Review Article

Neuro-Ophthalmologic Disorders That Mimic Glaucoma: A Review

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Abstract

Background: To summarize the neuro-ophthalmologic disorders that mimic glaucoma.
Material and Methods: All studies from PubMed, Scopus, and Google Scholar were found using these keywords: optic atrophy, neuro-ophthalmologic disorders, glaucoma mimicker, glaucoma masquerader, and mimic glaucoma.
Material and Method: All studies from PubMed, Scopus, and Google Scholar were found using the following keywords: optic atrophy, neuroophthalmologic disorders, glaucoma mimicker ,glaucoma masquerader, and mimic glaucoma .We obtained the patients' consent for this study.
Results: The main neuro-ophthalmologic disorders that mimic glaucoma are large physiologic cupping, optic disc cup asymmetry, hereditary optic neuropathy, tilted disc syndrome, morning glory anomaly, superior segmental optic nerve hypoplasia, optic disc coloboma, optic disc pits, optic disc drusen, high myopia, inflammatory conditions (optic neuritis), toxic-nutritional optic neuropathy, nutritional deficiency, toxins, traumatic optic neuropathy, compressive optic neuropathy, periventricular leukomalacia, hydrocephalus, perinatal asphyxia, ischemic optic neuropathy, and radiation optic neuropathy.
Conclusion: Repeated evaluations of visual functions such as visual acuity, visual field, and color vision are necessary for diagnosing glaucoma mimickers. Furthermore, a precise examination of the optic disc and retina is crucial. Notably, it is necessary to consider normal-tension glaucoma as an exclusionary diagnosis.

Keywords: Optic Neuropathy; Glaucoma; Optic Nerve.

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Introduction

Glaucoma is a potentially progressive, irreversible, and blinding optic neuropathy characterized by RNF defects and corresponding visual field changes in the presence or absence of elevated IOP [1]. Non-glaucomatous optic nerve cupping may be a normal variation or a congenital optic disc anomaly. However, it can also occur due to hereditary, inflammatory, nutritional, toxic, compressive, or ischemic optic neuropathies and retinal or CNS disorders [2]. Distinguishing glaucomatous from non-glaucomatous neuropathy can be challenging in clinical practice, even for professionals. Up to 20 % of non-glaucomatous cupping cases have been misdiagnosed and treated as glaucoma[2]. When the IOP is elevated, diagnosing and treating glaucoma is relatively simple. The difficulty arises in patients with normal-tension glaucoma. Misdiagnosis can result in ineffective diagnosis and treatment, which has a detrimental effect on the patient's overall health and psychological well-being. It may also cause significant financial consequences [3].

Material and Method

All studies from PubMed, Scopus, and Google Scholar were found using the following keywords: optic atrophy, neuro-ophthalmologic disorders, glaucoma mimicker, glaucoma masquerader, and mimic glaucoma. We obtained the patients' consent for this study.

Results

The main neuro-ophthalmologic disorders that mimic glaucoma include normal conditions such as large physiologic cupping and optic disc cup asymmetry. They also cover hereditary optic neuropathy, such as autosomal dominant optic atrophy or Leber hereditary optic neuropathy. The mentioned disorders also include optic disc anomalies, for example, tilted disc syndrome, superior segmental optic nerve hypoplasia (topless disc syndrome), morning glory anomaly, optic disc coloboma, optic disc pits, optic disc drusen, high myopia, inflammatory conditions (optic neuritis), toxic-nutritional optic neuropathy, nutritional deficiency, toxins, traumatic optic neuropathy, compressive optic neuropathy, periventricular leukomalacia, hydrocephalus, perinatal asphyxia (hypoxic ischemic encephalopathy), ischemic optic neuropathy (specifically the arteritic type), and radiation optic neuropathy.

The various types of Glaucoma mimickers

Large physiologic cupping

A large physiologic cup is frequently associated with a large disc, even without typical glaucoma signs, such as focal thinning or notching, RNFL defects, beta zone peripapillary atrophy, disc hemorrhage, or VF defect. As a result, clinicians must employ the optic disc size measurement technique during a slit-lamp examination or note the optic disc surface area on the OCT printout. In this situation, a normal visual field, NFL thickness, and attention to the large disc size help distinguish this normal entity from glaucoma [4, 5].

