

ABDOMINAL ASCITES IN ELECTRIC EELS (*ELECTROPHORUS ELECTRICUS*) ASSOCIATED WITH HEPATIC HEMOSIDEROSIS AND ELEVATED WATER pH

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Abstract: Six electric eels (*Electrophorus electricus*) from various centers that house aquatic organisms presented clinically with abdominal distension following prolonged exposure to elevated environmental pH. Postmortem examination revealed marked ascites. Culture of the abdominal fluid from three of the eels yielded either *Aeromonas hydrophila* or *Citrobacter freundii*, which were most likely secondary invaders. Histopathology showed marked iron accumulation in both hepatocytes and hepatic macrophage aggregates.

Key words: Ascites, eels, liver, pH, hemosiderosis, *Electrophorus electricus*.

INTRODUCTION

Electric eels (*Electrophorus electricus*) have a compact coelom immediately caudal to the head followed by a long muscular tail. When excessive coelomic fluid accumulates, distending the visceral cavity of an electric eel, it can appear to have an enlarged head. This is the basis of the inappropriate lay term "big head" (Fig. 1). The exact etiology of this condition is unknown, but it has been observed in captive eels in a number of public aquariums, and may be related to environmental factors. Little information has been published concerning husbandry and disease conditions of captive electric eels. Maintaining eels in soft water with a pH of 6.0-6.8 has been recommended.^{2,18} Wild electric eels are found in acidic freshwater (pH 3.8-6.0) of the middle and lower Amazon basin.^{2,6,10,12,20,21} The clinical history and histopathology of six eels that were maintained in freshwater with an average pH of 7.9 follows.

CASE REPORTS

Clinical histories

The University of Maryland, Baltimore, Aquatic Pathobiology Center (UMB APC) performed complete necropsies or evaluated fixed tissues from six electric eels with histories of coelomic distension between 1985 and 1993. These cases were submitted by the National Aquarium in Baltimore (NAIB),

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the Maryland Science Center in Baltimore (SCIB), and the Seattle Aquarium (SA). A seventh eel with no coelomic distension or ascites from the Pittsburgh Zoo (PZ) is included for comparative purposes.

Eel 1, 1.12 kg in weight and 63 cm long, resided at NAIB for several months before developing acute coelomic distension. Radiographs revealed a large quantity of 1-cm-diameter substrate rocks from the exhibit in the stomach. These were removed by gavage under tricaine methane sulfonate (MS222, Crescent Research Chemicals, Phoenix, Arizona 85055, USA) anesthesia, reducing the apparent coelomic distension. The substrate in the tank was changed to include only larger stones. A few days later the coelomic distension returned, and radiographs revealed extensive coelomic fluid accumulation but no stones. Chloramphenicol (40 mg/kg i.m. s.i.d., Chloromycetin®, Parke Davis & Co., Detroit, Michigan 48232, USA) therapy was initiated and the eel continued to eat normally. Chloramphenicol therapy had no impact on the clinical course and the animal was found dead in its tank 1 wk later. Retrospective evaluation of the water quality records for the tank showed a fluctuating increase in pH (7.0-8.5) that stabilized between 8.0 and 8.1 for 1 mo before the onset of the problem. No elevations in nitrite, nitrate, or ammonia were recorded.

Eel 2, a 1.8-kg female, was housed at the NAIB for a few months in the same tank as eel 1. Two weeks before death, a 4- to 6-cm-long strand of mucoid fecal material was noted clinging to the vent. Direct microscopic examination of the feces did not reveal any parasites. After 1 wk, slight coelomic distension was noted that dramatically increased several days later. Systemic chloramphenicol therapy, 40 mg/kg s.i.d. i.m. was initiated. No improvement was apparent the following day. Ra-

