

VARSHA VAHINI

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From the Editor's Desk

We are dealing in this issue about two important disease of clostridial origin. We feel apt to deal with these diseases as they are highly prevailing today. We need to underline that both are sequale of underlying immunosuppression. therefore improving bird immune status specilly for immunosuppressive problemslike IBD can be of great help in prevention

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Necrotic enteritis

Necrotic enteritis was first described in chickens in England in 1961. Since then it has been reported in most poultry producing countries around the world. As its name suggests, the condition is characterised by the death or necrosis of the intestinal lining, predominantly of the upper small intestine. Here, clostridial bacteria produce large amounts of damaging type C toxin which, when combined with digestive enzymes, destroy the lining of the gut.

Necrotic enteritis is an acute illness is usually very short and often the only signs are a sudden increase in mortality. It is caused by *Clostridium perfringens*. *C perfringens* is a nearly ubiquitous bacteria readily found in soil, dust, feces, feed, and used poultry litter. It is also a normal inhabitant of the intestines of healthy chickens.

Trigger factors

The pH and high oxygen content of the healthy small intestine do not support growth of the organisms. So,

for necrotic enteritis to occur, there needs to be a trigger factor that tips the balance in favour of the clostridial bacteria, allowing them to proliferate and migrate to the upper intestines. Spores of the causative organism are highly resistant

The clinical disease most often occurs either following an alteration in the intestinal microflora or from a condition that results in damage to the intestinal mucosa (eg, coccidiosis, mycotoxycosis, salmonellosis, ascarid larvae). High dietary levels of animal byproducts (eg, fishmeal) and high viscosity diets predispose birds to the disease. Anything that promotes excessive bacterial growth and toxin production or slows feed passage rate in the small intestine could promote the occurrence of necrotic enteritis. Immunosuppression may be due to chicken anaemia, IBD or MD reduces resistance to gut infections.

Clinical signs: Most often the only sign of necrotic enteritis in a flock is a sudden increase in mortality.



Mortality may be 5-50%, usually around 10%. Infection occurs by faecal-oral transmission

- Depression.
- Ruffled feathers.
- Inappetance.
- Closed eyes.
- Immobility.
- Dark coloured diarrhoea.
- Undigested feed getting excreted
- Sudden death in good condition

Lesions : The gross lesions are primarily found in the small intestine (jejunum), which may be ballooned, friable and contain a foul-smelling, brown fluid. The mucosa is usually covered with a tan to yellow pseudomembrane often referred to as a “Turkish towel” in appearance. This pseudomembrane may extend throughout the small intestine or be only in a localized area.

- Small intestine (usually middle to distal) thickened and distended. Intestinal mucosa with diphtheritic membrane.
- Intestinal contents may be dark brown with necrotic material.
- Reflux of bile-stained liquid in the crop if upper small intestine affected.
- Affected birds tend to be dehydrated and to undergo rapid putrefaction

Diagnosis

A presumptive diagnosis may be made based on flock history and gross lesions Confirmation is by smear examinations from affected tissues and a good response to specific medication, usually in less than 48 hours.

Prevention and treatment

Because *C. perfringens* is nearly ubiquitous, it is important to prevent changes in the intestinal microflora that would promote its growth. This can be accomplished by adding antibiotics in the feed such as virginiamycin (20 g/ton feed), bacitracin (50 g/ton feed), and lincomycin (2 g/ton feed). The addition of anticoccidial compounds, especially of the ionophore class, has been extremely helpful in preventing the coccidial damage that leads to necrotic enteritis.

Administration of probiotics or competitive exclusion cultures has been used to both prevent and treat clinical necrotic enteritis (presumably by preventing the proliferation of *C. perfringens*). Probiotics may limit multiplication of bacteria and toxin production. Digestive enhancers, which will also suppress bacterial overgrowth in the intestines, can help. Effective cleansing and disinfection with broad spectrum disinfectants regularly can prevent immunosuppressive diseases and return NE.



Severe clinical outbreaks usually respond to specific antibiotic treatment Treatment includes Penicillins (e.g. phenoxymethyl penicillin, amoxycillin), in drinking water, or Bacitracin in feed (e.g. 100 ppm). Water medication for 3-5 days and in-feed medication for 5-7 days depending on the severity

Conclusions

Necrotic enteritis is a frustrating problem as it repeatedly affect the flocks and can cause significant mortality in broilers in their rapid growth phase. Inclusion of ionophore anticoccidials, effective growth promoters and digestive enhancers will help to ensure gut stability and thereby prevent overgrowth

Ref:

1. Mercks Veterinary Manual
2. *Stephen A Lister, Crowshall Veterinary Services*
3. The Poultry Site Quick Disease Guide



Gangrenous dermatitis (GD)

Gangrenous dermatitis (GD), other wise known as “wing rot” is yet other Clostridial disease, associated with *Staphylococcus aureus*. Combined infections are often more severe.

For gangrenous dermatitis (GD) to occur in a large population of birds, generally three things are required:

- Some type of injury to the skin
- Some type of immune suppression
- The disease causing organism -Clostridium or other species

Skin damage due to trauma, wet litter, and pecking may provide entry sites for causative bacteria. Some simple factors can result in an outbreak of hysteria/nervousness in the flock leading to an increase of cuts and scratches. That could be an electrical storm, longer day length, increased light intensity, light restriction programs and low dietary sodium levels. Management problems that could contribute to flock hysteria include predators entering the house, improper fan cycling, flashing lights and/or loud sudden noises. As a general rule, any alteration in management, which reduces stress or reduces the possibility of a wound, will reduce the probability of having a GD outbreak.

