Sudden Death Syndrome in chicken: A review

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Abstract: Sudden death is a condition in apparently healthy fast growing broilers. Chickens die suddenly without any previous signs. Broilers of all ages are subjected to sudden death starting as early as 2 days of age and up to market age. Sudden death may occur due to infectious or non infectious causes. Infectious causes include viral and bacterial causes are the most predominant. Also, Mycotoxicosis may cause sudden death due to ingestion of ration contaminated with mycotoxins**.** Among non infectious causes, nutrition, management and high intensity and lighting which appear to increase the incidence of Sudden death. This condition is characterized by a short wing beating convulsions prior to death and majority of affected broilers are found dead lying on their backs, Condition often been referred as "Flip-Over Disease". This condition causes major economic losses in broilers industry worldwide for many years. The mean mortality rate due to sudden death syndrome was 3 - 9 % and peak mortality usually occurs between 3 and 4 weeks of age. There is no proper treatment and preventive measures for control of sudden death syndrome but incidence can be reduced by massive vaccination program, hygienic measurements and managemental techniques.

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Introduction

Sudden death may occur due to several infectious and non infectious causes. Among infectious causes viral, bacterial and mycotic causes are the most predominant, (**Peiris *et al.*, 2007).** The major viral causes of sudden death are *influenza virus A*, Avulavirus **or** Avibirnavirus **(Julian, 2005)**.

The main bacterial causes of sudden death include clostridium. Avian cholera **(Kuhnert and Christensen, 2008),** or *salmonella typhmurium* **(Richardson *et al.*, 2003).** Also, Mycotoxicosis may cause, sudden death due to ingestion of ration contaminated with mycotoxins **(Zaki *et al.*, 2012).**

Sudden death causes world-wide great economic losses in the broiler industry for many years **(Olkowski *et al.*, 2008).**

1. Causes of Sudden Death in broilers

Sudden Death in broilers may occur due to several causes, among which, bacterial, viral,, mycotic and multi-factorial causes. Age, immune status, nutritional and Managemental factors.

Infectious causes:

1-Viral Causes.

a-Avian influenza (AI):

Influenza ***A*** viruses are classified into subtypes based on two surface antigens, the hem agglutinin (H) and neuraminidase (N) proteins. There are 16 hemagglutinin antigens (H1 to H16) and nine neuraminidase antigens (N1 to N9). **(Saif *et al.*, 2003)**.

Avian influenza viruses AIV mainly infect birds. In wild species, these viruses are especially common among birds that live in wetlands and other aquatic environments. Waterfowl and shorebirds appear to be the natural reservoirs for the influenza A viruses, and carry all of the known subtypes **(Swayne *et al., 2006)***.

The incubation period in poultry is one to seven days. Occasionally a virus can replicate and be transmitted in the new host species and a permanent jump is made **(Brooks *et al.*, 2009**).

In birds, AI virus are shed in the feces as well as in saliva and nasal secretions. Fecal transmission is facilitated by the persistence of AIV in aquatic environments for prolonged periods particularly at low temperatures **(Desvaux *et al.*, 2009)**.

Virus isolation is performed in embryonated eggs; hemagglutinating activity which indicates the presence of influenza virus. Serological tests including agar gel immunodiffusion, hemagglutination, he magglutination inhibition and ELISAs are useful as supplemental tests. RT-PCR assays can identify AIV in clinical samples, and can replace virus isolation in some cases (**Balish *et al.*, 2010).**

b-Newcastle disease (ND):

ND viral strains are differ according to the degrees of pathogenicity (e.g. velogenic, mesogenic and lentogenic strains). Infected birds showed gastrointestinal, respiratory and nervous signs, with mortality up to 100%, depending upon pathotype of the virus (**Romer *et al*., 2006).**

Infection may inhalation or ingestion and that spread from one bird to another depends on the availability of the virus in an infectious form the gross lesions and the organs affected in ND virus infected birds are dependent on the strain and patho type of the infecting virus, in addition to the host and all the other factors that may affect the severity of the disease (**Alexander, 2000)**.

The pathogenicity of ND is determined chiefly by the strain of virus, although doses, route of administration, age of the chicken, and environmental conditions all have an effect. **(Alexander *et al.*, 2004)**.

Immunohistologic techniques offer a rapid method for the specific demonstration of the presence of virus or viral antigens in organs or tissues. lnununofluorescence techniques for thin sections of trachea or impression smears **(Huang *et al*.,2004)**.

