Bovine Diseases

By LTC Dana E. McDaniel
Agenda

- Anthrax
- Brucellosis
- Clostridial Diseases
- Foot and Mouth Disease
- Hemorrhagic Septicemia
- Bovine Tuberculosis
Anthrax

• Etiology
  – Zoonotic disease of livestock causing sudden death in grazing animals and serious economic loss to farmers
  – Caused by spore forming bacterium, *Bacillus anthracis*
  – Incidence of the disease may be high during drought or following flooding

• Transmission
  – Transmission via infective spores in soil; spores remain infective for many years
  – Spores ingested while grazing
  – Pastures infected by animals that died of anthrax and spores released into soil as the carcass decomposes
  – Spores may also enter the body by inhalation or through the skin
Anthrax

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Anthrax

• Species affected
  – Affects wild and domestic herbivores: cattle, sheep, goats, camels
  – Can also affect humans exposed to tissues from infected animals, contaminated animal products, or directly by the spores (zoonotic)
  – Never eat the meat of animals suspected of dying from anthrax
Anthrax

• Clinical signs
  – Incubation period is 3-7 days
  – Peracute form has sudden onset and rapid death
  – Acute form has abrupt fever and a period of excitation followed by depression, stupor, respiratory or cardiac distress, staggering, convulsions and death
  – Body temperature may reach 41.5C, rumination ceases, milk production is reduced, and pregnant animals may abort
  – There may be bloody discharges from body openings
Anthrax

• Pathologic findings
  – Never open the body of an animal that is suspected of dying of anthrax (do not butcher)
  – Bacteria survive in the pasture for many years as spores
  – Rigor mortis is absent or incomplete
  – Dark, tarry blood may ooze (fails to clot) from body openings with marked bloating and rapid body decomposition
  – Enlarged, dark red or black, soft, semifluid spleen is common
  – Liver, kidneys, and lymph nodes are congested and enlarged
  – Meningitis may be found if the skull is opened
Anthrax

Multiple foci of hemorrhages and fibrin debris (submaxillary Inn.)
Anthrax

- Diagnosis
  - Difficult to diagnose by clinical signs alone
  - Confirmatory lab exam should be attempted if anthrax is suspected
  - Diagnosis by lab confirmation: submit cotton swab dipped in the blood and allowed to dry
  - Lab tests may include bacterial culture, PCR, and fluorescent antibody stains to see the agent in blood films
  - Differentiate from other “sudden death” diseases such as: clostridial infections, bloat, and lightning strike
  - Also, consider acute leptospirosis, bacillary hemoglobinuria, anaplasmosis, and acute poisonings by bracken fern, sweet clover, and lead
Anthrax

• Treatment
  – Implement a preventive program to reduce losses among livestock
  – Livestock at risk should be treated with a long-acting antibiotic such as oxytetracycline and then vaccinated 7-10 days after the antibiotic treatment
  – Any animals becoming sick after initial treatment and or vaccination should be retreated immediately and revaccinated one month later
Anthrax

• Prevention and Control
  – Through vaccination programs, rapid detection and reporting, quarantine, treatment of asymptomatic animals (postexposure prophylaxis), and burning or burial of suspect and confirmed cases.
  – Vaccinate livestock 2-4 weeks before the grazing season
  – Vaccination protection lasts for about one year and should be repeated annually
Brucellosis

• Etiology
  – Caused by *Brucella abortus* bacterium; also called Bang’s disease

• Transmission
  – Spread by contact with aborted tissues and fluids
  – Discharges then contaminate pasture and feed
  – Infection usually occurs via ingestion, but may also occur through the skin or eye
  – Zoonotic disease, so wear gloves when handling aborted fetuses and burn or bury any placentas and fetuses not needed for diagnostics and pasteurize milk for human consumption
Brucellosis

- **Clinical signs**
  - Causes abortions in the second half of gestation (usually about 7 months), produces weak calves, retained placenta or causes cows to have trouble breeding back
  - Abortion or stillbirth occurs 2 weeks to 5 months after initial infection
  - Orchitis and inflammation of the accessory sex glands may occur in males
Brucellosis

• Pathologic findings
  – Affected cotyledons may be normal to necrotic, and red or yellow
  – The intercotyledonary area is focally thickened with a wet, leathery appearance
  – The fetus may be normal or autolytic with bronchopneumonia
Brucellosis

Fibrin on lungs of bovine fetus

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Brucellosis

Chronic active purulent periorchitis

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Brucellosis

• Diagnosis
  – Diagnosis should only be attempted if fetal loss is >3-5% per year or per month due to low diagnostic success rate and high cost of lab work
  – Diagnosis made by maternal serology combined with fluorescent antibody staining of placenta and fetus or isolation of *B. abortus* from placenta, fetus, or uterine discharge

