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Forage-Related Cattle Disorders

Acute or Atypical Interstitial Pneumonia (AIP)

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Introduction

Acute or atypical interstitial pneumonia (AIP) is terminology used by veterinary pathologists to describe a characteristic pattern of damage in the lungs of cattle. At necropsy, the first indication of AIP-affected lungs is they fill the thorax and do not collapse like normal lungs when the chest cavity is opened. On further investigation, the AIP lungs are found to be semi-firm with a rubbery texture; not hard and consolidated as occurs in bacterial bronchopneumonia. Large air bubbles are found between portions of the lung (see Figure 1, “interlobular” or “bullous” emphysema) and the cut surface of the lung may appear wet or shiny due to edema. Affected lobules are dark red to purple and may be interspersed with normal looking lobules, creating a “patchwork” or “checkerboard” appearance.

Causes

A variety of agents, directly or indirectly, will damage the walls of the air sacs in the lungs and cause AIP. One of these agents is tryptophan, an amino acid present in forages that is metabolized by certain organisms in the rumen to 3-methyl indole (3-MI). The 3-MI is absorbed into the bloodstream, transported to the lungs and metabolized to a new compound 3-methyleneindolenine (3MEIN) that causes the widespread cellular injury in the lung. Nursing calves are not at risk and yearlings are less susceptible than adults. Tryptophan-induced AIP is called acute bovine pulmonary edema and emphysema (ABPEE) or “fog fever,” a condition first reported in Europe more than 200 years ago. The disease is associated with the grazing of “fog lands” which are pastures that have

lush new growth after being cut for hay or silage. In the western United States, ABPEE is most often seen in mature beef cows when they are moved from dry summer mountainous pasture to a lush, irrigated lowland pasture. Clinical signs develop within days and up to two weeks after the pasture change. Studies have found that tryptophan levels in pastures associated with fog fever are not necessarily higher than unaffected pastures. Instead, it is the abrupt change from the low plane of nutrition when grazing the poor quality dry forage to lush pasture that increases the rumen organisms capable of metabolism of tryptophan to 3-MI, resulting in its rapid formation. Similarly, brassicas including kale, rape,

and green turnip tops are rich sources of tryptophan that can be converted in the rumen to 3-MI and cause AIP when given sudden access to them following relatively dry, high roughage diets.

In the Southeastern United States, acute interstitial pneumonia has been produced by ingestion of the leaves and seeds of perilla mint (*Perilla frutescens*) (Figure 2). Perilla ketone is the toxin absorbed from the rumen into the bloodstream and carried to the lungs where it damages the lung tissue. Perilla mint is a common weed also known as purple mint, wild coleus, and beefsteak plant. *P. frutescens* thrives in late summer, when pastures are frequently dry and dormant. Cattle normally avoid it but may be forced

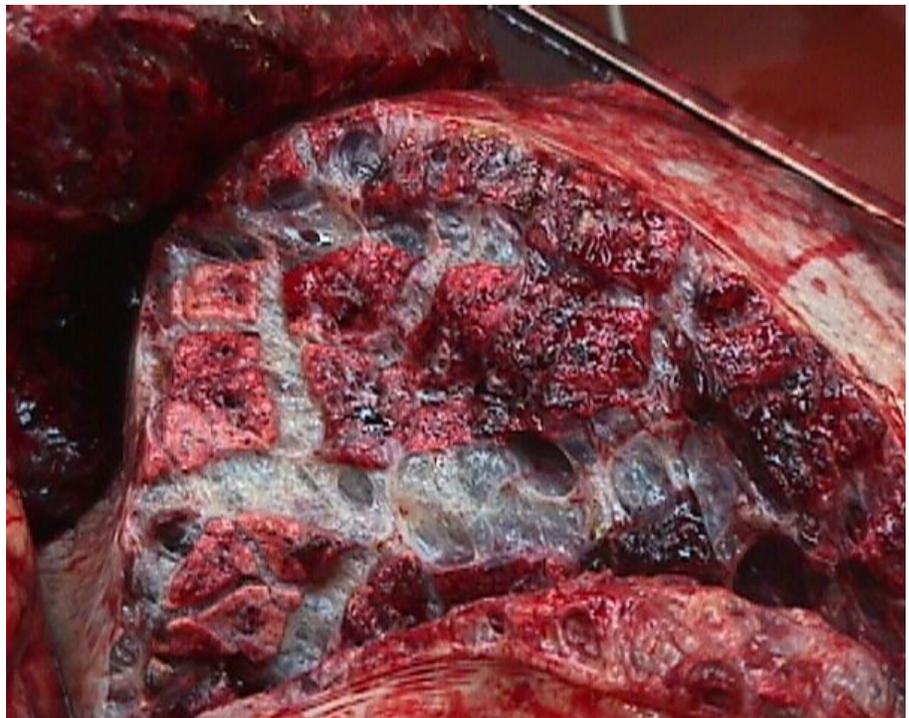


Figure 1. AIP lung with bullous emphysema.

