The Review of DIABETIC STUDIES
Reprint from
The Review Of
DIABETIC
STUDIES
OPEN ACCESS

Interprofessional Management OF Diplopia AND Visual Health-An Updated Review For Optometrists, Pharmacists, Radiologists, AND Nursing Professionals

Samar Mohammad Alghamdi[1],Maha khalofah Alghamdi[2],Fahad Bader Alharbi[3],Majed Salah Almarwani[4],Hassan Mohammd Alshehri[5] ,Reem Saleh Alamri[6],Bayan Hajij Almarwani[7],Saad Mushabbab Saad Alqadham[8],Mubarak Muhathil Faleh Alqahtani[9],Mohammed Abdullah Alsahli[10],Abdulrahman Abdullah Saif Alsahla[11],Sultan Mohammed Sayf Alsahli[12] ,Ali Dhifallah Bakheet Alzahrani[13],Sami Abdullah Alwagdani[14]

[1]*Ohud Hospital*
[2]*Ohud Hospital*
[3]*Ohud Hospital*
[4]*Ohud Hospital.*
[5]*Ohud Hospital*
[6]*Ohud Hospital*
[7]*Ohud Hospital*
[8]*General Administration of Health Programs and Chronic Diseases, Ministry Headquarters*
[9]*Rawdat Al Ardah General Hospital, Riyadh Al Quwaiiyah*
[10]*Primary Health Care Center, Al Muzahimiyah, Riyadh*
[11]*Ruwaydat Al Ard General Hospital*
[12]*Rawidat Al Ardah General Hospital*
[13]*Assistant Administration for Compliance, Riyadh Health Affairs*
[14]*King Faisal Complex, Taif, Ministry of Health*

Abstract:

Background: Diplopia, or double vision, is a common yet clinically significant visual complaint with diverse ocular, neurological, and systemic etiologies. It may arise from monocular optical disturbances or binocular ocular misalignment, the latter often linked to cranial nerve dysfunction, orbital pathology, or neuromuscular disorders.
Aim: This review synthesizes updated evidence on interprofessional approaches to classification, diagnosis, and management of diplopia.
Methods: The article evaluates epidemiological data, etiological classifications, diagnostic pathways, and therapeutic options across published clinical studies and specialty guidelines.
Results: Binocular diplopia is most frequently associated with microvascular cranial nerve palsy, thyroid eye disease, trauma, and systemic conditions including malignancy and infectious processes such as COVID-19, with acute cases requiring urgent imaging to exclude life-threatening pathology. Diagnostic precision relies on structured history, motility testing, and targeted neuroimaging, while management ranges from optical correction and prism therapy to surgical realignment and treatment of underlying disease.
Conclusion: Interprofessional collaboration among ophthalmology, neurology, radiology, and rehabilitation services is critical for timely identification of etiologies, reduction of complications, and optimization of functional outcomes for patients with diplopia.

Keywords: Diplopia, binocular misalignment, cranial nerve palsy, thyroid eye disease, neuro-ophthalmology, strabismus, management.

Introduction:

Diplopia, commonly referred to as double vision, is characterized by the perception of two images of a single object, which may be separated vertically, horizontally, or obliquely. This condition can be classified as either monocular or binocular in origin, depending on whether it persists when one eye is covered. Monocular diplopia typically arises from optical aberrations within the affected eye, such as cataracts, corneal irregularities, or lens dislocation, and does not resolve with occlusion of one eye. In contrast, binocular diplopia results from ocular misalignment, often due to strabismus or cranial nerve dysfunction, and usually disappears when either eye is occluded. Accurate classification of diplopia is critical, as the underlying causes range from benign refractive errors to neurologically or systemically significant pathology.[1] Patients presenting with binocular diplopia should undergo a thorough and systematic evaluation to identify all potential underlying causes. Acute onset diplopia requires urgent assessment, especially if associated with red flags such as headache, pupillary changes, or other neurological deficits, as these may indicate life-threatening conditions such as stroke, intracranial mass, or aneurysm. Standard clinical tests, including cover-uncover and alternate cover testing, prism testing, and ocular motility assessment, can help localize the source of ocular misalignment. In many cases, diplopia secondary to microvascular etiologies, such as diabetes or hypertension-induced cranial neuropathies, resolves spontaneously within six months without surgical intervention.[1] Early and accurate identification of diplopia etiology is essential for timely management and to prevent potential complications. Collaboration among optometrists, ophthalmologists, and other healthcare professionals ensures comprehensive care, including diagnostic imaging, corrective lenses, occlusive therapy, and, when necessary, referral for surgical or neurological intervention. This multidisciplinary approach optimizes patient outcomes while addressing both functional and quality-of-life concerns associated with diplopia.

