INTRODUCTION

The following diseases will be discussed in this paper: interdigital phlegmon, interdigital dermatitis, heel horn erosion, and digital dermatitis. They will be discussed as separate diseases although there has been considerable discussion about whether interdigital dermatitis and digital dermatitis are different manifestations of the same disease and also whether interdigital dermatitis and heel horn erosion are part of the same complex. There is ample evidence that these diseases occur under the same conditions, i.e. poor foot hygiene.

INTERDIGITAL PHLEGMON (FOOTROT, INTERDIGITAL NECROBACILLOSIS, FOUL IN THE FOOT)

The primary signs of interdigital phlegmon are fissures and caseous necrosis of the interdigital skin and diffuse, symmetrical digital swelling. Pain, moderate to severe lameness and pyrexia are also common signs of this disease. A characteristic, fetid odor is usually present due to the presence of \textit{Fusobacterium necrophorum}. A possible sequela is deep digital sepsis (Reinohl-DeSouza et al., 2004).

Clinical Signs and Diagnosis

Clinical signs are acute to peracute, severe lameness with marked swelling around the coronary band and in the interdigital space (Berg and Franklin, 2000, Bergsten et al., 1997, Stokka et al., 2001). It is commonly found in one foot and is more common on rear feet. The first signs of disease are erythema and swelling in the interdigital space and around the coronet. Animals show slight lameness for 18 to 24 hours, which is often missed (Baggot and Russell, 1981). Other signs are pyrexia, decreased feed intake, and decreased milk yield. A visible
necrotizing lesion in the interdigital space and a fetid odor are usually present by the time lameness is seen and diagnosis is based on clinical signs. Culture of *Fusobacterium necrophorum* is rarely used to confirm diagnosis. A form of the disease that is more rapid in onset and less responsive to antimicrobials (super footrot or super foul) has been reported in the UK (Cook and Cutler, 1995) and the US (Guard, 1997).

Septic arthritis might be mistaken for interdigital phlegmon but most septic arthritides involve swelling of only one digit and would not have a necrotizing interdigital lesion (Bergsten, 1999). Deep digital sepsis, however, can be a sequela to chronic or non-responsive interdigital phlegmon (Reinohl-DeSouza et al., 2004).

**Epidemiology and Pathogenesis**

Interdigital phlegmon has been known for centuries and is found worldwide. Occurrence is sporadic for beef and dairy cattle although outbreaks have been reported (Berg and Franklin, 2000, Stokka et al., 2001). It affects all age groups, occurrence is more common during wet conditions, and Jerseys had lower risk than other dairy breeds (Alban et al., 1995, Monrad et al., 1983). Tiestall cows had lower risk than loose-housed cows and first parity cows were highest risk (Alban et al., 1996, Alban et al., 1995). Interdigital phlegmon has a positive association with lameness from other causes (Alban et al., 1995). Very different incidences from year to year on the same pastures were observed with the crude incidence varying from 0.1% to 4.8% over a 12 year period (Monrad et al., 1983).

Trauma or conditions that predispose cattle to interdigital skin damage may allow entry of causative organisms (Baggot and Russell, 1981, Bergsten et al., 1997, Committee, 2006). Examples of such conditions are stones, stubble, pieces of wood, uneven ground, constant moisture, ice, manure slurry, or dried mud.

**Etiology**

The bacteria most commonly isolated from this infection are *Fusobacterium necrophorum* acting in synergy with *Porphyromonas levii* (Berg and Franklin, 2000, Committee, 2006). Other secondary invaders include members of the genera *Prevotella*, *Peptostreptococcus*, and other *Fusobacterium* (Dr. Richard Walker, personal communication). The presence of spirochetes has also been reported (Doherty et al., 1998). Different isolates of *F. necrophorum* have been found to have large differences in virulence, which may account for the occasional reports of “super footrot” or “super foul” (Nagaraja et al., 2005).

