

Visceral Gout (Uricosis) and Urolithiasis Caused by Dehydration in Laying Hen Farm, Necropsy and Histopathology Findings

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Abstract

This case report describes visceral gout in laying hens caused from deprivation of water supply as a consequence of limiting access for drinking water. In two different layer farms, nineteen week old pullets had a sudden and increased mortality rate. Gross pathology revealed typical uricosis lesions including: deposition of uric acid and its salts in kidney, pericardium, liver, muscle atrophy and dehydration. The changes seen microscopically were consistent with end stage renal failure due to chronic renal disease and other organs had lesions consistent with visceral gout. Subsequent to the diagnosis, owners instituted improved water access, and mortalities decreased. This case report emphasizes the importance of early diagnosis of chicken gout, need of training for poultry farmers and field veterinarians for early and proper prevention of visceral gout in laying hens.

Keywords: Visceral gout, Urolithiasis, Dehydration, Pullet

Yumurtacı Tavuk Çiftliğinde Dehidrasona Bağlı Visseral Gut (Ürikozis) ve Ürolitiazis, Nekropsi ve Histopatolojik Bulgular

Özet

Bu vaka sunumunda yumurtacı tavuklarda içme suyu eksikliğine bağlı olarak şekillenen visseral gut olgusu tanımlanmıştır. İki ayrı işletmede ani ve artmış mortalite oranı ile toplam 19 adet piliç bildirildi. Makroskopik bakıda böbreklerde, perkardiyumda ve karaciğerde ürik asit ve tuzlarının yığıntıları, kas atrofisi ve dehidrasyon ile karakterize tipik ürikozis lezyonları gözlemlendi. Mikroskopik bulgular kronik böbrek hastalığına bağlı son safha böbrek yetmezliği bulguları ile uyumlu olup diğer organlardaki lezyonlar visseral gut işaret etmekteydi. Teşhis takiben çiftlik sahiplerinin hayvanların su alımlarını iyileştirmeleri sonucunda ölümler azaldı. Bu vaka takdimi visseral gutun yumurtacı tavuklarda erken teşhis ile çiftlik yöneticilerinin ve veteriner hekimlerin konu hakkında eğitilmelerinin hastalığın erken ve uygun bir şekilde önlenmesi açısından önemini vurgulamaktadır.

Anahtar sözcükler: Visseral gut, Ürolitiazis, Dehidrasyon, Piliç

INTRODUCTION

The commercial laying hen industry is one of best developed branches of agriculture in Kosova, comprising about 1 million laying hens ^[1]. In former time egg production was mostly centralized in state cooperatives, and recently this industry is growing as private business. However, many farmers entering this field lack relevant experience, and so there are numerous health problems surfacing.

Visceral gout occurs secondary to kidney damage which can have numerous etiologic reasons, including: nutritional

and metabolic factors, infectious causes, toxicity and other factors. One prominent cause is water deprivation which leads to concentration of uric acid and other minerals in the blood and later in the kidney. The infectious causes, such as nephrotropic strains of infectious bronchitis virus and renal cryptosporidiosis; and noninfectious factors, such as vitamin A deficiency ^[2,3], treatment with sodium bicarbonate, mycotoxins, such as oosporein ^[4]. Another cause of visceral gout is also if the feeding growing birds layer rations that are high in calcium and protein ^[2,4,5]. Whenever there is kidney damage, excretion of uric acid gets affected and uric acid starts accumulating in the blood and later in tissues.



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Visceral gout and urolithiasis are reported as an important cause of renal failure in pullets and caged laying hens [6]. Lack of access to water is a primary contributing cause and this can happen during transport, blockage of nipples, inadequate number of nipples, overcrowding etc. Severe or persistent dehydration increases resorption of water causing a subsequent reduction in urine flow. As uric acid secretion decreases, urates may precipitate in renal tubules and ureters leading to impaction and potentially renal failure [6,7].

The cause of gout is often difficult to determine. The original kidney damage may occur long before the onset of gout mortality. Visceral gout is rarely diagnosed ante mortem and usually diagnosed only at necropsy and findings are generally sufficient to diagnose gout in poultry [2,7].

The purpose of this report is to describe importance of proper diagnosis of visceral gout caused by dehydration in order to reduce mortality outbreaks, and lessons from this study can be applied to further improvements of disease prevention and control.

CASE HISTORY

Clinical Signs

In 2012 and again in 2014, we were contacted by farmers having increased mortality in their large laying farms. The first farm had 17.000 pullets and the second had 20.000 pullets. The clinical signs were characterized by sudden onset of depression, low feed intake, dehydration, and rise in mortality. In both farms the onset of the mortality in flock of 19 to 28 weeks old is reported. In week 19 mortality rate was about 20 pullets per day with peak of mortality on weeks 20-26 with about 90 pullets per day. From week 26 until the mortality has reduced in week 28 compared to normal mortality rate. In the first farm the total number of dead pullets was 3.000 pullets (out of 17.000 initially), or 17.9%. At the second farm, mortality was 3.500 pullets (from 20.000 pullets initially), or 17.5%. Mortality has been reduced and stopped within short period after proper access to the water has improved for 2 to 4 weeks. In both farms pullets were vaccinated with complete program, including protection against infectious bronchitis virus.