Etiology

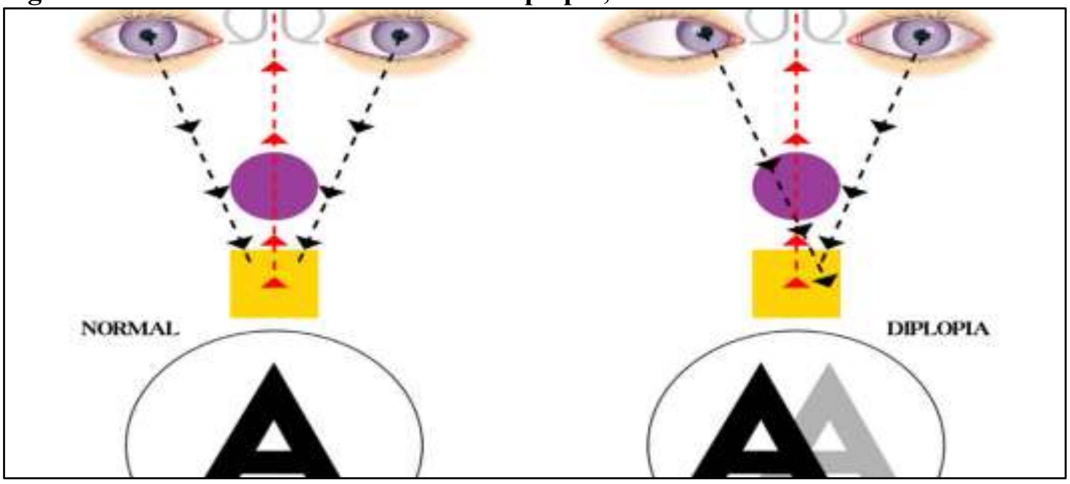
The etiology of diplopia is fundamentally divided into two categories based on its presentation: binocular or monocular. Binocular diplopia arises from misalignment of the eyes, whereas monocular diplopia is typically the result of optical abnormalities within a single eye. Understanding the underlying causes of binocular diplopia requires a systematic consideration of the anatomical and neurological pathways that maintain ocular alignment. Proper alignment depends on the coordinated innervation of all extraocular muscles, ensuring that the eyes remain in the primary position with equal antagonistic tension. Disruption at any level—from the cranial nerve nuclei to the muscles themselves—can result in double vision. Cranial nerves III (oculomotor), IV (trochlear), and VI (abducens) are responsible for innervating the extraocular muscles. These nerves originate in specific regions of the brainstem, with cranial nerves III and IV emerging from the midbrain and cranial nerve VI from the pons. The nerve fascicles traverse the brainstem and exit ventrally in the case of cranial nerves III and VI, and dorsally for cranial nerve IV. Along their course in the subarachnoid space, the nerves are susceptible to a variety of pathologies including inflammation, infection, hemorrhage, or malignancy. Any disruption in nerve function along this pathway can lead to diplopia, either through partial or complete extraocular muscle paralysis. Upon exiting the subarachnoid space, all three nerves converge in the cavernous sinus, a venous structure lateral to the pituitary gland. The proximity of these nerves in the cavernous sinus explains why lesions in this area often produce multiple cranial nerve palsies. Pathologies affecting neighboring structures, such as pituitary adenomas, sphenoid sinus disease, or internal carotid artery aneurysms, can exert compressive or infiltrative effects on the nerves, resulting in ocular misalignment. Following the cavernous sinus, the cranial nerves travel in close association through the superior orbital fissure, alongside the optic nerve (cranial nerve II). Lesions in this region frequently produce a combination of diplopia, visual deficits, and proptosis. Orbital diseases, including tumors, trauma, or inflammatory conditions, may directly interfere with nerve function or extraocular muscle movement, contributing to binocular diplopia. Neuromuscular junction disorders, such as myasthenia gravis, represent another potential cause, disrupting the transmission of neural signals to the muscles, thereby impairing ocular alignment. Finally, intrinsic pathology of the extraocular muscles, including myopathies or localized trauma, can independently produce diplopia. Therefore, a comprehensive understanding of diplopia etiology requires consideration of the full continuum from central neural control, cranial nerve course, neuromuscular transmission, and muscular integrity. Monocular diplopia, in contrast, is usually caused by abnormalities within the cornea, lens, or vitreous that generate distorted or duplicated images independent of ocular alignment. Recognizing the distinction between monocular and binocular diplopia is critical, as it guides the diagnostic approach, including imaging, laboratory assessment, and referral to appropriate specialists such as optometrists, ophthalmologists, or neurologists, to identify and address the underlying pathology effectively.[1]

Classification and Etiology

A study conducted in South China employed a novel classification system to evaluate diplopia according to extraocular muscle (EOM) dysfunction, categorizing patients into three distinct groups: single EOM (sEOM), multiple EOMs (mEOMs), and comitant strabismus [2]. The sEOM group is the most frequently observed pattern, often arising from microangiopathic processes and trauma, with the lateral rectus being the muscle most commonly involved. In certain cases, diplopia in this group was associated with nasopharyngeal carcinoma (NPC), while others developed sEOM due to radiation-induced neuropathy following therapeutic interventions [2]. These findings highlight the intersection of localized cranial nerve impairment and systemic or treatment-related factors in producing diplopia. The mEOM group primarily results from thyroid-associated ophthalmopathy, which causes inflammation and fibrotic changes in multiple extraocular muscles, producing complex motility disturbances and restricted ocular movement [2]. In contrast, patients in the comitant strabismus group commonly present with acute acquired comitant esotropia. In these cases, misalignment is consistent across all gaze directions, differentiating it from incomitant deviations where misalignment varies depending on the direction of gaze [2].

Monocular diplopia, distinct from binocular diplopia, persists when only one eye is open and is usually caused by intrinsic ophthalmologic factors. Common etiologies include cataractous changes in the crystalline lens, abnormalities of the corneal surface such as keratoconus or uncorrected astigmatism, and, in rare cases, cortical lesions affecting the occipital lobe, termed cortical polyopia. Cortical polyopia is frequently associated with homonymous visual field defects [2]. When no structural or optical etiology is identified and diplopia does not improve when looking through a pinhole, a functional cause is presumed [2]. Recent evidence suggests that infectious and postinfectious processes can also contribute to diplopia. Specifically, COVID-19 infection has been associated with a mild increase in abducens nerve involvement, and postvaccination diplopia has been observed in patients receiving both vector-based and RNA-based vaccines [3]. These findings indicate that viral or immune-mediated mechanisms may transiently impair ocular motor function, resulting in diplopia even in otherwise healthy individuals [3].

