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Sodium toxicosis in chickens: case series (2014–2023) and literature review

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Abstract. Cases of sodium toxicosis (ST), although reported infrequently, can result in acute morbidity and mortality and extensive losses in affected poultry. We analyzed the clinical, pathologic, and toxicologic findings of 7 diagnosed cases of ST in chicken autopsy submissions at the California Animal Health & Food Safety Laboratory System (CAHFS), University of California–Davis, from 2014 to 2023. We also evaluated the brain sodium concentrations in 10 clinically normal broiler chickens to elucidate potential differences with salt-intoxicated chickens, and reviewed the literature of field cases of ST in chickens and turkeys. Lesions of anasarca described in the 7 ST cases (66 chickens) identified from the CAHFS database included: ascites (62 of 66; 6 of 7 cases); hydropericardium and cardiomegaly (54; 6 of 7); edematous, congested lungs (24; 6 of 7); enlarged, pale kidneys (24; 6 of 7); subcutaneous edema (17; 4 of 7); cystic testes (14; 6 of 7); and cerebral edema (7; 4 of 7). Brain sodium concentrations exceeded 1,800 ppm in only 4 of 24 brains analyzed in our case series. In the feed samples analyzed from 5 ST cases, sodium concentrations exceeded the recommended 2,000 ppm; concentrations detected were 2,500–12,000 ppm. In brains from the 10 clinically normal chickens evaluated, brain sodium concentrations were 1,500–1,700 ppm.

Keywords: anasarca; brain; chickens; cystic testes; edema; feed; sodium toxicosis.

Sodium toxicosis (ST) caused by excess sodium ion intake has been intermittently reported in confinement-reared poultry and its occurrence can lead to significant economic losses in affected flocks.[2,14] In the literature, ST is also referred to as sodium ion intoxication, sodium intoxication, sodium chloride toxicity, sodium chloride intoxication, salt toxicity, salt poisoning, salt intoxication, salt toxicosis, and hypernatremia.[2–5,7,9,11,13–17] Direct or acute ST can develop secondary to excess intake of salt from feed and/or drinking water, with clinical signs often developing within 1–2 d of intake of excess sodium. Water availability and water intake in cases of hypernatremia have a significant influence of the pathophysiology of sodium toxicosis.[16] Excess sodium intake accompanied by restricted water intake is a common predisposing factor in delayed or indirect ST, with clinical signs often apparent within 4–7 d of restricted water intake.[1,4]

ST occurs when excess sodium intake increases serum sodium concentrations, which facilitates water loss from the intracellular fluid into the interstitium and extracellular fluid. Sodium passively diffuses across the blood-brain barrier, thus increasing the sodium concentration of the cerebrospinal fluid. Intracellular osmolarity of brain cells subsequently increases in an attempt to mitigate excess water loss and avoid cellular degeneration. In cases of rapidly increased sodium concentrations, failure of this protective mechanism results in cellular dehydration, necrosis, and vascular disruption in the brain, which can result in cranial hemorrhages. Variation in susceptibility and severity of ST-induced disease in avian species can be attributed to several factors, including species and age affected, renal function, concentration of sodium ingested, access to drinking water, and dietary factors.[4,7,8]

Macroscopic lesions of anasarca observed in acute ST in poultry include, cerebral edema, ascites, hydropericardium, cardiomegaly, edematous and congested lungs, pale and edematous kidneys, and enlarged cystic testes.[2–7,9,11–15,17] Severity of acute ST lesions in chickens can vary. In severe cases, intake of high concentrations of sodium can lead to acute intoxication that can result in severe diarrhea, dehydration, renal damage, weight loss, and death. Moderately high sodium intake produces a variety of clinical signs and pathologic findings depending on the duration of time that intoxicated birds survive with hypertension before heart failure occurs. In these cases, water retention and hypervolemia lead to cardiac overload, causing right ventricular hypertrophy and dilation, valvular insufficiency, generalized edema, and

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Table 1. Sodium concentrations in brain and feed samples in 66 chickens (7 cases) with diagnosis of sodium toxicity at the CAHFS Laboratory (2014–2023).

