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Original Article

Principal Lesions and Patterns of Mortality Observed in a Broiler Breeder Flock During the Laying Period

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ABSTRACT

In any broiler breeder flock, there is always some mortality that is considered to be "normal" during the life of the flock. The aim of this study is to summarize the prevalence and patterns of mortality during the production period. To evaluate the mortality, each dead hen was systematically and completely examined via necropsy. In addition to post mortem evaluation, weights (whole body, liver, spleen and heart) were recorded to correlate the bird's physical condition with the mortality category or lesion. In general, the variation in mortality was not associated with the specific weight of the organs. However, for specific diseases like fatty liver, septicemia and ascites, there was a correlation with the organ weight of the liver, spleen and heart respectively. Ovaritis was observed to be the most prevalent lesion found in dead hens during the production period followed by ovarian regression (atrophy), peritonitis, internal lay and vent pecking. The two periods with the higher total percentage of morality were onset of lay (25-30 weeks) and end-stage of the production period (56-61 weeks). During the laying period, we mainly observed metabolic disorders followed by infectious lesions. Infectious lesions, including ovaritis and septicemia, were the predominant cause for mortality at the onset production. Vent and feather pecking were also primarily concentrated during the first weeks of the production period. Metabolic diseases were observed in higher prevalence during the latter half of the production period.

INTRODUCTION

Principal Lesions and Etiology

Peritonitis is one of the most prevalent diseases causing mortality of laying hens during all stages of the production cycle. While there are many different causes for this disease in broiler breeder hens, the primary pathogens involved in peritonitis include *E. Coli,* Jordan *et al.* (2005), *Pasteurella multocida,* Goodman (2001) and *Salmonella,* Shivaprasad (2000). Ovaritis is often associated with peritonitis, and can be considered to be a consequence of the disease.

The exact route of colonization of pathogens in the ovary and the abdominal cavity of the hen is controversial. For example, some authors, consider an ascending route of infection from the cloaca to be the primary route of infection and cause of peritonitis. Other authors believe that a systemic infection is the most important route of infection and that other organs are posteriorly infected, including the ovary which may be infected due to the rich blood and nutrient supply that makes it a perfect substrate for microbial growth. With regards to the ascending route, this appears to be a likely course of infection due to the elevated estrogen levels in laying hens that induces a relaxation of the sphincter muscle between the vagina and the cloaca.

An additional problem to consider in laying hens is an anatomical "displacement" between the ovary and the location of the infundibulum (the structure that catches the ova). If this anatomical abnormality occurs, it can result in sterile peritonitis, also known as an 'internal layer' if the ovum is not captured by the infundibulum, but rather falls into the abdominal cavity. The displacement of the infundibulum can be caused by excessive fat in the hen, malformation of the oviduct, or inflammation and the consequential deformation of the ovary.

Ovarian regression or atrophy is often seen as a sign of physical discomfort and can be associated with any kind of chronic disease and management problems (lack of water, excessive ambient temperatures, insufficient ventilation, or an excessive percentage of males in the flock). Many authors have highlighted the observation that the reproduction cycle of the hen is a "luxury" that develops when the conditions are correct, and is one of the first functions to disappear when stress is present.

Bird pecking is one of the most common multifactorial problems in broiler breeder flocks during the rearing and laying periods. Various situations can provoke or incite curiosity in the hen that later results in pecking and subsequent cannibalism in the flock. These scenarios include prolapsed cloaca, diarrhea at the onset of production, or bloody wounds, particularly near the vent of the bird. Stressful situations are also implicated with this syndrome and can include elevated light intensity during the rearing period, excessive environmental heat, and high bird density. In recent years, there have been an increase in reports of male aggression and the resulting chain of hen pecking attributed to roosters. The consequences of this pecking can be diminished with beak trimming but this does not resolve the root problem of the pecking.