Fig. 1: Differences between normal and diplopia,



Acute-onset diplopia remains a rare and challenging presentation in ophthalmology. Most cases involve isolated cranial nerve palsies of the third, fourth, or sixth nerves [4]. Other contributing factors include mechanical restrictions, dysfunction of higher neurological control, decompensation of pre-existing heterophoria, idiopathic causes, and monocular visual disturbances [4]. Clinical assessment can identify the underlying cause in most patients, while only a small subset requires urgent radiological evaluation to exclude emergent pathology [4]. Diplopia has also been documented as a complication of surgical procedures, including neurosurgical, dental, endoscopic paranasal sinus, and ophthalmic surgeries [5].

Furthermore, diplopia may develop following surgical repair of orbital floor or blowout fractures, or due to dehiscence of scleral belt loop tunnels [6][7][8][9]. Such cases demonstrate that structural disruption of orbital tissues or direct trauma to extraocular muscles can lead to misalignment and double vision. Reviews of clinical cohorts have identified the common causes of binocular diplopia according to both etiology and clinical presentation. Microvascular disease, cerebrovascular accidents, neoplastic processes, myasthenia gravis, trauma, and decompensated phoria are the most frequent underlying causes [10]. Management of these cases is typically led by neurologists, followed by ophthalmologists, emergency physicians, and neurosurgeons [10]. This interdisciplinary approach underscores the importance of early and accurate assessment to guide timely intervention and optimize outcomes for patients with diplopia [10]. The study emphasizes that categorizing diplopia according to EOM involvement, distinguishing between monocular and binocular types, and considering systemic, infectious, and procedural etiologies allows for more precise diagnosis and targeted management. Such an approach improves patient outcomes, particularly in acute-onset cases or those related to systemic or surgical complications, by facilitating rapid identification of the underlying cause and appropriate referral [2][3][4][5][6][7][8][9][10].

Epidemiology

Diplopia is a frequently encountered clinical complaint in both outpatient and emergency settings. In the United States, studies have estimated nearly 805,000 ambulatory visits and 50,000 emergency department encounters annually with diplopia as the presenting complaint [11]. This high incidence highlights the significance of diplopia as a common and distressing symptom for patients, particularly when it presents acutely. Acute-onset diplopia often provokes considerable anxiety, as patients fear underlying serious pathology. Despite this concern, studies indicate that only a minority of cases are attributable to life-threatening etiologies, with one analysis reporting that just 16% of patients presenting with diplopia had conditions considered potentially life-threatening [4]. This demonstrates that while diplopia warrants prompt evaluation, the majority of cases are related to benign or self-limited causes such as microvascular cranial nerve palsies or functional misalignment. Diplopia also holds particular relevance in oncology, where it may serve as an early indicator of malignancy-related complications. Recent studies have identified diplopia as a debilitating symptom in patients with cancer, often resulting from orbital or intracranial metastases or local tumor invasion [12]. These manifestations may present as either paralytic strabismus or restrictive strabismus, with sixth cranial nerve palsy being the most commonly affected in paralytic cases [12]. Diplopia in this context is often associated with a progressive clinical course, reflecting the severity and extent of tumor involvement. Screening for metastatic disease in patients with new-onset diplopia is therefore critical to guide timely intervention and optimize life expectancy. These findings underscore the dual nature of diplopia: it is a prevalent and distressing symptom in the general population, but in specific high-risk groups, such as oncology patients, it may signify serious underlying pathology requiring immediate assessment and targeted management [11][12]. Understanding the epidemiology of diplopia, including its prevalence, common etiologies, and associations with systemic disease, is essential for clinicians to appropriately stratify risk, determine the urgency of evaluation, and plan diagnostic and therapeutic strategies in both general and specialized patient populations [4][11][12].

Pathophysiology

Binocular diplopia arises when the image projected onto the fovea of one eye does not coincide with the corresponding foveal image of the other eye, resulting in the perception of two distinct images. The direction of diplopia directly corresponds to the axis of ocular misalignment; horizontal deviations produce horizontal diplopia, whereas vertical misalignments result in vertical diplopia. Beyond simple misalignment, maculopathies may disrupt point-to-point foveal correspondence, creating diplopia through altered central sensory fusion. The effects of macular pathology on central versus peripheral sensory fusion differ significantly, challenging conventional approaches to diplopia management that primarily target motor alignment rather than sensory integration [13]. In adults, diplopia frequently presents in outpatient settings and is commonly classified based on the type of strabismus. Although a significant proportion of adult diplopia cases are idiopathic, diplopia can occasionally signal serious underlying pathology, necessitating prompt and thorough evaluation [14]. Epidemiological studies using magnetic resonance imaging (MRI) to assess orbital structures have revealed that elderly patients presenting with binocular diplopia more often exhibit vertical deviations compared with other strabismus types, including exotropia, esotropia, and combined deviations. Specific causes within each strabismus group have been identified: in the exotropia group, convergence insufficiency exotropia and basic exotropia are most common; in the esotropia group, orbital pulley disorders and sixth cranial nerve palsy predominate; vertical strabismus is frequently associated with fourth cranial nerve palsy or orbital pulley dysfunction; and combined strabismus typically results from orbital pulley disorders and fourth cranial nerve palsy [15]. Horizontal strabismus can also be complicated by dysfunction of the oblique extraocular muscles, particularly the inferior or superior oblique. This disruption often leads to compensatory head posture, vertical or torsional diplopia, and abnormal binocular fusion, reflecting the intricate interplay between muscular control, orbital mechanics, and visual perception [16]. Such cases underscore the complex pathophysiology underlying diplopia, where subtle neuromuscular deficits can translate into significant functional impairment. In pediatric populations, diplopia generally corresponds to nonemergent conditions; however, when diplopia presents acutely in children with accompanying neurologic or visual symptoms, it may indicate a life-threatening etiology, including cranial nerve palsy, intracranial mass, or vascular insult [17]. The pathophysiology in these acute cases often involves disruption of ocular motor pathways at the level of the cranial nerves, brainstem nuclei, or neuromuscular junction, highlighting the importance of rapid assessment. Across age groups, the pathogenesis of diplopia integrates ocular alignment, extraocular muscle function, orbital architecture, and central sensory processing, illustrating the multifactorial mechanisms that contribute to both acute and chronic presentations. Understanding these mechanisms is essential for accurate diagnosis, risk stratification, and targeted management strategies in patients with diplopia [13][14][15][16][17].

