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Fatal Envenomation of a Chilean Flamingo (*Phoenicopterus chilensis*) From Eastern Yellow Jacket Wasps (*Vespula maculifrons*)

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Abstract: A 37-year-old, female Chilean flamingo (*Phoenicopterus chilensis*) presented with severe facial angioedema, bilateral corneal and palpebral edema, nictitating membrane paralysis, bradycardia, bradypnea, hypothermia, and numerous stingers and remnants of eastern yellow jacket wasps (*Vespula maculifrons*) attached to the feathers of the head, palpebrae, and conjunctiva. Evaluation of 2 complete blood cell counts and results of plasma chemical analysis and serum protein electrophoresis revealed severe increases in creatinine phosphokinase and aspartate aminotransferase activity, electrolyte disturbances, and moderate increases in levels of α_1 , α_2 , β_1 , and γ immunoglobulins when compared with reference interval values and conspecifics. Despite intensive treatment, the bird died 19 hours after presentation. Results of histologic evaluation of tissues were compatible with envenomation. Response to envenomation in avian species is not documented but should be considered in birds presenting with angioedema.

Key words: anaphylaxis, envenomation, eastern yellow jacket wasp, *Vespula maculifrons*, serum protein electrophoresis, avian, Chilean flamingo, *Phoenicopterus chilensis*

Clinical Report

A 37-year-old, female Chilean flamingo (*Phoenicopterus chilensis*), weighing 2.8 kg, housed with 12 conspecifics and 7 lesser flamingos (*Phoenicopterus minor*), presented with a 45-minute history of being found recumbent in a shallow exhibit pool. The flamingos were housed in an open-air outdoor natural exhibit with an indoor holding facility and were maintained on a commercial flamingo diet. Physical examination demonstrated 3 dead wasps of the order Hymenoptera and numerous stingers in the integument of the forehead, nape, cranial cervical integument, chin, cheek, intermandibular integument, bulbar conjunctiva, palpebra, rhamphotheca, and cere. Two additional wasps were noted in the oral cavity, but no stingers were present. Edema of the oropharyngeal tissues was observed, but the glottis appeared normal. In addition, several

mats of calliphorid eggs were noted among the feathers of the cranium. Severe angioedema of the head and palpebral edema was observed (Fig 1). The bird was initially unresponsive with a cardiac rate of 180 beats/min and a respiratory rate of 4 breaths/min. Harsh pulmonary sounds were auscultated in the air sacs. Bilateral corneal edema and apparent paralysis of the third eyelid were observed. There was no indirect or direct pupillary response in either eye. Body temperature was below detectable levels with a cloacal thermometer.

Blood samples were obtained from the medial metatarsal vein for a complete blood cell count (CBC) and select plasma biochemical analysis and serum protein electrophoresis. Abnormal results of the CBC were lymphopenia and elevated packed cell volume (PCV) (Table 1). No eosinophils or basophils were observed. Increased aspartate aminotransferase (AST) activity, hyperphosphatemia, hypercalcemia, hypoglycemia, hypoproteinemia, hyponatremia, hypochloremia, and hyperkalemia were observed (Table 1). Elevation of α_1 , α_2 , β_1 , and γ -immunoglobulins (Ig) were

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Figure 1. Severe angioneurotic edema in a Chilean flamingo caused by envenomation from eastern yellow jacket wasps. Note the overall swelling of the head and periocular tissues, remnant stingers (S), and palpebral edema (E).

noted when compared to 7 conspecifics of the same flock and limited reference intervals (Table 2).

A 22-gauge intraosseous (IO) catheter was placed in the right ulna, and warmed 2.5% dextrose in saline (9 mL/kg IO) with dexamethasone sodium phosphate (2 mg/kg IO) and diphenhydramine hydrochloride (3.5 mg/kg IO) were administered over 15 minutes. An additional amount of warmed 2.5% dextrose in saline (10 mL/kg SC) was administered. After 30 minutes, slowed third eyelid movements were observed, but the bird remained sternally recumbent. Topical ophthalmic triple antibiotic ointment with hydrocortisone (neomycin and polymyxin B sulfates, bacitracin zinc, and hydrocortisone acetate ophthalmic ointment) was applied in both eyes. Thirty-seven stingers were manually removed (Fig 2). The bird was placed in a standard incubator at 40°C, supplemented with 100% oxygen. The bird stood unassisted 12 hours after presentation but exhibited cervical rigidity. The bird was ataxic but did not fall. Reevaluation at that time revealed ecchymotic hemorrhage in the integument of the intermandibular space with significant edema (Fig 3). Additional dexamethasone sodium phosphate (5 mg IM) and diphenhydramine hydrochloride (5 mg IM) were administered.