The presence of fatty liver syndrome in breeder birds kept on litter is not as common as caged birds. Nonetheless, in both cases, the syndrome is associated with high energy diets, birds with large pale combs and wattles, and at post mortem, the liver will appear to be enlarged, friable and pale due to the altered liver architecture. In advanced cases of fatty liver syndrome, the dead birds may also have large blood clots in their abdominal cavity. Sometimes the hemorrhage is encapsulated by fibrin produced by the rupture of intrahepatic portal veins associated with degenerative changes in the veins. High energy diets can also contribute to the hemorrhagic syndrome with fatty liver. Hens with fatty liver syndrome often have high levels of phospholipids C20:3n3, while otherwise healthy hens have higher levels of C18:3n. Phospholipids are essential cofactors in the biochemical reactions involved in thrombin generation. Thus, this change suggests that alterations in the composition of the phospholipids may be a contributing factor in the development of fatty liver hemorrhagic syndrome.

Cloacal prolapse is principally caused by abnormal oviposition, and is most commonly seen in overweight hens and is contributed to excessive fat accumulation within the pelvic area. However, prolapse can also be seen in young hens and in this case the main problem with oviposition is the narrowness of the pelvic opening combined with the production of double yolk eggs. This complication may be related to early increases in light duration or feed stimulation. In both cases, the hen has difficulty laying the egg and this often results in the prolapse of the cloaca. Due to the extended period of prolapse that can be associated with the oviposition, pecking of the prolapsed tissue can result which may lead to cannibalism, death and evisceration.

Uric acid is the main nitrogenous waste product in birds, and consequently birds can develop gout, secondary to an abnormal contamination of urates within the bird known as visceral gout or articular gout. Visceral gout is characterized by precipitation of urates in the kidneys and on serous surfaces and appears as a white chalky coating. Urate deposits in organs is caused by renal failure and the main cause is dehydration. Other causes include nephrotoxicity, infectious agents as IBV, avian nephritis virus, glomerulonephritis and vitamin A deficiency. Articular gout is normally associated with genetics and high protein diets.

Ascites and right ventricular failure is more common in broilers than breeders, because of the feeding regime followed. Pulmonary hypertension, due to hypoxemia, which causes right ventricular hypertrophy and failure, has been considered the main cause of ascites. Modern broiler strains are considered more susceptible because of the smaller ratio of lungs relative to body size, thicker blood-gas barrier, and larger and less deformable red blood cells Julian (1989). The heart is the primary organ affected, but other organs such as the liver or spleen can also suffer increases due to changes in their size.

Metabolic joint disorders in broiler breeders are not as frequently seen due to the use of controlled feeding regime during the rearing period of the chicken which results in less stress on the joints. Selection of broiler breeder males during rearing includes an evaluation of their joint structure and therefore it is not common to find roosters with tibiotarsal or toe deformities.

Similar work has been carried out on modern broilers and backyard poultry, but the number of similar studies done on broiler breeders is limited.

MATERIAL AND METHODS.

A 3500 hen flock of the Cobb male line grandparent breed was followed for 36 weeks during the production period, from 25 to 61 weeks of age. Every dead hen was collected and frozen as soon it was detected by the farmer. Once a week, all dead hens were completely and systematically post-mortem examined to evaluate different gross lesions. A total of 666 broiler breeder hens were grossly examined.

Because the fast autolysis of the digestive system, we decided not taking into account enteric lesions. Weights of the body, spleen, liver and heart were recorded for each dead bird to observe correlations with body condition, mortality and disease prevalence. To avoid non-significant results, the blood was washed out and auricles were removed, and only the ventricular weight was taken into account for the heart weight.

In this paper we did not consider histopathology and microbiology. Necropsies took place in the Pathology Department of the *Veterinary School of the Complutense at the University of Madrid.*

RESULTS AND DISCUSSION

Most poultry diseases are well understood, but their prevalence and distribution during the production period is not well known. Our purpose was to observe the 'normal' disease pattern in a broiler breeder flock that did not have any contributing infectious disease outbreaks or management failures.

Results are summarized in Table 1 and Figures 1, 2, 3, 4, and 5.

The type of lesions observed and prevalence is summarized in Table 1. The average weekly organ weights measured (liver (107.6 grams); spleen (1.7 grams) and heart (11.6 grams)) were not correlated to any increases or decreases in percentages of total mortality. However, some diseases like ascites, fatty liver and septicemia resulted in hens with respectively heavier organ weights (heavier heart (19.9 grams); liver (159.1 grams); and spleen (4.5 grams). Approximately 2% of the hens evaluated had no macroscopic lesions.

