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Cross-Infection Between Cats and Cows: Origin and Control of *Streptococcus canis* Mastitis in a Dairy Herd

L. L. Tikofsky[1] and R. N. Zadoks[1,2]
[1]Quality Milk Production Services, and
[2]Department of Food Science, Cornell University, Ithaca, NY 14850

ABSTRACT

Group G streptococci in animals usually belong to the species *Streptococcus canis* and are most commonly found in dogs and cats. Occasionally, *Strep. canis* is detected in milk from dairy cows. An outbreak of *Strep. canis* mastitis in a dairy herd is described. Based on results from bacterial culture and ribotyping, a cat with chronic sinusitis was the most likely source of the outbreak. Subsequent cow-to-cow transmission of *Strep. canis* was facilitated by poor udder health management, including use of a common udder cloth and failure to use postmilking teat disinfection. Infected cows had macroscopically normal udders and milk, but significantly higher somatic cell counts than *Strep. canis*-negative herd mates. The outbreak was controlled through antibiotic treatment of lactating cows, early dry-off with dry cow therapy, culling of infected animals, and implementation of standard mastitis prevention measures. Cure was significantly more likely in dry-treated cows (87.5%) and cows treated during lactation (67%) than in untreated cows (9%). Whereas mastitis due to group G streptococci or *Strep. canis* in dairy cows is usually limited to sporadic cases of environmental (canine or feline) origin, this case study shows that crossing of the host species barrier by *Strep. canis* may result in an outbreak of mastitis if management conditions are conducive to contagious transmission. In such a situation, measures that are successful in control of *Strep. agalactiae* can also be used to control *Strep. canis* mastitis.
(**Key words:** *Streptococcus canis*, mastitis, host species barrier, group G streptococcus)

Abbreviation key: BMSCC = bulk milk somatic cell count, **DCT** = dry cow treatment, **GGS** = group G streptococcus, **LCT** = lactating cow treatment, **MRSA** = methicillin-resistant *Staphylococcus aureus*, **QMPS** = Quality Milk Production Services.

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Corresponding author: Linda L. Tikofsky; e-mail: lg40@cornell.edu.

INTRODUCTION

Streptococci are a common cause of mastitis in dairy cows. In many areas, contagious mastitis caused by *Streptococcus agalactiae* has largely been controlled (Loeffler et al., 1995; Andersen et al., 2003), but other streptococci, specifically *Streptococcus dysgalactiae* and *Streptococcus uberis*, continue to be highly prevalent throughout the world (Wang et al., 1999; Zadoks et al., 2004). Identification of streptococcal species in mastitis diagnostics is usually based on hemolytic patterns, esculin splitting, and the CAMP reaction (National Mastitis Council, 1999). Serological grouping in accordance with the Lancefield system can also be used for typing of some streptococcal species from milk, most importantly for group B streptococci or *Strep. agalactiae* (Facklam, 2002). In addition, group G streptococci (**GGS**) are occasionally found in bovine milk samples.

Mastitis caused by GGS in dairy cows is relatively rare. In herd surveys from Iowa and New York State, the prevalence was 0.7% of 455 streptococcal cultures from 72 herds (McDonald and McDonald, 1976), 4 of 250 dairy herds (1.6%) (Hamilton and Stark, 1970), and 125 of 105,083 surveyed cows (0.1%) (Wilson et al., 1997). However, herd outbreaks due to GGS have been reported from many places, including Washington, DC (Miller and Heishman, 1940); Ontario, Canada (Barnum and Fuller, 1953); Denmark (Romer, 1948); New York (Hamilton and Stark, 1970); Pennsylvania (Eberhart and Guss, 1970); Israel (Bergner-Rabinowitz et al., 1981); Louisiana (Watts et al., 1984); The Netherlands (O. C. Sampimon, personal communication, 2003); and Italy (P. Moroni, personal communication, 2003). In 1986, the name *Streptococcus canis* was coined (Devriese et al., 1986) to describe GGS found in dogs and cattle. Animal GGS or *Strep. canis* differed in physiological, biochemical, and DNA hybridization characteristics from human GGS isolates which belong to the species *Strep. dysgalactiae* spp. *equisimilis* (Devriese et al., 1986). In fact, *Strep. canis* is more closely related to *Streptococcus pyogenes* or group A streptococcus than to GGS of humans (Facklam, 2002). In dogs and cats, *Strep. canis* is found on skin and mucosa of asymptomatic carriers and in many pathological conditions, in-

cluding infections of the skin, urogenital, and respiratory tract, polyarthritis, abortion, septicemia, canine streptococcal toxic shock syndrome, and necrotizing fasciitis (Devriese et al., 1986; DeWinter et al., 1999; Hassan et al., 2003).

