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REVIEW ARTICLE

Neurological pathologies in acute acquired comitant esotropia

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Abstract

Background Acute acquired comitant esotropia (AACE) is an uncommon subtype of esotropia characterized by sudden and usually late onset of a relatively large angle of comitant esotropia with diplopia in older children and adults.

Methods A literature survey regarding neurological pathologies in AACE was conducted using databases (PubMed, MEDLINE, EMBASE, BioMed Central, the Cochrane Library, and Web of Science) in order to collect data for a narrative review of published reports and available literature.

Results The results of the literature survey were analyzed to provide an overview of the current knowledge of neurological pathologies in AACE. The results revealed that AACE with unclear etiologies can occur in many cases in both children and adults. Functional etiological factors for AACE were found to be due to many reasons, such as functional accommodative spasm, the excessive near work use of mobile phones/smartphones, and other digital screens. In addition, AACE was found to be associated with neurological disorders, such as astrocytoma of the corpus callosum, medulloblastoma, tumors of the brain stem or cerebellum, Arnold-Chiari malformation, cerebellar astrocytoma, Chiari 1 malformation, idiopathic intracranial hypertension, pontine glioma, cerebellar ataxia, thalamic lesions, myasthenia gravis, certain types of seizures, and hydrocephalus.

Conclusions Previously reported cases of AACE with unknown etiologies have been reported in both children and adults. However, AACE can be associated with neurological disorders that require neuroimaging probes. The author recommends that clinicians should perform comprehensive neurological assessments to rule out neurological pathologies in AACE, especially in the presence of nystagmus or abnormal ocular and neurological indications (e.g., headache, cerebellar imbalance, weakness, nystagmus, papilloedema, clumsiness, and poor motor coordination).

Key messages

What is known

- Previously reported cases of acute acquired comitant esotropia (AACE) with unknown etiologies have been reported in both children and adults.
- AACE can also be associated with neurological disorders.

What is new

- Functional etiological factors for AACE have recently been rising for many reasons, such as the excessive near work use of mobile phones/smartphones and other digital screens. This rise has also been witnessed during the COVID-19 pandemic when people were forced to stay at home and work from home through digital devices.
- AACE can be associated with neurological disorders, such as astrocytoma of the corpus callosum, medulloblastoma, tumors of the brain stem or cerebellum, Arnold-Chiari malformation, cerebellar astrocytoma, Chiari 1 malformation, idiopathic intracranial hypertension, pontine glioma, cerebellar ataxia, thalamic lesions, myasthenia gravis, certain types of seizures, and hydrocephalus.

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Keywords Acute acquired comitant esotropia · Strabismus · Neurological pathologies · Intracranial tumors · Arnold-Chiari malformation · Hydrocephalus

Introduction

Acute acquired comitant esotropia (AACE) is an uncommon subtype of esotropia characterized by sudden and usually late onset of a relatively large angle of comitant esotropia with diplopia in older children and adults [1–13]. However, it can rarely occur at any time after the age of 6 months [8].

AACE with unclear etiologies has been reported in many studies [1, 2, 6, 8, 14–18]. Functional etiological factors for AACE can be due to many reasons, such as functional accommodative spasm [8, 19], the excessive near work use of mobile phones/smartphones, and other digital screens [5, 11, 12, 20–24]. However, other studies in patients with AACE have found neurological pathologies, such as astrocytoma of the corpus callosum, medulloblastoma, tumors of the brain stem or cerebellum, Arnold-Chiari malformation, cerebellar astrocytoma, Chiari 1 malformation, idiopathic intracranial hypertension, pontine glioma, and cerebellar ataxia [6, 19, 25–36]. Other studies have also reported an association between AACE and serious disorders, such as brain tumors, Arnold-Chiari syndrome, hydrocephalus, thalamic lesions, myasthenia gravis, and certain types of seizures [27, 30, 37–40].

