

## MANAGEMENTAL AND NUTRITIONAL DISEASE - SUDDEN DEATH SYNDROME IN BROILERS

**\*P.V. Meshram and Febi Bijoy**

Department of Pathology, Bombay Veterinary College, Parel, Mumbai-400 012

E-mail: pramodvetpath@gmail.com (*\*Corresponding Author*)

**Abstract:** Sudden death syndrome (SDS) is a condition in which healthy fast growing broilers chicks die suddenly from no apparent causes. It has developed into a major problem to the broiler industry in many parts of the world. Broilers of all ages are affected starting as early as 2 days of age and continuing through to market age. Peak mortality usually occurs between 3 and 4 weeks of age and males are more affected than the females. There is usually a short wing beating, convulsions prior to death, so that the majority of affected broilers are found dead lying on their backs. As a result, the condition often been referred to as "Flip-Over Disease". Lung edema is a prominent PM lesions. There is no proper treatment and preventive measures for control of SDS, but incidence can be reduced by management techniques. The causes of the Sudden Death Syndrome include; Managemental factors, Nutritional factors, Diet Composition and Role of Prostaglandins. Metabolic disorders affect internal body metabolism and development and are the cause of the large proportion of mortality in commercial poultry flocks especially. One of the main factors affecting these diseases is rapid growth rate.

**Keywords:** Metabolic disorders, Sudden Death Syndrome, Poultry broiler, Ascites, Mortality.

### Introduction

In the poultry industry, Broilers are raised on a highly concentrated energy diet to maximize growth rates and reduce the total number of days needed to reach market weight. Intensive production practices are implemented to maximize economic returns in the poultry industry. Regulating the feed intake of broiler chickens is not always practiced although the intense growth rate causes stress on the broilers and can result in metabolic diseases and skeletal disorders. Air quality, stocking densities and feed/water availability were not only harm the welfare of the broilers, but result in economic losses due to reduced animal performance, mortalities, and condemnations at slaughter plants. Amongst such diseases, one of the important diseases affecting the broiler industry is SUDDEN DEATH SYNDROME.

Sudden death syndrome has developed into a major problem to the broiler industry in many parts of the world. Broilers of all ages are affected starting as early as 2 days of age and continuing through to market age. Sudden death syndrome (SDS), also known as 'flip-

overDisease' or 'acute death syndrome', is a disease of young and fast-growing broiler chickens especially male which die suddenly and occasionally on their back, hence the name flip-over disease. It is a metabolic disease may be due to genetic, environmental and nutritional factors. Broilers of all age groups, as early as 2 days of age are affected. There is no proper treatment and preventive measures for control of SDS, but incidence can be reduced by management techniques.

### **Related factors**

A large number of factors have been studied with a view to isolate causative agents in SDS. Most studies have involved diet texture and composition. Hulan *et al.*, (1980) suggested that there are numerous nutritional and physiological factors which may lead to SDS. The level and type of fat (higher for saturated than for unsaturated) in the diet may be involved, the fact that death is apparently due to heart failure may suggest the involvement of electrolytes (Na+<-, K+,Cl-); sudden noise and high intensity lighting appears to increase the incidence of SDS. The possible factors which may be responsible for the occurrence of Sudden death syndrome are as follows:

### **I. Managemental Causes**

#### **a) Lighting**

A period of short day length during early growth has recently been shown to be beneficial in reducing SDS in birds. Classen (1991) indicates a 30-60% reduction in incidence of SDS where the broilers were subjected to only 6h light from 3-14 d or various incremental programs of 6h increasing to 23 h over the 3-35 d period. Early growth rate is reduced because birds have less time to eat, and this beneficial in reducing SDS. Blair *et al.*, (1993) recorded a comparable significant reduction in SDS using trials involving around 2500 birds.

#### **b) Stocking density**

Broilers chickens are generally reared at a considerably higher stocking density. Such rearing conditions may act on the birds as a stress that causes functional disorders in their organs including the heart (Kaul and Trangadia 2003).

