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Invited Review

A Glossary for ''Pseudo'' Conditions in Ophthalmology

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Abstract

The term ''pseudo'' refers to ''lying, false, fake, simulation, imitation or spurious.'' In ophthalmological literature, there are many diseases/conditions/signs/phenomena that are considered as ''pseudo.'' A literature search was conducted on the Medical Subject Headings website, and the keywords that were searched in the title and abstract were as follows: (pseudo-), (fake), (false), (mimicker), (simulator), (masquerade), AND (condition) AND(causes) AND (ophthalmology) OR (eye) OR (ocular) OR (ophthalmic) OR (cornea) OR (retina) OR (strabismus) OR (glaucoma). The search was restricted to English language. The major databases such as PubMed, Medline, Scopus, Google Scholar, OVID, EBSCO, and Cochrane Library were searched or investigated for information. The objective of this review is to summarize common ''pseudo'' conditions in ophthalmology and their respective common causes. We believe that the knowledge of these pseudo-conditions will provide significant benefits in the differential diagnosis of various ophthalmic disorders.

Keywords: Condition, fake, false, mimicker, masquerade, ophthalmology, pseudo, simulator.

Introduction

The term ''pseudo'' is a prefix that is derived from the word ''pseudes'' in Greek language. It means ''lying, false, fake, simulation, imitation or spurious'' (1, 2). In the search of databases, such as PubMed or Google Scholar, there is no article on pseudo-conditions found in ophthalmology that is published in a scientific journal. On this topic, only a slide presentation was detected in a web search (3). The literature search was conducted on Medical Subject Headings website and restricted to only English language. The keywords that were searched in the title and abstract included the following terms: (pseudo-), (fake), (false), (mimicker), (simulator), (maquerade), AND (condition) AND (causes) AND (ophthalmology) OR (eye) OR (ocular) OR (ophthalmic) OR(cornea) OR (retina) OR (strabismus) OR (glaucoma). Major databases such as PubMed, Medline, Scopus, Google Scholar, OVID, EBSCO, and Cochrane Library were searched for the abovementioned information. Here, the objective of this review is to summarize common ''pseudo'' conditions or phenomena that are mentioned or present in the ophthalmological literature, their respective common causes, and their distinguishing features from true ones in an alphabetical order.

Pseudo-Abducens palsy/Pseudo-Sixth cranial nerve palsy/Pseudo-Abduction deficit (thalamic esotropia) is a neurologic restriction in abduction with an intact abducens nerve. It can be manifested during voluntary eye movements with the impairment of lateral gaze and full abduction in the vestibular–ocular reflex (VOR) testing, the lack of ipsilateral esotropia in the primary gaze, and adduction nystagmus of the contralateral eye if the weakly abducting eye is used for fixation. The intact VOR shows the integrity of the infra-nuclear abducens nerve. Pseudo-abducens palsy is likely to be caused by supranuclear or thalamic pathology and does not present

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with typical infra-nuclear abducens palsy findings. The main causes of this pathology are myasthenia gravis, thyroid eye disease, Duane's retraction syndrome, medial orbital wall fracture, longstanding esotropia, and convergence spasm. It can be distinguished from a true abduction deficit via doll's head maneuver or by patching one eye for a short time (4-9).

Pseudo-Accommodation is defined as an increased depth-of-focus in the pseudo-phakic eye. It occurs due to the static optical properties, such as pupil size, astigmatism, and wavefront aberrations of cornea and the intraocular lens (IOL) that do not depend on ciliary muscle actions, of the pseudophakic eye. It is different from pseudophakic accommodation, which is the dynamic change in the refractive state of the eye because of the forward movement of the IOL–bag complex (10,11).

