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In-depth Analysis of the Pathogenesis and Research Progress in Cutting-edge Treatment of Type III Acute Acquired Comitant Esotropia

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Abstract: Type III acute acquired comitant esotropia (AACE) is a special type of binocular coordination disorder with sudden onset characteristics, but its pathogenesis and treatment strategies remain unclear. This article analyzes the incidence and classification characteristics of Type III AACE, and explores its pathogenesis from multiple perspectives including clinical medicine, neuroscience, and neuro-ophthalmology. It is found that this disease is associated with factors such as decompensation of phoria, dysfunction of the visual center, abnormalities in the physiological and anatomical structure of extraocular muscles, accommodative factors, and disorders of convergence and divergence. The prognosis of Type III AACE is excellent with treatments such as botulinum toxin injection and surgery, and adjunctive therapies like prism adaptation test and visual function training can enhance the management effect. This article provides an in-depth analysis of the pathogenesis and cutting-edge treatment techniques for patients with Type III AACE, which can offer guidance for the subsequent diagnosis and treatment of such patients.

Keywords: Type III acute acquired comitant esotropia; Treatment progress; Pathogenesis

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1. Introduction

Acute acquired comitant esotropia (AACE) is a special type of esotropia, often characterized by sudden onset of esotropia, which may be accompanied by diplopia. The diplopia presents with horizontal ipsilateral features. Patients have the same angle of deviation when gazing in different directions, without symptoms such as ocular movement disorders or extraocular muscle paralysis, but there is a risk of potential binocular single vision dysfunction. Type III AACE may initially present with intermittent eye position deviation and diplopia. Most patients experience binocular single vision when viewing near objects and diplopia when viewing distant objects, while a few patients have diplopia issues when viewing both near and distant objects. Type III AACE does not have self-limiting properties. For a few patients with mild strabismus, reducing the amount of time spent on

electronic devices or avoiding close work for extended periods can decrease the degree of strabismus and alleviate diplopia symptoms. Additionally, some patients with Type III AACE may not show significant signs or symptoms at the onset of the disease, leading to a delay in initial diagnosis that may span months or even years. This study provides a deep interpretation of the pathogenesis of Type III AACE and analyzes cutting-edge treatment options to guide future clinical diagnosis and treatment.

2. Incidence and classification

In the context of changing reading habits, the susceptible population for AACE has increased. Coupled with the prolonged home office hours for adults and extended learning time on electronic devices for children during the pandemic, the time spent on close-up eye use has increased, leading to a rise in the incidence of Type III AACE, especially in intermittent esotropia with small angles and myopia [1]. Based on Burian and Miller's research on AACE classification [2], it can be divided into the following types:

(1) Swan type AACE, which commonly occurs in infants and children, with or without hyperopia, and can be triggered by monocular vision loss or monocular patching that disrupts binocular fusion.

(2) Burian-Franceschetti type AACE, which is prevalent in children and adolescents, may be associated with mild hyperopia, and has a large esotropia angle, often related to psychological stress and physical fatigue.

(3) Bielschowsky type AACE, which commonly affects adults and older children, is associated with varying degrees of myopia.

Additionally, some patients may present with complications such as optic disc edema or nystagmus, or experience failure to restore binocular vision after treatment, or recurrent AACE after treatment, requiring vigilance for intracranial pathologies [3].

3. Pathogenesis

3.1. Decompensation of phoria

Before the onset of Type III AACE, patients may experience intermittent binocular diplopia, which progresses to persistent diplopia as the disease advances. In the initial stage of phoria, it relies on fusion reserve for compensation. However, under the influence of multiple factors such as aging, increased physical and psychological stress, and illnesses, the degree of phoria increases while the fusion reserve decreases. If the phoria cannot be compensated, it may suddenly transition into manifest esotropia.