**Treatment and Prevention**

Treatment consists of parenteral antibiotics given for 3-5 days (Baggot and Russell, 1981, Berg and Franklin, 2000, Cook and Cutler, 1995, Henke et al., 1994, Morck et al., 1998, Rebhun and Pearson, 1982). The following drugs are commonly used and have a label claim for treatment of interdigital phlegmon in
the US: amoxicillin (IM or SC), ceftiofur (IM or SC), erythromycin (IM), and sulfadimethoxine (PO, IV,) (Anonymous, 2005) Five to ten percent ZnSO₄ or CuSO₄ footbaths are reported to work well for prevention (Bergsten et al., 1997, Rebhun and Pearson, 1982). Prompt treatment is crucial to success. Other management practices to attenuate incidence are good corral and freestall hygiene, proper freestall design and bedding, and removing trauma inducing objects (Baggot and Russell, 1981). *Fusobacterium necrophorum* vaccines may be efficacious for prevention (Checkley et al., 2005, Clark et al., 1986). One Canadian study found that an *F. necrophorum* vaccine was efficacious in preventing foot rot when feedlot cattle were fed a forage based growing diet but was not efficacious when they were fed a grain based growing diet (Checkley et al., 2005).

**INTERDIGITAL DERMATITIS**

Interdigital dermatitis (ID) is an acute or chronic, superficial dermatitis of the interdigital skin (Blowey, 1994a, van Amstel and Bemis, 1998). Lesions are usually painful to touch but often do not cause lameness. The lesions occur on all areas of the interdigital skin, more commonly on the rear feet.

**Clinical Signs and Diagnosis**

Inflammation is confined to the epidermis. Diffuse, multifocal epidermal excavations and erosion in the interdigital cleft is seen in early cases. More chronic cases will show hyperkeratosis, which is visible on the dorsal and palmar commissural skin folds (Blowey, 1994a, Guard, 1995). Lesions are painful to touch and may have a serous exudates and fetid odor. This condition is frequently accompanied by heel horn erosion, and some authors consider interdigital dermatitis/heel horn erosion as one disease (Bergsten et al., 1997, Somers et al., 2005b).

**Epidemiology and Pathogenesis**

Interdigital dermatitis occurs in dairy cattle, especially in wet and/or dirty environments. It is usually an incidental finding since it rarely causes lameness. It often occurs concurrently with heel horn erosion and digital dermatitis.

**Etiology**

Some researchers have speculated that interdigital dermatitis and digital dermatitis may be different forms of the same disease complex (Blowey, 1994b, Read and Walker, 1994b, Walker et al., 2002). Interdigital dermatitis and DD share several histological characteristics; including spirochetal involvement (Blowey, 1994a, Read and Walker, 1994a, van Amstel and Bemis, 1998) and both can be successfully treated and prevented with the same topical antibiotics and/or footbaths (Blowey, 1994a, Blowey and Sharp, 1988). *Dichelobacter nodosus*
and *Fusobacterium necrophorum* may be primary or contributory pathogens (Blowey, 1994a, Guard, 1995, van Amstel and Bemis, 1998)

**Treatment and Prevention**

Prevention is usually accomplished by the use of 5 – 10 % ZnSO₄ or CuSO₄ footbaths. As with the use of footbaths to prevent interdigital phlegmon or digital dermatitis, care must be taken to insure that the footbath solution remains clean. It is possible for interdigital dermatitis to persist on dairies that practice regular footbathing since causative organisms may survive within deep heel cracks that are not permeated by footbath solutions; hence, heel cracks must be trimmed away during hoof trimming to allow for exposure to footbath solutions (Guard, 1995). It is also possible that footbath solutions do not reach the lesions when cows have manure plugs lodged in the interdigital space between the heels (Walker et al., 2002)

**HEEL HORN EROSION (EROSIO UNGULAE, SLURRY HEEL)**

Heel horn erosion is an irregular loss of heel bulb horn and may be mild or progress to complete loss of the heel bulb. It has been associated with prolonged contact with manure slurry. Oftentimes, it is an incidental finding but in the more severe forms, it will cause lameness. Loss of heel horn alters the natural balance of the foot and removes part of the concussion absorption mechanism, which may contribute to underestimating the secondary effects of heel horn erosion (Kempson et al., 1998).

**Clinical Signs and Diagnosis**

Often, heifers or cows with heel horn erosion are not lame but diagnosis is by observation of visible loss of heel horn (Collick et al., 1997). The pattern of erosion can vary from multiple pits in the bulbar horn (mild) to deep, oblique grooves (moderate) to complete loss of the bulbar horn with exposure of the corium (severe).