Weekly mortality was about 0.6%. The mortality pattern describes a peak (1.2-1%) at the onset lay, lower mortality levels during the mid-lay period (0.2%), and a second peak of mortality during the end of the production period (0.6-0.9%). During all periods of production, the mortality was associated with bodyweights slightly below the standard. In our opinion, if the flock is managed correctly, bodyweight does not seem to be an essential factor that can determine the mortality outcome of a flock. Obviously, hens that suffer from any disease will normally stop eating and their body weight subsequently decreases (Figure 1).

Table 1 – Type of Lesion and total percentage observed in dead hens from 25 to 61 weeks. Liver, spleen and heart (only ventricles) weight. Note spleen weight in septicemia, liver weight in fatty liver, and heart weight in ascites syndrome.

Type of lesion	Total lesion 25 to 61 weeks	Liver weight (grams)	Spleen weight (grams)	Heart weight (ventricle weight)
Ovaritis	21	110.7	2.1	9.9
Ovary Atrophy	14	105.3	2	12.3
Peritonitis Internal lay	11	111.4	4.5	11.4
Pecking signs	11	104.2	3.1	12.2
Fatty liver Ruptured liver	10	159.1	1.8	12.3
Pale kidney Urate deposit	9	99	1.3	10.3
Prolapsed cloaca	8	106.3	1.4	9.3
Joint diseases	7	115.2	1.7	11.5
Ascites Enlarged heart	5	112.1	1.9	19.9
Others	2	93.1	1.9	10.1
No lesions	2	101.4	2.3	8.6

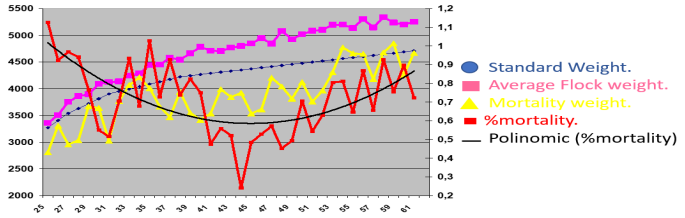

Figure 1 – Mortality, standard weight, real weight of the flock and weight of the dead hens (weekly data).

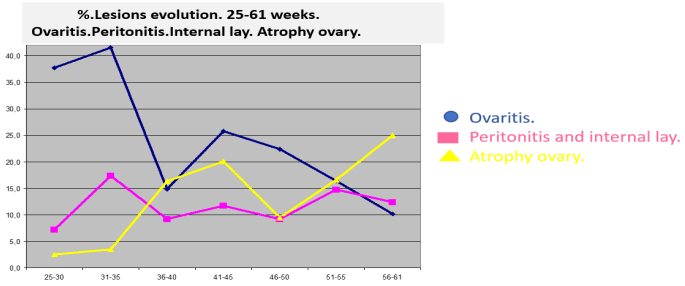

Figure 2 – Percentage of ovaritis, peritonitis and internal lay and ovary atrophy observed in dead hens (grouped every 5 weeks).

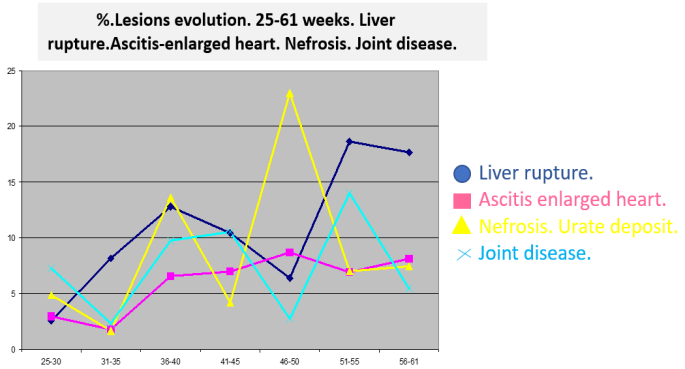

Figure 3 – Percentage of prolapsed cloaca and feather pecking (cannibalism) observed in dead hens (grouped every 5 weeks).

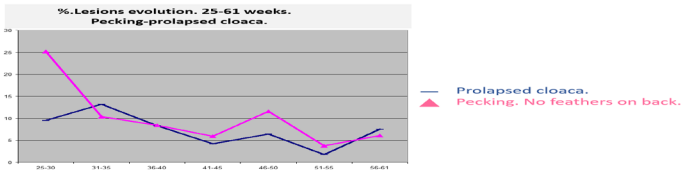

Figure 4 – Percentage of fatty liver/hemorrhagic syndrome, ascites and enlarged heart, urate deposit and bumble foot-joint diseases, observed in dead hens (grouped every 5 weeks).