History and Physical

A meticulous history and comprehensive clinical examination are essential in evaluating patients presenting with diplopia. The initial step involves characterizing the type of diplopia as either monocular or binocular. This distinction is critical, as monocular diplopia typically indicates a primary ocular or optical problem, whereas binocular diplopia reflects misalignment of the visual axes and may be associated with neurological, muscular, or orbital pathologies. Failure to establish this distinction early may lead to unnecessary investigations, delays in diagnosis, and increased patient anxiety [18]. Once the type is identified, the onset and progression of symptoms should be carefully documented. Acute-onset diplopia warrants urgent evaluation, particularly when associated with neurological symptoms. Patients should be assessed for accompanying brainstem signs, even though isolated brainstem strokes are uncommon. Diplopia may represent the primary manifestation of lesions affecting the diencephalon, brainstem, cranial nerve nuclei or fascicles of cranial nerves III, IV, or VI, or the medial longitudinal fasciculus and vestibulo-ocular pathways, potentially producing skew deviation [19]. Patients presenting with acute binocular diplopia and any additional signs suggestive of brainstem dysfunction—such as vertigo, dizziness, dysarthria, crossed motor or sensory deficits, ataxia, or imbalance—require immediate referral to an emergency department for MRI evaluation of the brain and brainstem. Imaging should include diffusion-weighted and susceptibility-weighted sequences to detect subtle ischemic or hemorrhagic lesions.

Fig. 2: Diplopia.

Further history should explore symptom variability, fatiguability, and potential triggers or relieving factors, as these can indicate neuromuscular junction disorders, such as myasthenia gravis. Patients should also be questioned regarding signs of increased intracranial pressure, including headache, nausea, and transient visual obscurations. For patients over the age of 50, screening for symptoms consistent with giant cell arteritis, such as temporal headache, jaw claudication, or scalp tenderness, is recommended. Epidemiological studies have identified common clinical characteristics in adults with acquired diplopia, including double vision, blurred vision, monocular diplopia, and eye strain. Past ocular history, including childhood strabismus or amblyopia, as well as systemic conditions such as hypertension and prior cranial nerve paresis, may predispose patients to symptomatic strabismus or age-related divergence insufficiency esotropia [20]. The physical examination begins with assessment of ocular motility. Each eye should be tested individually in all cardinal directions of gaze to detect deficits consistent with cranial nerve III, IV, or VI dysfunction. Alternate cover testing is performed to evaluate ocular alignment. In cases where vertical misalignment is suspected, measurements should be recorded in ipsi- and contralateral gaze, as well as in ipsi- and contralateral head tilt positions. The three-step test, which examines hypertropia increasing in contralateral gaze and ipsilateral head tilt, can help confirm fourth nerve paresis [19]. When ocular motility is full and eye misalignment remains uniform in all directions of gaze, the deviation is classified as comitant, often due to decompensated congenital strabismus. These patients typically do not require extensive further evaluation beyond ophthalmological referral. Comprehensive examination should also include assessment of pupils, eyelid position and movement during gaze testing, proptosis, orbicularis oculi function, and dilated funduscopy to identify optic nerve head edema or signs of venous stasis retinopathy. Quantitative documentation of motility deficits, ideally expressed in percentages of expected movement, ensures accurate monitoring and guides subsequent diagnostic or therapeutic interventions. This structured approach to history and physical examination forms the foundation for identifying the etiology of diplopia and for planning timely, targeted management.

