

MYCOTOXIN-INDUCED DISEASE IN CAPTIVE WHOOPING CRANES (*GRUS AMERICANA*) AND SANDHILL CRANES (*GRUS CANADENSIS*)

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Abstract: In 1987, an epizootic in cranes at the Patuxent Wildlife Research Center, Laurel, Maryland, USA, caused illness in 80% of 300 captive whooping cranes (*Grus americana*) and sandhill cranes (*Grus canadensis*) and death of 15 of these cranes. Gross pathology findings were inconclusive and consisted of dehydration, atrophy of fat, renal insufficiency, and small spleens. Extensive testing resulted in isolation of *Fusarium* sp. mold from constituents of the grain-based diet. Low levels of two mycotoxins, T² (1-2 ppm) and deoxynivalenol (0.4 ppm), were isolated from the pelleted feed.

Key words: Cranes, *Fusarium* sp., whooping crane, *Grus americana*, sandhill crane, *Grus canadensis*, deoxynivalenol, mycotoxin, trichothecene, T², vomitoxin.

INTRODUCTION

In September 1987 an epizootic with associated morbidity and mortality occurred in 80% of the captive cranes at the Patuxent Wildlife Research Center (PWRC), Laurel, Maryland, USA. At the time of the outbreak, PWRC housed 300 cranes, including 44 (20%) of the world's whooping cranes (*Grus americana*), 49 (40%) of the known Mississippi sandhill cranes (*G. canadensis pulla*), 126 Florida sandhill cranes (*G.c. pratensis*), 80 greater sandhill cranes (*G.c. tabida*), and one hybrid whooping crane × greater sandhill crane.

CASE REPORT

Clinical presentations

A juvenile Mississippi sandhill crane was found moribund on 18 September 1987. The bird was thin (2.6 kg) and had pale mucous membranes. The white blood cell count was elevated to 23,040 with a hematocrit level of 47% and total solids of 4.7 µg/dl. *Coccidia* (*Eimeria gruis*) and strongyles were present on fecal examination. Within 2 days another juvenile Mississippi sandhill crane and a juvenile Florida sandhill crane were found dead. The next day, two Florida sandhill cranes (one juvenile, one adult female) were observed to be weak, depressed, thin, dehydrated, and to have ruffled feathers. The

juvenile had a white blood cell count of 27,000, hematocrit of 37%, and total solids of 3.9 µg/dl. Fecal examination showed *coccidia* (*E. gruis*). The blood test results from the ill female and an apparently healthy male in the same pen were within normal ranges, and fecal examinations were negative for parasites. Suspecting an epizootic, 20 other juvenile cranes were examined, and 18 were found to have similar but less pronounced clinical signs of disease.

Physical examinations were subsequently performed on all the captive cranes and revealed that approximately 240 (of 300) had similar clinical signs of disease, including weight loss, dehydration, and necrosis of the tip of the tongue, and, in two cases, the tip of the beak. As birds became weak, they began to have difficulty moving and became ataxic when stressed. The weakest cranes were recumbent and generally died within 12 hr. The most severely affected birds exhibited a 10-20% weight loss.

Based on examinations and clinical pathology, supportive care was initiated. Because of the weight loss and dehydration, therapy included fluid replacement and nutritional supplementation. Cranes were gaged with a mixture of liquified crane pellets from a different mill date than those currently being used (Medium Crane Maintainer, 3/16", Zeigler Bros. Inc., P.O. Box 95, Gardners, Pennsylvania 17324, USA;

