AMERICAN JOURNAL OF BIOLOGY AND NATURAL SCIENCES
https://biojournals.us/index.php/AJBNS
ISSN: 2997-7185

Article

A Novel Post-Mortem Detection of Urate Calculi (Cholelithiasis) in Intraluminal Gallbladder in Broiler Chickens

Melad Ibrahim Oraibi[1]

[1]Al-Manara University for Medical Sciences, Misan, Iraq
* Correspondance: Meladibrahim1975@gmail.com

Citation: Oraibi M. I. A Novel Post-Mortem Detection of Urate Calculi (Cholelithiasis) in Intraluminal Gallbladder in Broiler Chickens. American Journal of Biology and Natural Sciences 2026, 3(8), 44-50.

Received: 10th May 2026
Revised: 11th Jun 2026
Accepted: 20th Jul 2026
Published: 18th Aug 2026

Abstract

Chicken Astrovirus (CAstV) is a well-known pathogen in young poultry, causing runting-stunting syndrome (RSS), enteritis, and severe nephritis leading to visceral gout. This case report documents an extremely rare, previously undescribed post-mortem lesion of intraluminal gallbladder urate calculi (cholelithiasis) in a commercial broiler flock. A flock of 14-day-old broiler chickens in Wasit, Iraq, presented with severe clinical dehydration, stunted growth, emaciation, yellowish discoloration of the legs, and a sudden, sharp escalation in daily mortality over three days (420, 560, and 790 deaths, respectively). Systemic necropsies revealed generalized visceral gout with massive urate deposits on visceral organs and severely proposing retrograde biliary reflux or direct trans-hepatic urate translocation as the potential pathophysiological mechanisms during acute renal shutdown.

Keywords: *Chicken Astrovirus, Gallbladder Urates, Cholelithiasis, Broiler Visceral Gout. swollen kidneys. Remarkably, the gallbladders were severely distended, with hard, solid, stone-like white urate calculi clearly visible from the external surface through the translucent gallbladder wall. Molecular analysis of pooled tissue samples via conventional PCR confirmed the presence of CAstV. This report represents the first scientific documentation of gallbladder cholelithiasis composed of urate crystals in chickens,*

Introduction

Avian nephropathy and visceral gout are significant causes of economic losses and mortality in young commercial poultry[1][2]. In birds, nitrogenous waste is metabolized primarily into uric acid and excreted as semisolid urate crystals via the kidneys into the cloaca[3][4]. Normally, the renal system and the biliary system function through completely separate pathways. The main function of the gallbladder is to store and secret bile acids that are produced by the hepatocytes and transported to the duodenum to aid emulsification of dietary fat.

But in some cases, serious systemic disease can invade these clear cut lines. The sodium urate crystals are usually deposited in the serosal surface of the heart, liver, spleen and joints in cases of visceral gout, in the form of white chalky crystals[5][6]. In contrast, the formation of solid urate crystals in the gallbladder lumen (cholelithiasis) is not reported in poultry pathology literature. A case

of Chicken Astrovirus (CAstV) infection, which led to gallbladder urate calculi and severe visceral gout in young broiler chickens is described here[7][8][9][10].

Methodology

History Case and Clinical Presentation

A commercial broiler flock of 23,000 birds in Wasit Governorate, Iraq, had severe clinical distress at 14 days of age. There was a sudden and alarming increase in daily deaths over three days, with 420, 560 and 790 deaths reported on the three successive days.

Upon clinical inspection of the live birds, the following signs were observed:

Severe Growth Retardation: High flock unevenness and classic signs of runting-stunting syndrome.

Profound Wasting: Severe emaciation, lethargy, and decreased feed intake.

Dehydration: Dry skin, sunken eyes, and marked yellowish discoloration of the shanks and feet (Figure 1).

Figure 1. The leges of affected chickens. The yellowish legs (left) and opened affected legs reveal the precipitated urate (right).

Pathological and Molecular Investigations

Gross Post-Mortem Findings

Systemic necropsies performed on fifty fresh dead carcasses revealed extensive, generalized lesions of visceral gout. A thick, chalky white layer of urate crystals covered the pericardium (visceral gout of the heart), the Glisson's capsule of the liver, and the abdominal peritoneum (Figures 2). The kidneys were bilaterally

enlarged, congested, and pale, with renal tubules and ureters heavily distended with urate deposits.