### **II. Nutritional Factors**

#### **a) Diet texture**

In broilers pelleted feed is extensively used. It has many advantages. It reduces bulkiness, minimizes wastage, toxin is destroyed while pelleting and processing and it has higher digestibility as compared to mass. Due to pelleted feed there is faster growth rate hence incidence of SDS and ascites are more in broilers (Kaul and Trangdia, 2003). Proudfoot *et*

*al.*, (1984) assumed that there was a pelleting factor influencing SDS that was independent of body weight, and that these factors developed in the feed during the steam and pressure treatment employed during pelleting. In testing this theory, various diet ingredients by-passed the pelleting process, and so were added as "mash" ingredients to the other pelleted diet components. When all the protein-rich ingredients (soybean meal, fish meal and canola meal) by-passed pelleting, there was a significant reduction in SDS of male broilers to 49 d (3.6 vs 0.9%). While **Proudfoot** *et al.*, (1984) conclude that toxic substances are produced by pelleting protein ingredients, and that these predispose to SDS, again it must be emphasized that reduction in SDS in this trial was associated with reduced growth rate (2160 vs 1950 g at 49 d).

### **III. Diet composition**

Because SDS occurs only in fast-growing birds that are assumed to be eating to near physical capacity, Bowes *et al* (1988) investigated the effect of physical feed restriction and no such mortality occurred in the feed-restricted birds. Julian and Lesson (1985) reported some interesting effects of using different carbohydrates sources in a diet. Mortality due to SDS was more than doubled to 6% SDS when glucose was the predominant energy source compared to 2.1% mortality when fat was a major contributor or 2.5% when corn starch was used.

Blair *et al.*, (1990) conducted studies to investigate field reports of higher SDS in birds fed wheat rather than corn. In controlled studies, wheat-based diets did produce more SDS, although as a proportion of total mortality there was no diet effect. In two other trials, either the incidence of SDS or its proportion of total mortality was reduced when meat meal was included in the diet. Blair *et al.*, (1990) conclude that meat meal supplies some previously unidentified factor that provides protection against SDS. Biotin has perhaps been singled out most frequently as a possible factor in SDS. Steele *et al.*, (1982) showed no benefit from supplying up to 100 µg/d (via the drinking water) within a commercial flock of 60,000 birds to 49 d. A liver analysis confirmed an increased biotin uptake by the birds, although the authors conclude that there is little if any association between normal biotin status and SDS. Whitehead and Randall (1982) made similar conclusions, although suggested that biotin may be a factor if SDS is complicated by the incidence of fatty liver and kidney syndrome (FLKS). A diet which is marginally deficient in the vitamin biotin may cause sudden unexpected death of young broiler chickens when they are exposed to stress. Chickens affected with this disorder have low levels of biotin in their livers (Johnson et al 1980).

#### **IV. Lactate metabolism**

Lactate accumulates when there is inadequate oxygen to fuel the normal aerobic metabolism and  $\text{NAOH}_2$  is not oxidized. Production of lactate rather than pyruvate, due to anoxia, leads to acidosis. Weil and Abdelmonen (1970) developed a relationship between blood lactate levels and the probability of survival, indicating that one hundred percent mortality is likely when lactate levels reach 10 times normal. Weil and Abdelmonen (1970) suggest that survival is related to the irreversibility of the metabolic disturbance which occurs during acidosis, and that  $\text{O}_2$  intake during recovery does not always lead to survival. This scenario is seen with SOS birds that are revived during an attack, where their ultimate survival is unpredictable.

Summers *et al.*, (1987) reported that on SDS type death, 100 per cent incidence could be induced in broilers by injecting 20 per cent lactic acid solution into the wing vein. Pipetting 5 ml. of same lactic acid solution into the crop had less consistent effect but gave high SDS mortality in general and concluded that broilers receiving diet high in glucose "flipped" within 30 minutes of dosing with lactic acid, while those receiving a diet high in corn-starch took over 1.5 hours to flipped.