Pseudo-Argyll Robertson pupil is an abnormal pupillary sign that is characterized by a normal near reflex but with the absence of a light reflex (light-near dissociation), absence of miosis, and presence of pupillary irregularity. Argyll Robertson pupil is a highly specific sign of neurosyphilis that is defined by the emergence of bilateral pupils, which are small and show a poorly constructive response to light beside a light-near dissociation. Its common causes include the aberrant regeneration of third cranial nerve palsy following acute traumatic and compressive but not vascular events, diabetes mellitus, multiple sclerosis, Wernicke's encephalopathy, neurosarcoidosis, tumor, hemorrhage, and spinocerebellar ataxia type 1 (12,13).

Pseudo-Cataract is a clinical entity similar to cataract that manifests from the delivery of drug particles to the posterior lenticular surface following the intravitreal injection of triamcinolone (14).

In *Pseudo-Chalazion*, the clinical entities mimic chalazion and include a neurogenic tumor of the eyelid (neurilemmoma). It is a tumor that emerges in the meibomian glands of the eyelid (sebaceous carcinoma) and is type of metastatic tumor in the eyelid (15-17).

Pseudo-Convergence Insufficiency is a condition in which there is a reduced point of convergence (NPC) and near exophoria. The important characteristics, such as improved NPC and reduction in near exophoria with the use of low-plus lenses, differentiate *pseudo-convergence insufficiency* from convergence insufficiency (18, 19).

Pseudo-Cystoid macular edema (non-angiographic, non-leaking cystoid macular edema) is probably caused by the accumulation of intracellular fluid (intracellular edema), but not by extracellular as developed in the true retinal edema, along with toxicity in the Müller cells and subclinical extracellular leakage. It may occur in conditions such as X-linked foveoschisis, myopic foveal schisis, Goldman Favre disease, the pseudo-hole with an epiretinal membrane, nicotinic acid maculopathy, some forms of retinitis pigmentosa, vitreomacular traction syndrome, and hydroxychloroquine and taxane maculopathy (20-23).

Pseudo-Dendrite is a misnomer. It means ''true dendrites'' excluding those observed in typically and classically in herpetic keratitis. It is defined as raised, branching epithelial lesions or corneal ulceration with/or without associated punctate epithelial staining. The main causes of pseudo-dendrites are *acanthamoeba* keratitis, corneal neurotrophic epitheliopathy, herpes/*varicella-zoster* keratitis, healing of a corneal epithelial defect or corneal abrasion, epithelial rejection in a corneal graft, contact lens wear, and toxic keratopathy secondary to topical medication (keratoconjunctivitis medicamentosa). Other causes include recurrent corneal erosion syndrome (stromal dystrophy or epithelial basement membrane degeneration), tyrosinemia type II, and meibomian gland disease (24-27).

Pseudo-Divergence excess is defined as the occurrence of near exodeviation that increases up to 10 PD of distance deviation after prolonged monocular occlusion (the presence of a larger exotropia) caused by an increased tonic fusional convergence. The distance angle initially appears to be larger than the near angle, but the deviation for near exodeviation distance is similar when the near angle is remeasured with the patient looking through +3.00 D lenses or after 30–60 minutes of monocular occlusion (28, 29).

Pseudo-Drusen Reticular is a clinical phenotype of drusenoid deposits that are located between the sensorial retina and retinal pigment epithelium (RPE) (the subretinal space and above the level of the RPE), unlike drusen. They are strongly associated with late age-related macular degeneration (AMD), particularly geographical atrophy, type 2 and 3 choroidal neovascularization. Reticular pseudo-drusen (RPD) may also be observed in Sorsby's fundus dystrophy, pseudo-xanthoma elasticum, and acquired vitelliform lesions. RPD is associated with an increased age and poor prognosis in AMD. RPD may be characterized as pale, irregular sinusoidal, or annular single yellow lesions that more commonly manifest in yellowish-white net-like patterns (reticular network pattern) because of the lobular anatomy of the choroid in color fundus imaging as compared to soft drusen. True drusen, an established marker for AMD, are the concentrated deposits of extracellular material found around the macula. It has been demonstrated that RPD is associated with a loss of choroidal small vessels and an increase in the spaces between large choroidal veins not typical of AMD-associated drusen. RPD is usually observed at the superotemporal quadrant of the macula except for fovea, whereas soft drusen are often localized in the area of fovea. RPD is generally not accompanied by changes in RPE, and a fairly uniform pattern appears over the large retinal areas. RPD

was not associated with the deposition of basal laminar or basal linear deposits. RPD can best be distinguished from standard drusen via optical coherence tomography (OCT). Tget appear as granular hyperreflective deposits located between the RPE layer and the ellipsoid zone on the OCT scans. However, cuticular drusen and soft drusen present as round punctate accumulations under RPE (30-34).

