

Case 1

Patient: Female, 62 years old.

Chief complaint: Gradual decrease in vision in both eyes for 2 years; inability to see clearly with the right eye for the past month; left eye pain and headache for the past 2 weeks.

History of present illness: The patient has experienced a gradual decline in vision in both eyes over the past 2 years, from 0.8 to 0.1, without ocular redness, eye pain, or tearing. One year ago, she was diagnosed elsewhere with "fundus hemorrhage" and was treated with oral hemostatic medication, after which her vision temporarily improved. During the past 6 months, her vision has fluctuated, accompanied by dark shadows/floaters in front of the eyes. One month ago, vision in the right eye deteriorated further. Two weeks ago, the left eye became red and painful, with tearing and headache, prompting this visit.

Past medical history: Diabetes mellitus for 10 years, controlled with medication.

General physical examination: Blood pressure 140/90 mmHg. General condition fair. Cardiopulmonary auscultation: negative. Abdominal palpation: liver and spleen not palpable. No abdominal mass, tenderness, or shifting dullness.

Ophthalmic examination:

Visual acuity: Right eye 0.1, left eye 0.05; J7 not visible in either eye. Intraocular pressure: R 5.5/5 = 17.30 mmHg; L 10/4 = 43.39 mmHg.

No eyelid edema in either eye. Right eye: conjunctiva not injected; cornea and anterior chamber clear; KPs (-); flare (-); iris neovascularization (-); mild cortical lens opacity. Left eye: ciliary injection (++); corneal epithelial edema (+); anterior chamber clear; KPs (-); flare (-); fine neovascular vessels visible at the pupillary margin at the 3 and 9 o'clock positions; mild lens opacity. Fundus: Right eye shows vitreous hemorrhage, more prominent inferiorly. Part of the superior retina is faintly visible. A proliferative membrane extends from the optic disc along the superotemporal vessels, with neovascularization on its surface. The retina shows patchy hemorrhages of varying sizes, cotton-wool spots, and hard exudates. In the left eye, the optic disc is hazily visible with a clear margin and slightly pale color. Retinal hemorrhages of varying sizes, circinate exudates at the posterior pole, microaneurysms, and a localized neovascular membrane in the superotemporal mid-periphery are visible. (See 1 fundus photograph.)

Laboratory results:

Complete blood count: (-)

Urinalysis: Pro 150 mg/dL, Glu 150 mg/dL, Ery 25/mL.

Liver function: (-)

Renal function: blood urea nitrogen 9.40 mmol/L; creatinine 122 mmol/L.

Blood glucose: 10.7 mmol/L

Preliminary diagnosis — Primary diagnosis:

Secondary diagnosis:

Diagnostic basis:

Differential diagnosis:

Diagnosis and treatment plan:

Answer:

Primary diagnosis: Stage V diabetic retinopathy in both eyes. (Proliferative vitreoretinopathy in both eyes, most likely diabetic in origin.)

Secondary diagnoses: 1. Secondary neovascular glaucoma, left eye.
2. Senile cataract in both eyes (early stage).

Diagnostic basis:

Primary diagnosis: History of diabetes mellitus; bilateral retinal hemorrhages, exudates, proliferative neovascular membranes, and vitreous hemorrhage.

Secondary diagnosis: Elevated intraocular pressure in the left eye, corneal edema, and iris neovascularization.

Mild lens opacity in both eyes.

Differential diagnosis:

1. Hypertensive retinopathy: Hypertension is present; optic disc edema is more common, with preceding arteriosclerotic changes.
2. Nephropathic retinopathy: Mainly characterized by retinal edema and exudative retinal detachment.
3. Central retinal vein occlusion: Usually unilateral; retinal hemorrhages are predominantly flame-shaped and patchy. Optic disc edema may also be present.
4. Retinal vasculitis: More common in young men; peripheral retinal veins show hemorrhage and perivascular white sheathing. Hemorrhage may recur and lead to vascular occlusion. Both eyes may be involved.

Diagnosis and treatment plan

Further examinations:

1. B-scan ultrasonography — to rule out retinal detachment.
2. FFA (fundus fluorescein angiography) — to assess areas of retinal nonperfusion and stage the disease.
3. Systemic evaluation: Renal function to rule out diabetic nephropathy; electrocardiogram; blood glucose.

Treatment recommendations

1. Systemic blood glucose control.
2. Lower intraocular pressure: topical medications (timolol, dipivefrin) and systemic treatment (acetazolamide, hyperosmotic agents). If the intraocular pressure cannot be controlled, cyclocryotherapy may be performed.
3. Retinal photocoagulation after the intraocular pressure has decreased.
4. Once the systemic condition is well controlled, vitrectomy with endolaser photocoagulation may be performed in the right eye.

Case 2

Medical record summary:

Patient: Male, 58 years old.

Chief complaint: Floating dark shadows and flashes in the right eye for half a month, with a curtain-like obstruction on the nasal side for 3 days.

(Past history: No other medical history. Denies history of trauma. Myopia of -2.00 D in both eyes.)

General physical examination: Normal.

Ophthalmic examination:

Visual acuity: Right eye: counting fingers at 1 foot; Jr7 not visible. Left eye: 0.7, Jr3. Intraocular pressure: R 5.5/9 = 8.54 mmHg; L 5.5/5 = 17.30 mmHg.

External examination of both eyes is normal. Right eye: conjunctiva not injected; cornea and anterior chamber clear, with the anterior chamber slightly deeper than the fellow eye; KPs (-); flare (++); mild cortical lens opacity. Left eye: no injection; cornea and anterior chamber clear, medium depth; KPs (-); flare (-); mild lens opacity. Fundus: In the right eye, floating blood is present in the vitreous, more prominent inferiorly. The temporal half of the retina is elevated, forming a gray hemispherical detachment. A localized brown-gray elevation is visible in the temporal periphery extending to the ora serrata. Left fundus is normal.

Laboratory results:

Complete blood count: (-)

Urinalysis: (-)

Liver function: (-)

Renal function: (-)

Blood glucose: (-)

Preliminary diagnosis — Primary diagnosis:

Secondary diagnosis:

Diagnostic basis:

Differential diagnosis:

Diagnosis and treatment plan:

Answer:

For diagnosis, the fundus should be examined for a retinal break. (On examination, a small horseshoe-shaped retinal tear was found in the superotemporal right retina.)

Preliminary diagnosis — Primary diagnosis: 1. Rhegmatogenous retinal detachment with choroidal detachment, right eye, PVR-B.

Secondary diagnoses: 2. Vitreous hemorrhage, right eye; 3. Senile cataract in both eyes (early stage).

4. Refractive error in both eyes.

Diagnostic basis:

1. History/symptoms: Dark shadows in front of the eye and a curtain-like obstruction.

2. Ocular examination: Vitreous hemorrhage, retinal detachment, and a retinal break are present. The anterior chamber is deep, intraocular pressure is low, flare is (++) , and

choroidal detachment is present. Mild lens opacity is present in both eyes.

Differential diagnosis:

1. Choroidal mass: Metastasis or choroidal melanoma may present as a solid brown-black, non-transilluminating choroidal elevation, with secondary retinal detachment on the surface. Choroidal melanoma is generally mushroom-shaped and has no retinal break. Intraocular pressure is normal or elevated. B-scan ultrasonography can help rule this out.
2. Vitreous hemorrhage from other causes: Diabetic retinopathy, hypertensive arteriosclerotic retinopathy, and other disorders generally show manifestations of the underlying disease, such as retinal vascular abnormalities, retinal hemorrhage, and exudation, without retinal detachment; these are usually not difficult to distinguish.
3. Uveal effusion syndrome: Choroidal effusion/detachment occurs first, followed by secondary retinal detachment without a retinal break. Subretinal fluid shifts with body position; there is no anterior segment inflammation, and intraocular pressure is normal.

Diagnosis and treatment plan:

Further examinations: Detailed fundus examination to locate retinal breaks. B-scan ultrasonography to confirm whether choroidal detachment is present. Assessment of visual function.

1. Pupil dilation and systemic or local corticosteroids: to treat the choroidal detachment as preoperative therapy.

2. Preoperative preparation: Complete blood count and urinalysis, general physical examination, topical antibiotics, intraocular pressure assessment, and lacrimal drainage system evaluation.

3. Surgical plan: Conventional retinal detachment surgery. Rationale: short duration of retinal detachment, no proliferative changes, and a retinal break is present.

Case 3

Medical record summary:

Patient: Female, 30 years old.

Chief complaint: Recurrent redness and pain in the left eye for 2 years, worsening over the past 10 days.

Two years ago, after a cold, the patient developed redness and pain in the left eye, photophobia, and tearing. She was diagnosed with keratitis at a local hospital and improved after treatment (medications unknown). During the following 2 years, redness and eye pain in the left eye recurred repeatedly. Ten days ago, after fatigue, redness and pain in the left eye recurred, with tearing and blurred vision, prompting this visit.

Past history: Denies history of trauma.

General physical examination: (-). Preauricular gland: (-).

Ophthalmic examination: Visual acuity: Right eye 1.0, Jr1; left eye 0.2, Jr4. Eyelids are normal in both eyes. Left eye: mixed conjunctival injection; central punctate corneal opacity/scarring (with one neovascular vessel growing into it); nasal corneal opacity and edema with stromal infiltration; fluorescein (FL) staining (+) (morphology shown in photograph); anterior chamber of medium depth; KPs unclear; flare (+); pupil round, 2 mm in diameter, with

sluggish light reaction; lens clear. Right eye: no injection; cornea and anterior chamber clear; KPs (-); flare (-); lens clear. Fundus: right eye normal; left fundus difficult to visualize.

Laboratory results:

Complete blood count: (-)

Urinalysis: (-)

Liver function: (-)

Renal function: (-)

Blood glucose: (-)

Preliminary diagnosis — Primary diagnosis:

Secondary diagnosis:

Diagnostic basis:

Differential diagnosis:

Diagnosis and treatment plan:

Answer:

Preliminary diagnosis — Primary diagnosis: Corneal ulcer, left eye; herpes simplex viral keratitis (dendritic keratitis).

Secondary diagnoses: Corneal opacity/scar, left eye; secondary iridocyclitis, left eye.

Diagnostic basis:

Primary diagnosis: History of recurrent redness and pain in the left eye. Ocular examination shows ciliary injection, corneal opacity and edema in the left eye, a right dendritic ulcer [as stated in the source], and positive fluorescein (FL) staining.

Secondary diagnoses: Corneal opacity/scar: punctate corneal opacity with neovascularization. Iridocyclitis: ciliary injection, small pupil, and flare (+).

Differential diagnosis:

1. Bacterial corneal ulcer: Often associated with a history of trauma, rapid progression, marked ocular irritation, and prominent photophobia and tearing. The corneal ulcer is irregular in shape and may progress in a creeping fashion; hypopyon may occur. Ulcer scraping may reveal bacteria or increased neutrophils. Bacterial culture may identify the causative organism.
2. Fungal corneal ulcer: May have a history of plant-related trauma, a prolonged course, relatively mild irritation, or prior corticosteroid use. The corneal ulcer appears as a gray-white infiltrate with a rough, elevated surface; shallow grooves or pseudopods may be present around the ulcer. Hypopyon is common. Ulcer scraping may reveal fungal hyphae and spores, and fungal culture may be positive.

Diagnosis and treatment plan:

Diagnosis:

Cytologic examination of corneal-ulcer scrapings: to differentiate bacterial, fungal, or viral infection and to rule out secondary bacterial infection.

Bacterial and fungal cultures of the corneal ulcer: etiologic testing to guide selection of

sensitive antimicrobial agents.

