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Pathological Manifestations of Naturally Occurring Gout in Broiler chickens reared in North Kashmir

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Abstract
Chicken is more susceptible to gout due to uricotelic mechanism for excretion of nitrogenous waste and the absence of the enzyme uricase, which converts uric acid into less harmful substances. Uric acid itself is not toxic but precipitated crystals can cause severe mechanical damage to tissues like kidneys, heart, lungs and intestine leading to visceral gout and also articular gout in joints. Gout is a complex metabolic disease influenced by infectious agents, nutritional factors and management practices. The gross examination revealed swollen kidneys with chalky white deposits on the surface. Microscopically, the kidneys showed tophi formation or urate granulomas in the interstitial spaces, containing areas of caseous necrosis surrounded by urates. The histological sections stained with H & E highlighted the presence of urate crystals in the interstitial, intertubular and intraglomerular areas, surrounded by epithelioid cells. The duplicate sections stained with De-Galantha's method displayed urate particles as black masses, needles or acicular crystals arranged in a radiating pattern. Based on this study, it was concluded that gout in birds caused marked pathological changes in the visceral organs of the affected birds causing huge economic loses to poor farmers.

Keywords: Gout, Poultry, Kidney, Uric Acid, Gross Pathology, Histopathology

Introduction
Avian species often have multiple kidney diseases and most diseases in poultry are either bacterial, viral or parasitic (Cojean *et al*., 2020) [5]. Gout is mainly due to disorders of secretion through the kidneys, severe dehydration and use of nephrotoxic drugs, fungal toxins, viral etiology, reabsorption of uric acid through secretion, dietary phosphorus or calcium imbalances, excess salt, urea adulteration and management stressors such as high temperature and higher ammonia in the barn may contribute to urate accumulation in internal organs and joint surfaces (Sahu *et al*., 2017) [17]. In poultry, there are two main forms of gout, which differ according to where the uric acid accumulates i.e. visceral gout and synovial gout. In both forms, the deposits consist of needle-shaped crystals, called tophi. Visceral gout is a condition in which white crystals of uric acid or urate accumulate in the soft tissues of various body organs. Visceral gout is considered an acute form of the disease, causing massive mortality, characterized by urate deposits on serous surfaces, mainly in the liver, kidneys, pericardium, heart and air sacs. Visceral gout occurs more often in 2-3 day old broilers. Young birds older than 14 weeks are more likely to become affected in farm. Whenever kidney damage occurs, the excretion of uric acid becomes difficult and uric acid begins to accumulate in the blood and then in tissues (Banday *et al*., 2009) [3]. Gout is a chronic condition where uric acid and urates build up, often causing inflammation in ligaments, tendons, and especially in the joints and synovial fluid of the toes. The wing joints experience chronic inflammation due to urate crystal deposits, forming granulomas known as tophi. Monosodium urate (MSU) crystals deposits can also be seen around joints known as periarticular gout. The joints swell, redden, swell and feel warm to the touch. This condition is rarely seen and is usually a chronic condition. The kidneys remain normal but may develop white urate deposits if the bird experiences dehydration. A bird with gout tends to avoid

perching due to pain, preferring flat surfaces instead. Walking causes noticeable distress, often accompanied by vocalization due to discomfort. The bird may appear depressed, dehydrated, and exhibit greenish diarrhea. Additionally, it may appear lethargic with ruffled feathers and a moist vent. Gout usually affects one or two joints at a time, typically forming urate crystals on both the legs and wings simultaneously. The wrist joint of the wing is commonly affected in gout-related injuries (Ali and sultana, 2012) [1].

