Nutritional diseases(I)

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Vitamin Deficiencies in Poultry

- Vitamin deficiencies are most commonly due to inadvertent omission of a complete vitamin premix from the birds' diet. Multiple signs are therefore seen, although in general, signs of B vitamin deficiencies appear first. Because there are some stores of fat-soluble vitamins in the body, it often takes longer for these deficiencies to affect the bird, and it may take months for vitamin A deficiency to affect adult birds.

Vitamin A Deficiency

- Depending on liver stores, adult birds could be fed a vitamin A–deficient diet for 2–5 mo before signs of deficiency develop. Eventually, birds become emaciated and weak with ruffled feathers. Egg production drops markedly, hatchability decreases, and embryonic mortality increases. As egg production declines, there will likely be only small follicles in the ovary, some of which show signs of hemorrhage. A watery discharge from the eyes may also be noted. As the deficiency continues, milky white, cheesy material accumulates in the eyes, making it impossible for birds to see (xerophthalmia). The eye, in many cases, may be destroyed.

- The first lesion usually noted in adult birds is in the mucous glands of the upper alimentary tract. The normal epithelium is replaced by a stratified squamous, keratinized layer. This blocks the ducts of the mucous glands, resulting in necrotic secretions. Small, white pustules may be found in the nasal passages, mouth, esophagus, and pharynx, and these may extend into the crop. Breakdown of the mucous membrane usually allows pathogenic microorganisms to invade these tissues and cause secondary infections.

- The yellow pigment in the shanks and beaks is usually lost, and the comb and wattles are pale. A cheesy material may be noted in the eyes, but xerophthalmia is seldom seen because chicks usually die before the eyes become affected. Secondary infection may play a role in many of the deaths noted with acute vitamin A deficiency.
- Young chicks with chronic vitamin A deficiency may also develop pustules in the mucous membrane of the esophagus that usually affect the respiratory tract. Kidneys may be pale and the tubules distended because of uric acid deposits, and in extreme cases, the ureters may be plugged with urates. Blood levels level of ~5 mg of uric acid can rise from a normal

Vitamin D3Deficiency

- **Vitamin D3 is required for the normal absorption and metabolism of calcium and phosphorus. A deficiency can result in rickets in young growing chickens or in osteoporosis and/or poor eggshell quality in laying hens, even though with the diet may be well supplied calcium and phosphorus.**

- Laying hens fed a vitamin D3–deficient diet show loss of egg production within 2–3 wk, and depending on the degree of deficiency, shell instantly. quality deteriorates almost Retarded growth and severe leg weakness are the first signs noted when chicks are deficient in vitamin D3. Beaks and claws become soft and pliable. Chicks may have trouble walking and will take a few steps before squatting on their hocks. While resting, they often sway from side to side, suggesting loss of equilibrium. Feathering is usually poor, and an abnormal banding of feathers may be seen in colored breeds. With chronic vitamin D3 deficiency, marked skeletal disorders are noted

- Dry, stabilized forms of vitamin D3 are recommended to treat deficiencies. In cases of severe mycotoxicosis, a water-miscible form of vitamin D3 is administered in the drinking water to provide the amount normally supplied in the diet. In cases of impaired liver function, metabolites of vitamin D are the usual choice for treatment.

Vitamin E Deficiency

- The three main disorders seen in chicks deficient in vitamin E are encephalomalacia, exudative diathesis, and muscular dystrophy. The occurrence of these conditions depends on various other dietary factors. and environmental

1- Encephalomalacia

- Encephalomalacia is seen in commercial flocks if diets are very low in vitamin E, if an antioxidant is either omitted or is not present in sufficient quantities, or if the diet contains a reasonably high level of an unstable unsaturated fat. and Encephalomalacia may respond to vitamin E supplementation, depending on the extent of the damage to the cerebellum.

