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Original Article

Possibility of Neurological Diseases Associated with Acute Acquired Comitant Esotropia

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Purpose: This study investigated the possibility of neurological etiologies causing acute acquired comitant esotropia (AACE) and to evaluate the differences in clinical features between younger children, older children, and adults.

Methods: In this retrospective analysis, patients who had been diagnosed with AACE between July 2017 and June 2021 were included. Data on clinical findings, medical history, brain or orbital imaging, and ophthalmological and orthoptic examinations were retrieved from medical records and analyzed. Patients were divided into three groups based on their age: younger children (<10 years), older children (10–18 years), and adults (>18 years).

Results: Overall, 41 patients with AACE (15 females and 26 males) were examined. Most patients were children. Mild hyperopia was observed in children, while adults had moderate to high myopia. The mean angle of esotropia at a distance fixation was 43.57 ± 9.77, 51.54 ± 8.75, and 30.14 ± 12.39 prism diopters (PD) in younger children, older children, and adult groups, respectively. The mean angle of esotropia at a near fixation was 43.57 ± 9.37, 51.15 ± 9.39, and 31.43 ± 12.15 PD in younger children, older children, and adult groups, respectively. Significant differences were found in the mean angles of esotropia in patients with AACE at both near and far distances according to their age (all $p < 0.001$). Among 36 patients with previous neuroimaging data, none had AACE secondary to intracranial lesions. Over 2 years, five patients who were under continuous observation did not develop any neurological abnormalities.

Conclusions: AACE was more common in children than in adults. The angle of deviation was larger in children than in adults. Coexisting or underlying neurological diseases were not present in patients with isolated AACE, which eliminated the need for neuroimaging. Continuous follow-up evaluations are warranted when signs of intracranial disease are observed in patients who have not undergone an imaging investigation.

Key Words: Acute acquired comitant esotropia, Nervous system diseases, Refractive errors

Sudden onset of esotropia with an equal angle of deviation in all fields of gaze is the characteristic feature of acute acquired comitant esotropia (AACE), which is rare. No statistical data is available regarding its actual incidence or prevalence. AACE can occur in both children and adults [1–3] and has been reported to account for 0.3% of all childhood strabismus cases [4,5].

Historically, AACE has been categorized into three

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types based on the clinical features and apparent etiology. Type 1, or Swan type, is an acute onset esotropia following occlusion. Type 2, or Burian–Franceschetti type, is a refractive error defined as minimal hypermetropia without an accommodative element, which is usually precipitated by physical or psychological stress. Type 3, or Bielschowsky type, is AACE associated with myopia [6]. The number of AACE cases has increased significantly in recent years. Many recent studies have reported the varied etiologies and clinical courses of AACE. The reported underlying etiologies range from functional, systemic diseases to serious, intracranial diseases. Some studies have reported intracranial diseases, such as elevated intracranial pressure, brain glioma, Chiari malformation, and thalamic or cerebellar tumors as etiologies, all of which require neuroimaging studies [1–4,7–9]. However, the decision of when to perform further neuroimaging and/or neurological evaluation is controversial. A few studies reported the differences in the clinical course between age groups [3,8]. Therefore, this study aimed to review the possibility of neurological etiologies causing AACE, examine its clinical course, and investigate the differences between its clinical features in younger children, older children, and adults.

Materials and Methods

Ethical statements

The Institutional Review Board of Naresuan University approved the study protocol (No. NU-IRB 1719). The requirement for informed consent was waived due to the retrospective nature of the study.

Patient selection

The medical records of patients diagnosed with AACE between July 2017 and June 2021 at the Department of Ophthalmology, Naresuan University Hospital were retrospectively reviewed. Patients who met the following criteria were included: (1) acute onset of esotropia with photographic evidence of previously aligned eyes; (2) onset after 1 year of age; (3) comitant esodeviation with deviation in all directions of gaze differing by <5 prism diopters (PD) at both near and distant fixation; and (4) patients with corrected visual acuity better than 20 / 30 in both eyes.

The exclusion criteria were as follows: (1) having incomitant esotropia, meaning limited abduction and larger deviation in the lateral gaze (particularly more than 5 PD); (2) having a reduction in the esotropia with hypermetropic spectacle correction or showing hyperopia of more than +2.00 D; (3) a history of ocular occlusion; and (4) a history of ocular trauma or surgery.

Age, sex, presenting symptoms, duration of deviation, past medical history, average daily time spent looking at near objects like smartphones and tablets before the onset of AACE, duration and findings in the follow-up period, best-corrected visual acuity, cycloplegic refraction, ocular deviation during near and distant fixation by alternate prism cover testing, ocular motility, and the results of brain and orbital imaging (computed tomography [CT] and/or magnetic resonance imaging [MRI]) were extracted from the patients' records. The duration of AACE was determined from the medical history and old photographs. The follow-up period was from the first visit to the last appointment. In patients aged <40 years, cycloplegic refraction was performed 40 minutes after instillation of cyclopentolate 1% twice in both eyes, 10 minutes apart. In patients aged ≥40 years, refraction was performed without the use of a cycloplegic agent. Spherical equivalents of refractive errors were calculated using the algebraic sum of the dioptric powers of the sphere and half the cylinder. The angles of deviation were assessed by the alternate prism cover test at distant and near fixation in the primary, secondary, and tertiary (if possible) gaze positions.