One of the most prevalent lesions found in a male line flock is ovaritis (11%), which can be associated with salpingitis, peritonitis and perihepatitis, and internal laying (Barnes *et al.*, 2003). These lesions have a constant prevalence during the production period. Particularly during the early production period, most of the peritonitis and internal laying problems are due to a high male to female ratio or a lack of synchronization between the male and female bodyweights (Figure 2). Additionally, malformations due to infectious problems can modify yolk shape and cause inflammation of the oviduct which can make it more difficult for the oviduct to capture the ovum when it is released from the ovary (Ridell, 1996).

Mortality during the first weeks of the production period was mostly associated with the male to female ratio. After decreasing the number of males, the female mortality decreased, not only on poorly-feathered hens but also on feathered hens. The stress created by males chasing females can also be related to the higher mortality observed. A stressed flock is more susceptible to any kind of infectious diseases. Increased hen stress has also been described as one the most important causes for internal laying.

In our opinion, most of the internal laying was caused by the stress created by the males. This is also been reported in heavy hens at the onset of production when they are more susceptible to superovulation.

Ovarian atrophy can be the result of any kind of chronic disease. Its prevalence increases with age and represents an important number of the non-laying hens. These hens also had bodyweights below the flock average.

Ovaritis seems to be the most prevalent lesion found in hen mortality with 21% of the total percentage of lesions found during the production period. Ovaritis is particularly important at onset production since it can be seen in 40% of all dead hens. Estrogenic changes predispose the hen to relax the sphincter making it easier for bacteria to enter into the vent cavity. This is probably one of the main causes of ovaritis due to the high metabolic rate of the ovary coupled with an intense blood flow during septicemic infections make it easier for microorganisms to invade it (Figure 5).

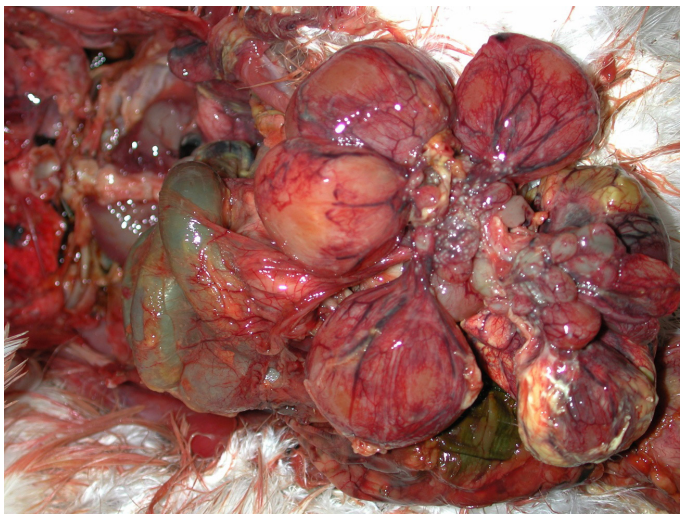

Figure 5 – Ovaritis and peritonitis in a 26 week old hen, note congestion and fibrino--purulent exudate over follicles.

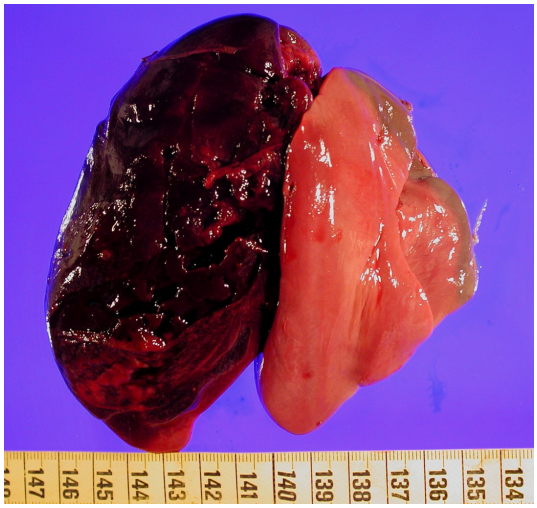

Figure 6 – Fatty liver and hepatic rupture and hemorrhagic syndrome in a 53 week old hen. Note the pale hepatic lobe in contrast with the other hemorrhagic lobe.