Case	Age, d	Year	Production type (company)	Chickens per case	Brain sodium, ppm*	Feed sodium, ppm
1	6	2023	Broilers[a]	8 (6 live, 2 dead)	1,500 (3), 1,600 (1)	5,700
2	25	2021	Broilers[b]	10 (5 live, 5 dead)	1,700 (2), 1,300 (1)	2,500
3	25	2021	Broilers[b]	10 (5 live, 5 dead)	1,500 (1), 1,600 (1), 1,900 (1)	2,600
4	18	2016	Broilers[b]	6 dead	1,700 (1), 1,900 (1)	NA
5	12	2016	Broilers[b]	4 dead	2,800 (1), 1,700 (1)	NA
6	7	2016	Broilers[a]	20 (18 live, 2 dead)	1,400 (3), 1,500 (1), 1,600 (1)	12,000
7	28	2014	Backyard[c]	8 (2 live, 6 dead)	1,300 (1), 1,500 (1), 1,600 (1), 1,800 (1), 2,800 (1)	4,300

NA = not available; feed was not submitted for analysis in these cases.
a–c Indicate ST cases from 2 commercial broiler companies (A, B) and 1 organic pasture-raised farm (C).
* Numbers of brains analyzed for sodium are in parentheses.

ascites. Finally, mildly elevated sodium intake can produce loose droppings and weight gain due to water retention for at least 1–2 d after the start of exposure. In mild intoxication, reduction in feed intake and poor growth may occur but, in some cases, feed consumption and weight gain are unaffected.[4]

The recommended dietary sodium concentration is 1,500 ppm in egg-laying chickens 0–6-wk-old and white-egg layers in full production; 1,700 ppm in 0–4-wk-old turkeys, and 2,000 ppm for 0–3-wk-old broilers, based on feed with dry matter of 90%.[4] The range of normal brain sodium concentrations in chickens is 1,600–1,700 ppm on a wet-weight (WW) basis[10]; concentrations >2,000 ppm WW are highly suggestive of sodium intoxication.[14] Serum sodium concentrations >150 mEq/L are considered to be elevated.[5]

Diagnosis of ST typically entails the detection of elevated sodium concentrations in feed and/or drinking water, serum, and/or brain, together with a clinical history and gross and microscopic lesions compatible with ST.[14] Our aims were: 1) to characterize the clinical, pathologic, and toxicologic findings of ST cases in chicken autopsy submissions to the California Animal Health & Food Safety (CAHFS) Laboratory System from 2014 to 2023, 2) to review the literature of field cases of ST in chickens and turkeys, and 3) to provide preliminary data on baseline brain sodium concentrations in healthy broiler chickens to elucidate potential differences with brain sodium concentrations from sodium-intoxicated chickens.

Case series

ST cases and autopsy

We searched the electronic database of CAHFS from January 2014 to July 2023 for chicken cases with a diagnosis of ST in which brain sodium analysis was performed. We retrieved and analyzed 7 ST cases in chickens submitted from 6 commercial broiler premises and 1 pasture-raised, organic, backyard chicken flock. Each case included 8–20 chickens; sixty-six 6–28-d-old chickens were evaluated (Table 1). Five ST cases originated from premises in California; cases 4 and 5 were submitted from commercial Louisiana broiler farms owned by company B. The in-state commercial broiler cases were submitted from broilers farms in the Central Valley of California. The clinical history in most cases reported acute increases in mortality, ataxia, abdominal distension, and abdominal turgidity. Gasping and pasty vents were described in a few cases. Live chickens were euthanized with CO_2 in a gas chamber, according to standard operation procedures of CAHFS and following AVMA guidelines. Blood samples from each bird were collected individually to obtain serum for routine serology. Postmortem examination and diagnostic work-up were carried out in all chickens.

Gross findings in the 66 chickens autopsied from the 7 ST cases included, ascites (62 chickens, 6 of 7 cases), hydropericardium and cardiomegaly (54, 6 of 7), edematous, congested lungs (24, 6 of 7), enlarged, pale kidneys (24, 6 of 7), subcutaneous edema (17, 4 of 7), and cystic testes (146 of 7). Macroscopic lesions described in a small number of cases included distended, thin-walled intestines with watery content (6- and 7-d-old commercial broiler chicks). Moderate necrotic enteritis of the small intestine was noted in chickens submitted from the backyard case.