Fundoscopy and slit-lamp examinations were performed on all patients. All patients were also examined by one pediatric ophthalmologist with a subspecialty in strabismus. All patients were suggested to undergo neuroimaging to rule out central nervous system pathologies. Additionally, neurological examinations were performed, and comprehensive medical histories were taken from patients who had clinical features suggestive of central nervous system pathologies and abnormal neuroimaging findings. The patients were divided into three groups based on their age: younger children (<10 years), older children (10 to 18 years), and adults (>18 years).

Statistical analysis

Categorical data are shown as frequencies and percentages. The Shapiro-Wilk test was used for normally distrib-

uted data. Continuous variables are presented as the mean ± standard deviation and range. Paired *t*-tests were also used. One-way analysis of variance test and a subsequent Bonferroni post hoc analysis were used for multiple between-group comparisons. Statistical analyses were performed using Stata ver. 12.0 (StataCorp). A *p*-value of <0.05 was considered statistically significant.

Results

Overall, 41 patients with AACE (15 females and 26 males) were included in this study. The mean ± standard deviation age of patients during their first eye examination was 10.8 ± 8.6 years (range, 3 to 44 years). The patients comprised 21 younger children (51.2%), 13 older children (31.7%), and seven adults (17.1%). None of the patients had any recent history of trauma, systemic disease, medication use, febrile illness, or a family history of strabismus. None of the patients had neurological signs or symptoms at their last follow-up. The average follow-up period was 1.5 ± 0.8 years (range, 0.1 to 3.8 years).

The duration of AACE presentation ranged from 2 weeks to 48 months before the first visit. Patients presented with symptoms such as esotropia (34.1%), diplopia (22.0%), or diplopia with esotropia (43.9%). None of the patients had abduction nystagmus or latent nystagmus. No V patterns, inferior oblique muscle overactions, or dissociated vertical deviations were observed. None of the patients showed any delay in abduction in either eye. Anterior segment and fundus examinations revealed no abnormalities.

Visual acuity was good in all patients, none of whom had a vision <20 / 30 in the worse eye. Nine patients with myopia were using refractive corrections at presentation. The refractions of patients with AACE were significantly different between the age groups ($p < 0.001$). The mean cycloplegic refractive error in younger children was +0.75 ± 0.58 (range, –0.75 to 1.63) in the right eye (OD) and +0.82 ± 0.57 (range, –0.38 to 1.75) in the left eye (OS). The mean cycloplegic refractive error in older children was +0.21 ± 1.39 (range, –3.75 to 1.75) in the OD and +0.35 ± 0.78 (range, –1.38 to 1.25) in the OS. Younger and older children exhibited mild hyperopia. In the adult group, moderate to high myopia was observed with a mean myopia of –3.27 ± 2.86 D (range, –7.63 to –0.38 D) in the OD and –2.98 ± 2.00 D (range, –6.63 to –0.63 D) in the OS. Spherical equivalents of both younger and older children were significantly different from those of adults (all $p < 0.001$ for OD and OS in younger and older children). No significant difference in mean spherical equivalent could be observed between younger and older children (OD, $p = 0.908$; OS, $p = 0.573$) (Table 1).

Nineteen patients (46.3%) had alternating esotropia, 12 (29.3%) had left esotropia, and 10 (24.4%) had right esotropia. The mean angle of esotropia for distant fixation (43.80 ± 12.08 PD; range, 16 to 70 PD) did not significantly differ from that for near fixation (43.90 ± 11.70 PD; range, 20 to 70 PD; $p = 0.789$). The mean angles of esotropia in patients with AACE were significantly different between age groups both at near and far distances (all $p < 0.001$). The older children group showed the largest angle of deviation. The mean angle of esotropia at distant and near fixation in older children was significantly larger than those in adults ($p = 0.001$ and $p < 0.001$, respectively). Similarly, the mean angle of esotropia at distant fixation in younger children was significantly larger than those in adults ($p = 0.011$).

Table 1. Refraction of patients with acute acquired comitant esotropia

Cycloplegic refraction	Younger children (<10 yr) (n = 21)	Older children (10–18 yr) (n = 13)	Adults (>18 yr) (n = 7)
Right eye (D)			
Average	+0.75 ± 0.58*	+0.21 ± 1.39*	–3.27 ± 2.86
Range	–0.75 to 1.63	–3.75 to 1.75	–7.63 to –0.38
Left eye (D)			
Average	+0.82 ± 0.57*	+0.35 ± 0.78*	–2.98 ± 2.00
Range	–0.38 to 1.75	–1.38 to 1.25	–6.63 to –0.63

Statistical analysis was performed using one-way analysis of variance with the Bonferroni test.
D = diopters.
*$p < 0.016$ (vs. adult).

