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

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.
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.

**Macrosocical 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 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
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.
REFERENCES