• Treatment
  – Treatment is unsuccessful
Brucellosis

- Prevention and Control
  - Use vaccines for prevention and control
  - Test and slaughter of carrier cows combined with calfhood vaccination are required for eradication
  - New bulls should be quarantined for 10-14 days before introducing to the herd in order to evaluate their health status and prevent transmission of venereal disease
  - Purchase bulls only from herds with a good herd health program and with a known health status
Clostridial Diseases

• Etiology
  – Caused by *Clostridia* bacteria which are large, anaerobic, spore-forming, rod-shaped organisms
  – Usually fatal; rapid death with blackquarter and pulpy kidney disease
  – Many names, depending on specific bacteria
    • *Cl. chauvoei* – blackleg/blackquarter; affects cattle and sheep
    • *Cl. haemolyticum* – bacillary hemoglobinuria; affects cattle and sheep
    • *Cl. perfringens* – types B, C, and D – enteroxemia, pulpy kidney disease; affects cattle, sheep and goats
Clostridial Diseases

• Transmission
  – Clostridial organisms are common in soil and the intestinal tracts of animals and are usually harmless
  – Under the right conditions, the bacteria grow rapidly and release toxins, quickly destroying tissue and often causing death
  – Flooding of low lying pasture may also bring the bacteria to the surface and increase the risk of exposure
  – These diseases are not contagious
Clostridial Diseases
Blackleg/Blackquarter

• Clinical signs
  – High incidence in summer and fall
  – Often affects the biggest and healthiest animals
  – In cattle, mostly affects those 6 months to 2 years old
  – In sheep, usually follows an injury or development of a wound
  – Sudden onset with a few animals found dead without signs
  – Acute lameness and marked depression
  – Initial fever, but normal to subnormal temperature once clinical signs begin
  – Edematous and crepitant swellings develop in hip, shoulder, chest, back, neck and elsewhere
  – Swelling is small, hot, and painful at first
  – As progresses, swelling enlarges, there is crepitation on palpation, and the skin becomes cold and insensitive as the blood supply diminishes
  – Death occurs in 12-48 hours
Clostridial Diseases
Blackleg/Blackquarter

• Pathologic findings
  – Edematous and crepitant swellings in hip, shoulder, chest, back and neck
  – Affected muscles are dark red to black, dry and spongy
  – Sweetish odor to muscle and is infiltrated with small bubbles, but with little edema
  – Lesions are small in sheep and in deep tissues, so may be overlooked
Clostridial Diseases
Blackleg/Blackquarter

Dark red to black of muscle often with a distinct odor of sour milk.
Clostridial Diseases
Blackleg/Blackquarter
Clostridial Diseases
Bacillary hemoglobinuria

• Clinical signs
  – Cattle may be found dead without any signs
  – Sudden onset of severe depression, fever, abdominal pain, dyspnea, dysentery, and hemoglobinuria
  – Anemia and jaundice in varying degrees
Clostridial Diseases
Bacillary hemoglobinuria

• Pathologic findings
  – Dehydration, anemia, sometimes subcutaneous edema
  – Bloody fluid in abdominal and thoracic cavities
  – Trachea contains bloody froth with hemorrhages in the mucosa
  – Small intestine and occasionally large intestine are hemorrhagic with free or clotted blood in their contents
  – An anemic infarct in the liver is virtually pathognomonic; it is slightly elevated, lighter in color, and outlined by a bluish red zone of congestion
  – Kidneys are dark, friable and usually studded with petechiae
  – The bladder contains dark urine
Clostridial Diseases
Bacillary hemoglobinuria
Clostridial Diseases
Bacillary hemoglobinuria
Clostridial Diseases
Enterotoxemia

• Clinical signs,
  – *Clostridium perfringens* Type B/C
    • Severe enteritis, dysentery, toxemia, and high mortality in young
    • Sudden death often first/only sign in lambs and kids
    • Some young may cry out before death, grind teeth, have muscular tremors, froth at mouth, have yellowish or bloody diarrhea, and convulsions
    • High levels of starchy foods in the diet and slowing of gut movement are predisposing factors
    • In calves, acute diarrhea, dysentery, abdominal pain, convulsions, and opisthotonos
    • Death may occur in a few hours
    • Less severe cases may survive a few days
    • Recovery period of several days is possible
Clostridial Diseases