- **Materials and Methods:** The focus of this study was on 1,500 chickens brought for postmortem examination from various places in the Ganderbal district of Kashmir to the Division of Veterinary Pathology, Faculty of Veterinary Medicine and Animal Science, Kashmir. For histopathological analysis, samples were meticulously preserved in 10% formalin following the excision of affected parts. Tissue sections exhibiting lesions along with adjacent normal tissue were selected for fixation and subsequent histopathological examination.
- **Histopathology:** Initially, all tissue samples sized at 1 cm x 0.5 cm underwent careful trimming and were fixed for a minimum of 24 hours. Subsequently, to remove the fixative, the tissues were submerged in running tap water overnight. Next, the tissues underwent dehydration using ascending concentrations of alcohol: 50%, 70%, 80%, 95%, and absolute alcohol, each solution applied for 1 hour. Following dehydration, the tissues were cleared in chloroform through two changes, with each change lasting one and a half hours. The tissues were then embedded in molten paraffin wax at 56°C, with two changes, one and a half hours each, and paraffin blocks were created using templates. Sections of 5 µm thickness were obtained using a rotary microtome. These sections were carefully spread on a warm water bath at 45°C and transferred onto oil and grease-free glass slides. Gelatine was added to the water bath to ensure better adhesion of the sections to the slides. After air drying, the slides with sections were stored in a cool place until ready for staining (Lillie, 1965). De-Galantha's staining procedure for uric acid began with deparaffinizing the tissue sections by using xylene, followed by bringing them to the water level. Subsequently, the slides were immersed in a 20% silver nitrate solution and exposed to direct sunlight for a period ranging from 2 to 4 hours. To develop the stain, a freshly prepared solution containing 3% gelatin in 10 ml of hot water, 3 ml of 20% silver nitrate solution and 2 ml of 2% hydroquinone solution was poured over the sections until urates appeared black and connective tissues took on a yellow hue. Afterward, the slides were washed in hot water at 58°C. Following this step, dehydration, clearing and mounting in DPX were carried out. The urate crystals were visibly stained black, contrasting with the yellow coloration of the connective tissues.

Results and Discussion: In this investigation, broiler chickens affected with naturally occurring colibacillosis in North Kashmir exhibited characteristic clinical signs and pathological changes. The observations have been presented under the following subheadings.

(A) Clinical Symptoms: The clinical signs observed in affected chicks may include dullness, dehydration, ruffled feathers, segregatory behavior, restlessness, a moist vent with whitish pasty droppings, difficulty in movement and standing, painful joints with shifting of weight between feet and a shuffling gait (Ali and Sultana, 2012) [1]. The affected birds may be unable to perch, spending most of their time on the cage floor, and if wings are involved, they may lose the ability to fly. There are often reddened, swollen feet progressing to blisters and sores, alongside joint pain and immobility caused by urate deposits (Patel *et al*., 2014) [20]. The postmortem findings reveal emaciated, dehydrated carcasses with moderate swelling of hock and phalangeal joints. The other observed signs may include decreased appetite, lethargy, general weakness, weight loss, feather plucking, dull plumage, self-trauma, abnormal droppings with chalky urates and changes in temperament (Jana, 2008) [10].

(B) Pathomorphology: Postmortem findings revealed extensive lesions in affected chicks. Grossly, dry, platery patches of white chalky urate deposits were evident on the serosal surfaces of the pericardium, air sacs, peritoneum, liver, kidneys and ureters. The congestion of internal organs was likely exacerbated by the emaciation and dehydration of the birds (Satalkar, 2007) [19]. Nephropathy was consistently observed, characterized by unilateral to bilateral enlargement protruding from bony depressions, alongside moderate to severe kidney congestion. The kidneys appeared frosted due to accumulated urate crystals, accompanied by pinpoint hemorrhages and chalky deposits on their serosal surfaces. Ureters on both sides exhibited distension with retained semi-fluid to semi-solid white urates, giving them a cord-like appearance and often enlarged due to urolith blockages, with varying degrees of associated kidney degeneration (Lakkawar *et al*., 2018) [12]. Microscopically, marked congestion and hemorrhages involved glomeruli, cortical and medullary tubules, collecting ducts, and medullary tracts, with focal to diffuse degenerative changes observed in tubular epithelium, such as vacuolar degeneration and necrosis (Akter and Sarkar, 2015) [2]. Uric acid crystal aggregates characterized by needle-shaped crystals surrounded by inflammatory cells were noted in kidney parenchyma (Chandra and Balwant, 1998) [6]. Additionally, hyaline casts were found in some tubular lumens, with glomerular changes including atrophy, distortion, and segmentation (Feizi, *et al*., 2011) [7]. The liver exhibited enlargement, friability and white chalky urate deposition on its capsule surface. Liver sections showed severe sinusoidal congestion, parenchymal hemorrhages, fatty changes and areas of necrotic hepatocytes (Ramzan *et al*., 2015) [16]. Liver parenchyma contained radiating uric acid crystals mixed with necrotic debris and surrounded by inflammatory cells (Sandhyarani *et al*., 2019) [18]. Subcapsular hepatocytes showed flattening with elongated nuclei, intense cytoplasmic basophilia, and fibrous connective tissue proliferation in Glisson's capsule (Mir *et al*., 2005) [14]. In the heart, the entire pericardium displayed varying degrees of urate deposits on its serosal surface, indicating uric acid pericarditis (Yewale, 2010) [21]. The pericardium was firmly adhered to the heart. Myocardial lesions included urate deposition, destruction of myocardial cells and infiltration of inflammatory cells. Heart lesions further exhibited myocardial congestion due to severe blood