Treatment plan:

1. Subconjunctival injection of 0.2% cytarabine; 0.1% acyclovir eye drops q2h; 0.1% cycloctidine ophthalmic ointment qn.
2. Pupil dilation and oral nonsteroidal anti-inflammatory drugs for treatment of iridocyclitis.
3. Corticosteroids are contraindicated in epithelial lesions.

If the ulcer remains unhealed for a prolonged period, cauterization and debridement of the ulcer surface may be performed.

Case 4

Medical Record Summary

Patient: Female, 6 years old.

Chief complaint: Strabismus accompanied by decreased vision for 4 years.

History of present illness: At age 2, the child's parents noticed intermittent inward deviation of the left eye. At age 3, she was examined at a local hospital and was told that surgery could be performed later. At age 4, cycloplegic refraction at another hospital showed R: +6.00 DS +1.50 DC $\times$ 90; L: +5.50 DS +1.25 DC $\times$ 90. Visual acuity could not be tested. Glasses were prescribed, but she did not wear them consistently. At age 5, she was examined at another hospital and prescribed R: +3.00 DS = 0.5; L: +2.50 DS = 0.1. Visual acuity did not improve with glasses, and the strabismus has persisted to the present.

Past history: No remarkable maternal pregnancy or delivery history. Growth and development were normal. There was a history of upper respiratory infection with fever before onset. No other systemic disease. Vaccinations were received on schedule.

Family history: Unremarkable.

General physical examination: Normal.

Ophthalmic examination: Visual acuity: uncorrected R 0.5, L 0.1; with glasses R 0.5, L 0.1. Bilateral epicanthal folds. No congestion in either eye; corneas and anterior chambers clear. Pupils equal, 3 mm in diameter, with brisk light responses. Lenses clear. Undilated fundus examination: both optic discs relatively small, reddish, with clear margins; maculae (-), vessels (-). Ocular alignment without glasses: when the right eye fixates, left esotropia 30°; when the left eye fixates, right esotropia 30°. With glasses: alternating esotropia 20°, +45Δ; in upgaze, esotropia 15°, 30Δ; in downgaze, esotropia 40°, +75Δ. Ocular motility: no limitation in any direction in either eye. Both eyes elevate on adduction; elevation in adduction is excessive, while depression in adduction is normal.

Laboratory Results

Complete	blood	count:	(-)
Urinalysis:			(-)
Liver	function:		(-)
Renal	function:		(-)
Blood glucose:	(-)		

Preliminary Diagnosis / Answer

Primary diagnosis: Concomitant esotropia of the left eye/both eyes (partially accommodative),

associated with V-pattern and inferior oblique overaction.

Secondary diagnoses: 1. Bilateral refractive error (hyperopic astigmatism). 2. Bilateral amblyopia (refractive and strabismic).

Diagnostic Basis

Primary diagnosis: 1. Alternating esotropia with no limitation of ocular movements. 2. Esotropia decreases after correction of hyperopia. 3. The amount of esotropia changes between upgaze and downgaze in a V pattern (less esotropia in upgaze and greater esotropia in downgaze, with a difference greater than 15Δ).

Secondary diagnoses: 1. Bilateral cycloplegic refraction findings. 2. Corrected visual acuity remains below normal after wearing glasses, with both refractive and strabismic factors present.

Differential Diagnosis

1. Paralytic esotropia: the secondary deviation is greater than the primary deviation; abduction is limited and diplopia may occur.
2. Accommodative esotropia: esotropia associated with hyperopia. When hyperopia is uncorrected, excessive accommodative convergence causes esotropia, with an abnormal AC/A relationship. After wearing glasses, accommodation relaxes and the esotropia can be completely corrected.
3. Non-accommodative esotropia: generally no refractive error or only mild hyperopia; the amount of esotropia is the same with and without glasses.
4. Congenital esotropia: onset within 6 months after birth; the esodeviation is large and stable, without significant refractive error, and is not corrected by glasses. It may be associated with V-pattern, dissociated vertical deviation (DVD), inferior oblique overaction, etc.

Diagnostic and Treatment Plan

1. Cycloplegic refraction: 1% atropine bid for 3 days. R +5.50 DS +1.75 DC $\times$ 90 = 0.6; L +4.75 DS +1.25 DC $\times$ 90 = 0.1.
2. Prescription principle: reduce the spherical power by +0.5 to +1.0 DS in each eye, with the aim of reducing the accommodative demand and thereby decreasing the esodeviation.
3. Amblyopia training: improve visual acuity in both eyes in preparation for surgery. Rationale: surgery after improvement and equalization of binocular visual acuity makes acquisition of fusion more likely. Method: mainly occlusion therapy. Assess fixation pattern; use after-image therapy for central fixation and a red filter for eccentric fixation.
4. Surgery: timing—when visual acuity in both eyes has improved and become similar. Method—horizontal rectus recession/resection. For V-pattern: upward transposition of the lateral rectus muscles and downward transposition of the medial rectus muscles. For inferior oblique overaction: inferior oblique recession, tenotomy, or anterior transposition.

Case 5

Medical Record Summary

Patient: Male child, 2 years old.

Chief complaint: Admitted because a white reflex had been noticed in the left pupillary area 3 months earlier.

History of present illness: Three months earlier, the parents incidentally noticed a yellow-white reflex in the child's left pupil under lamplight at night. At that time there was no redness, swelling, or pain in the left eye. The next day he was examined at a local hospital. Because of his young age, visual acuity was not tested, and leukocoria was diagnosed. Further examination at a larger hospital was advised, but this was not pursued for various reasons. The child's general condition remained unchanged. About 1 week ago, the left eye became red and painful with tearing; he was irritable and crying, and the cornea appeared hazy, prompting this visit.

Past history: Second child, born at term by spontaneous delivery. Normal growth and development, no systemic diseases, and vaccinations received on schedule. One older sister is healthy.

Family history: Parents are healthy; no family history of hereditary disease.

General physical examination: General condition good. Heart and lung auscultation normal. Abdomen soft and flat; liver and spleen not palpable. No abnormalities of the limbs or spine.

Ophthalmic examination: Visual acuity could not be tested. Left eye: mixed conjunctival injection, mild conjunctival edema, mild diffuse corneal edema, anterior chamber of medium depth with a 1-mm gray-white opacity inferiorly; KPs unclear, flare $\pm$; pupil 4 mm with sluggish light response. Lens clear. A yellow-white reflex was seen in the vitreous behind the lens. A yellow-white elevated lesion was visible at the posterior pole, with shallow retinal detachment over its surface and vessels coursing over it. Mild vitreous opacity with several floating white clumps of varying sizes. Digital intraocular pressure T+1. The left eye did not fixate and had 15° esotropia. Right eye: no congestion; cornea and anterior chamber clear; undilated fundus showed normal optic disc and macula. Ocular movements were normal in all directions in both eyes.

Laboratory and Ancillary Tests

Complete blood count: (-); urinalysis: (-); liver and renal function: (-).
Chest fluoroscopy: (-); orbital plain X-ray: (-).

Preliminary Diagnosis / Answer

Primary diagnosis: Retinoblastoma (Rb) of the left eye, glaucomatous stage.

Secondary diagnoses: Secondary retinal detachment of the left eye; pseudohypopyon; concomitant esotropia of the left eye (sensory/disuse type).

Diagnostic Basis

Young child with left leukocoria; a yellow-white solid elevated fundus lesion with shallow retinal detachment is visible. Intraocular pressure is elevated. Hypopyon is present. The left eye is esotropic, does not fixate, and ocular motility is normal.

Differential Diagnosis

1. Congenital cataract: complete or partial lens opacity, without hypopyon or secondary glaucoma; ocular ultrasonography is normal.
2. Endophthalmitis: low intraocular pressure, anterior chamber and vitreous opacity; the history may include infectious factors such as fever or a systemic infectious focus.
3. Coats disease: large areas of outer retinal exudation at the posterior pole with subretinal cholesterol crystals, vascular dilation and tortuosity, neovascularization, and vascular tumors. Secondary exudative retinal detachment may cause leukocoria. Most common in children aged 6–

10 years, usually unilateral, without a hereditary family history. Fundus fluorescein angiography and B-scan ultrasonography can confirm the diagnosis.

4. Retinopathy of prematurity (ROP): history of prematurity and oxygen therapy; may be associated with cataract. A vascular proliferative membrane behind the lens extends toward the peripheral retina. Usually bilateral, with peripheral vascular tortuosity and dilation, neovascular proliferation, vitreous hemorrhage, and even retinal exudation/detachment causing leukocoria. The pupil may dilate poorly and iris vessels may be dilated. Regression leaves scar tissue, and vision is often severely impaired.

5. Persistent hyperplastic primary vitreous (PHPV): a relatively dense central fibrovascular membrane in the vitreous cavity behind the lens. The affected eye is often slightly smaller than the fellow eye; the lens is often opaque. Traction of the retrolental fibrous membrane may elongate the ciliary processes, and secondary glaucoma and retinal detachment may occur. It is present from birth but often detected during childhood, usually unilateral and without a hereditary family history. (The original notes state that full credit is given for the first three items plus either one of the last two.)

Diagnostic and Treatment Plan

1. B-scan ultrasonography of both globes to confirm a solid intraocular space-occupying lesion in the left eye and assist in differential diagnosis.
2. Detailed dilated fundus examination to exclude early disease in the right eye.
3. Optic foramen X-ray, or CT/MRI, to exclude orbital and intracranial metastasis. Calcification on orbital X-ray supports the diagnosis of retinoblastoma; a negative result does not exclude it.
4. Examine the preauricular gland/nodes and left supraclavicular lymph nodes. (The source notes may provide: preauricular gland/nodes [-], left supraclavicular lymph nodes [-].)
5. Complete systemic examination and preoperative preparation.

Treatment

1. After confirmation of the diagnosis, enucleation of the left eye may be performed. Avoid compressing the globe during surgery. The optic nerve stump should be longer than 10 mm, and hemostasis should be ensured. After enucleation, inspect the globe wall for tumor extension and the optic nerve for thickening. If present, postoperative radiotherapy or chemotherapy should be performed.
2. During surgery, perform a detailed dilated fundus examination of the right eye to exclude early lesions. If lesions are present, cryotherapy or radiotherapy should be performed.
3. Send the enucleated globe for postoperative pathological examination to determine whether optic nerve metastasis is present. If present, perform cranial CT to exclude intracranial metastasis. If intracranial metastasis is found, obtain a neurology consultation; radiotherapy may be performed.
4. Postoperative follow-up should be continued, with observation of the right eye to the age specified in the original plan (the source leaves the age blank). Genetic counseling is recommended.

Case 6

Medical Record Summary

Patient: Male, 72 years old.

Chief complaint: Intermittent distending pain in the right eye with colored halos for 3 years.

History of present illness: Three years ago, without an obvious trigger, the patient developed distending pain and colored halos in the right eye, which resolved spontaneously after rest. He was subsequently examined at a hospital; no abnormality was found, but glaucoma was suspected. During the past 2 years, he has had multiple episodes at night of right-eye distending pain, blurred vision, and colored halos, generally improving after sleep and rest. One year ago, after becoming anxious and angry, he suddenly developed severe right-eye pain with a sharp decrease in vision, accompanied by nausea and vomiting. At the hospital, intraocular pressure was as high as 81.65 mmHg; corneal edema and an extremely shallow anterior chamber were noted. After treatment with hyperosmotic agents, oral Diamox, and topical 2% pilocarpine, the intraocular pressure returned to normal. At present, the right eye is treated with 2% pilocarpine eye drops 6 times daily and 0.5% timolol bid. The left eye receives 2% pilocarpine bid and has never had an attack. Appetite, sleep, urination, and bowel movements have been normal since onset.

Past history: Diabetes mellitus for 3 years, currently well controlled with medication. Hyperopia for 40 years. Denies cardiovascular or infectious diseases, trauma, or drug allergy.

Personal history, family history, and general physical examination: No special findings recorded.