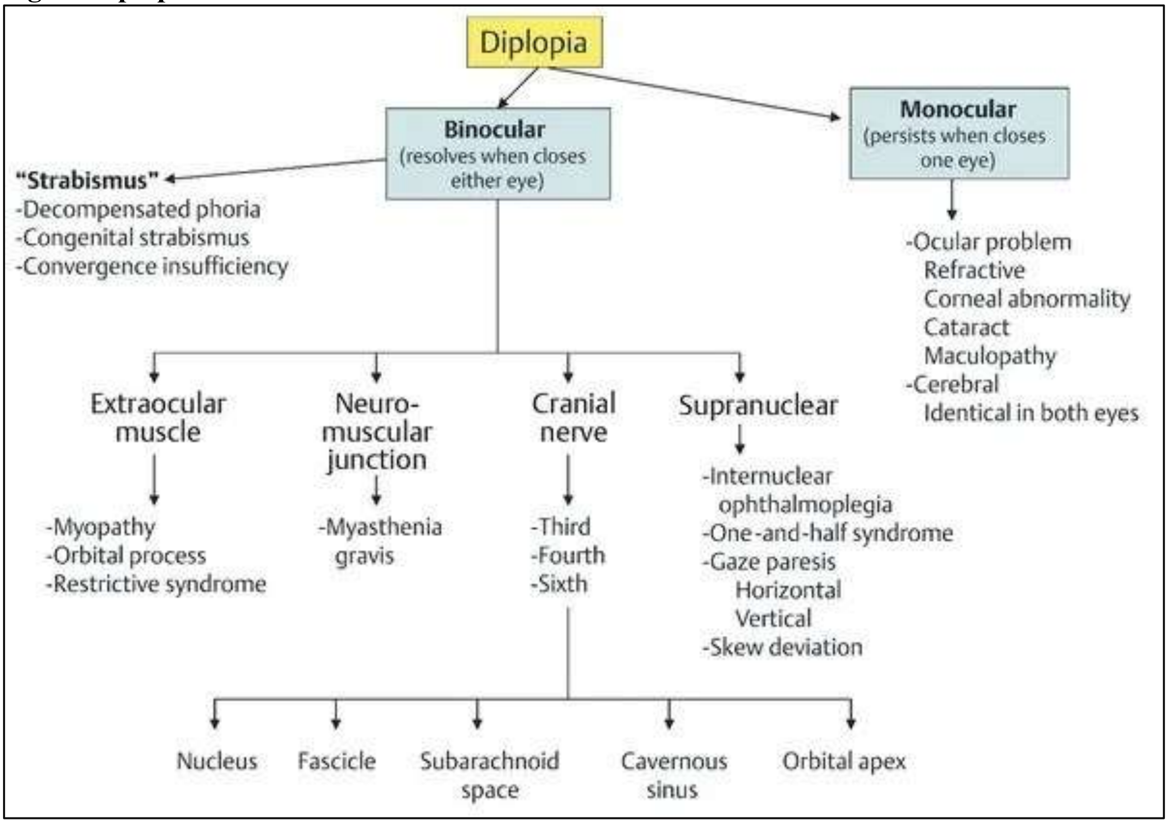
Evaluation

The evaluation of diplopia begins with a structured and detailed assessment of extraocular motility in each eye individually, followed by careful ocular alignment testing. Alignment is assessed using alternate cover testing, and abnormalities should be recorded in all cardinal positions of gaze, ideally using prism diopters to quantify deviations. These findings are integrated to determine whether the diplopia can be attributed to an isolated palsy of the third, fourth, or sixth cranial nerves. Distinguishing between complete and partial nerve palsies is critical, as the management for each differs substantially. Diagnosing complete third or sixth nerve palsy is relatively straightforward, whereas partial third nerve paresis can be challenging to identify. Fourth nerve palsy requires a nuanced understanding of the three-step test, which evaluates the hypertropia in contralateral gaze and ipsilateral head tilt to confirm superior oblique involvement [21]. When multiple cranial nerves are affected, urgent neuroimaging becomes mandatory. Magnetic resonance imaging (MRI) of the brain and orbits with contrast is indicated to assess for lesions along the course of the oculomotor nerves, including the cavernous sinuses, superior orbital fissure, and extraocular muscles. Early MRI evaluation in these scenarios enhances diagnostic accuracy for binocular diplopia, avoids unnecessary radiation exposure, and does not prolong emergency department length of stay or delay clinical decision-making [21]. Patients with signs of fatiguability, variability in diplopia, or decreased orbicularis oculi strength should be evaluated for ocular or generalized myasthenia gravis. Acetylcholine receptor antibody testing may be performed, though its sensitivity ranges from 50% to 70% [22]. In cases where antibody tests are negative but clinical suspicion remains high, single-fiber electromyography (EMG) of the orbicularis muscle is recommended. When performed by an experienced operator, single-fiber EMG demonstrates approximately 95% sensitivity for detecting ocular myasthenia-specific abnormalities [23].

All patients presenting with third cranial nerve palsy, whether complete, partial, pupillary-sparing, or pupillary-involving, require emergent computed tomography angiography (CTA) to exclude a compressive aneurysm along the subarachnoid course of the nerve. CTA is widely available and reliably detects aneurysms larger than 2 to 3 millimeters, although aneurysms generally need to reach approximately 4 millimeters to produce significant third nerve compression [24]. Interpretation accuracy depends on the radiologist's experience; if pupillary involvement is present and CTA is read as normal, clinicians must communicate directly with the radiologist to ensure careful review of the internal carotid–posterior communicating artery junction, the most common site for aneurysms causing third nerve palsy [25]. If CTA is normal and the patient is over 50 with vascular risk factors, a microvascular ischemic etiology is likely, and the patient can be observed for 2 to 3 months for expected improvement or resolution. Patients younger than 50 or those without vascular risk factors should undergo urgent MRI with contrast and steady-state sequences to evaluate the entire course of the third nerve and exclude compressive or demyelinating lesions [24]. Fourth nerve palsy is evaluated based on vertical deviation in gaze. If the hypertropia is greater in upgaze than downgaze, it is likely due to decompensated congenital strabismus, trauma, or microvascular ischemia and can be managed conservatively with prism correction or observation for spontaneous improvement [26]. If deviation worsens in downgaze, contrast-enhanced MRI with steady-state sequences is recommended to assess the fourth nerve along its course and exclude compressive lesions.

Acute sixth nerve palsy in patients over 50 with vascular risk factors and isolated deficits may initially be observed, with imaging reserved for cases that do not improve within 2 to 3 months. Younger patients and those without microvascular risk factors require MRI with contrast and steady-state sequences to evaluate for demyelinating, compressive, or infiltrative causes along the nerve's course [19]. In the emergency department, several strategies streamline the evaluation of diplopia. Unenhanced CT of the head or orbits is generally unhelpful, whereas MRI is preferred for isolated ocular motor nerve palsies. Patients with isolated fourth or sixth nerve palsies without additional neurological deficits are often referred to a neurologist or ophthalmologist for further evaluation due to limited ED resources. Acute third nerve palsy warrants immediate CT and CTA to rule out aneurysmal compression. Contrast-enhanced imaging is indicated in suspected orbital apex syndrome, retro-orbital masses, thyroid eye disease, or ocular trauma. CT and CT venography are valuable in suspected cavernous sinus thrombosis. For patients over 60 with a recent history of diplopia, inflammatory markers should be assessed to exclude giant cell arteritis [21]. This structured approach—integrating detailed ocular motility testing, alignment evaluation, laboratory assessment, and targeted imaging—ensures accurate identification of underlying etiologies. Early differentiation of microvascular, compressive, infectious, or neuromuscular causes enables timely intervention, reduces the risk of vision-threatening complications, and improves long-term functional outcomes for patients presenting with diplopia.