- **The classic sign of encephalomalacia is ataxia. The results from hemorrhage and edema within the granular layers of the cerebellum, with pyknosis and eventual disappearance of the Purkinje cells and separation of the granular layers of the cerebellar folia. Because of its inherently low level of vitamin E, the cerebellum is particularly susceptible to lipid peroxidation. In prevention of encephalomalacia, vitamin E functions as a biologic antioxidant. The quantitative need for vitamin E for this function depends on the polyunsaturated amount of linoleic acid and fatty acids in the diet.**

2- exudative diathesis

- **Exudative diathesis results in a severe edema caused by a marked increase in capillary permeability. Electrophoretic patterns of the blood show a decrease in albumin levels, whereas exudative fluids contained a protein pattern similar to that of normal blood plasma.**

3-muscular dystrophy

- Vitamin E deficiency accompanied by sulfur amino acid deficiency results in severe muscular dystrophy in chicks by ~4 wk of age. This condition is characterized by degeneration of the muscle fibers, usually in the breast but sometimes also in the leg muscles. Histologic examination shows Zenker's degeneration, with perivascular infiltration and marked accumulation of infiltrated eosinophils, lymphocytes, and histocytes.

Vitamin K Deficiency

- Impairment of blood coagulation is the major clinical sign of vitamin K deficiency. With a severe deficiency, subcutaneous and internal hemorrhages can prove fatal. Vitamin K deficiency results in a reduction in prothrombin content of the blood, and in the chick, plasma levels are as low as 2% of normal young
- Hemorrhagic syndrome in day-old chicks has been attributed to a deficiency of vitamin K in the diet of the breeder hens. Gross deficiency of vitamin K results in such prolonged blood clotting that severely deficient chicks may bruise or other injur bleed to death from a slight

- A vitamin K deficiency in poultry may be related to low dietary levels of the vitamin, low levels in the maternal diet, lack of intestinal synthesis, extent of coprophagy, or the presence of sulfur drugs and other feed additives in the diet. Chicks with coccidiosis can have severe damage to their intestinal wall and can bleed excessively.

Vitamin B12Deficiency

- Vitamin B12 is an essential part of several enzyme systems, with most reactions involving the transfer or synthesis of methyl groups. Although the most important function of vitamin B12 is in the metabolism of nucleic acids and proteins, it also in functions carbohydrate and fat metabolism

- In growing chickens, a deficiency of vitamin B12 results in reduced weight gain and feed intake, along with poor feathering and nervous disorders. Although deficiency may lead to perosis, this is probably a secondary effect due to a dietary deficiency of methionine or choline as sources of methyl groups. Vitamin B12 may alleviate perosis because of its effect on the synthesis of methyl groups. Other signs reported in poultry are anemia, gizzard erosion, and fatty infiltration of the heart, liver, and kidneys. Laying hens initially appear to be able to maintain body weight and egg production; however, egg size is reduced. In breeders, hatchability can be markedly reduced

Pyridoxine (Vitamin B6) Deficiency

- A vitamin B6 deficiency causes retarded growth, dermatitis, and anemia. Because a major role of the vitamin is in protein metabolism, deficiency can result in reduced nitrogen retention

- Anemia is often noted in ducks but is seldom seen in chickens and turkeys. Young chicks may show nervous movements of the legs when walking and often undergo spasmodic convulsions, leading to death. During convulsions, the chicks may run about aimlessly, flapping their wings and falling with jerking motions

- **In adult birds, pyridoxine deficiency results in reduced appetite, leading to reduced egg production and a decline in hatchability. Severe deficiency can cause rapid involution of the ovary, oviduct, comb, and of the testis in wattles, and cockerels**

Thiamine Deficiency(B1)

In the initial stages of deficiency, lethargy and head tremors may be noted. A marked decrease in appetite is seen in birds fed a thiamine-deficient diet. Poultry are also susceptible to neuromuscular problems, resulting in impaired digestion, general weakness, star-gazing, and frequent convulsions.

- **Polyneuritis may be seen in mature birds ~3 wk after they are fed a thiamine-deficient diet. As the deficiency progresses, birds may sit on flexed legs and draw back their heads in a star-gazing position. Retraction of the head is due to paralysis of the anterior neck muscles. Soon after this stage, chickens lose the ability to stand or sit upright and topple to the floor, where they may lie with heads still retracted**

Riboflavin Deficiency(B2)

- Many tissues may be affected by riboflavin deficiency, although the epithelium and the myelin sheaths of some of the main nerves are major targets. Changes in the sciatic nerves produce "curled-toe" paralysis in growing chickens. Egg production is affected, and riboflavin-deficient eggs do not hatch