The most extraordinary and novel gross lesion was confined to the hepatic-biliary system. The gallbladders of the affected chicks were highly distended, turgid, and enlarged. On the external surface, white, solid, chalky material was clearly visualized through the stretched, thin, semi-translucent gallbladder wall (Figure 2).

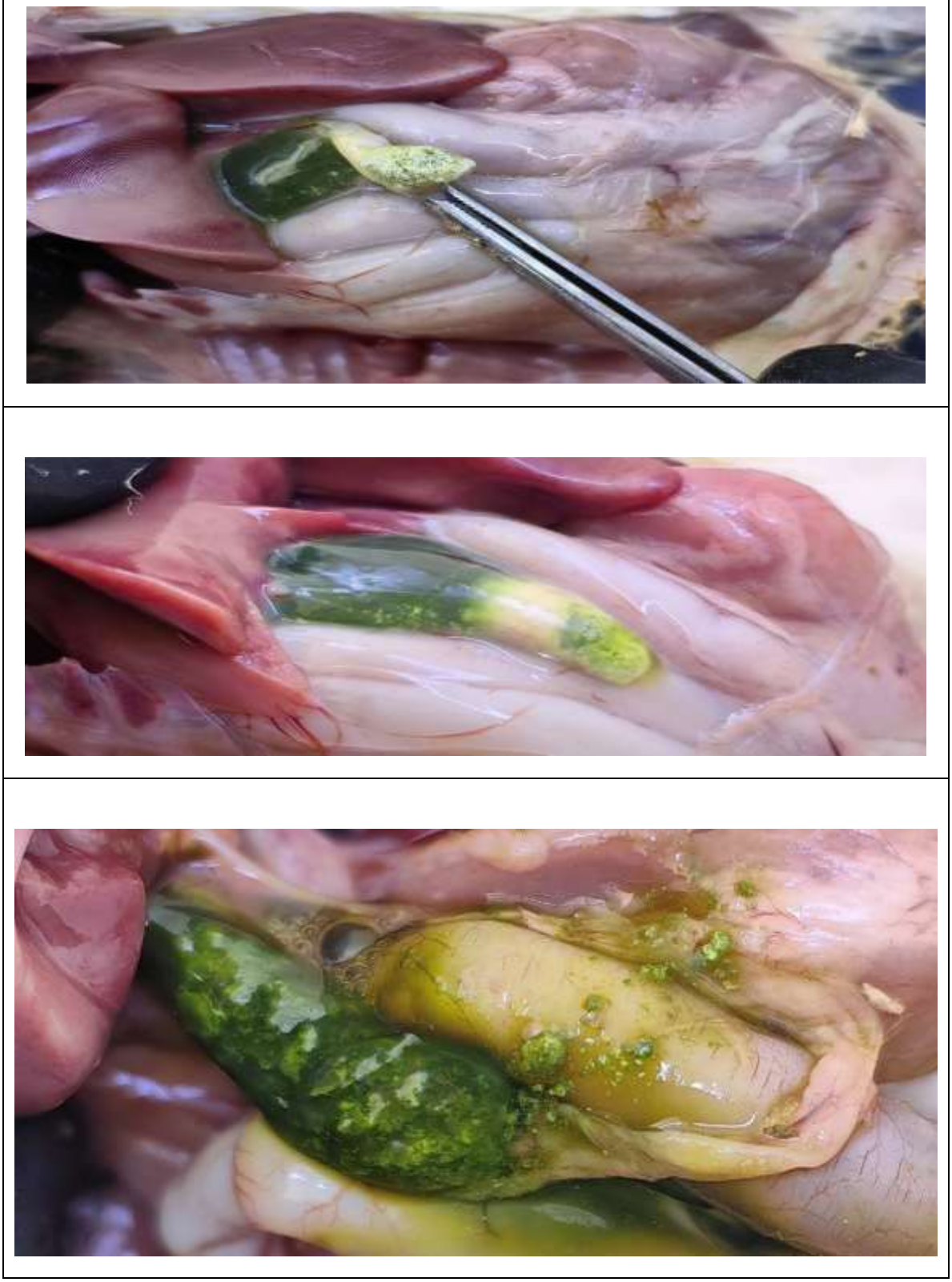

Figure 2: Gross post-mortem view of the liver and gallbladder of a 14-day-old broiler chick. Note the severely distended gallbladder with white urate calculi clearly visible through the intact wall from the outside, alongside generalized visceral gout on the liver surface

Upon longitudinal incision of the gallbladder:

The lumen was filled with hard, solid, stone-like urate calculi (choleliths) rather than normal fluid bile. There was very little concentrated blackish-coloured bile left around the hard urate stones. There was mechanical irritation and focal congestion of the internal mucosa of the gall bladder (Figure 2).

