Slide Recognition

Group 1
Q. Abnormalities?
A. Optic disc coloboma, choriodal hypoplasia
Q. 6 month old dog. Describe gross abnormalities. Most likely diagnosis?

A. Retinal detachment, 10x10mm bulbous protrusion from optic nerve; optic nerve coloboma with secondary retinal detachment
Q. Describe abnormalities. What is the pathogenesis of the retinal abnormality?

A. Two foci or peripapillary hyporeflectivity (retinal detachment), 0.5 of lateral disc has grey, out of focus portion (coloboma), Rhegmatogenous retinal detachment from poor attachment of neural retina to coloboma margin.
Q. Abnormality; anatomic location of abnormality
   A. Two, 0.25 disc diameter, hypo or depigmented foci; RPE (RPE “coloboma”)
Q. Most likely etiologic diagnosis; order (category) causing lesions
   A. Ophthalmomyiasis interna; diptera (flies).
Q. Etiologic diagnosis; preferred treatment
A. Dirofilaria immitis (in anterior chamber); surgical removal under dim light

Ocular larval migrans due to nematode
Surgical removal of worms + anthelmintic
Q. Conjunctival biopsy from a horse. Etiologic diagnosis? What ocular conditions may this etiology cause in the horse?

A. *Onchocerca cervicalis* microfilaria; conjunctival nodules/depigmentation, sclerosis/pigmentary keratitis, anterior uveitis, +/- peripapillary chorioretinitis.
Q. Appearance of the eye and forelimb in a horse. Most likely etiologic diagnosis? Life cycle of the causative agent? A biopsy of the affected area would demonstrate what abnormalities? Preferred treatment?

A. *Habronemiasis* (*H. muscae*); Adult in stomach, eggs passed in feces, face fly, aberrant deposition of larva in medial canthal region or limbs; PMNS, eosinophils, macrophages and other MNCs if chronic; systemic ivermectin, systemic NSAIDs, topical therapy as needed.
Q. Appeared in the conjunctival sac of a horse following flushing of the nasolacrimal system. Scale is in millimeters. Etiologic diagnosis?

A. Thelazia lacrimalis
Q. Appearance of the eye of a budgie. Most likely etiologic diagnosis? Preferred treatment?

Knemidokoptes pilae; Systemic ivermectin is curative

A. Knemidokoptes pilae; oral or parenteral ivermectin
Q. Abnormalities? Pathogenesis?

A. geographic, peripapillary pale area (hypopigmented or depigmented) with pigment clumping; retinal or chorioretinal degeneration, post inflammatory or ischemic
Q. Abnormalities? Pathogenesis and significance of findings?

A. Punctate, multifocal pale areas with central pigmented foci (depigmentation/hyperpigmentation); post inflammatory (viral?) retinal degeneration; common incidental finding

Multifocal chorioretinopathy/chorioretinitis (bullet hole lesions)

Peripheral depigmentation and central hyperplasia of the RPE due to presumed inflammatory damage, suggestive of historical uveitis
Q. Species?

A. Goat (caprine)

Sheep (ovine)
Slide Recognition

Day 2
Q. Young goat. What are the abnormalities. What is the etiologic diagnosis. What is the prognosis and treatment. If this were a sheep, what would the most likely etiologic diagnosis be?

A. Deep corneal vascularization, stromal WBC, edema; prognosis often favorable, oxytetracycline, mycoplasma spp.; sheep=chlamydial spp.
Q. 7 year old Holstein cow. What is the most likely etiologic diagnosis? What other organs might be involved.

A. Lymphosarcoma (bovine leukemia virus); uterus, spinal cord, heart, abomasum

Etiologic diagnosis: Orbital Lymphosarcoma

Bovine leukemia virus

Other organs involved
Heart – cardiac arrhythmias, intestinal melena, Uterus and kidney – uterine and renal masses, lymph nodes – lymphadenomegaly
Q. Abnormalities?

A. Cataract, deep AC (resorption of lens)
Q. Funduscopic abnormalities? Most likely etiology?

A. Pale foci, pigment clumping, white, translucent foci adjacent to pecten and in periphery (widespread chorioretinal degeneration); trauma

Funduscopic abnormalities
Chorioretinal scaring with depigmented areas and degeneration
Pigment clumping
Vitreal condensation

Etiology: West Nile Virus
Secondary potential etiology: trauma
Q. Cuban Tree Frog. Describe the abnormality. What is the most likely etiologic diagnosis? What are the possible pathogenic mechanisms of the disease? What is the prognosis?