Fig. 3: Diplopia.



Treatment / Management

Diplopia represents a multifaceted clinical problem with both ocular and neurological origins, requiring a targeted approach for effective management. Its presentation varies from monocular to binocular, and treatment strategies are largely determined by the underlying etiology and severity of the misalignment. For monocular diplopia, the primary focus is on correcting optical or media-related abnormalities. Comprehensive refraction and biomicroscopic examination of the crystalline lens, cornea, and other optical structures are essential. Cataract surgery may be indicated when lens opacities are present, and uncorrected refractive errors should be addressed with corrective lenses. Functional visual improvement in monocular diplopia often depends on optimizing the optical pathway to restore single-image perception [4]. Binocular diplopia arises from ocular misalignment and requires careful identification of the responsible extraocular muscle or cranial nerve involvement. Initial management typically emphasizes noninvasive interventions aimed at symptom relief. Prism lenses incorporated into spectacles can effectively palliate diplopia, especially when the deviation is small, generally up to 10–12 prism diopters. For larger misalignments, temporary occlusion of one eye, using a patch or taping one lens of spectacles, can eliminate diplopia and provide functional visual relief. In patients with stable, symptomatic misalignments, referral to a strabismologist is indicated to assess the potential for surgical correction. Surgical intervention aims to restore ocular alignment and binocular vision, particularly in patients whose diplopia significantly interferes with daily functioning [27].

Botulinum toxin injections represent another therapeutic option for patients with paresis of extraocular muscles. By injecting the antagonist of the affected muscle, ocular alignment can be temporarily improved, though the effect is dose-dependent, variable, and not permanent. Monovision techniques, where a contact lens is used to alter the refractive state of one eye, can also provide relief for small-angle misalignments by disrupting binocular fusion [28]. Diplopia secondary to structural lesions, such as skull base meningiomas affecting the abducens nerve, has shown improvement following stereotactic radiosurgery. Studies demonstrate that early intervention, particularly with gamma knife surgery, is associated with better resolution of diplopia. The timing of intervention plays a crucial role in preserving ocular motor function and preventing long-term misalignment [29]. Advances in understanding vertical diplopia highlight the role of skew deviation versus trochlear nerve palsy. Supine positioning can differentiate between these etiologies, with skew deviation demonstrating improvement when supine. For ocular myasthenia gravis, therapeutic strategies, including corticosteroids or thymectomy, have been shown to reduce conversion to generalized disease and improve ocular motility [30]. Surgical options for vertical deviations include procedures such as temporal slant recession of the inferior rectus muscle (TSRIRM). This intervention has demonstrated efficacy for small-angle vertical strabismus, offering reliable correction in outpatient settings. TSRIRM is particularly advantageous due to its accessibility and minimal invasiveness, making it suitable for patients with persistent diplopia who do not respond to noninvasive measures [31]. In conclusion, the management of diplopia requires an individualized, etiology-driven approach. Monocular cases focus on optical correction and lens management, whereas binocular diplopia often necessitates a combination of prism therapy, occlusion strategies, botulinum injections, or surgical intervention. Structural lesions and systemic conditions require early recognition and specialized management to prevent permanent misalignment or functional vision loss. Emerging surgical techniques and neuro-ophthalmologic advances continue to improve outcomes, providing targeted, evidence-based approaches that enhance quality of life for patients with diplopia.

Differential Diagnosis

The differential diagnosis of diplopia is broad, particularly for binocular diplopia, and requires an anatomical and systematic approach to identify the underlying etiology. Binocular diplopia results from ocular misalignment, which may originate from lesions affecting the cranial nerves III, IV, and VI, their nuclei, fascicles, or pathways connecting them. Lesions in the brainstem affecting these cranial nerves, the vestibulo-ocular pathways, or the medial longitudinal fasciculus can lead to diplopia. Although lesions affecting horizontal or vertical gaze centers in the pons or midbrain are uncommon, they may also contribute to ocular misalignment and diplopia. Posterior reversible encephalopathy syndrome (PRES) can present with visual disturbances including diplopia, cortical blindness, hemianopia, quadrantanopia, seizures, headaches, and altered mental status, highlighting the importance of recognizing systemic and neurological associations early [32]. Lesions of the medial longitudinal fasciculus often produce diplopia and may arise from demyelination in patients under 50 or ischemic events in older patients. Superior oblique myokymia (SOM) manifests as monocular, high-frequency, low-amplitude torsional contractions of the superior oblique muscle, resulting in diplopia and oscillopsia [33]. Uveomeningitis and myelodysplastic syndromes can also present with new-onset diplopia, ataxia, and fluctuating consciousness, often with unremarkable brain imaging or blood chemistries [34]. Pathology involving cerebrospinal fluid, including inflammatory processes or malignancy, can produce oculomotor nerve palsies, while elevated intracranial pressure frequently affects the sixth cranial nerve. The Miller-Fisher variant of Guillain-Barré syndrome produces diplopia secondary to preferential involvement of the oculomotor nerves, accompanied by ataxia and areflexia in adults [35]. In pediatric patients, diplopia may indicate serious conditions such as Guillain-Barré syndrome, myasthenia gravis, botulism, or poisoning, necessitating rapid evaluation [36]. Wernicke encephalopathy due to thiamine deficiency can also cause diplopia and should prompt intravenous thiamine administration in patients presenting with confusion, nystagmus, or ataxia.