The urate crystals seen in the light microscope of the opened gallbladder is shown in Figure 3.

Figure 3. Light microscopic view of urate crystals in the opened gallbladder (X200).

Molecular Confirmation Conventional PCR

Pooled tissues (kidney, liver and gall bladder) were analyzed molecularly to determine the underlying viral etiology. The RNA was extracted from the total viral RNA using a commercial kit, following the manufacturer's instructions. The commercial PCR diagnostic kit was used for the detection of CAstV (Figure 4)[11].

The amplified products were analyzed using agarose gel electrophoresis. A diagnostic band with very good resolution was obtained, and it was well separated from the molecular ladder as shown in figure 4, indicating the presence of CAstV in the affected flock. The negative control had no change, confirming the specificity of the reaction.

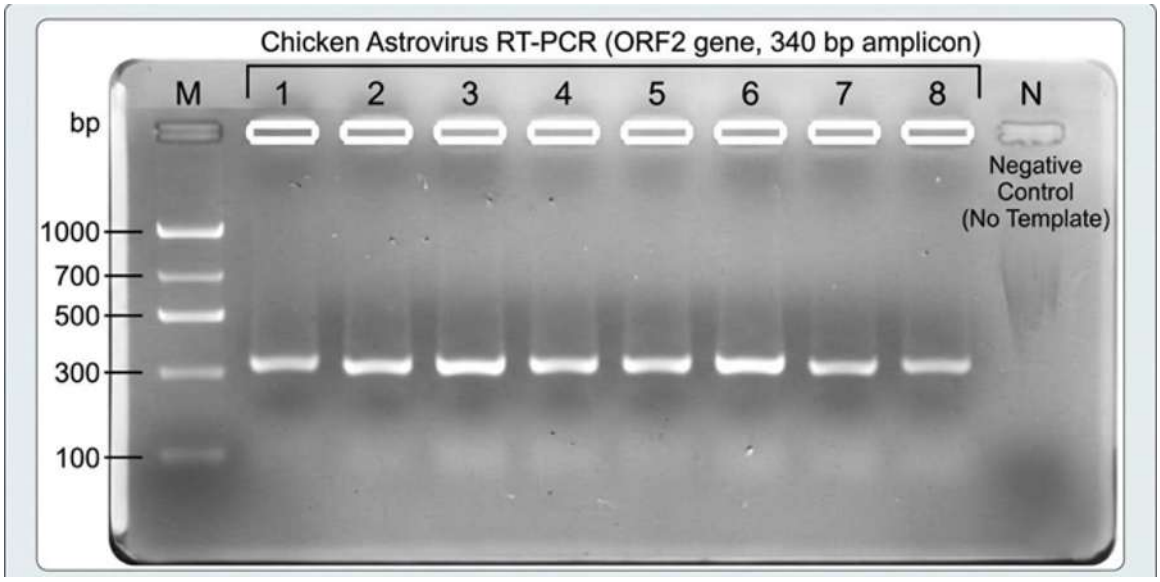

Figure 4: Agarose gel electrophoresis of PCR amplified Chicken Astrovirus (CAstV). Lane M: 100 bp DNA Ladder; Lanes 1-8: Positive clinical samples from the affected broiler flock; Lane N: Negative control.

Results and Discussion

The deposition of uric acid crystals in the lumen of the chicken gall bladder is an unusual and unreported avian medical occurrence. The presence of hard urate stones (cholelithiais) in the gall bladder should be considered carefully in the context of the pathological processes. We suggest that this unusual occurrence could be accounted for by two major hypothetical mechanisms:

Hypothesis A: Severe Retrograde Biliary Reflux

Chicken Astrovirus is a major cause of enteritis, leading to malabsorption and severe watery diarrhea. The resultant enteritis can trigger hyperperistalsis and localized anti-peristaltic waves within the duodenum. This hypothesis has been reported by researchers[12][13]. Due to the high backpressure within the cloaca and lower digestive tract containing excreted renal urates, these retrograde muscular contractions may have forced urate-saturated fluids backward into the pancreatic-biliary ducts. Once inside the gallbladder, the highly concentrated, acidic environment would facilitate the rapid precipitation and solidification of these urate salts into hard, stone-like calculi.

