A Review of Necrobacillosis

Mell Wostoupal

Iowa State College

Follow this and additional works at: https://lib.dr.iastate.edu/iowastate_veterinarian

Part of the Large or Food Animal and Equine Medicine Commons, and the Veterinary Pathology and Pathobiology Commons

Recommended Citation

Wostoupal, Mell (1957) "A Review of Necrobacillosis," Iowa State University Veterinarian: Vol. 19 : Iss. 1 , Article 4. Available at: https://lib.dr.iastate.edu/iowastate_veterinarian/vol19/iss1/4

This Article is brought to you for free and open access by the Journals at Iowa State University Digital Repository. It has been accepted for inclusion in Iowa State University Veterinarian by an authorized editor of Iowa State University Digital Repository. For more information, please contact digirep@iastate.edu.
Necrobacillosis is an infectious disease caused by *Spherophorus necrophorus*, occurring in most animals as a secondary invader and characterized by sharply defined areas of liquefaction necrosis having hyperemic and hemorrhagic borders.

The term includes several disease entities which may be designated by various names, depending on the animal involved and the tissue affected. These entities are calf diphtheria, foot rot, necrotic hepatitis of cattle and sheep, necrotic rumenitis of cattle, and bull nose of swine. Others are intestinal lesions secondary to enteritis in swine and horses, bite and castration wounds, contagious pustular dermatitis and ulcerative dermatosis of sheep, necrotic dermatitis, necrosis following pneumonia, thrush, and quittor in the horse. The condition may also be present in lesions of avian diphtheria, secondary to the viral agent, beak ulceration of chickens and also may be found in the typical “button ulcers” of hog cholera.

It should be noted here that the organism has been found in the ceca of normal swine, and it is likely that it exists in the alimentary tract of other animals also. It is possible that the organism may be introduced by eating out anaerobic condi-

**ETIOLOGY**

*Spherophorus (Actinomyces) necrophorus* is a slender, non-motile, gram negative organism varying from coccoid cells to slender filaments. The bacillus is sensitive to oxygen and under usual conditions does not grow unless good anaerobic conditions are obtained. In many types of necrobacillosis it has been shown that the bacillus is not the primary infectious agent, and that the infection must be initiated by viruses or other bacteria. It is highly possible that other organisms produce anaerobic conditions in the body tissue by cell destruction and normal oxygen consumption necessary for their metabolic processes. Thus, having established vesicles suitable for growth, *Spherophorus* will then proceed to grow and multiply in the anaerobic medium and commonly penetrates epithelial membranes on its way to the secondary lesions.

[This paper was written by Mell Westoupal, a junior, for a pathology course.]

*Issue 1, 1957*
tions are produced by other organisms. It does not seem likely that the bacillus multiplies outside the body but it remains viable in soil and manure for short periods of time. Marsh and Tunnicliff were able to demonstrate the organism in a wet pasture 10 months after sheep with foot rot had run on it, but could not demonstrate it after a second 10 month period. They also determined that under these favorable conditions, it could survive one winter in the rigorous climate of Montana.

Many workers have concluded that an exotoxin is produced by the organism due to the fact that rabbits die after great emaciation when the only lesion is a small area of edema and inflammation. An endotoxin is also produced and can be demonstrated by injecting heat killed cells into laboratory animals. A subsequent inflammation and necrosis follows intradermal injection in the rabbit which is evidence of a heat stable endotoxin. Since the tissue changes, regardless of their location, are essentially the same, only a few of the more common diseases will be described.

CALF DIPHTHERIA

This is one of the more common forms of necrobacillosis which occurs as a fairly acute, infectious and highly fatal disease of young calves. It often attacks whole herds, affecting only a few animals but later in the course of the disease may become widespread. The mucosal membranes of the throat and mouth are affected, thus the names necrotic stomatitis, laryngitis, and pharyngitis are used.

Most writers refer to the bacillus as a secondary invader that attacks only weakened tissue, that won’t develop in a mucous membrane, and that enters through a slight break in the mucosa. Predisposing causes are injuries to the mucosa from sharp objects, such as stubble and barley beards, exposure to filth and eruption of teeth in calves. Since the organism is a strict anaerobe, it does not develop on the mucous membranes but burrows into deeper tissues after gaining entrance through abrasions and waits there for suitable conditions to occur. This is one of the reasons for the 3 to 5 day incubation period in the disease.

