



Prevalence and Pathology of Egg Bound Syndrome in Commercial White Leghorn Chicken

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ABSTRACT

Prevalence of egg bound syndrome in commercial white leghorn layer chicken in Namakkal region of India was studied over a period of two years from June 2009 to May 2011. Out of 24,158 carcasses examined, the egg bound syndrome was noticed in 663 cases with a overall mortality of 0.5 %. The study revealed that heat stress (28.66%), asphyxia (23.23%), hypocalcemia (17.35%), salpingitis (7.54%), large size egg (6.18%), dehydration (5.73%), vent trauma (5.28%), obesity (3.62%), abnormal ovulation (1.81%) and oviduct neoplasm (0.60%) were the various causes of egg bound syndrome in commercial layer chicken. Microbial analysis of the samples revealed the presence of *Escherichia coli* from cases associated with salpingitis. The syndrome was recorded with highest occurrence in 21-30 wk and 61-70 wk age laying chicken and with a higher incidence in summer season (44.95%).

Keywords: Layer chicken, Prevalence, Egg bound

INTRODUCTION

Namakkal is the most thickly populated poultry zone in India with a layer population of 45 million birds and occupying second place in egg production at national level (Srinivasan et al., 2012a). As a result of continuous and rapid selection for more egg production and heavier egg weight, current commercial layer hybrids produce more than 350 eggs in the 60 wk laying period (Catli et al., 2012). Although it is well known that reproductive disease of poultry results in decreased egg production and increased mortality, avian reproductive pathology is treated rather briefly in literature (Solomon, 2002).

The avian oviduct is a tubular organ responsible for forming the egg by the secretion of components surrounding the yolk. Oviductal derangements such as atrophic oviduct (Srinivasan et al., 2012a), cystic oviduct and persistent right oviduct (Srinivasan et al., 2011a and 2011 b) are reported in commercial layer chicken and cause reduction in egg production and mortality. In egg bound syndrome a fully or partially formed egg is lodged in the shell gland or vagina, but can not be expelled by the bird at a normal rate. It is commonly noticed in pet birds (Worell, 1999) and broiler breeders (Eitan and Soller, 2009) resulting in life threatening symptoms and high mortality. Modern commercial layers are highly prolific and susceptible to

egg bound syndrome, however it was rarely diagnosed and reported (Batra and Singh, 1978) and their etiological factors were not investigated in relation to their age and season wise occurrence. To ensure persistent and maximum production in poultry flocks, it is imperative to investigate the egg bound syndrome in order to understand its prevalence, nature and significance of this disorder. Therefore present study was planned to find out the prevalence of egg bound syndrome associated mortality in layer chickens in Namakkal zone of India.

MATERIALS AND METHODS

Flock history

The study was carried out over a period of two year (June 2009 to May 2011). A total of 24,158 carcasses of white leghorn layers, above 20 weeks of age belonging to commercial poultry farms situated in and around Namakkal district of Tamil Nadu state, India were examined for the presence of egg bound syndrome. The flocks showing egg bound syndrome were inspected and information regarding shed capacity, flock size, cage size, strain of chicken, cage management and hygiene, feed additives, vitamins-mineral supplements, treatments and egg production

traits were collected from the owners of the farms. To study the seasonal variations in the incidence of the egg bound syndrome, the whole year was divided into four seasons namely summer (March, April and May), south west monsoon (June, July and August), north east monsoon (September, October and November) and winter (December, January and February). According to the age, layers were grouped as 21 – 30 wks, 31- 40 wks, 41 – 50 wks, 51 – 60 wks, 61 – 70 wks and 71 – 80 wks.

Pathological examination

The dead birds were surface disinfected and necropsies were performed as per approved procedure (Chauhan and Roy, 2007). The area of the vent and cloaca was examined for signs of inflammation and prolapse. The carcasses were thoroughly examined for gross pathological changes including inflammatory signs and presence of exudates in the peritoneal cavity, ovary, oviduct or all of them. Oviduct with egg bound was removed and opened along its longitudinal axis for examination of mucosal surface and shell formation, size, numbers and position of eggs.