AACE has also been reported in monozygous twins [16]. There have been reports of acute comitant esotropia at ages 6–9 years in four siblings [14]. Eight patients aged 6.5–72 years with esotropia and associated diplopia, of which one had a preceding illness, have been reported [1]. None of these patients had a neurological pathology [1]. Idiopathic intracranial hypertension has also been reported as an infrequent cause of comitant esotropia [35].

It is important to remember that concomitancy in acute onset esotropia does not exclude an underlying serious neurological disease [6, 9, 10, 27].

The neurological processes underlying comitant esotropia consist of hydrocephalus, bilateral disorder of the sixth cranial nerves, and injury to the vergence center in the brainstem, which are usually associated with nystagmus [38]. Injuries to supranuclear mesencephalic configurations that are in command of vergence eye movement, as well as infranuclear pathologies have also been proposed [38, 41].

In general, although previously reported cases of AACE with unknown etiologies have been reported in both children and adults, AACE can be associated with neurological disorders and this article discusses these potential neurological pathologies in AACE. Therefore, the purpose of this study was to provide an overview of the current knowledge of neurological pathologies in AACE based on the available literature.

Method

A literature survey regarding neurological pathologies in AACE was conducted using databases (PubMed, MEDLINE, EMBASE, BioMed Central, the Cochrane Library, and Web of Science) in order to collect data for a narrative review of published reports and available literature. Specific keywords (Acute acquired comitant esotropia, Strabismus, Neurological pathologies, Intracranial tumors, Arnold-Chiari malformation, and Hydrocephalus) were applied to search and evaluate potential articles in English language, regardless of the date of study. AACE was defined as a subtype of esotropia characterized by sudden onset of comitant esotropia of more than 15 prism diopters in patients who were older than the age of one year. However, it can rarely occur at any time after the age of 6 months, according to reports. Therefore, few old studies that reported early onset cases of AACE were also included, from which available data were collected.

The diagnosis of AACE was based on an acute onset of comitant esotropia with a deviation difference of less than 5 prism diopters (PD) in all gaze directions, evidence of the absence of strabismus before esotropia onset, and normal gaze movement and diplopia when indicated. The exclusion criteria included history of ocular surgery (except for refractive surgery), paralysis, ocular trauma, accommodative spasm, and accommodative strabismus (hyperopia ≥ +2.00 diopters, D) with resolution of deviation with full hyperopic correction.

Intracranial tumors

Acute onset comitant esotropia may be associated with brain tumors [25, 26, 42–47]. Although the type of brain tumor or location within the brain is unclear in many patients in reports, it is vital to review these reports to create a better framework for patients with unusual presentations [38]. This is due to the fact that more than one cause may be responsible for this diverse group of patients [38]. AACE can be linked to tumors in the cerebellum, brainstem, pituitary region, and corpus callosum [6, 38].

Several brain tumors (e.g., cerebellar astrocytomas, medulloblastomas, pontine gliomas, astrocytoma of the corpus callosum with hydrocephalus) have been reported to be associated with AACE in children with no neurological signs, and occasionally, acute-onset nonaccommodative esotropia was the only presenting manifestation [26, 27, 38, 41,

48]. In one study, 40% of children with acquired esodeviation have been found to have a particular neurologic injury and comitant esodeviation [49].

Medulloblastoma has also been reported in a 10-year-old patient with AACE [26]. The patient had negative neurological and neuroradiological findings on the first visit [26]. However, the diagnosis of the brain tumor was made 28 months after the onset of diplopia [26]. Unfortunately, this was the case when the diagnosis of the brain tumor may have initially been missed, but this is not certain.

AACE has been reported in six children with tumors of the brain stem or cerebellum [27]. None of them had manifestations of abducens nerve dysfunction [27]. AACE in twelve children was associated with neurological injuries [49]. Brain tumors, meningitis, and a basilar artery aneurysm as well as associated thrombosis were confirmed in these children [49].

