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Short Communication

Pathology of sudden death syndrome in chickens: Report on associated pectoral muscle lesions

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Abstract

A private broiler chicken farm reported 2.5% mortality per day in apparently healthy birds with clinical signs of sudden death and found upside down. Postmortem examination revealed oedema of lungs, congestion of spleen and kidney, mottling of liver and highly contracted ventricles and clotted blood-filled auricles of heart. Representative tissue samples were collected in 10% formalin for histopathology. Microscopically, heart revealed MNC infiltrations with vacuolar changes in myocardial fibers. Pectoral muscles revealed muscle fiber swelling, fragmentation, degeneration, vacuolation and myofibril flocculation within sarcolemma. Cardiac tachyarrhythmia was proposed to be the cause of death predisposed by various factors. This study described the gross and microscopic pathology and prominent pectoral muscle lesions for the first time in SDS affected birds and elaborated on various possible factors for the cause of sudden cardiac arrest.

Keywords: Broiler chicken, Pathology, Pectoral muscle, Sudden death syndrome

Highlights

- Pathology of SDS was documented with meagre lesions accounting primarily the changes in heart.
- Documenting elaborative histopathological changes in skeletal muscle of SDS affected broilers.
- Cardiac arrhythmia might play possible role in skeletal muscle lesions in SDS.

Sudden death syndrome (SDS) described as a condition of sudden mortality in apparently healthy broiler chickens without any discernible cause. It was described earlier as 'oedema of lungs' in England (Hemsley, 1965) and 'died in good condition' in Australia (Jackson *et al.*, 1972). After brief review by Riddell (1997), the disease was named as acute death syndrome (ADS) and as 'heart attack' in chicken. The dead birds usually found upside down after acute mortality and hence the syndrome is popularly known as 'flip-over'. The disease was frequently reported in broiler chicken from 1-8 weeks of age (Newberry *et al.*, 1987). The cause of the death was uncertain. Genetic, nutritional and environmental factors were predisposed for incidence of the disease. But the exact mechanism for SDS was unidentified and puzzling (Crespo and Shivaprasad, 2008). Pathology and its related lesions in SDS affected birds were also limited. Hence, the report described the gross and microscopic pathology of broiler chicken died of flip-over and correlated the possible factors for contribution of the disease.

A private broiler chicken farm holding a stock of 8000 Vencobb birds had a history of sudden mortality in 30 days old chickens. Mortality was 80-100 birds per day. Birds were reared in deep litter system with floor space of 4500 square feet for 4000 birds. Birds were fed with commercial broiler finisher ration @ 130-150 g per bird. Randomly selected, three broiler chicken carcasses were presented for postmortem examinations to the Department of Veterinary Pathology, Veterinary College and Research Institute, Orathanadu, Tamil Nadu, India. Clinical signs of the affected birds were gathered and recorded. It was reported that the malady was short and sudden. Systematic postmortem examination was carried out. Gross lesions were recorded. Representative tissue samples from affected organs were collected in 10% formalin for histopathology. Tissues were processed as per standard paraffin embedding technique. Tissue sections of 4 µm thickness were prepared and stained with haematoxylin and eosin (H&E) stain (Bancroft and Layton, 2019).

Clinical signs exhibited by affected broiler chicken were reported to be sudden vigorous flapping of wings and fluttering of body, producing raucous and stentorian sound and suddenly found dead by turning its body upside down. Few birds showed ventral recumbency before the clinical signs, followed by death.

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Externally, affected dead birds revealed severe subcutaneous ecchymosis over tip of the manus of the wing flap, breast and ventral and ventro-lateral abdomen (Fig. 1). Upon internal examination, breast muscle appeared enlarged and showed irregular pale areas. On incision, paleness extended deep into the muscle bundles. Liver was found to be mottled and enlarged. Liver parenchyma showed irregularly linear faint yellowish discolouration beside normal parenchyma. The heart showed distention of both the auricles with clotted blood (Fig. 2). The ventricles of heart were empty with obliterated chambers (Fig. 3). Lungs were congested and oedematous (Fig. 4). Kidneys were slightly congested and enlarged. Proventriculus and gizzard were filled with yellowish granular feed material. The intestine revealed mild congestion of serosa and mesenteric vessels.