vessel engorgement and focal to diffuse hemorrhages between muscle fibers (Mudasir *et al*., 2017) [13]. The lungs showed urate deposition in parenchyma, air capillaries, and parabronchi. The edematous fluid was present in air capillaries. The spleen exhibited splenitis and subcapsular hemorrhages, with urate deposits observed in both subcapsular regions and parenchyma (Harr *et al*., 2002) [9]. The dry, platery patches of white chalky urate deposits were also noted on breast muscles, neck and on serosal surfaces of pericardium, peritoneum, mesentery, proventriculus, gizzard, testes, ovaries, and over the abdomen and chest wall. The microscopic examination of the proventriculus revealed edema, hemorrhage and heterophilic infiltration in the serosa. The bursa of Fabricius showed mild depletion of lymphocytes in follicles (Patil *et al*., 2015). These uric acid crystals were observed in black color with De Galantha's stain (Mohan *et al*., 2012) [15]. In the articular form, white chalky nodules known as tophi were found in tissues, occurring in subcutaneous tissue outside joints, sometimes causing ulcers without severe pain (Hong, 2020) [8]. On opening joints, white semi-fluid urate deposits were present within joints. The urate deposits were also observed in almost all other joints, including shoulder, elbow, carpus and phalanges in wings and hip, knee and toe joints in limbs. Rarely, some urate deposits were observed in cervical articulations. Arthritis with segmental necrosis of periarticular muscles and intermuscular edema were also noted (Behtari *et al*., 2015) [4].

GROSS PATHOLOGY

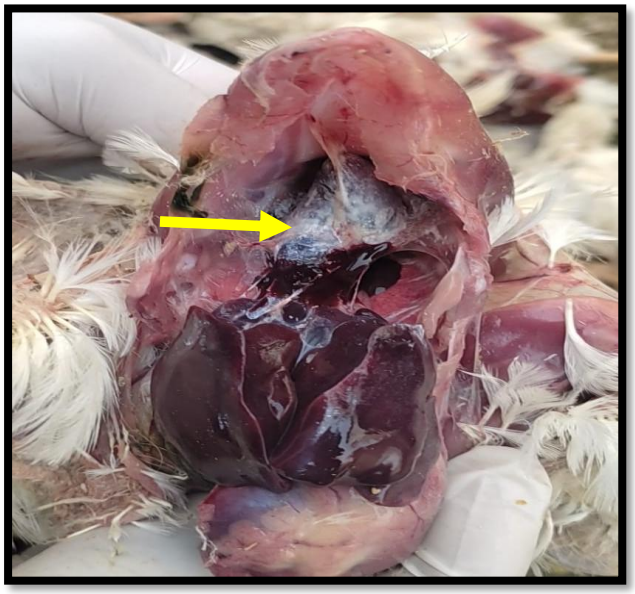

Fig A: Heart revealing deposition of white chalky material on the pericardial surface

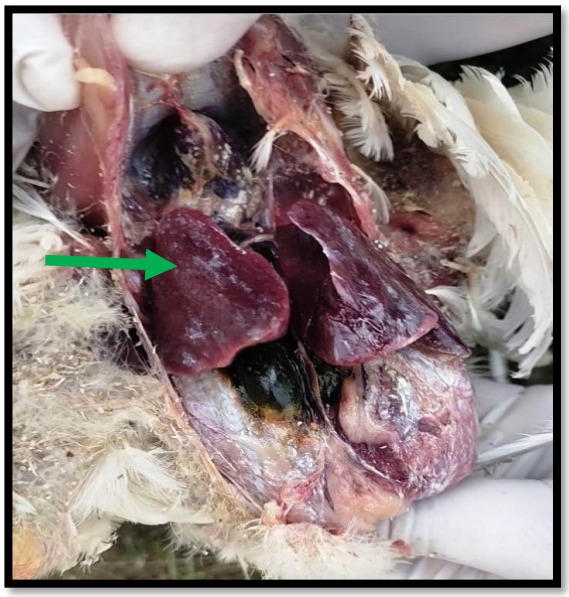

Fig B: Liver reveals presence of white, chalky material scattered diffusely across the surface of liver giving a distinct frost appearance

