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Disorders of the postpartum bovine uterus: A Literature Review

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Abstract

Uterine health condition plays a very important role in evoking cattle fertility or infertility. The major prerequisites to enhance and achieve postpartum bovine fertility are a normal physiological uterine involution and re-establishment of ovarian cyclicity. Uterine function is usually compromised in cattle by bacterial contamination of the uterine lumen after parturition, and pathogenic bacteria often persist, causing uterine disease, a key cause of infertility in cattle. The causes of uterine diseases are complex and multifactorial; therefore, a broad approach must be taken when trying to identify the causes or prevent them. The dairy cow undergoes a state of negative energy during the transition into lactation, which leads to immunosuppression and increased susceptibility to disease. The main risk factors for uterine diseases are dystocia, stillbirth, abortion, prolapsed uterus, retained placenta. Uterine diseases are associated with lower conception rates, increased intervals from calving to first service or conception, and more cattle culled for failure to conceive. Health and management strategies should be applied to avoid the various risk factors that may affect cattle around parturition and postpartum. This review article will through light on conditions associated with uterine health and disorders and their significance on future fertility of parturient dairy cows.

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Introduction

The bovine uterus is bicornuate in type having small uterine body and two long uterine horns. The major function of uterus is to retain and nourish the embryo or fetus. The endometrium provides a mechanism for attachment of the extra embryonic membranes (placenta), whereby nutrients are conveyed between maternal and fetal blood. After full term gestation, uterine contractions are responsible for 90% of the efforts in fetal and fetal membranes expulsion (Sloss and Dufty, 1980). With the absence of the postpartum

infection, the uterus returns to the normal non-pregnant size and function before pregnancy (Roberts, 1982).

Another important function of the uterus is that it controls duration of corpus luteum (CL) function by producing a luteolytic factor ($PGF_{2\alpha}$), near the end of diestrus phase of the estrous cycle, that is diffused to ovary from uterus by local rather than systemic circulation, inducing CL luteolysis and initiating subsequent estrous cycle (Jackson, 1995).

Fluid accumulation in the uterus, whether normal during gestation or abnormal pathological fluids associated with some uterine disorders, results in CL persistence and anestrum (Gustafsson, 1980).

Health condition of the uterus plays a very important role in evoking cattle fertility or infertility. This review article will through light on conditions associated with uterine health and disorders and their significance on future fertility of parturient dairy cows.

Uterine health

The major prerequisites to enhance and achieve postpartum bovine fertility are a normal physiological uterine involution and reestablishment of ovarian cyclicity. These factors influence the fertility rate in the postpartum cow and can subsequently increase or reduce the open period.

Normal uterine involution (normal puerperium)

Restoration of the uterus to its normal anatomical and physiological non-pregnant state after parturition is termed uterine involution. After parturition, the uterus is large flabby sac, thus reduction in size and reorganization of tissues are necessary before another pregnancy can be maintained (Arthur *et al.,* 1989).

During pregnancy, the uterus is in sterile conditions, but at parturition these conditions are disrupted. Both pathogenic and nonpathogenic bacteria enter the uterus through the dilated cervix and rapidly multiply in a favorable uterine environment. The postpartum uterus depends on three processes to eliminate bacterial contamination and to be prepared for the subsequent pregnancy: 1) A defense mechanism that includes a massive infiltration of lymphocytes into the uterine lumen to phagocytize the major pathogens.) A massive release of prostaglandin during the first two weeks postpartum that induces myometrial contractions and evacuation of intrauterine fluids and fetal membranes. 3) Estrogen secreted by the ovaries before the first ovulation makes the uterus more resistant to infection (Jainudeen and Hafez, 2000).

Under normal conditions, the uterus continues contraction after parturition to expel the placenta. Expulsion of the placenta involves loosening of the villi of the fetal cotyledons from the crypts of the maternal caruncles. This loosening is believed to be aided by removal of the blood from both fetal and maternal placenta as a result of the strong uterine contractions during expulsion of the fetus (Salisbury *et al.,* 1978). Placenta is usually discharged within half an hour to 8 hours, and considered retained if it is not expelled 8 to 12 hours postpartum (Roberts, 1982).

Prostaglandins have been reported to play a role in controlling uterine involution, although the postpartum rise in $PGF_{2\alpha}$ metabolite (PGFM) may be a reflection of the

process of involution rather than the cause (Noakes *et al.,* 2001). A positive correlation between PGFM concentration in the peripheral circulation and the diameter of uterine horn was reported by Eley *et al.,* (1981). Similarly, Toribio *et al.,* (1994) reported a marked linear decrease of PGFM levels from parturition until day 23 postpartum. They found a significant correlation between the duration of elevated PGFM and the time required for completion of uterine involution. However, Bekana *et al.,* (1996) stated that there was no significant relationship between the duration of $PGF_{2\alpha}$ release and the time required for completion of uterine involution. They added that the plasma levels of PGFM were significantly high for the first 12-18 days in the postpartum cow with normal puerperium.

