GANGRENOUS MASTITIS IN GOATS: WHY IS IT SO HARD TO CURE?

Sreeja S.

Mastitis is a disease complex having different causes, different degrees of intensity with variation in the duration and residual effects. Caprine mastitis is seen to be a more difficult problem to tackle when compared to that in bovines and that too of a gangrenous nature. It is the most severe form resulting in the animal's death or in complete or partial sloughing of the udder.

Gangrenous mastitis in goats is typified by a sudden onset immediately after parturition and in the beginning of lactation; good milking goats are particularly affected. The condition usually is restricted to the period of lactation. However sometimes gangrenous mastitis occurs during the last week of pregnancy, where often it causes loss of fetuses and death of the doe from toxemia. It is most frequently due to Staphylococcus aureus and the resulting toxemia is due to bacterial toxins and tissue destruction. Secondary invasion by Escherichia coli and Clostridium spp contributes to the severity of the lesion and production of gas.

Stages of gangrenous mastitis

Gangrenous mastitis can be clinically grouped in to three stages depending on the severity, extent of udder involvement and duration of disease. In the early stage, the skin of the teat or udder floor becomes cool and edematous, and the goat appears lame. This infection initially involves the teats, distal portion of the udder and then spreads laterally towards the attachment of the udder. Milk at this stage is blood tinged with minute clots. Palpation of the udder reveals rise in local temperature, tenderness and evidence of pain to the animal which stand with abducted hind quarters. The intermediate stage is characterized by distension of the udder to 2-3 times its normal size and the udder may be seen dragging on the floor in most cases. Palpation of the udder is apparently less painful at this stage and the udder pits on pressure. Many excoriations and a reddish blue discoloration are noticed on the skin of udder. The affected udder becomes cold and insensitive with an offensive smell and a thin zone of dark red hyperemia at the periphery. This is followed by excessive desquamation of the epithelium of the affected area, complete shedding of the epidermis and thereby exposing the underlying dermis (white area). Serous exudate ooze out from this area and the secretion becomes watery red with gas bubbles which produce a squeaking sound when the teat is stripped. In the late stage of the disease the whole skin from teat to attachment becomes dark blue in colour surrounded by a thin prominent hyperemic zone. Death may occur within 24 hours. If the animal survives the acute phase, a clear blue line of demarcation forms on the udder and the gangrenous portions are sloughed after several days or weeks. The udder is reduced in size and had thick dry hard mass with many cracks and fissures. The exposed mass contains yellow caseated material which could be easily detached from the udder attachment.

Predisposing causes

It is proposed that the browsing nature of goats predisposes it to teat abrasions and wounds which then lead to gangrenous mastitis. In addition, the relatively poor vascularisation of caprine udder compared to cattle and the lack of valves in large veins are the other predisposing factors. Gangrenous mastitis occurs mostly in lactating animals rather than dry ones mainly because the marked distension of the lactating udder predisposes it to trauma and thus becomes vulnerable for the acute necrotizing action of the alpha-toxin of S aureus. Thus histologically there is venous thrombosis and the initial inflammatory changes are replaced by necrosis and sloughing of epithelial cells. This thrombosis is mainly responsible for the edema of the mammary gland and ventral abdominal wall. As in other

domestic animals, the large veins of the udder have no valves which tend to slow down the venous return from the udder thereby increasing the chances of thrombosis. Moreover milk is a good medium for the growth of bacteria. All these factors make gangrenous mastitis a difficult disease to cure.

Treatment.

Treatment must be initiated at the early stage of the disease itself for the udder half to be restored to normal function. Local intramammary infusion of any antibiotic will not give the desired effect because the organism multiplies deep inside the parenchyma where the drug has no accessibility. Also the alpha-toxin produces ischemia and thrombosis of large vessels preventing the paranterally administered drug to reach the site of action. Intermediate stage of the disease may also be treated effectively by administration of diuretics, systemic antibiotics and topical application of antiseptic creams at the earliest. This is possible because in this stage necrosis and gangrene involves only a small group of lobules in the distal part of the udder and the treatment limits the spread of infection to the rest of udder. Thus granulation tissue may be formed after complete shedding of necrotized material leaving a small scar on the skin over the distal part of udder. In the later stages medicinal treatment is usually of no use and efforts to save the life of the doe may be resorted to. Amputation of the udder can be a life saving procedure if the goat is very toxemic with gangrenous mastitis. Another option is that the gangrenous gland can be infused with silver nitrate 3 % (20-50ml), copper sulphate 5% (20ml) or acriflavin 1:500 (100-200ml) which kills the pathogens and removes the necrotic tissues. The gland thus treated will not return to production but the life of the doe may be saved.

References

Abu-Samra, M.T., Elsanousi, S.M., Abdalla, M.A., Gameel, A.A., Aziz, M.A., Abbas, B., Ibrahim, K.E.E. and Idris, S.O. 1988. Studies on gangrenous mastitis in goats. Cornell Vet. 78: 281-300

Ameh, J.A., Addo, P.B., Adekeye, J.O., Gyang, E.O., Teddek, L.B. and Abubakar, Y. 1994. Gangrenous caprine coliform mastitis. Small Rumin. Res. 13: 307-309

Radostitis, O.M.., Blood, D.C., Gay, C.C. and Hinchcliff, K.W. 2000. Veterinary medicine. A text book of the diseases of cattle, sheep, pig, goat and horses. Ninth edition. W. B. Saunders Company Ltd, London, p.1877

S. Sreeja. 2005. Clinico – therapeutic studies on bacterial mastitis in goats. MVSc thesis, Kerala Agricultural University, Mannuthy, p. 122

Smith, M.C. and Sherman, D.M. 1994. Goat medicine. Lea and Febiger, Philadelphia, p. 620

Author

Dr.Sreeja S.
Veterinary Surgeon
Department of Animal Husbandry
Kerala

****