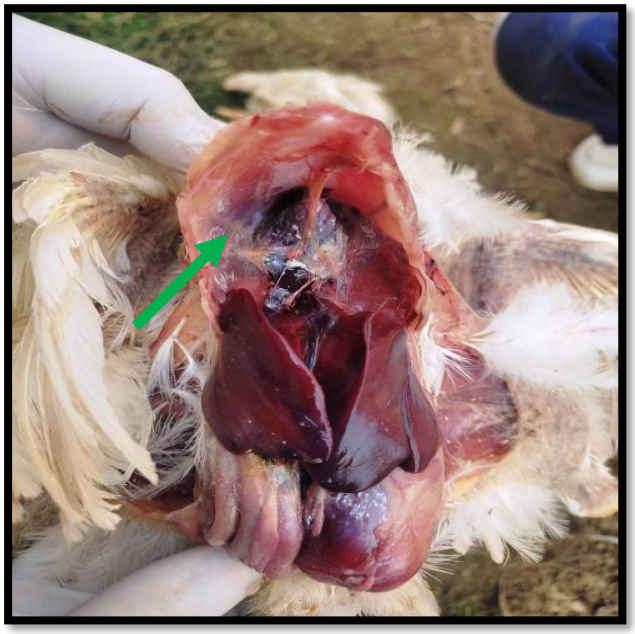

Fig C: Air sacs may display white chalky deposits on their surfaces, resulting in a cloudy appearance

Fig D: Kidneys appeared swollen, congested and enlarged in appearance

Histopathology

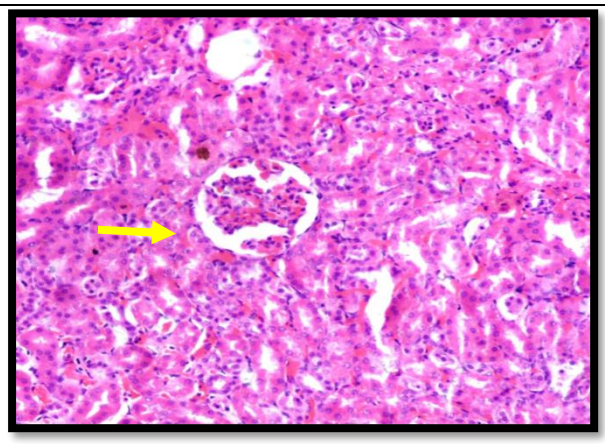

Fig 1: Photomicrograph revealing glomerular atrophy with sloughing off renal tubular epithelium (H&E ,10x)

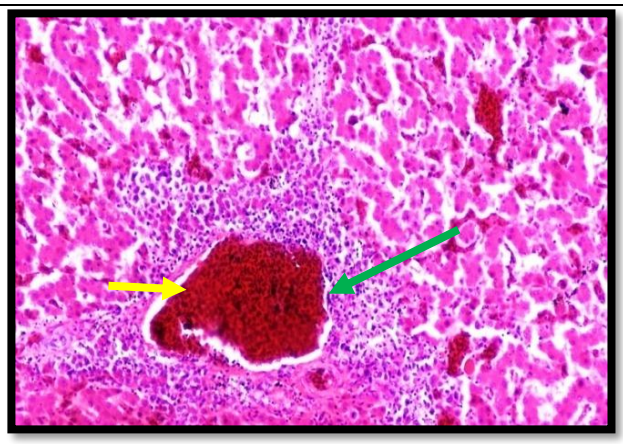

Fig 2: Photomicrograph of liver revealing congestion with perivascular infiltration (H&E, 10x)