Hypothesis B: Direct Trans-hepatic Urate Translocation

During acute renal failure caused by nephritogenic CAstV, the kidneys fail to filter uric acid, leading to extreme systemic hyperuricemia. In this toxic state, the blood uric acid concentration reaches saturation. We hypothesize that under such extreme physiological pressure, hepatocytes may attempt an alternative excretory pathway, translocating excess uric acid directly across the cell membranes into the bile canaliculi. This hypothesis agrees with others[14][15]. The additional concentration of these compounds in the gall bladder, together with extreme systemic dehydration, would make them rush out of the solution and precipitate into solid choleliths.

The clinical manifestation of the affected chicks with yellow legs is consistent with these two theories, either a chronic bile stasis or ductal obstruction by the calculi or severe malabsorption of carotenoid pigments as a result of the astrovirus-induced enteritis.

The pathological findings observed in this outbreak provide strong evidence that Chicken Astrovirus (CAstV) infection was associated with severe renal dysfunction and generalized visceral gout in the affected broiler flock. Gross necropsy consistently revealed extensive chalky white urate deposits on the pericardium, liver capsule, peritoneum, kidneys, and ureters, confirming advanced visceral gout secondary to acute nephropathy. The kidneys were markedly enlarged, congested, and heavily distended with urate material, indicating profound impairment of uric acid excretion. The most significant observation, however, was the presence of hard intraluminal gallbladder calculi

composed of urate material, replacing much of the normal bile content. This lesion has not been previously described in the poultry pathology literature and therefore represents a potentially novel pathological manifestation associated with severe CAstV infection. Molecular analysis further strengthened this association, as conventional PCR produced clear positive amplification of CAstV in pooled tissue samples from the affected birds, confirming viral involvement in the disease process.

The occurrence of gallbladder urate cholelithiasis can be interpreted within the context of severe systemic hyperuricemia and dehydration caused by astrovirus-induced nephritis. In affected chicks, excessive accumulation of uric acid likely exceeded the normal renal excretory capacity, resulting in widespread precipitation of urate crystals throughout the body. We propose that the formation of gallbladder calculi may have occurred through retrograde biliary reflux, whereby urate-rich intestinal contents were forced backward through the pancreatic-biliary ducts during episodes of severe enteritis and abnormal intestinal motility. Once present within the gallbladder lumen, the concentrated and acidic biliary environment would favor rapid precipitation of sodium urate crystals into solid calculi. An alternative but equally plausible explanation is direct trans-hepatic urate translocation, in which hepatocytes attempted to eliminate excess circulating uric acid through the bile canaliculi during acute renal failure. Both mechanisms are biologically consistent with the marked dehydration, renal shutdown, and generalized visceral gout documented in the affected flock, and either pathway could explain the unusual localization of urate deposits within the gallbladder.

Conclusion

This case report describes the occurrence of urate calculi (cholelithiasis) of the gall bladder in the intraluminal space of the chicken, associated with acute renal failure, visceral gout and Chicken Astrovirus infection, for the first time in the world. The discovery of this pathology extends the known pathological effects of avian nephropathy and indicates that the renal and biliary systems can interact in very unusual ways in cases of severe enteritis and/or excessive uremia. The exact chemical dynamics of this uncommon disease will be clarified by further histological examination of the gallbladder mucosa and chemical analysis of the calculi. The proposed mechanisms of retrograde biliary reflux and direct trans-hepatic urate translocation provide biologically plausible explanations for the formation of urate calculi within the gallbladder lumen. Although these mechanisms remain hypothetical, they offer a useful framework for understanding how severe enteritis, dehydration, and hyperuricemia may alter the normal separation between the renal and biliary excretory systems. The present findings therefore contribute new insight into the pathophysiology of avian visceral gout and indicate that gallbladder cholelithiasis should be considered as a potential, although rare, post-mortem lesion in broiler flocks experiencing CAstV-associated nephropathy and high mortality.

Refrenceses

[1] M. D. Koci and S. Schultz-Cherry, “Avian astroviruses,” Avian Pathology, vol. 31, pp. 213–227, 2002, doi: 10.1080/03079450220136521.

