AVIAN UROLITHIASIS
(Renal or Visceral Gout)

Dr. Bernie Beckman
Hy-Line International

Visceral gout is a condition of chickens that has been recognized for more than 30 years. Due to its striking lesions, many names have been used to describe visceral gout such as acute toxic nephritis, renal gout, kidney stones, nutritional gout, nephrosis, and others. Visceral gout is readily recognized by its distinctive lesions which are characterized by white chalk-like deposits covering the surface of various abdominal organs as well as the heart sac. Recently, gout has been sporadically reported in Great Britain, the United States, and other countries.

Gout: White chalky deposits over the heart, liver, and abdominal cavity

Gout is a condition in which kidney function has decreased to the point where uric acid (nitrogenous waste) accumulates in the blood and body fluids. The uric acid subsequently precipitates as calcium-sodium-urate crystals in a variety of locations, particularly in the kidneys and the serous membranes of the liver, heart, mesentery, air sacs, and joints. The damaged kidneys are characterized by atrophied or missing portions of kidney lobes, kidney and ureter stones, and remaining kidney tissue that is swollen and white with urates.

Stones in dilated ureter and kidneys

Although gout has been recognized for some time as a cause of pullet and layer mortality, it continues to be a diagnostic challenge. In some cases, birds are in full production and exhibit few external symptoms prior to death. Chickens affected by renal damage can continue to be productive until less than one third of their normal kidney mass remains functional. Compensatory enlargement of remaining normal kidney tissue is responsible for maintaining adequate renal function, at least for a time.
Top kidney is swollen and enlarged, compensatory hypertrophy

Due to the sporadic occurrence of outbreaks of gout, the question is often asked, “What is gout and why did this suddenly happen?” Layer mortality can double in an affected flock, but the birds with renal damage maintain their level of egg production until near time of death. The subsequent economic problems that are associated with mortality and loss of production can be significant. Gout is not a single disease entity, but rather a potential sequel to kidney damage from any of a number of causes, which can be infectious, nutritional, toxic, or possibly a combination of these.

Kidney Anatomy and Function

The long, paired kidneys of the chicken are located in bony depressions of the lumbar region of the abdominal cavity. They are normally reddish-brown in color and have three distinct lobes or divisions. The primary function of the kidney is to maintain the chemical composition of body fluids (blood). The kidney serves a variety of other functions in the body: removal of metabolic waste and toxic products, conserving fluids and vital electrolytes, regulation of blood volume, and production of hormones which regulate blood pressure.

The kidney is truly a vital organ. When renal function stops, uric acid, normally excreted by the kidney, is then deposited any place that blood is circulated. A higher percentage of blood is circulated to the abdominal organs, heart, liver, and kidney, thus the lesions associated with gout are more pronounced in those organs. A bird with no renal function will likely die within 36 hours.

Causes of Gout

The cause of gout is often multi-factorial. Like many diseases, it can be complicated by several factors and it is often difficult to determine the primary cause. The common factors that can cause or contribute to gout are nutritional, infectious, toxic, and others.

Nutritional

Nutritional or metabolic factors known to affect the kidneys are:

1. Excess dietary calcium (layer diets) fed to immature pullets over a period of time will result in kidney damage that may lead to gout. Research has shown that stones which develop are of calcium-sodium-urate composition. Layer diets that contain high levels of calcium, but low available phosphorus, are known to contribute to gout.

2. Sodium bicarbonate is sometimes used to improve egg shell quality or combat the effects of heat stress. At high levels, sodium bicarbonate can contribute to gout by making the urine more alkaline. An alkaline urine, with high levels of calcium, is an ideal medium for formation of kidney stones.

3. Water deprivation, because of mechanical error or salt deficient diets, may affect the kidneys, but seem to have less effect because of the birds’ tendency to consume less feed (i.e., less calcium) when water is not available or the stimulus to drink (sodium) is not present.

4. Vitamin A deficiency over a long period of time can cause damage to the lining of the ureters, but would be rare with modern vitamin formulation.

5. Excessive dietary protein (30-40%) can produce gout in experimental birds.

Infectious

Viral agents known to be involved in gout are infectious bronchitis, avian nephritis virus, and related viruses.

Infectious bronchitis is a highly contagious infection of chickens which normally involves the respiratory tract, but can also affect the reproductive and urinary systems. Certain strains of bronchitis such as Holte, Gray, Italian, and Australian T have a greater affinity for the kidneys than other strains. The strains of IB that cause kidney disease do not tend to cause respiratory symptoms or egg shell problems. Other unclassified nephropathogenic IB-type viruses have been isolated from field cases. While several known and unclassified strains of IB are nephropathogenic, the usual respiratory viruses such as Holland and other vaccine strains of infectious bronchitis tend to have no predilection for the kidneys.

One mechanism by which bronchitis may affect the kidneys is chronic viral persistence leading to nephritis and renal failure over time. As young birds are most susceptible to bronchitis kidney damage, the initial infection may occur long before renal function is critically reduced and mortality occurs. When pullets sexually mature, they are placed on higher calcium diets to meet the demands of egg production. If the kidneys were previously damaged, they may no longer function normally with the higher level of calcium excretion, and the result is likely to be gout.

Avian nephritis virus, an enterovirus which grows in the kidneys, has been associated with kidney disease and serious outbreaks of mortality in Europe and Asia. Serological surveys show that antibodies to ANV can be
detected in chicken and turkey sera from around the world. Although most information at this time suggests only a subclinical role for ANV, research has shown ANV inoculated into healthy chickens will cause inflammation of the kidneys.

