Cow postpartum uterine infection: A review of risk factors, prevention and the overall impact

Joshua Onyango

The University of Northampton and Moulton College, Department of Animal Management and Veterinary Health, Northampton, NN3 7RR, Great Britain.

Abstract

Several physiological changes occur in the cow’s uterus soon after calving. These changes are significant if a cow is to recover and come back into season, ready to conceive, thus any factors that interfere with the normal functioning of the uterus and other associated structures will affect overall reproductive performance. Research has shown variations in the prevalence of uterine infection between different countries due to several factors such as general environment, nutrition, calving season and breed. Infections such as puerperal metritis and endometritis are often caused by pathogens such as *Actinomyces pyogenes*, *Fusobacterium necrophorum* and *Bacteroides spp*, which can cause significant damage to the uterine endometrial tissue. Such infections require prompt diagnosis and attention in order to prevent their detrimental effect. Research has also documented other factors that may contribute to the incidence of infection of the uterus after calving. They include age, parity, poor nutrition, dystocia and retained foetal membranes (RFMs). Prevention of uterine infection should therefore focus on the causal factors, applying the best management practises in order to minimise their overall impact. Further studies into improved genetics focusing on reproductive immunology would be something that is worthwhile in the 21st century.

Keywords: Dairy cow, uterine infection, risk factors, postpartum, prevalence, prevention.

Physiology of periparturient uterus

According to Roche (2006), the uterus should return to its normal size with uterine weight loss (90%) by 30 days postpartum. The periparturient notable changes include the release of placentac membranes, ischemic necrosis and detachment of several layers of caruncular epithelium, convalescing of surface defects with a fresh epithelium, while the myometrium muscle fibres shorten. These changes occur within 14 days after calving, just before the normal ovarian cyclic activities are about to start taking place (Sheldon et al., 2009). Kask et al. (2003) reported that uterine involution starts as early as day 29 but Arlt et al. (2009) and Sheldon et al. (2009) have shown that, on average, it occurs by day 40 when influenced by periparturient complications such as uterine disease, resulting in delayed uterine involution. Crowe (2008) found that cows on good nutrition have their first postpartum ovulation between about day 15 and 30 in beef suckler cows, but between 70 and 100 for cows in poor body condition. Potter et al. (2010) have reported that it is normal for pathogens such as bacteria to infect the cow uterus in the first few days after calving. Polat et al. (2009) have also reported that 10-17% of cows have pathogenic bacteria two weeks post-calving while a striking study by Sheldon (2007) reported that intensively managed dairy cattle often have bacterial uterine contamination rates of 90 to 100% within the first two weeks postpartum. However, studies have shown that a healthy uterus is able to get rid of the majority of mild infections very effectively (Gautam et al., 2010). Many trials have revealed that a wide range of micro-organisms gain entry into the uterus causing infection. However, calving that experience less complications often report only mild isolates of pathogens, with up to 30% of them being non infective (Roche, 2006; Sheldon, 2007).

Definitions and aetiology of uterine infections
Despite a lack of precision and variations among research groups, the definitions of uterine infections encountered in cattle have been extensively reviewed and expressed (Foldi et al., 2006; Palenik et al., 2009; Potter et al., 2010). Whilst it is not always possible to group every animal with uterine infection, some definitions have been used. Puerperal metritis can be defined as an acute systematic illness due to infection of the uterus by pathogenic bacteria, usually within 10 days after parturition (Sheldon and Dobson, 2004; Melendez et al., 2004; Williams et al., 2008). Actinomyces pyogenes is considered to be the most common bacterium involved in the cause of the disease, due to its resistance and synergistic action with gram negative anaerobes such as Fusobacterium necroforum, Bacteroides spp (Mateus et al., 2002; Petit et al., 2009). It results in acute inflammation in the endometrial, peritoneal and myometrial layers of the uterus. Clinical signs include a foul smelling red brown watery uterine discharge which might be accompanied by pyrexia (increased body temperature, ≥39.5 °C) (Palenik et al., 2009; Sheldon et al., 2009). Serious cases are characterised by reduced milk production, lack of appetite, increased pulse and heart rate, abrupt ruminal stasis and dehydration (Foldi et al., 2006).

Endometritis involves the inflammation of the uterine layer (endometrium) characterized by the presence of mucopurulent vaginal discharge, three weeks or more postpartum (Sheldon et al., 2006; Arlt et al., 2010). Usually, the main causative agent is Arcanobacterium pyogenes, which persists for more than 3 weeks postpartum (Miller et al., 2007; Tsouisis et al., 2009; Westermann et al., 2010). Gram negative anaerobes such as Fusobacterium necroforum, Bacteroides spp, Prevotella spp have also been isolated in clinically diseased animals where, A. pyogenes act synergistically with the gram negative anaerobe bacteria to enhance the severity of endometritis (Williams et al., 2005; Drillilch, 2006). These organisms are acquired from beddings, environment and fecal contamination of the animal coat (Sheldon et al., 2008). Gilbert et al. (2005) reported that the incidence of lactational endometritis varied from 7.5% to 61.6% among dairy herds, consistent with Potter et al. (2010) who reported a 27% lactational incidence of endometritis in a study that involved four Holstein dairy herds.