Papilloedema in a 6-year-old patient with acute onset comitant esotropia, along with normal ductions and versions, was found to be associated with hydrocephalus caused by a cystic lesion in the corpus callosum and an astrocytoma of the corpus callosum [25]. In this patient, esotropia was suddenly resolved before surgery and radiotherapy [25].

In another case of an eleven-year-old patient with acute onset comitant esotropia, facial and abducens nerve palsies were found six weeks after strabismus presentation [44]. Then, the diagnosis of pontine glioma was confirmed using neuroradiographic probes [44]. This case is very educational case, as the patient's initial comitant esotropia was an early indication of the abducens nerve palsy that eventually became apparent [38, 50].

However, the paretic etiological reason is not responsible for all cases of acute onset comitant esotropia associated with a brain tumor [38]. For example, no neurological manifestations were observed in a 10-year-old patient with comitant constant esotropia when first examined [26], but the patient presented with posterior fossa dysfunction 28 months later, and was subsequently diagnosed with medulloblastoma involving the cerebellum. However, over this long observation period, the patient's esotropia did not become inconcomitant, and no other indications of paresis of the abducens nerve were observed [26].

Although the mechanism responsible for acute comitant esotropia in patients with brain tumors is unclear, comitant esotropia may have been caused by injury to supranuclear mesencephalic configurations, which are responsible for vergence eye movements [8, 51]. Infranuclear injuries, such as varying degrees of bilateral sixth nerve paresis, may be responsible for acquired comitant esotropia [8, 51].

Hydrocephalus

Hydrocephalus can occur for many reasons, such as subarachnoid hemorrhage, blood clots in the brain, meningitis, head injuries, stroke, and brain tumors [8]. Hydrocephalus, and not the brain tumor itself, may induce esotropia in patients with AACE [52–54]. A common link between comitant esotropia, as well as inconcomitant esotropia, with the hydrocephalic patient has been reported [52]. A case report described a 3-month-old infant with intermittent hydrocephalus [55]. This patient was shown to have a chronic downward deviation of the eyes and acute onset comitant esotropia whenever the intraventricular pressure increased [55]. They reported that the deviations in this infant completely resolved with the re-establishment of normal intraventricular pressure. Furthermore, abducens paresis was never observed in this infant [55]. Therefore, the frequently detected comitant esotropia in these patients does not appear to be indicative of abducens nerve dysfunction [38]. Although AACE rarely occurs at such an early age, and some old reports cannot be verified fully, this case was only presented to highlight the important effect of intraventricular pressure on eye alignment.

Co-existing hydrocephalus has been accounted to the involvement of acute onset comitant esotropia in Arnold-Chiari syndrome [53, 56, 57]. Considering the long-term comitancy and high occurrence of an A rather than a V pattern, esotropia in this circumstance is unlikely to be primarily due to abducens nerve dysfunction [38, 54]. Therefore, the link between hydrocephalus and esotropia does not support the simplified concept that all cases stand for abducens nerve dysfunction [38].

However, many patients with brain tumors and acute onset comitant esotropia have no indication of either abducens nerve paresis or hydrocephalus [38]. For example, one study found that only two of six cases with brain tumor and acute onset comitant esotropia showed elevated intracranial pressure [27]. None of these six patients showed indications of abducens nerve dysfunction [27]. Remarkably, three of these six cases had bilateral abduction nystagmus [27]. In this regard, seven of eight patients with posterior fossa tumors and acute onset comitant esotropia had abduction nystagmus, and six had asymmetric optokinetic nystagmus [47]. Brainstem or cerebellar dysfunction alone could be responsible for comitant esotropia in these patients [38, 47]. The vergence system in the mesencephalon may also be responsible for the origin of comitant esotropia, even in infantile esotropia [38, 54]. As the exact pathogenesis of acute onset comitant esotropia observed in some patients with brain tumors needs to be discovered, it is obvious that neither abducens nerve dysfunction nor hydrocephalus alone can be responsible for all of the cases, based on the above-mentioned reports and comments.