#### **Pathogenesis**

Stress is the main factor to contribute towards the pathogenesis of SDS. Ononiwu *et al.*, (1979a) explained that when the course of a disease is acute, most of the pathological lesions are associated with vascular disturbance. The process starts with circulatory lesions manifested by increased permeability of the peripheral circulatory system. Under the influence of physiological stresses even a healthy capillary may become permeable. This physiological permeability, caused by short-term increases in blood pressure, is usually reversible. However, when the stimulus surpasses the tolerance level, irreversible changes occur not only in the wall of the blood vessel, but also in the tissue which they supply. In SDS death would appear to be caused by heart damage which leads to lung edema so that the chickens are unable to breath. Sufficient fluid is lost from the circulatory system into the lung tissue spaces to result in peripheral circulatory failure or shock. The histological changes of intense congestion and edema in the lungs result in the tissue parenchyma becoming separated from fresh blood supply leading to hypoxia. The observation of shrunken gall bladder in 100% of the cases of SDS has not been reported before. Such gall bladders may indirectly confirm field observations that the broilers dying of SDS eat normally right up to

the time of death, since the physiological function of the gall bladder is to contract and supply bile rapidly to the intestine during the process of digestion. Vascular congestion is a constant feature of most of the tissues examined microscopically, particularly in the lungs where much of the effective air spaces were lost because of engorgement of pulmonary capillaries. Lymphocytic infiltration and inflammation involving the secondary bronchi and the presence of edema in the alveoli considerably reduce gaseous exchange and enhance respiratory distress.

### **Post mortem lesions**

Ononiwu *et al.*, (1979a) found that birds are in good health and flesh. The liver and kidneys are slightly enlarged and the latter have patchy areas of sub capsular hemorrhage. Lungs are often congested and oedematous. The ventricles of the heart are generally contracted and the thyroid, thymus and spleen congested. There may be hemorrhages in the kidney. Feed is present along the entire digestive tract particularly the crops and gizzards were full of recently ingested feed. Intact food particles are present in gizzard. Gall bladder is empty. The heart contains clotted blood in the atria which are of postmortem in origin but the ventricles are often empty and the left ventricle in particular assumes a hypertrophied appearance. Necropsy reveals the presence of a structure within the blood of the heart. Histologically these structures have been identified as "Jelly clots" or chicken fat clots both of which are PM in origin.

### **Microscopic lesions**

Lung:- Varying degree of vascular engorgement. There is presence of RBCs and edema of intestinal and interlobar connective tissue.

Liver:- At the portal triad, infiltration of leucocytes, there is distortion and reduction of lumen of bile duct.

Heart:-degeneration of fibers, separation of cardiac muscle fibers by edema and infiltration of heterophils.

Kidneys:- have subcapsular and parenchymatous hemorrhage.

### **Diagnosis**

Diagnosis is based on the history of sudden death (within one minute by violent wing flapping, convulsion and death) gross and microscopic pathology and no evidence of other disease. Riddell and Springer (1985) reported that diagnosis of SDS was made if the birds were well fleshed with congested lungs, as small gall bladder and no evidence of other disease. Diagnosis is based on the history of sudden death within one minute by violent wing

flapping, convulsion and death. Blood profiles and tissue analysis has little role in confirmative diagnosis.

### **Prevention and treatment**

There is no one treatment and preventive measures for control of SDS.

1. Condition is related to early faster growth rate. So, such management techniques should be used to reduce early maximum potential for growth.

Broilers are most commonly fed diets in pelleted form to maximize feed consumption. Broilers fed the same diets in crumble form consume less. Producers can slightly alter growth rates by temporarily substituting pelleted diets with crumble form early in production.

2. Use diets with 5-7 per cent reduction in nutrient density thereby tampering early fast growth rate up to 18-20 days which reduces incidence of SDS.