Pyometra is characterized by the accumulation of pus within the endometrial cavity, followed by persistent corpus luteum (Sheldon et al., 2008). Also there is a failure for cows to show oestrus (Foldi et al., 2006). The condition is most common in cows during first postpartum ovulation in the presence of pathogenic bacteria hence, the key issue is to identify such cows and administer the right treatment (LeBlanc, 2007).

Prevalence of uterine infections

Several studies have reported variations in the prevalence of uterine infections (Konyves et al., 2009a; Plontzke et al., 2010). The variations in prevalence rates have been attributed to different ways of classification, management systems, breed differences, environmental factors, nutrition, age and parity amongst others (Gautam et al., 2010; Potter et al., 2010). Gilbert et al. (2005) reported the prevalence rate of endometritis to be 53% among the dairy herds in the US. In Spain, the prevalence of endometrii varied from 2.6% to 4.5 % (Lopez-Gatius, 2003), in Japan 23.6% (Gautam et al., 2010), in Korea 47.6% (Kim and Kang, 2003), in Denmark 6.25% (Bruun et al., 2002) while in Australia it varied from 5.6% to 10.9% (Moss et al., 2002) and in Argentina 10% to 38% (Madoz et al., 2008; Plontzke et al., 2010) on pasture based extensive systems. In the UK, the prevalence of uterine infections was recorded to be 10.1% (Azawi, 2008), while in Brazil the prevalence of uterine infection in Zebu cows was 3.3% where, the low percentage prevalence was related to the ease with which the Zebu expels the calf. However, a higher prevalence was noted from a study conducted in Ethiopia by Bacha and Regasa (2009) who reported that sub-clinical endometritis in Zebu x Friesian cross breeds was 47.5% and 30.5% at week 4 and 8 postpartum, though there was a significant decrease as postpartum period increased.

Risk factors associated with uterine infections

The exact risk factors for cow postpartum uterine infection are unknown. However, several investigations have identified some of the risk factors and found that they vary among different regions or countries because of the differences in general management, environment and herd health conditions (Kim and Kang, 2003; Bell and Roberts, 2007; Potter et al., 2010). Various risk factors related to management and individual cows have been identified. They include RFMs (Abdelhameed et al., 2009), dystocia (Garry, 2004), age (Sheldon et al., 2006), parity (Gautam et al., 2009) calving season (Buckley et al., 2010), breed (Potter et al., 2010) and nutrition (Bell and Roberts, 2007) among others.

Retained foetal membranes

RFMs have been shown to be one of the most significant risk factors for uterine infection, causing up to about 50% cases of metritis (Goshen and Shpigel, 2006; Konyves et al., 2009b). Most cows will pass the afterbirth (placenta) within 6 hours of calving though in some cows it may extend up to 24 hours (Abdelhameed...
et al., 2009). Buckley et al. (2010) showed that the retention may vary between 4 and 48 hours. A high frequency of placental retention has been reported in cows compared to other species, supporting the fact that cows have intimate placental attachment (Smith and Risco, 2002).

The time frame used to diagnose RFMs is contentious. However, it is scientifically accepted that failure to expel the foetal membranes within 24 hours after parturition is regarded as abnormal (Smith and Risco, 2002; Gautam et al., 2010). The aetiology of RFMs is complex (Konyves et al., 2009b) but the pathogenesis has been linked with failure in prompt breakdown of the cotyledon-caruncle attachment after delivery of the calf (Abdelhameed et al., 2009). The condition can occur due to insufficient uterine motility, induced parturition, twin birth, stillbirth, dystocia (Konyves et al., 2009b; Majeed et al., 2009; Potter et al., 2010), abortion, mineral and vitamin deficiencies (Wilde, 2006; Alsic et al., 2008), hormonal imbalance and immune disease affecting degradation of placentomes at the end of pregnancy and negative energy balance during prepartum period (Fraser, 2005; Ingvartsen, 2006). It has been reported that animals with a problematic negative energy balance before calving and sub-optimal vitamin E status in the last few weeks before calving are more likely to have RFMs (Quiroz-Rocha et al., 2009; Beagley et al., 2010). However, a study by LeBlanc and co-workers (2002b) found no significant difference in the incidence of RFMs, metritis and endometritis between groups of cows given and those not given vitamin E a few weeks prior to calving.