Having said that the possible presence of a prior sixth nerve paresis that has partially been recovered by the time the patients presented should also be considered in some cases with AACE [6, 8].

From a practical point of view, associated nystagmus of any kind, especially nystagmus in abduction, can be a warning indication that necessitates further neurological evaluations [27, 38, 45, 47].

Arnold-Chiari malformation

Arnold-Chiari malformation has been reported to be associated with AACE [51]. Reports revealed that Arnold-Chiari malformation was associated with esotropia [30, 32]. Divergence palsy due to brainstem dysfunction may be the cause of esodeviation in another case report of two patients with craniocervical junction anomaly [28]. In one study [58], two patients with AACE were identified as having Chiari I malformation, which highlights the importance of a complete neurological assessment and neuroimaging in this group of patients, particularly when they are coexisted with neurological indications, such as headache, syncope, or papilledema [58].

Another report [59] found an 11-year-old girl with Arnold-Chiari malformation who was initially identified at age 10, with an esodeviation of 35 and 30 prism diopters for distance and near fixations, correspondingly, and gaze-evoked nystagmus [59]. Although normal ocular alignment was achieved after surgical treatment in this patient, binocular function failed to work [59].

Coexisting hydrocephalus was also found to be responsible for acute onset comitant esotropia in patients with Arnold-Chiari syndrome [38].

Cerebellar ataxia

Seven adult cases of cerebellar ataxia and esotropia have been reported [36]. Cerebellar esotropia may have been caused by extreme convergence tonicity, a supranuclear incident that may result from disorders of the central vestibular system [6].

Ocular myasthenia and AACE

The diagnosis of ocular myasthenia is based on a history of changeable weakness, with findings that agree with fatigue for the duration of the examination [8]. The clinical diagnosis of ocular myasthenia can be validated in several ways, including clinical examination (sleep, stare, ice, and Cogan tests), serum examination (anti-acetylcholine antibody test), pharmacological tests (e.g., edrophonium and neostigmine tests), and electrophysiological tests [8]. Therefore, it is important to rule out the presence of ocular myasthenia in patients with AACE before deciding for strabismus surgery [8].

Etiology of AACE in Asian populations may be different

Many studies on patients with AACE have been performed in Caucasian populations [2, 15, 16, 18, 60]. However, few studies have examined AACE in Asian populations [4, 11, 19, 59, 61]. In fact, there are differences between Caucasian and Asian populations in their eyes. For example, a higher incidence of myopia and glaucoma among Asians has been reported [62–65].

There are not many studies on patients with AACE in different races. Comparative research is also unavailable at this time. However, since there is a higher incidence of myopia in the Asian population, and the most common type of AACE in the Asian population is associated with myopia [4, 11, 59, 61], the author proposes that race may be an important factor to consider.

Risk factors for AACE with intracranial pathology

In some patients, comitant esotropia can be the only indication of neurological pathology for the length of time before other neurological indications progress (e.g., ataxia and sixth and seventh nerve paresis) [66]. Therefore, several attempts have been made in cases of AACE to characterize particular risk factors that can be used for screening to recognize the presence of potential intracranial diseases, and then supply a source for referral to brain imaging probes [66]. In this regard, children with intracranial pathologies were found to be considerably older at onset than children without intracranial pathologies, with a mean age of 7.5 years versus 3.8 years [66]. Other studies have also shown that the mean age of patients with AACE and intracranial pathologies was 7.1 years (range, 3–13 years) [17–19, 25, 26, 37, 38].

In one study [66], only one of three children (33%) with intracranial pathologies was reported to have an indication of neurological disease at acute presentation, which was papilledema [66]. Since the majority of recorded patients with brain disease and AACE had posterior fossa lesions or elevated intracranial pressure, the assessment of papilledema is imperative. Nevertheless, in this study [66], papilledema was not observed in 2/3 of the cases with intracranial pathology.

