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Risk factors for clinical endometritis in postpartum dairy cattle

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Abstract

Bacterial contamination of the uterine lumen after parturition occurs in most dairy cattle. The presence of clinical endometritis beyond three weeks post partum depends on the balance between microbes, host immunity, and other environmental or animal factors. The present study tested the hypothesis that clinical endometritis is associated with animal factors, such as retained fetal membranes, assisted calving and twins, as well as fecal contamination of the environment. The association between selected risk factors and the lactational incidence risk of clinical endometritis was examined in 293 animals from four dairy herds. Multivariate analysis was used to identify risk factors and quantify their relative risk (RR) and population attributable fraction (PAF) based on the proportion of cows exposed to each factor. The lactational incidence of clinical endometritis was 27% and significant risk factors for clinical endometritis were retained fetal membranes (RR = 3.6), assisted calving (RR = 1.7), stillbirth (RR = 3.1), vulval angle (RR = 1.3), primparity (RR = 1.8), and male offspring (RR = 1.5) but not the cleanliness of the environment or the animal. The highest PAF was associated with male offspring (0.6) so the use of sexed semen has the greatest potential to reduce the incidence of clinical endometritis. The dominant association between retained fetal membranes and clinical endometritis was supported by an expert panel of clinicians. The risk factors for clinical endometritis appear to be associated with trauma of the female genital tract and disruption of the physical barriers to infection rather than fecal contamination. (© 2010 Elsevier Inc. All rights reserved.

Keywords: Bovine; Clinical endometritis; Postpartum; Risk factors

1. Introduction

Bacterial contamination of the uterine lumen in the first two weeks after parturition occurs in 80–90% of dairy cattle [1]. For several weeks after parturition there is a cycle of bacterial contamination, clearance, and recontamination. In many animals this bacterial contamination is gradually resolved by uterine involution,

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passage of lochia out of the uterus, and through the mobilization of immune defenses. However, failure to resolve the contamination can compromise uterine function, and the persistence of pathogenic bacteria for at least 3 weeks postpartum causes clinical endometritis in 10–20% of postpartum dairy cattle [1–4]. Clinical endometritis is associated with tissue damage, delayed uterine involution, disruption of endometrial function, and perturbation of ovarian cycles [5–8]. Clinical endometritis is characterized by a purulent discharge from the uterus, which is often detected in the vagina of affected animals [1,3,4]. Clinical endometritis causes infertility at the time the disease is present, and

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subfertility even after successful resolution of the disease. In several studies, the impact of clinical endometritis on dairy herds was about 20% lower conception rate, 30 d longer median calving to conception interval, and 3% more animals culled for failure to conceive [1,3,4].

The bacteria associated with clinical endometritis are Escherichia coli, Arcanobacterium pyogenes, Fusobacterium necrophorum, and Prevotella species [7.9.10]. It is assumed that these organisms are acquired from the feces and fecal contamination of the animal coat, bedding, and the environment. However, the role of the animal's environment and, in particular, whether fecal contamination is a risk for clinical endometritis, has often been neglected. This is unfortunate because a key question for designing programmes to prevent disease is whether animal or environmental factors are most important. The risk factors that have been previously established for uterine disease include abnormal length of gestation, stillbirth, twins, assisted parturition, retained fetal membranes, or a caesarean section operation [11–16]. However, in many studies, the disease outcome that is examined is metritis during the first 3 weeks postpartum, rather than clinical endometritis, which is limited to disease after 3 weeks postpartum [17]. In some studies the case definition of the uterine disease encompasses metritis and clinical endometritis. In others, risk factors for uterine disease have only been identified individually by unadjusted or univariate analysis of their association with the disease

The present study tested the hypothesis that clinical endometritis is associated with animal factors, such as retained fetal membranes, assisted calving, and twins, as well as fecal contamination of the environment. We determined the association between selected risk factors and the lactational incidence risk of clinical endometritis as defined by the presence of >50% purulent material in the uterine discharge detectable in the vagina 21 d or more after parturition, or mucopurulent (approximately 50% pus, 50% mucus) discharge detectable in the vagina after 26 d postpartum [17]. In addition, expert veterinarian opinions of the risk factors considered important for clinical endometritis were examined. The purpose of the work is to generate knowledge that will inform the design of control programmes to prevent clinical endometritis.

2. Materials and methods

2.1. Animals

A prospective study enrolled all cows that calved during 1 yr from 4 Holstein-Friesian dairy herds. The convenience sample of herds in Somerset and Hertfordshire was selected based upon 2 main criteria. First, the farm had a good recording system with veterinary involvement in management of data using herd health computer software (Interherd; NMR, Chippenham, UK). Second, the farm had an established arrangement of weekly visits to the farm by a veterinarian for fertility management. The herds had all-year-round calving patterns with average calving to first insemination intervals ranging from 76-88 days, and calving to conception intervals of 92-140 days in the year preceding the present study. Cows received maize and grass silage while housed during winter, and grazed grass in the summer. Animals were enrolled at the weekly farm visits when animals 21-28 d postpartum were presented for veterinary examination.

2.2. Clinical examination

Animals were examined by a veterinarian (TP) and all procedures conformed to EC Directive 86/609/ EEC. The cleanliness of the hind quarters was assessed and assigned a cow cleanliness score based upon previously published criteria: (1) completely free of dirt or has very little dirt; (2) slightly dirty; (3) mostly covered in dirt; or (4) completely covered, caked in dirt [18]. Fecal consistency for each animal was assessed and assigned a numerical fecal consistency score based upon the following published criteria: (1) very dry, lumpy; (2) dry, stiff, semiformed pats; (3) circular, moist raised pat with petal like symmetrical rings surrounding a dipped centre; (4) flat, loose, thinly spread; or (5) liquid pools of feces [19]. Body condition was assessed by examination of eight areas of the cow's body, using established criteria [20]. Briefly, animals were assigned a body condition score along a 1 to 5 scale with 0.25 unit increments: score 1 indicated emaciated condition and score 5 an obese condition. The angle of the vulva was recorded as abnormal if less than 70° to the horizontal axis judged using a protractor.

The presence of clinical endometritis is usually determined by identifying pus in the lumen of the vagina with a speculum or by withdrawing the contents of the vagina by hand or with a Metricheck device [7,21–23]. There is a high level of agreement for diagnosis of clinical endometritis between the speculum method and manual examination of the contents of the vagina [23]. The presence of vaginal pus also correlates with the presence of polymorphonuclear cells detected

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