Single radial hemolysis, agar gel precipitin Enzyme-linked immunosorbent assays (ELISAs), Good correlation has been reported between ELISA and HI tests**. (Alexander, 2001).** Timing of vaccination of broiler chickens can be especially difficult due to the presence of maternal antibodies. Because of their short life, broiler chickens are sometimes not vaccinated in countries where there is a low risk of ND**. (Aldous *and Alexander,* 2001)**

c-Infectious bursal disease (IBD):

Infectious bursal disease (IBD) is an acute and highly contagious immunosuppressive disease in young chickens. It causes high mortality and immunosuppressant in susceptible birds, leading to a variety of secondary infections and a decreased response to vaccinations, which results in an important economic effect on the poultry industry worldwide. **(Abdel-Alim and Saif, 2001)**

The IBD virus has a worldwide distribution. It is resistant to a variety of disinfectants and is environmentally very stable, which accounts for its persistence in poultry houses (**Rubinellip and LIN, 2007)**

In birds that die at the peak of the disease outbreak, the bursa is enlarged and turgid with a pale yellow discoloration. Intrafollicular haemorrhages may be present and in some cases, the bursa may be completely haemorrhagic giving the appearance of a black cherry **(Abdel-Alim and Saif, 2001 and Habib *et al.*, 2006).**

The highly virulent strains of the virus cause a decrease in thymic weight and severe lesions in other lymphoid tissues. Lymphocyte necrosis is the most common histological lesion and is accompanied by edema, Hyperemia and accumulation of heterophils. Cystic cavities replace lymphocytes in follicles and later there is some regeneration of lymphocytes. Other lesions include dehydration and hemorrhages in the leg, thigh, and breast muscles **(Al-Natotur *et al*., 2004 and Kim *et al.* 2004)).**

Immunization of chickens with attenuated live or inactivated IBD virus vaccines is the principal method used for the control of disease. Live vaccines provide broad protection but also a proportional risk of reversion to virulence, whereas inactivated vaccines, although costly, are considered safer and are used successfully (**Gould *et al.*, 2001).**

2-Bacterial causes:

a-Gram positive bacteria: Clostridium perfringens:

Clostridium perfringens (C. perfringens) is a Gram-positive spore forming anaerobic bacterium that is commonly found in the environment and in the gastrointestinal tract of birds and humans as part of the normal gut microbiota (**Palliyeguru *et al.,* 2010**).

C. perfringens strains are classified into five types (A to E) on the basis of the production of four major toxins known as the alpha, beta, epsilon and iota toxins **(Younes *et al*., 2005)**. Although C. perfringens type A is also associated with necrotic enteritis (NE) in broilers **(El-Meneisy *et al*., 2007)**. The disease can occur in two forms; it may present as acute clinical disease or as sub-clinical disease**. (Saif *et al*., 2003; Olkowski *et al*., 2008**, **Lovland *et al*., 2004).**

C. perfringens is unique not only in terms of the number of toxins produced, but also in terms of their toxicity and lethal activity (**Vasfi Marandi, 2010).** *C. perfringens* toxigenic strains were isolated from both diseased and healthy chickens (**Saif *et al.,* 2003 and Timbermont *et al.,* 2009**).

The final identification of *C. perfringens* was carried out according to **Nauerby *et al. (2003)*** depending on: Microscopic appearance, Colonial morphology, Motility, hemolytic activity, gelatin hydrolysis, fermentation of Glucose, lactose, sucrose, maltose, salicin and mannitol, indol test, urease production, litmus milk test and lecithinase production.

Antibiotic growth promoters (AGPs) as lincomycin, virginiamycin, bacitracin, avoparcin, avilamycin in-feed have been long played an incidental part in clinical NE suppression, but now their use is being questioned in many countries. AGPs are effective against Clostridiosis by reducing numbers of *C. perfringens* shed in feces of chickens **(Devegowda*. and Murthy,* 2005**).

b-Gram negative bacteria:-

Salmonella Typhimurium:

Salmonella is a Gram negative bacterium that can survive and multiply in the environment as a result of fecal shedding. **(Poppe, 2000 and Lovland, *et al.*, 2004**).

Chickens are the natural hosts for both *Salmonella Gallinarum* (*S. Gallinarum) and Salmonella Pullorum (S. Pullorum).* cases of fowel Typhoid (FT) and Pullorum Disease (PD). Chicks may die in the earlystages of brooding without exhibiting any gross lesions. These include moribund and dead birds in the incubator or shortly after hatching if the chicks and poults are hatched from infected eggs. **(Byrd *et al.,* 2003 and McCrea *et al.*, 2005).** In peracute cases of FT and PD, chicks may die in the early stages of brooding without exhibiting any gross lesions. Both morbidity and mortality can be highly variable due to various factors such as age of the bird, strain of the bird, nutritional status of the bird, flock management and concurrent infections. Mortality can range from 0% to 100%, especially in chicks and poults. The greatest mortality is seen during the second week after hatching, with a rapid decline between the third and fourth week of age. Morbidity is generally higher than mortality