A. bilateral, yellow-gold medial stromal opacity; xanthomatosis; dietary or abnormal lipid metabolism; poor prognosis as other organs involved.

Abnormality: Bilateral, asymmetrical focal dense yellow corneal deposits
Etiologic diagnosis: lipid keratopathy, hypercholesterolemia, disseminated xanthomatosis
Possible pathogenic mechanism of disease: females – massive egg development suggesting that excessive lipid mobilization associated with oogenesis

High cholesterol diet
Prognosis: poor
Q. NZW rabbit. What is the abnormality? List two possible etiologic diagnosis.

A. Hypopyon, cataract, posterior synenchia, hemorrhagic white foci in iris (abscess); encephalitozoon cuniculi; pasteurella multocida
Q. Turkey with blindness. An epidemic of blindness exists in this farm production. List 4 possible etiologic diagnosis.

A. Marek’s disease, Avian Encephalomyelitis, Vitamin E deficiency, Genetic
Q. Clinical appearance and conjunctival cytology from a group of blue-fronted Amazon parrots. What are the clinical abnormalities? What is the cytologic findings? What is the etiologic diagnosis?

A. Chemosis/hyperemia, caseous lesion upper lid, severe blepharoedema and loss of filoplumes, heterophils and large inclusion bodies; pox virus
Q. Reticulated python. What is the diagnosis? What other non-ocular disease process might this animal exhibit (that is related to the ocular disease)? What is the preferred treatment?

A. Subspecular infection; stomatitis; systemic antimicrobials and excise small wedge from spectacle to allow drainage.

Diagnosis: subspectacular abscess/pseudobulphthalmos

Other non ocular disease process might this animal exhibit (related to ocular disease) - chronic stomatitis

Preferred treatment – surgical drainage by 30 degree wedge excision of inferior spectacle and flushing of the space with antibiotic solutions.
Q. Domestic ferret, Acute onset of blepharospasm and lethargy. The eyelids and appearance of the footpads are shown. What is the most likely etiology. What is the prognosis?

A. Canine distemper virus; grave
Q. 3 year old cat. Fine needle aspirate of the lesion stained with Dif Quik showed macrophages (giant cells), lymphocytes, and plasma cells. Clinical diagnosis? Preferred treatment?

A. Lipogranulomatous conjunctivitis; surgical excision.
Q. 3 year old Chow-Chow. List 6 autoimmune diseases that could cause these abnormalities.
A. P. vulgaris, P. foliaceous, E.multiforme, SLE, DLE, TEN
Abnormalities – Radial pigment dispersion on the anterior lens capsule, cataractous change, posterior synechia, dyscoria, iris hyperpigmentation, entropion uvea, mild conjunctival hyperemia

Breed – Golden Retriever

Q. Abnormalities? Breed?

A. Iris hyperpigmentation, pigment dispersion on ACL, lens membrane dorsally, dyscoria/posterior synechiae, cataract; Golden Retriever
Q. Species?

A. Galagos (Bush Baby)

Wild cat (Jaguar?)

ground squirrel

squirrel
Slide Recognition

Day 3
Q. List 7 methods of surgically treating this abnormality.

A. Robert’s technique, Dziezyc modification, Collagen injections, cryosurgery, lateral canthal transposition (Blogg), Lip to lid, oral mucosal grafts, Cross-lid flap
Q. List 6 surgical procedures that could be used to treat this neoplasm of the eyelid.

A. Sliding H graft, rotational graft from medial region, bucket handle, lateral transpositional graft, oral mucosal graft, lip to lid.
Q. What surgical procedure has been performed in this Chow-Chow?