Laboratory Results

Routine blood and urine tests: normal.
Liver and renal function: normal. Blood glucose: 7.3 mmol/L.

Ophthalmic Examination

Visual acuity: R 0.4 (+5.25 S +1.50 C $\times$ 6 $\rightarrow$ 0.8); L 0.3 (+3.50 S +0.75 C $\times$ 160 $\rightarrow$ 1.0). Intraocular pressure: R 30.39 mmHg; L 20.55 mmHg. Eyelids, lacrimal apparatus, and conjunctivae normal in both eyes. Corneas clear; a few brown KPs inferiorly in the right eye. Both anterior chambers shallow, peripheral depth 1/4 CT. Localized iris atrophy at 3 o'clock in the right eye. Right pupil 3 mm, slightly irregular, with sluggish light response. Right lens: punctate anterior subcapsular opacities and mild cortical opacity. Left iris normal; pupil 2 mm, round, with brisk light response. Left lens: mild cortical opacity. Vitreous normal in both eyes. Fundus: right optic disc pale, C/D 0.7; foveal light reflex visible, with a small amount of surrounding pigment; vessels normal. Left optic disc normal in color, C/D 0.2; foveal light reflex visible. Gonioscopy: right angle narrow grade III–IV with partial peripheral anterior synechiae (PAS); left angle narrow grade III. Visual field: moderate concentric constriction in the right eye; left normal.

Preliminary Diagnosis / Answer

Primary diagnosis: Acute angle-closure glaucoma in both eyes — chronic stage in the right eye and preclinical stage in the left eye.

Secondary diagnoses: Early senile cataract in both eyes; bilateral refractive error; diabetes mellitus.

Diagnostic Basis

1. History and symptoms: recurrent distending pain, decreased vision, and colored halos in the right eye; intraocular pressure previously reached 81.65 mmHg.
2. Examination and signs: typical glaucomatous triad in the right eye (pigmented KPs, iris atrophy, and glaukomflecken). Lens opacities in both eyes. Refraction demonstrates hyperopia.
3. Ancillary examinations: concentric visual-field constriction in the right eye and PAS on gonioscopy.
4. The left eye has never had an acute attack.
5. History of diabetes mellitus; blood glucose 7.3 mmol/L.

Differential Diagnosis

1. Chronic angle-closure glaucoma: distinguish from the chronic stage of acute angle-closure glaucoma. This case represents acute angle-closure glaucoma that became prolonged because it was not adequately treated. There is a history of acute attacks, the characteristic triad is present, and varying degrees of peripheral anterior synechiae are seen. In chronic angle-closure glaucoma, obvious ocular symptoms may be absent; intraocular pressure is generally moderately elevated, the anterior segment may remain unchanged until late disease, while the optic-disc cup/disc ratio becomes enlarged.

2. Open-angle glaucoma: lacks the characteristics of acute attacks and may have no obvious ocular symptoms. The anterior chamber is deep. In the chronic stage, optic-disc atrophy is obvious, the C/D ratio is large, and visual-field damage is typical. Gonioscopy should be performed for differentiation.

3. Acute gastrointestinal disease.

4. Migraine.

5. Intracranial disease.

Diagnostic and Treatment Plan

I. What additional examinations should be performed, and why?

1. Tonography of the left eye: because the left eye is in the preclinical stage. If the outflow facility (C value) is good, YAG laser iridotomy may be considered.

2. A- and B-scan ultrasonography of both eyes: because mild hyperopia is present in both eyes, these examinations can further measure axial length and calculate intraocular lens power. If the axial length is short, attention should be paid to the possibility of postoperative malignant glaucoma, and this should be emphasized when discussing the surgical prognosis. If necessary, lens extraction with intraocular lens implantation may be performed.

3. Corneal endothelial microscopy of both eyes: because the right eye has had glaucoma attacks for many years and the corneal endothelium has already been damaged, detailed preoperative examination is required to reduce the risk of postoperative corneal endothelial decompensation.

II. How should the treatment plan be formulated, and why?

Treatment should first be based on the current stage of glaucoma. The right eye has had recurrent attacks, topical medication is ineffective, intraocular pressure remains uncontrolled, and the disease is already at a moderate stage; therefore, filtering surgery should be performed. The left eye is in the preclinical stage; 1% pilocarpine eye drops may initially be used, and where available, YAG laser iridotomy should be performed.

Case 7

Case Summary

Patient: Male, 25 years old.

History: While striking a stone with a hammer, the patient felt that his left eye was injured by a flying fragment. He immediately experienced pain in the left eye, a flow of "hot tears," and blurred vision. He presented to the hospital half an hour later. Visual acuity was 0.8 in the right eye and 0.1 in the left eye. No wound was seen on the left eyelid. Ciliary injection was present in the left eye. At the 5 o'clock position, 1 mm inside the corneal limbus, a full-thickness corneal laceration approximately 2 mm long was seen. The wound was well apposed. Anterior chamber depth was normal, with aqueous flare (++). The pupil was 4 mm in diameter. Examination through the original pupil showed a clear lens and vitreous opacity. The fundus could not be clearly visualized; only the posterior pole of the retina could be seen and appeared reddish.

Laboratory Findings

Routine blood and urine tests: Negative / no significant abnormality.

Liver and renal function: Negative / no significant abnormality.

Ancillary Examination

Plain orbital X-ray and foreign-body localization radiograph.

Initial Diagnosis

Primary diagnosis: Penetrating injury of the left cornea.

Secondary diagnoses: Traumatic iridocyclitis of the left eye; intraocular foreign body to be ruled out.

Basis for Diagnosis

Primary diagnosis: Definite history of ocular trauma and a corneal wound.

Secondary diagnoses: Aqueous flare (+) and a history of injury by a foreign object; an intraocular foreign body must be excluded.

Differential Diagnosis

The history and signs of ocular trauma are clear, making differentiation from iridocyclitis due to other causes relatively straightforward. Further examination is required to exclude an intraocular foreign body.

Diagnostic and Treatment Plan

Examine the corresponding iris for a perforation. Perform a dilated examination of the lens, vitreous, and fundus. Purpose: to determine whether an intraocular foreign body is present and, if so, its location.

Perform additional imaging: X-ray examination; if a foreign-body shadow is detected, obtain foreign-body localization films. B-scan ultrasonography or CT may also be performed. Purpose: to determine the nature of the foreign body (metallic or nonmetallic, possibly magnetic) and its location.

If an intraocular foreign body is present, select the surgical procedure according to its location. Preoperative preparation should include lacrimal drainage irrigation, topical antibiotics, and a general physical examination.

Case 8

Case Summary

Patient: Male, 17 years old.

History: Decreased vision in both eyes for six months, worsening over the past two weeks, with a six-month history of unsteady walking. He denied headache but reported previous pain with eye movement. He smoked three cigarettes per day and had received no treatment.

Examination: Visual acuity was 0.1 in the right eye and 0.05 in the left eye, with no improvement by spectacle correction. Neither eye was injected. The corneas were clear and the anterior chambers were normal. Direct and consensual pupillary light responses were sluggish in both eyes. Fundus examination showed slightly indistinct optic disc margins with temporal pallor bilaterally. The foveal light reflex was absent. The arteriovenous ratio was normal, and no retinal hemorrhage or exudation was observed.

Past medical, personal, and family histories were unremarkable. General physical examination was normal.

Laboratory Findings

Routine blood and urine tests: Negative / no significant abnormality.

Liver and renal function: Negative / no significant abnormality.

Ancillary Examination

Visual fields: Bilateral central/paracentral absolute scotomas.

Visual evoked potentials (VEP): Prolonged latency and reduced amplitude in both eyes.

Initial Diagnosis

Primary diagnosis: Bilateral optic neuritis (suspected).

Secondary diagnoses: Partial optic atrophy; multiple sclerosis (neuromyelitis optica).

Basis for Diagnosis

Decreased vision, pain with eye movement, impaired afferent pupillary light responses in both eyes, slightly indistinct optic disc margins with temporal pallor, bilateral central/paracentral absolute scotomas on visual-field testing, and prolonged VEP latency with reduced amplitude in both eyes.

Differential Diagnosis

Intracranial space-occupying lesion: Papilledema may occur, usually with elevation of less than 3 D. Symptoms of raised intracranial pressure and meningeal irritation may include headache, nausea, and vomiting. CT or MRI can establish the diagnosis.

Tobacco-related toxic retinitis/toxic optic neuropathy.

Optic disc vasculitis: The optic disc may be swollen and hemorrhagic. The visual field may show enlargement of the physiologic blind spot or irregular defects. Visual loss is generally less severe than in optic neuritis.

Retrobulbar optic neuritis: Symptoms are similar, but there is no optic disc swelling. Recurrent attacks may lead to optic atrophy.

Diagnostic and Treatment Plan

Neurology consultation. The neurology consultation diagnosed neuromyelitis optica in the

setting of multiple sclerosis.

Medication: Prednisone 60 mg as a single dose for 6 days, followed by gradual tapering; vitamin C 0.1, vitamin B1, vitamin B12, V.S.K., ATP, and cephalosporin No. 6, as recorded in the source.

Follow-up one month later: Visual acuity improved to 0.4 in both eyes, and gait improved markedly.

Case 9

Case Summary

Patient: Male, 65 years old.

History: Progressive decrease in vision in the right eye for two years, without ocular redness or pain. Vision in the right eye worsened further over the past three months. For the last two days, the right eye had become red and painful, accompanied by headache.

Past history: No history of diabetes mellitus. Hypertension for 10 years, with blood pressure around 160/100 mmHg; antihypertensive medication was taken intermittently.

General examination: Blood pressure 150/100 mmHg; otherwise normal.

Ophthalmic Examination

Visual acuity: Right eye—light perception, with light projection tested at 3 m as recorded in the source; left eye—0.6. Red and green color perception was present.

Right eye: Mixed conjunctival injection, corneal edema, and a shallow anterior chamber. Peripheral anterior chamber depth was approximately 1/3 corneal thickness (CT), while the central chamber was markedly shallow, approximately 1 CT. Keratic precipitates could not be clearly assessed; aqueous flare was (+). The pupil was moderately dilated and fixed. Iris markings were indistinct. The lens was uniformly opaque. On fundus examination, the optic disc was only faintly visible and appeared reddish.

Left eye: No injection; clear cornea. Anterior chamber depth was approximately 1/2 CT peripherally and 3 CT centrally. KPs were absent and flare was absent. The pupil was normal in size and reacted briskly to light. Wedge-shaped cortical lens opacities were present. The optic disc and macula were normal; C/D ratio was 0.3.

Intraocular pressure: Right eye $10/3 = 37.19$ mmHg; left eye $5.5/5 = 17.30$ mmHg.

Laboratory Findings

Complete blood count: Negative / no significant abnormality.

Urinalysis: Negative / no significant abnormality.

Liver and renal function: Negative / no significant abnormality.

Initial Diagnosis

Primary diagnosis: Bilateral senile cataract—intumescent stage in the right eye and incipient stage in the left eye.

Secondary diagnoses: Secondary glaucoma of the right eye; hypertension.

Basis for Diagnosis

History of painless progressive visual loss; lens opacities in both eyes, with total opacity in the right eye and peripheral opacity in the left eye; shallow anterior chamber in the right eye, especially centrally; and elevated intraocular pressure in the right eye.

Differential Diagnosis

Primary acute angle-closure glaucoma: There may be a history of minor attacks and a family history. Predisposing anatomical factors include a small globe and shallow anterior chamber, and intraocular pressure may rise after dilation. In this case, there was no history of minor attacks, anterior chamber depth differed between the two eyes, and the lens was opaque and swollen, supporting differentiation.