Histology

Samples of skin, nasal cavity, eye, eyelid, cerebrum, cerebellum, heart, trachea, lung, liver, kidney, adrenal glands, testis, ovary, spleen, kidneys, air sac, yolk sac, navel, pancreas, intestines, and bone marrow were collected and fixed by immersion in 10% neutral-buffered formalin (pH 7.2) for 24–48 h. All tissues were processed by standard histologic techniques to produce 4-µm thick, H&E-stained sections.

Microscopic lesions included interstitial pulmonary congestion and edema; myocardial edema with dilation of right ventricle and expansion of the pericardial sac; and interstitial congestion and edema, and necrosis of renal tubule cells in most of the evaluated chickens. Less commonly, dilation of

Table 2. Brain sodium concentration in each of 2 clinically normal commercial broiler chickens of various ages submitted to CAHFS, August–October, 2023.

Age, d	Sodium concentration in brains, ppm
52	1,500; 1,600
41	1,500; 1,700
35	1,500; 1,500
35	1,500; 1,500
35	1,600; 1,600

testicular seminiferous tubules, and multifocal perivascular edema with distension of the Virchow–Robin space in cerebrum were noted in the studied chickens. There was no microscopic evidence of endoparasitemia in autopsy cases.

Salt analysis

Sodium concentrations were determined (CAHFS Davis Toxicology) on individual brains from 24 chickens from the 7 ST autopsy submissions, from feed samples in 5 of 7 autopsy cases (Table 1), and from brains from 10 clinically normal broilers (Table 2). Briefly, for the salt screen, the entire brain sample was homogenized and subsampled for analysis. Feed samples were mixed well and, if needed, a portion was ground by Stein mill and/or IKA mill until homogenized. Feeds were prepared in triplicate with results averaged. Both brain and feed samples were digested with nitric acid by hot-block digestion. Following digestion, hydrochloric acid was added to samples, which were then brought to a final volume of 10 mL with 18-Mohm water and filtered with a syringe filter. Filtered samples were run by inductively coupled plasma optical emission spectroscopy (ICP-OES) to measure the metals included in the mineral screen. Method blank and certified reference materials were prepared with samples for QC. A standard curve and blank were run at the beginning of the sample batch to calculate sample concentration. Continuing calibration verification and blanks were run every 10 samples to ensure drift was not >10% throughout the run. Sodium concentrations were reported on a WW basis.

Brain sodium from 19 of 24 chickens were <1,800 ppm, with concentrations of 1,300–1,700 ppm ($\bar{x}$ = 1,500 ppm) in 19 cases. In 5 of 24 brains, sodium concentrations were ≥1,800 ppm (1,800–2,800 ppm; Table 1). Sodium concentrations in the 5 feed samples analyzed were all higher than the recommended 2,000 ppm (2,500–12,000 ppm; Table 1).

For assessment of baseline sodium concentrations, we collected 10 brains from randomly selected, 35–52-d-old, clinically normal, commercial white Cobb/Ross broilers submitted from healthy flocks for routine surveillance testing. Brain sodium concentrations were <1,800 ppm in the 10 clinically normal broiler chickens (1,500–1,700 ppm, $\bar{x}$ = 1,500 ppm; Table 2).

Bacteriology

The number of cultures and organs selected for bacterial culture from autopsies varied by case and lesion presentation. From the 7 ST cases, aerobic bacterial cultures were performed on 2 brains (1 case), 4 sinuses (2 cases), 6 tracheas (3 cases), 5 lungs (3 cases), 6 air sacs (3 cases), 9 hearts (5 cases), 24 livers (7 cases), 1 bone marrow, 1 yolk sac, and 1 tarsal joint. Pooled cecal samples were screened for *Salmonella* spp. in 2 cases. Bacterial species isolated from aerobic cultures included *Escherichia coli* (7 of 7 cases) from 3 livers (2 cases), 1 sinus, 1 heart, 1 trachea, 1 lung, and 1 yolk sac; *Enterococcus cecorum* (3 of 7 cases) was isolated from 2 hearts (2 cases), 2 lungs (2 cases), 5 livers (2 cases), and 2 air sacs (one case); *Salmonella* Enteritidis (1 of 7) was isolated from 1 heart, 1 liver, and 1 cecal pool from the 6-d-old broiler chick case; and *Gallibacterium anatis* biovar *haemolytica* (1 of 7) was isolated from 1 heart, 1 trachea, and 1 liver culture.

