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Streptococcosis Contributed to Higher Mortality After Salt Toxicosis in a Specific Pathogen Free Breeder Flock

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Abstract

An outbreak of sudden death occurred in a research-specific pathogen-free breeder layer flock. Following the report of such a drastic loss in the flock, a comprehensive investigation was conducted to identify the causative agent of the event, including a review of history and clinical signs, as well as serology and postmortem examination. According to the serology results, highly pathogenic avian influenza and virulent Newcastle disease were not detected. Gross lesions and history indicated a toxic condition, and the gross examination was consistent with salt poisoning, but the condition worsened due to a second opportunistic pathogen. In bacterial culture of internal organs and Gram staining, Gram-positive cocci in pairs or chains were identified. Molecular investigations detected *Streptococcus gallolyticus*. It can be concluded that ignorance of salt balance in the diet could lead to salt poisoning and suppression of the immune system. Following toxicosis, opportunistic bacterial growth can occur rapidly, leading to septicemia in chicken flocks.

Introduction

Specific pathogen–free (SPF) flocks are maintained under strict biosecurity measures to support vaccine production and research. Mortality outbreaks in such flocks are rare and require thorough investigation because they can affect research reliability and vaccine effectiveness (Murray *et al*., 2022).

Although dietary sodium chloride is essential, excessive intake—particularly under water deprivation—can induce salt toxicosis in poultry. Hypernatremia causes a shift of water from intracellular to extracellular compartments, leading to cerebral dehydration and potentially intracranial hemorrhage (Yadala *et al*., 2019). During prolonged hypernatremia, adaptive intracellular osmolyte accumulation occurs in neurons, limiting rapid osmotic correction. If the hypernatremia condition is quickly fixed, water is restored but rushes back into brain cells and can cause swelling. According to the severity of the toxicosis, it can be acute with sudden death or may not show any visible signs of damage. The mortality rate may exceed 50% in affected animals, regardless of treatment (Gornatti-Churria *et al.,* 2025). Streptococcosis is a bacterial disease that affects many birds, including turkeys, ducks and geese, and chickens. Turkeys and waterfowl are typically affected within the first 3 weeks after hatch. Chickens tend to be affected later, usually between 5-7 weeks of age or again around 44–55 weeks. If a flock is affected, mortality can be very high—up to about 50%. The infection spreads mainly through three routes: by mouth (oral), through the air (aerosol), and through skin wounds (Crispo et al., 2018). Streptococcal bacteria are opportunistic pathogens. Many *Streptococcus* species are normal inhabitants of the microbiota in humans and animals, including poultry. Streptococci can cause a range of acute and chronic diseases in both commercial and noncommercial poultry. Under permissive conditions, streptococcal infections in poultry—whether arising in commercial production or backyard flocks—may present as acute or chronic disease (Hassan, 2014). This report describes an acute mortality event in an SPF breeder flock housed at the Razi Vaccine and Serum Research Institute in Iran.

Case Description

A total of 200 SPF chickens Hy-Line W-36 were reared in positive-pressure isolators with ad libitum access to feed and water at the Poultry Diagnostics

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Department of Razi Vaccine and Serum Research Institute. All equipment, water, and food were sterilized before moving to the houses. At 60 weeks of age, the flock experienced a sudden increase in mortality (>20%) and a sharp drop in egg production within a single day. Mortality occurred in both sexes, but the onset in roosters was delayed by approximately two days. Mortality continued to rise, exceeding 60% of the flock by the fifth day after observing initial signs. Prior to the onset of clinical signs, the birds showed no weight loss, emaciation, skin lesions, or behavioral abnormalities. Overall, more than 20% death in a single day was considered the outbreak onset. The highest mortality rate lasted for 5 days and declined in two weeks; all chickens that showed depression died eventually, survived birds had normal egg production within 2 weeks.

Clinical signs:

- Ruffled feathers and huddling
- Listlessness, Ataxia, and apathy
- Occasional watery droppings and vent pasting

Necropsy Findings

Gross lesions observed during post-mortem examination included (Figure 1A-C)

- Reddish skeletal muscles, consistent with dehydration
- Cyanosis of combs and wattles
- Generalized visceral congestion but in the absence of any exudate in sinuses, nasal cavities, or trachea
- Occasional accumulation of serous fluid in the abdominal cavity and Hydropericardium
- Blood was seen on the tip of the proventricular glands
- No congestion, consolidation, or hemorrhage was observed in the lungs, trachea, or buccal sinuses
- The kidney and liver were swollen, and the spleen showed various degrees of hemorrhages
- Hyperemia and hemorrhages on the brain, suggesting a systemic problem- cerebral edema and inflammation of the membranes surrounding the brain (meningitis).
- Watery and reddish content in the intestine and moderate necrotic lesions in the intestine
- When the gizzard was checked, some erosion was evident on its mucosa.



Figure 1A. Pinpoint hemorrhages on the tip of the proventricular glands



Figure 1B. Hemorrhages in the brain membrane



Figure 1C. Hyperemic internal organs and muscles

Diagnostic assessment

Laboratory examination

Blood samples were collected from the wing vein. Serum was separated and subjected to a panel of diagnostic tests to determine the cause of the outbreak.

-Internal organs culture

Food and water were investigated for possible bacterial contamination, but no pathogens were isolated. Aseptically, samples were taken from the heart, liver and spleens of freshly dead birds, then the samples were cultured on blood agar, and surprisingly, a pure α-hemolysis bacteria was grown and followed by Gram staining. Gram-positive cocci were detected. The same results were observed in direct Gram staining of a blood smear.

