Technical Update

THIAMIN DEFICIENCY IN PULLETS

BACKGROUND

Thiamin, also known as vitamin B1, is an essential vitamin involved in metabolic energy supply and critical for normal growth and development of poultry. While cases of thiamin deficiency are rare, it is important to recognize the clinical signs and resolve the issue quickly in order to avoid mortality. Deficiency is caused by three primary routes: omission from the diet, inhibition of thiamin absorption, or destruction of the thiamin molecule. Omission from the diet usually occurs from complete failure to either add the vitamin to the premix or add the premix to the ration. Inhibition of thiamin absorption may occur from intestinal disease, amprolium toxicity, or mycotoxins. Destruction of the molecule can occur from thiaminase enzymes present in poor quality fish meal (3,6).

Many issues related to the feed milling process can lead to thiamin deficiency. Improper storage of the premix relating to temperature, humidity, length of storage, and packaging is most common. Failure to include thiamin in the premix or improper mixing procedure may also contribute to deficiencies. Evaluation of the feed milling process should be the primary step when managing suspected cases of vitamin origin.

Figure 1. Late stage deficiency with large numbers of affected birds.

Amprolium is a commonly used coccidiostat or treatment for active coccidiosis. Amprolium is very similar in structure to thiamin and can competitively exclude absorption of the vitamin from the intestinal tract. Further, amprolium is known to block phosphorylation of the vitamin which is required for proper function (4).

Thiamin availability is affected by breaking down the vitamin B1 molecule due to naturally present thiaminase enzymes. Poor quality fish meals can contain high levels of amines (potentially caustic compounds derived from nitrogen) and/ or thiaminases (3). High amine concentration in the meal often impacts the lining of the gizzard, causing erosions and is a more common indication of poor quality fish meal. Signs of gizzard erosions warrant an investigation of the fish meal source and usually occur before the clinical signs of thiamin deficiency develop. Fish meals can vary greatly in their concentration of thiaminases, and raw ingredients should be tested before inclusion as a protein source in the diet.

Figure 2. Affected bird exhibiting squatting posture (paresis).

CLINICAL SIGNS

In pullets and adult birds, clinical signs of thiamin deficiencies take approximately six weeks to develop and represent a chronic problem. For chicks, the onset of clinical signs is more rapid due to low carry over of thiamine from the breeding hen.

Eggs derived from parent stock fed low levels of thiamine will have compromised levels of thiamin. This will result in high 18-day embryonic mortality and subsequent hatch. Surviving chicks will have compromised levels of thiamin, resulting in polyneuritis typically demonstrated as muscle paralysis which causes extended legs and retraction of the head (1,5).

In older pullets, early signs of thiamin deficiency present as decreased feed intake, lethargy, and head tremors (6). As the condition worsens, a polyneuritis causes paresis, which progresses to eventual paralysis of the birds (Fig. 1–3).

Figure 3. Affected bird with full paralysis.

Due to the association with feed supply, manifestation of thiamin deficiency tends to occur across the entire farm as opposed to one single house. Morbidity can vary greatly depending on the level and chronicity, but has been observed in excess of 60%. Starvation and dehydration are sequalae of paralysis and the level of mortality related to culling is significant.

Thiamine deficiency is typically a diagnosis of exclusion. Primary differentials to address first include Marek's disease, influenza, Newcastle disease, monensin (ionophore) toxicity, peripheral neuropathy, and other vitamin deficiencies including riboflavin.

NECROPSY

Necropsy of culled birds or mortality is unrewarding, showing no specific gross lesions. Severely impacted birds will show signs of starvation and dehydration. Histopathology may show degeneration of the cells lining the duodenal crypts of Lieberkühn, with dilation and filling of the crypts with cellular debris and necrotic cells, and vacuolation of the pancreatic acinar cells with hyaline body formation (2).

TREATMENT

When a diagnosis of thiamin deficiency is suspected, quick action is advisable. On farms with progressive disease showing paralysis and paresis, it is possible to give birds an injectable form of thiamine. While exact dosages for intramuscular injection of thiamin in poultry have not been determined, successful recovery of mildly and moderately paralyzed birds was achieved with 5–8 mg/kg (11.0–17.6 mg/lb) body weight. Severely affected birds are unlikely to recover due to extreme starvation and dehydration and should be euthanized accordingly.

Given that thiamin is typically considered a multiple farm concern, it is recommended that all farms receiving feed from the same source be immediately supplemented in the water with high doses of thiamin, either in combination with other water-soluble vitamins or alone. Excess thiamin is readily excreted through the urine or feces in poultry. Over supplementation is not a concern, as the toxic dose is 700 times the requirement level. Maintain regular supplementation in the water until the source of thiamin deficiency has been determined and corrected.

Removing old feed and replacing with a fresh batch of adequately supplemented feed will also help to ensure a rapid return of thiamine to the flock. Treating the birds in this manner will help the flocks to recover quickly. In cases of thiamine deficiency shortly after hatch, this same procedure should be utilized in affected parent flocks.

PREVENTION

Thiamin requirements for poultry diets range between 2.2–3.0 mg/kg (2.2–3.0 g/ton; 4.9–6.6 mg/lb) (Hy-Line, 2019). A minimum of 2.2 mg/kg feed (4.9 mg/lb) in rearing phase and 2.5 mg/kg (5.5 mg/lb) of feed during the laying period is recommended for commercial birds. Ensure breeder diets are adequately supplemented with thiamin, providing 3.0–3.5 mg/kg (6.6–7.7 mg/lb) feed. Although microbial synthesis of thiamin occurs in the intestine, this is not a dependable source and should not be relied upon during feed formulation.

Ensure the thiamine supplemented in the diet is in a suitable form (typically thiamine mononitrate [98%] is used), and adequately mixed in a vitamin premix. The vitamin premix should be from a reputable source and provided at a suitable inclusion rate to reflect the capability of the feed mixing system. Thoroughly mix the premix with other feed ingredients to ensure even distribution of thiamin and other vitamins. Do not use old or improperly stored premix. Thiamin is particularly sensitive to heat, trace minerals, and a high pH. When heat treatment is utilized, consider adding additional premix as thiamine is sensitive to thermal processing. Store premixes in cool facilities and away from direct sunlight. When possible, provide vitamins in a separate premix relative to trace minerals. Keep retention samples of the premix and finished feed for future reference in the event of feed-related problems in the flock.

The presence of mycotoxins and parasitism can also contribute to thiamin deficiency. Minimize mycotoxin levels in feeds, as some *Fusarium*-based toxins have been associated with thiamin deficiency. The presence of endoparasites such as coccidia and helminths (worms) compete with the host for thiamin; therefore, reducing the susceptibility of the bird to parasite infestation can be critical to preventing nutrient deficiency.

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