A greater angle of deviation at distance (>40%) was reported to be a considerable risk factor for the presence of intracranial pathology [66]. This indication should therefore be used when deciding to refer for further brain probes [66]. The risk factors for intracranial pathologies in children are not similar to those in adults. For example, a larger esotropic deviation at distance, which was a significant risk factor for intracranial pathology in this study [66], was associated with intracranial disease in 93% of children compared with 23% in adults in another study [67].

Oblique inferior muscle overaction with a small V pattern and oblique superior muscle overaction with an A pattern have been proposed for the potential presence of intracranial

pathologies, including intracranial tumors, Arnold-Chiari malformation, and hydrocephalus [27, 38, 68]. However, overaction of the oblique inferior muscle in patients with intracranial tumors was not a considerable risk factor in another study [66].

Nystagmus has been reported in patients with Chiari malformation type 1, hydrocephalus, and brain tumors, and thus may suggest subclinical paresis of the abducens nerve [69]. On the other hand, gaze-evoked nystagmus was not a considerable risk factor in one study [66].

In addition, the recurrence of AACE in hyperopic children was found to be another imperative risk factor for intracranial pathologies [66]. Therefore, hyperopic patients with AACE may initially appear to be managed by maximum plus lenses; however, esotropia may recur and/or other neurological indications may develop later [66]. For this reason, only if fusion can be demonstrated with plus lenses with full responses to spectacles, and in the absence of other risk factors and/or in the presence of causative occlusion, the probability of an underlying intracranial tumor can be minimized [9, 10, 66]. Therefore, it is important to further investigate patients with AACE who cannot express fusion after an appropriate prism intervention or surgical repositioning [9, 10, 66].

The average age of onset

Case reports [4] have discovered coexisting or underlying neurological injuries in patients with AACE. The mean age at onset was 26.6±12.2 years and three cases had cerebellar diseases [4]. These findings highlight that the cerebellum, as a part of the extrapyramidal system, functions significantly in preserving normal ocular alignment [4].

The mean age of onset was reported to be 4.7 years in 48 children with AACE over a 13-year period, and intracranial pathologies (e.g., hydrocephalus, pontine, and thalamic glioma) were found in 6% of these children [66]. They reported several risk factors for intracranial pathologies, including larger esodeviation at distance, recurrence of AACE, neurological manifestations (e.g., papilledema), and older age at onset (>6 years) [66]. The average age of onset was reported to be 6.14 years and intracranial pathologies were discovered in 8.33% of patients in another study [51]. These findings imply that AACE in childhood should be differentiated from that in adulthood.

In one study [61], which examined both children and adults (age at esotropia onset ranged from 3 to 62 years), the mean age ± SD at presentation was 23.1 ± 11.7 years. With regard to the age of the patients, 8.7%, 33.3%, and 58.0% were less than 10 years, 10 to 18 years, and more than 18 years, correspondingly [61]. These findings showed that AACE was more common in older children and adults than in younger children in the Chinese population [61].

The time-span from the onset of esotropia to the initiation of treatment was not found to be a decisive factor in the re-establishment of normal stereoacuity [3, 70, 71].

Different types of refractive errors have been reported among different age groups [2, 18, 61, 70, 72]. Based on these reports of patients with AACE [2, 18, 70, 72], younger children had mild hypermetropia, and adult patients always had myopia, but no clear conclusion could be drawn from older children (10–18 years old). In this regard, in one study of Asian cases [61], patients aged < 10 years showed mild hypermetropia, whereas older children (10-18 years old) and adults had moderate-to-high myopia [61]. The mean angles of esotropia were also found to be significantly larger in young children than in older children and adults. In contrast, no significant difference was found in stereoacuties among different disease durations [61].