PCR

PCR tests were performed for *Avibacterium paragallinarum* (2 cases), avian influenza A virus (avian IAV, 5 cases), infectious bursal disease virus (IBDV, 3 cases), infectious bronchitis virus (IBV, 4 cases), and enteric viruses (1 case). PCR tests detected IBDV from bursa tissue pools (2 of 3 cases tested), and parvovirus and chicken nephritis virus from large intestinal content pool (1 case tested). PCR tests for avian IAV, *A. paragallinarum*, and IBV from tracheal swab pools were negative. Enteric PCR screening for chicken astrovirus, reovirus, rotavirus, turkey astrovirus 1, and turkey astrovirus 2 from large intestinal content analyzed in 1 of 7 cases were also negative.

Serology

Serum samples collected from the live chickens from 5 of 7 cases were tested by ELISA (Idexx) for the detection of antibodies against avian IAV, IBV, reovirus, IBDV, Newcastle disease virus, *Mycoplasma synoviae*, and *M. gallisepticum* following the manufacturer's recommendations. All serum samples were negative for antibodies against avian IAV, IBV, reovirus, IBDV, Newcastle disease virus, *M. synoviae*, and *M. gallisepticum*.

Literature review

We searched PubMed, CAB Direct, Google Scholar, and Web of Science for published field cases of sodium toxicosis in chickens and turkeys. A limited number of field cases of ST in poultry with compatible clinical signs and postmortem findings and supported by sodium feed analysis have been reported in the literature. Field cases reviewed include ST in 5–11-d-old poults, and 13-wk-old Nicholas tom turkeys in the United States[15,17]; in 1-d-old and 6-wk-old broilers in

New Zealand[6]; and in 10–30-d-old broilers, 6–9-d-old broilers, and 24-wk-old broiler breeders in Israel.[9] A suspected case of ST in broilers that culminated in 80% mortality at the end of week 6 of the grow-out period was also briefly described in 2019 in a flock of 320 broilers in India.[11]

In 1986, ST was described in Georgia USA in 5–11-d-old poults experiencing a 4% increase in mortality, depression, abdominal distension, respiratory distress, drooping wings, outstretched head and neck, and reluctance to stand and move.[15] At postmortem examination, ascites, dilation of the right ventricles of heart, hydropericardium, edematous and congested lungs, and swollen, pale kidneys were noted in most poults evaluated. Microscopically, interstitial edema in lungs and kidneys and increased separation of cardiomyocytes and edema of the pericardial sac were noted. Sodium chloride analysis of feed and water revealed concentrations of 1.85% (18,500 ppm) and 0.00235% (23.5 ppm), respectively. Peracute onset of respiratory signs, dilated right ventricle, hydropericardium, ascites and sudden death were consistent with sodium chloride toxicosis, and ST was confirmed by detection of high sodium concentrations in the feed.[15]

ST was also described in 1-d-old broiler chicks and 6-wk-old broiler chickens with increased mortality, lethargy, watery droppings, and labored breathing in New Zealand in 1991.[6] Grossly, both ages of affected broilers had hydropericardium, cystic testes, ascites, and pulmonary congestion and edema at postmortem examination. Sodium concentrations were measured in feed samples and livers from dead birds. The starter feed contained 1% sodium, although it was labeled as 0.21%, and salt concentrations of 4.0 and 4.2 g/kg on a WW basis were detected in livers of 2 of the affected birds.[6]

ST involving 13-wk-old Nicholas tom turkeys with increased mortality, depression, polydipsia, diarrhea, ataxia, tremors, and torticollis was reported in the United States in 1995.[17] Grossly, round hearts, ascites, watery content in intestine, hepatic congestion, and pale, dehydrated skeletal muscles were observed in affected turkeys. Microscopically, there were lesions of cerebral malacia and glomerulopathy. Feed analysis detected 3.31 ± 0.09% of sodium (33,100 ± 900 ppm) and 3.12 ± 0.01% (31,200 ± 100 ppm) of sodium chloride. Salt toxicosis was believed to have been caused by an error at the feed mill and surviving toms were marketed when 16-wk-old.[17]

