

Review Article

UNDERSTANDING THE PATHOPHYSIOLOGY OF PYOMETRA AND IT'S TREATMENT IN BOVINES – AN OVERVIEW

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Introduction: Postpartum uterine infections severely affect the productive and reproductive performance of the animals which may cause severe economic losses both to the farmers and the dairy industries. Metritis, endometritis and pyometra are the most common uterine infections. Effective use of treatment protocols requires proper knowledge of both normal reproductive endocrinology and pathophysiology of these conditions. The present paper reports a brief review regarding the pathophysiology of pyometra and its treatment in bovines.

Pathophysiology: The uterus of all postpartum animals (90%) is contaminated with bacteria following parturition but this need not necessarily indicate infection or development of uterine disease. Most of these animals eliminate this contamination during the process of involution of uterus but if the contamination is not eliminated due to disruption of uterine defence mechanisms it results in persistent infection. Pyometra is one such postpartum uterine infection which is characterised by the accumulation of purulent or mucopurulent material in the uterine lumen with the presence of corpus luteum and failure of estrus. As a result the cervix remains closed but in some cases the lumen is not completely occluded and the purulent discharge can be noticed in the vagina when the animal lies down, urinates or defecates. In most cases pyometra develops as a consequence of endometritis because the postpartum animals with this problem ovulate and develop pyometra due to the presence of active corpus luteum. So the early postpartum ovulation may predispose the animals to this condition because *A. pyogenes* and anaerobic gram negative bacteria remain in the uterus after ovulation enabling continued bacterial growth after corpus luteum formation whereas post service pyometra is due to *Trichomonas foetus*, a flagellate protozoan which colonizes in the uterus. It does not prevent fertilization but causes embryonic death at early stages of

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gestation, sometimes early embryonic death is followed by pyometra and persistent corpus luteum.

Treatment: Success in the treatment of uterine infections depends on, evacuation of the uterine fluids, susceptibility of the infectious agents to the drug used, concentration and number of times the drug is used, and the exposure of entire endometrium to the drug. Several studies indicate that the presence of aerobic and anaerobic bacteria in the uterus contributes to reduce fertility in cows with uterine infection. Therefore, ideally, therapy for uterine infection should eliminate pathogens from the uterus and result in a short withdrawal periods. Evacuation of the uterus contributes to the success of further antibiotic therapy which can be done by repeated palpations of the uterus by the veterinarian and the use of hormones to expel the fluid or hasten the onset of estrus (Lewis, 1997). Estrus is usually the best way of stimulating uterine contractions and expelling the fluids (Roberts, 1986). When fluids are expelled, the effectiveness of antibiotics in clearing the remaining infection is improved. Estradiol has been recommended by some to stimulate myometrium contractions, phagocytosis and mucus production (Roberts, 1986) however, Overton *et al.*, 2003; Risco and Hernandez, 2003 concluded administration of estradiol did not have beneficial effects on reproductive performance. Administration of prostaglandin F₂ at postpartum shortens the postpartum interval in cows and Buffalo cows and may influence days to first estrus in dairy cows by enhancing lochia evacuation. The decrease in progesterone and increase in estrogen concentrations associated with luteolysis and follicular growth result in maximal resistance of the uterus to bacterial infection. PGF₂ has the least harmful effects and milk does not have to be discarded.

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