Isolation of causative agent

Heart blood, liver and oviduct swabs were collected from 663 dead birds with egg bound condition for screening of bacterial agents. The samples were placed in Brain Heart Infusion (BHI) broth and incubated at 37°C for 24 h and cultured aerobically in Brian heart infusion agar (BHIA) MacConkey's agar and eosin methylene blue agar (EMBA) for isolation of bacteria. Bacterial isolates were identified on the basis of their morphology, growth characteristics, sugar fermentation and biochemical characteristics (Quinn et al., 2011). Trachea, lung, spleen, caecal tonsil, kidney and oviduct collected from egg bound cases were subjected to haemagglutination (HA) test for detection of Newcastle disease virus (NDV) (Alexander and Senne, 2008; Mohammad et al., 2013) infectious bronchitis virus (IBV)(Villarreal, 2010) and egg drop syndrome – 76 (EDS-76) virus (Alam et al., 2009). Serum samples collected randomly from ten birds from flocks affected with egg bound syndrome were examined by haemagglutination inhibition (HI) test for the presence of antibodies to NDV, IBV and EDS virus and by ELISA for the *Mycoplasma gallisepticum* (Mg) and *Mycoplasma synoviae* (Ms).

RESULTS

Egg bound syndrome in commercial layer chicken was diagnosed based on the presence of partially or fully formed egg in the oviduct especially in the shell gland or vagina on necropsy examination and laboratory investigation (personal observation). The syndrome was recorded in 663 (2.74 per cent) out of 24158 birds examined over a period of two years (June 2009 to May 2011) as shown in the Table 1. Among the various causes large sized egg, heat stress, hypocalcemia and asphyxia induced by infectious laryngotracheitis and diphtheritic pox caused sudden death whereas the others caused chronic death. Ovarian

follicles were congested, fully developed with follicular hierarchy in suddenly died birds where as in chronic cases follicular regression and rupture with spillage of yolk materials in the abdominal cavity and occasionally the oviduct was atrophied and covered with thick exudates.

Large sized eggs due to double yolk in young layers was longer, broader and bigger in size (60 to 65gm) compared to normal egg (40 to 45 gm), fully formed and lodged in uterus or vagina (Fig. 1) (personal observation). In abnormal ovulation, an oviduct contained two eggs in different region of oviduct at different states of formation (Fig. 2) or in a distended shell gland (Fig. 3). Heat stress affected birds, uterus contained mostly thin shelled or shell less eggs with ovarian congestion (Fig. 4). In hypocalcemia an active, congested ovaries with partially or fully formed egg in the shell gland (Fig. 5) were observed. In few cases only thin shell membrane was noticed in the lower part of the oviduct and the femur was very fragile with little or no medullary bone. Traumatic injury of cloaca the vent region was thickened by blackish crusty materials with fully formed egg in the lower part of the oviduct (Fig. 6). Obese birds, showed enlarged, pale yellowish and friable liver, thick abdominal fat and fully formed egg in shell gland (Fig. 7). In dehydration the condition of the carcasses was fair to poor and the oviduct wall was tightly adhered on the egg surface (Fig. 8). Asphyxia associated egg boundness was commonly noticed in infectious laryngotracheitis and diphtheritic pox, in this the birds showed normal follicular hierarchy, completely formed egg in the uterus and haemorrhagic or caseous exudate in the upper part of the trachea (Fig. 9). In salpingitis cases oviduct contained fully formed egg coated with albuminous exudate and the mucosa showed mild thickening and congestion (Fig. 10). Neoplastic condition of oviduct especially uterine adenocarcinoma the mucosa was markedly thickened and the lumen was narrowed with multiple eggs in various stages of formation in different regions of oviduct (Fig. 11). The first egg acquires a white band and chalky appearance, while the second egg is flattened on its contiguous surface (ie, slab-sided). In few birds internal laying of thin shelled eggs was also noticed.



Fig. 1. Egg bound syndrome in a 28 wk old layer with double yolk



Fig. 2. Egg bound syndrome in abnormal ovulation revealed two eggs in different region of oviduct of 25 wk old white leghorn chicken.