Pseudo-Duane's Retraction Syndrome (DRS) is defined as the presence of some amount of abduction. The globe retraction and the narrowing of the palpebral fissure in the affected eye occurs during abduction. True DRS is characterized by the deficiency of abduction in the affected eye, globe retraction with adduction, and narrowing of palpebral fissure on the attempted abduction (35-37).

Pseudo-Duplication of the optic disc is defined as a normal optic disc and a disc-like lesion with vascularity and chorioretinal atrophy adjacent to the normal optic disc. The causes of the view of the doubled optic disc are optic disc coloboma, peripapillary chorioretinal coloboma, and scarring (38-42).

Pseudo-Endophthalmitis is a clinical entity that simulates the manifestation of endophthalmitis after the intravitreal injection of triamcinolone acetonide and it often resolves without specific treatment (43).

Pseudo-Enophthalmos may occur due to microphthalmia or phthisic globe, upper eyelid ptosis and/or lower eyelid reverse ptosis, and proptosis or pseudo-proptosis in the opposite eye, superior sulcus volume loss, or elevated eyelid crease. Enophthalmos is a relative posterior displacement of a normal-sized eye that concerns the bony orbital margin (44).

Pseudo-Epithelium cornea is defined as the multilayering of corneal endothelium that underlies the thickened Descemet's membrane. Its typical sample is posterior polymorphous dystrophy characterized by vesicles along with geographical or band-like opacities on Descemet's membrane (''tram-tracks'') (3-6).

Pseudo-Epitheliomatous hyperplasia is benign epithelial hyperplasia in conjunctiva or cornea. It may develop as a response to various inflammatory conditions such as vernal keratoconjunctivitis (45,46).

Pseudo-Esotropia is characterized by the false appearance of esotropia in the alignment of visual axes. It is the most common type of pseudo-strabismus and may be caused by myopia (due to negative angle kappa), a flat and broad nasal bridge, prominent epicanthal folds, or a narrow interpupillary distance that causes the observer to see less sclera nasally than expected (3-6,47,48).

Pseudo-Exfoliation is a systemic syndrome whose ocular manifestations are characterized by the deposition of whitish-gray fibrilo-granular protein on the lens capsule, iris, ciliary body epithelium, corneal endothelium, zonules, and trabecular meshwork. It is not true exfoliation on the lenticular capsules. True exfoliation is defined as the splitting of superficial zonular layer from the deeper layer because of high heat resulting from glassblowing or infrared radiation exposure of the anterior lenticular capsule (49,50).

Pseudo-Exotropia is most commonly caused by hypertelorism or abnormal interpupillary distance, temporal dragging of the maculadue to the retinopathy of prematurity, ectopic macula due to high myopia, congenital Toxocara scar or folds in the retina, or positive angle kappa. Angle kappa is the angle between the visual and anatomical axes. Normally, the fovea is located temporally toward the anatomical center of the posterior pole. A positive angle kappa defines a nasal reflex to the center of both corneas because of the slightly abduction of the eyes to obtain a bifoveal fixation (3-6,51).

Pseudo-Foster Kennedy Syndrome is defined as the presence of optic atrophy or pallor in one eye and optic disc edema in the other eye in the absence of an intracranial mass. The literature has reported the cases of benign intracranial hypertension (pseudo-tumor cerebri), ischemic optic neuropathies, optic nerve hypoplasia, and diabetic papillopathy. In contrast, true Foster Kennedy syndrome defines the presence of optic disc edema in one eye and optic atrophy in the other eye due to compressive optic neuropathy and increasing intracranial pressure typically arising from an olfactory groove meningioma and frontal lobe tumor (52,53).