The occurrence of RFMs in complicated parturition has been found to range between 20% and 50% (in cases of dystocia, caesarean section, premature calving and foetotomy) and 3% to 12% in uncomplicated parturition, although the figures may vary between regions and breeds of cattle (Smith and Risco, 2002). Bellows et al. (2002) reported an 8% occurrence of RFMs in normal parturition though this figure went up to 45% to 70% during twin birth. Alsic et al. (2008) reported 16.5% diet influenced cases of RFMs, following a trial which looked into the levels of selenium, and vitamins E and D post calving. Goshen and Shpiegel (2006) showed 9% and 13% cases of RFMs with 18% and 30% cases of metritis in primiparous and multiparous cows, respectively. Recent studies by Gunduz and co-workers (2010) and Jeremejeva et al. (2010) reported a 28% incidence of postpartum uterine infections where RFMs were found to be highly significant. Majeed et al. (2009) reported that the incidence of RFMs in Iraq was between 5% and 23%. Ahmed et al. (2009) reported the incidence of RFMs to vary between 5% and 10% in Egypt while in Ethiopia the incidence of RFMs was 14.7% though this varied between regions (Shiferaw et al., 2005).

Bruun et al. (2002) and Ahmed et al. (2009) demonstrated that cows with a RFMs have a higher chance of contracting uterine infections and the probable reason was that RFMs act as a good media for bacterial multiplication. A more recent study by Potter et al. (2010) found that RFMs were a highly significant risk factor of endometritis while, Han and Kim (2005) found a high occurrence of endometritis in dairy herds following cases of RFMs which they further linked with increased postpartum uterine infections. Similarly, Bakena (1994) showed that RFMs were associated with over 90% cases of endometritis while Dolezel et al. (2008) and Konyves et al. (2009b) reported increased odds of puerperal metritis in cows with retained RFMs. Other studies have failed to establish any link between RFMs and postpartum disorders (Amer et al., 2010).

**Dystocia**

Dystocia also defined as complications developed during calving often result in postpartum uterine infections (Han and Kim, 2005; Foldi et al., 2006; Tsousis et al., 2009). A relatively oversize calf may cause complications in the uterus during or after calving (Larson and Tyler, 2005; Abdelhameed et al., 2009). Difficult births have been strongly associated with uterine infections and reduced reproductive performance in most of the research work (Bell and Roberts, 2007; Dolezel et al., 2008; Potter et al., 2010). Assisting an animal with dystocia often results in damage to the uterine wall, creating wounds which later get invaded with pathogens. Mee (2008) showed that dystocia rates in the dairy industry with similar genotypes currently vary between 2% and 7% internationally, although the figures are higher in the US. It is thought that in the US, dairy animals are not rigorously selected for calving ease and most dairy farms do not direct their management towards reducing dystocia risks (Garry, 2004) paying little attention to the welfare implications of dystocia. Proudfoot and co-workers (2009) found that dystocia resulted in a reduced daily dry matter intake in postpartum cows and thus a reduced body condition with a higher risk for uterine infection. Dolezel et al. (2008) and Gunduz et al. (2010) found that dystocia was significant in the occurrence of severe puerperal metritis in cows.

There is little published information regarding the connection between twinning as one of the predisposing factors for dystocia and postparturient uterine infections, but a study by Smith and Risco (2002) and Hossein-Zadeh (2010) showed that multiple births increased the odds for dystocia which was also involved in the aetiology of part-parturient infections. Bell and Roberts (2007) showed that a difficult calving
was associated with male and twin births. Cows with twins have a higher risk of metritis (Mills, 2006) while still births and foetal-maternal disposition, characterised by a relatively large foetus compared to the maternal pelvis, has been shown to result in dystocia (Hickson et al., 2003; Gustafsson et al., 2004). Cady (2010) and Zaborski et al. (2009) reported that, for maximum profit without compromising on a heifer’s health and welfare and to minimise cases of dystocia, breeding should be programmed in such a way that heifers calve at 24 months of age when their pelvic development would be enough to calve relatively easily.

High incidences of dystocia have been reported in up to 40% of heifers compared to 20% of cows with the increased incidence in heifers being attributed to foeto-maternal disposition damaging the pelvis (Dohmen et al., 2000). A recent study by Micke et al. (2010) reported that a longer gestation period in 3-year-old Bosindicus x Bostaurus heifers was significantly associated with an increased risk of dystocia at calving and was also associated with increased uterine infections such as metritis and pyometra. Hence, a well defined management program to reduce cases of difficult calving and pay attention to identify cows experiencing dystocia is critical to cow health, welfare and farm profitability (Bell and Roberts, 2007; Potter et al., 2010).