3.2. Dysfunction of visual cortex

Prolonged exposure to screens such as mobile phones and computers late at night can continuously stimulate the visual cortex, disrupting biological rhythms and impairing brain function. Acute circadian rhythm disorders and reduced sleep can lead to uncoordinated eye movements, triggering AACE. fMRI technology enables qualitative analysis of functional brain regions, exhibiting advantages in displaying fine structures. It assists doctors in understanding the composition of visual information processed by the cerebral cortex and provides feedback on the neural mechanisms underlying visual formation. In a study conducted by Wang *et al.*, fMRI technology was used to aid in the diagnosis of AACE patients [4]. The results revealed that AACE patients had higher activation intensity in the middle frontal gyrus and right lingual gyrus compared to healthy individuals, while activation

intensity in other regions was lower. Future research can further explore the application of fMRI technology to assist doctors in analyzing the etiology of Type III AACE and assess changes in the visual cortex of patients.

3.3. Abnormal physiological and anatomical structure of extraocular muscles

Tightness of the medial rectus muscle and relaxation of the lateral rectus muscle can both induce Type III AACE. Prolonged close-up eye use in daily life, combined with biochemical and mechanical stimuli, can lead to skeletal muscle pathologies, resulting in disordered muscle strength of the medial and lateral rectus muscles, ultimately manifesting as eye position deviation. Additionally, anterior displacement of the insertion of the medial rectus muscle can cause the converging power of both eyes to be stronger than the diverging ability, triggering AACE. Especially for those who frequently engage in close-up vision tasks, multiple factors can strengthen the medial rectus muscles of both eyes, leading to a disturbance in the balance between convergence and divergence.

3.4. Accommodative factors

Most patients with Type III AACE suffer from myopia, and those with a higher degree of myopia tend to have more lagged accommodative ability compared to those with lower myopia or non-myopic individuals. Furthermore, people with myopia often have poor eye usage habits. The closer the viewing distance, the more severe the accommodation lag becomes. Under the influence of accommodation lag, hyperopic defocus may occur, causing the patient to enter a hyperopic state and leading to increased convergence. However, it's important to note that presbyopic and pseudophakic individuals, who have no accommodative ability, may also develop esotropia.

3.5. Convergence and divergence disorders

In a study by Huang *et al.*, it was found that over 70.51% of patients with Type III AACE spent more than 7 hours per day on close-up eye use [5]. According to a study by Zheng *et al.*, long-term viewing of electronic screens is associated with a higher risk of developing video terminal syndrome, which can induce ocular discomfort and extraocular symptoms, including dry eyes, blurred vision, and musculoskeletal disorders [6]. As the course of video terminal syndrome progresses, it can impair ocular accommodative ability and trigger diplopia. Evidently, excessive close-up eye use is a high-risk factor for the development of Type III AACE, leading to increased muscle strength of the medial rectus and stimulating spasms, thereby disrupting the balance between divergence and convergence of both eyes. Additionally, electronic displays possess unique characteristics, especially when viewing 3D displays. To achieve a stereoscopic visual effect, crossover and non-crossover parallax techniques are employed, which can cause fatigue in both eyes more easily. During crossover parallax viewing, images formed on the screen induce binocular convergence, but the accommodation focus of both eyes remains on the screen, resulting in disordered binocular convergence and accommodation, thereby exacerbating ocular discomfort and fatigue. Therefore, prolonged viewing of electronic screens, coupled with disordered convergence and divergence, can further disrupt binocular fusion function and increase the risk of diplopia.

4. Treatment methods

4.1. Botulinum toxin treatment for Type III AACE

Some patients with Type III AACE exhibit large deviation angles, and conventional conservative treatments may not be effective. In such cases, botulinum toxin type A can be injected into the extraocular muscles to enhance the

management of Type III AACE. Botulinum toxin is a neurotoxin that acts on cholinergic motor nerve endings. It antagonizes calcium ions and blocks the release of acetylcholine from nerve endings, preventing muscle fibers from contracting and, thus paralyzing the muscles. The injected toxin can maintain its efficacy for up to three months. Stimulated by its active ingredients, binocular single vision function can be rebuilt, reducing the risk of strabismus recurrence after discontinuation.

