GANGRENOUS DERMATITIS A THREAT TO BROILER CHICKENS

February 2023

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Gangrenous dermatitis (GD) was first reported in the United States in the early 1930s (Niemann, 1930). Although GD has been recognized for many years as a sporadic disease, the prevalence and severity of this condition has increased over the past two decades in the U.S. and elsewhere (Gornatti-Churria et al., 2018). It is a disease that affects primarily commercial broiler chickens and turkeys and is responsible for severe economic losses in the poultry industry worldwide (Li et al., 2010). GD is generally fatal, often within 24 hours. When an outbreak occurs, it often appears that growers may have healthy birds and dead birds, but few birds in between. It often occurs in fast-growing broiler chickens between 4 and 8 weeks of age although GD has been reported in broiler breeders, commercial layers and turkeys. Economic losses are substantial because much of the mortality occurs late in the flock when much of the feed has already been consumed. Death losses that range from 50 to 400 birds per house per day are not uncommon. The disease has been known by a variety of names, including avian malignant edema, gas edema, necrotic dermatitis, gangrenous cellulitis, red leg, blue wing and wing rot.

What causes GD?

A wound to the skin usually starts the process in motion. This is quickly followed by a secondary bacterial infection with Clostridium perfringens type A, Clostridium septicum or Staphylococcus aureus (alone or in combination). These bacteria are usually not able to penetrate intact skin. They need help from a scratch or a wound to gain entrance; however, infectious agents can be ingested if healthy birds peck at dead birds that have died with the disease or if litter and feces are contaminated with large numbers of disease-causing organisms. Staphylococcus aureus is often associated with milder forms of GD. Cases are generally much more severe and have much higher mortality when Clostridium is the cause. Clark et al. (2008)
indicated that for GD to occur and affect large numbers of birds, generally three things were required:

1. **Some type wound or injury to the skin,**
2. **Disease organisms (Clostridium or some other species) present in sufficient numbers to cause disease, and**
3. **Some type of immune suppression.**

Immunosuppression can be triggered by a variety of infectious factors in the poultry house environment such as infectious bursal disease virus, chicken anemia virus, Marek’s disease virus, reoviruses, etc., and environmental factors such as skin injuries (Clark et al., 2010) and wet litter (Martland, 1985).

There is no shortage of sources for potential skin injuries, even on well-managed poultry farms. Chicken toenails are a source of numerous cuts and scratches that broilers receive during the growout period. Growers should monitor their houses and eliminate any potential injury risk (sharp edges along walls, exposed nails at bird level, ladders left in the poultry house, etc.). It would be virtually impossible to remove all disease-causing organisms from the poultry house. *Clostridia* and *staphylococci* are ubiquitous (present everywhere) in the poultry house environment and in the intestine and on the skin of the birds. Fortunately, it is not necessary to remove all disease-causing organisms. We only need to maintain numbers below the threshold where the birds would be overwhelmed by the magnitude of disease-causing organisms.

Presence alone of disease-causing organisms does not automatically result in sickness or a disease outbreak. Other contributing factors likely play a role in development of clinical disease. *Clostridia* are extremely hardy and capable of surviving harsh environmental conditions for long periods of time. It is, therefore, unlikely that they can ever be totally eliminated from the poultry house environment. In addition, they are **spore-forming bacteria** that, when they detect that their environment is becoming unfavorable, form a spore that helps them survive until conditions improve. The spore is resistant to desiccation, practically all disinfectants, heat and cold.

**What to do?**

Birds with a strong, or even a reasonably competent, immune system are generally not affected by GD. Therefore, it is often believed that factors that result in immunosuppressive effects such as diseases mentioned earlier like infectious bursal disease, inclusion body hepatitis, reoviruses, chick infectious anemia, Marek’s disease, etc., may set a flock up for GD. In addition, mycotoxins (particularly aflatoxin) in the feed can result in immune suppression, as can birds subject to stressful situations like overcrowding, heat stress or wet litter conditions. Additional stressors such as coccidiosis, nutritional deficiencies, environmental extremes and management issues may also suppress the immune system, leading to greater risk of GD. It is **common for farms that have had GD before to have repeat outbreaks** unless corrective action is taken. This is often because there are large numbers of *Clostridia* present in the litter, capable of exceeding the threshold the birds can manage and overwhelming the immune system.

Corrective action may take several forms. A complete cleanout and disinfection of the house(s) is usually the best choice to reduce bacterial numbers and spore counts after an outbreak; however, even a complete cleanout may not completely eliminate the problem on farms with a history of GD. In addition, a complete cleanout is not always an option. Short downtimes
between flocks, availability and cost of new bedding materials and unfavorable weather conditions may make a total cleanout impossible. As an alternative, there are field reports that indicate different degrees of success using various litter treatments to cause a dramatic reduction in pH of the litter, which may restrict microbial growth. Sodium bisulfate, aluminum sulfate, salt and other products have been used to reduce the occurrence of GD.

**NOTE OF CAUTION TO GROWERS:** Do not apply any product to the litter or poultry house pad without first consulting with your flock supervisor or live production manager and receiving the green light from your integrator that the product in question is safe to use and approved by your integrator. Furthermore, growers should read the label on any product they use and understand all the consequences associated with any product they apply and how that product may affect the litter for future use.

Windrowing litter between flocks is another alternative to a total cleanout to reduce the total pathogen load of the litter. Remember, it’s not necessary to kill all the pathogens in the litter; however, it is critical to reduce pathogen numbers below the critical threshold so that the overall pathogen count is not a threat to the flock. In-house windrowing can eliminate most (not all) of the pathogens present in the litter, including *Clostridium*, when done correctly (Macklin et al., 2007). Construct the windrows as soon as possible after the birds leave. Do not wait 3-4 days to start. Windrowing takes advantage of primarily heat generation to kill microorganisms although competitive exclusion (replacing “bad” bugs with “good” bugs) and high ammonia levels may also play a role in reducing pathogen numbers.

