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Lead Poisoning in Grey-headed Fruit Bats (*Pteropus poliocephalus*)

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There are about 130 species of fruit bats throughout the world. One species, the grey-headed fruit bat, is common in coastal areas of eastern Australia, where its principal sources of food are the blossoms of native hardwoods, native fruits such as figs and a wide range of cultivated fruit (Hall and Richards, 1979, Bats of Eastern Australia, Queensland Museum Booklet No. 12, 66 pp.). While it may be expected that chemical toxicity, as a result of various pesticide and fungicide treatments of fruit, is a distinct possibility in this species, the likelihood of lead toxicity would, because of these specialized feeding habits, be expected to be low.

Two adult male grey-headed fruit bats (A and B) were presented for veterinary treatment after being found in suburban backyards in Brisbane, Queensland. Bat A was found in a moribund condition and at the time of presentation it showed severe muscle fasciculation, excess salivation, diarrhea and ataxia. It cried when approached and handled and appeared to be in a very apprehensive state. There were no external lesions.

A tentative diagnosis of insecticide poisoning was made. Initial treatment with atropine and valium proved ineffective in controlling the fasciculation. However some relaxation was obtained with the administration of xylazine (0.1 ml, 20% Rompun). Complete relaxation was obtained with a 0.2 ml dose 30 min later and maintained for 2 days with 0.05 ml doses when required. During this time it was fed orally by tube with a mixture of strained baby apple food and glucose. It was fed four to five times daily and given 10 to 30 ml per feed. Further treatment included the subcutaneous administration of balanced electrolyte solution, corticosteroids, procaine penicillin, multi-vitamins and anabolic steriods. From Day 7 it was able to feed itself and by Day 10 it had normal motor and sensory function, would flap its wings, but was reluctant to fly. No further change was noted until the morning of Day 18 when it was found dead in its cage and immediately submitted for necropsy. A whole body radiograph was taken at this time as part of a general skeletal study.

Approximately 12 mo later Bat B was found crawling around in a yard, with no apparent external injuries. It was unable to fly, but seemed alert and aggressive. This was considered to be normal behavior for a wild bat. Shortly after hospitalization and before a detailed clinical examination was performed, it was found dead in its cage, and submitted for necropsy. No drugs were administered to this bat prior to death.

Tissues were fixed in neutral buffered 10% formalin and prepared for histologic examination. All sections were stained with hematoxylin and eosin and kidney tissue sections were also stained with the Ziehl-Neelsen and Perl's Prussian Blue stains. Subsequently, formalin-fixed kidney tissue from Bat A was prepared for electron microscopy by rinsing in cacodylate buffer, post-fixing in osmium tetroxide, and embedding in Epon. Ultrathin sections were stained with uranyl acetate and lead citrate and examined in a Philips transmission electron microscope. Additional formalin fixed kidney and liver tissue from both bats were analyzed for lead and the liver from Bat B was analyzed also for arsenic (Stahr, 1977, Analytical Toxicology Methods Manual, Iowa State University Press, Ames, Iowa, pp. 42–46).

Macroscopic examination of Bat A revealed that the carcass was poorly nourished. The only gross abnormality was the presence of ecchymotic hemorrhages in the left middle lobe of the lung. All other body organs including the brain appeared normal.

Histologically, the left lung showed scattered

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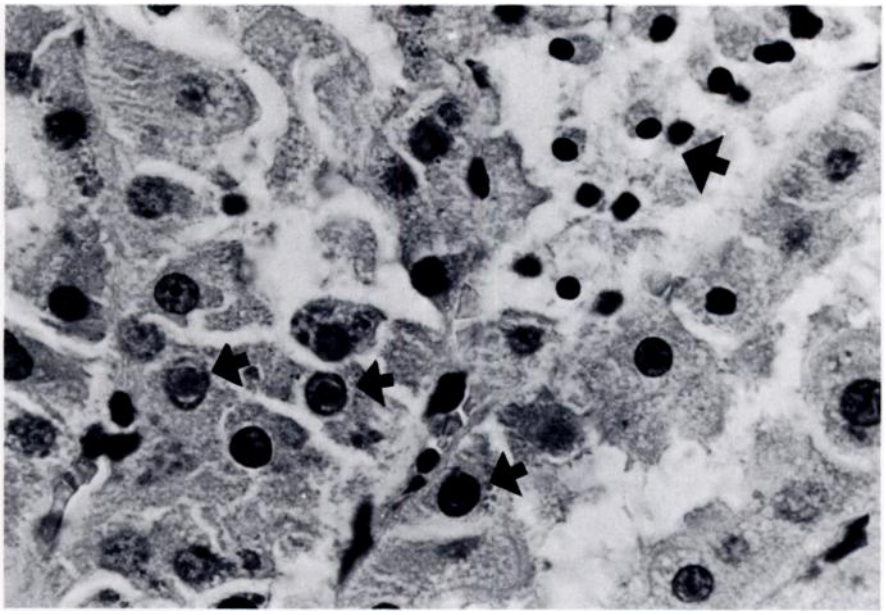

FIGURE 1. Fruit bat renal tubules showing large intranuclear inclusion bodies, some of which are indicated by the small arrows. Pyknosis and separation of cells in the distal tubules indicated by the large arrow. H&E, ×200.

areas of suppurative bronchopneumonia with associated hemorrhage. Apart from heavy neutrophil infiltration, there were some areas where macrophages were present in large numbers both in the alveolar spaces and interstitial tissues. Some bronchioles had plugs of necrotic cells and fibrin. The brain showed scattered small hemorrhagic foci, most of which were perivascular. The meninges were generally normal, but in the occasional sulcus there was macrophage and neutrophil accumulation. The liver was moderately congested with occasional enlarged vesicular hepatocyte nuclei. In the kidney the proximal tubular epithelial cells were swollen with brown-staining granular intracytoplasmic pigment. Some of this pigment stained positive for iron. Many of the nuclei were enlarged and vesicular in appearance. Eosinophilic intranuclear inclusion bodies were quite frequent (Fig. 1), but only a few were acid-fast positive with Ziehl-Neelsen stain. The distal tubular cells showed some coalescence, lifting of basement membrane, and pyknosis (Fig. 1).