Postpartum ovario-uterine axis

Ovarian activity during the early postpartum period exerts an important influence on the ability of the uterus to resist or eliminate bacterial infections. Jainudeen and Hafez, (2000) have stated that, the postpartum cows can resist uterine infection during the estrogenic phase but are very susceptible during the progesterone phase, which is due to decreased leucocytic activity. Dhaliwal *et al.,* (2001) reported an increased concentration of immunosuppressant proteins in the uterine lumen under progesterone dominance, which inhibits lymphocyte proliferation, making the uterus more susceptible to infection. Uterine infections can delay the onset of postpartum ovarian cyclicity by delaying the initiation of folliculogenesis and suppressing the rate of follicular growth in dairy cows during the early puerperium by inhibiting LH release (Jainudeen and Hafez, 2000). Zraly *et al.,* (1989) have assumed that the delayed and asynchronous growth of follicles in cows with pathological puerperium is a consequence of disturbed repairing processes of endometrium and of endocrine dysfunction. Mateus *et al.,* (2002) reported that ovarian activity was abnormal (prolonged anestrus, prolonged luteal phases, and ovarian cysts) in cows with severe endometritis.

During pregnancy, follicular waves occur periodically, but these follicles are of the anovulatory type because of the prolonged period of inhibition during pregnancy, due to the continuous negative-feedback effect of progesterone by the corpus luteum and placenta. Subsequently the pituitary becomes refractory postpartum (Noakes et al., 2001). Eley *et al.,* (1981) have reported that the ovario-uterine axis exerts an inhibitory effect on pituitary LH secretion during the early postpartum period.

After parturition, cows enter a period of postpartum anestrus that ends by the first postpartum ovulation which is not often associated with overt estrus. Hafez and Hafez, (2000) have reported that silent ovulations can occur in cows 2 to 3 weeks postpartum. The duration of the postpartum anestrus is affected by the rate of uterine involution, rate of development of ovarian follicles, pituitary gonadotrohins and levels of estrogen and progesterone (Stevenson and Britt, 1980).

Uterine disorders

Delayed uterine involution (abnormal puerperium)

Disorders and complications that occur in the uterus during or after parturition will subsequently result in delay or prolongation in the involution process. Uterine disorders

could be divided in to 2 major headings based on the time of their occurrence: disorders that occur during the peripartum period, and those occurring during the postpartum period.

Peripartum uterine disorders

Dystocia is the major problem that faces the cow at parturition, and it is greatly caused by or results in disorders and complications in the uterus during the peripartum period. Dobson *et al.,* (2001) have reported that cows with dystocia are known to have a delay in uterine involution, and increased intervals to first estrus and conception. They added that the consequences for fertility were worse (more inseminations per conception, prolonged calving to conception interval, and lower pregnancy rates) in cows that required a caesarian operation.
Wehrend *et al.,* (2002) reported that the main cause of dystocia was the incorrect position/orientation of the fetus (38.9%) as well as relatively or absolute too large calves (25.2%). Atallah *et al.,* (1999) have stated that 56.25% of dystocia cases in crossbred (Friesian x Native Egyptian) cows were attributed to fetal causes compared to 43.75% due to maternal factors. They added that 21-41% of all dystocias were due to faulty posture. Noseir and Hattab, (1999) have found that all dystocia cases (10.0%) in Friesian cows were associated with poor reproductive performance.

1. Uterine distention

1.1. Fetopelvic disproportion

This important cause of dystocia occurs when the fetus is larger than normal or the pelvis is smaller than normal or there is a disproportion between them. It usually occurs in cases of fetal monsters or fetal oversize (Jackson, 1995).

Fetal monsters

They arise from adverse factors affecting the fetus in the early stages of its development. These factors are mostly genetic in origin but may also include physical, chemical, and viral factors. Not all anomalous fetuses or monsters cause dystocia. The monsters that are characterized by an increased size of the fetus include: hydrocephalic, anasarcous, ascitic and monsters with marked skeletal defects such as Schistosomus reflexus and Perosomus elumbis, also monsters due to conjoined twins, double monsters, or monsters due to severe form of achondroplasia (bulldog calf) (Roberts, 1982). The more common fetal monsters are conjoined twins (33.1%) Schistosomes (31.8%), bulldog calves (8.4%), and other monsters account for 26.6% of cases (Jackson, 1995). Atallah *et al.,* (1999) reported that 5.5% fetal monsters and 11.1% oversized fetuses, were the causes of fetal dystocia in cows.