Pseudo-Fovea is defined as an area other than the true fovea centralis in the retina that is used for fixation on an image. The individuals who have squint eyes since their childhood may have developed abnormal retinal correspondence or a sensory adaptation in strabismus. In these cases, the fovea is suppressed and another point (*pseudo-fovea*) in the retina close to the visual center is perceived as the visual center (54,55).

Pseudo-Fluorescence is defined as non-fluorescent–reflected light that is visible before fluorescein injection. Additionally, the blue light reflected from highly reflective fundus lesions emits yellow-green light when stimulated by blue light in the presence of mismatched filters without fluorescein. The main causes of pseudo-fluorescence are myelinated nerve fibers, white areas of the fundus, such as high myopia, hard exudates, and chorioretinalatrophy or scars (56,57).

Pseudo-Graefe's (pseudo-von Graefe, pseudo lid lag) sign is a bizarre defect in ocular motility. It is defined as the elevation of the upper eyelid on attempted adduction or depression. It can occur as the aberrant regeneration or reinnervation of the incorrect extraocular muscle by misdirected regenerating axons in third cranial nerve palsy and paramyotonia congenita. von Graefe's sign is defined as the lagging or failure of the upper eyelid on the downward rotation of the bulbus and it is a sign of Graves' disease (4,58).

Pseudo-Gerontoxon is characterized by a paralimbic band of superficial scarring that resembles arcus senilis (segmentary arcus senilis). It can manifest in recurrent or previous allergic eye diseases such as limbal, vernal (spring catarrhal), or atopic keratoconjunctivitis. Gerontoxon (arcus senilis) is commonly observed among the elderly people. It is developed by the deposition of lipids at the peripheral cornea without any pathological significance. However, it can also occur in familial hypercholesterolemia-anemias (59,60).

Pseudo-Glaucomatous cupping is the emergence of optic disc cupping in the absence of elevated IOP along with other signs of glaucomatous optic neuropathy. The most common causes are congenital cupping (physiologically large optic cup), anterior or posterior ischemic optic neuropathy, traumatic optic neuropathy, tumoral compressive optic neuropathy (due to the fusiform aneurysms of the intracranial carotid arteries or tumors compressing the anterior visual pathway), Leber's hereditary optic neuropathy, and congenital optic disc anomalies such as coloboma, pit, or hypoplasia. Additionally, cilioretinal artery occlusion is associated with central retinal vein occlusion, anterior shock optic neuropathy, syphilis, radiation optic neuropathy, and methanol poisoning. It may also be a cause of pseudo-glaucomatous cupping (61-64).

Pseudo-Guttate (secondary guttata) is a transient, reversible corneal endothelial edema that is commonly associated with anterior segment pathology. It presents as a hyporeflective elevated shape without clear borders on confocal microscopy, and as dark lesions on a slit-lamp exam with specular illumination. It resolves over time and does not involve Descemet's membrane; these characteristics differentiate pseudo-guttate from primary corneal guttata. Primary guttata (guttae or true guttata) is characterized by the outpouchings of the Descemet's membrane, whereas pseudo-guttata are transient and completely reversible areas of endothelial edema without Descemet's involvement. The conditions and surgeries associated with pseudo-guttata include all the infectious types of keratitis iritis; endotheliitis; post-surgical inflammation such as corneal conductive, laser, or incisional refractive surgery interventions; YAG laser iridotomy/capsulotomy, pterygium surgery, cataract surgery, IOL explantation/implantation, glaucoma surgery, vitreoretinal procedures and medication toxicity (fortified vancomycin, benzalkonium chloride, toxic anterior segment syndrome, miostat, mitomycin C, intravitreal injection, anti-glaucoma medications, angiotensin-converting-enzyme inhibitors); endophthalmitis; glaucoma; and blunt traumatic, thermal, chemical, UV, and infrared injuries of cornea and contact lens keratopathy (65).