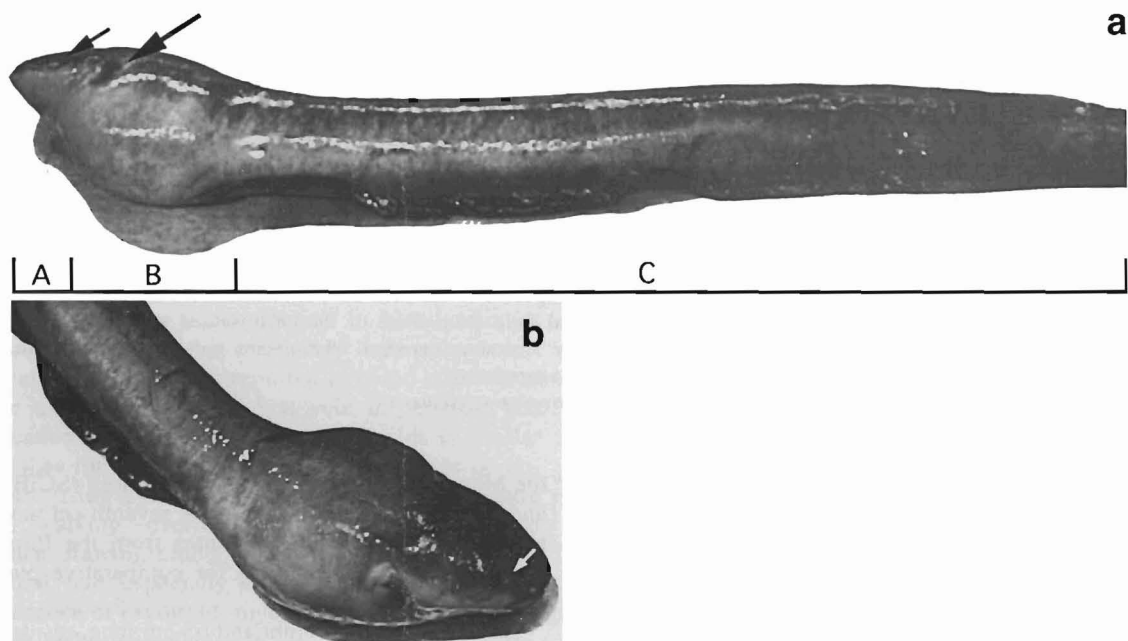


Figure 1. a. Eel with coelomic distension (incorrectly termed "big head"). A = head, B = trunk, C = tail, small arrow = eye, large arrow = operculum. b. Higher magnification of eel with coelomic distension. Arrow = eye.

diographs confirmed coelomic fluid accumulation and the presence of a single 1-cm substrate rock in the stomach. Coelomic centesis obtained several milliliters of clear straw-colored fluid that grew no organisms on aerobic bacterial and fungal media. The rock was removed by gastric lavage and the stomach was flushed twice with neomycin sulfate solution (20 mg/kg Neovet®, Med-Tech Inc., Elwood, Kansas 66024, USA). The antibiotic therapy was empirically changed to gentamicin (2.5 mg/kg i.m. every other day; Gentocin®, Schering Corp., Kenilworth, New Jersey 07033, USA), but the eel died the following day. Retrospective evaluation of the tank water quality records revealed a relatively steady pH of 8.0–8.1.

Eel 3, weighing approximately 1 kg, was obtained to replace the first two eels lost at NAIB. This animal was quarantined for 6 wk, and then transferred to the exhibit tank (unknown pH). The eel exhibited acute coelomic distension approximately 5 wk after being placed in the exhibit. Radiographs showed a large quantity of coelomic fluid; centesis removed clear straw-colored fluid that yielded no growth when cultured for aerobic bacteria and was unremarkable on cytology. Within 1 day the eel's coelomic cavity refilled with fluid. Treatment with furosemide (2.5 mg/kg i.m. s.i.d., Lasix®, National Laboratories Corp., Somerville, New Jersey 08876, USA) was initiated with some

subjective improvement noted. On the third day of therapy the eel died. At this time the aquarists were instructed to lower the pH to 3.8–6.0.⁶ Subsequent eels were maintained at a pH of 6.5–6.8 and no further cases of coelomic distension have occurred.

Eel 4, weighing less than 1 kg, from the SCIB, died with coelomic distension, but no other history was available.

Eel 5, also from the SCIB, weighed 5.8 kg and was 76 cm long. The eel had lost weight when the food was changed from goldfish to smelt. Two small lesions were noted on the back. Tetracycline (125 mg s.i.d., Pfizer Inc., New York, New York 10017, USA) was placed in the food for 3 days. Before death, the eel became anorectic and had a distended coelom. The aquarist estimated water pH to be 7.1, but subsequent measurements were 7.9. Because records were not maintained, the length of time that the pH had been elevated is unknown.

Eel 6 from the SA was approximately 91 cm long. It had recurrent bouts of coelomic distension, lethargy, and reduced appetite, with each bout lasting approximately 4–8 wk, beginning late October–November and resolving December–January. Treatment included slowly increasing the tank temperature to 25.6–26.7°C (78–80°F) and frequent water changes. The date of the first occurrence was not known, and no other health problems were noted.