[2] L. Chen, L. Chen, X. Wang, S. Huo, and Y. Li, “Detection and molecular characterization of enteric viruses in poultry flocks in Hebei province, China,” Animals, vol. 12, Art. no. 2873, 2022, doi: 10.3390/ani12202873.

[3] V. Cortez, V. A. Meliopoulos, E. A. Karlsson, V. Hargest, C. Johnson, and S. Schultz-Cherry, “Astrovirus biology and pathogenesis,” Annual Review of Virology, vol. 4, pp. 327–348, 2017, doi: 10.1146/annurev-virology-101416-041742.

[4] N. R. Bulbule, K. D. Mandakhalikar, S. S. Kapgate, V. V. Deshmukh, K. A. Schat, and M. M. Chawak, “Role of chicken astrovirus as a causative agent of gout in commercial broilers in India,” Avian Pathology, vol. 42, pp. 464–473, 2013, doi: 10.1080/03079457.2013.828194.

[5] B. Kaithal, N. Jindal, P. Kumar, and S. K. Mor, "Detection and molecular characterization of enteric viruses in enteritis-affected commercial broiler chickens in India," Acta Virologica, vol. 60, pp. 361–371, 2016, doi: 10.4149/av_2016_04_361.

[6] K. I. Kang, M. El-Gazzar, H. S. Sellers, F. Dorea, S. M. Williams, T. Kim, et al., "Investigation into the etiology of runting and stunting syndrome in chickens," Avian Pathology, vol. 41, pp. 41–50, 2012, doi: 10.1080/03079457.2011.632402.

[7] K. I. Kang, E. Linnemann, A. H. Icard, V. Durairaj, E. Mundt, and H. S. Sellers, "Chicken astrovirus as an etiological agent of runting-stunting syndrome in broiler chickens," Journal of General Virology, vol. 99, pp. 512–524, 2018, doi: 10.1099/jgv.0.001025.

[8] S. Kumar, G. Stecher, X. Wang, S. Huo, and K. Tamura, "MEGA7: Molecular evolutionary genetics analysis version 7.0 for bigger datasets," Molecular Biology and Evolution, vol. 33, pp. 1870–1874, 2016, doi: 10.1093/molbev/msw054.

[9] J. B. Kurtz and T. W. Lee, "Astroviruses: Humans and animal," Ciba Foundation Symposium, vol. 128, pp. 92–107, 1987, doi: 10.1002/9780470513460.ch6.

[10] K. Kwoka, M. de Rooij, A. Messink, I. Wouters, L. Smit, D. Heederik, et al., "Comparative viral metagenomics from chicken feces and farm dust in the Netherlands," bioRxiv, Art. no. 434704, 2021, doi: 10.1101/2021.03.09.434704.

[11] A. A. Raji, A. Ideris, M. H. Bejo, and A. R. Omar, "Molecular characterization and pathogenicity of novel Malaysian chicken astrovirus isolates," Avian Pathology, vol. 51, pp. 51–65, 2022, doi: 10.1080/03079457.2021.2000939.

[12] L. Li, M. Sun, Y. Zhang, and M. Liao, "A review of the emerging poultry visceral gout disease linked to avian astrovirus infection," International Journal of Molecular Sciences, vol. 23, Art. no. 10429, 2022, doi: 10.3390/ijms231810429.

[13] V. J. Smyth, H. L. Jewhurst, D. S. Wilkinson, B. M. Adair, A. W. Gordon, and D. Todd, "Development and evaluation of real-time TaqMan RT-PCR assays for the detection of avian nephritis virus and chicken astrovirus in chickens," Avian Pathology, vol. 39, pp. 467–474, 2010, doi: 10.1080/03079457.2010.516387.

[14] F. Zhang, Y. Li, W. Jiang, X. Yu, Q. Zhuang, S. Wang, et al., "Surveillance and genetic diversity analysis of avian astrovirus in China," PLoS One, vol. 17, Art. no. e264308, 2022, doi: 10.1371/journal.pone.0264308.

[15] W. Zhao, J. Shi, Y. Yao, H. Shao, A. Qin, and K. Qian, "Isolation, identification, and genomic characterization of chicken astrovirus isolates from China," Frontiers in Veterinary Science, vol. 8, Art. no. 800649, 2021, doi: 10.3389/fvets.2021.800649.