Pathological Study of the Experimentally Visceral Gout in Nasiriya City Broilers

Diyaa A. Auda
Department of Basic Science/ Collage of Nursing/ Thi-Qar University

Abstract

Pathological study (crossly and histologically) of experimentally induced of visceral gout syndrome was studied for 35 days after intake of high dietary calcium and protein concentration in Nassirya City Broilers. Forty healthy birds (aged 1 day) were used and divided into two groups. The first group had given normal diet {NCNP, 8.5g ca kg and 175g cp kg}, while the second group had given high concentration of dietary calcium and crude protein {HCHP, 30G CA KG AND 250G cp kg}. All birds at 35 days examined grossly and histopathologically. Typical visceral gout was induced by the HCHP in which caused severe kidney damage (failure of urinary excretion) leading to accumulation of urate crystals in various parts of the body like pericardium, peritoneum, air sacs and liver capsule. Distended ureters often containing white calcium urate calculi (uroliths), besides histological changes in the myocardium, kidney, and ureters.

Key words: Pathology, visceral gout syndrome, kidney damage, urate.

Introduction

A broiler is a type of chicken raised specifically for meat production. Modern commercial broilers, typically known as Cornish crosses or Cornish rocks are specially bred for large scale, efficient meat production and grow much faster than egg or traditional dual purpose breeds they are noted for having very fast growth rates, a high feed conversion ratio, and low levels of activity. Broilers often reach harvest weight of 4- 5 pounds dressed in only eight weeks (1).

They have white feathers and yellowish skin. This cross is also favorable for meat production because it lacks the typical hair which many breeds have that necessitates singeing after plucking. Both male and female broilers are slaughtered for their meat (2). Broiler are hatched from eggs laid by breeding stock (broiler breeders).Broiler breeders are usually housed on deep litter (soft wood shavings, chopped straw, etc) (3). Each broiler breeder produces up to 140 chicks. While broiler breeders are growing to adulthood their food is severely restricted, they therefore suffer from severe hunger. The restriction of food is carried out to prevent them from growing as fast as the meat broilers, breeder broilers are required to survive to adulthood in order to produce chicks(4). Of the many health concerns plaguing birds, one that seems to appear often enough to cause some concern is gout (5). Gout in birds can be in two forms, visceral gout and articular gout. Visceral gout is the most common form in poultry. Where in the plasma uric acid levels are high and urates are deposited in the visceral organs (6).

Gout is a metabolic disorder that results in hyperuricemia and the deposition of posively birefringent monosodium urate crystals in various parts of the body. Identification of the causative agent in field cases is difficult since one or more factors can contribute to

the infection, namely; nutrition problems(vitamin A deficiency, high vitamin D, excess levels of dietary calcium combinded with low dietary phosphorus in non- laying chicken rations, treatment with sodium bicarbonate and excessive protein levels) ,management factor (dehydration due to water deprivation), infectious agents (Infectious bronchitis virus and Gambaro), and toxins (mycotoxins, heavy metal poisoning and the inappropriate use of antibiotics, anticoccidials, chemicals ,pesticides, etc….) (7,8,9).

Materials and Methods

Forty healthy birds aged one day divided in to two groups. The first group had given normal diet (NCNP, 8.5 g ca kg and 175 g cp kg), while the second group had given high concentration of dietary calcium (Ca) and crude protein (cp0(HCHP,30g Ca kg and 250g cp kg) for 35 days. After that all birds were examined grossly and histopathologically. Gross tissue changes were observed and recorded carefully by observation in naked eye. Besides, samples were collected in 10% buffered neutral formalin for histopathology.

Histopathology

The tissue samples were trimmed properly and fixed for 24 hr. To remove the fixative, the tissues were kept in running tap water for overnight. The tissues were dehydrated in ascending grades of alcohol. The tissues were cleared by two changes in chloroform the tissues were embedded with molten paraffin wax. Paraffin blocks containing tissue pieces were made using templates. The tissues were sectioned with a rotary microtome at 5 um thickness. Then the sections were allowed to spread on warm water bath (45c) and taken on oil and grease free glass slide. A small amount of gelatin was added to the water bath for better adhesion of the sections to the slide .The slides containing sections were air dried and kept in cool place until staining. The sectioned tissues were deparaffinized in three changes of xyline. Then the tissues were rehydrated through descending grades of alcohol. The sections were stained with Harris hematoxylin and eosin. Finally the sections were mounted with cover slip using DPX and dried (10). The tissues were examined under microscope and photomicrograph was taken using compound microscope and photomicrographic camera (Olympus PM-C 35 MODEL).

