Hypermature Cataract in a Crested Mynah, Leucopsar rothschildi

Author: Schmidt, R. E.

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and Parsons, 1977, The Vertebrate Body, W. B. Saunders Co., Philadelphia, Pennsylvania, 624 pp.). These mummies are 3 mo in development as evidenced by the presence of placentas (Wrass, 1973, Pac. Sci. 27: 305–318). The total counts of vertebrae for the mummified fetuses ranged from 150–157 ($\bar{x}$ = 153.5). The counts for the normal fetuses ranged from 176–178 ($\bar{x}$ = 177.0). The mummified precaudal vertebrae ranged from 76–79 ($\bar{x}$ = 72.2) whereas the normal fetuses' precaudal vertebrae ranged from 80–82 ($\bar{x}$ = 81.0).

Based on our observations of total and precaudal vertebral counts, it appears that the development of these fetuses prior to mummification was abnormal. It is difficult to interpret these findings in terms of maternal physiology since only one specimen was collected and all of our observations are postmortem. Mummification in other vertebrates has been attributed to genetic factors, specifically a lethal gene (Loje, 1930, Tidskr. Landokonomi 10: 517–549; Hertig and Sheldon, 1943, Ann. Surg. 117: 596–606; Gilmore, 1952, Dairy Cattle Breeding, J. B. Lippincott, New York, 604 pp.; Asdell, 1955, Cattle Fertility and Sterility, Little, Brown Co., Boston, Massachusetts, 277 pp.; Roberts, 1971, Veterinary Obstetrics and Genital Diseases, Edwards Bros., Ithaca, New York, 776 pp.; Lauritsen, 1976, Acta Obstet. Gynecol. Scand. 54: 261–264), but this may not be the case (Deaton et al., 1959, J. Dairy Sci. 43: 312; Davidson and Roberts, 1961, Cornell Vet. 51: 34–46; Novak and Woodruff, 1979, Novak's Gynecologic and Obstetric Pathology, 8th Ed., Philadelphia, W. B. Saunders Co., 795 pp.). Environment factors, more specifically the uterine environment, should be considered at the time of early and late fetal death, premature birth, and anatomical malformations (Erb and Morrison, 1957, J. Dairy Sci. 40: 1030–1035; Roberts, 1962, J. Am. Vet. Med. Assoc. 40: 691–698; Novak and Woodruff, 1979, op. cit.).

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R. E. Schmidt, Veterinary Sciences Division, USAF School of Aerospace Medicine, Aerospace Medical Division, AFSC, United States Air Force, Brooks Air Force Base, Texas 78235, USA

As cortical cataracts develop, there can be liquefactive necrosis of lens fibers. If the necrotic cortical material is lost through absorption or extrusion, hypermature cataract results, with a smaller than normal lens and wrinkled capsule (Hogan and Zimmerman, 1962, Ophthalmic Pathology, An Atlas and Textbook, 2nd Ed., W. B. Saunders, Philadelphia, Pennsylvania, pp. 666–671; Yanoff and Fine, 1975, Ocular Pathology, A Text and Atlas, Harper & Row, Hagerstown, Maryland, pp. 371–375). Descriptions of cataracts in birds are infrequent, although they have been seen in older canaries, parrots and birds of prey (Arnall and Keymer, 1975, Bird Diseases, T.F.H. Publications, Neptune City, New Jersey, pp. 305–308). Inbreeding and local trauma have been suggested as possible causes (Petrak, 1969, Diseases of Cage and Aviary Birds, Lea & Febiger, Philadelphia, Pennsylvania, pp. 351–355). Other reported possible causes of avian lens opacity or cataract include avian encephalomyelitis virus, Vitamin E deficiency and possible genetic

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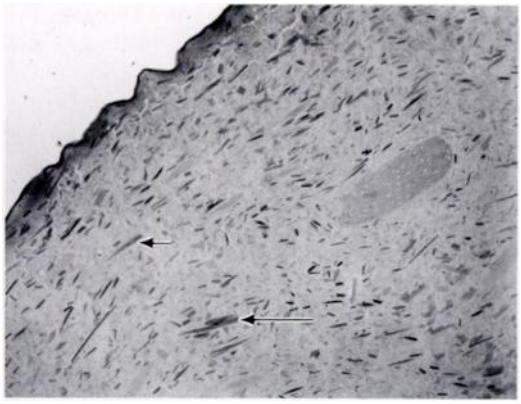

FIGURE 1. Cortex of affected left lens. Note wrinkled capsule, absence of epithelium, loss of normal lens structure and fragments possibly representing degenerated lens fibers (arrows). ×160.

FIGURE 2. Right eye with portion of relatively normal nuclear fibers. ×420.

defect in canaries (Mustaffa-Babjee, 1969, Vet. Bull. 39: 681–687). Keymer (1977, Avian Path. 6: 335–341) observed cataracts in 18 of 4,287 avian necropsies; most of the cataracts were associated with senility. Keymer did not attempt to determine the types of cataracts observed.

An adult female crested mynah, blind for approximately 4 mo, developed osteomyelitis of the first phalanx of the right foot and was euthanatized. The only gross lesion was a 1.0 × 0.7-cm mass involving the right foot. Histologically this was chronic, non-specific inflammation with new bone proliferation. Both eyes were examined histologically. The lenses were abnormal, consisting of amorphous, eosinophilic material containing darkly eosinophilic fragments. Each capsule was wrinkled, and lens epithelium was not seen (Fig. 1). In the right eye, a portion of relatively normal nucleus was seen (Fig. 2). In both eyes, there were foci of retinal degeneration with loss of photoreceptor nuclei and degeneration of inner and outer segments. The morphologic appearance of the lens lesions was compatible with hypermature cataracts, although the nuclear remnant in the right eye was somewhat suggestive of a morgagnian cataract. Keymer (1977, op. cit.) cited one report of morgagnian cataracts in a bird. No description was found of a hypermature cataract in birds.

No cause of the condition was suggested by the history or morphologic findings. The exact age of the bird was unknown.

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