Table 1. Clinical and pathologic findings in seven electric eels.^a

	Eel no.						
	1	2	3	4	5	6	7
Water pH	7.9–8.2	7.9–8.2	7.9–8.2	7.9–8.2	7.9–8.2	6.9–7.5	5.8–6.6
Coelomic distention with ascites	+	+	+	+	+	+	–
Bothwell score of liver iron	+++	+++	++	++	++	NA	–
Iron accumulation in kidney	–	+++	++	++	–	NA	–
Iron accumulation in spleen	–	+++	++	–	++	NA	–
Iron accumulation in intestines	+++	–	++	++	–	NA	–
Thick arterial walls	+++	+++	++	++	++	NA	++

^aNA = tissue not available.

Water pH ranged from 6.9 to 7.5 in the month before death.

Eel 7 was a female weighing 5.2 kg. Only a reduction in appetite was noted before death. Tank pH was maintained between 5.8 and 6.6. This case is included for comparison with the eels with coelomic distention.

Pathology

Eels 1 through 5 were necropsied at UMB APC, and tissue samples were placed in 10% phosphate-buffered formalin. Samples for aerobic bacterial culture were taken from the coelomic fluid (eels 1, 2, 5), spleen (eel 1), kidney (eels 2, 3, 4), and skin lesions (eel 4). Selected tissues from eels 6 and 7 were submitted to UMB APC already preserved in formalin. All tissue samples were processed and embedded in paraffin wax, and 6- μ m-thick sections were stained with hematoxylin and eosin (H&E).¹⁶ Selected sections also were stained with Perls' iron stain.¹⁶ Tissue positive for iron were graded according to a modified Bothwell technique.^{4,9}

Gross lesions included coelomic distention due to ascites in eels 1 through 6 (Table 1, Fig. 1). In addition, reddening of the peritoneal lining was noted in some of the eels examined. Coelomic fluid cytology was nonremarkable; specific gravities were 1.013–1.016. Microscopic lesions included large amounts of iron-containing pigment in hepatocytes (Fig. 2a–c), macrophages of the liver, spleen, kidney, and gut. Macrophages containing golden brown pigment (Perls' iron stain negative) were seen in the heart, kidney, and spleen. Thick arterial walls were present in the spleen and kidney (Fig. 3a) and extensive areas of fibrosis were present in the heart (Fig. 3b, c). Meninges, heart, liver, spleen, stomach, and kidney were congested in some animals.

Aeromonas hydrophila was cultured from eels 1 (spleen and coelomic fluid) and 3 (kidney), and *Citrobacter freundii* was cultured from kidney and

skin of eel 4. No organisms were cultured from eels 2 and 5.

Eel 7 had blood in the coelom but no significant distention. Microscopically, eel 7 had myocarditis, thick arterial walls in the spleen and kidney, and severe congestion in the liver, spleen, and caudal kidney. Numerous macrophages containing golden brown pigment (Perls' iron stain negative) were present in the heart and liver.

DISCUSSION

All of the eels with coelomic distention had been maintained in water with pH levels far above those found in their natural habitat. Electric eels have subsequently been maintained in water pH between 6.5 and 6.8 with no further cases of coelomic distention, suggesting a relationship between alkaline water pH and coelomic distention in captive electric eels. In freshwater fish, hydrogen ions appear to act as counterions to sodium uptake. It is not clear whether this transport exchange is active or passive, but decreasing environmental pH results in increased sodium efflux.¹⁵ For fish adapted to low pH (high hydrogen ion content) water but held in high-pH water for prolonged periods, the diffusional gradient against hydrogen ion influx may contribute to accumulation of body sodium, excess fluid retention, and the development of cardiac overload, ascites, and tissue edema.

Iron accumulation in these eels was quite extensive. The histopathologic findings are similar to reported findings associated with excessive iron storage in humans, rats, and several species of birds, particularly mynah birds.^{7,9,11,13,14,17,19} In mynah birds, iron appears to progressively accumulate in hepatocytes throughout life. Deposition of iron-positive pigment also has been noted in renal tubules, pancreas, heart, and lung.^{7,11} Abdominal distention due to ascites and hepatomegaly may follow excess iron accumulation. In general, ascites may develop due to a variety of causes, including congestive heart