Pecking higher prevalence (11%) was established at the onset of production. Wounds, physiological duodenitis (which causes diarrhea at the onset production due to changes in feed programs) and prolapsed vents can stimulate pecking together with high mortality levels. Once a hen is mature (post 35 weeks of age), this kind of behavior decreases (Figure 3).

Male line males normally show more aggressiveness, and in some cases the male-female ratio is the same as female line flocks. Therefore, in our opinion, having fewer males, especially at the onset of lay, can have positive results on hen mortality and bird pecking without having any decrease in hatchability.

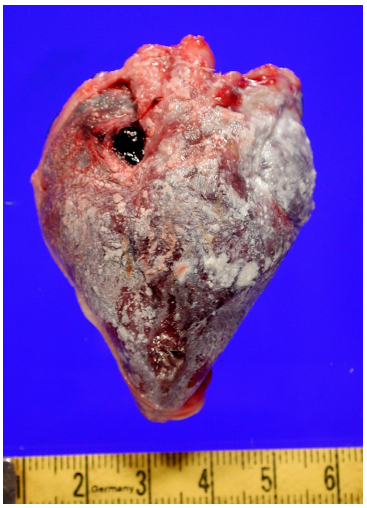

Figure 7 – Urate deposits in the pericardium (visceral gout) of a 41 week old hen. Note white mineralized deposits.

Prolapsed vents (8%) represented an important problem at early stages of production, but decreased from 15% to 5% at 62 weeks and did not show any correlation with the heaviest bodyweights. Therefore, this issue is not related to a fatty oviduct that could prevent the egg transit but is probably related with the capability of the oviduct to expel the egg or the large egg size that makes oviposition more difficult (Figure 3).

The lesions observed showed a time distribution pattern. Metabolic diseases like fatty liver (10%) (Figure 6), urate deposit (9%) (Figure 7), ascites and enlarged heart (5%) (Figure 8), were predominant during the late production period due to the normal aging process and the high metabolic rate that these breeders were subjected throughout all the production period (Figure 4).

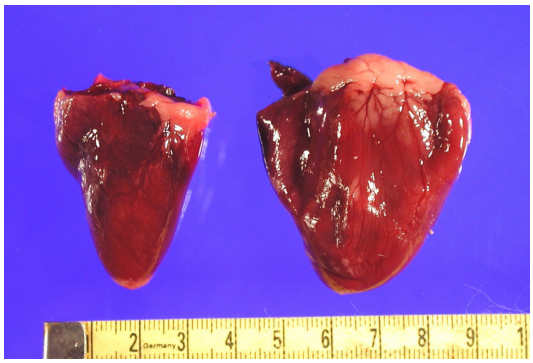

Figure 8 – Enlarged and normal hearts (only ventricles) of two 50 week old hens. Note size difference between normal heart (left) and dilated (right).

Bumble foot and joint lesions were predominant during the late part of production and are a common disorder. Being in contact for weeks with shavings and ammonia can increase the prevalence of joint problems. Folds on foot pads are reservoirs for litter bacteria and lack of uniformity on hens determine the presence of injuries due to running and stress. Staphylococcus infection seems to be the main cause, as this bacterium is capable of invading not only foot pads but also joints, liver, ovary and coelomic cavity, Pizarro *et al.* (2003). No leg malformations were found on post-mortem examination (Figure 4).

CONCLUSION

Understanding the lesion patterns and normal mortality during the production period of a broiler breeder flock, could assist technical staff in determining if there is a pathological problem or if the mortality is part of the normal evolution of the flock. This knowledge can also help veterinarians and avoid the use of unnecessary treatments or drugs for 'normal' mortality patterns.

Ovaritis was observed to have a higher prevalence, especially at onset lay. Once the hen reached peak production, and the stressful situation of feeding

program was gone, lesions associated with weight gain and metabolic failures (joint, hepatic and renal diseases) acquired importance.

The average bodyweight of dead hens was slightly below the standard production, but was not related to the mortality pattern. High liver and heart weights were associated with fatty liver and enlarged heart diseases respectively.

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