These teeth appeared normal on gross, external examination. Jaws are seldom radiographed as a part of the clinical work-up in young dogs suffering from life-threatening renal disease. Even if the jaws were radiographed, standard lateral views cause superimposition of the teeth and identification of distinct mineralized lesions such as the ones seen in these dogs would be difficult. Occlusive dental radiography of the first mandibular molar of a series of young dogs suffering from severe renal disease should be done in order to determine the prevalence of dental dysplasia associated with congenital renal disease.

References


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Cerebellar Cortical Atrophy in a Baboon

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Cerebellar atrophy with Purkinje cell loss and Bergmann’s astrocytosis is a characteristic of several human syndromes including Friedreich’s ataxia, olivocerebellar atrophy, and cerebellar cortical atrophy of Holmes. A number of animal models of cerebellar atrophy also have similar morphologic features in dogs, mice, cattle, cats, and horses, most with autosomal recessive inheritance. Although cerebellar atrophy has been described in a Japanese macaque, we can find no published reports of spontaneous cerebellar atrophy in baboons. We describe a case of cerebellar atrophy with Purkinje cell loss in a clinically blind baboon.

Since 1980, four cases of blindness without apparent ocular lesions have occurred in juvenile baboons, *Papio cynocephalus anubis*, at the Primate Field Station, Regional Primate Research Center, University of Washington. All four animals were born and raised in an outdoor breeding colony consisting of approximately 200 baboons. Although clinical signs in each case were seen by 1 year of age, the exact age of onset

![Fig. 1. Cerebellum. Note reduced thickness of molecular layer and hypocellularity of granular layer. HE. Bar = 100 μm.](image)

![Fig. 2. Higher magnification of Fig. 1. Note absence of Purkinje cells and Bergmann’s astrocytes (arrow). HE. Bar = 15 μm.](image)
could not be determined because baboon infants are usually carried by the dams until 6–10 months of age. No neurologic lesions were detected in cases 1 through 3. Case 4 was a 2½-year-old male baboon with progressive ataxia, seizures, and apparent blindness.

Physical examination of the animal under ketamine sedation revealed nystagmus and myoclonus. The animal was oblivious to its surroundings and unresponsive to visual stimuli. Corneal and pupillary responses were normal in both eyes. Other cranial nerve responses were normal, as were segmental reflexes. Ophthalmoscopic examination was unrevealing. Response to visual evoked potentials was within normal limits, as were blood counts and serum chemistries.

The animal was euthanized. The only remarkable gross pathologic finding was a marginally small cerebellum with slightly thinned folia. Histopathologic abnormalities were limited to the cerebellum. The molecular layer in all sections of the hemispheres was abnormally thin, and the granular layer was diffusely hypocellular. Purkinje cells were absent throughout the cerebellar hemispheres (Figs. 1, 2), and Bergmann's astrocytes were markedly increased when compared with a normal baboon cerebellum (Fig. 3). The vermis was normal. No morphologic abnormalities were found in eyes, optic nerves, visual tract, or in the rest of the central nervous system. Pedigree information on this case is incomplete, as both of its parents were imported. Its sire was imported at the same time and from the same source as the sire of a previous similar case, so the two males may have been related.

Cerebellar atrophy can affect the visual system and cause functional blindness in the absence of primary lesions in the visual tract. Ocular flutter can prevent fixation of the gaze, and ciliary muscle flutter can prevent accommodation, resulting in the inability to form an image. Although visual tests in this animal showed adequate perception of light, the lack of usable sight was apparent from the animal's behavior.

Although no cerebellar tissue from any previous case of blindness or ataxia in the baboon colony was available for examination, the potential of this syndrome for development as an animal model of human disease merits further study.

Acknowledgements

Supported by NIH grants RR07019, RR01203, and RR00166. We acknowledge the assistance of Dr. Mark Sumi in the interpretation of the brain lesions.

References


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