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Gout Management in Poultry

Metabolic disorders arise as a result of improper metabolic processes within the birds' body, led by abnormal bio-chemical reactions due to improper functioning of the vital organs like the kidney, liver, heart and lung.

Of them, the kidney is one of the vital organs that carry out diverse metabolic and excretory functions such as maintaining the chemical composition of body fluids, removal of metabolic waste and toxic products, regulation of blood pressure and blood volume and conservation of fluids and electrolytes.

One of the important metabolic disorders associated with kidney damage in poultry is gout.

Types of gout :

Uric acid is the end product of purine and protein metabolism in poultry. Uric acid is formed in the liver and excreted by kidneys. Birds are uricotelic, lack the enzyme uricase and this along with the process of water conservation, allows them to excrete urine in the form of semi-solid uric acid in the faeces.

Gout is due to disruption in the metabolic process of excretion of uric acid. Gout can arise either due to production of uric acid over the capacity of kidneys to excrete it, or due to compromised kidney function

failing to excrete the produced uric acid, of which the latter is the more common reason for incidence. Reduced excretion of uric acid leads to its accumulation in blood and body fluids. This subsequently favours its precipitation in various tissues.

Growth depression and mortality can be due to high levels of uric acid in blood and/or due to uric acid deposit in tissues causing mechanical damage consequently affecting their function.

Depending on the sites of uric acid deposition, gout is classified as:

- **Visceral gout**– Deposition of uric acid crystals in visceral organs like kidney, liver, heart and gut. It is the acute and more common form of gout reported mostly in young poultry, so called as “Baby Chick Nephropathy”. It causes huge mortality in poultry ranging from 15-35 per cent.
- **Articular gout**– Deposition of uric acid crystals in joints, ligaments and tendon sheath. It is chronic form of gout, has some genetic predisposition and rarely seen in poultry.

In both forms, deposits consist of white chalky needle shaped shiny crystals called ‘*tophi*‘. In gout, blood levels of uric acid can be as high as 44mg/100ml as compared to 5-7mg/100ml in a normal bird.

Causes of gout:

A. Nutritional causes

- Minerals
- Calcium: Phosphorus (Ca:P) ratio – Excess dietary calcium (>3%) with low available phosphorus(<0.6%) results in precipitation of calcium-sodium-urate crystals. Phosphorus acts as urine acidifier and low phosphorus leads to urate crystal formation.
- Sodium – Sodium intoxication puts extra stress on kidneys. Excessive use of sodium bicarbonate increases alkalinity of urine leading to kidney stone formation. Gout due to sodium intoxication is seen in younger birds when the sodium levels exceed 0.4% in water and 0.8% in feed. This generally happens when fish meal is used in the diet (even with

normal salt content), since fish meal is rich in salt content. Total content of sodium chloride in feed should not exceed 0.3%.

- Vitamins
- High levels of vitamin D_3 increases calcium absorption from the gut which can favour further formation and deposition of urate crystals.
- Prolonged vitamin A deficiency causes sloughing of tubular epithelium and subsequent blockade resulting in accumulation of urates in the kidney.
- Protein
- In case of pre-existing kidney damage, feed containing more than 30 per cent of crude protein will lead to excessive uric acid production which further worsens the kidney function.
- Adulteration of protein supplements with urea increases the nitrogenous component, further enhancing the uric acid production

B. Infectious causes

- Viral causes
- Infectious bronchitis virus (IBV) – Nephropathogenic strains of IBV affects the kidneys leading to nephritis and high mortality. The disease, when vertically transmitted, affects the kidney of progeny leading to gout in young chickens.
- Infectious bursal disease (IBD) – Though not to a large extent, this disease has been one of the predisposing factors for gout.

C. Metabolic cause

- Ascites – Hypoxic conditions increases the production of uric acid. Ascites in initial stages can lead to symptoms of gout.

D. Mycotoxins

- Ochratoxin, citrinin, ochratoxin, oosporin and many of the pesticide/insecticide residues have ill-effects on kidney tissue, causing inflammation of kidney tubules and ureters.

