

Descriptive Study of an Outbreak of Avian Urolithiasis in a Large Commercial Egg Complex in Algeria

Hicham SID, Amine FETTAH, Abdelaziz LOUNAS

University of Blida, Department of Veterinary Sciences, Blida Road Of Soumaa BP 270 Blida, Algeria; sidhicham@gmail.com

Abstract

Avian urolithiasis is one of the major causes of mortality in poultry. However, in Algeria this condition has never been described. An outbreak of avian urolithiasis was observed on a large commercial egg complex in the department of Chlef (West of Algeria). The clinical features of this condition are to be described. Mortality associated to urolithiasis started at the onset of egg production, estimated to 0.7 % per week. Urolithiasis induced an egg drop estimated to 12%. Dead and live layers were both necropsied and examined for kidney lesions. Most of the birds examined presented enlarged ureters, renal atrophy and visceral gout deposition.

Keywords: Algeria, caged layers, mortality, outbreak, urolithiasis

Introduction

The development of poultry industry in Algeria has faced serious obstacles of various types, especially on the basis of intensive production systems. Infections of flocks are becoming more often with significant losses, such as avian urinary tract disease. This condition is generally just one element of a systemic bacterial, fungal, viral, or metabolic disease (Styles and Phalen, 1998).

The kidneys are involved in a wide range of essential functions; therefore it is not surprising that kidney dysfunction can cause profound problems for the poultry industry. These problems range in severity from increased manure moisture to catastrophic flock mortality.

Urolithiasis is the formation of large urate "stones" in the ureters of affected birds (Wideman *et al.*, 1983). Death occurs due to uricemia when functional kidney mass is insufficient to sustain a normal metabolism (Mallinson *et al.*, 1984; Randall *et al.*, 1977; Raymond *et al.*, 1988). This condition is primarily observed in caged layers and induces an increased mortality and a decrease in egg production; it has been described in Great Britain as an enzootic disease (Siller, 1981).

Kidney malfunction is associated to multiple etiologies (Darbyshire, 1979; Fitz-Coy *et al.*, 1988; Wideman *et al.*, 1983). Ansar *et al.* (2004) reported that the urinary system of the treated birds with high dietary calcium and low phosphorous displayed inflammatory lesions, showing abnormalities of color, size, shape and texture of kidneys and ureters. Julian (1982) noticed that water deprivation is a cause of renal disease in chickens. On the other hand, Chousalkar *et al.* (2007), Ratanasethakul and Cumming, (1983), Mallinson *et al.* (1984) reported that the nephropathogenic IBV (Infectious Bronchitis Virus) plays an im-

portant role in kidney damage. However, in Algeria, this condition has never been reported in the past. This work is a descriptive study that reports an outbreak of urolithiasis in a flock of ISA Brown caged layers in Algeria.

Materials and methods

Case history

This study was conducted in a laying hens complex located in Chlef (West of Algeria), housing 270.000 caged layers. Layers were reared in 6 modern industrial houses. Every data was registered since the creation of this complex in 2001 (weekly mortality, treatments, egg production and feed consumption).

Between 2008 and 2010, urolithiasis started to spread from primary flock to all other flocks. It has been noticed a higher mortality rate at the onset of egg production.

This work has been carried out from May 2010 to August 2010. A research group from Blida University (Algeria) investigated this outbreak of avian urolithiasis in a flock of forty five thousand hens of ISA Brown (19 to 35 weeks-old), in order to describe the clinical features and to discuss the possible cause(s) of this condition.

All chicks in the studied flock were from the same source. They were vaccinated in the hatchery for Marek's disease by injection. At day 1, they were vaccinated in the farm against Infectious Bronchitis (IB) and Newcastle Disease (ND) via spray (Nobilis[®] ND Clone 30+MA5). At day 18, against Gumboro disease via the drinking water (Nobilis[®] Gumboro E228). At 4 and 8 weeks of age via spray, boosters ND and IB were given (Nobilis[®] ND Clone 30 ND +MA5). At 10 weeks of age, pullets were vaccinated against Fowlpox via wingweb transfixion.

