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J Vet Diagn Invest 16:321–325 (2004)

Effect of feeding green onions (*Allium ascalonicum*) to White Chinese geese (*Threskiornis spinicollis*)

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Abstract. Sudden increase in mortality was observed in 2 different flocks of mature breeder geese fed green onions. At necropsy, birds had pale epicardium with random petechiation, sanguinous fluid accumulation in the pericardial sac, and mild swelling of the liver and spleen. Histologically, there was accumulation of hemosiderin in hepatocytes, Kupffer cells of the liver, macrophages, and renal tubules. There was also moderate to severe hepatic necrosis, vacuolation of hepatocytes, splenitis, and renal tubular nephrosis. To assess the effects of green onion ingestion, 2 feeding trials were carried out in 3 mature White Chinese geese. In the first trial, onions were thoroughly mixed with pellet maintenance ration. In the second trial, onions were offered in a separate trough from the pelleted diet. During the 21 days of experiments, the red blood cell count and hematocrit decreased, whereas the polychromasia and reticulocyte estimate increased. The blood changes were more marked in birds from the second feeding trial. Gross and histologic changes were similar in both trials. Mild swelling and severe darkening of the liver were the only significant findings at necropsy. Histologically, the liver looked similar to that seen from the field outbreak. The liver contained moderate amounts of hemosiderin in the hepatocytes and Kupffer cells, and had centrolobular necrosis and vacuolation of hepatocytes. This experimental study demonstrated that anemia and liver pathology could be caused by ingestion of onions. Furthermore, Heinz bodies are not a consistent finding in the blood of geese fed onions.

In areas where onions are grown commercially, it is common practice to use culled onions as a source of feed for livestock. Because the ingestion of onions has been associated with hemolytic anemia accompanied by the formation of Heinz bodies within the erythrocytes in cattle, cats, horses, dogs, and sheep,¹⁴ most farmers use onions in strictly limited quantities. However, there are no reports describing the effects of onions in avian species. This brief communication describes the pathology found in 2 independent goose flocks associated with ingestion of green onions and the re-

sults from an experimental trial in which geese were fed this vegetable.

Sudden increase in mortality was observed in 2 different flocks of mature breeder geese. The first case occurred in November 2001, where 6% mortality was observed in a flock of 1,400 breeders of 11 different goose breeds. Most of the birds (95%) that died were White Chinese geese (*Threskiornis spinicollis*). The second case was in August 2002, and mortality as high as 8.4% was observed in a 2-day period in a flock of White Chinese geese (634 birds). In both cases, birds had been fed a pelleted maintenance ration (110–160 g/day/bird) plus free choice of green onions (*Allium ascalonicum*) for 7–10 days. In both cases, after onions were removed from the diet, mortality in the flock went back to normal levels.

A total of 12 adult geese (6 birds from each of the flocks) were submitted to the California Animal Health and Food