Optic disc cup asymmetry

CD ratio asymmetry can occur in normal eyes due to asymmetric optic disc size without characteristic glaucoma signs. In this situation, the normal visual field, NFL thickness, and the care of the other disc size help distinguish this normal entity from glaucoma [6].

Hereditary optic neuropathy

Genetic testing can aid in diagnosing hereditary optic neuropathies, which can manifest as cupping. Autosomal dominant optic atrophy and Leber hereditary optic neuropathy are

the two most common hereditary optic neuropathies that mimic glaucoma [3, 7].

Autosomal dominant optic atrophy (ADOA)

Autosomal dominant optic atrophy (Kjer's optic atrophy) with a gradual and primarily accidental vision loss (20/40 to 20/60) tends to produce a moderate to the severe temporal excavated pallor of the disc. In this case, the rim is not as pink as in glaucoma. Bilateral central or cecocentral scotoma is visible in the visual field. The severity of vision loss typically peaks in adolescence and remains stable for many years in these patients. Loss of RNFL in the papillomacular (temporal) region, in contrast to a superior and inferior defect in glaucoma, and pallor of the neuroretinal rim, contrary to the pink appearance of the rim in glaucoma, can be used to distinguish these two abnormalities [8].

Leber hereditary optic neuropathy

Leber hereditary optic neuropathy is a mitochondrially inherited disease characterized by sequential acute or subacute vision loss (less than 0.1) in both eyes. This disease progresses to bilateral diffuse cupping, disc pallor, central and arcuate VF defects, and poor recovery. A history of acute blurred vision in both eyes sequentially and a more prominent pallor on the cup can help differentiate this inherited disease from glaucoma [3].

Tilted disc syndrome (TDS)

TDS (Fuch's Coloboma) is a rare congenital unilateral or bilateral disc anomaly with a 0.4–3.5 % prevalence. It is characterized by an inferonasal tilt of the optic disc. Due to incomplete closure of the embryonic fissure, the optic nerve appears to enter the eye at an acute oblique angle rather than perpendicularly. Clinically, the superotemporal part of the disc appears to be elevated, while the inferonasal part appears depressed. TDS is also associated with high myopia; approximately one-fifth of patients with myopia, greater than five diopters, have tilted discs.

Non-progressive visual field defects, which are uncommon in glaucoma (such as the bitemporal superior visual field defect), can aid in diagnosis [7].

Superior segmental optic nerve hypoplasia (topless disc syndrome)

It is a non-progressive congenital anomaly affecting the optic nerve head, with hypoplasia generally confined to the superior disc. Significant findings in these patients include superior disc pallor, superior RNFL thinning, localized inferior visual field defect, localized inferior visual field defect, the relative superior entrance of the central retinal artery, and maternal history of type 1 diabetes. The congenital nature of this problem, the superior location of the disc's vascular exit, and the stable local defect in the superior disc can aid in diagnosing this glaucoma mimicker [7].

Morning glory anomaly

Morning glory anomaly is a unilaterally enlarged disc excavation with an abnormal retinal vascular pattern, annular peripapillary chorioretinal atrophy and pigmentation, and central glial tuft. It is caused by embryonic developmental abnormalities of the lamina cribrosa and posterior sclera.

Visual acuity is frequently inadequate. Moreover, afferent pupillary and non-progressive visual field defects are frequently present. Anomalies of the face such as hypertelorism, cleft lip, or palate.

Encephaloceles, corpus callosum agenesis, and endocrine abnormalities are all examples of central nervous system (CNS) defects.

The vascular pattern, poor visual acuity, and persistent problems all help distinguish this congenital condition from glaucoma [9].