Microscopically, the liver revealed acinar formation (Fig. 5) with vacuolar degeneration of hepatocytes. Scattered areas revealed mononuclear cell infiltrations. The kidney revealed swelling of tubular epithelial cells (Fig. 6). The spleen showed reticulum cell hyperplasia. The thymus and Harderian gland revealed lymphoid necrosis with starry-sky appearance and plasma cell depletion, respectively. The heart revealed vacuolar changes in cardiac myocytes (Fig. 7). The myocardium revealed mononuclear cell infiltrations (MNC) separating the muscles bundles (Fig. 8). The skeletal muscles (pectoral) revealed muscle fiber swelling, breakage and fragmentations (Fig. 9, 10 and 11). The affected swollen muscle fibers showed dissolution of myofibrils and appeared slightly basophilic with lack of striations. Degenerated myofibrils appeared as floccules with in the sarcolemma. The broken end of the muscle fibers showed shrinkage on either pole (Fig. 12). A few of the affected muscle fibers revealed vacuolar spaces representing dissolved myofibrils. Scattered muscle fibers also revealed intact sarcolemma having prominent nucleus and dissolution of myofibril elements.

SDS is known for its sudden, acute form of death in fast growing, apparently healthy heavier broiler chicken without any discernable clinical signs (Newberry *et al.*, 1987).

Clinical signs observed in the affected flock of the present study were in parallel with Newberry *et al.* (1987) and Crespo and Shivaprasad (2008). SDS was reported to be a sudden attack lasting for 53 seconds prior to death with rapid violent flapping, loss of balance, convulsion and strong muscular contraction (Newberry *et al.*, 1987; Praveen Kumar *et al.*, 2020) and fall onto their back (Gall *et al.*, 2019). Subcutaneous haemorrhage over the manus of the wings and ventral abdomen might be due to rubbing and/or contraction of body in recumbent birds during strong violent wing flapping and convulsion prior to death. Such recumbent birds die of SDS are known as 'downers' (Hopkinson, 1991).

The gross and microscopic lesions observed in the study were in accordance with the previous documented evidence (Crespo and Shivaprasad, 2008; Siddiqui *et al.*, 2009; Meshram and Bijoy, 2017). The pulmonary oedema, firmly contracted ventricles and blood-filled, highly distended atrium of heart of the present observations were consistent with all previous reports, and these were found to be emblematic to SDS.

The spongy and vacuolar appearance of sarcoplasm of the myocardial fiber and MNC infiltrations in the ventricular myocardium observed in this study were in parallel with earlier reports (Olkowski *et al.*, 2008; Sosnowka-Czajka and Skomorucha, 2022). It was reported that lipid metabolism affecting cardiac sarcoplasmic reticular transport system was responsible for vacuolar degenerations (Chung *et al.*, 1993; Tekeli *et al.*, 2016). These myocardial changes affect the physiological integrity of structures of heart and whereby interfere with orderly propagation of cardiac muscle activity and responsible for ventricle arrhythmia (Olkowski *et al.*, 2008). Earlier, during 1975, Cassidy *et al.* (1975) postulated that dilatation with accumulation of blood clot in auricles of affected birds was a postmortem change which occurred after death. This seems to be true as strong contraction of ventricular myocardium due to tachyarrhythmia might pave way for accumulation of blood in auricles prior to or immediately after death. Altogether, the lesions of heart contributed to catastrophic cardiac arrhythmia (Olkowski and Classen, 1998; Olkowski *et al.*, 2008). The tachyarrhythmia which alter polarization activity of heart conduction potential impart sudden arrest of heart and hence the disease also known as acute cardiac arrest syndrome (Ononiwu *et al.*, 1979). Mutation and dysfunction of the *chRYR2* gene was reported to be associated with cardiac arrhythmia causing sudden death syndrome, which resulted changes in intracellular calcium cycling machinery in broiler chickens (Sosnowka-Czajka and Skomorucha, 2022).

Beside myocardial changes structural abnormality, skeletal muscle changes as myofiber swelling, breakage, fragmentation and dissolution of myofibril

Pathology of sudden death syndrome in broilers

Fig. 1. Thirty days old broiler chicken with subcutaneous haemorrhages over the manus of wings and ventral abdomen

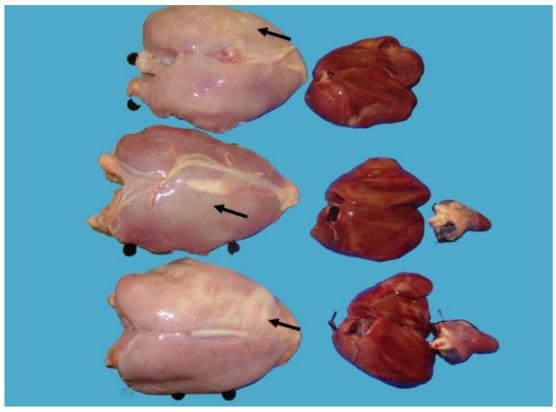

Fig. 2. Bulky pectoral muscle with scattered pale areas (arrow), mottled liver and auricles of heart dilated with blood