At 16 weeks of age, they were vaccinated against EDS (Egg Drop Syndrome), ND and IB by injection of an attenuated vaccine (Nobilis[®] ND+IB+EDS).

The flock was treated during the onset of egg production by two antibiotics (Enrofloxacin then Tylosin) for suspected RCD (Respiratory Chronic Disease). All treatments were added in the drinking water.

Management conditions

Single age birds were housed in a closed house with application of an all-in-all-out system. The house is built on well-drained land and has natural air movement. In order to ensure that birds receive the ideal amount of fresh air; a tunnel ventilation system is used. Ventilation, cooling and heat systems are controlled by a climate computer. Hot air generators are installed inside the building and uniformly distributed along its length.

Feed is stocked in silos outside the building. The silo can be loaded from above. Inside the house, feed is distributed by automatic hoppers which are controlled by a computer.

Before the transfer of pullets to the production site, all vaccinations were given a week before this operation. Transfer has been carried out at 17 weeks of age; each pullet was housed in less than 350 cm² floor space and able to reach at least 2 automatic drinkers (5 to 6 hens were housed per cage).

Forty five thousand hens were housed in this modern house; all environmental factors were controlled (lighting, temperature, humidity and level of toxic gases in the air). During the outbreak of urolithiasis, performance data were recorded (egg production, feed consumption and body weight). Pullet and layer feed formulations were examined carefully for their nutrient levels and analyzed for mycotoxins levels. The feed is locally prepared for all flocks with changes in nutrient levels for each phase of development. Vaccination program and medications were recorded.

Samples of water and feed from pullet and layer houses were collected for their concentration of heavy metals and calcium-phosphorous levels.

Necropsy observations

Dead layers (562 layers) aged between 19 to 35 weeks-old were necropsied on many occasions during the outbreak of urolithiasis.

Thirty three live layers were randomly selected (one layer per cage and one cage per 200 cages); they were euthanized by dislocation of cervical vertebrae and necropsied. Both live and dead layers were evaluated for the ureters

and kidney lesions in the same period (19 to 35 weeks). Depending on the severity of lesions, kidney atrophy was evaluated from slight to severe.

Statistical analysis

Test of association was made by means of a chi-square test depending on the number of dead or live hens examined; it was conducted at $P < 0.05$.

Results and discussion

Despite some differences in the age of the affected birds, necropsy observations and case history of this urolithiasis outbreak were similar to urolithiasis in caged layers described in United States (Brown *et al.*, 1987; Cowen *et al.*, 1986, Mallinson *et al.*, 1984). Authors investigated the pathology of same urolithiasis outbreak in caged layers. A variety of agents have been implicated, especially infectious bronchitis viruses and excessive dietary calcium

At the outbreak of urolithiasis, clinical signs at the affected birds were dehydration, pale combs and depression. It is probable that some hens died rapidly without prodromic signs. Some birds exhibited other signs like emaciation, swollen feet; additionally, they had a tendency to hide. These findings are in support with the observations of Fitz-Coy *et al.* (1988) who reported the same clinical observations in commercial white leghorn pullets.

The mortality at flock of 19 to 35 weeks old due to urolithiasis was relatively high, estimated to 11% compared to the normal rate (around 1.5%). Weekly mortality was estimated to 0.7% which is in agreement to Brown *et al.* (1987) who stated that losses from this disease range between 0.5 to 1% per week. Mortality was mostly associated to severe kidney atrophy and obstruction of ureters.

Necropsy findings for dead and live layers showed kidneys atrophy (Tab. 1) and uroliths deposition. The relationship between renal atrophy and uroliths deposition is showed in Tab. 2. Most of the examined birds presented enlarged ureters due to uroliths (Fig. 1). Several hens presented enlarged pale kidneys (Fig. 2), visceral gout and articular gout in the tibiotarsal joints.