In this paper, we describe an outbreak of bovine mastitis caused by *Strep. canis* in a New York State dairy herd. The source of infection and routes of pathogen transmission are identified through bacteriology, molecular typing of GGS isolates, and analysis of herd management. The impact on affected cattle and the outcome of antibiotic treatments and management changes that were instituted to curb the outbreak are presented. This case study serves both as a suggestion on how to deal with *Strep. canis* in dairy cattle, and as an example of the combination of traditional herd-health approaches with modern DNA-based methods for problem solving in a situation where crossing of the host-species boundary by a pathogen resulted in an unusual disease outbreak.

MATERIALS AND METHODS

Case History

In April 1999, Quality Milk Production Services (**QMPS**) personnel were requested to visit a dairy herd in central New York State for the first time to perform a whole-herd mastitis screening survey. The herd, consisting of 90 lactating head of Holstein-Friesian cattle with mean 305-d milk production of 6700 kg/cow, was in danger of losing its milk market because 2 of the last 4 official bulk milk somatic cell counts (**BMSCC**) were greater than 750,000 cells/mL. Bulk milk SCC had been 173,000/mL in December 1998, but counts had risen steadily since that time. The most recent BMSCC was 1,800,000/mL. Standard plate count was 41,000 cfu/mL.

The herd was housed in a tie-stall barn with concrete floors. Stalls were covered with rubber mats and minimal amounts of old hay. The milking system included a 5.08-cm (2-inch) pipeline around the barn with 8 milking units. Cows were milked twice daily by the producer and his wife. Cows' teats were forestripped and then washed with water and a common towel. Teats were not dried before attachment of the milking unit. Postmilking teat dip was not applied, and gloves were not worn by the producer or his wife. Cows were milked once a day for 3 d before dry off and then treated in each quarter with a long-acting penicillin-dihydrostreptomycin treatment. The herd had been closed for 40 yr and had always been housed at the same location. Several cats had access to the barn.

A second visit followed in May 1999. Bulk milk SCC on the latest test was 560,000/mL. Quarter samples for bacteriologic culture were collected from those lactating cows that were diagnosed with GGS at the whole herd survey in April. From the remaining 50 cows, composite cow milk samples were collected. In addition, swabs or samples were collected from milking unit inflations, nasal secretions, and hand surfaces of the producer's wife, udder wash towels, dip cups, and feline nasal and anal secretions. Personnel from QMPS returned to the herd in July and October 1999, for whole herd surveys. Bulk milk SCC were 560,000/mL and 470,000/mL, respectively, at those surveys. Because BMSCC was consistently below the legal limit and the producer planned to sell the herd in 2000, no further treatments or surveys were undertaken.

Milk Samples, Bacteriology, and SCC

Composite milk samples from each lactating cow were collected aseptically into sterile vials, in accordance with National Mastitis Council guidelines, at the morning milking. Samples were cooled rapidly and transported to the laboratory for immediate bacteriologic culture. Aliquots (0.01 mL) of each sample were plated on trypticase soy agar plates containing 5% sheep blood and 0.1% esculin (Becton Dickinson, Sparks, MD). Plates were incubated aerobically at 37°C and examined for growth at 24 and 48 h. Colonies were presumptively identified as streptococci by colony morphology, hemolytic patterns, and esculin reaction, and were confirmed by Gram stain and catalase-negative reaction. Representative colonies were tested for the CAMP reaction. Biochemical tests were performed on representative isolates with the API 20 Strep system (BioMèrieux, Hazelwood, MO), and serologic grouping was accomplished on all streptococcal isolates with the PathoDx latex agglutination system following the manufacturer's recommendations (Diagnostic Products Corporation, Los Angeles, CA). Based on this method, isolates could be identified as GGS, without differentiation between *Strep. canis* and *Strep. dysgalactiae* spp. *equisimilis*. Swab samples were inoculated in Todd-Hewitt broth upon collection and taken back to the laboratory for processing within a few hours. In the laboratory, samples were incubated for 3 to 4 h in a water bath at 37°C. Swabs were subsequently streaked onto trypticase soy agar plates containing 5% sheep blood and 0.1% esculin. Plates were processed and evaluated as described for milk samples.

Additional composite milk samples that were collected during the second herd survey (May 1999) were used to measure SCC (Fossomatic FC; Foss, Eden Prairie, MN). Antibiotic sensitivity of a limited number of isolates (n = 5) was determined using the Kirby-Bauer agar disk diffusion method in accordance with standards from the National Committee for Clinical Labora-

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