Pathological Findings

The necropsy is done in dead layers (45 layers respectively 22 in first farm and 23 pullets in second farm) aged between 19 to 24 weeks-old.

Macroscopical Findings

The most prominent change was observed on the serosal surface of organs of multiple organs, where there is a diffuse deposition of white chalky material (Fig. 1). Kidneys



Fig 1. Advanced visceral gout with extensive deposits of urates on the pericardium, surface of the liver and peritoneum

Şekil 1. Perikardiyum, karaciğer ve periton yüzeylerinde yoğun urat depozitleri ile karakterize ileri seviyede visceral gut



Fig 2. Urolithiasis involving right ureter and kidney atrophy with compensatory hypertrophy of the left side of the kidney

Şekil 2. Sağ üreterde ürolitiazis ve atrofi ile sol böbrekte kompanse hipertofi

were irregularly shaped and often markedly enlarged, but still with atrophy of selected lobules (Fig. 2). Dead birds were dehydrated and in poor body condition, the breast muscles were dry and atrophic (Fig. 3). The ureters were markedly enlarged with irregular white uroliths (Fig. 4). The large uroliths often completely filled and greatly expanded the affected ureter.

Histopathology findings: The changes seen in the kidneys were consistent with end stage renal failure due to chronic renal disease. In kidney the numerous tubules have degenerate heterophils in the lumen while other tubules have proteinous casts in the lumen. Some tubules were ruptured due to crystalline array formation with multinucleated giant cells (tophi formation). A few tubules were acutely necrotic with intact heterophilic infiltrates. Many



Fig 3. Pullet dehydration and musculature atrophy
Şekil 3. Piliçde dehidrasyon ve kas atrofisi



Fig 4. Picture showing, a- Bilateral urolithiasis and b- Urolith
Şekil 4. a- Bilateral ürolitiazis, b- Ürolit

of the organs showed lesions consistent with visceral gout. In ureters the lumen was dilated and lined by flattened epithelium that was often sloughing, with abundant eosinophilic material in the lumens, occasionally mineralizing.

There were multiple areas of fibrosis in the tubules near the ureters and surrounding the ureters. The proventriculus had a few submucosal glands with focal areas of necrosis and hemorrhage. In sections of spleen, there were multiple tophi throughout the parenchyma. The liver had sections with individual hepatocyte dropout/necrosis. Fibrin thrombi were evident in sinusoids multifocally. The heart had crystals on the epicardial surface.

DISCUSSION

Dehydration due to water deprivation is a common cause of visceral urate deposition in domestic poultry [6]. Dehydration in cage farming is generally caused by inability to reach the water or failure to provide adequate amount of water. In the cases presented here, mortality decreased after it was ensured that pullets had sufficient water.

In presented case report the onset of mortality in both farms was on week 19, and continued for 5 to 6 weeks with total mortality of 17.9% of pullets (3.050 from 17.000) in first farm and 17.5% from total flock (3.500 from 20.000) in second farm.

The mortality rate was slightly higher comparing with figures with case of uricosis described by Blaxland et al. [8], where mortality was 10 to 15% of the birds with highest mortality on 19 to 24 weeks old. The mortality rate it is also reported from total flock ranging between 2% and 50% in severely affected flocks [9].

The pathogenesis of visceral gout is not completely understood but generally is considered to be the acute form of disease causing huge mortality characterized by the urate deposits on serosal surfaces, most often in the liver, kidney, pericardium, heart and air sacs reported in different authors [2,4]. Similar necropsy and histopathology findings are presents in this case report. In many cases of second most evident finding were urolithiasis characterized by blockage of one or both ureters by urate concretions with attendant atrophy of one or more lobes of the kidney drained by the obstructed ureter. The presence of uroliths in the kidney leads to compensatory hypertrophy of remaining renal tissue. Affected birds often appear normal until ureteral flow from the contralateral kidney is blocked, leading to lethargy, straining, and death [6]. Urolithiasis is condition seen particularly in caged laying hens [9].

Visceral gout and urolithiasis as a cause of pullet and layer mortality continues to be a diagnostic challenge. For the field veterinarians still seem to be difficult to react until etiologic factors are better defined it is difficult to make specific recommendations. In order to prevent visceral gout Charlton *et al.* [9] recommended to observe reasonable limits of calcium and available phosphorus in rations during grow-out and to avoid electrolyte imbalance, mycotoxins and water deprivation.

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