**Epidemiology and Pathogenesis**

Heel horn erosion is a common lesion in dairy heifers and cows and has a higher prevalence in freestalls than tie-stalls (Bergsten and Herlin, 1996, Sogstad et al., 2005a, Sogstad et al., 2005b). Heel horn erosion is also more common as parity increases (Enevoldsen et al., 1991). Severe heel horn erosion is commonly associated with cows housed in freestalls, but it is also found in cows during the pasture season (Somers et al., 2005b).

One study found the variation in prevalence of heel horn erosion between dairies was very high (Manske et al., 2002). This same study found a strong correlation
of heel horn erosion with digital dermatitis (Manske et al., 2002). Other investigators have considered that interdigital dermatitis and heel horn erosion were one disease (Somers et al., 2005b). Heel horn erosion had lower prevalence when cows had better hygienic conditions for their feet (Bergsten and Herlin, 1996, Hultgren and Bergsten, 2001) and when animals had lower stocking density in deep bedding (Mogensen et al., 1997).

Manure slurry has an adverse effect on heel horn, which is exacerbated if the horn is poor quality (Kempson et al., 1998, Muelling and Budras, 1998). Degradation of the horn is also caused by urine, however, the degradation can be prevented by pretreating the heel horn with formalin but not with glutaraldehyde (Gregory, 2004). Once the heel horn is damaged by manure slurry and urine, it may be more susceptible to invasion by microorganisms.

**Etiology**

*Dicelobacter nodosus* has been implicated in the etiology heel horn erosion and may also be involved in interdigital dermatitis (Toussaint Raven et al., 1989). It is likely that heel horn erosion is a mixed bacterial infection, as are interdigital dermatitis, digital dermatitis, and interdigital phlegmon, with some of the same organisms being involved in all of these.

**Treatment and Prevention**

Heel horn erosion is reduced when footbaths are used (Bergsten and Herlin, 1996) but the finding that there is much variation in prevalence between farms with similar facilities points out that more than footbathing needs to be considered for control to be effective (Manske et al., 2002). Treatment includes paring away loose horn during hoof trimming (Collick et al., 1997, Toussaint Raven et al., 1989) and treatment with topical antibiotic or astringent especially if interdigital dermatitis is concurrent.

**Digital Dermatitis (Papillomatous Digital Dermatitis, Footwarts, Hairy Footwarts, Raspberry Footwarts, Strawberry Footwarts)**

Digital dermatitis (DD) is a multifactorial, superficial dermatitis of the digital skin of cattle and has been identified to be a major cause of lameness in dairy cattle in many countries (Brizzi, 1993, Read and Walker, 1998, Zemljic, 2002). Histopathology, response to antimicrobial treatment, and culture of lesions suggest bacterial etiology. The disease is reported primarily in housed dairy
cattle but there are reports of the disease in beef cattle or dairy cattle on pasture (Brown et al., 2000, Doherty et al., 1998, McLennan and McKenzie, 1996). Digital dermatitis was first described in Italy in 1974 (Cheli and Mortellaro, 1974) and was first reported in the United States in New York in 1980 (Rebhun et al., 1980). The disease has since been reported from many countries (Blowey and Sharp, 1988, Cruz et al., 2001, Kimura et al., 1993, McLennan and McKenzie, 1996, Peterse et al., 1982, Read et al., 1992, van Amstel et al., 1995, Vermunt and Hill, 2004). Economic losses are from reduced milk production (Hernandez et al., 2002), reduced reproductive efficiency (Hernandez et al., 2001), premature culling, and costs of treatment.

**Clinical Signs and Diagnosis**

The majority of DD lesions occur on the plantar, interdigital ridge of the rear foot (Mortellaro, 1994, Read and Walker, 1998). Less common sites for lesions are the plantar, interdigital ridge of a front foot and the dorsal aspect of any foot. Bilateral lesions are found frequently and some have multiple lesions on one foot, but lesions are confined to the digital skin and have not been reported to occur above the level of the dew claws, although there is evidence that udder cleft dermatitis (intertrigo) may share common pathogens (Beattie and Taylor, 2000, Stamm et al., 2009).