A. Brow-lift procedure
Abnormalities: anterior displacement of the third eyelid, pink fleshy multilobulated mass (1/3 of visible third eyelid) caudal to the third eyelid, serosanguinous discharge, bulbar conjunctival hyperemia.
Most likely neoplastic diagnosis: Adenocarcinoma of the gland of the TE
2 year old mixed breed dog. Most likely diagnosis?
Severe follicular conjunctivitis
Most likely diagnoses?
Adenoma with secondary chalazaion
Q. Most likely diagnosis?
A. Diffuse episclerokeratitis
Q. abnormalities; 2 differential diagnoses
A. Blue discoloration to sclera (scleral thinning); congenital staphyloma, post-inflammatory scleral thinning

A. Upper lid trichiasis/entropion; Stades procedure, incision near lid margin, second elliptical incision 15-25mm dorsal, excise skin; undermine dorsal incision slightly, pull dorsal incision line ventrally and suture 5mm from ventral incision.

Distchiae, epiphora;

Cryotherapy and epilation
Q. Two most likely clinical diagnoses?
A. Meibiomitis (esp staph spp); chalazion
Q. Describe the abnormalities. Diagnosis? Risk Factors?

A. Massive bullous lesion in central cornea with rim of adjacent edema, vascularization; Acute bullous keratopathy (eruptive bullous keratopathy); systemic cyclosporine or corticosteroids
Q. 2 year old mixed breed dog from Central America. Non-painful. Most likely diagnosis? Prognosis?

A. Florida Spots (Florida keratopathy), good prognosis (non-progressive)
Q. 8 year old dog. Most likely diagnosis? Preferred treatment?

A. Epithelial inclusion cyst; keratectomy
Q. Appearance of the cornea following a intracameral tPA injection. Abnormality and how did it occur? Prognosis?

A. 6-7mm dense white spiculated opacity in cornea (Stromal fracture), corneal vascularization; Intrastromal injection of air or medication. Good prognosis, resolves in 24h.
Q. Two examples of a 4 week old puppy, examined for a non-painful eye. Corneal abnormality and most likely diagnosis? Prognosis?

A. faint, relucent, geographic corneal opacity; “Infantile corneal dystrophy”; self-resolving.

Subepithelial generalized corneal epithelial hazy opacity  
Infantile corneal dystrophy (subepithelial)  
Good
Q. Most likely diagnosis?
A. Eosinophilic keratoconjunctivitis
Q. Species?

A. Lion

Feline

rabit

Rabbit
Slide Recognition

Day 4
Q. 3 year old feline. What is the most likely diagnosis? What would cytology from this lesion demonstrate.

A. Eosinophilic keratitis; lymphocytes, PCs, pockets of eosinophils, mast cells, PMNs if necrotic or acute

a) eosinophilic proliferative keratoconjunctivitis
b) epithelial cells, eosinophils, neutrophils, mast cells
Q. Abnormalities? Clinical diagnosis? Breed?

A. White, crystalline, spiculated central corneal opacity; corneal lipid dystrophy; S. Husky.

a) Axial oval iridescent white corneal opacity
b) Crystalline corneal dystrophy
c) Siberian Husky
Q.: Describe the abnormalities. What is the most likely diagnosis.

A. Extensive corneal vascularization, diffuse edema, multifocal punctate grey to white corneal opacities, multifocal ulceration; superficial punctate keratitis
Q. Dog with chronic anterior uveitis. Describe the abnormalities. What is the pathogenesis of the lesions?

A. white, granular infiltrate in peripheral (arcus) and central cornea, associated vascularization. Alteration in corneal lipid metabolism, corneal vascularization, extracellular lipid deposition, need to rule out systemic hyperlipidemia.

a) Arching geographic iridescent white corneal opacity from medial limbus extending to paraxial cornea over 6 clock hours and multifocal iridescent white opacities less dense axially, few branching thin blood vessels

b) inflammation causing cellular death and release of lipid into the stroma from fibroblasts and keratocytes; additionally, changes in pH and temperature from chronic inflammation lead to precipitation of calcium. Lastly, chronic use of topical steroids promotes binding of calcium.
Q. Dog with hypothyroidism. Describe the abnormalities. What is the common term for this ocular condition. What is the pathogenesis.

A. White granular, arc-shaped corneal infiltrate in temporal, peripheral cornea. Arcus lipidosis; increased lipid levels, lipid diffuses from conjunctival vessels, deposited in avascular peripheral cornea

a) Arching geographic iridescent white corneal opacity from lateral limbus extending to paraxial cornea over 7 clock hours
b) corneal arcus lipidosis
c) hypercholesterolemia & hypertriglyceridemia lead to pathological deposition of fat in cornea from perilimbal blood vessels
Q. What surgical procedure has been performed.
A. thermokeratoplasty
Q. What procedure has been performed on this dog?