The cavernous sinus is a frequent site for lesions causing multiple cranial nerve palsies. Etiologies include idiopathic or inflammatory conditions such as sarcoidosis, IgG4 disease, granulomatosis with polyangiitis, vascular lesions like aneurysms and carotid-cavernous fistulas, neoplastic processes including pituitary apoplexy or metastatic spread, and infectious causes such as cavernous sinus thrombosis. Lyme neuroborreliosis may present with diplopia and oculomotor palsy, often with radicular neck pain, lymphocytic pleocytosis in cerebrospinal fluid, and intrathecal antibody synthesis [37]. Other rare tumor-related causes include extrarenal rhabdoid tumors in the carotid space [38] and metastatic lung squamous cell carcinoma [39]. Osteomas involving paranasal sinuses have been reported to present with proptosis and diplopia [40]. Superior orbital fissure lesions, whether due to inflammation, infection, or trauma, may also compromise multiple cranial nerves, producing diplopia. Systemic lymphohistiocytic infiltration can manifest with diplopia, orbital mass, and acute vision loss, often associated with autoimmune disease, viral infections, malignancy, or drug reactions [41]. Orbital diseases such as thyroid eye disease, tumors, or vascular lesions are notable causes, with progressive proptosis and diplopia as hallmark features. Studies indicate that teprotumumab is more effective than intravenous methylprednisolone in reducing diplopia in thyroid eye disease [42]. Gradenigo syndrome, characterized by abducens nerve palsy, retro-orbital pain, and chronic otorrhea, occurs due to apical petrositis following chronic otitis media [43]. Convergence insufficiency, a binocular vision disorder, produces asthenopia, diplopia, headaches, and difficulty maintaining near focus [44].
Rare genetic and inflammatory ocular conditions, such as keratoendotheliitis fugax hereditaria, may cause recurrent unilateral corneal hyperemia, corneal edema, visual impairment, and diplopia, accompanied by photophobia, lacrimation, and pseudoguttata [45]. Finally, disorders of the neuromuscular junction and myopathies affecting extraocular muscles, including seronegative ocular myasthenia gravis, may manifest as transient diplopia, particularly in older patients [46]. In conclusion, diplopia has a complex differential diagnosis encompassing neurological, ophthalmological, systemic, and rare genetic conditions. Accurate evaluation requires detailed anatomical knowledge of ocular motor pathways, careful assessment of cranial nerve function, and consideration of systemic and orbital etiologies. Early recognition of life-threatening or rapidly progressive causes, such as aneurysms, cavernous sinus lesions, or demyelinating disease, is critical for preventing permanent visual impairment or neurological deficits. A structured approach that integrates clinical, radiological, and laboratory findings enables clinicians to systematically narrow the differential and initiate appropriate, timely management.

Prognosis
The prognosis of diplopia varies widely and is entirely dependent on the underlying cause. In cases where diplopia is secondary to benign conditions, such as decompensated congenital strabismus or minor extraocular muscle imbalance, the condition may persist chronically but can often be addressed surgically. Patients with acquired strabismus may require long-term prism therapy or strabismus surgery to correct ocular misalignment and restore binocular vision. In cases of systemic or neurological conditions, prognosis is closely tied to the progression and management of the primary disease. For instance, diplopia caused by myasthenia gravis often improves with appropriate immunosuppressive

therapy or thymectomy, and ocular symptoms may resolve entirely in some patients. Similarly, patients with diplopia resulting from Guillain-Barré syndrome, particularly the Miller-Fisher variant, usually experience resolution of ocular motility deficits over weeks to months as part of the overall recovery from the neuropathic process. Neurological lesions, such as small brainstem strokes, may produce diplopia that improves spontaneously over time, although some patients may retain residual deficits depending on the extent of the infarct. In compressive lesions, such as aneurysms affecting the third cranial nerve or tumors in the orbit or cavernous sinus, timely intervention is critical to prevent permanent deficits; early surgical or endovascular treatment can lead to full or partial recovery of ocular alignment and resolution of double vision. Diplopia secondary to orbital pathologies, thyroid eye disease, or trauma may persist despite medical or surgical intervention, and management is often focused on symptom palliation, including prism therapy, occlusion, or rehabilitation exercises. Overall, while benign and medically treated conditions generally have a favorable prognosis, acute neurological, vascular, or compressive causes require urgent recognition and intervention to prevent permanent impairment. Long-term outcomes are closely tied to early detection, precise diagnosis, and targeted management strategies, emphasizing the importance of a systematic, interprofessional approach to patient care. Patients should be monitored closely, and follow-up assessments are necessary to ensure improvement or to plan for corrective measures if symptoms persist. The individualized prognosis should always consider the patient's age, comorbidities, and underlying pathophysiology [45][46].