Age

The effect of age on reproductive performance has been studied but, results are not definitive (Pugh et al., 1994). A relationship between age and incidence of postpartum uterine infection, abortion and overall poor reproductive perfomance has been reported (Sheldon et al., 2006; Rafati et al., 2010). The higher milk yield in older cows is thought by some to result in more clinical abnormalities, which may delay the time of first postpartum ovulation and involution of the cervix and uterus (Kawashima et al., 2007; Kafi and Mirzaei, 2010). Some studies have shown association between age and incidence of metritis (Bruun et al., 2002) while, others have failed to establish any association (Emanuelson et al., 1993; Pugh et al., 1994). Smith and Risco (2002) found that the lowest incidence of postpartum metritis occurred in cows between 2 and 4 years of age and the highest incidence was in cows older than 7 years. In contrast, Lewis (1997) reported that cows between 2 and 4 years of age were more likely to have dystocia or RFMs and thus an increased incidence of uterine infections though the findings were consistent with Smith and Risco (2002) who reported increased postpartum problems in cows over 7 years of age.

Parity

The relationship between parity and postpartum disease has been supported in most research (Bruun et al., 2002; Benzaquen et al., 2007; Konyves et al., 2009a). Groehn et al. (1990) did not establish any association between parity and uterine infection though higher parity was significant for increased cases of RFMs and dystocia while Hajurka et al. (2004) and Tsousis et al. (2009) reported that advanced parity was associated with delayed uterine involution, body condition loss, reduced conception rates and increased cases of uterine infection. On the other hand, Ghanem et al. (2002) and Konyves et al. (2009a) reported increased risk of uterine infection with cows in early parity. A study by Ghanem et al. (2002) showed that first parity cows had an increased incidence of endometritis, where as those in the six parity and above range had the lowest. One possible reason for this is that first parity cows require more personnel assistance and there could be injuries inflicted on the genital tract (Kaufmann, 2009; Tsousis et al., 2009). A high risk of uterine infection with reduced conception rate has been reported in heifers when compared to cows at second calving (Quintela et al., 2004; Yusuf et al., 2010). Kaufmann (2009) found an increased incidence of subclinical endometritis with increased odds for conception in first parity cows. Similarly, Tsousis et al. (2009) found out that cows in their first parity had persistent purulent discharge compared to other groups which could have been as a result of calving assistance leading to uterine lesions. Interestingly, findings by Huffman et al. (1984) reported that cows in their first parity had reduced postpartum uterine infections.

On the other hand, studies by LeBlanc et al. (2002b) reported that clinically relevant endometritis was more prevalent in mature cows, where cows in their third or higher lactation had a prevalence of 21% compared to 13% for cows in first and second lactation. Lee and Kim (2006) showed that increasing parity was associated with increased milk yield and reduced body condition which increased the risk of pregnancy loss, RFMs and endometritis. Similarly, Quintela et al. (2004) reported that more reproductive abnormalities occur with the increase in the number of lactations while other reports have failed to establish if there is any significant association between parity and the incidence of uterine infections (Bartlett et al., 1986; Gilbert et al., 2005).

Calving season

The influence of calving season on postcalving problems has been documented with the majority of postpartum infections being reported to occur immediately after calving (Erb and Martin, 1980; Smith and Risco 2002; Majeed et al., 2009). Warmer months of summer and autumn calvings have been
associated with increased reproductive abnormalities due to the effect of heat stress (Quintela et al., 2004; Khan et al., 2009) while others have reported increased postpartum disorders, mostly during winter months, due to reduced exercise and body stress associated with the cold months. Dolezel and co-workers (2008) reported a high incidence (50%) of puerperal metritis in the spring months (March to May), albeit there was no significant effect. Conversely, Bartlett et al. (1986), Kim and Kang et al. (2003) and Gautam et al. (2009) reported that calving season was not significant in the presentation of postpartum uterine infections.

A study conducted by Buckley et al. (2010) in Northern Ireland found that calving season was positively correlated to increased incidence of RFMs and uterine infections. They further found that uterine infections were higher in the month of January and February (3%) compared to March (1.6%) and April or later (1.8%). Similarly, Majeed et al. (2009) reported increased incidence of RFMs during the months of January and March. In contrast, a study by Smith and Risco (2002) in the US showed that higher cases of RFMs occur in early winter (October to December) and the results were related to the fact that more calvings took place just before this time period. Findings by Markusfeld (1984) on a comparison of calving from three winter months (December to February) with three summer months (June to August) found that cows that calved in the summer had a higher risk of RFMs, predisposing them to uterine infections. In contrast, Grohn et al. (1990) reported increased risk of uterine infections in cows calving between September and February compared to other months. Interestingly, Kim and Kang (2003) reported up-to 36.6% cases of season associated cases of uterine infection though this was not significant. Others have reported that postpartum uterine infections are management rather than seasonal related (Lewis, 1997).