E. Managemental causes

- Water deprivation

- Improper brooding temperature – too high or too low brooding temperature, heats up or chills the water respectively – thereby reducing the water
- Inadequate number of waterer/nipples
- Improper height of waterers
- Water withdrawal during vaccination
- Too low water pH – leads to water rejection by poultry or irritates the epithelium
- Improper hatchery management
- Improper egg storage
- Inadequate incubation conditions
- Improper conditions in chick holding room
- Chicks held for a long in hatchery or transported for a long distance without water

F. Other causes

- Drugs and chemicals
- Antibiotics in high doses like gentamycin, sulphonamides and nitrofurosones are known to cause renal damage especially in young chicks. The drugs which get excreted through the kidneys have their own imbalancing effect on pH and renal metabolism.
- Phenol and cresol derivativeshave residual toxic effects on kidneys
- High doses of copper sulphate in water can lead to gout subsequent to reduced water intake by birds.
- OBSTRUCTION IN THE URETER OF THE BIRD.

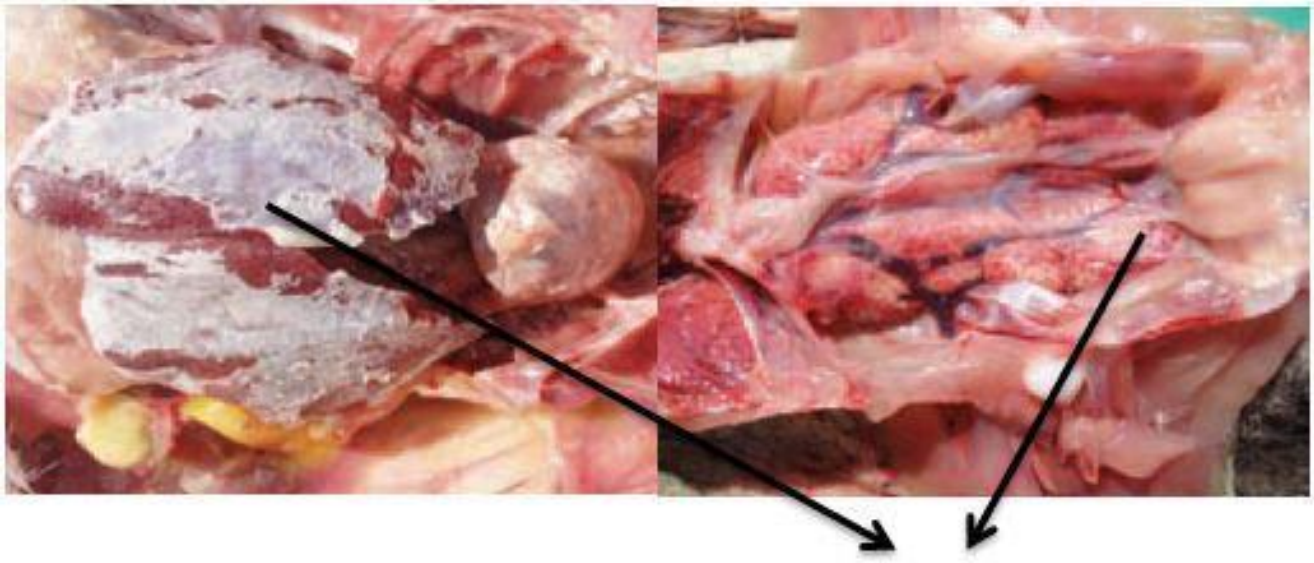
Clinical signs of gout:

- Depression
- Reduced feed intake
- Ruffled feathers
- Emaciation
- Lameness

- Moist vent
- Enteritis

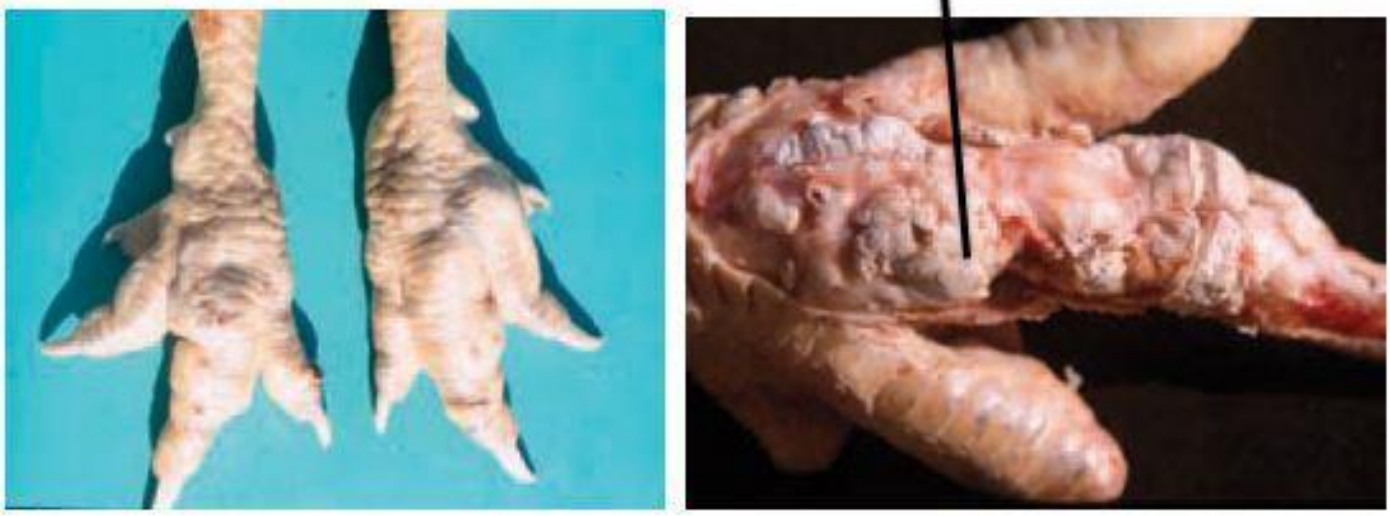
  Post-mortem lesions show urate deposition in kidney, liver, heart, gut,joints, ligaments and tendon .There is irregular and excessive enlargement of kidney lobules.

Visceral gout



Uric acid crystals

Articular gout



(Source: Diseases of poultry – Ivan Dinev, 2010)

Treatment, Prevention and control:

When having a sight upon the causes, the following prevention ideas can be suggested:

1. Disease management

1. Infectious bronchitis (IB) vaccination in time.
2. Proper screening of raw materials for mycotoxins, feed need to be supplemented with good quality toxin binders and acidifiers to take care of mycotoxin and microbial (bacterial and fungal) load respectively
3. Proper use of antibiotics, disinfectants, chemicals, anticoccidial etc.

2. Hatchery and farm management

1. Optimal incubation conditions – correct temperature and humidity need to be provided.
2. Optimal egg storage and handling conditions.
3. Chicks should not be held for long without water
4. Optimal brooding temperature
5. Adequate ventilation

3. Feed and water management

1. Balanced feed with respect to optimal calcium: phosphorus ratio, ideal electrolyte balance should be adopted. The sodium content in feed should not exceed more than 0.5 per cent.Use of jaggery (concentrated sugar) also seems to be beneficial.
2. Ensure adequate levels of A, D_3, K and B complex vitamins
3. Feed protein should not exceed the standard breed requirements. The raw materials should be carefully monitored for adulteration with urea, which might be one of the reasons for increased uric acid production. Feed dilution should be practiced when gout is observed. This can be carried out either by partial dilution of feed with grain source or by entire replacement of feed with pure grain sources (ground maize) for three to five days to reduce the exertion of kidneys to expel uric acid.
4. Provision of Ample numbers of waterers, urine acidifiers (like Vinegar @1-2 ml per litre water up to 24 hours, Potassium chloride @ 1 gm per litre water up to 24 hours, Ammonium chloride @2.5 kg/ton feed for 7 days, Ammonium sulphate @ 2.5 kg/ton feed for 7 days),kidney revitalisers

The following drugs have been successfully tried to treat gout:

1. Allopurinol@2 mg/Kg body weight in 24 hrs drinking water. Allopurinol decreases both uric acid formation and purine synthesis.
2. Probenecid:@2 mg per kg Body weight in 24 hrs drinking water. Probenecid works by interfering with the kidneys organic anion transporter (OAT), which reclaims uric acid from the urine and returns it to the plasma. If probenecid (an organic acid) is present, the OAT binds preferentially to it (instead of to uric acid), preventing re-absorption of the uric acid.

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