Photo courtesy of Dr. Louis Pittman, Pathologist-Breathitt Veterinary Center



Figure 2. Perilla mint.

Photo courtesy of the School of Veterinary Medicine/University of Pennsylvania

to eat it when other pasture forages are limited. The vegetative growth stage is of relatively low toxicity; the green seed stage plant is most toxic, especially the seeds. Dried hay is less toxic than green plants but can be lethal while frosted plants have relatively low toxicity.

Moldy sweet potato toxicity is also a known etiology of AIP. It is caused by ingestion of 4-lpomeanol, a toxin produced by sweet potatoes in response to infestation with the fungus *Fusarium solani*. The toxicity is usually severe and irreversible when it occurs so mold-damaged sweet potatoes should not be utilized for feed.

Ingestion of perilla mint or moldy sweet potatoes initiates acute respiratory distress syndrome that frequently results in death. Mature cattle are most often affected but it can occur in yearlings and calves. Treatment is of limited value, and cattle seldom survive severe cases.

Finally, there is feedlot-associated AIP, which is the second most common cause of respiratory death in feedlots after bronchopneumonia. It demonstrates a seasonal peak, with the highest incidence in the summer. It affects heifers at a much greater rate, it's more likely to affect heifers that have been on feed for a longer time, and it has a high case fatality rate. The cause of this syndrome is unknown and is probably multifactorial. It is thought to result from an interaction

of factors such as high ambient temperature, airborne dust, infection, endotoxin exposure, and antioxidant depletion due to consumption of rancid fat. This type of AIP affects cattle in some feed yards but not others so management factors likely play a significant role in its occurrence.

Clinical Signs

The clinical signs of AIP are often grouped together and described as an acute respiratory distress syndrome (Figure 3). This syndrome includes a sudden onset of open-mouth breathing with the head and neck extended, nos-

trils dilated, a sway-back appearance, foam coming from the mouth, an open shouldered stance, and sometimes aggression. Breathing is shallow and rapid at 35-75 breaths per minute (normal is 10-30 breaths in adult cattle) and may have a loud expiratory grunt. Temperature may be slightly elevated due to the severity of the condition. In extreme cases, air under the skin (subcutaneous crepitation) may be felt over the upper portions of the neck, shoulders and back. Mild exercise may cause the animal to collapse and die. Generally there is an absence of coughing and no signs of infection such as high fever (>104°F) or depression. Severely affected animals usually die two to three days after initial onset of clinical signs. Death occurs in approximately 30 percent of cases; less severely affected animals may improve without further consequences. The stress of handling cattle can cause further death loss so care must be taken when removing cattle from the offending pasture. Those that survive show dramatic improvement after an average of three days with recovery spanning approximately 10 days. However, severely affected animals that survive may develop chronic emphysema or heart failure. Treatment usually is ineffective after development of clinical signs. Treatments by a veterinarian may include diuretics, nonsteroidal anti-inflammatories and corticosteroids used in an extra-label manner.



Figure 3. Acute Respiratory Distress Syndrome.

Photo courtesy of Dr. Alan Doster, University of Nebraska-Lincoln

Prevention and Control

Prevention of “fog fever” is based on management strategies that incorporate gradual forage change and the addition of ionophores in the diet. Administration of monensin has been shown to reduce the conversion of tryptophan to 3-MI in the rumen by as much as 90 percent. For monensin to be effective, it must be present in the rumen at the time of exposure and should be fed continuously for at least 10 days after turn-out. If cattle have been on poor quality dry forage, slowly introduce them to lush pastures (including brassicas) by allowing access for only a few hours a day for the first five to seven days. This is best accomplished by strip grazing pasture utilizing portable electric fence. Gradually increase grazing time over a 10-12 day period.

Prevention of other types of AIP is based on lessening the exposure of susceptible cattle to known causes. Cattle should be offered sufficient forage so they are not forced to graze weeds such as perilla mint. Moldy sweet potatoes should not be utilized for feed. Avoid exposure to gases including hydrogen sulfide and nitrogen dioxide that may similarly damage the lungs. Feedlot-associated AIP is difficult to prevent because not much is understood regarding the cause of this condition.

Summary

Atypical interstitial pneumonia (AIP) is a disease characterized by the sudden onset of severe respiratory distress or sudden death associated with:

1. A sudden dietary change from dry to lush green forages or brassicas
2. Consumption of perilla mint
3. Ingestion of moldy sweet potatoes

Affected cattle should be removed from the suspected source as soon as possible but with extreme caution to avoid further respiratory distress. Treatment is of little value once clinical signs develop. Diagnosis of AIP is easily confirmed on necropsy; the lungs are puffed up, semi-firm, heavy and rubbery, and will not float in water or collapse as a normal lung would. Control of tryptophan-induced AIP is through slow introduction to lush pastures or brassicas and feeding monensin. Prevention of other types of AIP includes avoidance of areas with perilla mint when pasture is scarce and not including moldy sweet potatoes in feed. Feedlot associated AIP is not well understood and therefore difficult to control. It is most likely due to a combination of genetic and hormonal influences along with feed and management practices that result in severe disease.

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