Prevention of uterine infections

Despite the many disagreement on the treatment of postpartum uterine infections, there is a strong accord on the various measures necessary to prevent their occurrence (Brozos et al., 2009; Galvao et al., 2009). Most postpartum problems can be prevented if dry cow nutrition and management are well attended to and this should result in an increased herd pregnancy rate, reduced mortality, and fewer related diseases but the preventive strategy should only be justified if it will result in at least one of the aforementioned benefits (Mills, 2006; Bademkiran et al., 2009; Abdelhameed et al., 2009).

Management during the transition period

According to O’Conner (2009), cows should begin the transition phase while in good physical shape as poor body condition has been shown to suppress dairy cow immunity making them more vulnerable to postpartum problems. Hoedemaker et al. (2009) and Zaborski et al. (2009) reported that cows in poor body condition at calving were at higher risk of dystocia, metabolic disturbances, RFMs and subsequently developing endometritis. Indeed, body condition scoring has been correlated to cow uterine infection where over-conditioned cows have been shown to exhibit a higher incidence of dystocia with increased risk of uterine infection (those with a body condition score (BCS) greater than 4 on a 5-point scale) (Urton et al., 2005; Bacha and Ragasa, 2010). Similarly, under-conditioned cows are even more likely to develop uterine infection (those with a BCS of 2.5 or less). Bell and Roberts (2007) showed that monitoring of body condition scores in late lactation and during transition is very important so that at calving, cows have a BCS of about 3.5. However, Berry et al. (2007) found that BCS was not associated with the incidences of difficult calving and uterine infection. Huzzey et al. (2007) found increased incidence of metritis in the postpartum period following low feed and water intakes close to calving time, concluding that one of the most effective ways to avoid postpartum uterine infections is by ensuring high dry matter intake (DMI) in the transition period.

A retained placenta is a significant risk factor for uterine infection and because the cause is multifactorial, it is important to recognize that no one preventive measure will be universally effective (LeBlanc, 2007). Aspects suppressing the immune function in late pregnancy and early postpartum period predispose cows to RFMs (Abdelhameed et al., 2009). Kolb and Seehawer (2002) found that the expulsion of RFMs was inhibited by a diminished accumulation of leucocytes in the placentomes, following increased cortisol levels during the peripartum period while, Alsic et al. (2008) reported an increased incidence of RFMs when the prepartum provision of vitamin E and D were insufficient. A preventive strategy which will accelerate the expulsion of placenta from the uterine cavity and minimise uterine bacterial contamination would be the best goal in preventing cases of RFMs thus, reducing uterine infection (Kolb and Seehawer, 2002; LeBlanc et al., 2008). Published work has shown that a prepartum diet of 0.1-0.5mg/day selenium, 100g/day calcium and 2000 international units (IU) of vitamin E is an effective way of reducing incidences of RFMs (Sattar et al., 2007; Cook and Green, 2007). Erskine et al. (1997), LeBlanc et al. (2002b) and Brozos et al. (2009) showed that the administration of 3000 mg α-tocopherol subcutaneously or intramuscularly...
1-2 weeks prior to calving led to a 4% significant reduction on cases of RFMs and the incidence of uterine infection. The findings might be explained by the fact that a combination of vitamins tends to reduce oxidative stress in animals (Spears and Weiss, 2008; Konyves et al., 2009b). Vitamin E is responsible for the activity of glutathione peroxidase in the placenta and its deficiency results in a diminished activity of glutathione peroxidase and as a result, the content of lipid peroxidase increases and the synthesis of PGF2α (prostaglandin) is lowered affecting myometrial activities (Gajewski et al., 1999; Abdelhameed et al., 2009). In contrast, Gupta et al. (2005) and a meta-analysis by Bourne et al. (2006) found that vitamin E and Selenium injections 14 days prior to calving and within a day to calving did not reduce the incidence of RFMs and uterine infection. Similarly, LeBlanc et al. (2002a) found that vitamin E given as a single dose of 3000 IU one week before calving did not reduce the incidence of RFMs and postpartum uterine disease.

