Common themes in many endocrine diseases involving the eye include:

- Hypertension
- Neuropathy
- Lipid metabolism dysregulation

1. **Diabetes mellitus**

Dogs are much more likely to be affected vs. cats

Ocular complications relatively common

Possible complications:
- Cataract formation (most common)
- Dry eye (more common than previously thought)
- At risk for infected corneal ulcers
- Other neuropathies
- Retinopathy

**Diabetic cataracts**

**Pathophysiology:** Shunting of glucose into the aldose reductase pathway causing a buildup of insoluble sorbitol and an osmotic gradient drawing fluid into the lens.

**Risk:** 75% of diabetics will have cataracts within 1 year, 80% by 18 months.

**Clinical presentation:** diabetic will present with sudden onset blindness

**Clinical findings:**
- Bilateral mature intumescent (swollen) cataracts (no tapetal reflection).
- PLR/dazzle intact.
- ± Inflammation (hyperemia of conjunctiva/sclera, signs of discomfort, miosis, hypotony, pigment on lens) – indicates presence of uveitis which may be associated with leakage of lens proteins out of the cataractous lens or rupture of the lens resulting in acute severe uveitis (* suspicion of this warrants urgent referral)

**Good surgical candidates:** prompt referral after onset of blindness, no lens capsule rupture, minimal uveitis, no secondary glaucoma

**Poorer surgical candidates:** long duration of blindness prior to referral (long-standing uveitis increases risk of secondary glaucoma), lens capsule rupture, glaucoma preceding surgery, poor retinal responses.

**Clinical case management:** begin topical non-steroidal anti-inflammatories (q12-q6h dependent on perceived amount of uveitis). Discuss referral at the earliest convenient opportunity. Ideal to control other issues (dental disease, UTI etc.) before referral. If referral is not an option, continue on topical NSAIDS indefinitely, measure pressure intermittently. Monitor tear production (see below)

**Prognosis:** with surgical management, 85-95% success rate quoted, most common complications are glaucoma and retinal detachment. Without surgery – poor for vision, occasionally lens resorption can allow vision after a protracted period of time.

Possible future management options: “Kinostat” – topical aldose reductase
inhibitor, showed promise in initial clinical trials, but not licensed for use. Must be
given prior to cataract onset and consistently to prevent cataract onset.

Diabetic corneal disease
Pathophysiology: corneal neuropathy leading to reduced corneal sensation and
reduced reflex tearing.
Risk: post-phacoemulsification diabetics 35.7% incidence
Clinical presentation: no clinical signs to infected melting corneal ulcers
Clinical case management: tear supplementation (gels/ointments better than
drops due to longevity), consider adding cyclosporine topically if clinically
affected (discomfort, keratitis, discharge)

Other diabetic ocular diseases
Lipid
Ocular consequences of hyperlipidemia include
Lipid aqueous (secondary to uveitis)
Lipid keratopathy
Retinopathy (rare)

Diabetic ocular neuropathy
Usually subclinical
A feature of corneal disease, Horner’s syndrome may be increased incidence in
diabetics

Diabetic retinopathy
Although this is a very common complication in people, companion animals
rarely develop retinal complications of diabetes related to microvascular
disease.

2. Thyroid disease
Hypothyroidism: ocular disease related to hyperlipidemia, neuropathy
Hyperthyroidism: hypertensive ocular disease (hyphema, retinal detachment,
retinal hemorrhages)

3. Hyperadrenocorticism
Hypertension
Hyperlipidemia
Poor corneal wound healing
Central blindness: compression of chiasm due to pituitary macroadenoma

4. Sudden Acquired Retinal Degeneration Syndrome (SARDS)
Connection with endocrinopathy is unclear as underlying pathophysiology is
unknown.
Clinical presentation: More common in middle aged spayed female small-breed
dogs. Sudden onset (days to weeks) bilateral blindness. Associated symptoms
include: PU/PD, polyphagia, weight gain, hair coat changes – indicative of
hyperadrenocorticism (but only a small proportion will be confirmed as having
HAC).
Clinical findings:
- Resting mydriasis with sluggish PLR
- Normal fundic appearance in the early stages
- Hyper-reflective tapetum in advanced stages

Definitive diagnosis: electroretinography/chromatic PLR

Treatment: none proven, reported treatments include high doses of steroids, intravenous/intravitreal human immunoglobulin

Clinical study at NCSU: recruiting NOW until Fall 2017. Recruiting cases of SARDS, progressive retinal atrophy and hyperadrenocorticism (pituitary dependent) – trying to find out more about pathophysiology. Owner incentives are available – free diagnostics (treatment for pituitary-dependent HAC). Treatment trial for SARDS will hopefully be funded soon. Contact Dr. Freya Mowat (fmmowat@ncsu.edu) for more information on recruitment.

References