Streptococcus gallolyticus was confirmed by PCR assay targeting the Mn-dependent superoxide dismutase gene (forward primer SgsodA-F (5′-CAATGACAATTCACCATGA-3′; positions 17 to 34) and reverse primer SgsodA-R (5′-TTGGTGCTTTTCCTTGTG-3′; positions 424 to 407) reported by Sasaki *et al* (Sasaki, Osawa, Nishitani, & Whiley, 2004). The *Streptococcus* was isolated from the organs as a pure culture, indicating that the bacteria probably were the main causative agent of the outbreak.

Differential Diagnosis

Because of the cyanosis in combs and wattles and hemorrhages on the tip of proventricular glands, avian influenza and/or Newcastle disease was suspected. Therefore, a hemagglutination Inhibition test was done to detect any possible infection with avian influenza and/or Newcastle disease (Figure2). The results were reconfirmed by ELISA. *Mycoplasma spp*. infection was ruled out by a rapid agglutination test.

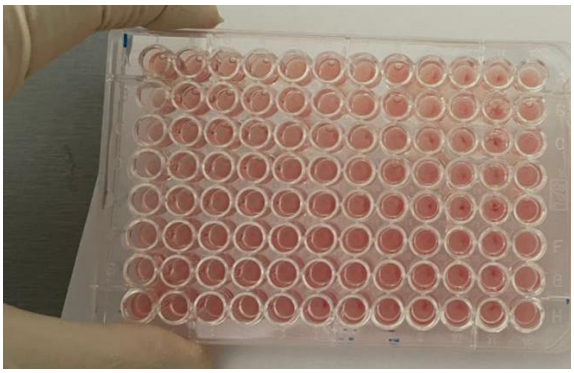


Figure 2. Negative HI results for Newcastle disease investigation

Final Diagnosis

Based on the clinical observations and laboratory examinations, streptococcosis predisposed by salt toxicosis was diagnosed, which led to a high mortality rate within 3 to 7 days.

Treatment/outcome

In total, 80% of the flock was lost after 2 weeks and because of the severity and acuteness of the outbreak, none of the birds with clinical signs survived, but the rest of the flock received vitamin E Selenium as supportive treatment. Egg production recovered 2 weeks after the last case of death.

Discussion

The present case is a report of an acute occurrence of high mortality in SPF breeder chickens emphasizes the vulnerability of SPF flocks despite strict biosecurity measures, the importance of continuous monitoring of food and water, especially food analysis, and the salt content of the diet. Salt limits in diets differ according to the breed and species, e.g. in egg-laying chickens (0-6 weeks old) and white-egg-laying hens at peak need, 1500 ppm sodium and 2000 ppm for Broilers (0-3 weeks old) in their feed. Normal brain sodium levels in Chickens are 1,600-1,700 ppm on a wet-weight (WW) basis. Serum sodium concentrations above 150 milliequivalent per liter (mEq/L) are considered elevated and indicate sodium intoxication (Gornatti-Churria *et al*., 2025).

Looking deeply into the history and the diet without any evidence of water deprivation showed a prominent alteration in what the birds were eating, specifically a diet with a very high salt content around 1.6%. This high-salt concentration is suggested as a possible cause of salt poisoning; therefore, salt toxicosis likely worsened the birds' condition, hence immunosuppression (Anil Kumar *et al*., 2000). The rapid progression, systemic signs, and hemorrhagic brain lesions suggested a highly virulent infectious or toxic situation. As mentioned above, the sudden and high mortality rate, along with a history of changes in diet, warned us of a toxic chemical and/or bacterial contamination. Looking at clinical signs and post-mortem signs such as pinpoint hemorrhages on the tip of proventricular glands and hemorrhages on the brain membranes, are common signs of highly pathogenic avian influenza, virulent

Newcastle, but were taken out after serological investigation. Swollen and fragile liver, marble appearance, hyperemia in the spleen and hyperemic muscles were indicators of septicemia infections and were confirmed in blood culture (Saumya *et al.*, 2014; Sekizaki *et al*., 2008).

The main conclusion is that sodium toxicosis has the potential to cause a sudden outbreak in a poultry flock, leading to a substantial loss because of a secondary infection, even though it is not common in poultry operations.

So far, no evidence proves *Streptococcus* species are transmitted from poultry to humans. However, one human case of endocarditis caused by *Streptococcus* gallolyticus subsp. *gallolyticus* disclosed a bacterial isolate that shared features with streptococci found in his layer flock that the patient was caring for (Dumke *et al.*, 2015).

Conclusion

Salt toxicosis (ST) happens in all animal species, but in cattle, pigs, and poultry, it is the most common. It can be tolerated if birds have enough access to a water supply. Malfunctioning water supplies, overcrowding, unfamiliar environments, or frozen water may create an appropriate ground for developing an ST condition. Poultry growing on standard diets display elevated susceptibility under water deprivation or when subjected to high-salt diet conditions, even in moderate water restriction, and so even a subclinical or acute ST can easily lead to secondary infection and significant death and economic loss; proactive management strategies with strict monitoring of diet are key to significantly reducing or preventing salt poisoning toxicity.

Limitations of the study

Because of the small size of the flock and the inability to repeat the case, the inability to generalize from a single outbreak is one of the limitations.

Another limitation is that the salt concentration of the water was not measured. This shortens our knowledge of the possible water salt content effect.

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