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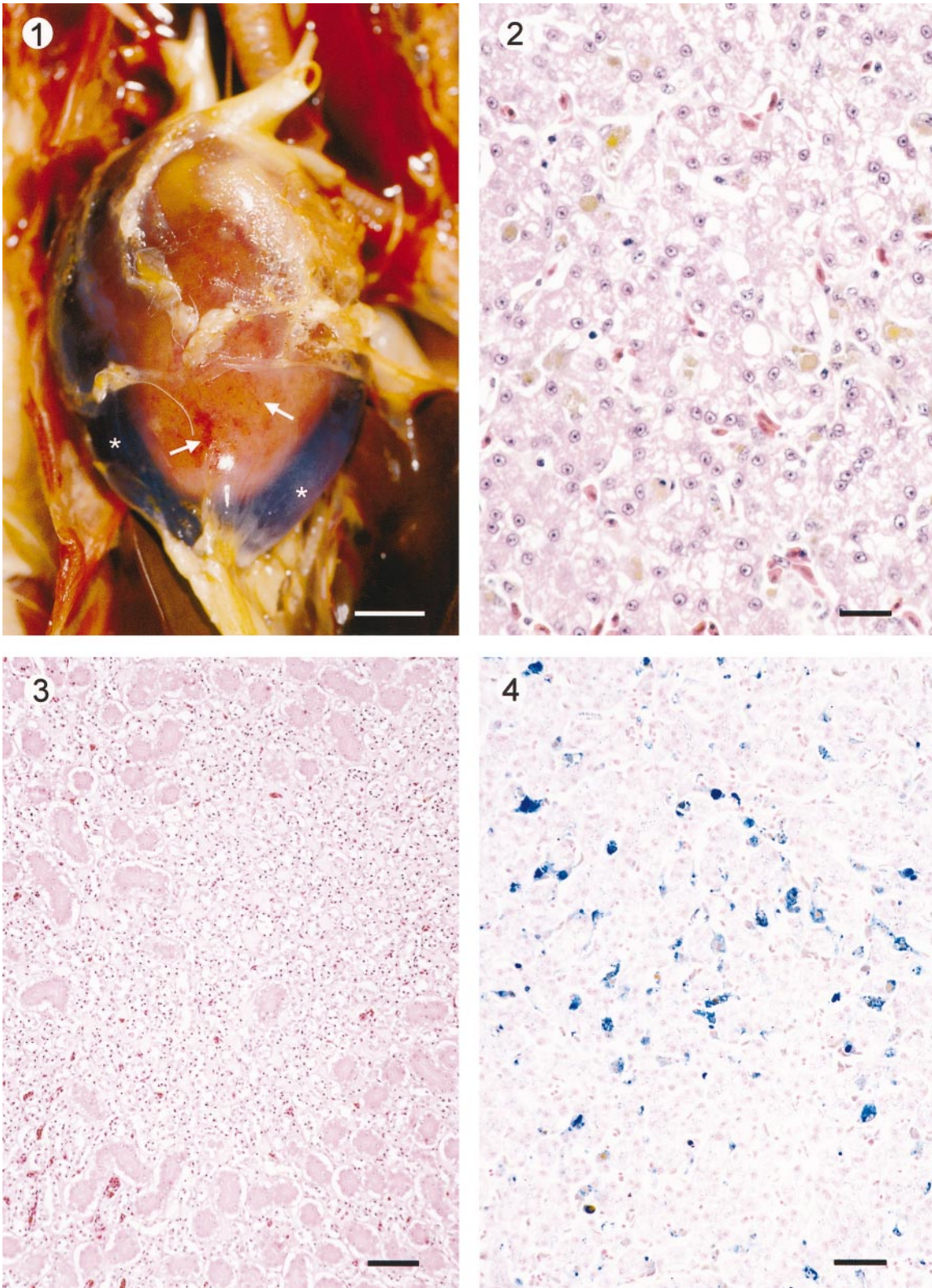


Figure 1. Heart from a goose submitted dead with accumulation of sanguinous fluid in the pericardial sac (*). Note the random petechiation on the epicardium (arrows). Bar = 1 cm.

Figure 2. Hepatocellular loss, macrovesicular hepatocellular vacuolar change, degeneration, and accumulation of hemosiderin in hepatocytes and in Kupffer cells from the liver of a goose fed green onions on the farm. Hepatocellular loss is suggestive of necrosis or apoptosis. Hematoxylin and eosin. Bar = 11 μ m.

Safety Laboratory System (CAHFS), Fresno, California. Grossly, all birds from both submissions had pale epicardium with random petechiation and liver and spleen were mildly swollen. Also, all 6 birds from the second submission had sanguinous fluid accumulated in the pericardial sac (Fig. 1). No feed was found in the crop, proventriculus, or gizzard.

Sections of heart, liver, spleen, kidney, skeletal muscle, thyroid, parathyroid, adrenal, and sections throughout the respiratory tract, digestive tract, and nervous system were collected.