- **When chicks are fed a diet deficient in riboflavin, their appetite is fairly good but they grow slowly, become weak and emaciated, and develop diarrhea between the first and second weeks. The characteristic sign of riboflavin deficiency is a marked enlargement of the sciatic and brachial nerve sheaths; sciatic nerves usually show the most pronounced effects. Histologic examination of the affected nerves shows degenerative changes in the myelin sheaths that, when severe, pinch the nerve.**

- **Signs of riboflavin deficiency in hens are decreased egg production, increased embryonic mortality, and an increase in size and fat content of the liver. Hatchability declines within 2 wk when hens are fed a riboflavin-deficient diet but returns to near normal when riboflavin is restored. Affected embryos are dwarfed and show characteristically defective "clubbed" down. The nervous system of these embryos shows degenerative changes much like those described in riboflavin-deficient chicks**

Choline Deficiency

In addition to poor growth, the classic sign of choline deficiency in chicks and poults is perosis. Perosis is first characterized by pinpoint hemorrhages and a slight puffiness about the hock joint .

- followed by an apparent flattening of the tibiometatarsal joint caused by a rotation of the metatarsus. The metatarsus continues to twist and may become bent or bowed so that it is out of alignment with the tibia. When this condition exists, the leg cannot adequately support the weight of the bird. The articular cartilage is displaced, and the Achilles tendon slips from its condyles. Perosis is not a specific deficiency sign; it appears with several nutrient deficiencies. Although choline deficiency readily develops in chicks fed diets low in choline, a deficiency in laying hens is not easily produced. Eggs contain ~12–13 mg of choline/g of dried whole egg. A large egg contains ~170 mg of choline, found almost entirely in the phospholipids.

Niacin (Nicotinic Acid) Deficiency

- **Niacin deficiency is characterized by severe disorders in the skin and digestive organs. The first signs are usually loss of appetite, retarded growth, general weakness, and diarrhea. Deficiency produces enlargement of the tibiotarsal joint, valgus-varus bowing of the legs, poor feathering, and dermatitis on the head and feet. Niacin deficiency in chicks can also result in "black tongue." At ~2 wk of age, the tongue, oral cavity, and esophagus become distinctly inflamed. In the niacin-deficient hen, weight loss, reduced egg production, and a marked decrease in hatchability can result.**

- **Ducks and turkeys with a niacin deficiency show a severe bowing of the legs and an enlargement of the hock joint. The main difference between the leg seen in niacin deficiency and perosis as seen in manganese and choline deficiency is that with niacin deficiency the Achilles tendon seldom slips from its condyles.**

Pantothenic Acid Deficiency

- The major lesions of pantothenic acid deficiency involve the nervous system, the adrenal cortex, and the skin. Deficiency may result in reduced egg production; however, a marked drop in hatchability is usually noted before this event. Embryos from hens with pantothenic acid deficiency can have subcutaneous hemorrhages and severe edema, with most mortality showing up during the later part of the incubation period. In chicks, the first signs are reduced growth and feed consumption, poor feathering with feathers becoming ruffled and brittle, and a rapidly developing dermatitis. The corners of the beak and the area below the beak are usually the worst affected regions for dermatitis, but the condition is also noted on the feet. In severe cases, the skin of the feet may cornify, and wart-like lumps occur on the balls of the feet. The foot problem often leads to bacterial infection.

Folic Acid (Folacin) Deficiency

- Poultry seem more susceptible to folacin deficiency than other farm animals. Deficiency results in poor feathering, slow growth, an anemic appearance, and sometimes perosis. As anemia develops, the comb becomes a waxy-white color, and pale mucous membranes in the mouth are noted.

- **The abnormal feather condition in chickens leads to weak and brittle shafts, and depigmentation develops in colored feathers. Although a folacin deficiency can result in reduced egg production, the main sign noted with breeders is a marked decrease in hatchability associated with an increase in embryonic mortality, usually during the last few days of incubation. Embryos have deformed beaks and bending of the tibiotarsus. Birds may exhibit perosis.**

Biotin Deficiency

- Biotin deficiency results in dermatitis of the feet and the skin around the beak and eyes similar to that described for pantothenic acid deficiency .Perosis and footpad dermatitis are also characteristic signs. Although signs of classic biotin deficiency are rare, occurrence of fatty liver and kidney syndrome (FLKS) is important to commercial poultry producers.