The Results and Discussion

A. Macroscopical findings

The most prominent change was observed on the serosal surface (liver,heart,kidneys and ureters). The pericardium, liver and air sacs appeared to be dusted with a pale yellow powder (Figure 1). Both kidneys were pale and slightly enlarged, with numerous white nodules on the external and sectioned surfaces (Figure 2). The ureters were often dilated in the whole length. They contained a small or large quantity of whitish fluid or muddy substance (Figure 3).

B. Microscopical findings

Mycocardium; multiple small, focal lesions were observed throughout the mycocardium. The lesions consisted of a central zone of radiating sheaves of eosinophilic crystal like material (Figure 4). This zone was surrounded by mononuclear and

multinuclear macrophages, with varying numbers of lymphocytes and heterophils on the periphery. Some necrosis was sociated with these lesions.

Kidney (Figure 5). The kidney lesions were similar to the cardiac lesions and were randomly distributed throughout the cortex and medulla .Some glomeruli were necrotic and there was focal dilatation of tubules; their lumina contained amorphous eosinophilic debris. Occasional infiltrates of lymphocytes and hetrophils were observed in the interstitial tissues at adistance from the granulomas.

Ureter (Figure 6).In both ureters, the lumen was dilated in almost the whole length and frequently contained fluid substances stained pooly or somewhat deeply with eosin. The epithelialcells of the uretres were hyperlastic to various degrees in of the ureters, an increase of lymphocytic cells was often observed, aproliferation of histocytic or fibrocytic cells was occasionally recognized.

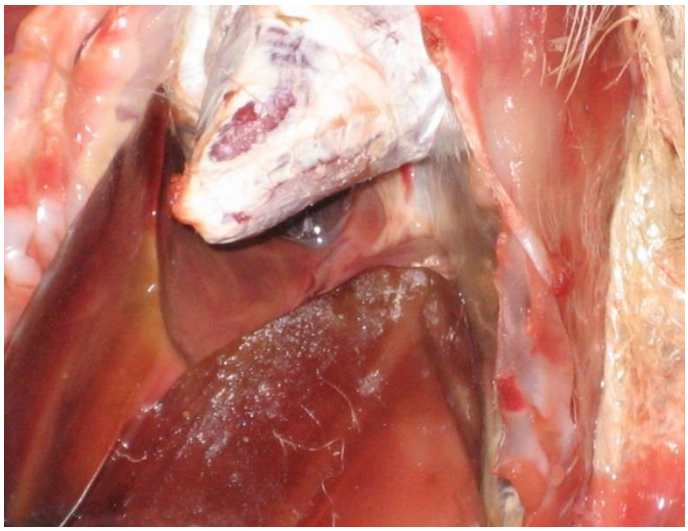

Figure (1): The heart of dead bird showing accumalation of dusted with a pale yellow powder

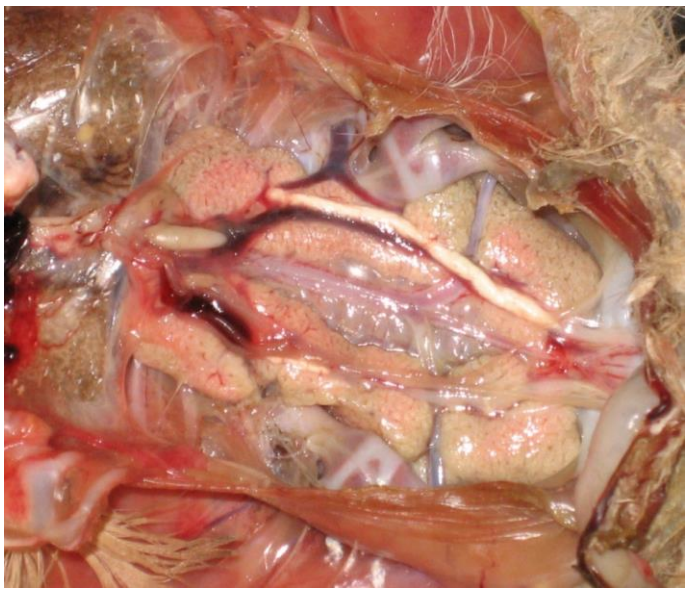

Figure (2): The kidney of dead bird showing numerous white nodules on the external and sectioned surfaces