Pseudo-Hole in macula mimics the clinical appearance of a macular hole. It is most commonly observed in association with the contraction of epiretinal membrane, vitreomacular traction syndromes, proliferative diabetic retinopathy, rhegmatogenous retinal detachment, intraocular inflammation, trauma, and venous occlusive disease. OCT demonstrates the steepening of foveal contour, the presence of full-thickness retinal tissue, and the reflective epiretinal membrane layer on the surface of the retina. Fluorescein angiography often reveals normal fluorescence except in the presence of traction-induced retinal vascular disruption (66, 67).

Pseudo-Hypertropia is the appearance of vertically misaligned eyes where one eye appears to be higher than the other. It can be caused by a vertically displaced macula from the retinopathy of prematurity or toxocariasis, eyelid retraction, facial asymmetry, orbital tumors, mucocele or trauma to the orbital floor via hypoglobus, or vertical and superior displacement of the globe. The light reflex test and covering test show it to be orthophoric (68).

Pseudo-Hypopyon is the appearance of similar hypopyon because of intravitreal or intracameral triamcinolone, emulsified silicone or phacolytic glaucoma (anterior pseudo-hypopyon) or retinoblastoma, leukemia, and Stage 3 best macular dystrophy (posterior pseudo-hypopyon) (69-73).

Pseudo-Inferior oblique overaction syndrome is defined as a strabismus having a Y pattern with exotropia in upgaze. There is a marked abduction and hypertropia of the adducting eye when elevation is performed in the side gaze, but there is no hypertropia of the adducting eye in the horizontal side gaze. The main theories proposed for the pathophysiology of this syndrome include an aberrant innervation between the superior and lateral rectus muscles as well as a heterotopic muscle pulley or the displacement of superior rectus muscle pulley toward the lateral rectus. In contrast, patients with true inferior oblique overaction syndrome present with the hypertropia of the adducting eye in the horizontal side gaze. Patients with pseudo-inferior oblique overaction syndrome do not respond to a surgical weakening of inferior oblique muscles (74).

Pseudo-Inflammatory (Sorsby's) macular dystrophy is a rare disease that typically occurs in late middle age and causes bilateral visual loss. Inheritance is autosomal dominant with full penetrance. Clinically, early, mid-peripheral, drusen, and color vision deficits are found in the affected patients. Some patients complain of night blindness. Most commonly, the presenting symptom is sudden acuity loss because of untreatable submacular neovascularization. Histologically, there is the accumulation of a confluent lipid-containing material having a thickness of 30 μm at the level of Bruch's membrane. The ERG is initially normal but may become subnormal in the later stage of the disease (75,76).

Pseudo-Internuclear Ophthalmoplegia (INO) is characterized by the weakness in muscles, adduction restriction, and con-

tralateral abduction nystagmus in the cases in which there is no lesion in the central nervous system. Pseudo-INO results from peripheral conduction defects or an intermittent blockage of neuromuscular conduction to the extraocular muscles. It can also be a clinical manifestation of ocular myasthenia gravis, Guillain–Barré syndrome (GBS), or the Miller–Fisher Syndrome (a variant of GBS). The extraocular muscle weakness can rarely produce a pseudo-INO. True INO is an abnormality of conjugate horizontal eye movement that is characterized by the failure of adduction in one eye and nystagmus in the abducting eye because of the damage caused often by multiple sclerosis (often bilateral) or ischemic damage or stroke (often unilateral) to the medial longitudinal fasciculus, which is a myelinated tract of fibers responsible for yoked eye movements (77-79).

Pseudo-Isochromatic color plate tests (Ishihara/Hardy Rand Rittler charts) include charts with the colored dots of various hues and shades indicating numbers, letters, or patterns. It is used for quickly and grossly testing color discrimination or acquired color loss and central visual dysfunction (80).

