Gout in Poultry/Birds

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Uric acid is produced in the liver and is the end product of nitrogen metabolism in birds. Consequently, birds can develop gout secondary to an accumulation of urates. Gout is not a disease entity but a clinical sign of severe renal dysfunction that causes hyperuricemia and accumulation of urates in tissues of birds. There are two different syndromes that differ in etiology, morphology and pathogenesis.

Cause, transmission, and epidemiology: The condition is due to a failure of urinary excretion, which can be as a result of:

- Obstruction of ureters
- Dehydration – most common cause
- Renal damage – kidney necrosis
- Vitamin A deficiency
- Secondary to urolithiasis
- Treatment with sodium bicarbonate

Except for the first cause, all occur due to turnover of nucleoprotein to cause hyperuricemia and precipitation and crystallization of urates. Other causes are mycotoxins such as oosporein, and renal cryptosporidiosis. Mechanisms of crystal precipitation in certain sites are unknown.

The two forms are visceral gout and articular gout.

A. Visceral Gout:

Clinical signs and lesions: The urate deposits appear as white chalky coating. Within the viscera they are observed microscopically as blue or pink amorphous material or as feathery crystals or basophilic spherical masses in tissues.

Diagnosis: This form is characterized by precipitation of urates in the kidneys, and serous surfaces of the liver, heart, mesenteries, air sacs or peritoneum. In severe cases surfaces of muscles and synovial sheaths of tendons and joints are affected. Precipitation of urates can occur within the liver, spleen and other organs.

Treatment: Correcting the cause of the renal damage will prevent other birds from developing gout.

Prevention: Visceral gout is prevented by feeding a correctly balanced diet and providing adequate water for chickens.

Recovery: Individual affected birds will not recover, but once the cause of the condition is corrected, there will not be an increased residual risk to new birds on the premises.

B. Articular Gout

Cause, transmission, and epidemiology: This form is generally restricted to individual birds and may be due to genetic defects in metabolism of uric acid. It may be a result of feeding high protein diets, which result in excess uric acid production. Affected chickens have a defect in kidney tubular secretion of uric acid.

Clinical signs and lesions: Affected birds have shifting lameness and an inability to bend the toes. The disease is characterized by tophi - deposits of urates around joints, especially the feet (confused with bumble foot). Joints are enlarged and feet are deformed. In chronic cases, urate precipitation is found on the comb, wattles, trachea and other areas.

Differential diagnosis: This form of gout has to be differentiated from other diseases causing joint and skin swellings and abscesses, such as bumble foot, lymphoid leukosis (osteopetrosis), infectious tenosynovitis, *Mycoplasma synoviae*, Staphylococcosus.

Diagnosis: On opening the joints, periarticular tissue is white due to urate deposition, and semifluid deposits of urates are seen.

Treatment: There is no treatment for this condition although providing a lower protein diet may be helpful.

Prevention: Provide a lower protein diet, especially for males.

Recovery: This is a sporadic problem of little economic importance in poultry. Individual birds will not recover.