Phacolytic (lens-protein–related) glaucoma: In a hypermature cataract, leakage of lens cortex may allow lens proteins and macrophages to obstruct the anterior chamber angle, impairing aqueous outflow and causing secondary glaucoma. Lens-protein debris and turbid aqueous may be present. In this case, the cataract was not yet hypermature, the aqueous was only mildly turbid, and no lens-protein debris was seen.

Pseudoexfoliation syndrome: Exfoliative material may be seen on the anterior lens capsule at the pupillary margin, with debris in the anterior chamber angle, and secondary glaucoma may occur. These findings were absent in this case.

Malignant glaucoma: More common after intraocular surgery. Contact between the ciliary ring and lens can cause posterior misdirection of aqueous into the vitreous, forward displacement of the lens–iris diaphragm, shallowing of the anterior chamber, and elevated intraocular pressure. Intraocular pressure may decrease after dilation.

According to the source, giving item 1 plus any one of items 2–4 was considered sufficient.

Diagnostic and Treatment Plan

Lower intraocular pressure medically. After excluding diabetes, administer an oral hyperosmotic agent and acetazolamide. Use topical mydriatics, antibiotics, and corticosteroid eye drops to reduce inflammation.

Perform general preoperative evaluation: internal-medicine assessment, blood-pressure measurement, routine blood and urine tests, blood glucose, ECG, lacrimal drainage irrigation, and intraocular-pressure monitoring.

Perform A- and B-scan ultrasonography to measure axial length and evaluate the posterior segment, exclude primary glaucoma and neovascular glaucoma caused by posterior-segment hemorrhagic disease, and prepare for intraocular lens implantation during cataract extraction.

Examine the anterior chamber angle and visual field of the left eye to exclude a primary mechanism. After lowering intraocular pressure, carefully examine the right fundus and exclude fundus hemorrhage.

Perform cataract extraction as soon as possible. Options include cataract extraction alone, cataract extraction combined with intraocular lens implantation, or phacoemulsification with intraocular lens implantation.

Case 10

Case Summary

Patient: Male, 38 years old.

Chief complaint: Decreased vision in both eyes accompanied by headache and nausea after a one-week cold.

Present illness: One week earlier, the patient developed a cold without fever, with rhinorrhea, cough, and generalized weakness. He improved after treatment, but then noticed blurred vision in both eyes with mild bilateral redness. There was no ocular pain, but slight tearing and photophobia.

were present. He also had marked headache and nausea without vomiting.

Past history: No similar previous episode. Previously healthy. General physical examination was normal.

Ophthalmic Examination

Visual acuity: Right eye 0.1; left eye, counting fingers in front of the eye. Bilateral ocular irritation (+) and ciliary injection were present. The corneas were clear. Gray-white, mutton-fat keratic precipitates (++) were visible inferiorly, with aqueous flare (++) . Partial posterior synechiae of the iris were present in the pupillary area of the left eye. The lenses remained clear.

Fundus: Both optic discs had clear margins and were slightly hyperemic. In the right eye, the retina appeared gray and elevated, with a large area of gray-white subretinal exudation. In the left eye, multifocal retinal elevations were present around the optic disc, with serous exudation beneath them and macular edema.

Laboratory and Ancillary Findings

Routine blood and urine tests: Negative / no significant abnormality. Liver and renal function: Negative / no significant abnormality.

Neurology consultation and cerebrospinal fluid examination: Increased lymphocytes and protein.

Initial Diagnosis

Primary diagnosis: Bilateral uveitis — Harada–Koyanagi syndrome (Vogt–Koyanagi–Harada syndrome).

Secondary diagnosis: Bilateral exudative retinal detachment.

Basis for Diagnosis

Decreased vision in both eyes after a cold, accompanied by meningeal irritation symptoms such as headache and nausea. Features attributed in the source to the Koyanagi form include anterior uveitis, positive KPs/flare, and posterior synechiae; features attributed to the Harada form include posterior uveitis with meningeal irritation. Choroidal exudation was present in the right eye, and subretinal serous exudation with macular edema in the left eye. The source also records systemic manifestations of tinnitus and alopecia.

Differential Diagnosis

Behçet disease: Recurrent uveitis (anterior disease often presenting as hypopyon iridocyclitis and posterior disease as retinal vasculitis), oral ulcers, polymorphic skin lesions, and genital ulcers. Treatment listed in the source includes immunosuppressants, cyclosporine, low-dose corticosteroids, and symptomatic treatment.

Rhegmatogenous retinal detachment: Symptoms may include floaters/dark shadows, metamorphopsia, visual-field defects, and decreased vision. Fundus findings include gray-white elevated detached retina, possibly vitreous hemorrhage and a retinal break. Treatment: surgery.

Acute retinal necrosis: Decreased vision with ocular pain, marked vitreous opacity, segmental retinal arterial occlusion, and patchy retinal necrosis at the equator and periphery. Treatment is mainly antiviral therapy (acyclovir), with vitrectomy in late stages.

Diagnostic and Treatment Plan

Ask about hearing and skin changes. The source records tinnitus and alopecia after onset, without skin changes.

Neurology consultation: Mild neck stiffness was found. CSF showed increased lymphocytes and protein.

Perform FFA and ICG to demonstrate choroidal leakage and help distinguish the condition from other diseases.

Treat with high-dose corticosteroids, initially with a loading/pulse dose followed by dose reduction after 5–7 days and low-dose maintenance. The source specifies prednisone 1–1.5 mg/kg/day for 1–2 weeks, reducing by 10 mg weekly; after reaching 30 mg/day, taper more slowly by 5 mg every 2 weeks; at 10–15 mg/day, maintain treatment for 3 months to 1 year. Also use mydriatics and nonsteroidal anti-inflammatory drugs.

Case 11

Case Summary

Patient: Male, 29 years old. He fell from a motorcycle and had loss of vision in the left eye for one day.

The previous day, he fell from a motorcycle and struck the left side of his head. The left temporal facial region became swollen and painful with slight skin oozing. There was no loss of consciousness. Left-sided epistaxis occurred. After opening his eyes, he found that he could not see with the left eye.

Past history: Previously healthy, with good vision in both eyes. General physical examination was normal.

Ophthalmic Examination

Visual acuity: Right eye 1.0; left eye, hand movements in front of the eye. Mild edema of the left eyelid and zygomatic-temporal region was present, with a skin abrasion superotemporal to the eyebrow. Ocular movements were good in both eyes and both corneas were clear. The left pupil was dilated to 6 mm; the direct light reflex was absent and the consensual reflex was present. The right pupil was 3 mm; its direct light reflex was present and consensual reflex was absent. Fundus examination was normal in both eyes.

Laboratory Findings

Routine blood and urine tests: Negative / no significant abnormality. Liver and renal function: Negative / no significant abnormality.

Initial Diagnosis

Primary diagnosis: Indirect traumatic injury of the left optic nerve.

Secondary diagnosis: Soft-tissue contusion of the left eyelid.

Basis for Diagnosis

A history of direct trauma to the left fronto-orbital region followed by blindness in one eye. The injured eye had extremely poor vision, a dilated pupil, absent direct light reflex but preserved consensual response. In the healthy eye, the direct response was normal while the consensual response was absent. The fundus of the injured eye was normal in the early stage.

Differential Diagnosis

Intraocular injury: Abnormal findings would be present in the vitreous or fundus.

Unilateral oculomotor nerve palsy: Both direct and consensual light reflexes are absent,

without severe visual impairment.

Diagnostic and Treatment Plan

Optic canal X-ray: The source describes deformity of the optic canal opening, discontinuity of bone, and fracture of the lesser wing of the sphenoid.

CT of both optic canals: Discontinuity of the bony wall of the optic canal, a teardrop-shaped shadow, and deformation of the sphenoid sinus.

VEP: Reduced P-wave amplitude and prolonged peak time, indicating impaired optic-nerve conduction.

Treatment: Surgery as early as possible in an attempt to rescue vision.

Comprehensive medical treatment: dehydration therapy, vasodilators, neurotrophic agents, antibiotics, and corticosteroids. The source also lists hyperbaric oxygen and 'light quantum' therapy.

Case 12

Case Summary

Patient: Male, 38 years old. Chief complaint: Diplopia for three weeks.

Three weeks earlier, the patient was punched in the left eye. The eye immediately became swollen, and vertical diplopia developed the next day. Walking became difficult. The diplopia disappeared when either eye was closed. He had previously been healthy, and general physical examination was normal.

Ophthalmic Examination

Visual acuity was 1.0 in both eyes. Both eyes were orthotropic in the primary position. Elevation and depression of the left eye were restricted, with limitation of elevation being more pronounced. The corneas and pupils were normal bilaterally. The optic discs and maculae were normal. Forced-duction testing of the left inferior rectus was positive.

Laboratory Findings

Routine blood and urine tests: Negative / no significant abnormality. Liver and renal function: Negative / no significant abnormality.

Initial Diagnosis

Primary diagnosis: Left orbital floor blowout fracture.

Secondary diagnosis: Left traumatic paralytic hypotropia.

Basis for Diagnosis

Primary diagnosis: Typical trauma history and clinical findings, including diplopia and restricted vertical ocular movement, especially elevation. Resistance on forced-duction testing of the inferior rectus indicates restriction of the inferior rectus. The diagnosis is confirmed by CT.

Secondary diagnosis: Clear history of trauma, vertical diplopia, especially marked limitation of upward movement of the left eye, and restriction of the inferior rectus on forced-duction testing.

Differential Diagnosis

Extraocular muscle palsy: Orbital floor fracture may be mistaken for superior or inferior rectus

palsy, while medial orbital wall fracture with limited abduction may be mistaken for lateral rectus palsy due to abducens nerve dysfunction. CT is the most definitive method of differentiation, but the forced-duction test is simpler. In an orbital blowout fracture, forced-duction testing is positive; in extraocular muscle palsy, it is negative.

Extraocular muscle rupture: The globe deviates toward the side opposite the ruptured muscle. Forced-duction testing is negative, with no resistance from the ruptured muscle. Local contusion signs such as subconjunctival hemorrhage and edema may be present. CT shows no fracture; surgical exploration can confirm the diagnosis.

Diagnostic and Treatment Plan

Complete routine systemic preoperative preparation.

Perform timely surgical repair of the left orbital floor fracture.

Case 13

Case Summary

Patient: Female, 50 years old, farmer. Right-eye proptosis for 10 years and decreased vision for 1 month.

The right eye had gradually protruded for 10 years without obvious visual decline or pain, and she had never sought medical attention. During the past month, mild distending pain and decreased vision developed in the right eye, prompting consultation. She had previously been healthy. General physical examination was normal.

Ophthalmic Examination

Visual acuity: right eye 0.08; left eye 1.0. Intraocular pressure was normal in both eyes. The eyes were orthotropic and ocular movements were normal in all directions. Exophthalmometry was recorded as 23 mm on the right and 13 mm on the left with a 100-mm base. Resistance to retropulsion was (++). No mass was palpable at the orbital rim. The anterior segment of the right eye was clear; the pupil was normal in size and both direct and consensual light reflexes were brisk. Fundus examination showed a slightly pale optic disc with somewhat indistinct margins; the posterior-pole retina was mildly wrinkled, without obvious edema, hemorrhage, or exudation. Orbital palpation, anterior segment, and fundus examination of the left eye were normal.

Laboratory and Imaging Findings

Routine blood and urine tests: negative / no significant abnormality. Liver and renal function: negative / no significant abnormality.

B-scan ultrasonography: An oval, medium-to-high intensity mass echo was detected within the right retrobulbar intraconal space, temporal to the optic nerve. It had clear margins, homogeneous internal echoes, and compressibility, measuring 27.6 x 18.86 mm. The optic nerve was compressed and displaced nasally, and the fundus echo band was flattened by compression.

CT: An oval, hyperdense soft-tissue mass was present within the right retrobulbar muscle cone. It was homogeneous, with clear and smooth margins. CT value: 56.9 HU. No abnormality of the orbital wall was seen.