The favorite locations of the lesions are on the cheeks adjacent to the molar teeth, at the base of the tongue and on the buccal mucosa, but in more severe outbreaks necrotic lesions may be found in the larynx, trachea, lungs, esophagus and rumen. At first the lesions resemble small scabs but later coalesce to form a necrotic area surrounded by a reddened zone. A offensive odor similar to that of limburger cheese and sulfur is present. Yellow to greenish necrotic material is present at the base of the ulcers and is firmly attached to the underlying tissue. A thick wall of granulation tissue usually surrounds the ulcer. The occurrence of diphtheritic membranes covering the lesions and loss of papillae are common and the membranes may completely block the respiratory efforts.

A similar condition occurs in swine also but usually affects the hard palate and teeth. It may loosen the teeth and invade the bones of the head as well as causing large caseous nodules on the skin of the legs similar to those found in the mouth of the calf.

Microscopically, the infection shows coagulation necrosis of the cells and pyknosis and karyolysis of the nuclei. The inflammatory exudate is usually of the fibrinous type but at times deep coagulation necrosis of the mucous membrane occurs and a fibro-necrotic or diphtheritic membrane is formed. The organism is usually located at the edge of the living tissue in a zone of inflammation where many white blood cells and fibroblasts are also found.

Since the disease is one of young calves, the animal often experiences difficulty in nursing. Loss of appetite occurs and due to the painful buccal mucosa a tuft of roughage may be taken, mouthed slightly, and then dropped dejectedly to the ground. Temperature elevation, salivation, swelling of the cheeks, protrusion of the tongue and drooling are some of the early symptoms. Respiration may become difficult or accelerated due to a na-
sal discharge and coughing occurs if lung involvement is present. Calves become very dehydrated, emaciated and stand with a slightly arched back. Animals affected are those confined to areas which may be contaminated with the organism, such as stables, while cases on the range are few in number. Filth, damp and unclean stables, and traumatic agents tend to increase the incidence of the disease.

FOOT ROT

Necrobacillosis of the foot as defined by Flint and Jensen is a necrotizing infection of tissues immediately proximal to the coronary band or of interdigital tissues often complicated by arthritis of the coffin joint, and caused in part by Sphero- phous necrophorus, penetrating from the surface. This definition is based on work done in Wyoming experiments and does not necessarily adequately describe the primary etiological agent in all parts of the country. In the Midwest and especially in Iowa the organism Corynebac- terium pyogenes is found more consistently than Sphero- phorous necrophorus in the lesions of foot rot and therefore the latter organism is considered by many to be only a secondary invader. It is possible that there is often confusion here between actual foot rot and similar lesions of the foot termed foot abcesses or non-contag- ious foot rot. Reports from Australia state that the primary causative agent of foot rot is Fusiformis nodosus, but Sphero- phorous necrophorus enters before typical lesions develop. Fusiformis nodosus is a strict parasite that does not survive for more than a few days away from the feet of infected sheep and goats. A similar condition may be present in the previously mentioned case.

In the past it was thought that in order for the organism to gain entrance into the tissues a maceration of the thin skin of the interdigital area due to wet, filthy conditions and traumatic agents was necessary. In the past few years however, foot rot has been seen more often in cattle on bluegrass pastures during the dry season of the year. It seems that the moisture problem is of primary im- portance along with traumatic agents in serving to weaken and erode the epithelial and horny structures of the foot which can later be penetrated by the organism if it is brought in contact with the foot. Another explanation of the dry pasture type of the disease evolves around the fact that Sphero- phorous itself is not capable of producing the lesions unless certain conditions are established. If an animal is subjected to wet conditions in the spring of the year it may have only a few organisms penetrating the hoof and may require several weeks or months to develop suitable conditions for adequate growth of the organism, in order for lesions to develop. These lesions may not be evident until after the animal is on dry pasture later on in the summer or in the fall.

A recent survey in New Zealand showed that the incidence of foot rot in pigs was considerably higher in concrete piggeries than those pigs raised on dirt or board floors. Foot rot appeared to increase as the floor surface increased in roughness and was generally the highest, about 19 per cent, in those pigs which were allowed to run "on and off" of the concrete. The explanation may be that pigs feel are worn and injuries develop on the concrete, allowing the bacteria to enter the damaged hoof, especially when the pigs are allowed to run out in muddy conditions.

In conjunction with the above data some additional studies were made attempting to determine the effect of alteration of the normal diet with milk products and garbage. It was determined that pigs fed garbage and whole milk were less susceptible to the disease than those fed on skimmed milk alone. Those on the deficient diet were evidently low in hoof forming elements, allowing entrance of the organism.