**Antimicrobials and hormonal therapy**

Antimicrobials and hormonal treatments have been applied in the prevention of postpartum uterine disorders (Bademkiran et al., 2009). For example prostaglandins have been documented as causing uterotonc activities through myometrial contraction hence the expulsion of the RFMs (Takagi et al., 2002; Olson, 2003; Dolezel et al., 2008). Abdelhameed et al. (2009) reported that the use of prostaglandins reduced the damaging effect of RFMs on a cow’s genital tract. Conversely, Mejia and Lacau-Mengido (2005) reported that the use of prostaglandins delayed conception, impacting overall cow reproductive performance. Gross et al. (1986) showed that the treatment of cow’s with PGF2α during the early days postpartum was effective in the prevention of RFMs as PGF2α was reported to increase uterine contraction and myometrial activities. McDougall (2001a) reported an improved reproductive performance following intrauterine use of cepaphirin while, Heuwieser et al. (2000) reported a shorter first service interval, increased oestrus detection efficiency and reduced days open in cows on PGF2α treatment programme. On the other hand, Archbald et al. (1990) found that prostaglandin treatment in postparturient cows which had experienced difficult calving and RFMs did not have any beneficial effect on the post-parturient uterine infections. Likewise, Stevens and Dinsmore (1997) found that the use of prostaglandins during the post-calving period had no effect on the incidence of RFMs and subsequent reproductive performance. Also, Hendricks and co-workers (2006) showed that repeated doses of prostaglandins did not have any benefit on the prevalence of clinical endometritis.

Use of broad spectrum antibiotics has been of great benefit in the prevention of uterine infections (Dolezel et al., 2008; Gani et al., 2008). Dobsons and Noakes (1990) reported a significant reduction in the number of cows infected by A. pyogenes and in the number of those showing purulent uterine discharge following the infusion of uterine pessaries containing penicillin, streptomycin, formo-sulphathiazole and ethinyl-oestradiol given 24 hours postpartum. Königsson et al. (2001) reported that early oxytetracycline use in cases of RFMs was not significant on the treatment of uterine infection or involution but, instead it slowed down the expulsion of the RFMs. Nonetheless, it was evident that oxytetracycline treatment after RFMs had come off shortened uterine infection but otherwise did not affect the overall clinical outcome of uterine disease while flunixin had no effect on uterine infection recovery. Interestingly, Goshen and Shpigel (2006) found that a post-calving intrauterine use of chlortetracycline significantly reduced the detrimental effects of postpartum uterine infections in heifers.

Galvao and co-workers (2009) found that an intrauterine infusion of ceftiofur hydrochloride was significant in the reduction of prevalence of clinical endometritis. Nevertheless, a uterine infusion with ceftiofur hydrochloride had no affect on the prevalence of subclinical endometritis. A recent study by Gunduz et al. (2010) showed that a combination of enrofloxacine and penicillin G administered intrauterine to cows with RFMs following assisted calving reduced the incidence of metritis. A clinical trial conducted by Overton et al. (2003) found that a prophylactic administration of non-antimicrobials such as estradial-cypionate to dairy cows at high risk for metritis did not reduce the risk of metritis while the use of ceftiofur hydrochloride on cows with signs of pyrexia during the early postpartum period showed a significant decrease on the incidence of metritis.

Others studies have reported that cows which experienced RFMs recovered without any treatment (Gustafsson et al., 2004) while others have shown that manual removal of RFMs with a combination of ceftiofur hydrochloride administered at a dose of 1.1mg/kg/IM for 5 consecutive days resulted in an absence of fever (Overton et al., 2003). However, there is no concrete evidence to suggest that manual removal of RFMs has a beneficial effect and in fact some findings have shown that this is harmful to the cow as it can result in damage to the uterus unless combined with other measures such as insertion of intrauterine pessaries (Palmer, 2003; Drillich, 2006; Majeed et al., 2009). It is worth noting that the use of hormones and antibiotics requires knowledge of endocrinology,
pharmacokinetics and pharmacodynamics in order to get good results (Azawi, 2008).

**Periparturient hygiene**

General farm hygiene, especially on beddings and maternity stalls, have been reported as affecting the incidence of postpartum uterine infections (Lewis, 1997; Opsomer and Kruif, 2009). Palmer (2005) and Bell and Roberts (2007) reported that dirty beddings was a good place for bacterial multiplication, increasing the chance for both prepurum and postpartum diseases. An increased incidence of uterine infections has been reported in cows that calve in stalls (O’Connor, 2009) compared to those calving on pastures (Bruun et al., 2002; Plontzke et al., 2010) with a probable reason that pasture paddocks are slightly cleaner and not much calving assistance is required. It is therefore important that high standards of calving hygiene be observed in calving stalls in order to reduce the incidence of uterine infections, albeit, it is very difficult to quantify cleanliness within farms. Studies by Gustafsson et al. (2004) and O’Connor (2009) reported that disinfection of calving equipments, frequent replacement of beddings and general hygiene of the calving environment can help minimise cases of uterine infections. Interestingly, a recent study by Potter et al. (2010) found that cleanliness at the farm was not significant in the prevention of postpartum uterine infection, consistent with findings by Noakes et al. (1991) who reported that farm hygiene was not linked with postpartum uterine infection.