MRI: A round-to-oval intraconal mass was present in the right orbit, showing long T1 and long T2 signal characteristics (low signal on T1WI and high signal on T2WI), with homogeneous signal, measuring 2.0 x 1.5 cm. The optic nerve was displaced superomedially and the extraocular muscles were displaced by compression. Dynamic contrast-enhanced scanning showed

progressive enhancement of the tumor.

Initial Diagnosis

Primary diagnosis: Right orbital mass.

Secondary diagnosis: Partial optic atrophy of the right eye.

Basis for Diagnosis

Long, chronic course without prominent accompanying symptoms. Imaging confirmed a space-occupying lesion in the right orbit with benign features.

Differential Diagnosis

Cavernous hemangioma: More common in young and middle-aged adults and in women. Unilateral, chronic, progressive proptosis is the most common sign. Severe symptoms are usually absent early, so presentation is often delayed; visual loss may occur later. The tumor is typically intraconal and often cannot be palpated, with only increased resistance to retropulsion. Ultrasonography characteristically shows homogeneous medium-to-high echoes and compressibility. CT and MRI show a round or oval, homogeneous, well-circumscribed, smooth intraconal mass with marked enhancement; MRI demonstrates progressive enhancement. Based on the history, signs, and imaging features, this case is consistent with an orbital cavernous hemangioma.

Schwannoma: Can occur at any age, although most patients presenting for care are adults. Unless located at the orbital apex and causing early visual decline, it usually presents with chronic progressive proptosis, with visual loss appearing after the tumor reaches a certain size. Clinical manifestations are nonspecific. The tumor may lie within the muscle cone or to one side of the globe. In addition to proptosis, globe displacement and deep orbital pain are common. Ultrasonography may show a round or oval hypoechoic area with clear margins, low internal echoes, and frequent cystic spaces. CT and MRI often show a well-defined tumor on one side of the orbit or within the muscle cone, sometimes with a "tail-tip" appearance; the interior may be heterogeneous with cystic changes. The history, signs, and imaging in this case are less suggestive of schwannoma, but postoperative pathology is required for confirmation.

Diagnostic and Treatment Plan

Surgical excision of the mass followed by pathological examination.

Case 14

Case Summary

Patient: Male, 35 years old. Chief complaint: Decreased vision in the left eye for 5 months. History of hypertension for 2 years.

Ophthalmic Examination

Visual acuity as recorded in the source: right eye 0.04 #1.5; left eye 0.02 #0.5. Intraocular pressure was normal in both eyes. Anterior segments were unremarkable bilaterally; KPs were absent and aqueous flare was absent. The right fundus had a tessellated appearance with no other abnormality. The left fundus showed extensive retinal hemorrhage at the posterior pole and macular edema.

Laboratory and Ancillary Findings

Routine blood and urine tests: negative / no significant abnormality. Liver and renal function: negative / no significant abnormality.

FFA: Scattered hypofluorescent patches were present on the retina. Small peripheral vessels were dilated and filled, and a nonperfusion area was visible in the inferonasal periphery. In the late phase, fluorescence staining was present at the posterior pole.

Initial Diagnosis

Primary diagnosis: Central retinal vein occlusion of the left eye.

Secondary diagnoses: Refractive error in both eyes; hypertension.

Basis for Diagnosis

Adult male with unilateral, relatively mild visual reduction and no other ocular discomfort. Fundus findings included acceptable optic disc color and margins, macular edema, dilated and tortuous veins, and extensive retinal hemorrhages along the four major venous branches. Fluorescein angiography showed mild fluorescein leakage from venous walls and capillary dilation.

Differential Diagnosis

Diabetic retinopathy: Usually bilateral. Retinal veins may be dilated and tortuous but generally less severely; hemorrhages are scattered and fewer. Blood glucose is elevated and systemic manifestations may be present. Patients with diabetes are also prone to retinal vein occlusion.

Hypertensive retinopathy: Usually symmetric in both eyes. Retinal hemorrhages are superficial and sparse, mostly at the posterior pole. Veins may be dilated but are not markedly tortuous and dark. Cotton-wool spots and star-shaped macular exudates are common.

Diagnostic and Treatment Plan

Monitor blood pressure and intraocular pressure. Control blood pressure and lower intraocular pressure.

Check blood lipids and reduce blood viscosity.

Antiplatelet therapy: aspirin and dipyridamole.

Traditional Chinese medicines for promoting blood circulation and resolving stasis, as recorded in the source: intended to dilate vessels, inhibit platelet aggregation, and improve microcirculation.

Iodide preparations to promote absorption of hemorrhage.

Hyperbaric oxygen for cystoid macular edema.

Laser treatment to photocoagulate nonperfusion areas and neovascularization, prevent vitreous hemorrhage, and reduce cystoid macular edema.

Case 15

Case Summary

Patient: Male, 44 years old. Chief complaint: A dark shadow obstructing vision in the right eye for 1 month.

Visual acuity: right eye 0.5; left eye 0.9. The anterior segments were unremarkable bilaterally; KPs

and aqueous flare were absent. The left fundus was normal. The right fundus showed a disc-shaped detachment of the retinal neurosensory layer at the posterior pole; the remainder of the retina was unremarkable. The patient had previously been healthy, and systemic examination showed no abnormality.

Laboratory and Ancillary Findings

Routine blood and urine tests: negative / no significant abnormality. Liver and renal function: negative / no significant abnormality.

FFA: A hyperfluorescent point approximately 1.5 disc diameters superotemporal to the foveal center showed progressively enlarging leakage over time. In the late phase, a hyperfluorescent spot was present in the macular area with surrounding pooling of fluorescence.

Initial Diagnosis

Primary diagnosis: Central serous chorioretinopathy of the right eye.

Secondary diagnosis: None.

Basis for Diagnosis

Fundus examination showed a disc-shaped neurosensory retinal detachment at the posterior pole with no other retinal abnormality. FFA showed a pinpoint hyperfluorescent focus approximately 1.5 disc diameters superotemporal to the foveal center, with progressive leakage and enlargement over time; in the late phase, a hyperfluorescent macular spot with surrounding pooling was present.

Differential Diagnosis

The following conditions can produce serous detachment of the sensory retina in the macular region:

Central exudative choroidal lesion and other disorders with macular choroidal neovascularization: More common in young and middle-aged women, with gray-yellow macular exudative lesions accompanied by hemorrhage. FFA shows leakage from the neovascular membrane in the early arterial phase.

Age-related macular degeneration (ARMD): Usually occurs in patients older than 50 years. Drusen and retinal pigment epithelial changes may be present, as may choroidal (subretinal) neovascular membranes. The disease is often bilateral.

Macular detachment caused by rhegmatogenous retinal detachment, especially inferior detachment: A retinal break can be identified.

Choroidal tumor: A mass can be seen with indirect ophthalmoscopy and may cause secondary serous macular detachment. FFA can help establish the diagnosis.

Retinal pigment epithelial detachment (RPED): The margin of RPED is sharper than that of CSCR. Occasionally, RPED may coexist with this disease or with ARMD.

Optic disc pit: A small defect or depression in the neural tissue of the optic disc. It may cause a serous retinal detachment connected to the optic disc.

Diagnostic and Treatment Plan

Most patients can recover visual acuity spontaneously to above 0.7 and have a very good prognosis. A small number of recurrent, multifocal, or prolonged cases have a poorer prognosis.

Examine the macula after dilation using a three-mirror lens, contact/noncontact fundus lens (60D or 90D), and indirect ophthalmoscopy to exclude associated choroidal neovascularization. Also examine the optic disc for an optic disc pit and exclude choroidal

tumor or rhegmatogenous retinal detachment. If the diagnosis is uncertain or atypical, if choroidal neovascularization is suspected, or if laser treatment is planned, FFA may be considered. OCT is useful for diagnosis and for determining the extent of the lesion.

Advise the patient to stop smoking and drinking alcohol, rest adequately, and maintain good sleep.

For most patients, review every 6-8 weeks, monitoring visual acuity and fundus changes until spontaneous resolution.

Laser photocoagulation may be considered if: (1) serous detachment persists for more than 4-6 months; (2) recurrence causes persistent reduction of vision in the affected eye due to the original lesion; (3) after persistent visual decline in the affected eye from the original lesion, the fellow eye also becomes affected; or (4) the patient insists on rapid visual improvement, for example because of occupational requirements.

Glucocorticoids are contraindicated in this disease.

Case 16

Case Summary

Patient: Male, 62 years old.

Chief complaint: Recurrent episodes for more than one month of distending pain in the left eye accompanied by headache, marked loss of vision, nausea, and vomiting.

Present illness: One month earlier, the patient developed gradually worsening distending pain in the left eye with ipsilateral migraine-like headache and a sharp decrease in vision. He was diagnosed at an outpatient clinic with "acute angle-closure glaucoma" and treated with systemic and topical antiglaucoma medications. Because the intraocular pressure remained elevated, anterior chamber paracentesis was performed. Acetazolamide and topical pressure-lowering medications were continued after the procedure, and the intraocular pressure was temporarily controlled. One day before admission, the same symptoms recurred and worsened and did not respond to medication.

Past history: No history of glaucoma or other ocular disease and no family history of glaucoma.

General examination: Blood pressure 140/80 mmHg. General condition was satisfactory. Cardiopulmonary examination was unremarkable. The abdomen was soft and flat without masses or tenderness; the liver and spleen were not palpable. The extremities and spine were normal.

Ophthalmic Examination

Visual acuity: Right eye 1.0; left eye 0.1. Intraocular pressure was normal in the right eye and 59 mmHg in the left eye.

Right eye: No conjunctival injection; clear cornea. Central anterior chamber depth was approximately 4 corneal thicknesses (CT), and peripheral depth approximately 1/2 CT. Iris pattern was normal. The pupil was round, 2 mm in diameter, with a light response. Fundus examination was normal.

Left eye: Mixed bulbar conjunctival injection and hazy corneal opacity. Fine brown KPs (+) and aqueous flare (+). The anterior chamber was shallow, approximately 1 CT centrally, with no peripheral anterior chamber. Iris markings remained fairly clear, with no atrophic patches or iridodonesis. The pupil was 3 mm and reacted sluggishly to light. The lens remained clear. Fundus examination showed an optic disc of normal color with clear margins and a physiologic cup of 0.4; no abnormality was seen in the posterior pole.

Gonioscopy: Right anterior chamber angle was narrow grade I-II; normal angle structures could not be seen in the left eye. Visual fields were normal in both eyes.

Course After Admission

Medical therapy was first used to lower intraocular pressure. Oral glycerol, intravenous mannitol, oral acetazolamide, and topical pilocarpine and timolol reduced the left intraocular pressure to approximately 30-40 mmHg after intravenous mannitol, and visual acuity improved to 0.8. However, when the medication effect wore off, intraocular pressure again rose to 50-60 mmHg. After eight days without significant improvement, left trabeculectomy with peripheral iridectomy was performed.

On the first postoperative day, slit-lamp examination showed a diffuse filtering bleb and clear cornea. The anterior chamber was grade 3 shallow, with the cornea completely apposed to the peripheral iris and lens; only slit-like anterior chamber spaces remained at the pupillary margin and around the minor iris circle. After full dilation, the inferior lens equator from 3 to 7 o'clock was exposed, and a shallow peripheral anterior chamber formed inferiorly. Digital intraocular pressure was T-1.

On further questioning, the patient reported that more than ten days earlier the left eye had been struck by a "flying disc," causing tearing, ocular pain, redness, and blurred vision, which gradually improved after more than ten days.

Laboratory Findings

Complete blood count, urinalysis, and liver and renal function: Negative / no significant abnormality.

Diagnosis

Primary diagnosis: Acute angle-closure glaucoma of the left eye, probably secondary.