Discussion

The purpose of this study was to provide an overview of the current knowledge of neurological pathologies in AACE based on the available literature. The results of the literature survey were analyzed to provide an overview of the current knowledge of neurological pathologies in AACE.

The results revealed that AACE with unclear etiologies can occur in many cases in both children and adults [1, 2, 6, 8, 14–18]. Functional etiological factors for AACE were found to be due to many reasons, such as functional accommodative spasm [8, 19], the excessive near work use of mobile phones/smartphones, and other digital screens [5, 11, 12, 20–24, 73]. For example, the rise of AACE has been witnessed during the coronavirus disease 2019 (COVID-19) pandemic when people were forced to stay at home and work from home through digital devices [23, 24].

In addition, AACE was found to be associated with neurological disorders, such as astrocytoma of the corpus callosum, medulloblastoma, tumors of the brain stem or cerebellum, Arnold-Chiari malformation, cerebellar astrocytoma, Chiari 1 malformation, idiopathic intracranial hypertension, pontine glioma, cerebellar ataxia, thalamic lesions, myasthenia gravis, certain types of seizures, and hydrocephalus [6, 19, 25–40]. This calls for comprehensive neurological assessment to rule out neurological pathologies associated with AACE.

As a result, since AACE can be of varied etiology ranging from functional factors to those harboring serious intracranial diseases, patients' medical history and records should be evaluated thoroughly [7, 8, 19]. Regular neurological and ophthalmic assessments are necessary in patients with an

unconfirmed etiology and unclear precipitating history [7, 8, 19, 59]. It is worth noting that previous studies [18, 28, 38] have reported incidences of up to 10% for neurological diseases associated with AACE. However, there are no single clinical symptoms or signs that signify the presence of neurological diseases [7, 8]. The author recommends neuroimaging in patients with AACE if they have an unclear precipitating history or abnormal ocular and neurological indications, such as headache, cerebellar imbalance, weakness, or nystagmus.

Risk factors for AACE with intracranial pathology have been reviewed in this article. However, it is important for clinicians to have experience in the field of neuro-ophthalmology when dealing with strabismus. For example, based on the author's experience, if sensory or motor fusion cannot be demonstrated in patients with AACE when examined with prisms or a synoptophore, the possibility of an underlying neurological disease should be suspected. Cycloplegic refraction should be performed to rule out the accommodative component, especially in young patients. Careful ocular motility analysis in all gaze directions is also important to rule out a paretic deviation.

The main limitation of this study is that most of the reported studies and the available literature are old and based on case reports, which are not considered as high levels of evidence. Therefore, the concluding and commentary lines cannot be generalized. They are just comments based on the available and limited literature in this regard. This calls for further studies with higher levels of evidence to fill the current gap in this area of science.

Conclusion

Although isolated AACE can be a benign form of strabismus, it can also be the first indication of a serious neurological disorder. Various studies have disclosed that AACE can be associated with intracranial diseases, such as elevated intracranial pressure, brain glioma, Arnold-Chiari malformation, and thalamic or cerebellar tumors, which necessitate neuroimaging probes, particularly in older individuals. Nevertheless, there are no clinical symptoms or signs that specify the presence of a neurological disease.

Since a variety of intracranial diseases (e.g., hydrocephalus, Chiari type I malformation, and tumors of the cerebellum, brainstem, or sellar region) have been associated with AACE, it is vital to consider complete neurological assessments in these patients, especially in the presence of nystagmus or abnormal ocular and neurological indications (e.g., headache, cerebellar imbalance, weakness, or nystagmus).

In conclusion, the uncommon but potential presence of a coexisting or underlying neurological pathology in patients with AACE should be sufficient to justify neurological and /or neuroradiological examinations in this group of patients. This is a particular concern for patients with AACE who present with other neurologic indications (e.g., headache, papilloedema, clumsiness, and poor motor coordination) or accompanying nystagmus.

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