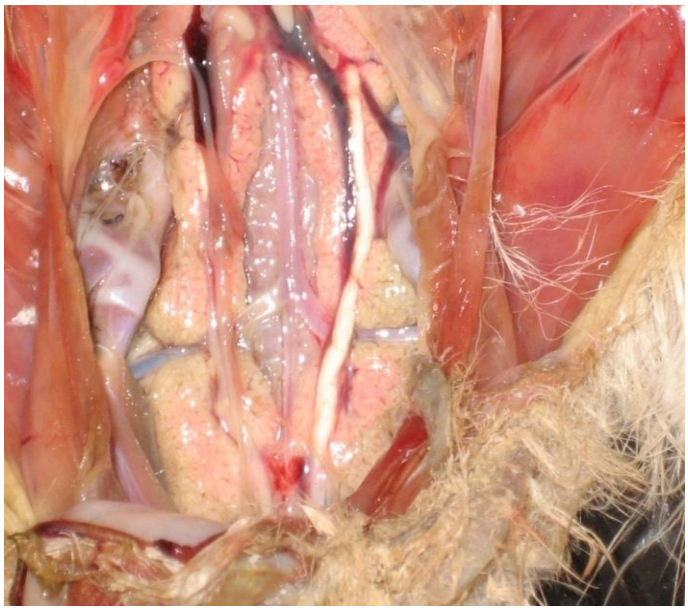

Figure (3): The ureter of dead bird showing dilated in the whole length and contained a small or large guantity of whitish fluid or muddy Substance

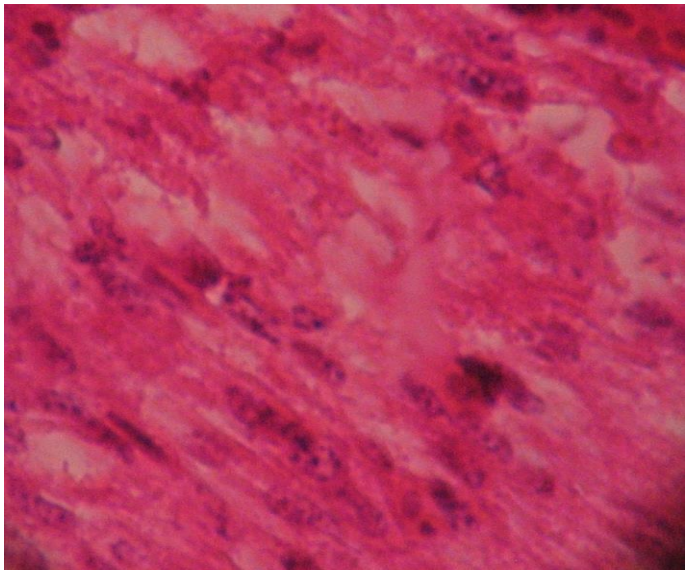

Figure (4): Heart section of dead bird showing the lesions consisted of a certral zone of radiating sheaves of eosinophilic crystal. (H&E 400).