The gross appearance of the lesions, characteristic location on hind limbs, and predilection for skin-horn junctions, especially those bordering the heel bulbs and interdigital space distinguish this disease from other types of bovine dermatitis (Blowey and Sharp, 1988, Read and Walker, 1998). Most lesions are 2-6 cm across at their greatest dimension, circular or oval, and have clearly demarcated, raised borders with hypertrophied true hairs often surrounding the border (Read and Walker, 1998). Lesion surfaces may have filiform papillae varying in length from 1 mm to 3 cm and 0.5 - 1 mm in diameter and/or may have a granular surface (Brizzi, 1993, Read and Walker, 1998). Washed surfaces are generally very painful and bleed easily if traumatized.

The erosive lesions slowly enlarge and become proliferative masses 2 to 6 cm in diameter that are red, grey, or black; and oval, spherical, or U shaped (Read and Walker, 1998). Histopathology suggests that the erosive and proliferative forms are the same disease.

Histopathology is helpful to confirm a diagnosis of DD; but is not necessary since lesions are easily recognized by their characteristic appearance and location (Blowey and Sharp, 1988, Read and Walker, 1998). Histopathological criteria to establish a diagnosis of DD are: 1) circumscribed plaque of eroded acanthotic epidermis attended by parakeratotic papillomatous proliferation profusely colonized by spirochete-dominant bacterial flora, 2) loss of stratum granulosum, 3) invasion of stratum spinosum by spirochetes, and 4) infiltration of neutrophils, plasma cells, lymphocytes, and eosinophils in the dermis (Read and Walker,
If biopsies are performed, they should be full thickness 4 or 6 mm punch biopsies, washed off with sterile saline, and placed in buffered formalin.

Results of a biopsy study on treatment and recurrence of DD indicated that the gross visual and histopathological diagnoses were in agreement for active lesions prior to treatment but histopathology on day 28 after treatment with lincomycin or oxytetracycline found a high percentage (53%) of lesions that visually appeared to be healed but still had histologic evidence of infection (Berry et al., 2009). It was not known whether these lesions were recurrent or incompletely healed infections.

Most lesions have a fetid odor due to secondary bacterial growth in the exudate covering the DD lesion. Swelling of the pastern and fetlock regions is not present in uncomplicated cases. Lameness is a herd characteristic on dairies where DD has a high prevalence but is an inconsistent finding on individual infected cattle and is not consistently related to lesion size or stage (Laven and Proven, 2000). Claws of cows with chronic lesions may develop a clubbed appearance, since the cow prefers to bear weight on (and wear down) the toes.

**Epidemiology and Pathogenesis**

Digital dermatitis spreads rapidly when introduced into a naïve herd, often affecting the majority of adults within the first year of infection. When endemic in a herd, lameness is more commonly seen in lower parity cows (Holzhauer et al., 2006, Somers et al., 2005a). Bulls and yearling heifers can also be affected but usually comprise a small fraction of clinical cases. In California, the disease is most severe during the spring and summer months (Read and Walker, 1994b), but in Europe it is most severe in the winter months (Blowey, 1994a, Brizzi, 1993). Freestall herds have higher prevalence than tie stall herds (Manske, 2002, Sogstad et al., 2005b). Cattle confined to pasture are rarely affected (Frankena et al., 1991, McLennan and McKenzie, 1996). One study found that the Holstein breed was at higher risk for DD than a dual purpose breed (Holzhauer et al., 2006).

Epidemiological studies have indicated the following cow and herd level risk factors for DD: 1) muddy or wet conditions (Rodriguez-Lainz et al., 1996b, Wells et al., 1997, Wells et al., 1999), 2) purchasing replacement cattle from off premises (Rodriguez-Lainz et al., 1996b, Wells et al., 1997, Wells et al., 1999), 3) lower parity (Somers et al., 2005a, Wells et al., 1999), 4) the presence of other infectious claw disorders (Holzhauer et al., 2006, Walker et al., 2002), and 5) the use of outside hoof trimmers and not washing hoof trimming equipment between cows (Wells et al., 1999). One study found that animals that had >8 h per day of access to pasture were at higher risk for DD than those that had no access to pasture (Holzhauer et al., 2006) while another study found that cows that had full access to pasture during the summer or had cleaner conditions during the housing period had less risk of DD (Somers et al., 2005a).
Constant moisture and low access to air were necessary for experimental transmission of the disease to calves (Read and Walker, 1996, Read, 1997). These conditions are present on confinement dairies if manure management and hygiene are not adequate. Poor freestall or bedding area management will exacerbate the problem by forcing cows to stand in manure slurry for longer periods and will not allow feet of cattle to dry out periodically.

