Gangrenous mastitis in a ewe flock

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Introduction

Mastitis in ewes can result in significant losses of both ewes and lambs in New Zealand (West, Bruere and Ridler 2009). It is considered the most important mammary disease of sheep even though it may be of minor importance to the New Zealand sheep industry at large. Although this may be the case, the economic impact is still unknown and is potentially a serious loss due to reduced lamb growth rates and survival, because of decreases in milk yield and quality, as well as increased ewe culling rates (Quinlivan 1968, Barber 2015). Mastitis by definition is inflammation of the mammary gland tissue. Often mastitis is caused by an infection of bacterial origin, but can also be due to other types of pathogens or trauma (Barber 2015, Menzies 2000). Infectious causes of mastitis can cause disease resulting in a variety of clinical signs and levels of severity. The most common cause of acute mastitis in sheep in New Zealand and Australia is due to the bacteria *Staphylococcus aureus* (*S. aureus*) (Barber 2015, West, Bruere, and Ridler 2009, Clark 1972, Ekdahl 1972). The highest incidence of acute clinical mastitis occurs during spring when the ewe is lactating (Quinlivan 1968) with *S. aureus* occurring most often in the third and fourth weeks postpartum (Jones, Lanyon and Watkins 1989). While *S. aureus* is the main cause of gangrenous or black mastitis in ewes in New Zealand, there are other organisms that may cause similar disease: *Mannheimia spp* and *E. coli* are the second and third most common diagnosed causes of mastitis in New Zealand (Ekdahl 1972, Barber 2015, Quinlivan 1968, Jones, Lanyon and Watkins 1989). Acute gangrenous mastitis is a disease primarily identified when ewes become lethargic, febrile followed by hypothermia, dehydration, anorexia and a swollen, painful blue coloured udder. Very few descriptions exist of gangrenous mastitis outbreaks in ewes in New Zealand – most reports (Allen 2011, Clark 1972, West, Bruere and Ridler 2009) are anecdotal and accompanied by little investigation. One survey (Quinlivan 1968) of clinical mastitis in sheep reported an incidence rate of 1.4% during August and September in the North Island and an overall clinical mastitis incidence of 1.65% over a two year period. *S. aureus* made up 44% of these cases when only accounting for acute forms of mastitis during the spring pre-weaning months (Quinlivan 1968). Other studies describe the incidence of acute mastitis in sheep to range from 0.65% (Clark 1972) to 10% (Jones, Lanyon and Watkins 1989). Mortality rates can be high with acute gangrenous mastitis caused by *S. aureus*, varying from 0.2% to 80% in untreated toxemic ewes (Menzies 2000, Clark 1972). The wide range of reported mastitis rates indicates the need for more surveillance amongst New Zealand ewe flocks to allow for an assessment of the impact on production and health in New Zealand and the consequential economic losses due to outbreaks.

Presentation

An outbreak of gangrenous mastitis occurred on a Canterbury meat and wool production sheep farm in spring 2015. The farm consists of 227 hectares made up of perennial ryegrass and clover permanent pasture with chicory and plantain paddocks as well. Shelter belts include hedges of both *Pinus radiata* and *Cedrus deodara* pines. Rare sporadic cases of gangrenous mastitis in ewes had occurred over the last 50 years of farming on this property. The farm purchased 159, five year old and older Corriedale ewes, with 257 lambs at foot, ranging from three to five weeks old on 13 September 2015. The animals had travelled a distance of maximum 15 kilometres by transport truck. On arrival, the animals were treated with the farm’s standard protocol for newly purchased stock: triple active quarantine drench; crutching of the ewes; and tailing, clostridial vaccination, insect growth regulator application, and treatment with injectable selenium and B12 for the lambs.
Outbreak

In the five days following their arrival, 9 of 159 (5.7%) ewes died. Three ewes were found recumbent in the yard on the day of arrival and treated for milk fever by the farmer, using calcium borogluconate, dextrose, and magnesium hypophosphite hexahydrate subcutaneously (SC). All three ewes appeared to respond positively to treatment but died on 15 September 2015. Six more ewes died over a three day period, between 16 and 18 September 2015, or were humanely euthanized by the farmer due to recumbency and lack of response to milk fever treatment. Some of these ewes were noticed to have a black ventrum and udder, and were recumbent or showed hind limb paresis. However, not all ewes that died were noticed to have these signs.