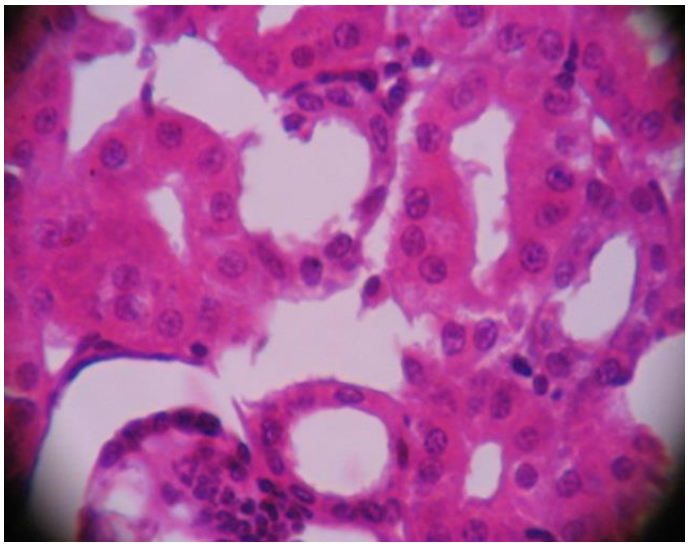

Figure (5): Kidney section of dead bird showing some glomeruli were necrotic and there was focal diltation of tubules.(H&E 400).

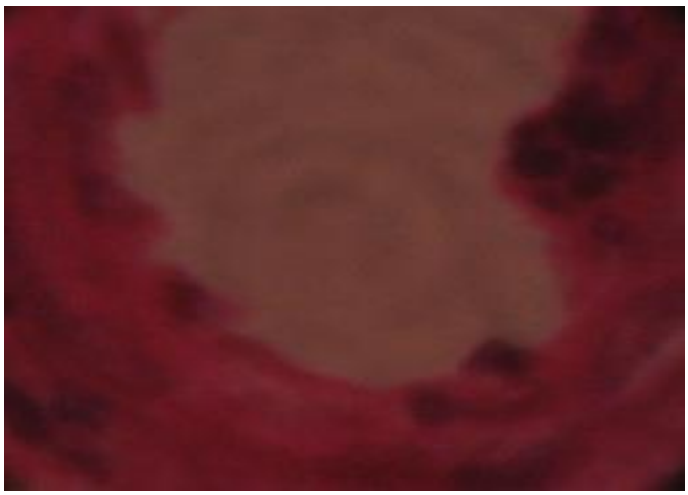

Figure (6): Ureter section of dead bird showing the lumen was dilated in almost the length and frequently contained fliud substances stained pooly. (H&E400).

Birds are uricotelic, the bulk of nitrogenous waste being excreted by the kidney as uric acid. Avian gout is usually associated with hyperuricemia (11). Stated that factors causing hyperuricemia can be placed in to two general categories: 1-Impaired blood clearance of uric acid as a result of renal insufficiency (secondary hyperuricemia) 2-Formation of increased amounts of uric acid (Primary hyperuricemia) (11). Specific condatios known and suggested to cause visceral gout in birds are numerous. The renal lesions associated with vitamin deficiency were reported by Coles (12) and others to cause hyperuricemia and gout. Pyelonephritis (6) and renal neoplasia (11) are associated with gout. Many investigators, including Bechade (13) and Coles (12), blame excessive protein in diet. Siller (6) suggests that diet incorrect in amino acid balance may result in hyperuricemia (13,12) mention,among other factors,decreased solubility of blood urates. Nephrotoxic substances such as potassium dichromate and mercuric chloride are reported to result visceral deposition of urates. In this broilers the main renal lesions seen microscopically

were the tophaceous urate deposits. The remaining kidney tissue appeared to be functional. The concretions found in the cloaca also indicate that renal clearance of uris acid had been greater than normal for some time. Thus the general factor responsible for the disease in this bird probably was not renal insufficiency. Pathologic evidence suggests an overproduction of uric acid, or a decrease in the solubility of normal blood levels of uric acid. The pathological changes observed in the present study various organs are similar to the already existing reports (13,14,15). In this study the histopathological changes in myocardium section are similar to the already reports (16,17,18), also the histopathological changes in kidney and ureters are similar to the already existing reports(19,20).