Fetal oversize

When the fetus is larger than normal it may have difficulty in passing through the birth canal. In this instance, assistance may be needed. Breeding of first-calf heifers to bulls

that are known to sire small calves is often practiced to minimize this problem in heifers which may not be fully mature themselves before delivery of their first calf.
Fetal overgrowth syndromes have arisen from chromosomal abnormalities, spontaneous mutations or experimental genetic manipulation, especially after exposure to unusual environments both in vivo and in vitro (Young *et al.*, 1998).

1.2. Hydrops uteri

It is the accumulation of excessive amounts of fetal fluids within the pregnant uterus. The fetus itself may or may not be edematous and may show anasarca, hydrothorax, or ascites. Two forms of hydrops uteri have been described (depending on the site of excessive fluids): hydrops amnion and hydrops allantois. Hydrops amnion is often associated with abnormalities of the fetus, especially cleft palate, pituitary hypoplasia and bulldog calves, while placental abnormalities and interference with sodium metabolism contribute to hydrops allantois (Jackson, 1995).

1.3. Twinning

Dobson et al. (2001) have reported that 26% of calves from twin-bearing cows required assistance at birth, and 13% of calves died. Echternkamp and Gregory, (1999) stated that the interval from parturition to first estrus was prolonged and the pregnancy rate was reduced in the postpartum cow birthing twins. They added that the higher incidence of dystocia with twins than with singles (46.9 vs. 20.6%) was primarily due to abnormal presentation of one or both twin calves at parturition.

2. Uterine inertia

The most important peripartum uterine disorders are those that primarily interfere with normal coordinated myometrial contractions. Two conditions were reported for weakness or absence of uterine contractions; primary and secondary uterine inertia (Jackson, 1995).

Primary uterine inertia, is mostly a deficient myometrial contraction which delays propulsion of the fetus toward cervix and vagina (Roberts, 1982). Arthur *et al.*, (1989) attributed its incidence to hormonal causes whether due to reduction in the level of hormones (estrogen, oxytocin) or due to inability of the uterine wall to respond to hormones. Sloss and Dufty, (1980) and Arthur *et al.*, (1989) have reported that this primary weakness of labor expulsive power could be attributed to metabolic disorders like hypocalcaemia, hypomagnesaemia, and ketosis, or due to debilitating nutritional deficiencies, toxemia, and septicemia. In addition, the overstretching of the myometrium was reported to be one of the important causes of primary inertia of the uterus. This condition is usually associated with abnormally large fetus, fetal ascites, hydrops allantois or twinning (Jackson, 1995).

Secondary uterine inertia, results from exhaustion and is a result of, rather than a cause of dystocia. This condition develops when an obstructive dystocia have existed for longtime and the fetus has become tightly impacted in the uterus (Roberts, 1982).

Secondary uterine inertia is frequently followed by retention of the fetal membranes and retarded involution of the uterus, factors which predispose to puerperal metritis (Arthur *et al.,* 1989).

Postpartum uterine disorders

1. Retention of placenta

Parturition is completed only after expulsion of fetal membranes (3rd stage of labor), which normally detach from uterus within 12 hours following birth of the fetus, and if not expelled within 24 hours it is regarded as being retained (Sloss and Dufty, 1980).

Most investigators (Roberts, 1982; Arthur *et al.,* 1989; Jackson, 1995) revealed that the retention may be associated with three main factors: 1) Insufficient expulsive myometrial efforts after the second stage of labor. 2) Failure of separation between cotyledons and caruncles as a result of inflammatory changes, hormonal imbalances, lack of polymorph migration to the site of attachment and possibly immune deficiencies. 3) Mechanical obstruction due to partial closure of the cervix.

Infectious diseases including brucellosis, campylobacteriosis, tuberculosis, Leptospirosis and various molds, causes abortion and are associated with retention of fetal membranes (Sloss and Dufty, 1980). Laven and Peters, (1996) have reported a number factors that were associated with increased incidence of retained placenta; heat stress, prolonged or shortened gestation, induction of parturition, dystocia, hypocalcaemia, twin births, abortion, fatty liver, and nutritional deficiencies of carotene, vitamin E and selenium.

Dohmen *et al.,* (2000) have stated that cows with retained placenta had significantly higher lipopolysaccharide (bacterial endotoxins) levels in uterine lochia as compared to dystocia and healthy postpartum cows. Konigsson *et al.,* (2002) reported that all cows with retained fetal membranes have high levels of prostaglandin metabolite (15-ketodihydro-PGF2alpha) immediately after parturition but these levels fall rapidly within 2 weeks postpartum. They suggested a bacterial contribution to this elevated level of prostaglandin metabolite.