It has been demonstrated by the Montana Veterinary Research Laboratory that the infectious agent lives on the ground or bedding only a short period of time after infected sheep have been removed from the premises. These findings were altered when it was found that a

Issue 1, 1957
pasture severely infected produced necrotic lesions on the soles of sheep pastured there 1 year later. Spherophorus was isolated from the lesions but they were not typical lesions showing that certain unknown conditions or organisms are necessary for actual foot rot to occur.

The incubation period has been experimentally estimated at about 10 to 14 days in sheep but in experimental injections of cattle feet, symptoms were observed in 48 hours after injection of Spherophorus into the common digital artery. The resistance of the animal and secondary conditions present, account for the difference in the artificial lesions.

One of the most evident symptoms of the disease is severe lameness in one or more feet which often throws an animal off feed and causes him to stand with the foot elevated. There is usually no involvement of tissues above the hoof and in advanced cases the horn may become deformed and overgrown.

The initial lesion is a tenderness and reddening of the medial face of the claw or a surface necrosis of the soft horn of the heel. The lesions progress to other areas of necrosis on the bulb of the heel and the medial face of the digits. After extending through the soft tissue it may extend to the corium of the sole and about 2 weeks after infection the entire sole may become involved. The process then extends from the sole to the laminae of the wall so that within 30 days after the initial lesions, the laminae are involved. Exudate is usually absent but chronically infected coffin joints discharge exudate continuously through tracts which open into the interdigital space and occasionally a tract opens at the coronary band on the abaxial surface. This exudate has an odor typical of the lesions.

Histologic sections of naturally infected feet were made in Wyoming experiments and on the basis of pathologic changes, they were classified into eight groups: dermatitis, necrosis of the interdigital tissue, arthritis of the coffin joint, osteitis, laminitis, inflammation of connective tissue, arthritis of the pastern and fetlock joints and miscellaneous. The first three groups are related because they are in close anatomic proximity, contained Spherophorus, and were infected by penetration from the surface. Therefore these three conditions were termed necrobacillosis while the others were considered secondary involvements and didn’t necessarily contain the organism. Death is due to the infections of the feet and complications such as toxemia although in fatal cases there are no lesions on the viscera or internal organs.

Foot rot must be distinguished from the foot lesions of: (1) foot and mouth disease, (2) contagious ecthyma, (3) ulcerative dermatosis, (4) other suppurative conditions of sheep called foot abscesses or non contagious foot rot and (5) infections from Erysipelothrix rhusiopathiae. Differential diagnosis is difficult and is usually based on the location of the primary lesions, whether or not the hoof is involved and the exudate, if present.

HEPATIC NECROBACILLOSIS AND RUMENITIS-LIVER ABSCESS COMPLEX

Hepatic necrobacillosis is an infectious noncontagious disease characterized by single or multiple abscesses of the bovine liver and is caused by Spherophorus necrophorus which enters as a secondary invader. The above statement is intended to mean that the hepatic abscesses occur secondarily to gastric injuries which constitute what is known as the rumenitis-liver abscess complex.

Hepatic abscesses occur in fattened cattle and lambs primarily and only occasionally in unfattened animals. The abscesses are responsible for economic losses to packers by necessitating condemnation of livers and to producers by causing occasional deaths and by reducing food utilization in feeder animals.

Because the internal lesions of necrobacillosis have been limited to the liver and because Spherophorus is normally found in the alimentary tract it is logical to assume that lesions of the alimentary tract may permit the organism to penetrate into the portal blood. It is possible
for the organism to enter the portal circulation by traumatic gastritis, nontraumatic gastritis or by points of entrance posterior to the stomach. The organism is known as a secondary invader of vesicular lesions which serve as mediums from which the bacillus can multiply. The finding of vesicular lesions in the rumens of fattened cattle causes this condition to conform to the other types of necrobacillosis.

Experimentally infected cattle and sheep using a bacterial extract injected by way of the portal route showed a relatively short incubation period of 2 to 3 days with maximum nodule formation in 20 days. This period is undoubtedly shorter than the natural infection due to the relatively poorer permeability of the rumen wall.

The disease is usually sporadic in cattle and often affects animals of from 2 to 3 years of age. There is often a reduced milk flow, depression, constipation, abdominal pain, anorexia, pulse of 80–100 per minute, respiration of 30–40 per minute, and a temperature of 104–106 degrees. In acute types there may be grunting, an arched back, stiffness and a tendency to remain down. There may be associated lung lesions due to metastasis involving the pleura which results in fast breathing, slight nasal discharge, cough and pain on percussion over the heart.