Necropsy observations revealed that kidney atrophy in live chickens was slighter (24%) than severe (9%). On the other hand, 45% of the dead chickens presented severe kidney atrophy. Significant association have been found ($P < 0.05$) between severe kidney atrophy in dead chickens (146/253) and the presence of uroliths which suggests the possibility of the implication of kidney atrophy in uroliths formation. However, most of the live chickens necropsied

Tab. 1. Kidney lesions of caged layers during the urolithiasis outbreak

	Kidney atrophy				Number of chickens examined
	Severe	Moderate	Slight	Normal kidney	
Dead chickens	253(45%)	151 (27%)	102(18%)	56(10%)	562(100%)
Live chickens	3(9%)	5(16%)	8(24%)	17 (51%)	33(100%)

Tab. 2. Severity of kidney atrophy and its relationship with uroliths formation

	Kidney atrophy		
	Severe	Moderate	Slight
Dead chickens			
Uroliths	146/253	128/151	0/102
No Uroliths	107/253	23/151	0/102
Live chickens			
Uroliths	3/3	3/5	3/8
No Uroliths	0/3	2/5	5/8



Fig. 1. Live 30 weeks-old layer. Large uroliths filling both ureters with moderate atrophy of the right kidney

presented a slight kidney atrophy (24%). Our results coincided with the findings of (Siller, 1981) who reported that kidney atrophy contribute to the formation of uroliths.

Feed analysis revealed normal calcium and phosphorus levels. However, it has been noticed that pre-layer and layer formulas contain higher levels of calcium (respectively 2% and 3.5%) than the growing formula (1%), as this high level is needed for egg production. We have noticed that mortality triggered just after replacing the growing formula by the pre-layer formula. Our results coincided with the findings of Wideman and Cowen (1987); Ansar *et al.* (2004) who reported that high calcium diets may play an important role in urolithiasis outbreak and modifications of feed formulas can be a factor of physiological stress.

Cage density was relatively high (less than 350 cm² per layer) which may play the role of stress factor.

Water samples showed acceptable limits of heavy metals concentrations. However, it has been noticed an increase in water intake during this outbreak. According to Julian (1982), water deprivation possibly contributed to outbreaks of urolithiasis in two flocks of laying hens by decreasing fluid flow through the kidneys. Additionally, electrolyte balance may be etiologically involved, as is suggested by the apparent role of water deprivation in outbreaks of urolithiasis and in increased serum urea and uric acid levels (Losing, 1980).



Fig. 2. Live 20 weeks-old layer. Enlarged pale kidneys

Egg production was affected during this outbreak; the egg drop was estimated to 12%. This result doesn't correlate to the observations of (Cowen *et al.*, 1986) who reported that layers affected by urolithiasis remain productive.

During the outbreak, urolithiasis was observed in caged layers 19 or more weeks of age, but later this condition was seen in pullets too (14 to 18 weeks of age).

Despite vaccination program for IBV (Infectious Bronchitis Virus), a variant nephrotropic IBV may be involved in this urolithiasis outbreak. Results of similar outbreaks in Unites States (Cowen *et al.*, 1986; Brown *et al.*, 1987) showed that variant nephrotropic IBV may be involved in kidneys damage. Lee (2002) found that nephropathogenic strains of IBV cause lesions in the kidney, based on several tissue samples collected at various times.

Conclusions

Urolithiasis is a degenerative renal disease involving urolith formation in the ureters of caged layers. It may be associated with multiple etiologies (high dietary calcium, low dietary phosphorus or nephropathogenic IBV). In Algeria this condition has never been described before the current study. As a conclusion, we can say that urolithiasis occurs in caged layers at the onset of egg production. Mortality caused by urolithiasis outbreak during this study was important (0.7% per week). The egg drop induced by this condition was estimated to 12%.

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