Tissues were fixed in 10% neutral buffered formalin, embedded in paraffin, sectioned at 4 μm , stained with hematoxylin and eosin, and examined by light microscopy. Furthermore, sections of liver, spleen, and kidney were stained with Congo red and Perls' iron. The most striking lesions were moderate to severe hepatic necrosis, centrolobular vacuolation of hepatocytes, splenitis, and renal tubular nephrosis, and accumulation of hemosiderin in hepatocytes, Kupffer cells of the liver, macrophages, and renal tubules (Figs. 2, 3). All sections of liver and spleen had multifocal, extensive foci that stained positive to Perls' iron. Congo red staining revealed moderate amounts of amyloid in the hepatic sinusoids and mild amounts in the spleen.

Three livers from each submission were cultured for aerobic bacteria on 5% sheep blood and MacConkey agars^a and incubated at 37 C under 7.5% CO₂ for 48 hours. Three livers from the first case were cultured for anaerobic bacteria on PRAS PEA and PRAS Brucella agars^b and incubated in an anaerobe box that contained an AnaeroGen^c pack and an anaerobe indicator^c at 37 C for 5 days. Intestinal contents from both cases were selectively enriched in selenite enrichment broth at 42 C for 18–24 hours. After enrichment, selenite broth was plated on brilliant green and XLT4 agars^a and incubated at 37 C for an additional 24 hours. No significant aerobic bacteria were isolated from the liver. A few *Clostridium perfringens* colonies were isolated from one liver. *Salmonella* sp. from group E was isolated from the intestinal contents in both submissions. Bacteria were considered opportunistic because the isolates were different between birds.

Liver and kidney samples were analyzed for heavy metals (arsenic, cadmium, copper, iron, lead, manganese, mercury, molybdenum, and zinc) by inductively coupled plasma spectrometry (ICPS). In both submissions, heavy metals were within the expected concentration range, except above normal, but not toxic, concentrations of copper (47.78 ± 13.33 ppm, mean \pm SD) and iron (803.60 ± 67.64 ppm) in the liver.

Five days after the second case was submitted to the laboratory, blood from 6 geese in the affected flock was collected for complete hematology. No evidence of anemia was

observed, and no Heinz bodies were detected in the erythrocytes.

To confirm the diagnosis of onion poisoning, 2 feeding trials were performed. In the first trial, 3 White Chinese geese were fed an ad libitum ration consisting of 75% (wet weight basis) green onions and 25% pelleted maintenance ration for 21 days. The onions and pelleted maintenance ration were thoroughly mixed, so the birds could not select one portion of the diet alone. In the second trial, 3 geese were fed the same proportions of onion and maintenance ration, but both portions of the diet were offered in separate trays so birds could choose what to eat. Finally, 2 additional White Chinese geese were selected as controls and were fed 100% maintenance ration ad libitum. Blood for complete hematology and biochemistry studies was collected in heparinized tubes^d from the birds on days 0, 6, 9, 13, 16, and 21.

It took about 1 week for the birds to start eating the onions. Blood changes were observed from day 9 to the end of the experimental period and were more obvious in the birds that were given the separated onions and maintenance ration than in those in which onions had been thoroughly mixed with the pellets. The red blood cell count and hematocrit decreased, whereas polychromasia and reticulocyte estimate increased during this period (Table 1). In the geese fed the mixed onions and pellets, a few Heinz bodies and slight anisocytosis were observed in one sample on day 13 and in 2 samples on day 21. Slight to moderate anisocytosis was a consistent finding in the geese fed the onions separately. No Heinz bodies were ever observed in this group of geese. In both feeding trials, the blood biochemical values were maintained within the normal range throughout the experimental period.

On day 21, the birds were euthanized with carbon dioxide gas¹ and a full necropsy was performed. Except for the mildly enlarged and severely darkened livers, no other significant gross changes were observed in the birds from either feeding trial. No significant gross findings were observed in the control birds.