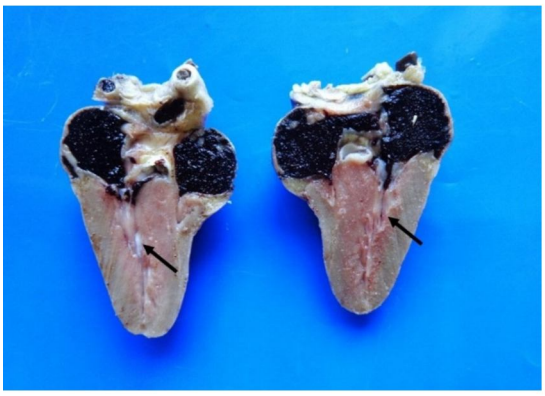

Fig. 3. Strong contraction of ventricles (arrow) and dilated auricles with blood clot

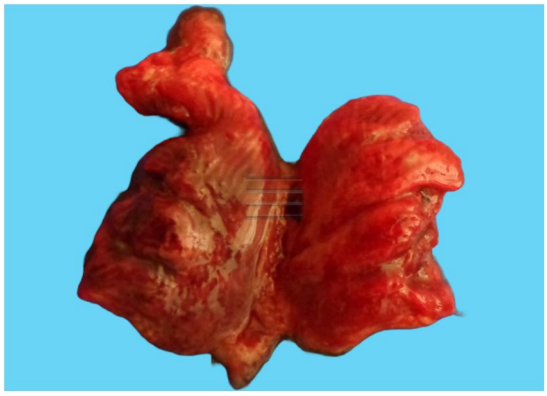

Fig. 4. Oedema and congestion of lungs

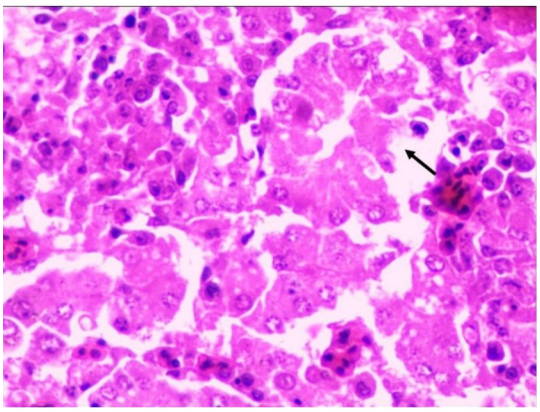

Fig. 5. Acinar formation of hepatocytes Liver x400

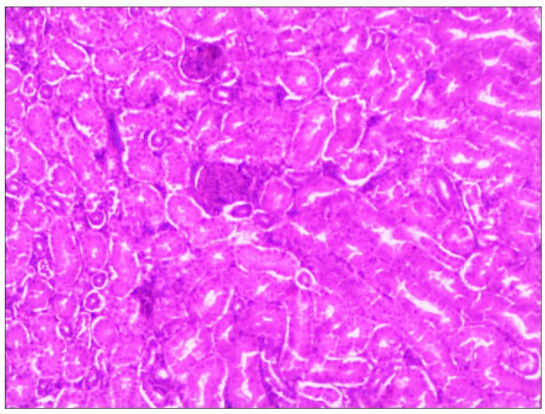

Fig. 6. Cell swelling of tubular epithelial cells Kidney x100

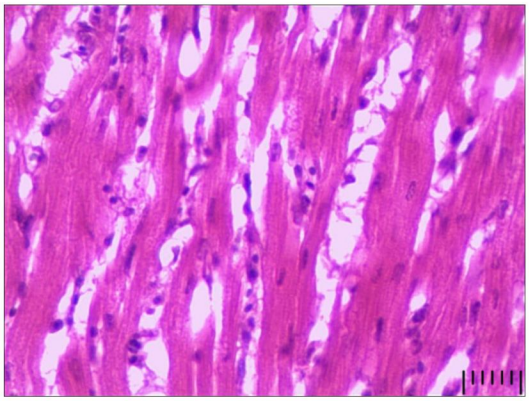

Fig. 7. Vacuolar changes in myocardium of heart H&E x400

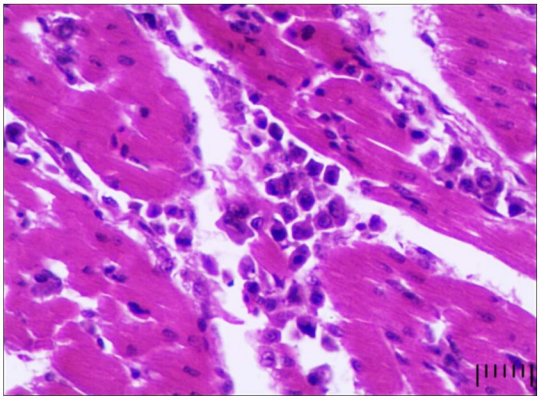

Fig. 8. Mononuclear cell infiltrations in cardiac muscle fibers H&E x400