Both FT and PD can be detected serologically by use of a macroscopic tube agglutination test, rapid serum test, stained antigen whole blood test or microagglutination test

Both S. Gallinarum and S. Pullorum grow readily on beef agar or broth or other nutrient media. The bacteria are aerobic or facultative anaerobic and grow best at 37C. The organisms will grow in selective enrichment media including selenite Fand tetrathionate broths, and on differential plating media including MacConkey, bismuth sulphite and brilliant green (BG) agars.*S. Gallinarum* and *S. Pullorum* can be controlled and eradicated by use of serological testing and elimination of positive birds. Vaccines may be used to control the disease and antibiotics for the treatment of FT and PD. Although FT and PD are widely distributed throughout the world, the diseases have been eradicated from commercial poultry in developed countries such as the United States of America, Canada and most countries of Western Europe. Both *S. Gallinarum* and *S. Pullorum* are highly adapted to the host species, and therefore are of little public health significance **(Saif *et al.,* 2003)**.

Pasteurella / Avian cholera (Pasteurellosis)*:*

Avian cholera is a contagious disease resulting from infection by *Pasteurella multocida*. Suspicion of avian cholera must be considered when a large number of dead animals are found in a short period of time. All domestic species are susceptible to the disease, Avian cholera outbreaks often manifest as acute fatal septicaemia **(McCrea *et al.,* 2005 and Ppopff, 2009)**.

Pasteurella multocida (*P. multocida)* is a gram-negative non-motile cocco-bacillus with bipolar staining. *P multocida* strains are classified into five capsular antigen types (A, B, D, E and F) and 16 somatic antigen types the organism may enter the body through the digestive tract or the respiratory system. The disease is not transmitted through the egg. **(Jamshidi *et al.,* 2008).**

All breeds. in all ages are suseptible but aged birds are more susceptable than younger birds. Acute pasteurellosis can result in death 6–12 h after exposure. Susceptibility to infection and the course of the disease depend upon many factors: sex, age, genetic variation, immune status, concurrent infections, nutritional status and strain virulence. **(Shivaprasad, 2000)**.

Pasteurella multocida (P. multocida) usually presenting a rapid septicemia with high mortality. *Pasteurella* grows on blood agar under aerobic conditions. **(Kuhnert and Christensen, 2008).**

Incubation period of *P. multocida* in naturally acquired infections is from 1-3 days. The course usually varies from per acute to sub acute. Peracute infections are characterized by sudden death, while acute cases showed swollen sinuses and wattles Untreated cases usually end fatally Mortality up to 100% due to stress condition **(Bastianello and Henton, 2004).**

Diagnosis depends on isolation and identification of the causative bacterium, Pasteurella multocida. Presumptive diagnosis may be based on the occurrence of typical signs and lesions and/or on the microscopic demonstration of myriad bacteria in blood smears, or impression smears of tissues such as liver or spleen. Pathological Change Abscess in the wattle, Hemorrhage in the duodenal mucosa (enteritis), Increase size of the liver and necrotic fuci in the liver are observed (**Jamshidi *et al.,*2008)**

the bird were treated with Sulphamethaxazole and Trimethoprim at the dose rate of 25 mg/kg body weight for five days, administered in drinking water. Clinical signs and mortality associated with fowl cholera were reduced from second day after the commencement of the treatment and birds completely recovered on the 4th day of treatment. (**Mbuthia *et al.*, 2008)**.

3-Non infectious causes

Mycotic infection:

Molds develop from spores that are found in the environment. Mold growth on grain under field conditions or during storage can occur at moisture levels above 16% and at temperatures above freezing. The growth of molds on grain can affect the

nutritional quality of grain in several ways. First, they decrease the nutritional value of the commodity as they consume fats, protein and carbohydrates that are present in the grain. Thus these nutrients are no longer available for the animal. Secondly, some species of mold are able to produce highly toxic compounds called mycotoxins**. (Zaki *et al*., 2012)**.