mention of manufacturer does not imply U.S. Government endorsement), liquid hypoallergenic soybean formula (Isomil Soy Formula, Ross Laboratories, Columbus, Ohio 43216, USA), a high-calorie dietary supplement (Nutri-Cal, Evsco Pharmaceuticals, Buena, New Jersey 08310, USA), mixed dry cereal (Mixed Cereal for Baby, Gerber Products Co., Fremont, Michigan 49413, USA), and vitamin powder (Vionate for Pets, ARC Laboratories, P.O. Box 18884, Irvine, California 92713, USA). Subcutaneous injections of fluids (and intravenous injections in the weakest birds), primarily 2½% dextrose with half-strength lactated Ringer's solution, were given to the dehydrated cranes at the rate of 3% of body weight per day. Fluids calculated to restore hydration were divided into four doses over a 48-hr period. Vitamin B complex (Phoenix Scientific Inc., St. Joseph, Missouri 64506, USA) was given as an adjunct to the fluid therapy. Cranes with intestinal nematodes were given a single treatment with ivermectin (10 µg/kg, Ivomec, Merck & Co. Inc., Rahway, New Jersey 07065, USA). Because of the elevated granulocyte counts in many of the early cases, antibiotic therapy consisting of ampicillin injections (100 mg/kg b.i.d. i.m. for 5 days, Polyflex, Aveco Co. Inc., 800 5th Street N.W., Fort Dodge, Iowa 50501, USA) was given to all birds being treated with the other supportive care. Fifteen cranes (5% of the captive population), including four members of endangered species, had died by October 5, and the remaining cranes had returned to normal. During the 17-day period (September 18–October 5), 240 cranes had received therapy.

Clinical Pathology

Blood was collected from 80 of the cranes during examinations. The most significant finding was an elevation of the serum uric acid concentration above 17 µg/dl in 31.3% of the cranes.³ Young Mississippi sandhill and greater sandhill cranes had a higher incidence of elevated serum uric acid. Ele-

vated uric acid concentrations (29.5 and 38.2 µg/dl) were seen in two of the three whooping cranes that died. Eight other cranes with high initial serum uric acid concentrations were rechecked 5 days post-treatment, and only one still had an elevated concentration (>17 µg/dl).

Hematologic evaluation showed elevations in the granulocyte counts in most sick birds. Three juvenile cranes, seen early in the course of the epizootic, had highly elevated granulocyte counts (>40,000). Hematocrit levels on all cranes tested were initially within the reference range of 38–48%.³ The erythrocytes were mature with little evidence of enhanced erythropoiesis. Follow-up blood samples taken 5–7 days later showed a decrease in hematocrit levels (to the 30–35% range), and erythrocyte morphology had shifted to more immature cell types.

Pathology

Of the 15 cranes that died, complete necropsies were performed on 13. The gross pathologic changes generally reflected debilitation with some lesions related to secondary complications. The first crane found dead, a Florida sandhill crane found on 19 September 1987, had no unusual external signs. Internally there was severe visceral gout with diffuse mineral deposits over the liver capsule, air sacs, pericardial sac, and epicardial surface of the heart. Extensive mineral deposits were found throughout the kidneys, liver parenchyma, and splenic parenchyma, with lesser amounts in the tracheal adventitia and submucosa. Articular gout was found in the shoulder and stifle joints. Joint surfaces and capsule were intact but covered with thick chalky deposits. Additional mineral deposits coated the pleural lung surfaces near the air sac orifices, myocardium, and muscles of the thighs. The esophagus and proventriculus were empty, while the ventriculus contained a small amount of yellow ingesta mixed with grit. Abundant creamy, pink material was found throughout the intestines. The colonic mu-

cosa was severely hyperemic in longitudinal streaks. Subcutaneous, abdominal, and coronary fat deposits were normal, as was pectoral muscle development. No lesions were seen in the brain, gall bladder, testes, adrenal glands, thymus, thyroid glands, or pancreas. Very few coccidia oocysts (*E. gruis*) were found in the gastrointestinal tract. The most prominent histologic finding was nephrosis.

The second death was an 87-day-old Mississippi sandhill crane chick being reared by surrogate Florida sandhill crane parents and found dead on 20 September 1987. The keel was moderately prominent, and there was a thick, white material coating the tongue and mildly adherent to the mucosa surrounding the nasal choanae. A small white pustule was found on the margin of the glottis. The kidneys were diffusely severely pale and had a rubbery texture. Moderate urate deposits were found in the shoulder and stifle joints, with urates streaking skeletal muscles in the thighs. Mild urate deposits were found on the pericardial sac and submucosa of the trachea. Severe urate deposition occurred on left atrioventricular valves. Fat depositions in subcutaneous tissue, abdomen, and coronary groove were absent. The liver was firm and mahogany colored. The spleen was small. The esophagus and proventriculus were empty, while the proventricular mucosa was coated with cloudy mucous and had white mineral granules in a 3-cm-long streak on the mucosal surface. The underlying mucosa was severely reddened. The ventriculus contained grit and a small amount of cracked grain. Intestinal contents were creamy and semisolid. No lesions were seen in the brain, cardiac muscle, lungs, air sacs, liver, gall bladder, esophagus, ovary, adrenal glands, bursa, thyroid glands, thymus, or pancreas. Hepatocellular atrophy and nephrosis were found on histologic examination.