Pseudo-Membrane in the conjunctiva is characterized by a coagulated fibrin-rich exudate that adheres to the inflamed conjunctival epithelium without blood or lymphatic vessels. Its removal does not causes bleeding because it can be peeled away while leaving the underlying conjunctival epithelium intact. The main causes of a conjunctival pseudomembrane are adenoviral or bacterial conjunctivitis *(Streptococcus family, Corynebacterium diphtheriae)*, Stevens–Johnson syndrome, ligneous conjunctivitis, graft versus host disease, toxic exposure to the conjunctival surface. The true conjunctival membrane is formed by the coagulum of exudate over substantia propria, and its removal causes bleeding and tearing in the conjunctival surface (81,82).

Pseudo-Myopia is often caused by the spasm of the near reflex that may most frequently occur in young females. During excessive near work, there is an occurrence of a transient ciliary muscular spasm, which relaxes the zonular fibers. However, the ciliary muscle cannot relax even during the distant gaze. This functional condition causes the eye to appear to be myopic. Its main signs include miosis, diplopia, visual blurring, and headache. Ciliary spasm may be triggered during the examination of eye movements (83).

Pseudo-Ocular Pemphigoid (medication-induced pemphigoid) is clinically identical to ocular cicatricial pemphigoid (OCP) because of the long-term use of some topical medications including echothiophate iodide, pilocarpine, epinephrine, practolol, timolol, idoxuridine, D-penicillamine, and demecarium bromide. The characteristics such as the observation of its resolution following discontinuation of the relevant agent differentiate pseudo-ocular pemphigoid from OCP (84,85).

Pseudo-Operculum is a semi-translucent pre-foveal tissue resulting from spontaneous vitreo-foveal separation. It contains vitreous condensation without neurosensory retinal components. Histopathological examination suggests the presence of proliferative and reparative fibrous astrocytes and Müller cells. An early recognition of pseudo-operculum points toward the presence of underlying impending macular holes (86,87).

Pseudo-Orbital/preseptal cellulitis is a clinical entity mimicking orbital cellulitis that can occur after sub-tenon antineoplastic drug (carboplatin) injections. Diseases such as noninfectious inflammation (idiopathic orbital pseudotumor, sarcoidosis, Graves orbitopathy, Wegener's granulomatosis), ruptured dermoid cyst, rhabdomyosarcoma, lymphangioma, neuroblastoma, extrascleral spread, and necrosis of intraocular melanoma, metastatic disease to the orbit should be considered in the presence of pseudo-orbital/preseptal cellulitis (88, 89).

Pseudo-Papilledema is not a true disc swelling. Optic discs appear like swollen or raised without edema of the retinal nerve fiber layer secondary to ocular disorders such as peripapillary masses, astrocytic hamartomas, optic disc drusen, congenitally anomalous discs, hypoplastic optic discs, hypermetropia, Leber's hereditary optic neuropathy, tilted as optic disc, small optic discs (<2 mm^3), peripapillary myelinated nerve fibers and crowded optic disc in hypermetropia, epi-papillary glial tissue in Bergmeister's papillae, optic disc infiltration by neoplastic or inflammatory cells, or scleral infiltration, vitreo-papillary traction, orbital hypotelorism, and Alagille syndrome (arterio-hepatic dysplasia). The vessels surrounding the disc are not obscured, the disc is not hyperemic, and the peripapillary nerve fiber layer is normal. Spontaneous venous pulsations, if present, strongly suggest pseudo-papilledema. Nerve fiber layer hemorrhages are absent in pseudo-papilledema (90).

Pseudo-Parinaud syndrome is a clinical entity that mimics Parinaud syndrome. It is characterized by the presence of the binocular elevation palsy and bilateral eyelid retraction instead of ptosis (91).

Pseudo-Phakia occurs following the implantation of an artificial lens after the surgical extraction of crystalline lens.

Pseudo-Plasticity is defined as the ability to turn from gel to liquid or liquid-like substance under pressure. Most ocular viscoelastic devices (OVD) such as sodium hyaluronate and methylcellulose behave in that way because of their pseudoplasticity with lower viscosity at higher shear rates, whereas some OVDs such as chondroitin sulfate do not exhibit pseudo-plasticity because of their constant viscosity. Pseudoplasticity provides the substance to easily inject and remove at increasing flow rates through a small gage cannula (92).