Complications

The primary complication of diplopia is the functional and psychological burden it imposes on patients. Persistent double vision can significantly impair daily activities, including reading, driving, or performing visually guided tasks. Patients with diplopia may experience frequent headaches, eye strain, or nausea due to the constant misalignment of visual inputs. The severity of these symptoms is often correlated with the degree of ocular misalignment and the patient's ability to compensate with head posture or suppression of one eye. In severe cases, diplopia may cause safety concerns, such as difficulty navigating stairs, operating vehicles, or performing tasks requiring precise depth perception, increasing the risk of accidents or falls. Complications can also arise indirectly from the interventions used to manage diplopia. For example, surgical correction of strabismus or orbital decompression carries risks of infection, hemorrhage, scarring, or over- or under-correction of eye alignment. Prism lenses and occlusion therapy, while non-invasive, may lead to visual discomfort or temporary loss of stereopsis. In systemic or neurological causes of diplopia, untreated primary disease may lead to additional complications, such as progressive neurological deficits, visual field loss, or exacerbation of underlying vascular or autoimmune conditions. Psychosocial complications are also notable. Chronic diplopia can lead to frustration, anxiety, and decreased quality of life. Patients may develop social withdrawal or depression due to persistent visual disturbance and dependence on adaptive strategies. Pediatric patients with diplopia may experience delayed learning or impaired academic performance due to difficulty focusing on visual tasks. Overall, complications of diplopia extend beyond the ocular system to functional, neurological, and psychosocial domains. Early recognition, appropriate intervention, and patient education are crucial to minimizing these effects and improving long-term outcomes. Monitoring for complications related to both the underlying cause and therapeutic interventions is an essential component of comprehensive patient care [43][44][45].

Patient Education

Patient education is a critical component in managing diplopia. Patients must understand the nature of their condition, including the distinction between monocular and binocular diplopia, as this directly influences the diagnostic approach and treatment plan. Educating patients on the anatomical and physiological basis of ocular misalignment helps set realistic expectations regarding symptom resolution, particularly in chronic or neurologically based cases. Clinicians should explain the rationale behind each diagnostic test, including neuroimaging, antibody testing, or motility assessments, to reduce patient anxiety and enhance adherence to evaluation protocols. For patients with binocular diplopia, education should focus on symptom management and prevention of exacerbation. Patients should be instructed on the correct use of prism lenses, occlusive patches, and appropriate head posture techniques to improve functional vision. They should also understand that some interventions, such as surgical correction or botulinum toxin injections, may require repeat procedures or adjustment over time. In monocular diplopia, patients should be educated about refractive correction, lens options, or potential cataract surgery, depending on the etiology. Patients should also be informed about the importance of follow-up evaluations. Diplopia may indicate underlying systemic, vascular, or neurological conditions that require ongoing monitoring. For example, patients over 50 with acute-onset diplopia should be aware of symptoms related to giant cell arteritis, whereas patients with neurological deficits should understand the signs of progression that warrant immediate attention. Education should empower patients to recognize red flags, including sudden visual loss, severe headache, or neurological symptoms, prompting urgent evaluation. In addition to clinical information, patients should be counseled on adaptive strategies to minimize the functional impact of diplopia. These strategies may include environmental modifications, limiting activities requiring fine depth perception until symptoms are managed, and guidance for occupational adjustments. By understanding the complexity of their condition and the reasoning behind treatment recommendations, patients are more likely to comply with interventions and achieve improved outcomes [45].

Enhancing Healthcare Team Outcomes

Management of diplopia requires a coordinated interprofessional approach to ensure accurate diagnosis, appropriate treatment, and optimal patient outcomes. Initial evaluation often occurs in the emergency department or primary care setting, where clinicians, nurses, and allied health professionals identify acute presentations, differentiate between monocular and binocular diplopia, and determine the urgency of referral. Nurses play a pivotal role in initial assessment, documenting visual symptoms, assisting with motility testing, and coordinating imaging studies. Referral to an ophthalmologist, ideally a neuro-ophthalmologist, is critical for definitive diagnosis. The ophthalmologist integrates findings from the history, motility examination, ocular alignment assessment, and imaging to determine etiology and treatment strategies. Collaboration with neurologists, particularly in cases involving cranial nerve palsies, demyelinating disease, or cerebrovascular lesions, ensures comprehensive evaluation. In systemic or autoimmune cases, input from rheumatologists, endocrinologists, or infectious disease specialists may be necessary for targeted therapy. Pharmacists contribute by reviewing and optimizing medication regimens, particularly in cases of myasthenia gravis, inflammatory neuropathies, or systemic therapies affecting ocular function. Occupational therapists and vision rehabilitation specialists support functional adaptation, providing strategies to manage diplopia in daily activities, including reading, driving, and work-related tasks. Radiologists play an essential role in interpreting neuroimaging accurately, identifying aneurysms, masses, demyelinating lesions, or orbital pathologies that may contribute to diplopia. Effective communication among team members ensures timely interventions, prevents duplication of testing, and enhances patient understanding. Structured care pathways, frequent updates on patient progress, and shared decision-making facilitate coordinated management. Interprofessional collaboration improves diagnostic accuracy, reduces delays in treatment, and optimizes long-term functional and visual outcomes. By integrating clinical expertise across multiple disciplines, healthcare teams can provide patient-centered care that addresses both the ocular and systemic aspects of diplopia, ensuring comprehensive management and improved quality of life [46].

Conclusion:

Diplopia represents a multifactorial visual disorder requiring precise differentiation between monocular and binocular causes, as this distinction guides all subsequent diagnostic and therapeutic decisions. While many cases stem from benign microvascular or optical etiologies, others indicate serious neurological, orbital, or systemic disease, necessitating urgent evaluation—particularly when associated with cranial nerve involvement or acute neurological symptoms. Prognosis varies widely based on underlying pathology, with microvascular and inflammatory causes often improving spontaneously, whereas compressive, malignant, or traumatic etiologies demand early intervention to prevent permanent dysfunction. Long-term outcomes are optimized through coordinated interprofessional care integrating ophthalmologists, neurologists, radiologists, and rehabilitation specialists, ensuring comprehensive evaluation and tailored management strategies. Patient education, timely referral, and structured follow-up are essential components of effective management. Ultimately, early recognition and targeted, multidisciplinary intervention remain central to improving visual function, reducing complications, and enhancing quality of life for individuals affected by diplopia.

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