Within one month in 2015, extensive salt intoxication was reported in 6 commercial broiler farms and 2 broiler breeder flocks in southern Israel.[9] The initial field cases involved 3 farms raising 10–30-d-old broiler chickens. Affected birds had increased water consumption, watery droppings causing wet litter, acute mortality, inability to walk and stand, depression, respiratory distress, apathy, ataxia, and opisthotonos. Analysis of feed detected concentrations of 0.9% sodium. The subsequent Israeli field cases was described in 6–9-d-old broiler chicks with increased water consumption, acute mortality, inability to stand or walk, depression, apathy, ataxia, and opisthotonos. The analysis of feed detected concentrations of 1.2% sodium. Macroscopic lesions described in the affected commercial broilers include swollen and edematous shanks, subcutaneous edema, enlarged hearts, hydropericardium, ascites, enlarged pale kidneys without increased urates, and pale, soft skeletal muscles. ST was also detected in 24-wk-old broiler breeders with increased water intake, and wet litter was reported; the sodium concentration detected in feed was 0.6%. The second broiler breeder ST case involved 45-wk-old broiler breeders experiencing an 8% drop in egg production, increased water intake, and mildly increased mortality. Grossly, swollen kidneys were noted. Feed sodium analysis detected 0.7% sodium. The addition of excess dietary sodium was attributed to technical problems at the feed mill in the 2015 Israeli cases, which ultimately affected >1,000,000 broilers and 50,000 broiler breeders.[9]

Discussion

Given the paucity of data of normal brain sodium concentrations in poultry, we analyzed brain sodium concentrations from 10 clinically normal broiler chickens. Brain sodium concentrations from the 10 clinically normal chickens were 1,500–1,700 ppm ($\bar{x}$ = ~1,600 ppm). These values align with the reported range of normal brain sodium in chickens of 1,600–1,700 ppm WW.[10]

We report here a case series of ST affecting commercial broilers and backyard chickens submitted to the CAHFS lab over a period of 9 y. Gross and microscopic lesions compatible with ST along with feed sodium concentrations exceeding the recommended values were detected in our case series. Although brain sodium concentrations exceeding 2,000 ppm WW has been documented as highly suggestive of ST,[10] only 2 of 24 brains in our case series exceeded this value and only 5 of 24 brains exceeded 1,700 ppm sodium. We hypothesize that the lower-than-expected brain sodium concentrations in most of our ST cases may be attributed to ad libitum drinking water access and the dilution of sodium from intracerebral edema in affected birds. This is also supported by the findings of experimental trials of ST in turkeys.[5] In these trials, when water was provided ad libitum, the mean and range of brain chloride concentrations in poults receiving 5% NaCl was not significantly different than controls fed 0.5% NaCl. In another group receiving 5% NaCl and restricted drinking water, brain chloride concentrations were significantly higher than both the group with ad libitum water access and the control group.[5] These findings indicate that salt analysis on representative feed samples may be a more reliable indicator of ST in suspect cases than brain sodium concentrations. Although serum and liver sodium analysis have also been described in diagnostic ST cases, these samples were not analyzed for sodium concentrations in our case series.

In our case series, gross or microscopic findings associated with dehydration or visceral gout were not detected. Mild mineralization in the lumens of scattered renal tubules was observed in our cases 1 and 3–5, without evidence of mineral deposition in other organs examined. The lack of visceral gout and histologic renal lesions of interstitial edema and congestion, nephrosis, distension of renal tubules with mild multifocal mineral deposits, and distended lumens of ureters is in accordance with studies on sodium intoxication occurring with access to drinking water.[12] The detection of additional pathogenic agents such as *Clostridium perfringens* producing necrotic enteritis, IBDV, *E. coli*, and *E. cecorum* were only occasionally identified in a few cases, and the exact role of these agents in exacerbating morbidity and mortality in ST-affected chicken flocks is unknown. Sodium concentrations exceeding recommended values in the feed samples analyzed in our cases series may have occurred due to errors in mixing feed ingredients at the feed mills.

Despite only mildly elevated feed sodium concentrations in cases 2 (2,500 ppm) and 3 (2,600 ppm), affected chickens had compatible and microscopic gross lesions of ST. Lower-than-expected feed sodium concentrations can occur if the feed analyzed was not a representative sample of the feed that initiated toxicosis. In addition, excess sodium intake may be attributable to alternative sources of sodium, such as drinking water additives. We recommend analysis of sodium concentrations in representative feed and water samples, in conjunction with assessing gross and microscopic postmortem findings, for the diagnosis of ST in poultry.

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