Secondary diagnoses: Traumatic lens dislocation/subluxation with secondary glaucoma (with a pre-existing primary anatomic factor); blunt ocular trauma.

Basis for Diagnosis

The history of a glaucoma attack together with markedly elevated intraocular pressure, hazy cornea, ciliary injection, and sharp visual loss supports the diagnosis of glaucoma.

The typical triad of primary acute angle-closure glaucoma was absent, and anterior chamber depth was asymmetric between the two eyes, which did not support a purely primary glaucoma diagnosis.

Postoperative dilation revealed lens subluxation, and further history disclosed ocular trauma, supporting the diagnosis.

Differential Diagnosis

During treatment, the condition failed to improve when managed as primary acute angle-closure glaucoma. Intraocular pressure was controlled after surgery, and postoperative dilation revealed lens subluxation causing anterior chamber shallowing. The source therefore attributes the condition to primary acute angle-closure glaucoma combined with secondary traumatic glaucoma due to lens subluxation.

Acute attack of chronic angle-closure glaucoma: There was no previous history or prodrome of glaucoma attacks and no fundus, visual-field, or anterior chamber angle changes typical of chronic angle-closure glaucoma.

Simple primary acute angle-closure glaucoma: The right anterior chamber angle was narrow grade I-II, indicating a primary anatomic predisposition, but the traumatic lens abnormality

supports a secondary component.

Case 17

Case Summary

Patient: Female, 15 years old.

Chief complaint: Decreased vision, pain, and redness of the left eye for one month, worsening over the last three days.

Present illness: One month earlier, the left eye became red and painful with decreased vision. At another hospital, visual acuity was recorded as 0.1, Jr 1. Slit-lamp examination showed "punctate and linear epithelial and subepithelial infiltrates in the inferonasal paracentral cornea with fluorescein staining." Dendritic keratitis was diagnosed, and topical acyclovir, cyclosporine, and tobramycin were given, without obvious improvement. Two weeks later, symptoms worsened and vision decreased further. Examination showed enlargement of the corneal lesion, linear infiltrates in the nasal subepithelium and superficial stroma running radially, patchy stromal infiltrates nasally and inferiorly, gray-white KPs, and aqueous flare (++) . In addition to the topical drugs, subconjunctival tobramycin and cytarabine and intravenous ciprofloxacin were administered, but the condition did not improve. Three days before presentation, severe left-eye pain and a sharp decrease in vision developed.

Past history: Myopia of -5.00 D in both eyes. The patient had continuously worn orthokeratology (OK) lenses for one year until the onset of disease. No previous ocular disease.

General physical examination: Normal.

Ophthalmic Examination

Visual acuity: Right eye 0.2, Jr 1; left eye, hand movements, unable to see Jr 7. The left eyelid was hyperemic and swollen. Ocular irritation symptoms were (++) , with marked mixed conjunctival injection. The cornea was edematous, with a central 8 x 8 mm ring-shaped infiltrate and ulcer. Anterior chamber depth was normal, with a 2 mm hypopyon inferiorly. The remaining structures could not be clearly visualized. Digital intraocular pressure was T+1.

Laboratory and Ancillary Findings

Routine blood and urine tests, liver function, and renal function were normal.

Corneal ulcer scraping: Cytologic examination for pathogenic microorganisms. Bacterial culture and antimicrobial susceptibility testing.

Diagnosis

Primary diagnosis: Acanthamoeba keratitis of the left eye.

Secondary diagnoses: Hypopyon of the left eye; secondary glaucoma; refractive error in both eyes.

Basis for Diagnosis

Primary diagnosis: One-year history of OK-lens wear. Early ocular findings included epithelial and subepithelial infiltration, pseudodendritic keratitis, and radial keratoneuritis. Late findings included the typical ring-shaped corneal infiltrate and ulcer of Acanthamoeba keratitis. Cytologic examination of corneal ulcer scrapings may show Acanthamoeba cysts and trophozoites, and Acanthamoeba culture may be positive.

Secondary diagnoses: Hypopyon was present on examination, digital intraocular pressure was T+1, and there was a history of myopia.

Differential Diagnosis

Herpes simplex viral keratitis: Early Acanthamoeba keratitis is easily misdiagnosed as herpes simplex keratitis. The amoebic dendritiform lesion is described as a slightly elevated linear opacity, unlike the grooved opacity with terminal bulbs in herpes simplex keratitis; hence it is termed pseudodendritic keratitis. Acanthamoeba infection may involve corneal nerves, causing radial keratoneuritis and severe ocular pain. A history of corneal trauma or contact-lens wear is common, antiviral therapy is ineffective, and the disease may progress to a typical stromal ring infiltrate. Herpes simplex keratitis more often has recurrent episodes and responds to antiviral therapy; if untreated or unresponsive, it may progress to a geographic corneal ulcer.

Bacterial keratitis: Acute onset, severe ocular irritation, abundant discharge, and early corneal infiltration and ulceration. Antibiotic therapy is effective.

Fungal keratitis: Ocular irritation is relatively mild. The corneal ulcer is irregular with a comparatively dry surface, and pseudopods or satellite lesions may occur at the lesion margin.

Diagnostic and Treatment Plan

Corneal ulcer scraping for cytology and identification of pathogens. Giemsa or PAS staining of scrapings may demonstrate Acanthamoeba cysts.

Acanthamoeba culture to further establish the diagnosis, using a non-nutrient medium seeded with Escherichia coli.

Bacterial culture and antimicrobial susceptibility testing because Acanthamoeba keratitis may be accompanied by bacterial infection.

Confocal microscopy may reveal reflective oval bodies within the cornea and can aid diagnosis; a negative result does not completely exclude the disease.

Medical treatment: The source states that effective anti-Acanthamoeba drugs were limited domestically and recommends combined fluconazole, metronidazole, and chlorhexidine eye drops. Nonsteroidal anti-inflammatory drugs may be used for severe pain. The patient should stop wearing OK lenses and use a mydriatic.

Complete preoperative evaluation and plan penetrating keratoplasty. Because this is a late-stage lesion with secondary glaucoma, the source recommends penetrating keratoplasty, followed postoperatively by 0.02% chlorhexidine and 0.5% fluconazole for anti-Acanthamoeba treatment.

Case 18

Case Summary

Patient: Male, 6 years old.

Chief complaint: Inability to raise both upper eyelids since childhood.

Present illness: The child did not open his eyes during the first week after birth. The parents later noticed that both upper eyelids could open only a narrow slit. He habitually tilted his head backward to look at people. At age 3, he was examined at a local hospital and advised to undergo surgery later. At age 4, cycloplegic refraction at another hospital showed no abnormality, but visual acuity could not be tested. At age 5, visual acuity was 1.0 in both eyes at another hospital.

Past history: The mother's pregnancy and delivery were unremarkable. Growth and development of other organs were normal. No other systemic disease. Vaccinations were received on schedule.

Family history: The mother had the same ocular appearance. Information about other family members was unavailable. General physical examination was normal.

Ophthalmic Examination

Uncorrected visual acuity: Right eye 1.2; left eye 1.0. Both eyebrows were elevated. Horizontal palpebral fissure length was 17 mm bilaterally. Complete bilateral upper-eyelid ptosis covered three quarters of the cornea. Levator function was 0 in both eyes. Bilateral inverse epicanthus and a wide inner canthal distance of 39 mm were present.

There was no ocular injection. The corneas and anterior chambers were clear. The pupils were equal, 3 mm in diameter, and reacted briskly to light. The lenses were clear. Undilated fundus examination showed normal optic discs with normal color and clear margins; macular and vascular findings were recorded as negative. Ocular alignment was orthotropic. Eye movements were unrestricted in all directions. Bell's phenomenon was positive bilaterally, and there was no lagophthalmos.

Laboratory Findings

Complete blood count, urinalysis, liver function, renal function, and blood glucose: Negative / no significant abnormality.

Diagnosis

Primary diagnosis: Bilateral blepharophimosis syndrome (congenital blepharophimosis syndrome).

Secondary diagnosis: None specified in the source.

Basis for Diagnosis

Bilateral short palpebral fissures.

Complete upper-eyelid ptosis.

Bilateral inverse epicanthus.

Increased intercanthal distance.

Positive family history.

Differential Diagnosis

Bilateral congenital ptosis: Apart from upper-eyelid ptosis, there are no other eyelid abnormalities.

Myasthenia gravis: Ptosis is milder in the morning, worsens in the afternoon and after activity, and is an acquired disorder.

Blepharophimosis syndrome: Characterized by ptosis, short palpebral fissures, and inverse epicanthus.

Diagnostic and Treatment Plan

Surgical correction of ptosis, epicanthus, and blepharophimosis. Procedures listed in the source include frontalis suspension, levator shortening and advancement, medial canthal Z-plasty, and medial canthal Y-V correction.

Case 19

Case Summary

Patient: Male, 32 years old.

Chief complaint: Diplopia after a head injury in a traffic accident three months ago.

Present illness: Three months ago, the patient sustained a head injury in a traffic accident and was unconscious for 20 minutes. After regaining consciousness, he noticed diplopia and inability to abduct the right eye. Symptoms improved slightly after medical treatment, but diplopia persisted. The separation of the double images decreased on left gaze and increased on right gaze. Since onset, the right eye has frequently been red. Previous CT examinations of the head and orbit showed normal intracranial structures and no orbital fracture.

Past history: Unremarkable. General examination: Blood pressure 120/70 mmHg; otherwise normal.

Ophthalmic Examination

Visual acuity: 1.0 in both eyes; Jr1 bilaterally. The left external eye was normal. The right eye showed incomplete eyelid closure with 2-3 mm lagophthalmos. The right nasolabial fold was shallow.

Ocular alignment and motility: With the right eye fixing, the left eye showed 30° esotropia; with the left eye fixing, the right eye showed 15° esotropia. Adduction was normal bilaterally. On attempted right-eye abduction, the temporal corneal limbus remained 6-7 mm from the lateral canthus. Left-eye abduction was normal. Vertical movements were normal in both eyes.

The right conjunctiva was mildly injected. The inferior cornea showed a band-like superficial stromal opacity with mild edema; fluorescein staining was negative. The left cornea and anterior chamber were clear. Both lenses were clear. Optic discs and maculae were normal bilaterally.

Diplopia testing: A red lens was placed before the right eye.

Laboratory Findings

Routine blood and urine tests, liver and renal function, and blood glucose: Negative / no significant abnormality.

Diagnosis

Primary diagnosis: Right lateral rectus palsy with paralytic esotropia (right abducens nerve palsy).

Secondary diagnoses: Right facial nerve palsy with incomplete eyelid closure; exposure keratitis of the right eye.

Basis for Diagnosis

Primary diagnosis: Right esotropia with impaired abduction and a secondary deviation greater than the primary deviation. Diplopia is uncrossed and the separation increases in the direction of action of the right lateral rectus. There is also a history of head trauma.

Secondary diagnosis: Mild right conjunctival injection, an inferior band-like superficial stromal corneal opacity, mild edema, and negative fluorescein staining.

Differential Diagnosis

Comitant esotropia: Usually begins in childhood. The angle of deviation is stable, the primary and secondary deviations are equal, ocular movements are unrestricted in all directions, and diplopia is usually absent.

Restrictive strabismus caused by an orbital wall fracture: A medial orbital wall fracture may cause esotropia and limited abduction, with the secondary deviation greater than the primary deviation. Forced-duction testing is positive, and CT shows a medial orbital wall fracture with entrapment of orbital contents and the medial rectus. In this case, CT already showed no orbital wall fracture, so forced-duction testing was considered unnecessary.

Diagnostic and Treatment Plan

For cranial nerve palsy caused by craniocerebral trauma, provide neuroprotective and neurotrophic treatment to promote recovery of lateral rectus function. The source lists vitamins B1 and B12, inosine, ATP, vasodilators, and nerve growth factor.