A visit by the veterinarian (author) was made on 18 September 2015. A postmortem examination was performed on the ninth ewe that had been euthanized by the farmer just prior to the farm visit. Black discolouration and necrosis of the skin of the entire udder and ventrum was noted extending cranially to the level of the xiphoid process, where serosanguinous fluid oozed from these cut skin surfaces. No other gross significant findings were noted. Fresh and fixed issues were submitted to the laboratory for culture and histological examination.

A single live ewe from this recently purchased flock was also presented, having been identified by the farmer that day with a black udder. On physical examination she was found to be bright, alert and responsive, eating and walking stiffly in the back end. The entire udder and ventrum was dark purple in colour, cold and very painful when palpated. The right half of the udder had a clotted bloody secretion and the left udder half had normal looking milk. A milk sample was taken, using standard milk sampling techniques, from both udder halves and pooled. The milk sample and fresh tissue from the postmortem ewe were immediately submitted to the laboratory for aerobic culture.

Treatment

An off label dosage (1,200,000 IU) of procaine penicillin was administered intramuscularly (IM) to the ewe along with a single dose (40mg) of meloxicam given SC as an off label use in sheep. The farmer continued treatment of this ewe two days later using the same dosage (1,200,000 IU) of procaine penicillin IM. The ewe survived following treatment but was observed to lose significant milk production. In the event of further cases, the farmer was advised to give three doses of procaine penicillin IM, repeated every 48 hours, and a single dose of meloxicam SC. No additional cases were identified and thus none were treated.

Outcome

Samples of mammary gland, liver, heart muscle, lung, kidney, lymph node and spleen were all submitted for histological examination. The mammary gland revealed neutrophil infiltration, thrombosis of small vessels, large numbers of large coccoid bacteria, and interlobular septa were widened by oedema and fibrin effusion. Mainly large gram-positive cocci and some smaller gram-positive cocci were identified. There was nephrosis of the kidney and plasma cells were found in large numbers within the
lymph node. Lastly, there were many neutrophils in the red pulp, which surrounded the white pulp, of the spleen.

A heavy and predominant growth of *S. aureus* was cultured from fresh tissue samples, while a heavy growth of *S. aureus* was also obtained from milk samples. All isolates were sensitive to all antibiotics (amoxclav, cephalothin, erythromycin, oxacillin, penicillin, tetracycline, trimethsulpha) in the antimicrobial sensitivity profile. The incidence of disease caused by *S. aureus* was 6.3% (10/159). The case mortality rate was 90% (9/10).

