

Feedlot Sessions

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Interpreting Post-Mortem Lesions in Feedlot Cattle: Theories, Speculations and Conclusions

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Introduction

Changes seen at necropsy of feedlot cattle can be major indicators of processes and diseases presented in the lot. Careful interpretation of the lesions must be made to arrive at accurate and meaningful conclusions. Many lesions are etiologically specific, most are not. It is important to recognize which are which. The following presentation will discuss how to interpret specific lesions seen at necropsy of feedlot cattle based on the author's experience.

Respiratory System

Ulcerative laryngitis

Anything that will cause a persistent edema of the larynx may result in bilateral contact ulceration. Some possibilities include viral infections such as PI-3, IBR and BRSV, persistent or chronic pneumonia especially productive bronchopneumonias (i.e. H somnus), environmental irritants, dust, ammonia fumes, allergens.

Diphtheritic laryngitis

A direct result of contact ulcerations that have become secondarily infected with bacteria such as *Fusiformis necrophorum*, *Actinomyces (Corynebacterium) pyogenes* and perhaps others. Very difficult to heal these lesions even if you kill the bug. These cattle may be very susceptible to subsequent laryngeal edema.

Tracheal edema ("honker syndrome")

Cause is unknown. Theory: Airflow in trachea is usually laminar or mildly turbulent. With any occlusion of the trachea or with forced inspiration and/or expiration the airflow increases its turbidity and becomes less efficient. Turbulence is also greatest along the margins of the airflow resulting in irritation to the mucosa.

Irritation results in edema, thereby decreasing the diameter of the tracheal lumen and increasing flow resistance. This becomes a vicious cycle much like the asthmatic with bronchoconstriction and may result in asphyxiation due to partial tracheal occlusion.

Hyperemic tracheitis

Don't try to make too much out of this lesion (change). At best it indicates respiratory distress, which you probably already knew from clinical signs or other lesions. It does not indicate a specific etiology.

Diffuse interstitial pneumonia

Although BRSV and dietary causes of interstitial pneumonias are likely the major etiologies of most feedlot interstitial pneumonias, there certainly may be other causes. Occasional allergic interstitial pneumonias may occur due to inhaled antigens (i.e. mold spores, pollens, etc.) and also the endotoxins of *Haemophilus somnus* (HS) have produced the lesions experimentally. *Haemophilus somnus* bronchopneumonias in anterior ventral locations are frequently accompanied by interstitial lesions in the caudal lobes.

Focal to multifocal lung necrosis in lobules with bacterial consolidation

Although severe *Pasteurella hemolytica* pneumonias often result in fibrinohemorrhagic consolidation of the lungs, when areas of necrosis are seen within the consolidated areas, examine the trachea and larynx for evidence of fibrinonecrotic surface lesions such as those occurring with IBR or calf diphtheria.

Diffuse serofibrinous pleuritis

Nearly always the result of *Pasteurella hemolytica* infection breaking into the pleural cavity from affected lung.

Diffuse fibrinous pleuritis

Although *P hemolytica* is often the cause of this lesion, *Haemophilus somnus* is also very capable of producing this change. This lesion will also often extend to the external surfaces of the pericardium, but rarely results in extensive pericarditis involving the epicardial surfaces of the heart.

Central Nervous System Lesions

Yellowing /softening of the cerebral cortices

Polioencephalomalacia. A means to view this lesion even in the very early stages of the disease is with the use of a Wood's light (UV light source). Etiologies for this lesion include disruption of the rumenal microflora, excessive sulfates (in feed, water and other mineral sources), miscalculations in anticoccidial drugs (such as Amprolium) and excessive total dissolved solids in water source. Chronic lead intoxication may also induce polioencephalomalacia lesions. Acute brain edema and salt intoxication are less common possible causes.

Multifocal hemorrhage and necrosis

Randomly scattered foci of hemorrhage and necrosis are generally the result of thromboembolic meningoencephalitis caused by the septicemic form of *Haemophilus somnus* infection. Occasional disseminated fungal infection (i.e. aspergillosis, mucormycosis) arising from fungal rumenitis, may produce similar brain lesions. Septicemic HS infections generally manifest other lesions in addition to TEME, some of the more common lesions include multiple inflammatory foci seen as petechial hemorrhages in the skeletal and cardiac muscles, submucosal hemorrhages in the esophagus and fibrinopurulent polyarthritis (most easily seen in the stifle joints).

Bilateral malacia of the midbrain and brain stem

Lesions similar to those seen in subacute enterotoxemia in sheep have been seen in feedlot cattle. These are often fairly alert downers. Little experimental work has been done on this condition, but it is suspected to be the result of sublethal *Clostridium perfringens* type D enterotoxemia.