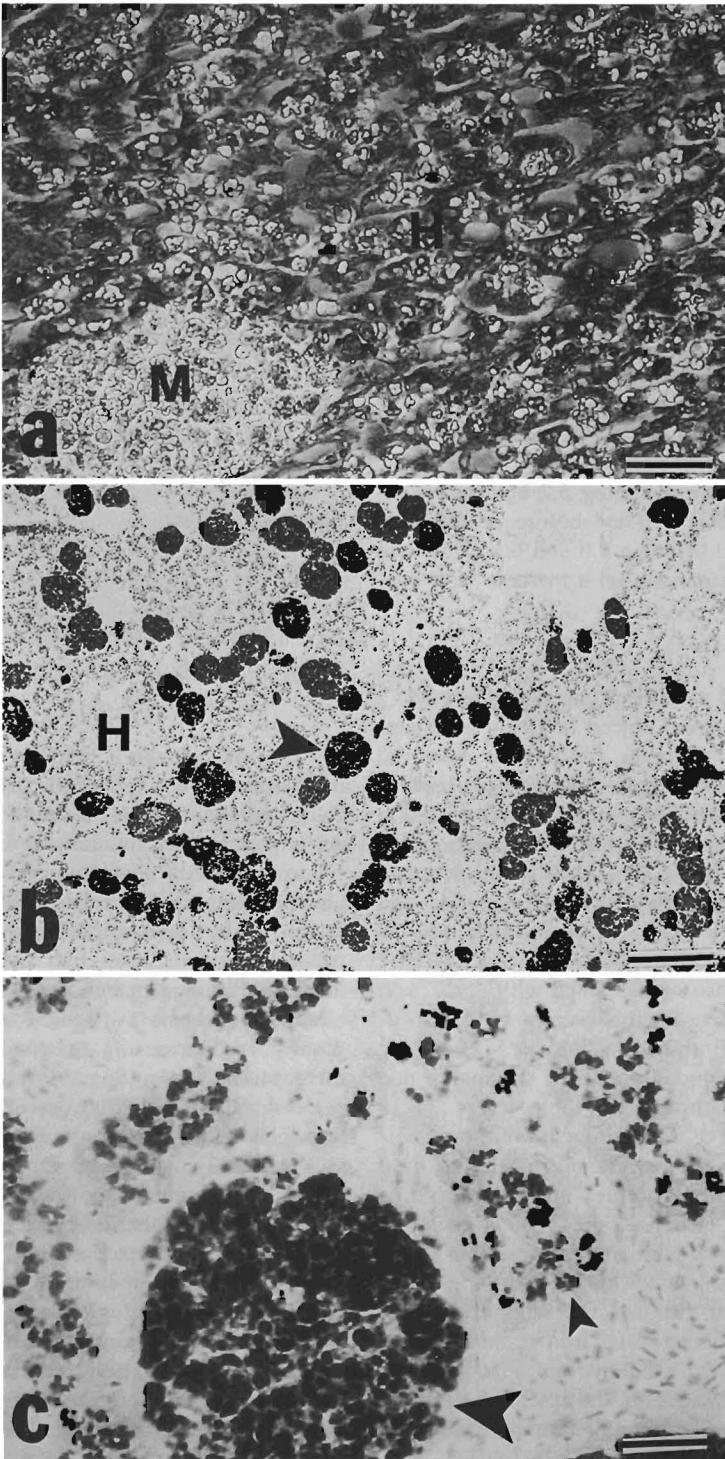


Figure 2. a. Liver. Refractile bodies (iron pigment) in hepatocytes (H) and macrophage aggregate (M). Phase contrast. H&E, $\times 640$. b. Liver. Iron pigment in hepatocytes (H) and macrophage aggregates (arrow). Perls' iron stain, $\times 64$. c. Liver. Higher magnification of Figure 2b showing iron deposition in hepatocytes (small arrow) and macrophage aggregates (large arrow). Perls' iron stain, $\times 640$.