The lesions of the rumen are often as important as those of the liver and should be examined in making a diagnosis. Gross pathology of the rumen shows both an acute and chronic rumenitis. Acute rumenitis is characterized by hyperemia, hemorrhagic mucosa, edema, gas bodies and necrosis. Chronic rumenitis is characterized by depigmentation, scars from healed vesicles, thickening of mucosa and submucosa from fibrosis, pits in the epithelium, nodules or vesicles and ulcers. A few necrotic foci in the liver do no harm but extensive nodules with accompanying peritoneal adhesions often cause death. An enlarged liver with rounded edges and dark yellow nodules two inches in diameter with a clay consistency are usually scattered throughout the organ. Subperitoneal emphysema, distension of the gall bladder with bile from pressure exerted by the nodules, necrotic foci in the pleura, diaphragm, heart, spleen and kidneys also may be evident on post mortem examination.

Histopathological examination of the liver nodules shows necrosis near the periphery of the nodules with the organism abundant there. The rumen epithelium is necrotic and contains vesicles which are filled with leucocytes and bacteria, including Spherophorus necrophorus. Necrosis and thrombosis are extensive and the exudate consists primarily of fibrin, serum and neutrophiles. In chronic rumenitis the epithelium becomes thickened and the submucosa contains epithelial nodules composed of concentric layers of keratinized epithelial cells which are liberated in response to the chronic irritation. Eosinophiles, lymphocytes, macrophages and giant cells are
prominent in the submucosa, especially surrounding the lymphatics.

In early stages of acute multiple abscess formation, affected animals are sick clinically and may die. In chronic stages of multiple abscess formation and during the entire course of single abscess formation affected animals are not sick clinically and, consequently continue to eat and gain in body weight. After the symptoms are distinct, death occurs within a period of 2 weeks due to the severe toxemia developed from the liver malfunction.

Recent experimentation has been done on the causes of rumenitis occurring in fattened beef cattle which subsequently lead to bacterial invasion and hepatic necrobacillosis. Although not specifically identified, the irritant was introduced into the anterior ventral sac of the rumen by, (1) feeding a ration in which the ratio of concentrate was high and (2) by rapidly changing from a ration high in concentrate.

Necrobacillosis of the liver has been recognized for many years as the cause of deaths in lambs from 1 to 3 weeks old. The bacteria enter the body of the lamb by way of the umbilical cord soon after birth and spread from the umbilical to the portal vein causing multiple necrotic foci of infection in the liver. This type of infection does not involve the digestive tract.

Another condition involving considerable losses of lambs showing necrobacillosis often occurs but the lesions involve the mucosa of the rumen as well as adhesions of the liver to the diaphragm and extension of the necrotic processes to the lung. This condition appears to be similar to the one occurring in feeder lambs as complications of contagious ecthyma.

The above type of necrobacillosis is typical in that a primary infection of the rumen occurs with a secondary invasion of the liver, diaphragm and lungs. Favorable conditions are those of poor sanitation in early spring lambing where excess moisture makes it possible for the lamb to ingest considerably large amounts of organisms from the udder and contact with the ewe. At this early age the rumen mucosa is not well developed and is susceptible to invasion by the bacteria which also may be picked up from the ground.

**Clinical and Laboratory Studies of Novobiocin—a New Antibiotic.** The results of laboratory and clinical studies of a new antibiotic, novobiocin, were presented. Staphlococci were remarkably sensitive in vitro, and there was no cross resistance with other antibiotics. Pneumococci and Group H streptococci were also inhibited by low concentrations, but they were less sensitive than staphlococci. Among the Gram-negative bacilli, members of the proteus group were moderately sensitive.

Novobiocin was less effective in the presence of a larger inoculum of bacteria, and serum exerted a marked inhibitory effect. Serum concentrations performed by dilution of patients serum in broth were considerably higher than those obtained with other antibiotics although the method used did not reflect the factor of serum inhibition. The daily dose was two grams administered orally, and side effects were minimal. Two patients developed rashes which may have been caused by novobiocin.

Results in 75 patients with a variety of clinical infections were in general favorable, and they appeared comparable to those obtained with erythromycin. In staphlococcal infections in which prompt healing did not occur, the infecting organisms rapidly became resistant to novobiocin. For this reason it is recommended that novobiocin be used exclusively for treatment of infections caused by antibiotic-resistant staphlococci. Whenever possible another antibiotic to which the organism is also sensitive should be administered simultaneously.