A. Cyanoacrylate glue application for corneal ulcer
Q. Describe the abnormalities. What is the most likely etiologic diagnosis? What is the preferred treatment.

A. raised, red mass in peripheral cornea and temporal conjunctival, corneal edema. hemangioma/hemangiosarcoma; surgical excision( keratoconjunctivectomy) +/- ancillary therapy
Q. What surgical procedure was performed in this dog, and what postoperative complication is seen?

A. Parotid duct transposition. Calcium deposits from saliva.
Q. 8 year old cat with change in appearance of eye. Most likely diagnosis? Suggested diagnostic or follow-up plan?

A. Rule out iris melanosis or melanoma; Plan is controversial, could include monitoring, cytology, biopsy, or enucleation.

a) Feline diffuse iridal melanoma

b) Diagnostics: FNA, biopsy.
Follow up - let client decide to monitor with photographs in 1-2 months or enucleation.
Q. 6 year old cat. Most likely diagnosis? Treatment options?

A. Iris melanoma (note dyscoria); iridectomy, laser, monitor for progression
Q. Prognosis?

A. Uncertain?, lesion does not appear to have reached opening of ciliary cleft, but may well involve deeper layers of iris stroma.

Guarded, pigment encroaching on ICA but remains confined to iris.
Q. 6 year old Labrador retriever. Most likely diagnosis? Treatment options?

A. Iris melanoma (melanocytoma); iridectomy, laser ablation, monitor
Q. Species?

Alpaca
A. Llama

Deer

Slide Recognition

Day 5
Q. Fixed specimen from a 8 year old dog. What is the gross pathologic abnormality

A. Nodular mass in region of ciliary body, lens displacement

Nodular, well circumscribed, tan/pale mass arising from the ciliary body and displacing the lens
Q. What is the gross pathologic abnormality. What is the most likely diagnosis. What is the prognosis (biologic behavior) of this condition

A. Pigmented mass on posterior pole of globe, retinal detachment; choroidal melanocytoma; locally invasive, but benign, not metastatic
Q. 14 year old cat, appearance of eye 14 months following an evisceration. What is the abnormality. What is the most likely diagnosis.

A. Pink mass in ventral corneal region; recurrence of intraocular neoplasia for which globe eviscerated, here a ciliary body adenocarcinoma.
Q. Feline eye. What is the most likely clinical diagnosis. What cell layers contribute to this lesion.

A. Uveal cysts; posterior iris epithelium and/or ciliary epithelium
Q. Slit-lamp photograph. What is the abnormality?

A. Keratic precipitates
Q. Abnormalities, pathogenesis?

A. Turbid, milky appearance to aqueous; hyperlipidemia + uveitis

Lipid aqueous; breakdown of blood aqueous barrier (uveitis) with hyperlipidemia
Q. Equine eye. The eye is non painful and the abnormality was noted incidentally. What is the most likely, non-neoplastic clinical diagnosis?

Iris hypoplasia

A. Iris stromal cyst (stromal hypoplasia)
Q. Most likely etiologic diagnosis?
A. Diabetes mellitus
Q. Abnormalities?

A. Microphakia, aphakic crescent, elongated ciliary processes
Q. Canine 7 years old. Camera is focused on the lens. What is the most likely etiologic diagnosis?
A. Hypocalcemia
Q. Biomicroscopic abnormalities?

A. Complete (intumescent, hypermature) cataract, shallow AC, capsular or subcapsular plaques

Shallow anterior chamber, hypermature, intumescent lens with prominent y sutures
Q. 9 year old cat with intermittent anisocoria. What is the most likely etiologic diagnosis? What is the anatomic location of the lesion. What is the proposed pathogenesis?