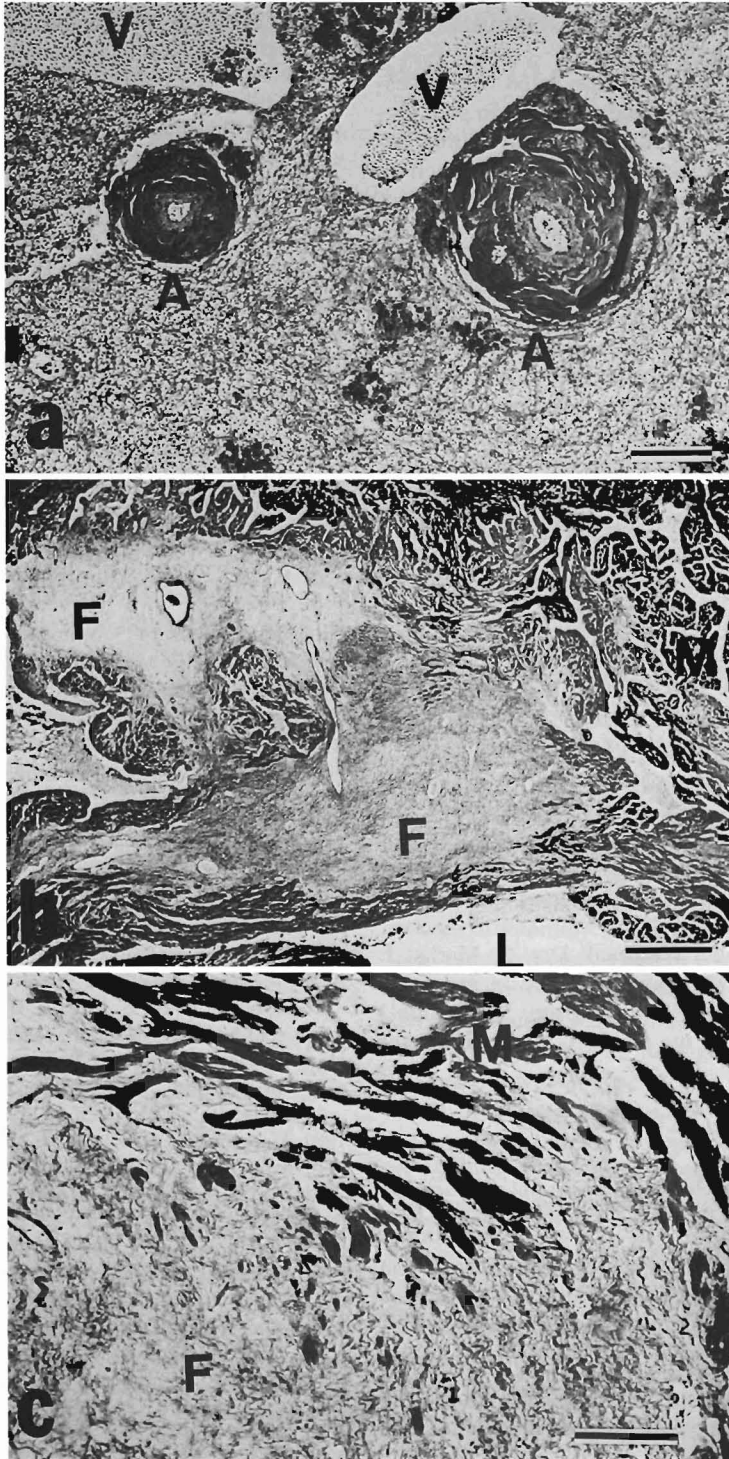


Figure 3. a. Spleen. Thick arteries (A) adjacent to veins (V). H&E, $\times 32$. b. Heart. Areas of fibrosis (F) in myocardium (M). Lumen of ventricle (L). H&E, $\times 32$. c. Heart. Higher magnification of Figure 3b showing area of fibrosis (F) adjacent to normal myocytes (M). H&E, $\times 160$.

failure, peritonitis, hepatic venous obstruction, and hepatic cirrhosis.^{5,19}

In humans, complications associated with excessive iron accumulation include changes in skin pigmentation; diabetes mellitus as a result of iron deposition in the pancreas; impaired liver function; and congestive heart failure with microscopic findings of iron pigment in myocardial fibers, necrosis of cardiac myocytes, and interstitial fibrosis.^{8,19} The cardiac function of the eels in this case study was unknown, but iron-bearing macrophages and fibrosis were present in the myocardium. Thickened arterial walls were noted in the spleen or kidney of all eels for which these tissues were available. The cardiac lesions and a hypertensive state due to volume overload may have contributed to these very thick arteries.

The excess iron levels in these eels may have made them more susceptible to bacterial infection. Virulence of some eel pathogens has been shown to be dependent on iron availability from host fluids.¹³ However, the etiology of the iron accumulation in these electric eels was not determined.

CONCLUSION

A correlation may exist between alkaline water pH and the development of ascites in electric eels that may have caused excess fluid retention and cardiac overload. Iron accumulation in the hepatocytes and macrophages of the liver, kidney, spleen, and gut may contribute to ascites formation.

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