Sudden Death Syndrome – An Overview

Siddiqui, M. F. M. F.*, M.S. Patil, K.M.Khan, and L.A.Khan

Post Graduate Institute of Veterinary and Animal Sciences,
MAFSU, Akola (M.S.)
* Corresponding author Email: mfmf@rediffmail.com

Abstracts

Metabolic disorders affect internal body metabolism and development and are the cause of the large proportion of mortality in both commercial and backyard poultry flocks. One of the main factor affecting these diseases is rapid growth rate. Two of the more important types of metabolic disorders are the "Sudden Death Syndrome" and Ascites. Sudden death syndrome (SDS) is a condition in which apparently healthy fast growing broilers chicks die suddenly from no apparent causes. 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". 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. Peak mortality usually occurs between 3 and 4 weeks of age. Males are more affected than the females. 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 includes; Managemental factors, Nutritional factors, Diet Composition and Role of Prostaglandins.

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

Incidence

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. Peak mortality usually occurs between 3 and 4 weeks of age. (Gardiner *et al.*,1988)

The syndrome has been reported to cause between 1.31 and 2.46per cent mortality with males more commonly affected than females (Riddell and Orr, 1980), however proudfoot and Hulan (1982) reported 0.90 to 3.61 per cent mortality due to SDS in broilers. From 0.71 to 4.07 per cent as reported by Riddel and Springer. (1985) where as Ononiwu *et al.* (1979) reported 1.0 per cent mortality due to SDS is broilers. Brigden and Riddell (1975) reported that 70-80 per cent of male mortality and 20-25 per cent of female broilers chickens mortality could be attributed to acute death syndrome or "Flip-Over Diease".

Thus, SDS is a leading contributor to mortality in broiler chicken production. Although the precipitating event has yet to be ascertained," Cardiovascular failure" appears to be the immediate cause of death.

Etiology

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 presence of blood clot in the heart may suggest that level or absence of certain vitamins in the diet might be involved also suggest the evidence of genetic variation with regards to the incidence of SDS. Thus, we can categorised the possible etiological factors involved in SDS as under.

I. Managemental Causes

a) Lighting

Ononiwu *et al.* (1979) hypothesized of that cannibalism, excitement, fighting and pilling induced by light intensity above optimum would place broilers under stress and implied that these could lead to death from sudden death syndrome, where as Caves (1981) found no significant difference in a similar test. Hulan *et al.* (1980) stated that sudden noise and high intensity light appears to increase the incidence of SDS.

Under low light intensity Dateon *et al.* (1976) observed better broiler performance because the chickens are less active and therefore waste less

energy in exercise. No significant effect of light treatment on feed conversion and mortality was reported by Darminey and Nakalle (1977).

Riddell and Springer (1985) found no correlation between mortality due to SDS and light intensity, whereas Newberry *et al.* (1985) observed that chickens were no more likely to die from SDS in area of relatively high light intensity than in darker areas.

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).

Stress: (Lighting and stocking density)

- Catecholamine (Adrenal galnd)
- Increased Ca^{+} in cardiac muscles
- Cardiac arrhythmia

c) Exercise

Systolic, diastolic and mean blood pressures significantly decrease during exercise. Exercise and stress release catecholamines into circulation when combined with hypotension, severe congestion of abdominal organs would results in to SDS.

II. Nutritional factors

a) Diet texture

In broilers pelleted feed is extensively used. It has many advantages. It reduces bulkiness, minimizes, wastage, destroys toxin 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) proposed that one or more toxic factors were produced when protein supplements were subjected to pelleting and this may be involved in causing SDS. Thus, he speculated that the toxin must be produced when soyabean meal and not meat meal is pelleted, since reduction in the quantity of soyabean meal in the diet reduced the incidence of SDS. It is more likely, however that the inclusion of meat meal supplies a previously unidentified factor present in animal protein which provides some protection against the occurance of SDS.

III. Diet composition

Summers *et al.*(1987) reported that on SDS type death of 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.

Julian and Lesson (1985) observed that birds feed diet high in glucose are more likely to show SDS and recorded significantly higher blood lactate levels in the birds that died of SDS than survived. In the year 1990 Jacob and Blair, evaluated the effect of deitary lactate and glucose on the incidence of SDS in male broilers chickens by using four levels of dietary lactate (0, 2.5, 5.0, 7.5 % calcium lactate) and four levels of dietary glucose (0, 15, 30, 45 % cerelose) in 1280 male broiler chickens at 4 weeks of age of recorded. Overall mortality to be 6.64 per cent of which 32.9 per cent was attributed to SDS and revealed no significant difference between treatment in either total mortality or mortality due to SDS.

