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Review Article

The Overaction of the Inferior Oblique Muscle and Associated Vertical Strabismus: Prevalence, Etiology and Diagnosis

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ABSTRACT

Introduction: Vertical strabismus, commonly caused by the overaction of the inferior oblique (IO) muscle, is a frequent oculomotor disorder. It affects one-third of all patients with strabismus, with 70% of these cases coexisting with esotropia. Currently, there is no universally accepted method for quantifying the magnitude of IO overaction (IOOA), as existing classifications are subjective and may not be entirely appropriate. Standardization of IOOA classification is crucial to better understand its severity, develop effective treatment strategies, and predict surgical outcomes. The mechanism of action of the IO muscle is complex and varies with the globe's position during contraction. Beyond horizontal and vertical movements, the IO muscle also contributes to torsional eye movements, impacting the diagnosis and treatment of vertical strabismus and cyclotropia.

Materials and Methods: The biomechanical properties of the IO muscle and its triple-action characteristics (horizontal, vertical and torsional movements) were analyzed. A review of current diagnostic practices was conducted, emphasizing the need for comprehensive clinical examinations to identify the signs and symptoms of IOOA and determine its severity.

Results: Disorders of the IO muscle often result in vertical strabismus and cyclotropia, complicating strabismus treatment. The variability of the IO muscle's mechanism of action highlights the inadequacy of subjective classification systems. Incorporating the triple-action characteristics of the IO muscle into diagnostic evaluations enables a more accurate understanding of IOOA and its clinical manifestations.

Conclusion: Standardizing the classification of IOOA is essential for accurately assessing its severity, guiding treatment strategies, and predicting surgical outcomes. Comprehensive diagnostic examinations that consider the triple-action nature of the IO muscle are necessary to improve the management and treatment of patients with IOOA.

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Introduction

Strabismus is a binocular vision disorder where one eye deviates from the normal fixation point. The prevalence of strabismus ranges from 0.5 to 5% [1]. Strabismus is more than a cosmetic defect associated with impaired binocular vision and stereopsis. It can negatively impact the patient's self-esteem and interpersonal relationships [2].

Deviation of the eyeball upward in the adduction position is a consequence of the overaction of the inferior oblique muscle [3]. It has been reported in 70% of patients with esotropia and 30% of patients with strabismus [4, 5]. This anomaly can be either unilateral or bilateral. In addition, the inferior oblique overaction (IOOA) can be categorized into primary and secondary deviations [6]. Primary IOOA is most common in children. The prevalence of this form of vertical strabismus is around 72% in cases of infantile esotropia and 30% in cases of acquired strabismus [7]. Among all variations of vertical strabismus, IOOA is observed in 16.7% of cases [7].

Materials and Methods

In the current study, Google Scholar, PubMed and Science Direct search engines were used for finding the related articles using the following keywords: "vertical strabismus," "inferior oblique overaction," "strabismus," and "overelevation." Papers published between 1965 and March 2023 that were relevant to the topic of this review were included. The selected articles and scientific evidence were collected, summarized, categorized, evaluated and concluded upon.

Results

The etiology of primary IOOA is still under debate. However, Duane suggested that excessive innervation of vergences could lead to the development of strabismus [8]. According to his theory, overelevation of the eye in adduction occurs due to an increased innervation of the adductive eye by the inferior oblique (IO) muscle compared to the contralateral superior rectus muscle. In contrast, Liesch and Simonsz hypothesized that ocular overelevation in adduction could be observed in previously healthy eyes after prolonged monocular occlusion [9]. This event may indicate a natural tendency for the eye to lift on adduction, but under normal binocular conditions, these kinds of ocular movements are controlled by fusion [10, 11]. Chavasse considered it reasonable to compare the features of the superior oblique (SO) muscle and the IO muscle. He proposed that the releasing effect of the SO in adduction was less than the lifting effect of the IO, which was explained by their anatomical features [12]. In support of this theory, Berens noted that the IO as an elevator spent 42% of muscle energy, while the SO as a depressor spent only 37% [13]. In 1952, Scobee also supported the theory that IOOA was caused by muscle imbalance [14]. He believed that to take the globe away from the nose, an increased impulse is applied to the IO of the adducted eye and an impulse with the same strength directs to its synergist, ie, the ipsilateral superior rectus muscle. The author also stated that the elevator action of the IO in adduction was greater than the depressor effect of the SO. As a result of the upcoming imbalance, overelevation of the adducted eye develops. Lancaster also agreed with this point of view [15]. The overaction of IO can be explained by the oblique location of the insertion of the medial rectus muscle or by excessive adhesion between the IO and the inferior or lateral rectus muscles. Guibor suggested that the excessive lifting effect of the IO muscle might be due to its synkinesis with the ipsilateral medial rectus muscle due to an impulse distribution in the central nervous system [16].