Optic disc coloboma

It is defined as a congenital condition caused by incomplete closure of the embryonic fissure that is sporadic or autosomal dominant, unilateral or bilateral (half of the cases). This anomaly exhibits defects in the inferior retinal nerve fiber layer and superior visual field. Optic discs that are enlarged, sharply circumscribed, bowl-shaped, glistening white, and deeply excavated are most frequently found inferiorly or inferonasal of the disc that mimics glaucoma. Occasionally, however, the retina, sclera, lens, ciliary body, and choroid may be colobomatous concurrently, helping in diagnosis.

The blood vessels appear to originate entirely on the periphery of the excavation.

Progressive vision loss and VF defects can occur due to fluid leakage into the sub-macular area, which can be misinterpreted as progressive glaucoma. The blood vessels appear to originate completely on the periphery of the excavation. A sizeable optic disc coloboma results in a superior field defect resembling arcuate glaucoma. However, the unique pattern of disc vessels can aid in accurate diagnosis [7, 10].

Optic disc pits

A congenital optic disc pit is a round or oval, gray or yellowish depression located in the temporal sector of the optic disc. It is commonly unilateral and located in the temporal sector of the optic disc. In 85 % of cases, the optic nerve with the pit is larger than without the pit. Optic pits are herniations of the dysplastic retina into a collagen-lined pocket that extends posteriorly with varying depths through a developmental defect in the lamina cribrosa near the subarachnoid space (SAS). SAS is anterior to the pia mater. OCT depicts the entire length of pits from their opening to their bottom. Although arcuate scotoma is the most common visual field defect, almost any visual field defect can occur because of nerve fiber displacement. Certain optic disc pits are associated with serous retinal detachment maculopathy, resulting in decreased visual acuity and central or cecocentral defects, particularly in the third and fourth decades of life [7, 10]. The pattern produced by enhanced depth optical coherence tomography can assist in distinguishing these two entities.

Optic disc drusen

Optic disc drusen may be associated with NFL and visual field abnormalities such as nasal depression and arcuate scotoma with or without AION. Without excavation and true cupping, sectoral or diffuse disc pallor develops over time. The B scan, autofluorescence, and EDI OCT are all sensitive methods for detecting disc drusen [11].

High Myopia

Myopia has a large disc and a shallow cup with a thin neuroretinal rim. The discs may be tilted (typically temporally) and exhibit peripapillary atrophy, making them glaucoma suspects. Certain field defects, such as arcuate or paracentral defects caused by myopic chorioretinal scars and arcuate scotomas caused by tilted discs, are present in high myopes. With RNFL abnormalities on OCT, follow-up of patients can rule out a progressive glaucoma pattern [7].

Inflammatory conditions (optic neuritis)

Although optic disc pallor is the most

frequently observed old change in the optic nerve head following optic neuritis, a larger cup is expected in the affected eye compared to the non-affected eye. While acute optic neuritis is easily distinguished from glaucoma, the diagnostic dilemma occurs with optic atrophy and cupping in advanced stages.

The critical clues for differentiation are a detailed history and, most likely, a brain MRI and increased thinning of the peripapillary NFL temporal area compared to the inferior and superior glaucoma [11, 12].

Toxic-nutritional optic neuropathy

Toxic and nutritional optic neuropathies are both acquired mitochondrial optic neuropathies with overlapping genetic susceptibility and toxic environmental influences.

The characteristic pattern of toxic-nutritional optic neuropathy is painless, progressive, bilateral, and dose and duration-dependent visual loss. It also covers decreased contrast perception, dyschromatopsia (particularly faded red color), sluggish pupils, and symmetrically central or cecocentral scotoma with negative RAPD.

In most cases, the disc exhibits a typical pattern during the acute phase because the primary lesion is not localized to the optic nerve and may originate from the chiasm or optic tracts. However, disc edema or hemorrhage may occur during certain acute phases, such as those associated with methanol or amiodarone toxicity.

A detailed history of possible toxic exposure and dyschromatopsia not seen in glaucoma is required to differentiate between the two entities [13, 14].

Nutritional deficiency

The predominant cause of nutritional optic neuropathy is a deficiency of B-complex vitamins (particularly B1 and B12) or folic acid, which happens most frequently in alcoholics and smokers. The most common treatment is intensive vitamin use with variable results [11].