Sections of heart, lung, spleen, kidney, liver, intestine, pancreas, bone marrow, and trachea were collected and prepared for histologic examination as described above. Histologic changes were similar in both experimental trials. The liver contained moderate amounts of hemosiderin in the hepatocytes and Kupffer cells. The bone marrows were between 60% and 90% cellular and contained numerous immature erythrocytes with no abnormal cells. Perl iron stain revealed large amounts of iron in the livers (Fig. 4). Histology was unremarkable in the tissues from the control birds. In addition, the liver from each bird was analyzed for heavy metals by ICPS. Birds exposed to onion in the diet had high, but not toxic, levels of copper and iron in the liver (Table

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Figure 3. Renal tubular loss and degeneration, suggestive of necrosis, from the kidney of a goose fed green onions on the farm. Hematoxylin and eosin. Bar = 450 μm .

Figure 4. Histophotograph of the liver from an experimental goose fed green onions thoroughly mixed with a pellet maintenance ration. Note the numerous hepatic foci stained positive for iron. Perls' iron. Bar = 23 μm .

Table 1. Changes in select hematologic values from experimental geese fed green onions.

	ID*	Day 0	Day 6	Day 9	Day 13	Day 16	Day 21
RBC ($\times 10^6$)	1C	3.2	2.15	4.07	2.94	2.89	2.96
	2C	2.45	2.32	2.09	2.02	2.46	4.24
	1M	3.07	3.18	2.43	2.34	2.3	1.56
	2M	3.3	2.4	2.27	2.03	1.9	1.56
	3M	3.3	2.28	2.25	ND†	2.29	2.3
	1S	3.69	3.2	1.61	1.03	0.97	1.03
	2S	2.76	1.9	0.95	1.3	0.99	1.08
	3S	3.34	1.65	0.93	0.74	1.11	1.35
	HCT (%)	1C	34	37	47	43	43
2C		36	38	41	40	40	48
1M		36	36	35	34	35	27
2M		41	35	31	30	29	32
3M		42	40	33	ND	36	34
1S		40	41	24	29	31	24
2S		38	34	23	23	26	27
3S		39	42	23	29	28	24
Polychromasia		1C	slight	slight	slight	slight	slight
	2C	slight	slight	slight	slight	slight	slight
	1M	slight	slight	moderate	marked	moderate	marked
	2M	slight	moderate	marked	moderate	marked	marked
	3M	slight	slight	marked	ND	moderate	slight
	1S	slight	slight	marked	marked	marked	marked
	2S	slight	slight	marked	marked	marked	marked
	3S	slight	slight	marked	marked	marked	marked
	Reticulocyte estimate	1C	+1	+1	+1	+1	+1
2C		+1	+1	+1	+1	+1	+1
1M		+1	+1	+2	+3	+2	+3
2M		+1	+1	+3	+2	+2	+3
3M		+1	+1	+2	ND	+2	+1
1S		+1	+1	+3	+3	+3	+3
2S		+1	+1	+3	+3	+3	+3
3S		+1	+1	+3	+3	+3	+3

* Identification of experimental geese. The letter after each bird number indicates the group: C = control; M = mixed onions and pellet diet; S = separated onions and pellet diet; RBC = red blood cell count; HCT = hematocrit.

† ND = Not done.

2). Heavy metal concentrations in control birds were within the expected limits.

Heinz bodies have been described as a characteristic change of red blood cells in animals fed onions.^{6,12,13} Susceptibility to onion poisoning has been correlated with the animal's propensity for Heinz body formation.² Interestingly, Heinz bodies were not a consistent finding in the blood smears of the experimental geese. Heinz body anemias do

not regularly occur in birds, although the condition has been induced experimentally in fowl.⁵ Heinz bodies have also been observed in domestic fowl fed rapeseed meal⁹ and in shore birds suffering from oil spill ingestion.^{4,7}

On the other hand, hematology results indicated a progressive drop in red blood cell count and an increased polychromasia and reticulocyte estimate. By day 21 of the trial, most of the geese fed onions had anemia. Furthermore, his-

Table 2. Heavy metal concentrations (ppm) in the liver of geese from feeding trials.