3. Feed a low protein/ low energy diet during first 14 days to lead reduced oxygen demand in growing broilers.

4. Protein supplements with soyabean meal, canola meal and fish meal which are not pelleted decrease incidence of SDS.

5. All birds can be subjected to restricted feed up to 8-10%, and feed to twice daily only. Supplementation with glucose containing electrolyte, liquid toxin binders, immunomodulator, and simple broad-spectrum antibiotics has to be provided in water. (Kedar Karki *et al.*, 2009)

### **References**

- [1] Bowes, B.A.; R.K. Julian; S. Lesson and T. Stirtzinger (1988): Effect of feed restriction on feed efficiency and incidence of SDS in broiler chickens. *Poultry Sci.* 67: 1102-1104.
- [2] Blair, R., J.P. Jacob and E.E. Gardiner, 1990. Effect of dietary protein source and cereal type on the incidence of sudden death syndrome in broiler chickens. *Poult. Sci.* 69: 1331-1338.
- [3] Blair, R., R.C. Newberry and E.E. Gardiner, 1993. Effects of light pattern and dietary tryptophan supplementation on growth and mortality in broilers. *Poult. Sei.* 72:495-502.
- [4] Classen, H., 1991. Increasing photoperiod length provides better broiler health. *Poultry digest.* June 1991, pp.15-28.
- [5] Hulan, H.W.; F.G. Proudfoot and K.B.M. MacRae (1980): Effects of vitamins on the incidence of mortality and ADS ("Flip-over") in broiler chickens. *foultry Sci.* 59: 927-931.
- [6] Johnson, A. R.; Hood, R.L and J.L. Emery (1980): Biotin and the sudden infant death syndrome. *Letters-Nature* 285, 159-160 (15 May 1980)
- [7] Julian, R.J. and S. Leeson (1985): The effect of diet on Sudden Death Syndrome in

broiler chickens. Proc. Poultry Industry School, Guelph Agricultural Conference, Guelph, Ontario, Canada.

[8] KaulLalita and Trangadia (2003): Sudden death syndrome in broilers. *Pashudhan*, April: PP.1. Mollison, S.; W. Guenter and B. R. Boycoll (1984):

[9] Kedar, K.: Poornima M., Banshi S, Salina M,: Pragya K (2009) Mycosis in Broiler Chicken: Outbreak of Sudden Death Syndrome in Nepal(journal?)

[10] Ononiwu, J.C., R.G. Thomson, H.C. Carlson and R.J. Julian, 1979a. Pathological studies of sudden death syndrome in broiler chickens. *Can. Vet. J.* 20:70-73.

[11] Proudfoot, F.G., H.W. Hulan and K.B. McRae, 1984. The effects of dietary micronutrient, fat and protein components in pel/eted feeds on the incidence of sudden death syndrome and other traits among male broiler chickens. *Can. J. Anim. Sci.* 64:159-164.

[12] Riddell, C. and R. Springer (1985): An epizootiological study of acute death syndrome and leg weakness in broiler chickens in Westem Canada. *Avian Dis.* 29 : 90-102.

[13] Summers, J. D.; M. Bedford and D. Spratt (1987): SDS : is it a metabolic disease? *Feed stuff.* 59: 20.

[14] Steele, P., J. Edgar and G. Doncon, 1982. Effect of biotin supplementation on incidence of acute death syndrome in broiler chickens. *Poult. Sci.* 61 :909-913.

[15] Weil, M.H. and A.A. Abdelmonen, 1970. Experimental and clinical studies on lactate and pyruvate as indicators of the severity of acute circulatory failure. *Circulation* 41 :909.

[16] Whitehead, C.C. and C.J. Randall, 1982. Interrelationships between biotin, choline and other B vitamins and the occurrence of fatty liver kidney syndrome and sudden death syndrome in broiler chickens. *Br. J. Nutr.* 48:177-184.