The consequence of placental retention may be most serious in parturient cows that it is frequently leading to acute or chronic metritis and subsequent delay in uterine involution (Jackson, 1995).

2. Uterine infections

The term uterine infection is used to indicate that the uterus is contaminated with pathogenic organisms. Endometritis indicates that the endometrium is inflamed; metritis indicates that all layers of the uterine wall are inflamed, and pyometra indicates that purulent exudates have accumulated in the uterine lumen (Lewis, 1997). Jainudeen and Hafez, (2000) have stated that, metritis is the inflammation of the entire thickness of the uterus (i.e. endometrium and myometrium), and of the several bacteria that have been implicated, *Actinomyces pyogenes* is the most frequently encountered organism in the cow. Laven and Peters, (1996) reported that puerperal metritis is usually associated with retained placenta. They added that metritis is usually a consequence of retained placenta rather than a cause of it. Sloss and Dufty, (1980) have reported that the condition is

critical when the animal is recumbent, severely dehydrated and also when generalized peritonitis has developed. They postulated that the future fertility is bad and peritoneal adhesions between uterus and other pelvic and abdominal organs are common sequel.

Nonspecific pathogens that could be isolated 3-5 weeks postpartum from cows with endometritis were *E.coli, Streptococci, A.pyogenes, Pasteurella* species, *Proteus* species, and Gram-negative anaerobes (Konigsson *et al.,* 2001; Mateus *et al.,* 2002). While, specific infections that could cause endometritis include: *Trichomonas fetus*, *Campylobacter fetus*, *Brucella abortus*, and Tuberculosis of the uterus (Arthur *et al.,* 1989).

Recent work has highlighted the importance of *E. coli* on the development of metritis and endometritis, especially the fact that it predisposes to infection with other pathogenic bacterium such as *F. necrophorum* and *T. pyogenes*, increases the likelihood of developing metritis and endometritis, and decreases the likelihood of conception (Bicalho *et al.,* 2012; Machado *et al.,* 2012a; 2012b).

In addition to bacterial and viral risk factors, various risk factors related to management and individual cows, are associated with uterine infections. Dystocia, retained placenta, stillbirths, twinning, milk fever, and ketosis were associated with increased risk of endometritis (Correa *et al.,* 1993; Lewis, 1997). Mollo *et al.,* (1997) reported that endometritis occurred in 51.6% of cows following placental retention, compared to 10.4% of those with normal expulsion of fetal membranes.

Nonspecific uterine infections reduce the reproductive efficiency of dairy cows. In some herds, 40% of the postpartum cows may be diagnosed with, and treated for, uterine infections (Lewis, 1997). In Egypt, the incidence of endometritis ranged from 42.6% to 54.2% in infertile cows (Abd-Elgawad *et al.,* 2000). A high incidence (81.25%) of chronic endometritis in native cows was reported by Atallah, (1984).

Lewis, (1997) reported that the consequences of uterine infections vary considerably among cows. The magnitude of the consequences varies with severity of infection, time postpartum, and herd health management. He added that effects on reproductive performance included; reduced rate of uterine and cervical involution, prolonged calving to conception interval, increased interval to first estrus, and increased culling rates.

Uterine infections in cows are associated with elevated circulating levels of prostaglandin-F2alpha metabolite (Thompson *et al.,* 1987). In a study by Del Vecchio *et al.,* (1992), bacteria (*E.coli, Actinomyces pyogenes*) were inoculated into uterine horns. They found that mean prostaglandin-F2alpha metabolite concentration had increased after bacterial infusion. Konigsson *et al.,* (2002) have reported that the prostaglandin metabolite elevation is significantly correlated to duration of uterine infection. They suggested a bacterial contribution to this elevated level.

Although complete elimination of uterine disease does not seem possible with the current understanding of the pathophysiology of uterine diseases, there are management strategies that can be taken to alleviate the problem. Prevention strategies should be focused on maximizing cow comfort, preventing late term abortions with appropriate vaccination programs, preventing nutritional disorders, and the use of prophylactic treatment with prostaglandin F2-alpha, oxytocin, estradiol, nonsteroidal anti-inflammatory drugs, and antibiotics as needed (Galvão, 2013).

On conclusion, the postpartum period is a critical period in the annual reproductive cycle of the dairy cow and directly influences the duration of this cycle. Uterine disorders have

negative effects on various measures of productivity of dairy cows. They reduce reproductive efficiency, increase herd health costs, cause an appreciable reduction in milk production, and force producers to cull cows that would otherwise be productive and remain in the herd.

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