The next death occurred on 22 September 1987 in a 2-yr-old Florida sandhill crane. A moderately prominent keel and mild right lateral beak deviation were the only external

observations. A slit-like, 8-mm-long skull fracture over the caudal right cerebral hemisphere had perforated the dura and the right cerebral hemisphere. However, there was no cerebral or dural hemorrhage, possibly indicating that the skull fracture was a post-mortem lesion caused by one of the other cranes in the pen pecking at this crane's head. There was a lack of subcutaneous, abdominal, and perineal fat. Mild petechial hemorrhage occurred in the coronary fat and subcutaneous tissue of the legs. The proventriculus contained some cloudy mucous. Grit and dense, yellow/green fibrous material was found in the ventriculus. Several small, unidentified nematodes and an *Acanthocephalia* nematode were found in the gastrointestinal tract. No lesions were seen in the skeletal muscle, heart, pericardial sac, heart valves, trachea, lungs, air sacs, liver, gall bladder, ovary, oviduct, adrenal glands, kidneys, spleen, thyroid gland, thymus, or pancreas.

Also on 22 September 1987 a young Florida sandhill crane was found recumbent in its pen. It was removed to the hospital, where it received treatments, including intravenous fluids, but it died the next day. External findings included a reddening and abrasion around the nares and a moderately prominent keel. Internally there were a mild hemorrhage along the jugular furrow (possibly from intravenous injections) and a lack of subcutaneous, abdominal, coronary, or perirenal fat. Several nematodes were found in the ventriculus and the intestines. The esophagus, proventriculus, ventriculus, and upper third of the intestines were empty, while the lower third of the intestines contained abundant watery, brown material. Spleen, liver, and bursa were all small. The kidneys had a milky gray capsular surface and a dark red cut surface that was mildly edematous. No lesions were seen in the brain, musculoskeletal system, heart, pericardial sac, heart valves, trachea, lungs, air sacs, testes, thyroid glands, thymus, or pancreas.

Another young Florida sandhill crane was

found dead in its pen on 24 September 1987. The keel was severely prominent. Approximately 10 ml of watery brown fluid was found in the abdominal cavity. There was 5 ml of watery, pale brown fluid in the esophagus and proventriculus. There was a 1-cm-diameter pale and mildly hyperemic plaque on the mucosa of the proventriculus. The intestines contained watery, tan to brown fluid with flocculent brown material toward the caudal small intestine. The colon was severely distended with abundant fetid, watery, brown fluid. Both the spleen and liver were small. Caudal kidney lobes were mildly hyperemic, and adrenal glands mildly reddened. There was abundant fluid in the lungs along the costal attachments, and pink foamy fluid was seen on cut lung surfaces. There was a lack of subcutaneous, abdominal, coronary, and perirenal fat. All subcutaneous tissues and muscles had a sticky, dry texture. Hepatocellular atrophy, vacuolation in cerebellar neurons and kidney tubular cells, and swollen Kupfer cells were seen on histologic examination. *Salmonella* sp. was isolated from the gastrointestinal tract. No lesions were seen in the brain, skeletal system, joints, heart pericardial sac, heart valves, trachea, testes, thyroid glands, or pancreas.

On 24 September 1987 a 20-yr-old male whooping crane was found depressed, emaciated, dehydrated, and weak. There seemed to be a slight improvement with initial treatments, but the bird died the next day. External findings included bilateral reddened conjunctiva and a severely prominent keel. Severe diffuse edema was found over the head and cervical region with more moderate edema over the remainder of the body. Mild hemorrhage was seen over the jugular furrow. Petechial to ecchymotic hemorrhage was seen in the subcutaneous tissues of the inguinal area. Muscles in the pectoral and femoral regions were mottled with pale patches. There was a lack of subcutaneous, abdominal, coronary, and perirenal fat. There was a moderate amount of blood-tinged fluid within the abdominal air sacs.

