and appropriately, especially in first-lactation cows. Tissue damage can be minimized if animals are treated during the early stages of infection. As always, consult a veterinarian regarding off-label treatment options. The use of DHI program SCC records in addition to visual observation of forestripped milk and milk culture results will indicate effectiveness of treatment.

Many researchers have looked at the efficacy of pirlimycin treatment both in heifers prior to calving and in all animals as an extended therapy treatment during lactation. According to the manufacturer, pirlimycin is one of the most effective compounds against *S. aureus* because its chemical nature allows it to penetrate mammary tissues. In heifers, a single tube of pirlimycin treatment in each quarter six to 12 days prior to calving significantly reduced *S. aureus* infections at calving (Roy et al. 2007). Furthermore, mastitis data presented to the FDA suggests that two tubes, administered 24 hours apart to infected quarters of cows during lactation, resulted in a cure rate of 36.6 percent, whereas only 1.1 percent of nontreated controls recovered spontaneously. In field cases, the rate of cows cured during lactation increased to 49.4 percent. However, trials using the same treatment scheme at Louisiana State University and Iowa State University found cure rates of only 12 percent or less for chronically infected *S. aureus* cows during lactation.

Single-quarter, extended therapy with repeated label doses of pirlimycin has been examined as a means of providing drug levels beyond the expected life of the leukocytes that naturally fight off this infection. This protocol has been widely adopted for new intramammary infections with *S. aureus*, as it increases cure rates. Four-quarter extended treatment with repeated label doses will provide adequate therapeutic concentrations for many *S. aureus* bacteria. A cure rate of 50 percent at four weeks after treatment was found in more than 100 treated cows (Belschner et al. 1996). Whether these cure rates justify the additional expenses and effort, not to mention the potential risk of extra-label use and antibiotic residue, is unknown.

5. Dry cow therapy

Dry cow therapy (DCT) is more effective in eliminating infections than lactating treatment. However, DCT is not effective if the infections have become chronic by the end of lactation. When cows are not given DCT, spontaneous cures have been very low. DCT is cost-effective (Kirk et al. 1997). When a cow is dried off, it is recommended to treat all quarters with a commercially available DCT. Follow these steps when dry treating:

a. Cows must be milked out completely, teats dipped in postmilking teat dip and blotted dry after 30 seconds contact time.

b. Teat ends should be scrubbed with alcohol pads before partially inserting the antibiotic tube into the teat (1/8 inch). Although internal teat sealants do not prevent the spread of *S. aureus* infections, the commercially available product does help to reduce new environmental infections. Internal teat sealants are especially useful during the first two weeks and the last seven to 10 days of the dry period when cows are most susceptible to mastitis pathogens. This increase in susceptibility is in part due to the stress associated with drying and the precalving period.

c. Teats should be dipped again after treatment, prior to turning the cows out into a clean, dry environment.

6. Pregnant heifers

New infections are commonly found in heifers, either at calving or in early lactation. Up to one-third of these infections are caused by *S. aureus*. Often these *S. aureus* infections, if untreated, become clinical and recur throughout the first lactation and into the second lactation. Furthermore, these infections increase the chance of contagious spread to other animals in the herd. Several management practices can be used on heifers prior to calving to eliminate infections before the start of lactation.

Administration of dry cow therapy to heifers has been evaluated in several Louisiana studies. A dry cow product containing penicillin and dihydrostreptomycin was administered during the first, second, or third trimester of pregnancy in 35 bred heifers from four herds. Although prevalence of infection and SCC were reduced by treatment in all three groups of heifers, heifers dry-treated during the second trimester of pregnancy demonstrated the greatest reduction in mastitis and SCC at calving (Nickerson et al. 1995). It is recommended that heifers be treated with dry cow treatment at 60 days before expected calving date. Teat ends should be properly cleaned and disinfected before and after treatment. It is important to check milk for the presence of antibiotic residue at three to five days after calving or before the milk is allowed in the bulk tank.