**Genetic selection**

It has been widely documented that both sire and maternal effect can influence the occurrence of postpartum uterine complications (Joosten et al., 1991; Hickson et al., 2006; Bell and Roberts, 2007; Zaborski et al., 2009). A study by Steinbock et al. (2003) involving Holsteins at first calving reported genetic effects on cases of dystocia where the maternal effect was 6.2% whilst the direct effect was 4.4%. They further found that heritability for dystocia was 12.0% and 17.0% respectively. Such genetic effects can be reduced in a number of ways such as selection for ease of calving, which has been reported to reduce the incidence of dystocia in heifers and thus reduce uterine infections (Carnier et al., 2000; Potter et al., 2010). However, this should be practised with caution since it has also been reported that over use of a bull with calving ease may result in cows that are small in size, when practised over a long period of time (Senger, 2003). Larson and Tyler (2005) and Buckley et al. (2010) showed that it was necessary to breed heifers at 24 months of age, as by this time the pelvic diameter will have grown enough to minimise cases of dystocia and associated postpartum complications. Other studies have reported decreased postpartum uterine disease when cross breeding was practised compared to pure breeds, for example, Holstein x Jersey bulls showed less cases of dystocia compared with pedigree Holsteins (Maltecca et al., 2006), thus crossbreeding may be a good strategy in controlling some of the postpartum complications.

**Impact of uterine infections**

The main goal for the postpartum reproductive health in dairy cattle is for the uterus to undergo complete involution (return to its normal size and position), free from infection and for cows to have cyclic activities by the time they enter the breeding period (LeBlanc, 2002; Gautam et al., 2009). However, studies have reported that cows with RFMs and dystocia tend to have delayed uterine involution creating a greater risk for uterine infection due to the increased opportunity for bacteria to colonise the uterus (Abdelhameed et al., 2009; Opsomer and Kruif, 2009; Gunduz et al., 2010). Similarly, RFMs and difficult calving have been shown to result in a prolonged postpartum interval to conception, with increased days open and reduced milk production (Moss et al., 2002; Bereille et al., 2003; Quintela et al., 2004; Buckley et al., 2010). Interestingly, Kaneko et al. (1997) found no significance association in subsequent reproductive performance in cows which had RFMs and those without. The results suggested that injury to the uterus during cases of RFMs mostly heal within two months post-calving. Contrastingly, Mateus et al. (2002) found that puerperal metritis significantly slowed down uterine involution and caused a greater damage to the tissues of the uterus while Farin et al. (1989) concluded that pyometra affects ovarian activities, resulting in a lack of oestrus in cows. Sheldon et al. (2004) and Opsomer and Kruif (2009) showed that contamination of the uterus by pathogens such as bacteria in cows which had RFMs and dystocia can affect fertility due to the disruption of the normal endocrine control of ovarian activity, disrupted follicular development and more so, the tendency to prevent the establishment of pregnancy by the presence of pathogenic organisms in the uterine lumen. Thus, there is a demonstrable need to understand the risk factors for postpartum uterine infections and how best to reduce their effect on reproductive performance in dairy cows performance.

It has been estimated that for every 100 cows served for the first time, only about 30% live births occur, suggesting that uterine infection is one of the leading causes of reduced fertility in many dairy herds (Dobson et al., 2007). A study by Buckley et al. (2010) in Northern Ireland found a significant association between serious cases of dystocia and reduced...
conception rate after the end of breeding season compared to unassisted cows. This was because dystocia was highly associated with cases of clinical endometritis (Bell and Roberts, 2007). In addition, cows with dystocia had a significant increase in calving interval and repeated cases of dystocia in subsequent parturitions. Similarly, Bell and Roberts (2007) also reported that cows which experienced difficult calving had a reduced cycling rate in the preceeding breeding season compared to cows that experienced normal calving while Opsomer and Kruif (2009) reported that cows with RFMs were at 2 times a higher risk of metritis with a subsequent effect on fertility. LeBlanc (2002) found that conception rate was 20% lower in cows with endometritis, with a median calving to conception interval of 30 days longer and that about 35% more animals were culled for failure to conceive. Similarly, McDougall (2001b) and Kim (2006) showed increased culling and reduced pregnancy rates in cows with uterine infection while Gilbert et al. (2005) found endometritis was highly significant for reduced pregnancy rates where days open was 218 and 118, first service pregnancy rate 11% and 36% and pregnancy until day 300 postpartum 63% and 89% in cows with or without uterine infection respectively in a sample of dairy herd in the US. Gautam et al. (2009) found that clinical endometritis diagnosed in the late postpartum period (29-60 days) decreased pregnancy rate, albeit there was no significant effect in days to first insemination. Kasimanickam et al. (2004) found out that pregnancy rate after the second insemination in cows diagnosed with subclinical endometritis was 50% lower compared to healthy cows. Similarly, number of days open (100 vs 162) and pregnancy rate (35 vs 65) was worse for cows reported with clinical endometritis.