**Spastic pupil syndrome, ciliary ganglion, FeLV**

A. Spastic pupil syndrome; FeLV; ciliary ganglion; SPS is risk factor for future lymphosarcoma development
Q Most likely diagnosis? Other clinical signs this patient might exhibit? Most likely location for lesion? 5 Differential diagnosis

Cavernous sinus syndrome
Other cranial nerve deficits
Neoplasia, vascular, infectious, inflammatory, trauma, idopathic, metabolic

A. Total ophthalmoplegia, efferent pupillomotor dysfunction; ocular movement paralysis (check VOR) ventrolateral strabismus, neurologic (brainstem) signs; intracranial (not orbital) lesion; ddx = causes of menigoencephalitis (GME, infectious) diabetic neuropathy, trauma, vascular (i.e. hypertension, hemorrhage, ischemia), neoplasia,

14 cases of idiopathic CNIII paresis/paralysis (no other neurologic findings with extended follow-up)

MRI = CNIII enlargement (n=11), contrast enhancement (n=12)

Internal ophthalmoplegia (n=3), total ophthalmoplegia (n=11)

Clinical signs improved (n=7, 5 with no therapy), unchanged (n=7)
Q. Diagnosis?

A. Facial n. paralysis with “pseudoproptosis”
Q. Present since birth……Pathogenesis?

Abnormal crossing of temporal optic fibers at chiasm (normally stay ipsilateral), part of the pathogenesis is related to retinal hypopigmentation which leads to this misrouting

A. Misrouting of optic nerve fibers with some temporal fibers crossing over; creating visual ambiguity and binasal hemianopia; esotropia may be attempt to compensate for visual ambiguity
Q. Species?

Primate
A. Primate (human)

Wallaby
Chinchilla
Slide Recognition

Day 6
Q. Dog with past history of head trauma. What are the abnormalities. Most likely diagnosis? What is the pathogenesis or mechanism of the lesion.

A. Lobular mass in dorsal conjunctiva (orbital fat prolapse); disruption of orbital septum (age-related, trauma)

**Abnormalities:** focal well-demarcated dorsotemporal subconjunctival multilobular mass between 10 and 1 o’clock. Mild ventronasal displacement of the globe. Mild epiphora.

**Most likely diagnosis:** given history of trauma, orbital/retrobulbar fat prolapse.

Other differentials include orbital or retrobulbar neoplasm (lacrimal gland adenoma/carcinoma, canine lobular orbital adenoma).

**Pathogenesis:** unknown in dogs, likely related to a rent in Tenon’s capsule (potentially secondary to trauma) or senile weakening of Tenon’s capsule (in humans).
Most likely diagnosis: extraocular polymyositis. Pathogenesis: CD3+ predominant lymphocytic myositis and myonecrosis. Prognosis and treatment: Guarded prognosis, as atrophy and fibrosis may lead to enophthalmos and strabismus. Recurrence is rare but reported. Treatment involves tapering oral corticosteroids and azathioprine.

Q. 2 year old Golden Retriever. What is the most likely diagnosis. What is the pathogenesis of this condition. What is the prognosis and preferred treatment.

A. EOM myositis; lymphocytic/plasmacytic (immune-mediate) inflammation of EOM; good prognosis with immunosuppressive therapy although recurrence possible
Q. 1 year old SharPei. What are the abnormalities? What tissue is affected pathologically. Other breed affected?

A. Fibrosing EOM myositis; fibrosis of medial, ventral rectii, ventral oblique muscle belly; Iris Wolfhound
Q. Ultrasound from a dog with acute onset exophthalmos. What are the ultrasonographic abnormalities? What is the most likely diagnosis?

A. Hyperechoic lesion adjacent to posterior pole, acoustic shadow; foreign body.
Q. List the abnormalities. List possible etiologic diagnosis.

A. Pink, cellular subretinal mass (5, 1.5 o.d.d) with adjacent retinal detachment; hyporeflective foci in fellow eye; 1. any metastatic neoplasia (LSA, sarcoma, carcinoma) 2. Inflammatory/infectious esp. fungal but less likely

Two different fundic images are shown, assuming bilateral lesions in a single cat. 
Abnormalities: focal, well-demarcated, circular and variably sized (1.5 to 4 optic disc diameters), and mildly raised subretinal or intrachoroidal tan-brown lesion at the tapetal-nontapetal junction in both eyes. Lesions are surrounded by poorly demarcated regions of subretinal edema, tapetal hyperreflectivity, and retinal degeneration with out-of-focus retinal vessels. 
Possible etiologic diagnoses: infectious (cryptococcus, histoplasmosis, toxoplasmosis, disseminated bacteremia/sepsis, blastomycosis) versus neoplastic (multicentric lymphoma, multiple choroidal melanomas).
Q. Abnormalities?