Lungs were wet and exuded abundant watery pink fluid. The intrasternal portion of the trachea was reddened and contained a small amount of blood-tinged fluid plus flocculent, pasty, ingesta-like material. The pericardial sac was opaque and contained 3 ml of yellow fluid. The liver was pale, mottled, and congested. The kidneys had a pale mottled surface with faint urate deposits throughout the parenchyma. The spleen was small. The ventriculus muscle showed moderate petechial and ecchymotic hemorrhage. A small amount of pasty tan material was found in the cranial two-thirds of the intestine, while the caudal third contained fluid and brown mucoid material. The distal intestinal mucosa was hyperemic. Histologic findings included vacuolation of kidney tubular cells and cellular swelling in the liver. No lesions were seen in the brain, skeletal system, heart, heart valves, gall bladder, testes, thyroid glands, or pancreas.

On 25 September 1987 three additional cranes died. The first was a juvenile female Florida sandhill crane. The keel was moderately prominent, and pasty tan material was found coating the oral cavity. There was severe subcutaneous edema over the abdomen. A soft, multilobulated 1-cm nodule was found attached to the periosteum of the left humerus. The right clavicular air sac was mildly coated with yellow opaque material. The pericardial sac was opaque and contained a small amount of straw-colored fluid. The lungs were edematous at their costal attachments. Unclotted blood was found in both the lungs and heart. The spleen was mildly enlarged with a deep red parenchyma and prominent lymphoid follicles. The kidneys were swollen and moderately friable. The ventriculus was distended with grit, grain, and gray-yellow ingesta. There was mild serosal congestion of the intestines, with small amounts of watery material and mucous. The ceca were distended with granular yellow fluid, and the wall was diffusely hyperemic. Abundant semisolid, yellow feces was seen in the cloaca. There was a lack of subcutaneous, abdominal, cor-

onary, and perirenal fat. No lesions were seen in the brain, skeletal system, heart, heart valves, trachea, air sacs, ovary, adrenal glands, bursa, thyroid glands, or pancreas.

The second was a 5-yr-old male greater sandhill crane. The bird was weak on 24 September 1987, when intensive therapy, including intravenous infusions, was begun. The crane failed to respond and died the next day. The feathers surrounding the vent were soiled with grey and white feces, and the keel was moderately prominent. Massive subcutaneous edema was found over the head and neck, and there was some edema over the ventral torso. There was a gelatinous layer of edematous tissues along the costal area of the lungs, and each lung exuded abundant watery fluid when removed. Both the spleen and liver were small. Kidneys were pale brown with moderate accumulation of yellow-white urates within the ureters. The esophagus was mildly edematous, and the proventriculus was empty. Some grain and grit were found in the ventriculus. The intestines were turgid, filled with mildly fluid material in the cranial 10 cm and becoming more pasty and crumbly afterwards. No lesions were seen in the brain, skeletal system, heart, heart valves, trachea, gall bladder, testes, adrenal glands, thyroid glands, or pancreas.

The third crane that died on 25 September 1987 was a 3-yr-old male Florida sandhill crane found in its pen. There was a lack of subcutaneous, abdominal, and perirenal fat. The coronary fat was greatly reduced and had undergone partial serous atrophy. Subcutaneous tissues and muscle had a dry, sticky texture. There were petechial hemorrhages in the dura over the cerebral hemispheres, but no lesions were seen in the hemispheres. The spleen was small and dark red/brown. The liver was firm and enlarged, and the gall bladder was distended with slightly mucoid, green/yellow bile. The esophagus and proventriculus were empty except for some strands of grass. The ventriculus was severely distended with freshly ingested granular tan/yellow material and

cracked grain. Intestinal contents were watery, becoming fetid and watery in the caudal third, including the colon and cecum. There was some autolysis of the abdominal tissues. No lesions were seen in the skeletal system, skeletal muscle, heart pericardial sac, heart valves, trachea, lungs, air sacs, testes, adrenal glands, thyroid glands, or pancreas.