In Tennessee, a lactating cow antibiotic treatment containing either cloxacillin or cephapirin was administered to heifers at seven to 10 days before expected calving (Oliver et al. 1992). Cephapirin gave better treatment results than cloxacillin but resulted in antibiotic residue in milk at three days after calving. Treating heifers with cephapirin 14 days before expected calving eliminated the residue problem. It is therefore recommended that heifers be treated with lactating cow mastitis treatment at 14 days before expected calving, using precautions indicated under the preceding section about dry cow therapy.

7. Precautions at calving

Many mastitis infections (not specifically *S. aureus*) originate in the peripartum period. A well-drained pasture is preferred as a calving area, with no access to ponds, swampy areas, or drainage ditches. A clover-grass sod is desired, in contrast to fescue or muddy, beaten-up lots. Lots and pastures should be managed to prevent muddy areas where cattle would lie down. Filthy, damp, or muddy pens, lots, or pastures continually expose the teat end to a barrage of bacteria. Pens should be well-bedded, clean, dry, and comfortable. Selenium-vitamin E supplementation or injections at two to three weeks before expected calving have been shown to reduce mastitis after calving. Vitamin E levels of 1,000 IU/day during the dry period and 500 IU/day during lactation are recommended by the National Research Council. Other minerals and vitamins shown to reduce the incidence of mastitis include vitamin A/beta-carotene, copper, and zinc. By testing animals to identify micronutrient deficiencies, providing a balanced ration, avoiding poorly fermented silages, and including dietary supplementation of vitamin E and selenium, proper nutrition can be maintained to reduce incidence of mastitis.

Conclusions

The best treatment for *S. aureus* mastitis is prevention. Recommendations to prevent spread of contagious mastitis pathogens include:

1. Do not milk cows and heifers with the same teat cup/claw unit used to milk mastitis-problem cows.

2. Segregate *S. aureus*-infected cows into one group and milk them last. Another alternative is to sort out infected cows before each milking and restrain them in an isolation pen until all other cows have been milked. In a short period of time, these cows will become trained to sort.

3. If heifers or cows are purchased, segregate them until milk samples can be cultured and their mastitis pathogen status can be determined. If possible, examine the DHI SCC of cows before agreeing to make the purchase.

4. Cull infected cows, especially those with additional problems. Often, it is not economically feasible to cull a *S. aureus*-positive cow that produces 80-90 pounds of milk. However, any cow that has had clinical mastitis in the same quarter for three or more occasions, or any cow whose milk has been withheld from shipment for more than 28 days during the current lactation, should be considered for culling from the herd. It is important to keep mastitis records, including cows and quarters treated and treatment used.

5. Place *S. aureus*-infected cows on a do-not-breed list. Cull them when their milk is no longer needed (e.g., to make base) or they have been in milk for 305 days.

6. Biting flies traumatize the teat end. Flies also carry a number of mastitis-causing organisms that can colonize these teat lesions. Elimination of fly-breeding sites is one aspect of fly control. Flies breed in decaying feed or manure that has accumulated in exercise yards, calf pens, and box stalls. Another option for control is use of back-rubbers, feed additives, and ear or tail tags with insecticide. In one trial the use of tail tags containing insecticide resulted in only one of 100 tagged heifers with a mastitis infection, compared to 18 of 100 untagged heifers.

7. It is unclear whether waste milk from *S. aureus*-infected cows is a problem. One study showed a positive association between feeding *S. aureus*-infected colostrum and heifers calving with *S. aureus* mastitis (Roberson et al. 1998). Therefore, not feeding contaminated waste milk is a simple precaution that will most likely reduce a possible reservoir of bacteria.

8. Many *S. aureus* vaccines have been designed and tested over the years. One research study indicated little-to-no effect on new intramammary infections following vaccination with a commercial product (Middleton et al. 2009). However, another investigation demonstrated a 61 percent reduction in *S. aureus* mastitis using the same product (Nickerson et al. 2008). Future research endeavors may focus on alternative vaccine options that will successfully prevent *S. aureus* mastitis.

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