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# Gout in Domestic Fowl: A Comprehensive Overview

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Gout in domestic fowl is a complex metabolic disease that can impact birds of all ages. It is primarily characterized by the deposition of uric acid and urate crystals in various organs due to impaired renal excretion. When kidney function diminishes, uric acid accumulates in the blood and bodily fluids, subsequently precipitating as calcium or sodium urate crystals in the kidneys and serous membranes of the heart, mesenteries, air sacs, and joints. The disease is classified into two main types based on the site of uric acid deposition: visceral gout and articular gout.

**Types of Gout**

- **Visceral Gout:** This acute form of gout is characterized by the accumulation of urate crystals in internal organs, including the kidneys, liver, heart, mesenteries, air sacs, and peritoneum. In severe cases, urate deposits can also appear on the surface of muscles, synovial sheaths, tendons, and joints.
- **Articular Gout:** This chronic form of gout involves urate deposition around the joints, leading to enlargement and deformation. On examination, the periarticular tissue appears white due to urate deposits. Articular gout is often associated with high-protein diets or defects in the tubular secretion of uric acid.

Psittacine birds, such as budgerigars, parakeets, and parrots, are frequently affected by gout. In the poultry industry, gout can lead to significant economic losses due to mortality, poor growth, and decreased egg production.

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**Susceptibility**

Both broilers and layers are susceptible to gout, with higher incidences observed in broilers below 2-3 weeks of age. Visceral gout is more common in broilers as young as 2-3 days old and in layer pullets over 14 weeks.

**Etiology**

Gout in fowl can arise from various etiological factors, including nutritional/metabolic imbalances, infectious agents, toxicological substances, and environmental conditions.

- **Nutritional/Metabolic Causes:** These include excessive protein consumption, vitamin A deficiency, high sodium intake, excessive use of sodium bicarbonate, sudden dietary changes, low phosphorus, excess dietary calcium, amino acid imbalances, and water deprivation.
- **Infectious Origins:** Viruses, bacteria, and fungi such as Infectious Bronchitis (nephrotropic strain), Avian Nephritis Virus, Infectious Bursal Disease, Influenza A, Clostridium baratii, Ustilago maydis, and Chaetomium trilaterale can contribute to gout.
- **Toxicological Factors:** Various feed additives, antibiotics (e.g., aminoglycosides, sulphonamides, furazolidone), vitamins (particularly vitamin D), minerals (e.g., lead, calcium, phosphorus, sodium, fluoride), urea-mixed feed ingredients, fungal toxins (aflatoxins, ochratoxins, cyclosporins, ergotamine, oosporins), and disinfectants (phenols, cresols) can induce gout. Overuse of copper sulfate in drinking water can lead to water refusal, dehydration, and gout.
- **Miscellaneous Factors:** Environmental factors such as dampness in poultry sheds and cold exposure have also been linked to the development of gout.

**Pathophysiology**

In birds, uric acid is the end product of nitrogen metabolism and a primary nitrogenous waste from protein breakdown. Unlike mammals, birds lack the enzyme urease, making uric acid their final excretory product. Produced in the liver and excreted by the kidneys, higher levels of uric acid can precipitate in tissues. Normally, blood urate levels are 4-5 mg/dL, with 10% present in solution. Elevated uric acid levels can result from leukemia, pneumonia, or extensive cell disintegration due to increased purine biosynthesis. Clinical symptoms of gout appear when blood urate levels reach 21.4 mg/dL, and death occurs at levels around 47

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mg/dL.Uric acid itself is non-toxic, precipitated crystals cause mechanical damage to tissues. In cases of kidney damage, uric acid accumulates in the blood and tissues, leading to gout.

**Diagnosis**

Diagnosing gout involves assessing clinical signs, postmortem lesions, and conducting the Murexide test to confirm uric acid deposits.

**Clinical Signs**

Common clinical signs include loss of appetite, depression, emaciation, greenish diarrhea, lethargy, and sudden death. Affected chicks often appear dull with ruffled feathers and moist vents. In articular gout, birds exhibit shifting leg lameness, swollen and tender joints, and an inability to fly. As the disease progresses, pain and distress increase.

**Postmortem Examination**

Key lesions observed during postmortem examination include chalky white urate deposits on visceral organs, enlarged and swollen kidneys, distinct markings on kidney tubules, and thick, gray-white deposits in ureters. Joint cavities and tendon sheaths may be filled with thick, whitish material and desquamated epithelial cells with fatty degeneration.

**Therapeutic Management**

Effective treatment of gout involves a combination of therapeutic, nutritional, and management practices:

- Reduce dietary protein and sodium chloride levels.
- Provide vitamin A supplements (10,000 IU/bird).
- Administer molybdenum (400 mg/bird) and allopurinol to lower serum uric acid levels.
- Incorporate diuretics in feed and water to restore kidney function.
- Use ammonium sulfate (5 kg/ton) and ammonium chloride (10 kg/ton) in feed.
- Apply tincture of iodine to affected joints.
- Provide natural vinegar (15-50 ml/100 birds) in drinking water.
- Management Practices
- Maintain constant temperature.
- Ensure easy access to feed and water.
- Use smooth, flat, and broad perches made of softwood at low levels.
- Prevention and Control

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- Provide adequate water and avoid high-protein diets.
- Vaccinate against infectious bronchitis in areas where it is endemic.
- Control nephrosis-causing bacteria like E. coli.
- Avoid prolonged antibiotic use.
- Regularly analyze calcium and phosphorus levels in feed.
- Store feed properly to prevent fungal growth and mycotoxin contamination.
- Incorporate sodium carbonate in feed to improve egg quality.
- Use potassium chloride (1 g/L) in acute cases.
- Provide electrolytes to control mortality.
- Use urine acidifiers like ammonium sulfate and ammonium chloride.
- Ensure proper watering systems and adequate vitamin levels.
- Maintain proper temperature and humidity during chick transportation to avoid dehydration.