**Discussion**

While the precipitating factors that resulted in this outbreak are unknown, several risk factors can be identified that may play a part in the onset of acute and peracute mastitis events. Similar clinical signs to those seen in this outbreak have been described in other reports (Barber 2015, Quinlivan 1969, West, Bruere, and Ridler 2009). *S. aureus* is a bacteria that causes infection via the route of the teat canal and is also known to be found on the udders of healthy ewes. In fact, even flocks with low incidence of clinical mastitis can be found to have up to 8% culture positive for *S. aureus* on healthy udders. (West, Bruere, and Ridler 2009) *S. aureus* causes peracute and acute mastitis due to the release of the very potent toxins called alpha, beta and gamma-haemolysin, which all contribute to the pathogenesis of infection (Ekdahl 1972). The alpha-toxin is dermonecrotic and vasoconstrictive causing marked gangrenous necrosis of the udder tissue and ventral abdomen (Divers and Peek 2008). The extent of the discolouration of the skin of the udder is also notable, as it was in these affected ewes, where it starts in the mammary gland and continues up the ventrum and throughout the region of the groin. Red, serum-type secretions with or without clots or fibrin and gas is typical of *S. aureus* infected glands (Menzies 2000, Quinlivan 1968, Barber 2015). Several strains of *S. aureus* exist that cause a variety in severities of disease. In one study (Vauto 2009) the more virulent strain, causing gangrenous mastitis in dairy ewes, was found to be genetically different from the strain causing sub-clinical *S. aureus* mastitis in ewes, but they also shared a lot of genetic similarities also. Interestingly, these differences arose following sub-clinical disease, which implies that the strain causing gangrenous mastitis emerged genetically from or following the sub-clinical type (Vauto 2009). It is often reported that if the ewe survives an acute case of *S. aureus* within a few days, the gangrenous udder tissue may slough off and heal by second intention and likely acquire secondary bacterial infections of the wound and fly strike (Menzies 2000, West, Bruere, and Ridler 2009, Barber 2015). Fortunately, the single ewe that survived the disease did not undergo sloughing of the udder tissue and was not identified to acquire secondary health issues. Acute gangrenous mastitis is typically diagnosed on farm by farmers due to lame ewes, changes in colour of the udder, anorexia, and a hollow-appearance of the lambs. Lameness and recumbency is one of the presenting signs that may be most noticeable to the farmer when diagnosing these disease events (Barber 2015). Bacteriologic culture of milk is the most reliable diagnostic method in determining the cause of mastitis infection (Quinlivan 1968). The use of histology to diagnose a *S. aureus*
outbreak in ewes is a confirmatory tool displaying acute inflammation, septicaemia and the presence of bacterial infiltration into tissues. Although this can be useful, histology can be impractical when cost and turn-around time by the lab could be prohibitive to coming to an accurate diagnosis and treatment plan. It was, however, informative by demonstrating how severe \textit{S. aureus} toxaemia can be and how quickly infection can occur. Findings in the mammary gland were representative of acute suppurative and necrotizing mastitis. The kidney had nephrosis, which can be a common finding in sheep that reflects poor renal perfusion from dehydration and shock. The lymph node had large numbers of plasma cells which indicates its response to immune stimulation not of an acute origin. While the presentation of this outbreak was acute in all identified affected ewes, this could represent a more chronic disease process among the flock. The spleen had many neutrophils in the red pulp immediately around the white pulp, which is also a common finding during severe acute infections. With bacteriologic culture and antimicrobial sensitivity being the most useful diagnostic tool currently, an appropriate treatment protocol can be formulated. The treatment protocol used during this outbreak was developed based on the septicemia clinical presentation of the single ewe examined and the gross findings on postmortem examination. First generation beta lactam antibiotics were chosen as the parenteral antimicrobial therapy for these ewes because of its broad spectrum of activity. While \textit{S. aureus} strains may produce beta lactamase, making it resistant to penicillin, the isolate cultured during this outbreak was sensitive to penicillin, so the drug was recommended for any further cases (Quinlivan 1968). Intramammary antibiotics are often recommended in conjunction to parenteral antibiotic administration, but were not utilized as part of this treatment protocol because of the peracute and acute systemic signs caused by this \textit{S. aureus} outbreak. Evacuating the udder frequently of milk has been shown to assist in treating mastitis but may be impractical on meat and wool sheep production farms (Clark 1972). Lastly, the use of non-steroidal anti-inflammatory drugs (NSAIDs) has been shown to decrease the systemic effects of toxins from \textit{S. aureus} (Barber 2015) as well as reducing inflammation. Because of these effects, there is some evidence that NSAIDs can reduce the damage caused to the udder during the acute stage of mastitis and shorten the length of illness (Barber 2015). While only one treated ewe survived in the outbreak described, it is possible that along with the correct antimicrobial given, NSAIDs may have contributed to her survival. A carrier state of \textit{S. aureus} in ewes has been described in some studies (Barber 2015, Quinlivan 1968, Mcdougall 2000), which poses future concerns for this flock over the following lambing season. Without culling ewes that are known to have had clinical mastitis due to \textit{S. aureus}, there is a risk of chronic mastitis and the exposure of uninfected ewes by \textit{S. aureus} carrier animals during lambing. Some studies have shown that administering parenteral antibiotics to ewes over their dry period can reduce the incidence of clinical mastitis following lambing (Quinlivan 1968, Barber 2015). Although the prevalence of clinical mastitis during this outbreak is unknown, the research may suggest that the risk of this flock becoming a highly prevalent carrier of sub-clinical \textit{S. aureus} mastitis is extremely high. Once the ewe’s udder becomes infected with \textit{S. aureus}, this particular bacteria becomes well isolated within the mammary tissue and sheds intermittently in the milk. As teat-ends get damaged, possibly from vigorous lambs suckling, there is a potential route of entry for \textit{S. aureus} into the gland. Lambs could be one method of transmission between ewes, where the bacteria can reside in their oropharynx and cross suckling occurs (Barber 2015, Clark 1972).

