Clinical History: Left and right eyes of a female CD-1 mouse of approximately 8 weeks of age. The ophthalmologic exam revealed, microphthalmos and focal posterior subcapsular opacity (cataract).

Necropsy Findings: Small globes

Fixative: Glutaraldehyde

Microscopic images:

Figure # 1: Right globe
Figure # 2: Left globe

Figure # 3: Right globe
Histopathologic description:

The cornea and lens of the right globe had a distorted shape which was an artifact probably due to the osmolarity of the glutaraldehyde. There appeared to be an excessive amount of central retina which is a common finding in microphthalmic globes. The central retina contained numerous folds which appeared thickened due to tangential planes of sectioning (Figures # 1, 2, & 3 - left and right globes). The posterior capsule of the left globe was covered by a thin layer of small vessels (persistent tunica vasculosa lentis) (Figure # 2). The region of the optic disc of the right globe appeared attached to the posterior capsule of the lens which was covered by a thin fibrovascular membrane (persistent hyperplastic tunica vasculosa lentis) (Figure # 3).

Morphologic diagnoses:

Microphthalmos (right and left globes)

Persistent tunica vasculosa lentis (left globe)

Persistent hyperplastic tunica vasculosa lentis (right globe)

Multiple retinal folds (right and left globes)

Associated clinical conditions:

Microphthalmos

Cataract

Comments:

The developing lens is covered by a network of vessels (tunic vasculosa lentis) that communicate with embryonic vessels that extend from the optic disc (hyaloid vessels) with associated mesenchymal cells (primary vitreous). All of these vessels should disappear by the time the eyelids separate. Sometimes the vessels persist and if they persist with fibrous or cartilaginous tissue the condition is referred to as hyperplastic.

Persistent hyperplastic tunica vasculosa lentis (PHTVL)/ persistent hyperplastic primary vitreous (PHPV) is a congenital eye anomaly that has been described in humans, dogs and mice (TgN326f1Rpw) and sporadically reported in cats, horses, rabbits and rats. PHTVL/PHPV may be associated with microphthalmos and cataracts; loss of vision in severely affected individuals is due to the development of cataracts. The main feature of this alteration is proliferation
and persistence of retrolental fibrovascular tissue. In one ultrastructural study by Boeve et al. in Doberman pinchers (hereditary), it was found that disturbed development of the posterior capsule is the initiating factor and that a primary metabolic disorder in the lens may be the cause for this embryonic tissue to disappear; although a cause has not been determined. In Doberman Pinchers, this condition starts developing between days 30–37 of gestation that approximately corresponded to 43–66 of human gestation. Retinal dysplasia with/without detachment as it was observed in this case, has been described in dogs with PHTVL/PHPV.

In this case the principal finding is an excessive amount of sensory retina for the size of the globe which results in multiple folds. The sensory retina does not appear to have degenerative changes and the “rosettes” are actually tangential planes of section. If the retina is truly thickened, then some retinal dysplasia may be present. Retinal dysplasia is a common congenital eye abnormality in laboratory rats but less commonly observed in laboratory mice.

The focal posterior subcapsular opacity noted clinically may just be the adherence of the optic disc region to the posterior capsular region of the lens. If there were morphologic changes in the lenticular cortex, then a posterior subcapsular cataract would be an appropriate diagnosis.

References:


Please send your comments/questions to the whole LCPG list by hitting “reply to all”.

A final document containing this material with answers and a brief discussion will be posted on the C. L. Davis website by the end of the current month (http://www.cldavis.org/lcpg_english.html).