Enterotoxemia

• Clinical signs,
  – *Clostridium perfringens* Type D
    • Pulpy kidney disease
    • Occurs in lambs less than 2 weeks old or weaned in feedlots and on a high carbohydrate diet; or may occur when fed on lush green pastures or with goats/calves
    • Usually sudden death in best conditioned lambs
    • May see excitement, incoordination, and convulsions before death
    • Opisthotonos, circling, and pushing the head against fixed objects are common signs of Central Nervous System involvement
    • Frequently, hyperglycemia or glucosuria
    • May or may not develop diarrhea
Clostridial Diseases

Enterotoxemia

- *Clostridium perfringens* Type D
Clostridial Diseases

Enterotoxemia

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Clostridial Diseases
Enterotoxemia

• Pathologic findings
  – Hemorrhagic enteritis with ulceration of the mucosa is the major lesion in all species
  – Affected portion of the intestine is deep blue-purple and appears at first glance to be an infarction associated with mesenteric torsion
  – In young lambs, fluid-filled pericardial sac and hyperemic areas in the intestines
  – In older animals, hemorrhagic areas on the myocardium and petechiae/ecchymoses of abdominal muscles and intestinal serosa
  – Rapid post-mortem autolysis of the kidneys, but seldom found in affected goats or cattle
  – Hemorrhagic or necrotic enterocolitis may be seen in goats
Clostridial Diseases

• Diagnosis
  – Consider anthrax as a differential diagnosis for sudden death (if suspect anthrax, do not move or cut up the animal)
  – Confirm with lab testing to identify the bacteria or the toxin
  – Collect samples as soon as possible after death

• Treatment
  – Difficult to treat due to rapid progression
  – Use antitoxins when available in conjunction with antibiotic therapy, such as penicillin
Clostridial Diseases

• Prevention and Control
  – Proper management and vaccination
  – Booster mother with a multi-valent clostridial vaccine one month prior to birth date of young to increase the level of protection and period of time the young are protected
  – Ensure passive immunity through colostral transfer
  – Active immunity through two doses of vaccine 4-6 weeks apart; give first dose at 8 weeks of age or weaning time, when the protection from the dam’s milk begins to decline
  – Provide an annual booster before high risk periods to maintain protection
Foot and Mouth Disease

• Etiology
  – Also, AFTOSA
  – Highly contagious viral disease characterized by fever and vesicle formation in the mouth and feet
  – *Aphthovirus* in the Family Picornaviridae
  – Seven serotypes (A, O, C, SAT1, SAT2, SAT3, Asia1)
Foot and Mouth Disease

• Transmission
  – Most contagious disease known to exist
  – Infected animals exhale large quantities of virus which is then carried as an aerosol to other animals
  – FMDV can travel several miles on the wind
  – FMDV can survive within organic material such as bedding or manure
  – Animals can acquire the virus through oronasal exposure to the infected organic material
  – Affects all cloven-hoofed animals with cattle having a more severe form than sheep or goats
Foot and Mouth Disease

• Clinical disease
  – Incubation period 1-3 days
  – Morbidity approaches 100%
  – Fever, decreased activity, decreased feed consumption, small blisters on tongue, dental pad, feet, coronary band, interdigital cleft
  – Vesicles coalesce to become large, rupture and expose painful ulcers
  – Secondary infection occurs at exposed regions
  – Animals usually completely recover, but lose a great deal of condition during the short period of illness
  – Mortality is significant only in the very young due to heart muscle infection that leads to myocardial failure and sudden death
Foot and Mouth Disease

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Foot and Mouth Disease

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Foot and Mouth Disease

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Foot and Mouth Disease

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Foot and Mouth Disease

• Pathologic findings
  – Confirm with laboratory testing via complement fixation, ELISA, virus neutralization
  – Differential diagnoses include bluetongue, infectious bovine rhinotracheitis, bovine papular stomatitis, abrasive feed et al.

• Treatment
  – No specific treatment, but provide soft feed, dry environment to decrease problems from secondary infection
Foot and Mouth Disease

• Prevention and Control
  – Aimed at keeping infected animals and animal products from entering an area
  – Once endemic, control is by vaccination
  – Vaccines are serotype specific
  – Decontaminate infected premises using 2% acetic acid or sodium hypochlorite
Hemorrhagic Septicemia

• Etiology
  – Also, Pasteurellosis
  – Caused by bacterium, *Pasteurella multocida*, serotypes 6:B and 6:E (formerly B:2 and E:2)
  – 6:B is predominantly found in Asia
Hemorrhagic Septicemia

• Transmission
  – Transmitted by exposure to infected animals, carrier animals, or fomites
  – Precipitated by stress in animals harboring the organism subclinically
  – The bacteria do not survive well in the environment
  – Route of entry is presumed to be oronasal
  – After an outbreak, 20% of the survivors may be carriers down to less than 5% carriers after six months post-outbreak
  – Crowding/close contact facilitates spread
Hemorrhagic Septicemia

