

Pansteatitis in great blue herons

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FROM OCT 17 to Nov 14, 1985, 3 wild great blue herons (*Ardea herodias*) that were unable to fly or stand were brought by private citizens to the Chesapeake Wildlife Sanctuary for treatment. On the basis of physical examination, each heron was weak, lethargic, and 5% to 10% dehydrated. Each bird had large amounts of firm, lobulated, subcutaneous fat over most of its body, causing the overlying skin to have a nodular appearance. Firm masses were palpable in the abdominal cavity of each heron. Each bird was given 60 to 100 ml of 5% glucose solution orally and was force-fed fish and mouse parts.

The first heron evaluated (heron 1), an immature male, was found dead the day after initial examination. Heron 2, an immature female, was force-fed fresh, whole mice and pieces of thawed fish (spot and bluefish) daily for 15 days. Heron 2 improved

concentration was determined, and a biopsy specimen of subcutaneous fat was collected. Steatitis was diagnosed on the basis of histologic features of the fat specimen. The bird was hypoproteinemic (total protein, 3.6 g/dl; normal = 4.5 to 5.8 g/dl^a) and had a regenerative anemia (PCV, 17.5%; normal = 38% to 45%^a) with severe poikilocytosis and anisocytosis.

Each day, heron 3 was supplemented orally with 400 IU of vitamin E^b and force-fed mice and fish. Heron 3 had marked clinical improvement during the following 4 weeks. On day 28, hematologic results indicated that heron 3 was no longer anemic (PCV, 40%) but examination of a biopsy specimen of the subcutaneous fat indicated no change in the steatitis. Serum samples collected on day 28 were lipemic and thus unsuitable for vitamin E assays; evaluation of a

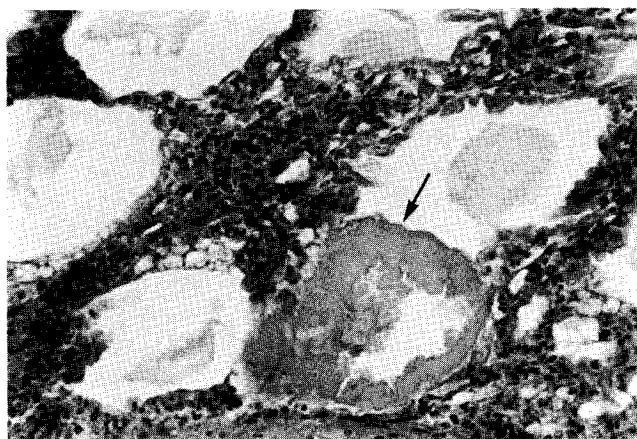


Fig 1—Photomicrograph of necrotic adipose tissue from heron 2. Notice the interstitial infiltration with heterophils and macrophages and the area of fat saponification (arrow). H&E stain; $\times 65$.

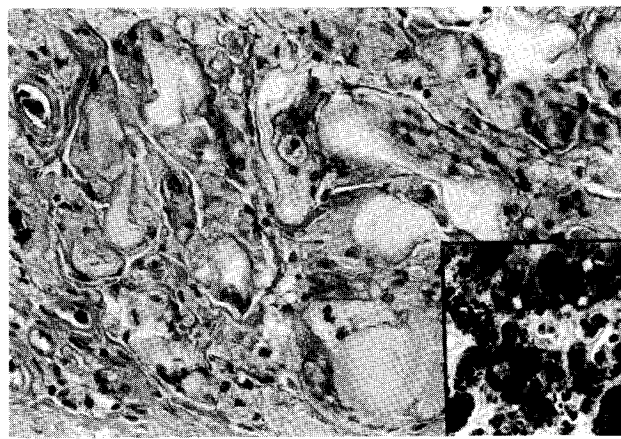


Fig 2—Photomicrograph of granulomatous steatitis in heron 3. Macrophages and giant cells predominate. H&E stain; $\times 65$. Inset—Notice the extracellular and intracellular globular deposits of darkly-staining acid-fast ceroid. Kinyoun's stain; $\times 65$.

clinically and was able to walk by day 7 after initial evaluation; however, on day 14, heron 2 again was unable to stand and died on day 15.

During the initial examination of heron 3 (an adult male), blood samples were collected, a hemogram was performed, serum vitamin E (α -tocopherol)

sample collected 3 weeks later (day 49) indicated a vitamin E concentration of 32.6 mg/L.

The daily oral dose of vitamin E for heron 3 was reduced from 400 IU to 200 IU beginning on day 61. The dosage was changed inadvertently to 200 IU twice a week from day 70 through day 81.

On day 82, heron 3 was found down and unable to stand. The bird was force-fed and was orally given supplements of 400 IU vitamin E daily; however, the

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^aNormal values for hemograms and serum protein were based on data from herons 4 and 5.

^bVitamin E, 200 IU capsules, Dart Drug Inc, Landover, Md.

bird's condition continued to deteriorate. Hematologic findings on day 86 included a moderate regenerative anemia (PCV, 29%), a marked leukocytosis (WBC count, 43,200/ μ l; normal = 9,000 to 16,000/ μ l⁸) and heterophilia (37,200/ μ l; normal = 6,000 to 11,000/ μ l⁸); serum samples were hemolyzed and unsuitable for biochemical analysis. On day 87, heron 3 was found dead.

Complete necropsies were performed on the 3 herons. Specimens of all body organs were fixed in 10% neutral-buffered formalin, histologically processed, and examined microscopically.