Twelve hours after presentation, an additional blood sample was obtained from the medial metatarsal vein for repeat CBC, plasma biochemical analysis (Table 1), serum protein electropho-

Table 1. Hematologic data from a Chilean flamingo envenomated from eastern yellow jacket wasps.

Test	Initial	12-h after presentation	Reference range ²⁹
WBC, cells/ μ L	4500	8600	10 200 $\pm$ 9783
Heterophils, cells/ μ L	3600	5280	3050 $\pm$ 2874
Lymphocytes, cells/ μ L	675	1118	6633 $\pm$ 8453
Monocytes, cells/ μ L	225	132	415 $\pm$ 571
Eosinophils, cells/ μ L	0	0	603 $\pm$ 1279
Basophils, cells/ μ L	0	0	415 $\pm$ 571
Packed cell volume, %	58	38	44.9 $\pm$ 5.1
Albumin, g/dL	1.2	1.1	1.6 $\pm$ 0.5
AST, U/L	2368	4228	212 $\pm$ 110
Calcium, mg/dL	15.6	9.6	11.1 $\pm$ 1.5
Chloride, mEq/L	92	104	111 $\pm$ 15
Cholesterol, mg/dL	152	55	320 $\pm$ 90
CPK, U/L	—	70 557	510 $\pm$ 655
Globulin, g/dL	3.0	2.0	3.7 $\pm$ 0.8
Glucose, mg/dL	18	445	196 $\pm$ 46
Phosphorous, mg/dL	23.4	8.7	8.5 $\pm$ 12.4
Potassium, mEq/L	15.8	5.9	2.8 $\pm$ 0.8
Sodium, mEq/L	124	133	156 $\pm$ 7
Total protein, g/dL	4.2	3.1	5.2 $\pm$ 0.9
Uric acid, mg/dL	—	49.4	5.5 $\pm$ 2.6

Abbreviations: WBC, white blood cells; AST, aspartate aminotransferase; CPK, creatinine phosphokinase.

Table 2. Results of prothrombin time, activated partial thromboplastin time, prothrombin time, and serum protein electrophoresis in an envenomated Chilean flamingo (F1) compared with a conspecific group of 7 Chilean flamingos (F2–F8).

Value	F1 _i	F1 _{12h}	F2	F3	F4	F5	F6	F7	F8
PT, s	—	>100	>100	>100	>100	>100	>100	42.7	>100
aPTT, s	—	>100	>100	78.4	>100	>100	>100	>100	>100
TP, g/dL	4.2	3.1	5.1	5.5	5.8	6.4	6.1	6.7	6.0
Alb, g/dL	1.2	1.1	3.44	3.32	3.92	4.24	3.94	4.21	4.15
Glob, g/dL	3.0	2.0	1.66	2.18	1.88	2.16	2.16	2.49	1.85
α_1 , g/dL	0.53	0.16	0.2	0.15	0.14	0.22	0.28	0.16	0.16
α_2 , g/dL	0.56	0.67	0.31	0.4	0.45	0.47	0.52	0.39	0.44
β_1 , g/dL	0.74	0.76	0.34	0.58	0.6	0.58	0.52	0.9	0.57
γ , g/dL	1.18	0.70	0.82	1.0	0.69	0.88	0.84	1.05	0.68

Abbreviations: PT, prothrombin time; aPTT, activated partial thromboplastin time; TP, total protein; Alb, albumin; Glob, globulin; F1_i, initial; F1_{12h}, 12 hours after presentation.

resis (SPE), prothrombin time (PT), and activated partial thromboplastin time (aPTT). The results of the SPE, PT, and aPTT were then compared with results from 7 conspecifics (Table 2). Increased AST activity, hyperglycemia, and extreme increase in creatinine phosphokinase (CPK) activity were found. The PCV, phosphorous, calcium, and chloride values had returned to within reference intervals. Hypoproteinemia, hypoglobulinemia, and hypoalbuminemia were documented. Hyperkalemia and hyponatremia persisted but were improved (Table 1). The results of the SPE demonstrated a decrease in α_1 Ig and an increase in α_2 Ig levels. The results of the PT and aPTT were greater than 100 seconds (Table 2). Frank hemorrhage was observed in a urate specimen and confirmed with an occult-blood test (Hematest, Fisher Scientific, Waltham, MA, USA). The bird was more alert, but ventroflexion

of the head was observed. Fluid therapy was repeated as before. The heart rate had increased to 224 beats/min, and respirations were 12 breaths/min. The bird became sternally recumbent again, and furosemide (2 mg/kg IO) was administered with prednisolone sodium succinate (10 mg/kg IM). An additional blood sample was obtained 7 hours later for measurement of total protein (TP) and PCV, and results were 4.9 g/dL and 45%, respectively, with a whole-blood glucose reading of >500 mg/dL. The bird died 19 hours after presentation.