Focal grayish discoloration of the medulla and pons

May be the only gross lesion seen in encephalitis listeriosis. Usually listeriosis is not recognizable grossly.

Cloudy meninges along the ventral cerebral surfaces

Bacterial meningitis. *Pasteurella multocida* or *Actinomyces (Corynebacterium) pyogenes* are two common isolates. You really have to look for this lesion. Touch a slide to the meninges, stain it with a blood smear stain and look for neutrophils if you suspect a meningitis.

Skeletal Muscles

Pale white muscles

High environmental heat, overheating, post-mortem autolysis and transport tetany can all pale out skeletal muscles. With a history of CNS signs, consider lead intoxication. Nutritional myopathy (vitamin E-selenium deficiency) may also result in focal white streaks in the musculature. Occasionally sarcosporidiosis may become so extensive that white streaks are noted grossly in the muscles. Histopathology of affected muscles may be helpful in sorting out the possibilities.

Hemorrhagic foci in skeletal muscles

Trauma, exertion, injury and generalized toxemias including enterotoxemia may all induce skeletal muscle hemorrhage. Septicemic HS may often result in multifocal skeletal muscle hemorrhage, necrosis and inflammation.

Dry hemorrhagic necrosis of skeletal muscles with gas bubbles

Blackleg, *Clostridium chauveoi*. Enough said.

Moist hemorrhagic necrosis with gas bubbles

Might be just trauma with post-mortem autolysis but also may result from malignant edema. Couple the history with other findings to avoid being misled.

Urinary system

Swollen pale kidneys

In feedlot cattle, the first thing to look for is a possible urinary tract obstruction (water belly, urolithiasis). Injection of oral nephrotoxic antibiotics (i.e. neomycin), may induce acute nephrosis. Rare toxicities may cause pale swollen kidneys in cattle including the mold toxin, ochratoxin and in some geographical areas, plant toxins such as pigweed.

Multifocal red to white foci in kidneys

This is usually a result of embolic showering of the kidneys with septic emboli breaking off the heart valve(s) or endocardial lesions in the heart. Open the heart and take a look when you see this lesion in the kidneys. If there are no heart lesions, follow the aorta from the heart to the renal arteries. May ultimately be linked to liver abscesses and feeding practices.

Purulent foci in kidneys accompanied by extensive necrosis and purulence of pelvis

Pyelonephritis caused by *Corynebacterium renale* or other bacteria.

Heart

Myocardial hemorrhage

Often is agonal or non-specific. However, when associated with sudden deaths, subendocardial hemorrhage, especially ecchymotic hemorrhage, may be an indicator of enterotoxemia.

Multifocal myocardial hemorrhage or necrosis

Acute to chronic HS septicemia. Blackleg can also affect the heart. White streaks may indicate also nutritional myopathy, rumensin toxicosis, or protozoan parasitism.

Liver

Focal to multifocal hepatic necrosis

If this is associated with sudden death with extensive petechial to ecchymotic serosal hemorrhages, consider *Clostridium novyi* or *Clostridium hemolyticum* infection, (especially if hemoglobinuria occurs also and the region has a history of sporadic problems of this type). Cattle from liver fluke areas have a higher incidence of clostridial hepatitis and toxemias.

Gastrointestinal tract

Oral, esophageal and abomasal ulcers

Although BVD infection should come to mind, this is not the only possible cause of these ulcers. Occasionally lesions induced by IBR or papular stomatitis viral infection can mimic BVD lesions.

Abomasal mucosal thickening

A worming program will probably pay dividends. Ostertagiasis is having a clinical effect if you can see lesions.

Full thickness necrotic, hemorrhagic rumenitis

These cattle have been overloaded or had lactic acidosis in the last few weeks. Check the feeding procedures. How fast were they brought onto concentrate?

Hemorrhagic, necrotizing enteritis

Enteric salmonellosis is a major cause of this lesion, but rarely gets this severe unless there is an underlying reason, i.e. stress, BVD, parasitism (coccidiosis) or other infections.

Hemorrhagic colitis

Most likely due to coccidiosis even if you cannot demonstrate the oocysts in this particular animal at death. Secondary bacterial invasion of the colonic lesion (i.e. *Clostridium sp.*) may accentuate the lesion.

Conclusions

Although much can be seen during a necropsy examination of a feedlot calf, it is imperative that the information be correlated with other data available to the feedlot veterinarian. A thorough understanding of the source of the cattle, the time in the lot, the vaccination history, the feeding practices of that operation and other similar information must be used when interpreting necropsy findings. Then correct interpretation of the post mortem findings is then often attainable even without the help of reference laboratory assistance.