Neurology consultation to exclude injury to other cranial nerves.

Protect the cornea with artificial tears, antibiotic eye drops, agents promoting corneal epithelial growth, and ophthalmic ointment at night.

If diplopia is severe and interferes with daily life, occlusion of one eye may be used.

If the angle of strabismus does not decrease during observation, botulinum toxin type A may be injected into the medial rectus to reduce the deviation and relieve medial rectus spasm.

If strabismus persists after 6-8 months, surgical treatment may be considered.

Case 20

Case Summary

Patient: Female, 62 years old.

Chief complaint: Blurred vision in the right eye for nearly one week.

Present illness: One week earlier, the patient suddenly developed painless blurred vision. One year earlier, she had experienced a sudden painless decrease in vision in the left eye, with no significant improvement after treatment.

Past history: Hypertension for 15 years, with a maximum blood pressure of 180/110 mmHg, controlled with medication. Diabetes mellitus for 3 years, treated medically.

General examination: Blood pressure 160/100 mmHg. General condition was satisfactory. Cardiopulmonary examination was unremarkable. The liver and spleen were not palpable, and the abdomen had no mass, tenderness, or shifting dullness.

Ophthalmic Examination

Visual acuity: Right eye 0.2; left eye 0.1; unable to see Jr7 in either eye. Intraocular pressure: 17.30 mmHg in both eyes. No eyelid edema or conjunctival injection was present. Corneas and anterior chambers were clear. KPs and flare were absent, no iris neovascularization was seen, and mild cortical lens opacity was present bilaterally.

Fundus: In the right eye, the optic disc margin was indistinct, especially superiorly; the disc was slightly pale and mildly elevated. In the left eye, the optic disc margin was clear and the disc was pale. Retinal arteries were narrow with increased light reflex bilaterally. The posterior poles showed hemorrhages of varying sizes, microaneurysms, and one or two ring-shaped exudates. No neovascular membrane was seen.

Laboratory Findings

Complete blood count, urinalysis, and liver function: Negative / no significant abnormality. Blood glucose: 8 (unit not specified in the source).

Diagnosis

Primary diagnosis: Ischemic optic neuropathy of the right eye.

Secondary diagnoses: 1) Left optic atrophy, most likely due to old ischemic optic neuropathy; 2) bilateral diabetic retinopathy, stage II; 3) bilateral senile cataract, incipient stage.

Basis for Diagnosis

Primary diagnosis: Blurred vision in the right eye for nearly one week; history of hypertension and diabetes; indistinct right optic disc margin, especially superiorly, with slight pallor and mild elevation; visual field showing an inferior arcuate scotoma connected with the physiologic blind spot.

Secondary diagnoses: The left optic disc has clear margins and pallor, with an inferior visual-field defect including the physiologic blind spot, supporting old ischemic optic neuropathy with optic atrophy. Diabetic retinopathy is supported by diabetes and bilateral posterior-pole hemorrhages, microaneurysms, and exudates. Incipient senile cataract is supported by mild bilateral lens opacity.

Differential Diagnosis

Optic papillitis: Usually occurs at a younger age, with very poor vision, marked optic disc edema, prominent peripapillary hemorrhage and exudation, and a central scotoma.

Foster-Kennedy syndrome: Marked optic disc edema; visual field shows enlargement of the physiologic blind spot on the side with disc edema and a central scotoma on the side with optic atrophy; symptoms of raised intracranial pressure are present.

Normal-tension glaucoma: Slow onset, optic disc cupping, and concentric visual-field constriction.

Papilledema due to intracranial hypertension: Marked optic disc elevation with extremely blurred margins; visual field shows enlargement of the physiologic blind spot.

Hypertensive retinopathy: Associated with hypertension, usually more marked disc edema, preceding arteriosclerotic changes, and fewer microaneurysms.

Diagnostic and Treatment Plan

Visual-field examination.

FFA to evaluate optic disc filling, retinal nonperfusion areas, and stage diabetic retinopathy.

Systemic evaluation to exclude an intracranial space-occupying lesion, particularly an inferior frontal lobe tumor.

Treatment with vasodilators and neurotrophic/supportive agents.

Actively identify and treat the underlying cause, including treatment of hypertension and control of blood glucose.

Use corticosteroids cautiously when clinically appropriate, as stated in the source.

Case 21

Case Summary

Patient: Male, 56 years old.

Chief complaint: Sudden decrease in vision in the right eye for 10 days.

Examination: Right visual acuity 0.02. Vitreous hemorrhage and opacity were present, and the

retinal structure could not be visualized.

Additional History Required

Ask about a history of hypertension, diabetes mellitus, ocular trauma, and high myopia.

Additional Ophthalmic Examinations

Measure intraocular pressure, examine the fundus of the fellow eye, and perform B-scan ultrasonography.

Initial Diagnosis

Primary/secondary diagnosis as recorded in the source: Vitreous hemorrhage and opacity; fundus condition to be determined.

Basis for Diagnosis

The source provides only the sudden severe visual loss and examination showing vitreous hemorrhage/opacity with inability to visualize the retina; a more specific underlying diagnosis requires further history and examination.

Differential Diagnosis

Diabetic retinopathy: Supported by elevated blood glucose and bilateral retinal changes.

Central retinal vein occlusion: Often associated with hypertension; the fellow eye may show hypertensive/arteriosclerotic fundus changes.

Retinal tear: Consider age and refractive status; B-scan ultrasonography may demonstrate posterior vitreous detachment.

Age-related macular degeneration: The fellow eye may show drusen and other degenerative fundus changes.

Diagnostic and Treatment Plan

Rest with the head elevated.

Treat the underlying systemic disease.

Use medication intended to promote absorption of the hemorrhage, as recorded in the source.

Follow the absorption of vitreous hemorrhage, monitor intraocular pressure, and use B-scan ultrasonography to assess the retina.

If the hemorrhage does not clear after 2-3 months, or if ghost-cell glaucoma or retinal detachment develops, consider vitreoretinal surgery.

Case 22

Case Summary

Patient: Female, 60 years old.

Chief complaint: Tearing from both eyes for more than ten years.

Present illness: Bilateral tearing for 15 years, gradually worsening.

Ophthalmic Examination

Visual acuity: Right eye 0.6; left eye 0.7. Mild entropion of the right upper eyelid was present, with the central eyelashes touching the cornea. Both conjunctivae showed a few papillae and follicles with linear scarring. Mild bulbar conjunctival injection was present bilaterally. A small amount of discharge was present in the right conjunctival sac. The superficial stroma of the superior central right cornea was opaque. Both lenses showed peripheral wedge-shaped opacities. Fundus examination showed narrowed retinal arteries bilaterally with an arteriovenous ratio of 1:2; the optic discs and maculae were normal.

Clinical Diagnosis

Primary diagnosis: Bilateral lacrimal drainage obstruction; right dacryocystitis suspected.

Secondary diagnoses: Bilateral trachoma (stage II); right upper-lid entropion with trichiasis; right corneal nebular/macular opacity; bilateral senile cataract (incipient stage); bilateral arteriosclerotic fundus changes.

Additional Ophthalmic Examinations

Lacrimal irrigation or compression of the lacrimal sac region; dacryocystography with iodized oil; corneal fluorescein staining.

Lacrimal irrigation results: In the right eye, irrigation through the lower punctum refluxed through the upper punctum with a large amount of purulent discharge. In the left eye, irrigation through the upper punctum refluxed through the lower punctum and vice versa; the passage remained obstructed even under pressure, without discharge.

Final diagnosis based on irrigation: Right dacryocystitis and left lacrimal drainage obstruction.

Treatment Plan

Lacrimal irrigation and anti-inflammatory treatment.

Plan right dacryocystorhinostomy (or dacryocystectomy as recorded in the source). Preoperative assessment should include blood pressure measurement, internal-medicine consultation, otolaryngology consultation, routine blood and urine tests, coagulation studies, and other necessary preoperative tests.

Elective surgical correction of right upper-lid entropion and trichiasis.

Case 23

Case Summary

Patient: Female, 65 years old.

Chief complaint: Decreased vision and metamorphopsia in the right eye for two months, worsening over the last seven days.

Present illness: Two months earlier, the patient developed decreased vision and visual distortion in the right eye. At another hospital, corrected visual acuity was 0.2. She was treated with cataract eye drops without significant improvement. One week ago, vision deteriorated further. Fundus visualization was poor, and B-scan ultrasonography showed vitreous opacity.

Past history: High myopia of -8.00 D in both eyes. No previous ocular disease. General physical examination was normal.

Ophthalmic Examination

Visual acuity: Right eye, hand movements and unable to see Jr7; left eye 0.05, corrected to 0.2 with -8.00 D, Jr2. There was no eyelid injection or swelling and no conjunctival injection. The corneas were clear, anterior chamber depth was normal, and the lenses were mildly opaque. Intraocular pressure was normal.

Fundus findings were documented in the source fundus image.

Laboratory Findings

Routine blood and urine tests and liver and renal function were normal.

Diagnosis

Primary diagnosis: Bilateral age-related macular degeneration (wet in the right eye and dry in the left eye).

Secondary diagnoses: Subretinal and vitreous hemorrhage in the right eye; bilateral senile cataract (incipient stage); bilateral refractive error/high myopia.

Basis for Diagnosis

History: Decreased vision and metamorphopsia in the right eye for two months, worsening over seven days.

Fundus findings described in the source: In the right eye, subretinal and vitreous hemorrhage extended from the posterior pole to the temporal mid-periphery, with an orange-yellow subretinal lesion in the macular area accompanied by yellow spots. In the left eye, the macular area showed retinal pigment epithelium (RPE) disturbance with focal yellow patches and yellow spots. The patient also had a history of -8.00 D myopia.

Differential Diagnosis

Choroidal melanoma with secondary hemorrhage: In an elderly patient, a brown-black spherical subretinal lesion may extend from the posterior pole to the temporal mid-periphery. FFA may show hypofluorescence with a double-circulation sign. Ultrasonography may demonstrate a solid choroidal mass with internal vessels and possible choroidal excavation.

Vitreous hemorrhage caused by posterior vitreous detachment: Posterior vitreous detachment and a retinal tear may be present.

Vitreous hemorrhage caused by retinal vascular disease: Consider diabetes, vasculitis, retinal vein occlusion, and related disorders.

Diagnostic and Treatment Plan

Further examinations: Fundus fluorescein angiography (FFA) and ultrasonography.

The source states that effective medical treatment was limited; because hemorrhage was extensive, medication could be used temporarily.

If the hemorrhage is absorbed and choroidal neovascularization (CNV) becomes visible, PDT/TTT may be performed.

If blood entering the vitreous cavity does not absorb, complete the preoperative evaluation and perform vitreoretinal surgery.

Case 24

Case Summary

Patient: Female, 72 years old.

Chief complaint: Left-eye proptosis for one year and diplopia for three months.

Present illness: The left eye gradually became proptotic one year earlier without redness, pain, or blurred vision. Three months ago, diplopia developed and progressively worsened, requiring occlusion of one eye for daily activities. Diplopia increased on upgaze, and the left eye could not elevate. Symptoms worsened over the past month, with tearing and photophobia.

Past history: Thyroid cancer was surgically removed 29 years earlier. A nodular adenoma was found five years ago. The patient had lost weight over the past six months. Hyperthyroidism was diagnosed three months earlier together with diplopia and was currently controlled with antithyroid medication. She denied hypertension, diabetes, and ocular or head trauma.

Ophthalmic Examination

Visual acuity: Right eye 0.7; left eye 0.4. Bilateral proptosis was recorded, with exophthalmometry values of approximately 18 mm in the right eye and 21 mm in the left eye at a 92-mm base. Both eyelids were mildly swollen, with 1–2 mm upper-lid retraction and lid lag, greater on the right.