Large amounts of subcutaneous and cavitary fat were found in each heron. All adipose tissue was firm and waxy and had a mottled yellow-to-brown coloration throughout. The adipose tissue often was lobulated and had a prominent fishy odor. Large red nematode parasites, identified morphologically as *Eustrongylides ignotus*,¹ were located in the abdominal cavity and mesenteric fat of herons 1 and 2. Atrophy of the pectoral muscles were found in herons 1 and 2. In heron 2, several dark fungal plaques were seen on the right thoracic and abdominal air sacs and a fungal granuloma was found in the right lung.

Microscopically, widespread necrosis of adipose tissue was found in each bird, accompanied by mild to moderate infiltrates of heterophils, with fewer macrophages (Fig 1). Hyaline, globular pigment often was associated with the histiocytic infiltrates and could be seen extracellularly and within the cytoplasm of macrophages and necrotic adipocytes. This pigment was yellow-brown when stained with H&E and was periodic acid-Schiff positive and acid-fast, which are findings consistent with those of ceroid. Often, reticuloendothelial cells of the spleen and liver also contained ceroid. In postmortem adipose tissue specimens from heron 3, inflammatory cell infiltrates usually contained more macrophages and ceroid pigment than were seen in antemortem fat specimens from this bird, and granulomas with giant cells were seen occasionally in the adipose tissue (Fig 2).

Myofiber degeneration and necrosis were seen scattered throughout the skeletal muscles of the 3 birds (Fig 3). These lesions often contained histiocytes. Similar changes were seen in the myocardium of each heron, but the lesions were smaller and less numerous than in skeletal muscles.

Pansteatitis and multifocal myodegeneration of cardiac and skeletal muscles were diagnosed in each heron. Heron 2 also had respiratory aspergillosis which was considered a secondary infection.

Clinical signs and pathologic findings in the 3 herons were compatible with hypovitaminosis E.^{2,3} Initial erythrocytic changes in heron 3 were similar to changes reported in mammals with vitamin E deficiency.^{2,4} The clinical and hematologic response of heron 3 while being given large supplements of vitamin E, followed by an exacerbation of clinical signs and death within 4 weeks after reducing the vitamin E dosage, also support a diagnosis of vitamin E deficiency.

Lesions of hypovitaminosis E have been correlated with low plasma α -tocopherol concentrations in dogs⁵ and with low serum α -tocopherol concentration

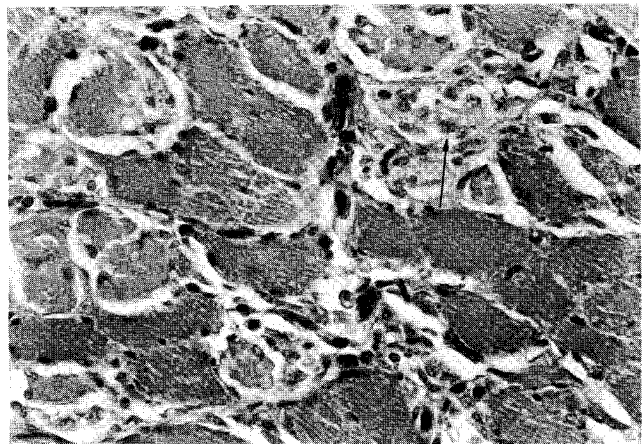


Fig 3—Photomicrograph of transverse section of skeletal muscle from heron 3. Notice the foci of myofiber degeneration. Low numbers of histiocytes are seen within degenerated fibers (arrow). H&E stain; $\times 65$.

in marmosets. The initial serum α -tocopherol concentration in heron 3 was 2.5 mg/L. After 7 weeks of vitamin E supplementation, serum concentrations had increased to 32.6 mg/L. In an attempt to determine approximate normal α -tocopherol concentrations for great blue herons, serum α -tocopherol concentrations were assayed in 2 other great blue herons (heron 4 and heron 5) that were brought to the Chesapeake Wildlife Sanctuary for problems other than steatitis. Heron 4 was an adult bird from Virginia with a gunshot wound and with a serum α -tocopherol concentration of 21.8 mg/L; heron 5 was an emaciated immature bird from Delaware with an α -tocopherol concentration of 18.3 mg/L. On the basis of data from herons 4 and 5, heron 3 initially had low serum α -tocopherol concentrations.

Diets composed primarily of fish usually contain high levels of polyunsaturated fats⁶ and have been associated with the induction of vitamin E deficiencies.^{2,3,6-10} Tissue concentrations of polyunsaturated fatty acids and vitamin E differ greatly between and within species of fish at different times of the year; diets based on a single fish species may have large fluctuations in fatty acid and vitamin E composition.⁶

Steatitis has been reported in several species of wild and captive fish-eating birds, including great blue herons.¹⁰⁻¹³ The 3 affected herons in the present study were found within a 10-mile radius of Annapolis, Md and probably fed along the Chesapeake Bay and its tributaries. The herons may have been feeding primarily on a species of fish that had high concentrations of polyunsaturated fatty acids. Or, if the herons had been consuming dead, rancid fish, oxidized fatty acids in this food source may have depleted their body stores of vitamin E.⁶ Vitamin E and selenium deficiencies have been experimentally induced in ducklings by the addition of various heavy metals to their diets.¹⁴⁻¹⁶ Reports of steatitis in great blue herons from California^{11,12} indicate that steatitis may be a widespread and common condition in this species.

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