- **Species affected**
  - Affected animals are cattle 6-18 months old
  - Infrequently occurs in sheep

- **Clinical disease**
  - Short incubation period with high morbidity and high mortality
  - Clinical disease usually lasts less than 72 hours
  - First signs are dullness and reluctance to move
  - There may be respiratory distress, with frothing at the mouth, and recumbency
  - Edematous swellings can be seen in the throat region, neck and brisket
Hemorrhagic Septicemia

Hemorrhagic Septicemia

• Pathologic findings
  – Lesions seen are those of severe sepsis, with extensive damage to the capillary bed
  – Widely distributed hemorrhages and edema, especially of the head, neck, and brisket region
  – Incision of the edematous swellings reveals a coagulated, serofibrinous mass with straw-colored or blood-stained fluid
  – Petechiation is present in multiple organs and serosal surfaces
  – There may be serosanguinous effusions in body cavities
  – There is an interstitial reaction in the lung, typical of a toxic state, and represented by a diffuse congestion and a rubbery feel to the lungs grossly
Hemorrhagic Septicemia

Hemorrhagic Septicemia

• Diagnosis
  – Epidemiological and clinical features aid in recognition of the disease
  – Characteristic necropsy lesions support clinical diagnosis
  – In endemic regions, acute salmonellosis, anthrax, pneumonic pasteurellosis, and rinderpest should be considered for differential diagnosis
Hemorrhagic Septicemia

• Treatment
  – Usually, too late to start treatment
  – Prophylactic antibiotics may be given to the rest of the herd that is not yet clinically ill
  – Sulphonamides, tetracyclines, are effective if administered early

• Prevention and Control
  – Vaccination in endemic areas, with bacterins or a modified live product
  – Avoid crowding, especially during wet conditions, to reduce the incidence of disease
  – Animals to be shipped should be vaccinated
Bovine Tuberculosis (TB)

• Etiology
  – Chronic infectious and debilitating granulomatous disease caused by *Mycobacterium bovis*
  – *M. bovis* is a hardy bacterium that persists in the environment
  – *M. bovis* causes a progressive disease in most warm-blooded vertebrates, including humans (zoonotic)
Bovine Tuberculosis (TB)

• Transmission
  – By inhalation of infected droplets expelled from the lungs
  – Also, by ingestion, particularly contaminated milk

• Species affected
  – Infects predominantly cattle, rarely affects other mammals
  – Humans are quite susceptible to bovine TB
  – Infections in humans occurs through drinking infected raw milk, raw milk products, and through inhalation
Bovine Tuberculosis (TB)

• Clinical Signs
  – Progressive emaciation, lethargy, weakness, anorexia, and a low-grade, fluctuating fever
  – Respiratory form with bronchopneumonia causes a chronic, intermittent, moist cough with later signs of dyspnea and tachypnea
  – Granulomatous form with bronchopneumonia may detect destructive lesions on auscultation and percussion of the lungs
  – Superficial lymph node enlargement may be a useful diagnostic sign when present
  – Affected deeper lymph nodes cannot always be palpated, but they may cause obstruction of the airways, pharynx, and gut, leading to dyspnea and ruminal tympany
Bovine Tuberculosis (TB)

• Pathologic findings
  – TB granulomas may be found in any of the lymph nodes, particularly in bronchial, retropharyngeal, and mediastinal nodes
  – In the lungs, miliary abscesses may extend to cause a suppurative bronchopneumonia
  – The pus has a characteristic cream to orange color and varies in consistency from thick cream to crumbly cheese
  – TB nodules may appear on the pleura and peritoneum
Bovine Tuberculosis (TB)
Bovine Tuberculosis (TB)
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Bovine Tuberculosis (TB)

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Bovine Tuberculosis (TB)
Bovine Tuberculosis (TB)
Bovine Tuberculosis (TB)

• Diagnosis
  – Most important diagnostic test: Intradermal tuberculin test
  – Diagnosis by clinical signs alone is very difficult
  – Microscopic exam of sputum and other discharges is sometimes used
  – Necropsy findings include “tuberculous” granulomas
  – Confirmation of diagnosis is by isolation and identification of the organism by culture, usually taking 4-8 weeks, or by PCR, which takes a few days
Bovine Tuberculosis (TB)

• Treatment
  – May be illegal in some countries
  – Destruction of TB positive animals should be attempted due to zoonotic nature of the disease

• Prevention and Control
  – Main reservoir of infection is cattle
  – Test and slaughter policy for eradication
  – Testing every 3 months in an affected herd to get rid of infected individuals
  – Pasteurization of milk reduces incidence of human infection
Questions???

• And, thank you for your attentiveness
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