Representative tissue samples from all organs were obtained, placed in 10% neutral-buffered formalin, embedded in paraffin, sectioned at 5 μ m and stained with hematoxylin and eosin. Histopathologic examination demonstrated extensive foci of acute necrosis and marked interstitial hemorrhage and edema in the cervical muscles.



Figure 2. Compilation of eastern yellow jacket remains and stingers removed from a Chilean flamingo (bar = 7 mm).



Figure 3. Ecchymotic hemorrhage and edema of the intermandibular skin of a Chilean flamingo from envenomation by eastern yellow jackets.

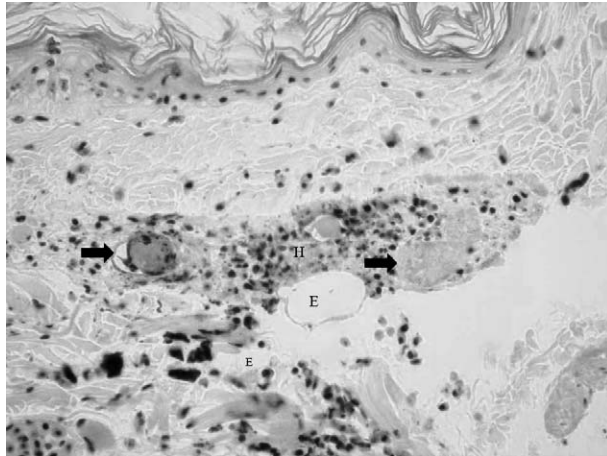


Figure 4. Photomicrograph of feathered skin from a Chilean flamingo envenomated by eastern yellow jackets. Note areas of subcutaneous edema (E) and thrombosis of superficial dermal vessels (arrows) and infiltrates of degenerate heterophils (H) (hematoxylin and eosin, ×400).

Marked edema was observed in sections of feathered skin obtained from the cranium with foci of vascular thrombosis and localized infiltrates of heterophils and few lymphocytes in the superficial dermis (Fig 4). Bulbar and palpebral conjunctiva demonstrated multifocal moderate foci of edema and marked infiltrates of heterophils and few lymphocytes and macrophages (Fig 5). The corneas had diffuse moderate fibrosis and edema with foci of necrosis and infiltrates of heterophils within the superficial stroma. The pulmonary tissues demonstrated diffuse marked acute congestion and marked air capillary edema. The epicardium had multifocal moderate infiltrates of macrophages and mild foci of granulation tissue proliferation. Accumulation of granular, pale basophilic material interpreted as urates was present within these foci (epicardial gout). The mesentery also demonstrated multifocal marked foci of acute hemorrhage with some areas demonstrating brightly eosinophilic globular material interpreted as yolk. Moderate foci of subacute inflammation were present in the portal areas of the liver. Extensive areas of acute hemorrhage and myonecrosis appeared to be related to envenomation.

Remains of 2 wasps were preserved in 90% isopropyl alcohol and sent in commercial-grade white vinegar for species identification (Department of Entomology, Kansas State University, Manhattan, KS, USA). The wasps were positively identified as the eastern yellow jacket (*Vespula*

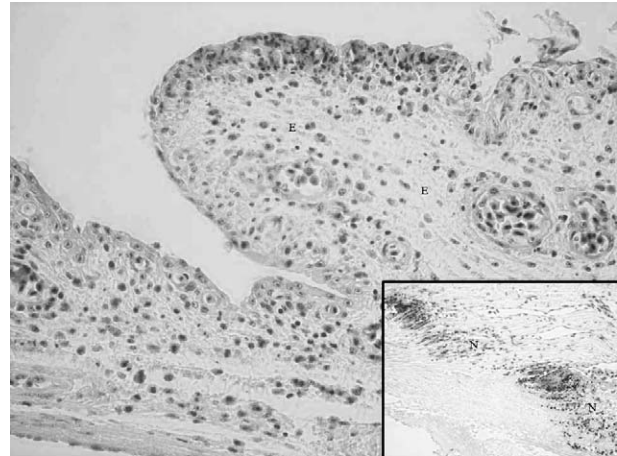


Figure 5. Eye of a Chilean flamingo envenomated by eastern yellow jackets. Extensive edema (E) with marked infiltrates of heterophils are present in the bulbar and palpebral conjunctiva (hematoxylin and eosin, ×400). Inset: Marked necrosis (N) and heterophilic inflammation in the stroma of the corneal limbus (hematoxylin and eosin, ×100).

maculifrons) based on taxonomic keys (H. Davis, written communication, December 2008).