References

1. Damerow, G.1995.A Guide to Raising Chickens. Story Books. ISBN 0882668978
2. WHICH BIRD SHALL IRAISH ? Genetic Options for pastured poultry producers: Meat – type Chickens and Turkeys By Skip Polson and Anne Fanatico December 2002 .APPPA.
3. Department for Environment Food and Rural Affairs www.defra.Gov.uk /Compassion in World farming Trust. 2005. The Welfare of Broiler Chickens in the EU .http:/www. ciwf. uk./puplications/reports/Welfare_ of_ Broiler _Chickens _in_the_EU_2005.pdf
4. Comparative Avian Nutrition; Kirk Klasing,CAB International ,New Youk, 1998
5. Sillers,W.G.(1959).Avian nephritis and visceral gout. *Laboratory Investigation* 8,1319-1346.
6. Quesenberry,K; Orosz,S;(1997) Dorrestein, GM. Musculoskeletal system. In Altman, RB; Clubb, SL;Dorrestein,GM;Quesenberry,K(ed).Avian Medicine and Surgery. W.b. Saunders Co.Philadephia, PA;
7. Roudybush ,TE.Psittacine Nutrition. In Jenkins, JR. (ED) The Veterinary Clinics of North America: Exotic Animal Practice. W.B Saunders Co.Philadephia, PA;January 1999.
8. Rupley. AE, Manual of Avian Practice. W.B. Saunders Co. Philadephia ;PA;1997.
9. Luna LG (1968).*Manual of Histologic Staining Methods of the Armed Forces Institute of Pathology*. 3 edn., McGraw Hill Book Co . New York.
10. Schlumberger,H.G.(1969)synovial gout in the parakeet.*Laboratory Investigation* 8,1304-1310.
12. Coles, B.H.(1985):Avian medicine and surgery Blackwell scientific Publication:O-Xford,London Experienta.
13. Bauck L,LaBonde J(1997);Toxic diseases. In Altman RB,Clubb SL,Dorrestein GM,Quesenberry K(eds):Avian Medicine and surgery.Philadelphia,WB Saunders Co, pp 604-613.
14. Samour J:Toxicology.In Samour J(ed):Avian Medicine.London,Mosby,2000,pp 180-193.
15. Dumonceaux G,Harrison GJ:Toxicology.In Ritchie BW,Harrison GJ,Harrison LR (eds): Avian Medicine: Principles and Application. Delray Beach,FL,HBD . Intl,2001, pp 1036-1041.
16. Shama GL(1997).Miscellaneous Pathological Conditions diseases.In:Lokeshwar RR, editor.*Handbook or Animal Husbandry*.2nd ed,New Delhl:ICAR Publication; .pp.450-1.
17. Uma CA,Vijayasarathi SK,Nalini TS,Satyanarayana ML,RAO S. Pathology of g- Out in poultry.*Indian J Vet Pathol*. 1999 ;23;94-5.
18. Arun PR, Azeez PA. Vulture population decline, diclofenac and avian gout. *Curr Sci*.2004;87:565-68.

19. Patel AK, Ghodasara DJ,Dave CJ, Jani PB, Joshi PB,Prajapati KS. Experimental Studies on etiopathology of visceral gout in broiler chicks. *Indian J Vet Pathol*. 2007;31:24-8.
20. Rao TB, Das JH, Sharma DR. An outbreak of gout in East Godavari District An- Dhra Pradesh.*Poultry Adviser*. 1993;26:43-5.

دراسة التغيرات المرضية العيانية والنسيجية في فروج اللحم المصاب تجريبيا بداء النقرص الأحشائي في مدينة الناصرية

ضياء عبد عوده
قسم العلوم الاساسية/ كلية التمريض/ جامعة ذي قار

الخلاصة

تمت دراسة التغيرات العيانية والنسيجية للأحشاء الداخلية لمرض داء النقرص في فروج اللحم تجريبيا في حقول مدينة الناصرية والناتجة عن زيادة تناول بمستوى الكالسيوم والبروتين في العليقة ولمدة 35 يوم . تم استخدام أربعين طيرا بعمر يوم واحد خالية من الإمراض وتم تقسيمها إلى مجموعتين تضم كل مجموعة عشرون طيرا. أعطيت المجموعة الأولى عليقة طبيعية والثانية عليقة عالية الكالسيوم والبروتين ولمدة 35 يوما اخضعت جميع الطيوربعدها للفحص العياني والمجهري حيث لوحظت وجود ترسب مادة طباشيرية في الأغشية المصلية وتجمع مادة اليوريت في الحالبين كذلك لوحظ تغيرات نسيجية في نسيج الكلية والقلب والحالبين وهي علامات تدل على الإصابة بداء النقرص الاحشائي في الطيور بسبب زيادة نسبة الكالسيوم والبروتين في العليقة عن الحد الطبيعي.