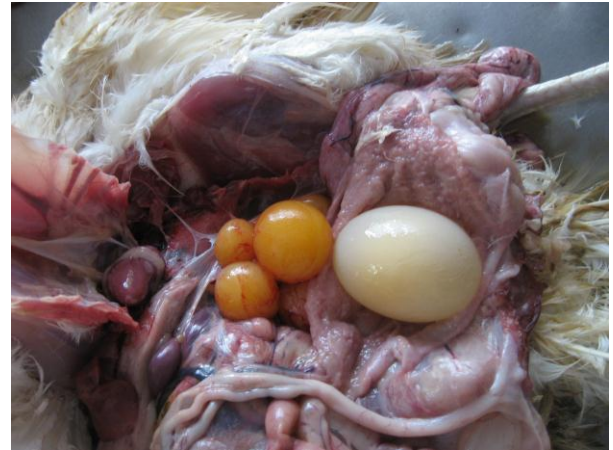


Fig. 5. Egg bound syndrome in hypocalcemia showed partially formed egg in the Uterus of white leghorn chicken.



Fig. 3. Egg bound syndrome in abnormal ovulation revealed two eggs in different stages of formation of white leghorn chicken.



Fig. 6. Egg bound syndrome in vent pecking showed fully formed egg in the uterus with wound in the cloacal region of white leghorn chicken



Fig. 4. Egg bound syndrome in heat stress showed thin shelled egg in the Uterus of white leghorn chicken.



Fig. 7. Egg bound syndrome in obesity showed thick abdominal fat and fully formed egg in the uterus of white leghorn chicken.



Fig. 8. Egg bound syndrome in dehydration showed enteritis and fully formed egg in uterus of white leghorn chicken.

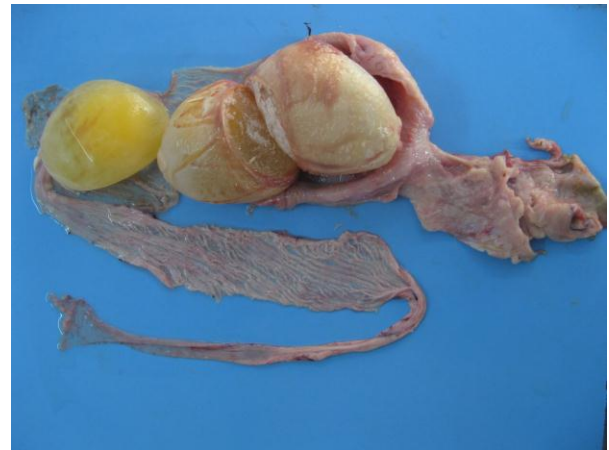


Fig. 11. Egg bound syndrome in uterine adenocarcinoma showed thickened uterine mucosa with multiple eggs in various stages of formation in white leghorn chicken.

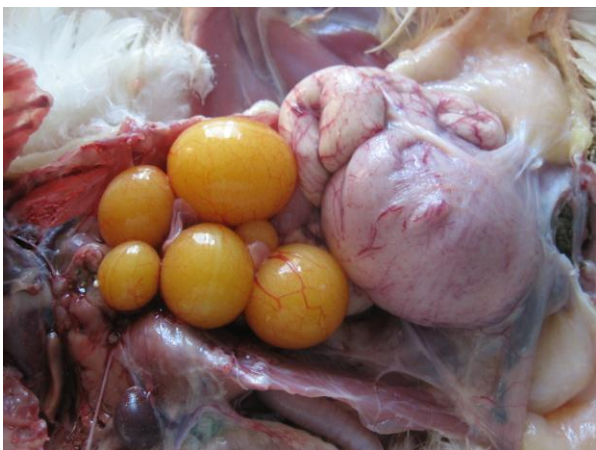


Fig. 9. Egg bound syndrome in asphyxia showed fully formed egg in the uterus of white leghorn chicken



Fig. 10. Egg bound syndrome in salpingitis showed albumin coated fully formed egg in the oviduct of white leghorn chicken.