In a study by Zhang *et al.*, patients with AACE who had onset within six months were treated with botulinum toxin injections, while those with onset exceeding six months underwent extraocular muscle transposition surgery [7]. The results indicated that both botulinum toxin injections and extraocular muscle transposition surgery were effective treatments for AACE, with significant outcomes. Botulinum toxin injection can be considered as a preferred treatment option for early-stage AACE patients.

In a study by Cui *et al.*, eleven AACE patients were treated with botulinum toxin type A injections and followed up for half a year [8]. The results showed that diplopia symptoms resolved within 1 to 2 months after injection, and there was no recurrence during the follow-up period. Shi's study found that surgery and botulinum toxin treatment had similar effects on AACE, with low recurrence rates for both [9]. Compared to surgical methods, botulinum toxin injection is easier to perform, reduces medical expenses, and shortens anesthesia time, providing a new approach for the treatment of Type III AACE.

4.2. Prism treatment for Type III AACE

Type III AACE often presents with the issue of "eating prisms." Conventional prism treatment based on past experience may increase the risk of under correction. Prism adaptation tests can improve the success rate of treatment for patients with Type III AACE, but the treatment cycle is long. Additionally, some patients have large strabismus angles, making it impractical to wear prisms continuously. However, prisms can be used for preoperative and postoperative correction in patients with Type III AACE to improve visual acuity, near stereoscopic sensitivity, and restore binocular visual function. In a study by Zhou *et al.*, 47 patients undergoing bilateral lateral rectus muscle recession surgery were subjected to prism adaptation tests [10]. The results indicated that prism tests can enhance surgical safety for AACE patients. Therefore, for AACE patients undergoing surgery, it is recommended to perform prism adaptation tests to ensure surgical safety.

4.3. Surgical treatment for Type III AACE

It is recommended that surgical treatment for Type III AACE patients be performed after six months of onset. Early treatment, when the strabismus angle is unstable, can increase surgical risks. In a study by Qiang *et al.*, patients with AACE were treated with unilateral medial rectus muscle recession and bilateral medial rectus muscle recession [11]. The results showed that unilateral medial rectus muscle recession could improve the orthophoria rate and optimize visual function in AACE patients. In a study by Xu *et al.*, modified Parks incision combined with rectus muscle folding surgery was used to treat AACE. This approach could restore the tear film function, enhance surgical effects, reduce postoperative pain, and improve ocular aesthetics [12]. In a study by Chao *et al.*, rectus muscle marginal myotomy was used to treat AACE, which could optimize binocular visual function, reduce surgical complications, and demonstrate high safety [13].

5. Visual function training

Visual function training, combined with surgical treatment, can assist patients in rebuilding visual function and reducing the risk of AACE recurrence. In a study by Wu *et al.*, the first stage of training focused on expanding the negative fusion range at near distances and optimizing monocular accommodation amplitude, using methods such as polarized vector diagrams and lens reading [14]. The second stage centered on expanding the negative fusion range at intermediate and far distances and improving monocular flexibility, utilizing tools like stereoscopes and flippers. Personalized visual function training can alleviate AACE symptoms and enhance binocular visual function.

6. Conclusion

The incidence of Type III AACE is increasing year by year, and treatments such as botulinum toxin injections and surgery are commonly used to improve patient outcomes. However, many patients with Type III AACE also suffer from myopia, which can affect the onset and progression of the condition through indirect and direct pathways. Additionally, long-term poor eye usage habits can alter brain region function, affecting binocular visual and motor functions. Therefore, future research should focus on exploring the impact of myopia on visual pathways and visual centers. Currently, most studies on the pathogenesis and treatment of Type III AACE by scholars are retrospective analyses. Prospective studies can be actively conducted in the future to deeply analyze the pathogenesis and cutting-edge treatment options for Type III AACE patients, aiming to improve their prognosis.

Disclosure statement

The author declares no conflict of interest.

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