Toxins

Methanol toxicity causes flashing, scotomas and scintillations, and occasionally basal ganglia degeneration. Other toxins with an adverse effect on the optic nerve are tuberculostatics (ethambutol and isoniazid), antibiotics (linezolid, ciprofloxacin, dapsone, and chloramphenicol), antiepileptics (vigabatrin: bilateral field constriction (tunnel fields) with preservation of central vision due to retinal and optic nerve toxicities), antiarrhythmic drugs (amiodarone), antimetabolites (methotrexate, cisplatin, carboplatin, vincristine, and cyclosporin), metals (lead, mercury, and thallium), solvents (ethylene glycol, and toluene), tamoxifen, sildenafil, TNF-alpha antagonists, disulfiram fumes, carbon monoxide, and potentially tobacco [14, 15]. Vitamin B or folate deficiency can amplify the effects of these toxins [14].

Traumatic optic neuropathy

Patients with traumatic optic neuropathy usually show optic atrophy three to six weeks after the initial trauma. They gradually increase optic disc cupping and, therefore, mimic glaucomatous cupping and arcuate defects in their visual fields that mimic glaucoma. However, after a sharp eye or head injury, acute onset of vision loss, decreased color vision, and a relative afferent pupillary defect can help differentiate the two entities [15].

Compressive optic neuropathy

Compressive anterior visual pathway lesions frequently mimic glaucomatous disc neuropathy, making it difficult to distinguish

from glaucoma.

Optic disc changes are more frequently observed in chiasmal and pre-chiasmal tumors. However, posterior lesions cause more visual field defects than optic disc anomalies, which in glaucomatous optic neuropathy typically respect the vertical rather than the horizontal midline. Displacement of the posterior LC and thinning of the LC occur more commonly in cupping due to glaucoma than in compressive optic neuropathy [16]. Measuring BMO-MRW reveals significantly fewer glaucoma values than other optic neuropathies [17].

Considering the following factors can help distinguish this condition from glaucoma: loss of central visual acuity, color vision defects, RAPD, and neuroretinal rim pallor of the optic disc, particularly the temporal part, with diffuse or temporal and nasal nerve fiber layer defect (in contrast to glaucoma, which has a pink rim and a thinner superior and inferior nerve fiber layer) [12, 18].

Periventricular Leukomalacia (PVL)

PVL is a structural loss of white matter pathways in prematurity. They may present with an enlarged cup-to-disc ratio, retinal nerve fiber layer thinning, and visual field defects, leading to a misdiagnosis of NTG. A small optic disc area is observed only in children with white matter damage before 28 gestational weeks. However, optic disc cupping is observed in children with a brain lesion after 28 gestational weeks. Prematurity in children, brain MRI, and the absence of normal-tension glaucoma can help differentiate PVL from glaucoma [19, 20.]

Congenital Hydrocephalus

While patients with congenital hydrocephalus do not typically develop papilledema, their optic nerves may be damaged by prolonged elevations in ICP and manifest as optic disc hypoplasia or cupping, depending on the time of injury [21].

Perinatal asphyxia (Hypoxic ischemic encephalopathy)

Perinatal asphyxia is a common cause of pediatric optic atrophy.

The enlarged cupping that mimics a glaucomatous disc occurs due to the loss of retinal ganglion cells because of hypoxic damage to the anterior visual pathway or retro-geniculate pathway, a condition known as trans-synaptic degeneration. Low Apgar scores, a history of seizures, cerebral palsy (CP), developmental delay (perinatal asphyxia signs), flare MRI hyperintensities in the periventricular and occipital lobe areas, and normal intraocular pressure can all aid in diagnosis [22]. Abnormal ONH morphology (pallor, cupping, or hypoplasia) is seen in 70 % of subjects with CP and is a predictor of poor motor outcome, indicating the significance of early referral for rehabilitation [23].

Ischemic optic neuropathy

Ischemic optic neuropathy results from acute hypoperfusion of the optic disc.