A. None, normal fundus

Variation of normal due to peripapillary tapetal hypoplasia
Q. Diagnosis?
A. Lipemia retinalis

Lipemia retinalis
Q. abnormalities? clinical diagnosis? stage?

A. Hyperreflective foci in area centralis, visual streak, “conus”; stage 4 taurine deficiency retinopathy

Abnormalities: horizontal band-like region of tapetal hyperreflectivity and retinal degeneration in the area centralis of a cat. Similar ancillary peripapillary and nasal fundic lesions. The lesions coalesce and are surrounded by thin rim of hyperpigmentation.

Clinical diagnosis and stage: feline central retinal degeneration (FCRD), or “taurine-deficiency retinopathy” (Stage 4).
Q. Fundus photograph, fluorescien angiographic appearance (15 seconds post injection) of a 12 year old cat with unilateral blindness. Describe the funduscopic abnormalities. Describe the angiographic abnormalities.

**Fundus abnormalities:** thick branching to patchy and coalescing regions of tapetal hyperreflectivity and retinal degeneration, also affecting and radiating from the peripapillary region. Lesions are surrounded by geographic to patchy hyperpigmentation. The optic nerve head is out-of-focus.

**Angiographic abnormalities:** perivascular and peripapillary extravasation (leakage) of fluorescein, early in angiogram.

A. Wedge-shaped areas of Tan to dark discoloration to tapetal fundus, tapetal hyperreflectivity, retinal vascular attenuation, peripapillary edema; hypofluorescence of tapetal fundus (non-perfusion), retinal vascular non-filling, peripapillary hyperfluorescence (pooling)
Fluorescein Angiography Phases

- Latency phase
- Pre-arterial (choroidal flush)
- Retinal arterial phase
- Retinal arterio-venous phases
- Retinal venous phase
- Recirculation phase
Fluorescein Angiography

Hyperfluorescence:
- Leakage from a vessel
- Accumulation in abnormal space or staining of existing (injured) tissues
- Greater visualization due to attenuation or loss of overlying structures

Hypofluorescence:
- Blocked fluorescence (hemorrhage, exudate, pigment)
- Vascular filling defects
Q. Describe the histopathologic abnormalities. What is the most likely etiologic diagnosis?

A. Neoplastic thrombi in choroidal vessel, one marginating or colonizing wall, panretinal necrosis, RPE hypertrophy and migration; angioinvasive pulmonary carcinoma with choroidal metastasis causing posterior pole ischemia

**Abnormalities:** suspected neoplastic cells in choroidal vessels, with associated RPE hypertrophy ("tombstoning") and true retinal detachment. The retina is severely diffusely degenerated down to a glial scar (i.e. rule out presence of chronic glaucoma versus chronic retinal detachment). Choroidal vascular necrosis and vascular endothelial hypertrophy and hyperplasia.

**Most likely etiologic diagnosis:** metastatic carcinoma (i.e. pulmonary carcinoma, mammary carcinoma, squamous cell carcinoma) or metastatic sarcoma (fibrosarcoma).
Q. Fluorescein angiogram 20 sec post injection. Abnormalities? Diagnosis?

A. Multifocal areas of hyperfluorescence (level of RPE), hyperfluorescence of the disc and peripapillary area; Multifocal chorioretinitis, optic neuritis.

Multifocal to coalescing punctate perivascular extravasation (leakage) of fluorescein.
Q. What are the funduscopic abnormalities. What is the most likely neoplastic etiologic diagnosis?
A. Diffuse hyporeflectivity to tapetal fundus (flat retinal detachment), multifocal hemorrhage; lymphosarcoma

Abnormalities: exudative retinal detachment with subretinal white exudate. Associated optic neuritis and multifocal intra- to subretinal hemorrhages. The optic nerve head is out of focus. **Most likely neoplastic etiologic diagnosis:** lymphosarcoma.
Q. Describe the abnormalities. Most likely non-metastatic neoplastic etiologic diagnosis.

A. Well demarcated, pink mass, (massive optic disc enlargement and protrusion), hyperemia. optic nerve menigioma….this was a glioma!
Q. Describe the gross abnormalities.