Bacteriological examination of liver, heart blood and oviduct swabs collected from egg bound cases associated with salpingitis revealed the presence of *E.coli*. The organism was identified based on lactose fermenting, pink colour round, smooth and glistening colonies on Mac Conkey's agar, black colonies with metallic sheen on EMB agar, indole production at 44°C, gas production in Eijkmann test and acid and gas production in differential sugar fermentation tests. Tissue samples collected for virological examination was found to be negative in haemagglutination test against NDV, IBV and EDS-76 virus. Moreover, it was confirmed in the serological tests also, since all sera were positive for Newcastle disease virus and Infectious bronchitis virus as a result of vaccination. The HI titer for EDS- 76 and ELISA value for Mg and Ms were found to be negative.

Age and season wise occurrence of egg bound syndrome in layer chicken was presented in Table 1. Age wise analysis on the overall occurrence of egg boundness in 663 commercial layer chicken showed highest occurrence in 21 to 30 wk followed by 60-70 and 70 – 80 wk age groups. Analysis of data on the season wise occurrence showed highest incidence during summer (44.45 per cent) followed by south west monsoon, winter and north east monsoon. Mortality in the affected flocks was 0.5 per cent however morbidity and drop in egg production was not observed.

DISCUSSION

Commercial layers in India are predominantly white egg producers (>95 per cent) with good farming practises, production is 350 eggs per hen housed in a 60 wk laying cycle. Expulsion of the fully formed egg (oviposition) from the oviduct involves the muscular contraction of the uterus (shell gland) if there is any delay or defect in the mechanism leads to egg bound syndrome in poultry. In the present study 2.74 per cent of dead birds showed egg bound syndrome which was in accordance with the results of Bhattacharjee et al. (1996) who also observed 2.80 per cent in layer chicken. However, Rahman and Samad (2003) and

Muthulakshmi et al. (2012) observed egg bound syndrome in 8.28 and 8.0 per cent respectively in layer chicken. The difference in the occurrence of egg bound

syndrome was due to variation in the duration of the study, management, climatic condition and type of birds utilized for the study.

Table 1. Egg bound syndrome: Age and season wise distribution and proportionate incidence

S.No	Cause of egg boundness	Age (weeks)						Season				No. of cases	Proportionate incidence (%) encountered
		21-30	31-40	41-50	51-60	61-70	71-80	Summer	SWM	NEM	Winter		
1.	Large size egg	17	03	--	--	11	10	11	06	09	15	41	6.18
2.	Abnormal ovulation	07	05	--	--	--	--	05	02	03	02	12	1.81
3.	Obesity	05	02	--	--	08	09	04	03	05	12	24	3.62
4.	Vent trauma	18	11	06	--	--	--	11	07	09	08	35	5.28
5.	Hypocalcemia	--	--	14	25	36	40	56	15	20	24	115	17.35
6.	Dehydration	5	6	4	10	7	6	16	05	04	13	38	5.73
7.	Asphyxia	45	25	31	23	16	14	56	40	36	22	154	23.23
8.	Heat stress	55	41	27	25	26	16	130	50	-	10	190	28.66
9.	Salpingitis	16	10	9	5	6	4	09	10	20	11	50	7.54
10.	Oviduct neoplasm	--	--	--	--	2	2	--	01	01	02	04	0.60
Total		168	96	93	91	112	103	298	139	107	119	663	100
Per cent		25.34	15.54	13.73	13.27	16.89	15.23	44.95	20.96	16.14	17.95		

SWM- South west monsoon, NEM- North east monsoon

Egg bound due to large sized egg was commonly noticed in young layers (21 to 30 wks) due to double yolked egg. The results are in consistent with Lewis et al. (1997) who also observed double yolked eggs during the start of a laying period and decreased as the birds mature. Ovulation normally occurs half an hour after oviposition. Simultaneous development and release of two follicles at a time occurs due to increased level of reproductive hormones by an over stimulated ovary in young layers (Lowry et al., 1979). Abnormal size egg will stretch and possibly weaken uterine muscle leads to egg bound. In hens, the next day's egg enters the shell gland (uterus) 5 h after oviposition (Warren and Scott, 1935). In few birds two eggs in different state of formation at different region of oviduct or two eggs in one distended shell gland was noticed due to erratic ovulation (Robinson et al., 1991).