McDougall (2001b) showed that delayed calving was associated with cows which had previously been diagnosed with endometritis, resulting in a pregnancy rate of 54%, four weeks after the start of breeding season compared to 68% in healthy cows in the herd. Garcia et al. (2003) reported a negative association in cases of puemeral metritis on the number of artificial inseminations per gestation, intervals partum-conception and partum-first insemination (0.54, 46 vs.15 and 18 vs. 24) respectively. Similarly, Santos et al. (2009) and Plontzke et al. (2010) showed that subclinical endometritis did not affect overall dairy cow reproductive performance. On the other hand, Hammon et al. (2006) reported decreased neutrophil activity in cows with subclinical endometritis. Chapwanya et al. (2008) and Bruno (2009) showed that the increased use of systemic antibiotics in cases of postpartum uterine infections resulted in antibiotic residues in meat and milk and, more so, antibiotic resistance, with a negative impact on public health while Bademkiran et al. (2009) and Bruno et al. (2009) reported that additional losses were as a result of discarding and the withdrawal of milk and meat.

A study conducted by Djemali et al. (1987) on dairy cows involving over 141,000 lactations with assisted calving showed that first-calf heifers experienced a dystocia score of 5 (very difficult) and produced less milk and milk fat of 465 kg and 20 kg respectively. The impact worsened with increased parity, where cows in their second and third parities with a dystocia score of 5 produced 1300 kg and 45 kg less milk and milk fat when compared with age contemporaries with a dystocia score of 1 (least difficult). Similarly, Dematawewa et al. (1997) showed that dystocia reduced average milk production by over 38 kg per cow in a study involving over 123,000 lactations. Rajala and Grohn (1999) found that cows which had experienced cases of dystocia, RFMs, and early postpartum uterine disease showed a significant reduction in milk yield while late uterine infections did not show any milk losses. A more recent study by Konyves et al. (2009a) found that cows with dystocia and RFMs showed a reduced milk, milk fat and milk protein production in the initial 100 days of lactation while Zaborski et al. (2009) reported that dystocia was significant for metritis and elevated somatic cell count. Thus, dystocia and RFMs have a significant effect on milk yield.

Urton et al. (2005), Huzzey et al. (2007) and Bell and Roberts (2007) reported that cows diagnosed with severe cases of uterine infections had a reduced dry matter intake and feeding time than cows that were healthy. In addition, sick cows had fewer aggressive interactions at the feed bins compared with healthy cows. The findings showed that uterine infection can have a great impact on dairy cow health and welfare and the subsequent reproductive performance.

Sheldon and Dobson (2004) highlighted that the financial losses associated with uterine infections are dependent on the reduced milk yield, cost of treatment and subfertility. A study by Kossaibati and Esslemont (1997) on the cost of postpartum complications in dairy herds in the UK found that the direct cost of a case of RFM, which is also a risk factor for metritis, was about £80 per cow per month. In addition, RFMs also resulted in reduced fertility, thus the estimated cost of treatment per cow was about £300, taking into consideration losses from culling and increased calving intervals. Herath et al. (2005) and Sheldon et al. (2008) showed that direct cost from low milk production and treatment of uterine disease in the UK on a national basis was more than £16 million per annum.

In one recent study, dystocia which has also been documented as being one of the potential risk factors for uterine infection, was reported to cost...
between US $96 to $397 in most dairy farms in the US, depending on the level of assistance required, while the estimated cost of a case of metritis ranged between US $330 and $386 (Bruno, 2009; Olson et al., 2009). Using a conventional incidence rate of 20% for metritis, the annual cost of uterine infection in the European Union has been estimated to be over 1 billion Euros and US $650 million in the US (Sheldon et al., 2009). It was estimated that a worldwide significant loss of 2.5 billion Euros per annum on the dairy industry was due to postpartum uterine infections (LeBlanc et al., 2002b).

**Conclusion**

Postpartum uterine infection remains a major challenge in the dairy cattle industry affecting fertility and overall reproductive performance thus, highlighting the need to look carefully into mitigating factors and thereby minimise losses. Herd owners should have a programme which involves frequent visit by farm veterinarians to monitor herd fertility with particular emphasis on detailed clinical examination of cows that have calved recently and more so those that show no signs of oestrus or have failed to conceive. Dairy farmers should also seek help from nutritional experts and adopt strategies on feeding management for different groups of cows, particularly the dry group during the transitional period. Additionally, farmers should seek advice from breeding experts on sire selection so as to use sires with less calving difficulty on heifers and on the selection of those breeds that have less history of post calving anomalies. Where necessary culling should be aimed at cows that have lost foetal membranes. Theriogenology, 34: 1025-1034.


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Onyango.....Cow postpartum uterine infection: A review of risk factors, prevention and the overall impact


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