**Toxins**

Poisonous substances (toxins), while widely distributed in nature, are not considered major causes of production loss or disease in poultry in most countries. There are, however, problems that can arise from nephrotoxic substances. Some potentially toxic substances are used on a routine basis, while others are the result of accidental exposure to flocks.

Products used on a routine basis that have potential for human error and resulting toxicity are antibiotics, anticoccidials, minerals, vitamins, manufactured chemicals, and pesticides.

1. **Antibiotics**, such as sulfas and aminoglycosides, are eliminated from the body via the kidneys and are potentially nephrotoxic.
2. **Minerals** and **vitamins** such as calcium, phosphorus, sodium, and vitamin D₃ are potentially toxic to chickens. Chicks, however, are much more sensitive to toxicity, probably because their kidneys are not fully developed.
3. **Disinfectants** and insecticides, when used according to manufacturers’ recommendations, are safe and effective, but a miscalculation of dosage can result in toxicity.
4. **Feeds contaminated by mycotoxins** and phytotoxins (plant toxins) may also result in toxicity. Citrinin, ochratoxin, and oosporein are mycotoxins that are nephrotoxic. Although these toxins will cause alterations in kidney function, they may not necessarily lead to gout.

**Control**

Gout, when recognized as a problem in field cases, is likely to be a multi-factorial problem and identifying a specific cause is often difficult. Another reason for diagnostic difficulty is that kidney damage occurring during the growing period often has no apparent signs until the birds later come into egg production and are fed high calcium diets. Field cases and research have demonstrated the importance of interactions between two or more contributing factors, especially nutrition and infectious bronchitis.

Some preventive areas concerning nutrition are:

1. Pullets need no more than 1% calcium up to 15 weeks of age. High calcium in pullets can cause serious kidney damage. Likewise, if available phosphorus levels are low, kidney damage can be exaggerated. If a pre-layer ration is being used around 16 weeks of age to 5% production, a calcium content of 2.25-2.50% should be sufficient.
2. Sodium bicarbonate is sometimes used to improve egg shell quality. Sodium bicarbonate makes the urine more alkaline and thus creates an excellent environment for calcium-sodium-urates to form in a damaged kidney. Make sure only a recommended level is being used in older flocks and only when needed for egg shell quality. A flock with gout should not be medicated with sodium bicarbonate or fed a feed which is highly alkaline (high levels of sodium or potassium).
3. Review calcium-phosphorus levels in the feeding program. Pullet and layer feeds should be routinely analyzed for calcium and phosphorus levels.
4. Feed samples could be assayed for the presence of the mycotoxins citrinin, ochratoxin, and oosporein.
5. Water deprivation should be avoided in growing and laying flocks.

The role of nephropathogenic bronchitis viruses is not totally defined, but combinations of nephropathogenic strains with other nutritional factors will produce gout in experimental birds. Most vaccine strains of IB are not nephropathogenic. To determine the possible role of IB in outbreaks of gout, the following areas should be investigated:

1. Review of IB vaccination program
2. IB vaccines administered according to the manufacturer’s directions and recommendations. To provide adequate protection against a field challenge, especially nephropathogenic strains, a vaccination program should include several live vaccinations of bronchitis (can be in combination with Newcastle) at about three weeks, eight weeks, and 14 weeks of age by spray or drinking water. The degree of reactivity of the vaccinations starts low and then is gradually increased each succeeding time by using either a less attenuated virus or by giving it by a more reactive method, such as spray instead of water. The vaccine strains used should be those known to be endemic in the area. A standard live vaccination program should provide adequate protection against most field challenges of IB. Killed vaccines or live boosters throughout lay may help keep bronchitis immunity high.
3. Due to the complex clinical signs associated with IB, a diagnostic laboratory should be enlisted to help in diagnosis and in virus isolation. An initial serological survey of chickens for IB by age group should be performed. Virus isolation may be assisted with the use of SPF sentinel birds placed in the housing complex and isolation attempts after two to three weeks of exposure.

**Treatment**

Current techniques for reducing gout mortality in pullets and layers rely on acidifying the urine to keep kidney stones dissolved and preserve functional renal mass of affected birds. Experimental diets containing ammonium chloride, ammonium sulfate, DL-methionine, 2-hydroxy-4-(methylthio) butanoic acid (HMB, Alimet) all
successfully acidify the urine to reduce Ca-induced kidney damage.

For flocks experiencing gout mortality, ammonium sulfate at up to 5 Kg/ton (10 Lbs./ton) of the diet, ammonium chloride at up to 10 Kg/ton (20 Lbs./ton), DL-methionine at up to 6 Kg/ton (12 Lbs./ton) of the diet, or Alimet at up to 6 Kg/ton (12 Lbs./ton) have been used to reduce mortality. Wet droppings may be a side effect of ammonium chloride usage, but not with the other treatments. A lower level of the above rates should be used initially and then gradually increased over several weeks to these final levels. After several weeks of the treatment level, if the desired results have been obtained, gradual removal may be started. However, some level of treatment may be needed for the flock’s life.

**Summary**

Research supports a strong link for bronchitis and high calcium as a major cause of gout mortality. While this is likely to be the underlying cause for most renal damage, other factors such as electrolyte balance, mycotoxins, and water deprivation need to be recognized as possible contributing factors. Likewise, any diet that increases urine alkalinity in combination with high calcium can contribute to mortality in pullets and layers. If gout does occur in a flock, mortality can be reduced by increasing the acidity of the urine to dissolve existing kidney stones or to prevent additional kidney stones from forming.