The secondary overaction of the IO muscle is more straightforward to explain. It is caused by paresis/paralysis of the ipsilateral SO or paresis/paralysis of the contralateral superior rectus muscle when the paretic eye is fixating. In the latter case, overelevation of the globe in adduction is caused by increased innervation transferring to the IO following Hering's law. However, in the first condition, overelevation in adduction is not caused by excessive innervation of the IO, but it is due to the lack of the tonus of the paralyzed SO antagonist; even a normal innervation impulse will be sufficient to overelevate the globe at the time of IO action [17]. Verhoeff believed that IOOA is always associated with paresis of the SO or hypoplasia of the trochlear nucleus [18]. Thus, two main theories exist regarding the development of the secondary IOOA. White and Brown believed that weakness of the superior rectus muscle leads to the development of secondary IOOA 28 times more often than the lesion of the SO [19].

On the contrary, Edward et al. believed that SO paresis is observed more often in secondary IOOA [20]. Adler noted that part of the disagreement might be because some authors consider only primary paresis [21], while others argued that paresis is associated with horizontal strabismus [22]. Adler also found that isolated primary vertical palsy studied by Davis was more often caused by SO paresis. However, the vast majority of vertical paralysis associated with horizontal strabismus studied by White and Brown was caused by weakness of the superior rectus muscle [19, 21].

Discussion

Classification of vertical strabismus due to IOOA

The clinically quantitative classification of the IOOA is based on the assessment of the elevation effect of this muscle [23, 24]. According to the degree of IOOA, the most widely used classification ranges from 0 to +4, corresponding to moving the globe at an angle of 45 degrees. Grade 0 corresponds to normal muscle function with no overelevation in the adductive state. Grade +1 indicates the minimum vertical deviation of the globe in adduction, while +4 corresponds to the maximum elevation of the adducted eye. The grades +2 and +3 are intermediate between these extremes [24]. Given that in the state of adduction at an angle of 45°, the globe can rise within 30°, other correspondences equal to 0-7°, 8-15°, 16-22° and 23-30° [25]. In 2006, Moon and Lee used not only degrees from +1 to +4 but also the corresponding values of hypertropia in PD (0–5, 6–10, 11–15 more than 15 PD) to assess the overaction value of the IO muscle, respectively [26]. According to the classification proposed by Kelkar et al. in 2015, grades 1+, 2+, 3+ and 4+ of IOOA approximately correspond to 1-15, 16-30, 31-45, and 46-60 PD hypertropia during globe adduction [27]. Some authors converted the degree of IOOA from 1+, 2+, 3+ and 4+ into 5, 10, 15 and 20 PD hypertropia when fixing into the distance, respectively.

There is no single approach for defining IOOA by degrees and the most common classifications are highly subjective and inappropriate. There is no unified classification of IOOA in the position of the adduction when we intend to determine the angle of deviation by the Hirshberg method. Due to the need to understand the severity of the disease in selecting treatment strategies and predicting the results of surgical intervention, it is necessary to carry out standardization in the classification of the IOOA.

Examination methods of vertical strabismus caused by IOOA

The anatomy of the IO is somewhat atypical compared to other extraocular muscles. The IO originates at the lower inner edge of the orbit (in the area of the entrance of the lacrimal canal) and is attached to the posterolateral part of the globe near the lower border of the lateral rectus muscle, passing between the wall of the orbit and the inferior rectus muscle 16 mm from the limbus.

The IO is the shortest extraocular muscle, with a length of only 37 mm. The width of the attachment site varies widely, ranging from 5 to 14 mm, on average about 9 mm. The attachment site forms a concave line. The anterior point of attachment is 10 mm from the attachment of the external rectus muscle; its posterior point is 1 mm lower and 1 to 2 mm lateral to the macula. Unlike other extraocular muscles, which have both muscular and tendon components, the IO is almost entirely muscular, with an average tendon length of 1 to 2 mm [28].