A. Optic nerve thickening, bilobed mass lesion extending into vitreous from optic nerve (optic nerve menigioma)

Severe diffuse pink-white expansion/thickening of the optic nerve and optic nerve head, causing the optic nerve head to extend or protrude into the vitreous chamber. Associated retinal detachment, which may be a true lesion or represent a fixation artifact.
q. Abnormalities?

a. Multifocal white-grey retinal foci in non tapetal fundus (deep retinal), focal alteration in tapetal reflectivity, optic disc swelling (out of focus to plane of retina)

Multifocal to coalescing gray-white, poorly demarcated and perivascular lesions in the nontapetal fundus. Focal serous retinal detachment associated underlying a retinal arteriole at the tapetal-nontapetal junction. Loss of detail and out of focus optic nerve head.
Q. H/E stained section. What tissue is being depicted? What are the microscopic abnormalities? What was the likely etiologic diagnosis? What would have been the preferred treatment in this animal? What is the term used to describe this set of lesions?

A. Lens; capsular rupture/scrolling, intralenticular PMN, degenerative lens fibers, Morgagnian globules; trauma; lens removal; phacoclastic uveitis or septic implantation syndrome

Tissue depicted: ruptured or perforated lens capsule and cataractous lens
Abnormalities: curling/scrolling of lens capsule, consistent with lens perforation and lens capsule rupture, with extralenticular extrusion of cataractous lens fibers. Severe intralenticular neutrophilic inflammation.
Etiology diagnosis: suspect cat claw penetrating (traumatic) ocular injury with second intralenticular bacterial infection
Preferred treatment: lensectomy
Term for “set of lesions”: septic implantation syndrome
Q. Canine eye. What are the microscopic abnormalities?

A. Closed iridocorneal angle (collapsed ciliary cleft), corneal vascularization/hypercellularity, pigment

Closed iridocorneal angle and collapsed ciliary cleft. The trabecular meshwork is not readily visible. The pectinate ligament is very thick/stout. Suspect goniodysgenesis. Associated moderate ciliary body atrophy (i.e. rule out presence of glaucoma) and moderate vascular keratitis.
Q. What species is represented. What are the characteristic features of the iridocorneal angle in this species?

A. Equine; prominent pectinate fibers; rudimentary scleral venous system; large ciliary cleft

Species: horse
Characteristic species features: prominent pectinate ligament and large ciliary cleft
Q. Species?

A. Cow

Pig

The answers for this question were already listed on the slide 😊
Slide Recognition

Day 7
Q. Abnormalities? What error in surgical technique is evident.

A. shallow AC (hypotony), focal anterior capsular or cortical cataract? possible corneal edema from endothelial damage?; tubing cut excessively long

Abnormalities:
Shallow anterior chamber, clear tubing with pigment deposition extending ~ 8 mm into anterior chamber, hyperpigmented uvea, cataractuous lens

Error in surgical technique: Gonio shunt tubing is advanced too far into the anterior chamber (avoid crossing the pupillary axis extending 3-4 mm into the anterior chamber)
Q. Equine eye. What are the abnormalities? What is the clinical diagnosis?

A. Diffuse corneal edema, corneal vascularization, stria; glaucoma

Abnormalities:
Dorsal and ventral straight 2-3 mm perilimbal vessels, diffuse corneal edema, two curvilinear Haab’s Striae in dorsonasal and dorsotemporal corneal quadrants, miotic pupil.

Clinical Diagnosis:
Glaucoma (most likely secondary glaucoma resulting from Equine Recurrent Uveitis)
Q. Abnormalities? Most likely diagnosis
A. Chemosis/hyperemia, corneal vascularization, multifocal punctate wqute foci esp. at termination of vessels (cellular infiltrate), conjunctival hyperemia; Immune Mediated Keratitis (IMMK)

**Abnormalities:** Conjunctival hyperemia and chemosis, prominent superficial and anterior stromal branching vascularization from the temporal limbus, faint perivascular edema, ventrotemporal focal yellow discoloration (consistent with fluorescein stain), few focal perivascular white opacities suggestive of cellular infiltrate or fibrosis

**Most likely diagnosis:** Immune mediated keratitis (IMMK) (epithelial vs superficial stromal)
Q. Slitlamp micrograph of a dog following extracapsular cataract extraction. The IOP is 55mm Hg. What postoperative complication is seen. What is the likely pathogenesis of this complication. What is the preferred surgical treatment of this complication.