Discussion

Envenomation due to eastern yellow jacket wasps caused the death of this bird. Characterization of the immune response to envenomation demonstrated increases in Ig, AST activity, CPK, electrolyte disturbances, angioedema, congestion, and hematuria similar to what is observed in mammals.^{1–3} However, the mechanism by which mediation of these responses occurred is unexplained.

Yellow jackets are members of the order Hymenoptera (bees and wasps) and are well known for their aggressive response when provoked.⁴ In addition, these wasps emit an alarm pheromone during envenomation, which attracts additional workers to attack.⁵ Three venom components—hyaluronidase, mastoparan, and phospholipase A1—have been demonstrated to stimulate prostaglandin E₂ and weakly enhance IgE and IgG1 in mice,^{6,7} and these may have stimulated the humoral response in this flamingo. Immunoglobulins documented in birds are IgG, IgM, and IgA.⁸ Immunoglobulin E is not found in birds but is a major inflammatory mediator in mammals.^{9,10} Yellow jackets construct subterranean nests in the spring, and colonies expand to include up to 2800 individuals by the end of summer.¹¹ Several yellow jacket nests were found

in the exhibit housing the flamingos. This bird had been observed to have a corneal scar in the right eye (OD) 5 years before presentation; a lenticular cataract with anterior synechiae was noted OD, and a lenticular cataract with anterior synechia in the left eye (OS) was found 4 years before presentation. Visual impairment may have led this bird to stumble into the path of a yellow jacket nest, invoking an immediate attack.

Initial hematologic values demonstrated increased AST and CPK activities, hypoproteinemia, hyperphosphatemia, hypercalcemia, hypoglycemia, and hyperkalemia. These values were compatible with tissue damage from envenomation and increased cellular permeability.¹² Phospholipase A causes red cell hemolysis, which supports the finding of hemoglobinuria in the urates.¹³ Hemolysis also allows intracellular potassium and phosphate to escape into the circulation, leading to hyperkalemia and hyperphosphatemia. Hyperkalemia depresses cardiac function, as observed clinically. Hyperuricemia was compatible with the histopathologic finding of epicardial gout. Eosinophilia and basophilia may be expected with envenomation hypersensitivity^{14–17} but were not observed in this case. Type I hypersensitivity reactions, typical of envenomation in mammals,^{17,18} are mediated by IgE isotypes that attach to basophils, which induces the release of vasoactive components, including histamine.^{13,17,19} The mechanism for angioneurotic edema observed in this bird remains unexplained, although vascular permeability likely contributed to the swelling.

Comparative blood samples for SPE were collected from 7 conspecific flamingos. Elevated α_1 , α_2 , β_1 , and γ -globulin levels and lower values for TP and albumin were observed when compared with conspecific birds (Table 2) and are compatible with an inflammatory reaction. Loss of albumin would be consistent with permeability of cell membranes caused by envenomation. Additional samples from 7 conspecifics were evaluated for PT and aPTT (Table 2). Most birds had PT and aPTT levels greater than 100 seconds. This diagnostic modality may not be useful in flamingos for evaluating clotting times through commercial laboratories unless species-specific thromboplastin controls are available.²⁰ Initial bradycardia, bradypnea, and recumbency were likely related to effects of envenomation on vascular endothelium, peripheral nerve endings, and myoneural junctions,²¹ as opposed to an allergic or anaphylactic reactions, which generally demonstrate tachycardia.²² Previous heart rates during routine examination in this bird averaged 320 beats/min.

Corticosteroids, antihistamines, and furosemide was used to alleviate reaction to envenomation. Doses were based on reference standards.^{23–26} An additional treatment that could be used is epinephrine, which provides sympathomimetic effects and may have reversed bradycardia and bradypnea.²⁷ Survey radiographs were not performed because of the instability of the flamingo at the time of presentation.

Hymenoptera stings account for more human fatalities than any other venomous bite or sting.^{10,28} Unique hypersensitivity accounts for severe systemic allergic reactions, which can be associated with any number of stings, or direct toxic reaction from multiple stings,^{10,18} as documented in this bird. In dogs, the lethal dose of hymenoptera stings is reported to be 20 stings/kg body weight.¹⁸ In this case, it is a reasonable conclusion that there were more than 37 stings to this bird because wasps are capable of stinging multiple times,¹³ and not all stings were likely observed.

Response to envenomation in avian species is not documented but should be considered in birds presenting with angioedema and elevated immunoglobulin levels.

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