On 26 September 1987 a 4-yr-old male whooping crane died. The bird was found down in its pen two days previously and treated. There was initial improvement following the onset of therapy, but the bird's condition later worsened. The keel was moderately prominent. There was diffuse subcutaneous edema over the thorax and abdomen. There was a lack of subcutaneous, abdominal, and perirenal fat. The coronary fat had undergone serous atrophy. Mild fluid accumulation was present at the junction of the lungs and their costal attachments. The spleen had prominent lymphoid follicles. There was a 2-cm-long, ischemic hemorrhage along the capsular surface of the right liver lobe. The gall bladder was distended with slightly mucoid, green/yellow bile. The esophagus, proventriculus, and ventriculus contained abundant watery fluid material (ingesta). The intestines were turgid and contained more water fluid with flocculent white and yellow material. The cecal walls were reddened, and two small petechial hemorrhages were seen. Histologic findings were renal tubular necrosis, vacuolated tubular cells, and swollen Kupfer cells. No lesions were seen in the brain, skeletal system, heart, pericardial sac, heart valves, trachea, lungs, air sacs, testes, adrenal glands, thyroid glands, or thymus.

On 26 September 1987 a 7-yr-old Florida sandhill crane was found dead. The keel was moderately prominent. There was a lack of subcutaneous, abdominal, and perirenal fat. The coronary fat had undergone serous atrophy. The wall of the terminal esophagus was severely and diffusely hemorrhagic and swollen. A 2-cm-diameter area of hemorrhage was seen on the mucosa of the pro-

ventriculus. The upper quarter of the small intestine was massively dilated and flaccid, filled with fetid, watery, brown fluid. There were numerous petechial hemorrhages on the intestinal wall in this area. Intestinal contents became more solid, approaching a claylike firm mass in the caudal two-thirds of the intestines. The ceca were distended with watery brown fluid and urates. The cloaca contained abundant watery tan fluid. The spleen was small but had prominent lymphoid follicles. Approximately 2 ml of straw-colored fluid was present in the pericardial sac. Coccidia were present in the intestines. Esophageal, proventricular, and hepatocellular atrophy was observed along with vacuolation in cerebellar neurons and hemorrhage in the intestinal submucosa. No lesions were seen in the brain, skeletal system, skeletal muscle, heart, heart valves, trachea, lungs, air sacs, testes, adrenal glands, thyroid glands, or pancreas.

Four additional cranes died between 27 September and 1 October 1987. The findings for these cranes were similar to those described above. During the same period, only one other animal died in the research colonies (approximately 2,500 animals at the time). This death occurred in the kestrel colony and was due to trauma.

Laboratory results

Extensive testing of the food, water, and environment of the cranes was initiated shortly after the onset of the epizootic and continued for several years. Analysis of the pelleted feed for nutrients, vitamins, and trace elements was conducted by the National Food Laboratory (6363 Clark Avenue, Dublin, California 94568, USA). In addition, the Patuxent Analytic Control Facility conducted a mass spectrophotometer analysis of the pelleted feed and the water for heavy metals (Pb, Hg, As, Cd, Se) and pesticides (organophosphates, chlorinated hydrocarbons, carbamates, polychlorinated biphenyls, and herbicides). Further screening of the water supply was performed at

the Environmental Testing Laboratory (1993 Moreland Parkway, Annapolis, Maryland 21401, USA). Screening of grasses and spilled old feed for aflatoxins and *Fusarium* spp. toxins was performed by the Department of Botany, University of Maryland (College Park, Maryland 20742, USA). All of the above tests were negative.

A series of bioassays of the pelleted diet using sandhill cranes (J. Carpenter, unpubl. data) and northern bobwhites (*Colinus virginianus*) (E. Hill, unpubl. data) were conducted and demonstrated a problem in the feed. Bobwhites offered the pelleted food refused to eat. Although most cranes refused to eat the pelleted food, some ate food or were gavaged with the diet and subsequently died with lesions similar to those found in cranes during the epizootic (two of six fed contaminated feed and all three gavaged with contaminated feed mixed with water died, J. Carpenter, unpubl. data). Therefore, further testing of the pelleted diet was indicated to determine the causative agent.

Samples collected from the gravity feeders and from unopened bags of feed were found to contain 1–2 ppm of T² mycotoxin (J. Richard, pers. comm.), 0.4 ppm deoxynivalenol (DON) (H. Casper, pers. comm.), and unquantified amounts of *Fusarium* sp. and *Aspergillus* sp. (P. Nelson, pers. comm.). The T² was identified in feed samples using thin-layer chromatography. The DON was identified using a basic analytic chemical method of methanol/water extraction followed by extraction with ethylacetate, florisil cleanup, trimethylsilylimidazole derivation, and analysis with a mass spectrophotometer.