Higher incidence of SDS in broiler chicks fed with wheat soyabean meal based diets than corn-soyabean meal based diets was recorded by Mollison (1983). Riddell and Springer (1985) conducted a survey of 51 broiler flocks in Canada and reported that the incidence was higher in flocks supplied by a feed company that use less corn and more wheat than other feed companies.

Blair and Jacob (1990) recorded significantly higher incidence of SDS for wheat fed birds while SDS as a percentage of the birds housed was reduced by the inclusion of meat meal in the diet, whereas, in brooder study, total mortality and the incidence of SDS were not affected by protein source but SDS as percentage of total mortality was reduced with the inclusion of meat meal in the diet.

Additional biotin (Hulan *et al.,* 1980) provided by corn or wheat may have contributed to the decrease mortality found with corns diet although the biotin requirement should have been met by the amount of premix added to each diet. Significantly lower incidence of SDS ($P<0.05$) in 29 to 56 days broiler chickens fed with 24 per cent protein finisher diet as compared to birds fed a 19 per cent finisher diet was recorded by Mollison and Guenter (1984).

Hunt and Gardiner (1982) compared various dietary factors (Wheat based Vs Corn based diets, supplemented K and supplemented biotin, Pyridoxine and Thiamin) and reported that neither total mortality nor mortality attributed to flip over was affected by diet.

Lactate metabolism

Relationship between blood lactate level and the probability of survival indicates that 100 per cent mortality is likely to occur when lactate level reaches 10 times higher than normal.

1. Broilers are fast growing birds having a large proportion of muscles compared to visceral organs like heart, lung and kidney which are not proportionally developed leading to inadequate supply of oxygen resulting in hypoxia and lack of aerobic metabolism

and utilization of $NADH_2$ in glycolytic pathway.

2. Production of lactate from pyruvate in presence of lactic acid dehydrogenase give rise to increase production of lactic acid resulting into systemic acidosis, change in blood pH, cardiovascular system disturbances leading to cardiac arrhythmia.

3. Lactic acid is also a fermentation product in the crop. It is observed that there is marked increase in lactic acid in cockerels which are 6 hours starved. If high protein diet containing excess level of sulphur is given to the birds, increased acid secretion results in higher level of lactic acid causing the condition SDS.

IV. Role of prostaglandin in SDS

Prostaglandin regulates the flow of blood and transmission of nerve impulses to visceral organs. In SDS, birds heart tissue reveals lower amount of linoleic acid and arachidonic acid hence decrease in the synthesis of prostaglandin which leads to deterioration of membrane structure and cardiovascular disturbances causing cardiac arrhythmia, heart function failure and increase the incidence of SDS (Kaul and Trangdia, 2003).

Pathogenesis

Stress is the main factor to contribute towards the pathogenesis of SDS.

Stress (lighting, stocking density, exercise, nutrition)
- Normal capillaries become permeable
- More lactic acid - Short term increase in blood pressure
- Less prostaglindin - Affect circulatory system
- Cardiac arrythmia - Heart function failure
- Cardiovascular failure - Lung edema
- Hypoxia - Death

Correlation between SDS and Ascites

SDS and ascites are metabolic diseases. Both have common etiological aspects. Males are more affected. They are different degree of metabolic condition but cardiac involvement and edema are common in both the conditions. When condition is acute results in SDS and if condition is chronic it results in ascites.

Clinical signs

1. Birds prior to death feeding, drink and walk normally. Then they extend their neck squawk and beat wings. Death occurs within minutes.

2. Newberry *et al.* (1987) studied behavioural changes in broilers by using videocamera. All affected birds exhibited a sudden attack prior to death characterized by loss of balance, violent wing flapping and strong muscular contraction. The apparent seitzure usually lasted just under one month and most of birds gave some type of squawk or high pitched cry during the attacks.

Biochemical changes: Biochemical changes that may accompany or precede SDS are not well documented; however, Rotter *et al.* (1985) reported calcium concentration of hard tissues was significantly lower in broilers succumbing to SDS then for broilers called from the same flock. Variation in herd calcium levels may be significant in view of the role calcium ions play in muscle contraction.

PM findings

Necropsy reveals the presence of a structure within the blood of the heart. Histologically this structure have been identified as "Jelly clots" or chicken fat clots both of which as PM in origin.

Lung :- Edema of lung

Kidney and liver :- Slightly congested and subcapsular petechial hemorrhage. Heavier liver with fatty infiltration.