Mycotoxins comprise of a family of fungal toxins, many of which have been implicated as causes of toxicity in man and animals. There are four classes of mycotoxins of major concern namely aflatoxins, zearalenone, ochratoxins and fumonisins. Formation of mycotoxins varied between species as well as within a given species **(Fredic and Hoerr,2010).**

a) Aflatoxins:

Acute aflatoxicosis causes clinical disease marked by hepatitis, icterus, hemorrhage and death. Aflatoxin can cause oncogenesis, chronic toxicity or peracute signs depending on the species and age of animal and the dose and duration of aflatoxin exposure **(Smith, 2003 and Nain *et al*. 2007**)

b) Ochratoxin:

The primary effect of ochratoxin A in all poultry species is nephrotoxicity. In poultry the proximal tubules are mainly affected and the kidney is pale and grossly enlarged. As with aflatoxin, fatty liver can also occur due to ochratoxin exposure **(Olkowski *et al.*, 2005a).**

***4-Nutritional factors*:**

a) Restriction program:

Since sudden death occurs only in fast growing birds that are assumed to be eating to near physical capacity. Physical feed restriction reduces the rate of mortality by 75% than the birds maintain on free choice feeding program **(Olkowski *et al.*, 2005a**).

b) Dietary protein and amino acids:

Dietary protein seems to have little effect on Sudden death. It has been observed that the meat meal supplies some unidentified factor that provides protection against Sudden death **(Lin *et al.,* 2017 and Collier *et al.* (2017)).**

c) Dietary minerals and vitamin:

Sudden death may relate in some way to mineral availability suggesting a potential hypomagnesaemia tetany caused by nutrient interaction. **(Rubart and Zipes, 2015)**.

5-Managemental factors:

a) Lighting:

A period of short day length during early growth has recently been shown to be benefited in reducing SDS in broilers. There is about 30-60% reduction in incidence of Sudden death when broilers are subjected to 6 hours light from 3-4 days or various incremental programmes of 6 hour increasing to 23 hour over the 35 day period. Early growth rate is reduced because birds have less time to eat thus reducing the incidence of Sudden death. **(Kaul and Trangadia,2013).**

b) Stock density:

Lampert *et al. (* 2016) reported that boiler chickens are generally reared at a considerably higher stocking density. Such rearing conditions may act on the birds as a stress causing functional disorders in their organs including the heart.

2- Pathogenesis of Sudden death

When the course of disease is acute there is vascular disturbance which starts with circulatory lesions manifested by increased permeability of the peripheral circulatory system. **Carramenhai *and Carregaro* (2016)** This physiological permeability caused by short term increase 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 appears to be caused by heart damage which leads to lung oedema, so that the chicks are unable to breath sufficient fluid is lost from the circulatory system into the lung tissue spaces causing peripheral circulatory failure. The histological changes of intense congestion and oedema in the lungs result in the tissue parenchyma becoming separated from fresh blood supply leading to hypoxia. Vascular congestion is a constant feature of most of the tissue examined microscopically particularly the lungs where much of the effective air spaces are lost because of engorgement of pulmonary capillaries. Lymphocytic infiltration and inflammation involving the secondary bronchi and the presence of oedema in the alveoli considerably reduce gaseous exchange thus enhancing respiratory distress. **(Kaul and Trangadia, 2013**).

3. Diagnosis of Sudden death

Diagnosis is based on the history of sudden death, gross and microscopic pathology and no evidence of other disease. Blood profile and tissue analysis has little role in confirmative diagnosis ***(Julian, 2005).***

4. Prevention and control of Sudden death:

There is no single treatment or preventive measure to control the sudden death syndrome. Feed a low protein/low energy diet during first 14 days to reduce the oxygen demand lab diagnosis. Followed by Vaccination, hygienic Measurement to prevent spread of infection, and Quarantine. Follow a step down lighting program from 5-18 day period of growth **(Rubart and Zipes,2015).**

5. Conclusion

Sudden Death is an acute heart failure disease that affects mainly male fast growing chickens that seem to be in good condition. Although a common

condition in fast growing birds, the pathogenesis remains unclear. Cardiac arrhythmias are involved in the pathogenesis of Sudden Death with ventricular arrhythmias (VA) being the most common observation representing premature ventricular contractions and fibrillation.

Sudden death occurs due to several infectious and non infectious causes. Among infectious causes viral, bacterial; and mycosis, Causes are the most predominant. The main bacterial causes of sudden death syndrome include clostridium, or *S. Typhimurium* The major viral causes of Sudden Death are Newcastle disease, Avian influenza and Infectious bursal disease Also, Mycotoxicosis may cause sudden death due to ingestion of ration contaminated with mycotoxins. Also, it is possible that the increased humidity and hot season favors the growth of mold and fungus in stored feeds increasing the risk of birds to Mycotoxicosis. Among non infectious causes, immune status, nutrition, management and high intensity lighting appears to increase the incidence of Sudden Death**.** Sudden Death has caused world-wide great economic losses in the broiler industry for many years. In general mortality from Sudden Death has been reported as varying from 3- 9% of the total mortality in affected flocks There is no one treatment or preventive system for the control of Sudden Death in broilers.

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