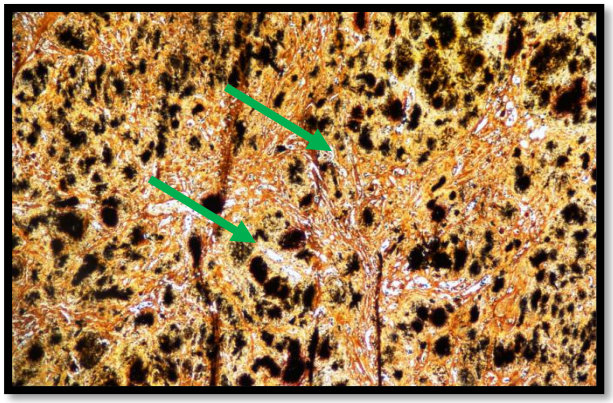

Fig 3: Photomicrograph of Kidney revealing black urate crystals deposits in the tissue section (De Galantha, 10x)

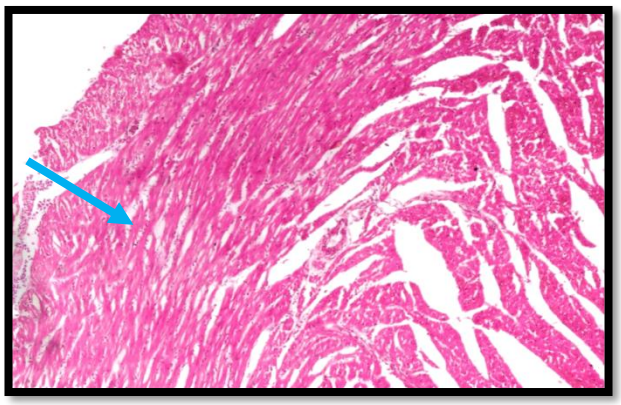

Fig 4: Photomicrograph of heart revealing separation and disruption of muscle fibres (H&E, 10x)

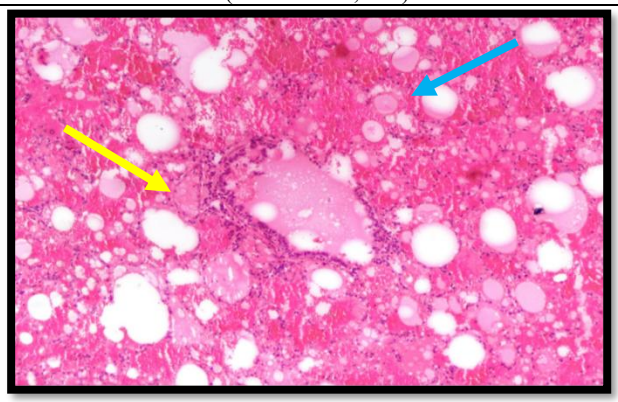

Fig 5: Photomicrograph of lung revealing massive influx of inflammatory cells with edematous changes (H&E, 10x)

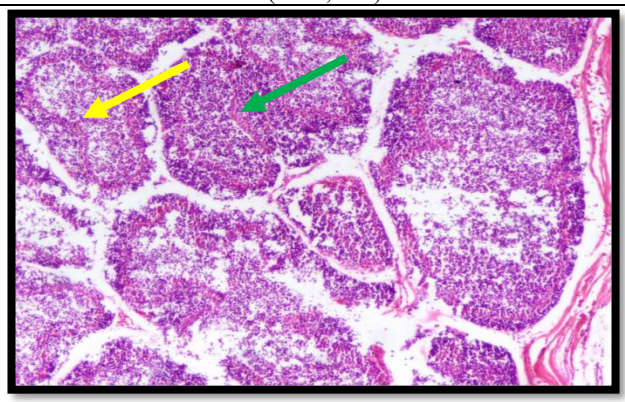

Fig 6: Photomicrograph of bursa reveals the deposition of needle-shaped urate crystals with infiltration of inflammatory cell (H&E, 10x)

Conclusion: The gross and histopathological examinations identify characteristic lesions such as tubular necrosis, interstitial nephritis, denudation of renal tubular epithelium and urate crystal deposition in visceral organs and joints. These findings underscore the importance of early diagnosis and management of gout to prevent mortality and substantial economic losses for poultry farmers.

Conflict of interest: The authors have declared no conflicts of interest

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