A. Pupillary block glaucoma (from synechia or vitreous displacement or both) or aqueous diversion glaucoma; disrupt synechia if present, possible anterior vitrectomy, to reestablish normal aqueous flow.
Q. What are the funduscopic abnormalities? What is the clinical diagnosis?

A. 3-4ODD? hyporeflective foci in the dorsal fundus, with multifocal brown foci, anomalous or abnormally curved retinal venule; geographic retinal dysplasia; Labrador

**Fundoscopic abnormalities:**
multifocal brown/orange foci within a geographic area of altered retinal reflectivity under a retinal vein

**Most common large breed:** Mastiff
Q. Diagnosis?
A. Geographic retinal dysplasia
Q. Canine, Cocker Spaniel. What is the funduscopic abnormalities. What are the possible pathogenic mechanisms for this animal’s lesion.

A. Retinal folds; genetic vs. imbalance of scleral and chorioretinal development

Funduscopic abnormalities:
Multifocal vermiform grey streaks (consistent with retinal folds) visible ventral to the optic disc segregated along the tapetal and non-tapetal junction

Possible pathogenic mechanisms for this animal’s lesions: Per Silverstein et al. 1971:
a) hyperplastic extension of the neural retina into abnormal sites away from the RPE
b) dysplastic processes within the neural retina that is detached from the RPE
c) occurring in neuroretinal areas devoid of RPE (i.e. colobomas) and
d) dysplastic processes in most or all of the neural retina, or only focal areas, without evidence of separation from the pigment epithelium; the latter (d) could possibly involve defects in the pigment epithelium itself, because this structure has been shown to be important during retinal development
Q. Photomicrograph. What is the species (or class of animal)? What are the microscopic abnormalities. What is the morphologic diagnosis? What is the most likely etiologic diagnosis?

Species/Class of animal: ruminant (most likely cow)
Microscopic abnormalities: cross sectioned retinal folds observed
Morphologic diagnosis: Retinal dysplasia
Most likely etiologic diagnosis: Bovine viral diarrheal virus (cow); also consider blue tongue (sheep)

A. Ruminant (prominent retinal blood vessels protruding into vitreous + fibrous tapetum); retinal folds, rosettes; retinal dysplasia, BVD
Q. Abnormalities?

A. Extensive punctate diffuse alterations in tapetal reflectivity (tapetal degeneration), multiple pale foci in nontapetal fundus, equivocal retinal vascular attenuation? equivocal demyelination of disc at 12:00 - 3:00? None observed; considered normal variation.
Q. Canine. What are the funduscopic abnormalities? What is the breed of dog?

A. Retinal folds, geographic area of hyporeflectivity with area of central hyperreflectivity, tapetal color alteration; geographic retinal dysplasia typical of that seen in English Springer Spaniel
Q. abnormalities?

A. Multiple retinal folds, 3o.d.d. foci of chorioretinal hypoplasia, optic disc coloboma; choroidal hypoplasia ~2.5x size of the optic disc; papillary coloboma; multifocal vermiform streaks concentrated in the non-tapetum
Axial posterior capsular cataract with a network of blood vessels and circumferential fibrillar extensions

**Seen in what breed as a familial trait:** Doberman Pinscher (Staffordshire Bull Terrier)

Q. What are the abnormalities. In what breed might you see this disorder as a familial trait?

A. Focal cataract/vascular network presumably on posterior aspect of lens (PHPV); Doberman, Staff. Bull Terrier, Bouvier
Q. Camera is focused posterior to the lens. What is the diagnosis (es)? What is the composition of these lesions?

A. Astroid hyalosis, nuclear sclerosis; calcium-lipid complexes
Q. Slitlamp photograph of an 8 year old, male cat. What is the biomicroscopic abnormality? What is the clinical diagnosis? What infection pathogen has been suggested to be associated with this lesion?

A. Relucent bodies/cells in anterior vitreous; cyclitis or pars planitis; FIV
Q. Breed? Pathogenesis?

Superficial corneal pigment deposition, secondary to chronic irritation. Corneal pigmentation develops due to migration of perilimbal melanocytes into the cornea, which deposit melanin granules within corneal epithelial cells

A. Pug. Pigment deposition from adnexal conformational abnormalities
Q. Species?

A. Ferret

Guinea pig

canine

Guinea pig