Acute phase:

Acute vision loss is associated with disc edema, peripapillary hemorrhages, cotton wool spots in anterior ischemic optic neuropathy (AION), or normal disc appearance in posterior ischemic optic neuropathy (PION).

Late phase:

Non-arteritic anterior ischemic optic neuropathy (NAAION), presence of sectoral disc pallor (infrequent cupping), attenuation of blood vessels, a small crowded disc (disc at risk) in the fellow eye, possible vasculopathy risk factors, or sleep apnea.

Table 1: Clinical features of glaucoma and also red flags for glaucoma mimickers

Clinical features of glaucoma	Red flags for glaucoma mimickers
Vertical elongation of the cup	VF defect inconsistent with optic disc appearance
Notching or Total cupping	Fast VF progression (1.5 dB per year or more)
Pink neuroretinal rim	Scotoma restricted by a vertical line
Deepened excavation	Low vision or unreasonable fast decrease VA
Peripapillary atrophy	Dyscromatopsia or asymmetrical color vision
Exposed lamina cribrosa	Unilaterality or RAPD
OCT: Superior or inferior thinning of RNFL	OCT: Temporal or diffuse thinning of RNFL
Disc hemorrhage	Normal IOP
Baring of the circumlinear vessels	Pallor of the remaining neuroretinal rim*
Family history of glaucoma	Age < 50 years

VF: Visual field, dB: Decibel, VA: Visual acuity, RAPD: Relative afferent pupillary defect, OCT: Optical coherence tomography, RNFL: Retinal nerve fiber layer, IOP: Intra ocular pressure.
*Certain glaucomatous conditions, such as acute angle-closure attack or secondary glaucoma with abruptly increased IOP, can be associated with a significant optic disc pallor due to ischemia [25-27].

Arteritic anterior ischemic optic neuropathy (AAION) is associated with increased cupping (up to 50 % of cases), weight loss, jaw claudication, elevated ESR, and C-reactive protein. These eyes exhibit marked neuroretinal disc pallor in contrast to glaucoma. Ischemic optic neuropathy is frequently related to superior or inferior altitudinal field defects that mimic glaucoma. However, sudden vision loss and neuroretinal rim pallor can help distinguish them [11, 12].

Radiation optic neuropathy

Radiation optic neuropathy (RON) can take place years after treatment because the optic nerve is a radiosensitive structure. However, most cases occur within three years of radiation, with a peak incidence after 1.5 years. RON is typically defined as a sudden and irreversible vision loss in one or both eyes due to anterior or posterior optic neuropathy. The RON can happen following radiation therapy for any tumor near the optic nerve, optic chiasm, or optic tract because of direct or indirect damage to the optic nerve or its vascular supply (radiation vasculitis). The clinical presentation is sudden and irreversible unilateral or bilateral vision loss with visual field defects associated with disc edema in the early stages and optic atrophy in the late stages, resulting in a misdiagnosis of glaucoma. Nevertheless, abrupt early phases and radiation history can help to differentiate them [24.]

Discussion

Clinicians should strongly consider the possibility of a neurologic etiology, such as masses, ischemia, or inflammation. When not associated with cupping, RNFL or papillomacular bundle RNFL loss are highly suspicious for a non-glaucomatous etiology. Additionally, the patient's young age, disc pallor, loss of visual acuity and color vision, and a poor correlation between the optic

nerve and VF findings all point to a condition other than glaucoma [25]. Glaucoma-related visual field defects are known to respect the horizontal raphe of the retina due to the anatomic distribution of the retinal nerve fiber layer. Respect for the vertical midline is highly specific for a neurological issue. Lower average RNFL thickness in the nasal and temporal quadrants and a lower GCC in non-glaucomatous etiologies, compared to glaucoma, even when cup disc ratio and overall RNFL thickness were comparable [3, 26]. Table 1 shows the clinical features of glaucoma and also red flags for glaucoma mimickers.

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Footnotes and Financial Disclosures

Conflict of interest:

The authors have no conflict of interest with the subject matter of the present manuscript