**Etiology**

Numerous obligate anaerobic or microaerophilic organisms have been associated with DD (Blowey et al., 1994, Borgmann et al., 1996, Choi et al., 1997, Collighan and Woodward, 1997, Demirkan et al., 1998, Demirkan et al., 1999, Doherty et al., 1998, Dopfer et al., 1997, Ohya et al., 1999, Walker et al., 1995). Spirochetes from the genus *Treponema* have been identified the most consistently, comprise the bulk of the colonizing bacterial mat found on active lesions, and are the organism found to invade the epidermis and dermis (Borgmann et al., 1996, Choi et al., 1997, Collighan and Woodward, 1997, Demirkan et al., 1998, Demirkan et al., 1999, Doherty et al., 1998, Dopfer et al., 1997, Ohya et al., 1999, Walker et al., 1995). These spirochetes also produce a humoral response in cows with active lesions (Walker et al., 1997).

Digital dermatitis is multifactorial involving environmental, microbial, host, and management factors (Brizzi, 1993, Read and Walker, 1998, Rodriguez-Lainz et al., 1996a, Rodriguez-Lainz et al., 1996b, Rodriguez-Lainz et al., 1999, Wells et al., 1999, Zemljic, 1994). Any factors that contribute to poor foot hygiene or trauma such as rough flooring, poor drainage, accumulation of feces and urine on floors, dirty, wet, or uncomfortable bedding areas, and overcrowding could increase the risk of DD. The mode of transmission between cows and between herds is currently unclear, although research has found concurrent spirochetal infection of feet and colon in cattle, which may indicate a source for the infecting bacteria (Shibahara et al., 2002).

**Treatment and Prevention**

The most common treatments for DD involve the use of topical antibiotics. Laven and Logue published an eloquent review of DD treatment (Laven and Logue, 2006). Commonly used antibiotics include: lincomycin, oxytetracycline, erythromycin, cefquinome, and valnemulin. There have been no reported antibiotic residue violations due to topical application of antibiotics (Blowey, 1994b, Britt et al., 1999, Brizzi, 1993). There are no antibiotics approved in the US for treatment of DD. In the UK, oxytetracycline in aerosol form and cefquinome as a parenteral treatment have been approved for treatment of DD (Laven and Logue, 2006). Parenteral antibiotics have not been consistently efficacious and require milk and meat withholding (Laven and Logue, 2006).

Footbaths using antibiotics, Cu or Zn sulfate, formalin, or various proprietary products are used on many dairies to control DD (as well as interdigital...
dermatitis and interdigital phlegmon). Most footbath products have not been rigorously evaluated (Laven and Logue, 2006). Some products are dangerous to handle (e.g. formalin) and some pose disposal problems (e.g. CuSO₄). Nonetheless, footbaths are commonly used on dairies to control the infectious lameness causing diseases.

A *Treponema* spp bacterin vaccine was developed in the US. Early field studies were encouraging, which allowed the manufacturer to obtain a full license for sale in the US. A recent blind field study of two commercial dairy herds where half the lactating herd was vaccinated with the bacterin and the other half with a placebo did not find the vaccine to be efficacious (Ertze et al., 2006). The vaccine has since been removed from the market. Recent studies have found several different *Treponema* phylotypes associated with DD, which would make the development of an efficacious vaccine more difficult (Elliott and Alt, 2009, Evans et al., 2009a, Evans et al., 2008, Evans et al., 2009b, Klitgaard et al., 2008, Nordhoff et al., 2008, Pringle et al., 2008, Schlafer et al., 2008, Stamm et al., 2009, Yano et al., 2009).

In spite of what we have learned during the last 30+ years about DD and the other infectious causes of lameness in cattle we still have the problems with no indication that they are under control. Poor foot hygiene (and adequate cow comfort) are still major problems on our modern dairies and footbathing appears to be helpful for prevention of the infectious causes of cattle lameness.

References


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