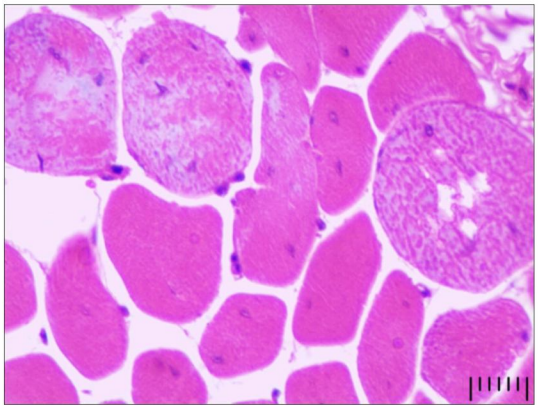

Fig. 9. Myofiber swelling of pectoral muscle of chicken breast H&E x400

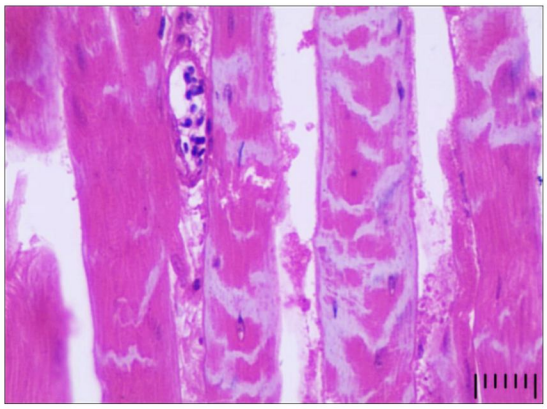

Fig. 10. Myofiber fragmentation with flocculations of myofibril of pectoral muscle of chicken breast H&E x400

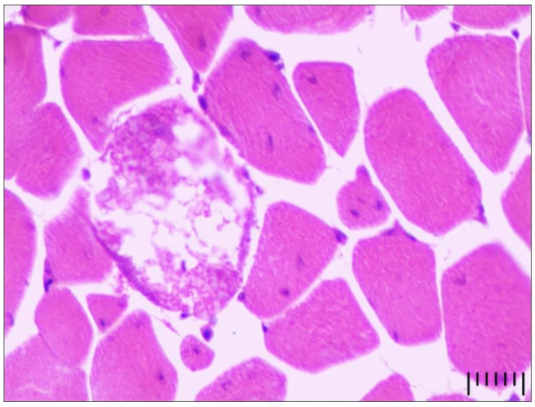

Fig. 11. Myofiber swelling, marked fragmentations and vacuolations of pectoral muscle of chicken breast H&E x400

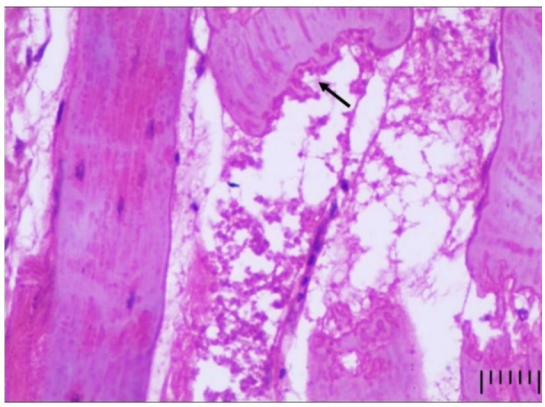

Fig. 12. Myofiber breakage with shrinkage of terminal ends (arrow) of pectoral muscle of chicken breast H&E x40