Pseudo-Plus-Minus Lid Syndrome is characterized by unilateral ptosis accompanied by a contralateral eyelid retraction

caused by a mesencephalic infarct. The first type is associated with the lesions of the midbrain region of the nucleus of the posterior commissure extending to the third nerve fascicle on the ptotic side. No change of lid retraction when the ptotic lid is manually raised can be observed. The second type is usually observed for lesions at or distal to the neuromuscular junction and occurs when the lid retraction of one eye is relieved by manually elevating the contralateral ptotic lid. It most commonly occurs in myasthenia gravis (93).

Pseudo-Polycoria is defined as the opening of accessory pupillary membrane. Pseudo-polycoria is often associated with Seckel syndrome, iridocorneal endothelial syndrome, posterior polymorphous dystrophy, juvenile glaucoma, ectropion uveae, iris atrophy, corectopia, iris trauma, or surgery. Polycoria is a pathological condition of the eye that is characterized by more than one pupillary opening in the iris. The presence of constriction of the accessory pupil in polycoria when the true pupil is dilated assists in differentiating between pseudo-polycoria and polycoria. True pupillary retains as an intact sphincter muscle, reacts to light, and synchronously contracts and can dilate with mydriatics (94-98).

Pseudo-Presumed ocular histoplasmosis syndrome (POHS) is a condition in which there is an occurrence of chorioretinal lesions that resembles those observed in patients with presumed ocular histoplasmosis. In contrast to patients with POHS, the patients with pseudo-POHS have associated vitreous inflammation. This entity includes multifocal choroiditis with panuveitis, punctate inner choroidopathy, and ocular ischemic syndrome (ischemic pseudo-iritis) (99-103).

Pseudo-Proptosis is defined as a false proptotic view of the globe without its anterior displacement from the orbit. It may be caused by contralateral enophthalmos, contralateral ptosis, facial asymmetry, shallow orbit, ipsilateral lid retraction, or contralateral enophthalmos, ipsilateral large globe (buphthalmos/myopia). Proptosis is caused by the abnormal protrusion or displacement of the globe, ipsilateral lid retraction, contralateral enophthalmos, contralateral small eye, contralateral ptosis, facial nerve palsy, bilateral asymmetric proptosis, or latrogenic pseudo-proptosis due to the lid retraction caused phenylephrine eye drops in a single eye or oversized prosthesis. It may arise from other vascular, endocrine, inflammatory, neoplastic ocular, or orbital pathologies (104).

Pseudo-Pterygium defines a conjunctival fold that may adhere to any quadrant of the cornea of the conjunctiva to the peripheral cornea. It is often stationary. Pseudo-pterygium may result from a peripheral corneal ulcer and ocular surface inflammation such as cicatrizing conjunctivitis, chemical burns, or chronic mechanical irritation from contact lens movement with an inadequate ocular surface lubrication. Pterygium is defined as a raised triangular growth on the corneal limbus, with an apex or head located on the cornea and a degenerative condition of unknown etiology. Ptergyum growths tend to be oriented laterally in the interpalpebral fissure on either the nasal or temporal side of the cornea and adhering to the corneal epithelium. In pterygium, a hook or probe cannot pass under the neck of pterygium tissue and it can be elevated with forceps, whereas this procedure can be performed in pseudo-pterygium (105).

Pseudo-Ptosis is a condition that mimics ptosis due to abnormalities other than resulting for the upper eyelid retractor muscles. It can be caused by hypotropia on the ptotic side, contralateral exophthalmos, contralateral lid retraction, blepharospasm, brow ptosis, double elevator palsy, dermatochalasis, enophthalmos, ipsilateral hypotropia, enophthalmos/phthisis bulbi, anophthalmos/microphthalmos, and severe dermatochalasis. Ipsilateral hypotropia disappears when the hypotropic eye assumes fixation on covering the normal eye. Ptosis is defined as the drooping or falling of the upper eyelid on bulbus in various types such as myogenic, neurogenic, mechanical, and aponeurotic (involutional) mechanisms (106).