Clostridium species are spore formers generally found in soil, but can be found in feed, feces, dust and any number of other places. Since Clostridium are very durable and able to survive extremely harsh conditions, it is unlikely that they will ever be completely eliminated from commercial animal facilities. The strategy with Clostridium is to keep the organism numbers as low as possible so that when animals are exposed, the chances of recovery are enhanced.

To keep conditions in the poultry production environment such that growth of the organism is discouraged, the following must be done:

- Removing dead birds often and proper disposal
- Keeping equipment as clean as possible particularly open type waterers
- Prevent other animals from entering the house.
- Routine disinfection programme

Immune competent birds generally are not get affected by GD. Therefore, the appearance of GD

may be related to immunosuppression due to IBD, Chicken anaemia, mycotoxicoses, stress and overcrowding. The disease may occur secondary to avian adenovirus or reticuloendothelial virus infections as well.

GD usually starts with the appearance of small pimples on the skin, soon progressing to involve large areas. The first sign is usually a sudden dramatic increase in mortality in the affected flock. Overall mortality is 10-60%. Affected chickens are extremely depressed, lethargic, and prostrate, and die within 8-24 hr. Red to black patches of moist, gangrenous skin are seen over



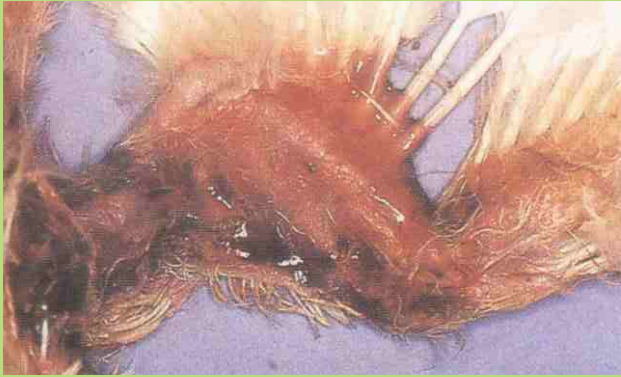
the breast, abdomen, wing tips, or thighs. Feather loss is common. The breast, wings, rump and abdomen are most commonly involved. There will be an accumulation of blood-tinged gaseous fluid beneath the skin and fluid can be jelly-like in consistency. The musculature looks pale with cooked appearance. The liver and spleen are enlarged and kidneys are usually swollen, and the lungs may be congested and edematous.

Prevention and control

Maintaining proper litter conditions, minimizing traumatic injury, and controlling cannibalism can help prevent the disease. Administration of oxytetracycline in the feed at 0.02% rapidly reduces mortality in field outbreaks of clostridial infections. Chlortetracycline, Oxytetracycline, Erythromycin in the water has proved beneficial for staphylococcal infections.

One method of reducing the incidence of GD following a break is the use of iodine disinfectant in the water. The solution is provided to birds every other day for three times. In other words, birds are to consume





iodine treated water a total of three times. This treatment is most effective when administered immediately after the onset of an outbreak or when an outbreak is expected (A. Rossi, personal communication).

If the birds in deep litter, there have been a number of field reports of treating litter with alum, aluminum sulfate, sodium sulfate, salt or other treatments to reduce the incidence of GD though there is no objective evidence that such treatments are effective. Salt is reported to be a drying agent, which assists with disinfection if used following cleaning and sanitizing of poultry houses. Other litter treatments are reported to produce a dramatic shift in litter pH, which may reduce or inhibit microbial growth.

After an outbreak, a complete clean-out, scrubbing and disinfection of houses is generally best so that spore counts are reduced. It is also advisable to test the pH of soil on the floor of farms with chronic GD problems. Frequently, soil is alkaline on such farms and, thus, should be adjusted to neutral or slightly acidic.

When GD is recurrent on farms, IBD titers should be checked to ensure that adequate immunity exists. If IBD titers are inadequate, IBD control programs should be re-evaluated and intensified. Better IBD control has, in some cases, resulted in a lower incidence of GD outbreaks.

The addition of elevated levels of fat soluble vitamins may enhance immunity as well as skin integrity. Therefore, fat soluble vitamins might be given to birds on farms with chronic problems. Thus, if water soluble preparations of vitamins A, D, E and K are used, they should be provided at half the recommended dose for three days and then clean water for four days.

Veterinary scientists explore poultry virus as cancer cure

US - Virologists in the Virginia-Maryland Regional College of Veterinary Medicine (VMRCVM) at Virginia Tech are looking at how a genetically modified variant of Avian Newcastle disease virus (NDV) can treat human prostate cancer.

Dr. Elankurmaran Subbiah, assistant professor in the Department of Biomedical Sciences and Pathobiology, was awarded a prestigious research grant by the Department of Defense. This "Congressionally Directed Medical Research Program" award will support the exploration and hypothesis development for an innovative approach to treating prostate cancer.

Prostate cancer is the second most common type of cancer in men, according to the American Cancer Society (ACS). The ACS estimates there will be almost 219,000 new cases of prostate cancer reported in the United States in 2007.

According to Subbiah, the use of poultry viruses as cancer therapy poses no threat to humans and several other oncolytic viruses are currently being explored to treat cancer. However, Subbiah's work is the first to alter Newcastle disease virus through a reverse genetic system to target prostate cancer specifically.

In the current investigation, Subbiah and his associates are altering the fusion protein of NDV to replicate only in the presence of prostate specific antigen (PSA), which is found exclusively in cancerous prostate cells.

Normal, healthy cells have an interferon antiviral system that activates upon infection with NDV thereby preventing replication of the virus, explains Dr. Subbiah. Cancer cells, however, have defective interferon antiviral systems, he said. NDV utilizes the defects to replicate in the diseased cells. The replication of NDV leads to the death of the cancer cell by a process called apoptosis - also known as programmed cell death or cell suicide- in the cell.

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