Pathogens grow most effectively at a temperature range similar to the body temperature of their host; with chickens, this is a temperature around 105-107°F. If the temperature can be increased a few degrees, growth rate of the pathogens can be inhibited. Raising the temperature 20-30°F above optimum can kill many microorganisms. Therefore, establish the goal of a minimum target temperature of 130-135°F inside the windrow. If this can be reached, this is hot enough to kill most pathogens. It is thought that if the litter temperature increase associated with windrow composting is sudden enough that it may catch *Clostridium* off guard and unprepared to form a spore in time to save itself. However, acting quickly after the flock leaves to construct the windrows is critical, as is determining litter moisture content. Litter that is too dry will not allow the windrow to heat. If less than 30 percent moisture is present in the litter, the windrow will have a difficult time achieving a temperature of 130°F or greater, and bacterial growth may be inhibited, but the bacteria will not be killed. Too much moisture will have a similar effect. Litter moisture greater than 60 percent may prevent the windrow from heating because the litter is too wet.

**What to look for**

Gangrenous dermatitis has characteristic lesions that help identify it once you know what to look for. GD often starts with small pimples on the skin that quickly progress to include much larger areas of the bird. What often first attracts attention are what appears to be small areas on the top of the wings that look like raw sores or bloody spots. These may appear small early on, but there is generally much more of the bird affected than just the wing; however, the top of the wing is easy to see as you walk the flock and can alert you to the fact that something is wrong. You may pick up on the wing damage before the mortality starts to increase. If you miss the wing
Damage, you won’t miss the rapid and steep increase in mortality as the disease spreads through the flock.

Birds with the disease never show clinical signs for long because they die very rapidly, generally within 24 hours. Lesions on the skin may appear in a variety of colors from dark red to purple to green (Figures 1 and 2). Lesions most often appear on the abdomen, breast, legs and wings. Gas or gelatinous fluid often accumulates under the skin, between the skin and muscle tissue. Therefore, the skin often has a “spongy” feel to it because of gas buildup between skin and muscle tissue. Muscle tissue is also affected and may appear as raw, moist areas in locations where the skin has deteriorated. Dead birds deteriorate and decompose extremely rapidly. You may pick up dead birds early this morning and know you have them all but then go back before noon and find numerous dead birds that look like they have been dead several days instead of only a few hours.

You must pick up dead birds multiple times each day should your farm be affected with a GD outbreak. Healthy birds pecking at dead birds can contract the disease, and live birds that have the disease can infect healthy birds via feces, feeding equipment, etc. Birds with the disease don’t live long, but they can do plenty of damage before they die. Often, a fairly accurate diagnosis can be made by the rapid increase in mortality and the characteristic gross lesions. It is not something you will easily forget if you ever have a case on your farm. Therefore, should you have it a second time (which often happens until the Clostridium numbers in the litter are reduced), you’ll quickly know what you are dealing with. Be advised, it can be tracked from house to house, so take precautions and practice sound biosecurity when working in your houses. If you have an outbreak in one house, work that house last and dedicate footwear for each individual house to help prevent tracking it to the other houses.

Growers should closely monitor feed inventory. Do your best not to run out of feed. If your feed inventory runs low, contact your flock supervisor or the feed mill to make sure you are scheduled for a feed delivery soon. Bird activity greatly increases when hungry chickens receive feed. This results in an increase in the number of cuts, scratches and skin damage. In addition,
make sure migration fences are in place on schedule to prevent overcrowding in some quadrants of the house that can lead to increased skin damage and a greater GD risk. Avoid loud noises or any activity that may frighten the birds and result in increased scratches or skin damage. Follow a lighting program that helps keep the birds calm and controls their activity level. Calm flocks will have fewer scratches and skin damage than nervous, flighty flocks. Anything you can do that reduces stress and/or lessens the possibility of a skin injury will reduce the risk of a GD outbreak.

As with many other aspects of broiler production, management factors play a critical role in preventing and controlling a GD outbreak. Reports of outbreaks are often greater in summer and fall than in spring and winter. Daily mortality collection is important throughout the year but is especially critical during hot weather. It’s easy to miss a dead bird from time to time, but do your best not to miss one single dead bird from one day to the next. Collection of all mortality at least once daily is critical to disease prevention. Unsanitary conditions, poor farm management practices and stressful environmental conditions (wet litter, high humidity, overcrowded conditions) may predispose flocks to GD. In addition, chicken house pads with a soil pH > 6 may also be at a greater risk of GD infection. Wet litter (> 60 percent moisture) should be considered a risk factor for GD. High in-house humidity resulting from cool cell usage throughout the summer and fall may increase litter moisture content, thereby increasing GD risk.

**Summary**

Gangrenous dermatitis is a serious health concern for many commercial broiler operations that has increased in recent years. Its somewhat come-and-go nature can make it more difficult to determine all the factors that play a role in its occurrence. Prevention should be the overall goal because managing a GD outbreak is stressful, labor-intensive, time-consuming and expensive. *Clostridium* and other disease organisms that cause GD are everywhere in the poultry house environment, making it unlikely they can ever be totally eliminated. Fortunately, total elimination is not necessary; we just need to maintain their numbers below the threshold that the birds are able to resist a challenge and remain healthy. It’s when pathogen numbers get above that threshold that we see sick chickens. Growers should focus on sound management practices, farm hygiene and good litter and house environment conditions to reduce the GD threat to their flocks.
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References