The mechanism of action of extraocular muscles is very complex and depends on the position of the globe at the time of their contraction. Clinically, the muscle's main (primary) action is evaluated, while the primary position of the eye and minor actions (secondary and tertiary) depend on the position of the globe. The primary action of IO is excycloduction. Moreover, elevation and abduction of the eye are minor actions for IO. Any impairment of the IO muscle leads to the development of cyclo-vertical strabismus, which is also the cause of the formation of torsion diplopia and torticollis, which reduces the effectiveness of strabismus treatment. IOOA generally leads to the development of the V-pattern strabismus [29]. To identify the entire symptom complex associated with IOOA, practitioners should consider the complexity of its triple action. From the clinical perspective, conducting a comprehensive diagnostic study is necessary. Simultaneously, to identify the IOOA, the displacement of the globe in 9 gaze positions is assessed [30].

An accurate differential diagnosis for the target muscle in cases of IOOA is critical. In particular, with the development of endocrine myopathy, frequent involvement of the inferior rectus muscles in the inflammatory process can also affect the IO muscle. This interaction may be related to the inferior anatomy of the orbit, as the inferior rectus and the IO are the only muscles in direct contact with each other. As a result, any inflammatory process will result in fibrosis of these two muscles and Lockwood's ligament. However, endocrine ophthalmopathy is a complex process in which surgical treatment of strabismus is performed only in cases of failure of pathologically oriented therapy [31].

Measurement of the magnitude of overaction of the IO muscle

The Hirschberg method, based on the observation of the position of the light reflex of the ophthalmoscope on the patient's cornea, is widely used to assess the deviation of the eyes [17]. The method was proposed by Hirschberg in 1886. While holding a candle 12 inches

from the patient, the examiner observes the reflex's position on the deviant eye's cornea. Then, the examiner expresses the results of observations in degrees. Hirschberg found that a 1 mm decentration of the corneal reflex corresponds to a 7° deviation relative to the visual axis of the examined eye [32]. At present, the angle of deviation is measured through the alternate cover test, with the occlusion of each eye separately while fixing an object located at a distance of 33 cm. Deviation and ocular motility are assessed in 9 gaze positions [33]. Another less commonly objective method for determining the magnitude of IOOA is achieving the absence of adjusting eye movements during the cover test and simultaneous deviation correction with the prism. In this case, the strabismus angle is corrected using loose prisms from the trial lens, Risley prisms, plastic Fresnel prisms, or prism bars. The study is carried out as long as eye movements are observed, and the angle of deviation is considered equal to the strength of the prism necessary to neutralize it. The magnitude of hypertropia, in this case, is expressed in PD. For the convenience of using various methods for studying the magnitude of the deviation between PD and Hirschberg degrees, the following relationship was found: 1°=2 PD [29, 33].

V-pattern syndrome

IOOA is often associated with V-pattern syndrome, regardless of whether there is a deviation in the primary gaze position or if IOOA is combined with esotropia or exotropia [34]. To diagnose V-patterns syndrome, when examining a patient, it is necessary to identify the difference in the magnitude of the horizontal deviation when changing the gaze from the upward toward the downward position when looking at an object at a distance of 33 cm. In cases where the deviation angle after changing the fixation decreases by more than 15 PD, or 10° indicates the presence of V-pattern syndrome [35].

Head tilt test

The head tilt test (Bielschowsky test) was first described by the German ophthalmologist Alfred Bielschowsky in 1914. The test detects changes in vertical deviation when the head is tilted to the right or left shoulder in paralysis of the vertical rectus and oblique extraocular muscles [36]. The author gave a physiological justification for the method. When the head is tilted towards the shoulder, endolymph movement occurs in the otolithic apparatus (semicircular canals of the labyrinth), as a result of which an impulse to contract is given to the corresponding groups of extraocular muscles [17]. Given the condition of normal muscle response, this mechanism allows the individuals to maintain the correct position of the eyes without disturbing binocularity.

In cases of IOOA, the Bielschowsky test allows differentiating between primary and secondary overaction, in which this phenomenon is considered positive and consists of the appearance and or increase in hypertropia when the head is tilted towards the affected eye [37].