Heat stress associated egg bound was noticed throughout the laying cycle however more common during the early laying period. Modern commercial layer strain attain peak production in between 25 to 30 wk (Srinivasan et al., 2012a) during this period metabolic heat production is increased to meet out the production demand makes them unable to cope up with the increasing environmental temperature. Presence of thin shelled egg or shell less egg in uterus is due to the death of the birds in the evening hours of day, since calcification occurs during night hours. Moreover if laying hens exposed high temperature plasma calcium level significantly reduced due to reduction in calcium use and uptake by the uterine and duodenal epithelial cells respectively leads to hypocalcemia (Mahmoud et al., 1996).

Egg-bound cases associated with hypocalcemia revealed partially or fully formed eggs in the uterus or sometimes only egg membranes. A calcium deficient bird may produce eggs whose shells are softer or more fragile than normal and it is very difficult for a bird to lay a soft-shelled egg, since the muscles that push the egg out tend to deform the egg rather than moving it. The egg may be stuck near the cloaca, or further inside. However, ovarian follicles were well developed with

follicular hierarchy, indicating that the birds were actively laying before death (Julian, 2005).

Incidences of obesity associated egg bounding were less common in commercial layers compared broiler breeders. Obesity caused egg bounding in young layers and above 60wk of age due to over feeding during growing period and excess energy intake compared to production in older birds. In obesity, increased amount of lipids also deposited in the glandular epithelium of the shell gland which impairs calcium utilization for the egg shell formation. Excessive abdominal fat in vent region will reduce the elasticity of the oviduct and predispose to egg boundness (Brake and Thaxton, 1979). Infectious laryngotracheitis and diphtheritic pox caused sudden death due to asphyxia with partially or fully formed eggs in the oviduct and these lesions are in conformity with earlier reports (Bhattacharjee et al., 1996; Srinivasan et al., 2012b). Oviposition activity in the affected bird might have aggravated the partial or complete obstruction of tracheal lumen and resulting asphyxia (Srinivasan et al., 2012b). Injuries on the vent lead to inflammatory condition and pain can hinder egg laying (Kaikabo et al., 2007). Salpingitis might cause chronic irritation and hyperactivity of oviduct leading to production and deposition of albuminous exudate on the formed egg (Srinivasan et al., 2013)

In uterine adenocarcinoma, the oviduct lumen was narrowed resulting in mechanical obstruction of egg movement leads to presence of two or more eggs in various stages of formation in the oviduct. As the second egg comes in contact with the first, pressure is exerted, disrupting the normal pattern of mineralization. The first egg acquires a white band and chalky appearance, while the second egg is flattened on its contiguous surface (ie, slab-sided) (Reynard and Savory, 1999). Uterine adenocarcinoma was commonly encountered above 61 wk, which concurs with the earlier report (Bwala et al., 2011) that ovarian and oviduct adenocarcinoma are frequently encountered in older white leghorn hens.

In the present investigation *E.coli* was isolated from egg bound cases associated with salpingitis. Among the various bacteria that cause primary or secondary reproductive tract infections *E.coli* is most common in commercial layer chicken (Srinivasan et al., 2013). Although the route of infection is not clearly known, contamination of vent, cloaca and oviduct with faecal material has been seen as an important source of such infection (Keller et al., 1995). Current study revealed lower incidence of infectious cause (30.77%) compared to non infectious causes (Uddin et al., 2011).

This disorder was recorded in all the four seasons; however the incidence in summer was higher than other seasons. This finding was in agreement with Uddin et al. (2011). High occurrence in this period might be due high incidence of hypocalcemia, heat stress and asphyxia due to ILT and pox. In affected flocks, birds were apparently healthy and may die due to exhaustion or from an infection. This may be the probable reason for sudden death in apparently healthy bird without any premonitory signs. The flocks maintained the standard egg production since affected birds died suddenly and the remaining birds appeared normal and maintained the production. In egg bound syndrome affected flocks 0.50 percent mortality was recorded. Similar observation was also made by Uddin et al. (2011). Mortalities from reproductive pathologies are rare and in most cases caused by other complications (Jordan et al., 2005).

Based on the above study, it may be concluded that egg bound syndrome is one of the pathological condition noticed in commercial layer chicken with an overall mortality of 0.5 %. Among the various causes of egg bound syndrome, the noninfectious factors played a major role and the condition was more prevalent during early and last stage of lay and in summer season.

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