Reports of gangrenous mastitis outbreaks describe most risk factors as anecdotal and can occur for a number of reasons. Most surveys completed, report no effects of the age of the ewe on susceptibility or incidence of infection (Quinlivan 1968, Mcdougall 2000). The main source of \textit{S. aureus} is the carrier animal, where it is shed in milk and can infect apparently healthy udders without causing clinical mastitis. It has also been described being shed from nasal secretions of ewes, as well as, lambs carrying the bacteria in
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the oropharynx and cross suckling between infected and uninfected ewes (Quinlivan 1968). Predisposing factors for disease are likely to be multifactorial but cold weather, windy conditions, and rain during lambing have been implicated (Quinlivan 1968). Moderately more wind and rain (total of 31mm) were received compared to historical averages during the month of September, when the outbreak occurred. Air temperatures were also below average of 8.3°C (0.5°C to 1.2°C below average) for the region through the entire month (Brandolin. 2015). These environmental factors may have played a role in increasing the ewe’s susceptibility to infection prior to their shipment to the farm where the outbreak occurred. Cold stress alone can cause spikes in cortisol secretion, reducing immune function and impairing antibody responses. Other predisposing conditions may have contributed to an increased risk of new infections in this flock, such as milk fever. Because those that were treated for milk fever by the farmer were never a true diagnosis of hypocalcaemia, it is unknown whether or not these ewes displayed any clinical signs of mastitis or if they truly died due to milk fever and not peracute mastitis.

Type of feed and feed availability also can play a role in the ewe’s susceptibility to infection. The environmental stressors of extreme weather conditions, such as, drought can also predispose ewes to develop clinical mastitis due to lack of feed stress, decreased milk yield leading to more excessive suckling by lambs, and causing damage to teat-ends (Barber 2015). Clover dominant pasture and improved pastures are said to be associated with increases in incidence of mastitis in sheep, possibly due to higher milk production and the estrogenic effects on lactation (West, Bruere, and Ridler 2009, Clark 1972). High stocking rates, trauma or damage to the teats because of vigorous lambs, shearing cuts and bruising, and udder conformation all contribute to the possibility of S. aureus infection of the udder (West, Bruere, and Ridler 2009, Quinlivan 1968). It is possible that the stress of transportation on the ewes, crutching and not being milked out as they normally would by their lambs on the day of arrival predisposed this flock to peracute and acute gangrenous mastitis. Because animals began to show signs of peracute infection on the day of arrival, it is possible that some or all of the affected animals were S. aureus carriers in the flock prior to their arrival to the farm. This was evident in the histology report of the lymph node showing a potential chronic form of the disease. The history of gangrenous mastitis on the property of origin is unknown. It is also possible that animals may have been exposed to the pathogen during transportation or on arrival. A heavily contaminated transport truck with S. aureus would have exposed all ewes and lambs to the bacteria. The most probable aetiology of this gangrenous mastitis outbreak is yet to be determined but it was likely causes of infection were either lambs cross suckling ewes, some form of teat-end damage, and an increase in milk production. Some future management decisions should be considered knowing that these ewes have all likely been exposed to or carry a high rate of the bacteria, even on healthy udders. These management changes might be routine monitoring of udders, such as observation and palpation of the udders themselves, at times of crutching, shearing, or after weaning (Clark 1972). Procedural changes will be made on the property to decrease the risk and severity of future outbreaks: purchased ewes will be crutched and lambs tailed prior to purchase; veterinary advice will be sought earlier in any outbreak; and affected animals will be treated with antibiotics and NSAIDs as soon as diagnosed.
References

For formatting purposes, all original long URLs have been condensed using the bit.ly format.


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