Ultrastructurally, the inclusion bodies consisted of a very dense and compact granular matrix. At the periphery there was a meshwork of fibrillar material some of which was radiating outwards (Fig. 2).

The carcass of Bat B showed no macroscopic abnormalities. The main histologic findings were in the kidneys where the proximal tubular epithelial cells showed marked variation in nuclear size, some vesiculation and considerable numbers of eosinophilic intranuclear inclusion bodies. In addition there were scattered small interstitial inflammatory foci consisting mainly of lymphocytes. The kidney changes overall were more severe than those observed in Bat A. A few small perivascular hemorrhages were noted in the brain.

The radiograph of the Bat A carcass showed no evidence of metallic lead. Lead levels in the kidney and liver tissue of Bat A were 20.5 mg/kg and 59.5 mg/kg respectively. In Bat B they were 44.6 mg/kg in kidney and 18.7 mg/kg in liver. The formalin fixative used for tissues from both bats was lead free.

In addition to high levels of lead in both kidney and liver, there were present in these cases some of the pathologic features of lead poisoning in domestic animals. The intranuclear inclusions, some of which were acid-fast, in the proximal tubular epithelium of the kidneys is one such feature. The ultrastructural appearance of a dense core and a fibrillar periphery are characteristic of lead inclusions (Richter et

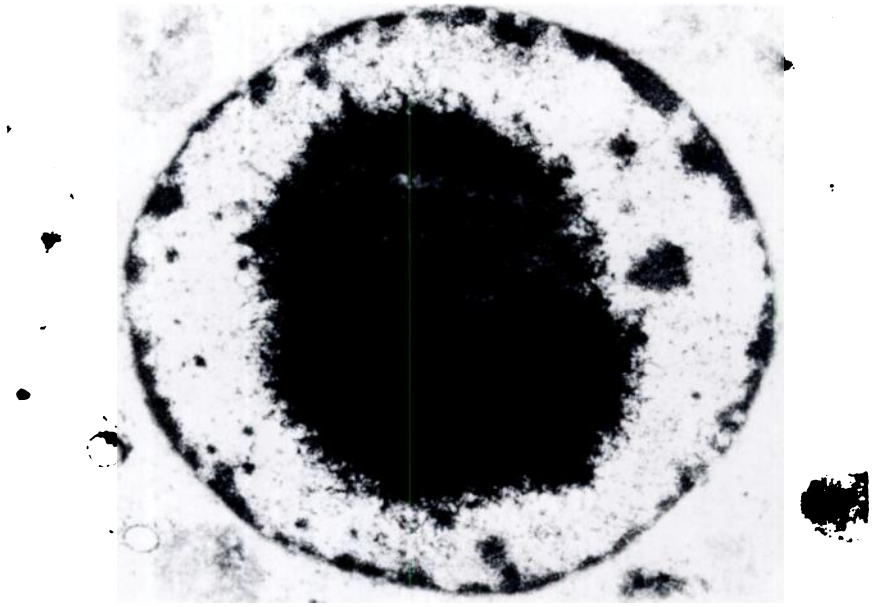

FIGURE 2. Electron micrograph of an intranuclear inclusion showing a dense compact granular core. The periphery has a fibrillar appearance; some of the fibrils radiate outwards. ×3,000.

al., 1968, Am. J. Pathol. 53: 189–217). The fibrillar nature is due to the composition of a protein-lead complex (Goyer et al., 1970, Lab. Invest. 22: 245–251). More recently it has been shown that inclusion development appears in kidneys of mice within 6 hr of intracardiac administration of lead, and that this involves protein synthesis (Choie et al., 1975, Beitr. Pathol. 155: 197–203). However the easy detection, by light microscopy, of large intranuclear inclusion bodies, as seen in these cases, is usually indicative of chronic poisoning (Choie et al., 1975, op. cit.). Other pathologic features of lead poisoning present in one or both of these cases included hepatocyte nuclear enlargement, liver congestion and vascular hemorrhage in the brain (Zook, 1972, Vet. Pathol. 9: 310–327). Neuronal degeneration or edema was not a feature although mild infiltration of inflammatory cells into the meningeal space was present.

The source of lead in these cases was not determined. Radiographic examination of Bat A failed to reveal any metallic lead. While little is known about the absorptive function of the alimentary tract of fruit bats, it is short and simple, allowing the passage of food in about 20 min (Yalden and Morris, 1975, The Lives of Bats, David and Charles, Newton Abbot, Vancouver, London, 247 pp.). Normally, the fruit is crushed in the mouth with only the juices and soft pulp being swallowed and the solids being compressed into a pellet and spat out. It is likely that metallic lead accidentally picked up during feeding would probably be disposed of before absorption could occur. Enquiries revealed that lead arsenate is used by some fruit growers in the Brisbane area. With the presence of a large fruit bat colony in the area, lead poisoning could be expected to be common. Because the fruit bat is considered to be a pest by orchardists and backyard fruit growers, it is probable that many deaths which may be observed are unlikely to be drawn to the attention of veterinarians. The negative arsenic result on Bat B however suggests that lead arsenate may not be the source of the lead. Despite this, the contamination, either malicious or otherwise, of specialized food by lead salts would appear to be the most likely cause of toxicity.

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