ID*	Arsenic	Cadmium	Copper	Iron	Lead	Manganese	Mercury	Molybdenum	Zinc
1C	<1	<0.3	39.4	127	<1	3.30	<1	1.0	43.5
2C	<1	<0.3	33.5	228	<1	2.93	<1	1.0	46.6
1M	<1	<0.3	61.6	1160	<1	3.27	<1	1.8	74.0
2M	<1	<0.3	65.9	1190	<1	3.22	<1	1.4	67.2
3M	<1	<0.3	71.1	918	<1	3.34	<1	1.5	60.3
1S	<1	<0.3	42	970	<1	2.53	<1	0.9	52.3
2S	<1	<0.3	59.7	1190	<1	2.47	<1	1.1	46.1
3S	<1	<0.3	40.9	1080	<1	2.78	<1	1.1	49.9

* Identification of experimental geese. The letter after each bird number indicates the group: C = control; M = mixed onions and pellet diet; S = separated onions and pellet diet.

tology of the liver from the 3 geese demonstrated abundant hemosiderin in hepatocytes and Kupffer cells, which may be associated with hemolysis. Hemolytic anemia due to denaturation of globin occurs in cattle, horses, and dogs fed onions.¹⁴ The toxic components in *Allium* species are sulfur-containing compounds, especially alk(en)ylcysteine sulfoxides.² These compounds interfere with the hexose monophosphate pathway and consequently lead to damage of red blood cell membrane, hemolysis, and denaturation of hemoglobin. The amount of these toxic compounds may vary with species of onion, time of year, and growing conditions. Culled onions may also be contaminated with other toxins, such as mycotoxins, which could contribute to the disease process.¹¹

In both field cases, green onions were offered in a separate pile, whereas pelleted feed was served in a trough; therefore, allowing geese access to an all-onion diet for approximately 1 week. In one trial, onions were served in similar fashion to that in the field cases. However, because of the minimal numbers of birds used in the experiment, all birds had access to some pelleted diet everyday. It is possible that in the field cases, because of pecking order, some geese did not have access to pellets and had to eat only onions. Furthermore, in the feeding trial where green onions were mixed in the same trough with the pellets, birds could not completely separate the pelleted feed from the onions. Livestock may be fed cull onions in large amounts without any real problems, as long as they are mixed with other vegetable wastes or other feed.^{6,8} This may explain why no overt clinical signs were seen in the experimental birds, even after 21 days of eating onions, compared with the increased mortality seen in the field cases.

Copper concentration in the livers of the geese from the field and the experimental trials were reported higher than normal but not of toxic levels. The normal liver copper in most birds has been reported between 3 and 15 ppm.¹⁰ On the other hand, average copper concentration in the liver of geese (57 birds) submitted to CAHFS during the past 10 years, that did not have evidence of copper toxicosis, was 54 ppm (between 3.3 and 214 ppm). Birds appear to tolerate higher levels of copper than many mammals, and clinical abnormalities associated with copper intoxication have rarely been reported in birds.³

Increased concentration of iron observed in the field cases as well as in the experimental birds probably was secondary to hemolysis and was compatible with the accumulation of hemosiderin observed histologically.

This experimental study demonstrated that anemia could be induced by ingestion of onions. However, Heinz bodies are not a consistent finding in geese fed onions. The feeding trials demonstrated that geese are quite resistant to onion intoxication. The data also indicated that a larger portion of the diet in geese might be onion, provided that the onions are mixed with a maintenance ration so the birds cannot

separate them. Finally, although the results from the feeding trials may suggest that onions were responsible for at least part of the changes seen in the outbreak, the cause of death could not be verified. Similar pathologic changes were seen in the liver of geese from the field outbreak and experimental birds, but no changes were observed in the heart or kidney of the experimental geese.

Acknowledgements. The authors would like to thank Mark Rezvani for his technical assistance and Dr. Peter Woolcock for reviewing the manuscript.

Sources and manufacturers

- a. Remel, Lenexa, KS.
- b. Anaerobe Systems, Morgan Hill, CA.
- c. Oxoid, Hampshire, UK.
- d. Vacutainer, Becton Dickinson, Franklin Lakes, NJ.

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