Pseudo-Retinitis Pigmentosa includes the conditions or diseases that can mimic the fundal pigmentary changes in retinitis pigmentosa. Most common causes include the presence of an intraocular foreign body; drug-induced pigmentary retinopathy due to thioridazine, chloroquine, hydroxychloroquine, quinine, and phenothiazine; infectious diseases such as toxoplasmosis, rubella, measles, syphilis, borreliosis; ocular inflammation such as optic disc vasculitis, chronic uveitis, and Vogt–Koyanagi–Harada syndrome; scars from chronic central serous chorioretinopathy, laser photocoagulation, old or treated retinal detachment, trauma, cancer-associated retinopathy, central retinal artery occlusion; and ophthalmic artery occlusion (107).

Pseudo-Retinoblastoma includes clinically similar lesions to retinoblastoma. It has been reported that approximately half of all patients referred to an ocular oncology center with the diagnosis of possible retinoblastoma had pseudo-retinoblastoma. Its common causes are Coats' disease and persistent fetal vasculature or persistent hyperplastic primary vitreous, retrolental fibroplasia, or retinopathy of prematurity and posterior cataracts. However, ocular toxocariasis, familial exudative vitreoretinopathy, fundus coloboma, or unattached retina may cause pseudo-retinoblastoma (108-109).

Pseudo-Rubeosis iridis (RI) is known as the occurrence of iris neovascularization due to the view of actually dilated or tortured normal iridial vessels. Abnormal iris vessels are very common in Fuch's uveitis. In RI, pathological iris neovascularization can occur in chronic inflammatory eye diseases, central retinal vein occlusion, posterior uveitis with retinal disperfusion, diabetic retinopathy, and neovascular glaucoma. Normal iris vessels course radially in contrast to the irreg-

ular distribution of neovascularization. Fluorescein angiography reveals the leakage from iris vessels in RI. Pseudo-RI does not show any extravasation or leakage of fluorescein. However, the leakage can also be rarely observed especially awith pseudo-RI in the eyes with active inflammation (110).

Pseudo-Trichiasis is caused by involutional entropion or longstanding entropion (3-6).

Pseudo-Tumor orbit is also known as an idiopathic orbital inflammatory syndrome that is characterized by a nonspecific idiopathic inflammatory, non-neoplastic, non-infective, space-occupying, and infiltrative disease of any or whole orbital soft tissues (muscle, the lacrimal gland, or sclera). Its clinical findings include eyelid erythema or edema, palpable mass, decreased vision, conjunctival hyperemia or edema, uveitis, hyperopic shift, and optic nerve edema. The imaging procedures may show the thickening of one or more extraocular muscles, such as the tendons, enlargement of lacrimal gland, or thickening of the posterior sclera. It simulates a tumor but gets resolved spontaneously (111,112).

Pseudo-Uveal Melanoma is defined as the conditions that simulate choroidal melanoma. Its most common causes are choroidal nevus, peripheral exudative hemorrhagic chorioretinopathy, congenital hypertrophy of the RPE, hemorrhagic RPE detachment, choroidal hemangioma, vaso-proliferative tumors of the retina, AMD, RPE hyperplasia, and any pathology that may cause choroidal hemorrhage (113-116).

Pseudo-Uveitis is a type of ocular masquerade syndrome. It can be caused by malignant conditions such as primary lymphoma in the central nervous system, intraocular lymphoma, leukemia, and also non-malignant conditions such as retained intraocular foreign body, rhegmatogenous retinal detachment, myopic degeneration, pigment dispersion syndrome, ocular ischemic syndrome, infectious intraocular inflammation, retinitis pigmentosa, multiple sclerosis, and drug and post-vaccination reactions (117-119).

Disclosures

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