observed in the pectoral muscles were the earlier record and must to enclose with the microscopic lesions in SDS of chicken. Hopkinson *et al.* (1983) recorded blanched musculature and the myopathic changes such as coagulative necrosis and sarcolemmal proliferation without cellular response in SDS affected 'downer' broiler chicken. Although the above explanation justifies the present pectoral muscle lesions from 'downer' chicken, the microscopic lesions of the present study, which was described above, were found to be unendorsed until now as per available documented evidence. It was hypothesized that fast growing broiler chickens have large proportion of muscle mass compared to visceral organs leading to muscular hypoxia. Lack of aerobic metabolism resulted in increased lactate production in skeletal muscle and lowering of pH, systemic acidosis and cardiovascular failure (Kumari *et al.*, 2016; Safaei *et al.*, 2021). High metabolic rate, elevation of body temperature, strong muscular contraction (Newberry *et al.*, 1987; Crespo and Shivaprasad, 2008) and hypoxia (Safaei *et al.*, 2021) of muscles prior to death might be responsible for the pectoral muscle lesions in 'downers'. This was further justified by severe contraction of ventricular myocardium and vacuolations of sarcoplasm (Sosnowka-Czajka and Skomorucha, 2022) of heart in the present study. Further investigation and experimental studies are warranted to know the exact mechanism behind the muscle lesions.

Various possible factors such as genetic, nutritional and environmental factors found to be linked with the occurrence of SDS. Six strains of commercial broilers videlicet Cobb-500, Arbor acres, Avian farms, Hubbard-Peterson, ISA and Ross were reported to be susceptible pointing hereditary involvement. This was found to be in-line with the present incidence of SDS, which was reported in Vencobb chicken. In contradiction, the incidence was reported in various broiler strains worldwide (Riddell, 1997; Crespo and Shivaprasad, 2008). High lightening intensity, stocking density and lack of exercise were proposed to be environmental and or management stress to broiler chicken (Ononiwu *et al*., 1979; Newberry *et al.*, 1985; Afolayan *et al.*, 2016). Commercial broilers were frequently subjected to various stress factors. Stress trigger life threatening cardiac arrhythmia and ventricular fibrillations (Olkowski *et al.*, 2008; Siddiqui *et al.*, 2009). Apart, physical form of feed was also suggested to increase the incidence of SDS. Pellet feed was most frequently used to increase body weight gain in broiler chicken (Azizian and Saki, 2020) especially to increase skeletal muscle mass (Ahmed and Abbas, 2013). It was reported that pellet feeding increases body metabolism and thereby SDS (Azizian and Saki, 2020). Further, ration containing low in energy and high in protein showed increased incidence of SDS with elevated blood lactic acid (Boroumandnia *et al.*, 2021). Additionally, high calcium, vitamin D and thiamine along with low phosphorus and potassium were found to be associated with the incidence of SDS (Crespo and Shivaprasad, 2008). Although the exact cause could not be ascertained in the present mortalities. We propose that physically induced stress associated with procedural and/or routine management activities in broiler management might posed higher risk among others.

In agreement with earlier workers, we propose that acute death (SDS) in broiler chicken may possibly be govern with controlled breeding strategies and selection of breeder broiler strain without *chRYR2* defect (Mosayyeb Zadeh *et al.*, 2022; Sosnowka-Czajka and Skomorucha, 2022). Additionally, restricted feeding by following low protein and energy ration during first14 days of age (Siddiqui *et al.*, 2009; Sahraei, 2014) and supplementation of feed with vitamin E and selenium and/or rapeseed containing long-chain omega-3 fatty acids EPA (eicosapentaenoic acid) and DHA (docosahexaenoic acid) to prevent cardiac arrhythmia (Mozaffarian, 2008) would reduce the risk of SDS. Further, addition of guanidinoacetic acid (GGA) in ration protect birds from reduced lactic acidosis (Boroumandnia *et al.*, 2021). As no curative is possible, preventing all possible stress factors is only way to reduce incidence of SDS (Afolayan *et al.*, 2016), especially shortening the photoperiod proved as a factor to overcome the stress in broiler chicken. It showed to increase secretion of melatonin, a defense mechanism against stress from various factors (Taati *et al*., 2019).

We conclude that the pathological changes observed in pectoral muscles during sudden death syndrome (SDS) in boiler chicken might be due to ventricular tachyarrhythmia which occurs shortly before death. Pectoral muscles pathology recorded in the present study is the earlier record and warrants further detailed experimental studies to understand the complete pathogenesis in SDS of broiler chickens.

Conflict of interest: Authors declare no conflict of interest.

Author's contribution: NBP: Conducted postmortem examination of dead birds, collected details regarding

the outbreak and prepared the manuscript; JS: Assisted in interpretation of histopathological lesions and done proof reading of the manuscript.

Data availability statement: All the data supporting the research findings have been presented in this paper. The corresponding author is willing to provide the raw data upon reasonable request.

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