DISCUSSION

Mycotoxins produced as secondary metabolites of molds, primarily *Fusarium graminearum*, will grow on corn in field situations. The amount of mycotoxin formation is dependent on environmental factors such as low temperature, high humidity, plant stress, and damage to the corn kernels. Deoxynivalenol is one mycotoxin produced

by a number of *Fusarium* spp.⁹ Death of snow geese (*Chen caerulescens*) and white-fronted geese (*Anser erythropus*) has been associated with DON levels of <5 ppm in moldy corn and 1–5 ppm in ingesta.⁵

Fusarium spp. mycotoxins have been cited as causing large-scale natural mortality in cranes.^{10,11} Clinical findings with *Fusarium* sp. have included progressive loss of motor control to the neck, wings, and legs. Pathologic findings include hemorrhage, granulomatous myositis, thrombosis, and vascular degeneration.¹⁰ Clinical pathology showed elevated serum levels of creatinine kinase, aspartate aminotransferase, and alanine aminotransferase. Even though *Fusarium* of three different species was found in the corn from which our pelleted food was prepared, the clinical signs and pathology seen at Patuxent were not consistent with earlier reports of mycotoxicosis in wild cranes. A tricothecene mycotoxin, probably T², was suspected but not identified in the earlier study of wild cranes.¹⁰

Deoxynivalenol is also a tricothecene mycotoxin, but most information about DON is for poultry; none was found for cranes. Signs of toxicity include feed refusal, vomiting, and hemorrhagic enteritis accompanied by a drop in general performance.⁷ The pelleting process had no effect on the level of DON found in the finished feed. After feeding chickens 0.02–1.87 ppm DON from hatch for 28 days, no significant effects on mortality were found.⁷

In short term oral study of DON with chickens, gasping, lethargy, assuming squatting position, and dropping of the wings and head were seen.⁶ Birds would later raise and shake their heads and swallow frequently, a behavior attributed to irritation of the upper gastrointestinal tract. Birds developed diarrhea and refused to eat or drink. Necropsy findings included ecchymotic hemorrhage throughout the intestinal tract, kidneys that appeared normal but actually had extensive urate deposits and visceral gout, crops distended with gas, and necrotic gizzard linings. Many of these findings are similar to

what we found in cranes. Our birds were often found dead in a squatting position (sternal recumbency). Gastrointestinal signs including diarrhea, gas in the intestines, and hemorrhagic enteritis were seen in our cranes. Also prominent in several cases was evidence of kidney malfunction in the form of extensive urate deposits (visceral and articular gout).

Deoxynivalenol is reported to be a sex-linked immunotoxicant in young chickens.⁴ In the cranes examined, five females and eight males died. Deoxynivalenol-contaminated wheat and corn used in poultry diets causes enteritis and mortality in chickens infested with *Eimeria* spp. Captive cranes at PWRC have always had low levels of *Eimeria grus* and *E. riechenowi*. In the late 1980s these levels increased due to a suspected resistance to the coccidiostat amprolium in use at that time.² However, *Eimeria* spp. was found in only one crane that died in this epizootic.

Significant decreases were found in hemoglobin, hematocrit, total red blood cells, magnesium, and sodium in male leghorn chicks fed DON-contaminated feed at relatively high levels (9 and 18 ppm) compared with controls (0 ppm).⁸ Hematocrit levels in the PWRC cranes were normal or lower than expected.

The 0.4-ppm concentration of DON was lower than the level generally shown to cause clinical disease and death in poultry. However, cranes are unusually sensitive to a number of disease agents. The immune system of cranes may not be as developed as those of other birds. Parasites, especially coccidia, were known to exist in the colony at low to moderate levels and may have exacerbated the toxicity of DON in some cranes. It is not known whether T² may have also interacted with DON. Clearly, additional research on such possibilities is needed.

Following the 1987 epizootic, protocols were instituted to bioassay all grain-based feeds used on research animals, including cranes, at PWRC. Storage conditions for

grain-based feeds have been improved and are monitored closely, and there has not been a repeat of the 1987 incident in spite of the continued problems with mycotoxins in all grain-based animal feeds.

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