Compensatory head position

Forced tilting of the head in the form of ocular torticollis is characteristic of most patients with paretic, paralytic strabismus. The mechanism of its occurrence lies in the fact that when the gaze is shifted towards the action of the affected muscle, diplopia increases, and when tilting the head, it becomes possible to avoid the field of action of the paretic muscle [38]. This condition avoids double vision and often preserves binocular vision.

In cases where torticollis does not provide fusion, patients turn or tilt their heads to increase the distance between "double" images or use their nose as an occluder. It is essential to differentiate between true torticollis and ocular torticollis. True torticollis is caused by pathology of the cervical vertebrae or fibrosis of the sternocleidomastoid muscle [39]. However, treatments specific to torticollis cannot correct ocular torticollis.

Secondary IOOA caused by paresis or paralysis of the SO is characterized by a tilt of the head to the opposite shoulder with the chin-down [40, 41]. It should be borne in mind that the degree of "correction" of the position of the eyes by tilting the head is much less than the degree of tilting the head. Long-term ocular torticollis contributes to the formation of facial asymmetry, which is an important clinical sign for differentiating congenital and acquired IOOA [42-46].

Evaluation of cyclodeviation

In addition to horizontal and vertical movements, IO muscles provide excyclotorsion for eye movements [47]. The occurrence of IOOA leads to the development of excyclodeviation. Patients with congenital or long-term IOOA have no complaints of torsional diplopia. This condition is due to adaptive compensatory mechanisms, which include ocular torticollis, compensation with cyclo-fusion reserves, and physiological suppression of one of the two monocular images. The value of physiological excycloduction does not exceed 12° and its large values are excessive and confirm the presence of muscle overaction [48].

Cyclotropia, when its magnitude exceeds the cyclofusion reserves, can prevent the development of fusion and stereopsis and compromise the functional results of strabismus treatment [49]. Therefore, along with a standard ophthalmological examination, evaluating cyclotorsion movements is critically important in diagnosing IOOA.

Cyclotropia assessment can be qualitative or quantitative. There are several ways to determine the presence of cyclotorsion displacement of the globe qualitatively [50-52]:

1) During eye movement examinations or performing a cover test, the presence of intorsion or extorsion is determined by observing the displacement of a large conjunctival vessel at the limbus; 2) During ophthalmoscopy and anatomical observation of the optic nerve head (ONH) level in relation to the macula, the macula lies 4 mm temporal to the ONH and 0.8 mm below the horizontal line. Thus, if the ONH is obviously above or below the macula, we can conclude a cyclotorsion displacement (displacement of the ONH above or below the fovea indicates the presence of excyclodeviation or incyclodeviation, respectively); 3) When using double Maddox's rod test; 4) Photographing the fundus can determine the relationship between the optic disc and the fovea.

Quantifying the cyclodeviation is necessary to confirm the diagnosis and evaluate the effectiveness of surgical intervention for disorders of the IO muscle. The subjective and objective assessments determine not only the position but also the degree of displacement of the macula relative to the optic disc. A subjective quantitative assessment can be made on a scale of a trial lens using a Maddox rod. The rotation of the perceived image represents cyclodeviation and can be compensated by lens rotation until the patient perceives the alignment of double images. However, this technique is difficult to implement in pediatrics. The most reliable methods for determining cyclodeviation in children do not require a patient response. These methods include the method of photographic recordings of images of markers in the fundus of both eyes during examination using a fundus camera, which allows for determining the position of the macula [53].

Conclusion

Any disorders of the IO muscle contribute to developing not only vertical strabismus but also cyclotropia, which may limit the efficacy of strabismus treatment. During the evaluation of patients with IOOA, it is necessary to conduct a complete diagnostic examination and assess torsional eye movements. In childhood, some diagnostic techniques are impossible since they require a conscious response from the patient. This limitation is the main reason for performing a complete diagnostic evaluation of the IOOA. It is necessary to develop new diagnostic procedures to overcome clinical shortcomings. To identify all the clinical signs and symptoms that result from IOOA, practitioners should consider the characteristics of its triple action. Therefore, it is necessary to conduct comprehensive diagnostic examinations on patients with IOOA.

Ethical Considerations

Compliance with ethical guidelines

There were no ethical considerations to be considered in this research.

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Authors' contributions

All authors equally contributed to preparing this article.

Conflict of interest

The authors declared no conflict of interest.

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