Ocular alignment and motility: With the right eye fixing, the left eye had approximately 10° esotropia and 20° hypotropia. Adduction and abduction of the left eye were mildly restricted. Elevation in adduction and abduction was markedly restricted, and the eye could not elevate above the horizontal midline. Depression was normal. Right-eye movements were normal.

The corneas and anterior chambers were clear, anterior chamber depth was moderate, KPs and flare were absent, and the lenses showed nonuniform opacity. Undilated fundus examination was normal, with normal optic disc color and margins and a C/D ratio of 0.3. Intraocular pressure was 17.7 mmHg in the right eye and 25.8 mmHg in the left eye.

Forced-duction test: Marked inferior restriction was present when the globe was passively pulled upward. General examination: Blood pressure 120/70 mmHg; otherwise normal.

Laboratory and Ancillary Findings

Routine blood and urine tests, liver and renal function, and blood glucose: Negative / no significant abnormality.

Thyroid tests: FT3 2.65 (reference 1.8–4.3); FT4 0.88 (reference 0.74–1.9); TSH 6.4 (reference 0.2–6.2); TGAbs 216 (++) ; TPOAb 719 (++) . Current treatment: methimazole (Thyrozol) 5 mg once daily.

UBM: Normal.

Diurnal intraocular-pressure curve (mmHg): 08:00—right 17.3, left 17.3; 11:00—right 14.57, left 15.88; 14:00—right 12.23, left 14.57; 16:00—right 9.36, left 12.23. Fluctuation: right 7.94, left 5.07. The source records the result as $\pm$ for the right eye and negative for the left eye.

Synoptophore: Primary position +2 R/L 13°. Nine diagnostic gaze positions were also recorded in the source. Diplopia testing demonstrated gaze-dependent image separation.

Imaging: Cranial CT was normal. Orbital MRI showed thickening of both medial rectus and inferior rectus muscles and the left lateral rectus, more pronounced on the left. Central and peripheral visual fields were normal bilaterally.

Diagnosis

Primary diagnosis: Bilateral thyroid-associated ophthalmopathy (Graves' ophthalmopathy).

Secondary diagnoses: 1) Left glaucoma to be excluded; 2) bilateral senile cataract (incipient stage).

Basis for Diagnosis

Primary diagnosis: One-year history of left proptosis and three-month history of diplopia, with a history of hyperthyroidism under treatment. The left eye showed hypotropia, restricted elevation, and mild horizontal movement restriction. Diplopia and ocular-motility testing demonstrated extraocular muscle dysfunction. Imaging showed thickening of the bilateral medial and inferior rectus muscles and the left lateral rectus.

Secondary diagnoses: The initial left intraocular pressure was elevated, but anterior chamber depth was normal, the fundus showed no glaucomatous changes, and visual fields were normal. Observation of the diurnal intraocular-pressure curve was considered to exclude glaucoma in the source. Mild bilateral lens opacity supported incipient senile cataract.

Differential Diagnosis

Oculomotor nerve palsy: May occur in older adults and usually produces exotropia with hypertropia or hypotropia; isolated depression may also occur with paralysis of both elevators. The affected eye may have limited elevation and ptosis. Proptosis is generally absent, the motility disorder is paralytic, and forced-duction testing is negative. In this case there were no other neurologic symptoms, cranial CT was normal, the motility disorder was restrictive, forced-duction testing was positive, and proptosis was present.

Isolated superior rectus palsy: The affected eye is hypotropic, elevation in abduction is limited, elevation in adduction and horizontal movements are normal, diplopia separation is greatest in the superotemporal field of the affected eye, and forced-duction testing is negative.

Orbital floor fracture: May cause hypotropia and limited elevation as a restrictive strabismus. There is usually a history of trauma, and orbital CT demonstrates a wall fracture. These findings were absent here.

Fixed strabismus: Usually congenital, generally without diplopia, and may show limited elevation with a positive forced-duction test. The clinical history in this case is inconsistent with it.

Myasthenia gravis: An acquired disorder that may affect multiple extraocular muscles bilaterally, with movement limitation and fatigability and sometimes generalized weakness. Forced-duction testing is negative.

Diagnostic and Treatment Plan

Orbital imaging to exclude an orbital space-occupying lesion or orbital inflammatory disease and to evaluate thyroid-associated ophthalmopathy.

Cranial imaging to exclude intracranial disease.

Thyroid-function testing to assess thyroid status and guide treatment.

Neurology consultation to exclude neurologic disease.

Visual-field testing to exclude early glaucoma and assess for optic neuropathy caused by thyroid-associated ophthalmopathy.

Internal-medicine/endocrinology consultation to formulate systemic treatment.

Treatment Recommendations

Formulate systemic treatment according to the internal-medicine consultation, treat hyperthyroidism, and maintain therapy after thyroid function normalizes.

Depending on the patient's systemic condition, consider systemic or local corticosteroids or immunosuppressants. The source notes potential adverse effects including hypertension, elevated blood glucose, and infection.

The source states that retrobulbar corticosteroid injection is not used for proptosis; intraocular pressure should be monitored.

For diplopia, occlusion of one eye may reduce symptoms. Strabismus surgery should be delayed until orbital inflammation has subsided; otherwise progression, undercorrection, or new strabismus may occur.

Case 25

Case Summary

Patient: Male infant, 6 months old. He was brought to the clinic because his parents noticed a white pupil in the right eye.

He was born at 29 weeks of gestation. History of oxygen therapy was unknown. Other history was unremarkable.

Ophthalmic Examination

External examination was normal in both eyes. The right eye had approximately 15 degrees of esotropia, with unrestricted movement in all directions. Both corneas were clear, anterior chambers were moderately deep, pupils were round with positive light reflexes, and both lenses were clear. Dilated fundus examination showed dilated retinal vessels at the posterior pole of the right eye. Temporal retinal vessels coursed straight toward the periphery. Retinal neovascularization and a gray-white fibroproliferative membrane were present in the far temporal periphery, with tractional retinal detachment around the membrane; the macula was not involved. In the left eye, posterior-pole vessels were mildly tortuous and dilated but followed a generally normal course. At the temporal equator, the retina formed an arcuate ridge; the retina anterior to the ridge was avascular, with no surrounding retinal hemorrhage or neovascularization.

Diagnosis

Primary diagnosis: Bilateral retinopathy of prematurity (ROP): stage 4A in the right eye and stage 2+ in the left eye.

Secondary diagnosis: Sensory (deprivation) esotropia of the right eye.

Basis for Diagnosis

Premature birth and characteristic fundus findings.

Differential Diagnosis / History to Clarify

Prematurity and history of oxygen therapy - retinopathy of prematurity.

Family history of tumors - retinoblastoma.

Maternal infection during pregnancy, especially rubella - congenital cataract.

Birth trauma - traumatic cataract or vitreous disease.

Postnatal fever or infection - endogenous endophthalmitis.

Diagnostic and Treatment Plan

Perform ocular B-scan ultrasonography to exclude an intraocular mass.

Treatment principle: Vitreoretinal surgery may be performed in the right eye; observe the left eye.

Case 26

Case Summary

Patient: Male, 20 years old.

Chief complaint: Left eye struck by a fragment while hammering a bearing two days ago, with redness, pain, and worsening visual loss for one day.

Present illness: Two days earlier, while working, he felt a foreign object strike the left eye. He occasionally noticed small black floating spots but did not seek care because vision remained relatively good. This morning, the left eye became red and painful and vision decreased sharply. He then presented for evaluation. He had previously been healthy, and general physical examination was unremarkable.

Ophthalmic Examination

Visual acuity: Right eye 1.0; left eye, counting fingers in front of the eye. Intraocular pressure was normal in the right eye and T-1 in the left eye. The right eye was normal. The left eye showed mixed conjunctival injection and corneal edema and opacity. At the 9 o'clock position there was a well-closed 1 mm full-thickness corneal wound with a corresponding iris perforation. The anterior chamber contained flocculent inflammatory exudate, KPs (++), aqueous flare (++), and a linear hypopyon. The pupil was 3 mm and fixed. Slit-lamp examination showed gray-white opacity in the vitreous behind the lens. The fundus could not be visualized.

Laboratory Findings

WBC: 12,000. Urinalysis: negative / no significant abnormality.

Diagnosis

Primary diagnoses: Intraocular foreign body in the left eye (possibly magnetic); endophthalmitis.

Secondary diagnoses: Penetrating corneal injury of the left eye; traumatic cataract.

Basis for Diagnosis

Primary diagnosis: Definite trauma history, a penetrating tract through the cornea and iris, inflammatory anterior-chamber exudation, gray-white vitreous opacity, and low intraocular pressure. Secondary diagnoses are supported by the corneal wound and cataract confirmed after dilation.

Differential Diagnosis

Iridocyclitis: Usually not accompanied by severe vitreous opacity and lacks a trauma history.

Vitreous hemorrhage: May occur with older age, systemic disease, or trauma; in the acute stage the vitreous opacity is red, and the anterior chamber does not show inflammatory exudation.

Diagnostic and Treatment Plan

Use X-ray and B-scan ultrasonography to confirm the presence, nature, and location of the foreign

body and to assess vitreous opacity.

Remove the foreign body as early as possible. Obtain intraocular specimens for microbiologic examination, administer intravitreal medication, and perform vitrectomy as indicated. After surgery, use systemic antibiotics and corticosteroids as recorded in the source.

Case 27

Case Summary

Patient: Female, 65 years old.

Chief complaint: Dryness and foreign-body sensation in both eyes for one year, accompanied by mild visual decline, worsening over the past three months.

Present illness: For one year, the patient frequently experienced bilateral dryness and foreign-body sensation without obvious discharge, together with mild visual decline. She had previously been diagnosed with chronic conjunctivitis in both eyes and treated with antibiotic eye drops. The drops temporarily relieved the dryness and foreign-body sensation, but symptoms gradually worsened during the past three months. Past history was unremarkable. General examination was normal.

Ophthalmic Examination

Visual acuity: Right eye 0.6 and left eye 0.5; manifest refraction improved vision to 0.7 in the right eye and 0.6 in the left eye. Eyelids were normal bilaterally. Both conjunctivae were mildly injected and the tear menisci were narrow. Scattered punctate fluorescein staining of the corneal epithelium was present in both eyes. KPs and flare were absent. Both lenses showed partial wedge-shaped opacities. Intraocular pressure: right 15 mmHg, left 17 mmHg. Schirmer I test: right 3 mm/5 min, left 2 mm/5 min. Tear-film breakup time (BUT): right 5 seconds, left 3 seconds. Fundus examination was normal bilaterally.

Diagnosis

Primary diagnosis: Dry eye disease in both eyes.

Secondary diagnosis: Bilateral senile cataract.

Basis for Diagnosis

Dry eye disease: Subjective symptoms of ocular dryness and foreign-body sensation; reduced tear secretion on Schirmer I testing (source normal value >10 mm/5 min); unstable tear film with shortened BUT (source normal value >10 seconds); and ocular-surface damage demonstrated by punctate corneal epithelial staining.

Senile cataract: Age 65 years, decreased vision, and partial wedge-shaped lens opacities on examination.

Treatment Plan

Dry eye disease: Use tear substitutes such as artificial tears. Tear conservation can be achieved with punctal or canalicular plugs, particularly when frequent instillation is required. Anti-inflammatory and immunosuppressive therapy may be considered because ocular-surface immune inflammation is associated with dry eye; the source lists 0.05% cyclosporine A and short-term low-concentration corticosteroids, while warning that corticosteroids should not be used long term because of adverse effects.

Senile cataract: Perform manifest refraction and prescribe spectacles if vision improves. Because current visual acuity remains approximately 0.5-0.6, surgery may be deferred for now.