Heart :- Firmly contracted with enlargement, ventricles were empty. Atria filled with blood clots. Clot was considered as post-mortem origin.

Crop and Gizzard :- Full of recently ingested food.

Gall bladder :- Empty, indicates birds prior to death eaten up food.

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 myofibrils, leukocytes infiltration which was lymphocyte and heterophils. There is edema which leads to separation of myofibrils.

Diagnosis

Riddell and Springer (1985) reported that diagnosis of SDS was made if the birds was 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

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

1. Condition is related to early faster growth rate. So, such management techniques are used to reduce early maximum potential for growth.
2. Use diets with 5-7 per cent reduction in nutrient density which tampering early fast growth rate upto 18-20 days. which reduces incidence of SDS.
3. K salts in feed given to broiler so incidence of SDS is lower (Ropkinson, 1983).

4. Wheat-soy diet supplemented with sunflower oil improved performance and reduces incidence of SDS (Flotter, 1985).
5. Protein supplements with soyabean meal, canola meal and fish meal which are not pelleted decrease incidence of SDS.
6. Dietary fat restriction from 0-7 days initially reduced growth rate and increased feed gain ratio.

   Lowering the energy : Protein ratio of finishing diet significantly increase feed gain ratio between 29 and 49 days and reduced fat pad size and incidence of SDS was significantly lower from 29 to 56 days of age.
7. Using quantitative feed restriction to decrease mortality. Feed restriction at 30-40 per cent for 7 days lowering the incidence of SDS mortality (Bowes *et al.,* 1988).
8. Feed a low protein/ low energy diet during first 14 days to lead reduced oxygen demand in growing broilers.

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. Brigden, J. L. and C. Riddell. (1975): A survey of mortality in four broiler flocks in Western Canada. *Can. Vet. J.* 16 : 194-200.
3. Cave, N. A. (1981): The effect of intermittent light on carcass quality, feed efficiency and growth of broilers. *Poultry Sci.* 60 : 956-960.
4. Deaton, J. W.; F. N. Reece; L. E. Kubena and J. D. May (1976): effects of varying light intensity on broiler performance. *Poultry Sci.* 55 : 515-519.
5. Dorminey, R. W. and H. S. Nakaue (1977): Intermittent light and light intensity effects on broilers in light proof pens. *Poultry Sci.* 56 : 1868-1875.
6. Gardiner, E. E.; J. H. Hunt; R. C. Newberry and J. W. Hall (1988): Relationship between age, body weight and season on incidence of SDS. *Poult. Sci.* 67 : 1243-1249.
7. Hopkinson, W. L.; W. Williams; G. L. Griffiths and D. Josesop (1983): Dietary induction of SDS in broilers. *Avian Dis.* 28(2) : 352.
8. 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. *Poultry Sci.* 59 : 927-931.
9. Jacob, J. P.; R. Blair and E. Gardiner (1990): Effect of dietary lactate and glucose on the incidence of SDS. *Poult. Sci.* 69(9) :1529-32.
10. 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.
11. Kaul Lalita and Trangadia (2003): Sudden death syndrome in broilers. *Pashudhan*, April: PP.1.
12. Mollison, B.; W. Guenter and B. R. Boycott (1984): Abdominal fat deposition and sudden death syndrome in broilers. The effect of restricted intake, early life caloric (fat) restriction and caloric : protein ration. *Poultry Sci.* 63 : 1190-1200.
13. Newberry, R. C. (1987): Behavioural study of chickens prior to death from SDS. *Poult. Sci.* 66 : 146-47.
14. Ononiwu, J. C.; R. G. Thomson; H. C. Carison and R. Julian (1979a): Pathological studies of SDS in broilers. *Can. Vet. J.* 20 : 70-73.
15. Ononwu, J. C.; R. G. Thomson; H. C. Carison and R. Julian (1979b): Studies on the effect of lighting on SDS in broiler chickens. *Can. Vet. J.* 20 : 74-77.
16. Produfoot, F. G. and H. U. Hulan (1982): Effects of reduced feeding time using all mash or crumble pellet dietary regimens of chicken broiler performance, including ADS. *Agri. Canada* 61 : 750-754.
17. Riddell, C. and R. Springer (1985): An epizootiological study of acute death syndrome and leg weakness in broiler chickens in Western Canada. *Avian Dis.* 29 : 90-102.
18. Summers, J. D.; M. Bedford and D. Spratt (1987): SDS : is it a metabolic disease ? *Feed stuff.* 59 : 20.

* * * * * * * *