However, the mean near angle of younger children was not significantly different from that of adults (p = 0.023). There was no significant difference in the mean angles of esotropia between younger and older children (distant, p = 0.086; near, p = 0.107) (Table 2). Patients with myopic correction did not have significantly reduced esotropia deviation for both near and far objects.

Thirty-six patients (87.8%) underwent brain and orbital neuroimaging studies (MRI, 29 patients; CT, seven patients). The remaining five patients (12.2%) refused investigations but remained under observation. The parents of three children refused imaging due to its requirement for anesthesia. Two adults denied neuroimaging because they had chronic deviation in only one eye with no other neurological symptoms. None of the 36 patients' scans showed neurological causes of AACE. One scan revealed a simple pineal cyst, and another revealed an encephalomalacic-gliotic change in the right corona radiata. None of the patients developed a neurological disease. The mean follow-up period was 1.5 ± 0.8 years, with a range of 0.1 to 3.8 years. Among the five patients who did not require imaging, none developed a neurological disease over a mean follow-up period of 2.1 years (range, 1.1 to 3.0 years).

Smartphone usage time was recorded in 30 of the 41 patients. Twenty-two patients (73.3%) used their smartphones for >4 hours a day. This was mostly the case with older children (90.9%) and adults (100%). In the group with daily smartphone usage <4 hours, the mean angle of esotropia was 38.75 ± 7.91 PD for distant objects and 38.75 ± 7.44 PD for near objects. In the group who used smartphones for >4 hours, the mean angle of esotropia was 43.23 ± 14.43 PD for distant objects and 43.18 ± 14.02 PD for near objects. There was no correlation between the angle of deviation and the duration of smartphone use with both distant and near fixation (p = 0.415 and p = 0.405, respectively).

Discussion

AACE, which typically presents as sudden onset esotropia, is an unusual ocular alignment disorder. The present study did not have any patients with a history of monocular occlusion or visual loss preceding the onset of esotropia as the exclusion criteria, namely Swan type AACE, was not achieved in any of the cases. Nine patients wore spectacles for myopia correction before the onset of esotropia. Therefore, it seems that uncorrected myopia is unrelated to AACE. Almost all patients, particularly younger and older children with mild hyperopia and large angle deviations, appeared to be part of a subgroup that resembles type 2 (Burian–Franceschetti type) AACE.

Recent studies have found that AACE can occur in any age group [1–4,7]. In the present study, AACE was more frequent in younger (51.2%) and older children (31.7%) than in adults (17.1%). However, some studies report that AACE is more common in older children and adults. [2–3,7].

The correlation of refraction in patients with AACE among different age groups has been reported. The present study showed that children <18 years had mild hyperopia and that adult patients had moderate to high myopia, which is consistent with that reported in prior studies [3,8]. In addition, older children (10 to 18 years) also had mild myopia to mild hyperopia. However, no statistical significance

Table 2. Mean angle of deviation in patients with acute acquired comitant esotropia

Deviation	Younger children (<10 yr) (n = 21)	Older children (10−18 yr) (n = 13)	Adults (>18 yr) (n = 7)
Near (PD)			
Average	43.57 ± 9.37	51.15 ± 9.39*	31.43 ± 12.15
Range	30–60	35–70	20–50
Distance (PD)			
Average	43.57 ± 9.77*	51.54 ± 8.75*	30.14 ± 12.39
Range	30–60	40–70	16–50

Statistical analysis was performed using one-way analysis of variance with the Bonferroni test.
PD = prism diopters.
*p < 0.016 (vs. adult).

could be found between this group and that of younger children (<10 years).

The mean deviations were 43.57 ± 9.37 PD for near and 43.57 ± 9.77 PD for distant vision among younger children, which was significantly less than those reported by Fu et al. [3]. However, the mean deviation in both younger and older children was larger than that in adults, similar to those reported by Fu et al. [3]. This is likely because younger and older children who had hyperopia met the criteria of type 2 AACE, which is characterized by a large angle of deviation. In contrast, other studies have reported small angle deviation in children [4,9]. The measurements in adults also showed a concordance with previous reports [3,10]. Based on the results of the present study, the refraction and angle of deviation were not statistically significant between younger and older children, but they were significantly different from adults.

The etiology of AACE remains unclear and varies from benign to serious intracranial disorders. Several reports state that it may be indicative of serious, intracranial diseases, including neoplasms in the cerebellum, brainstem, pituitary gland, or corpus callosum [11,12]. The mechanism of AACE is still unclear, and no single central nervous system pathology can account for all AACE cases.

Coexisting or underlying intracranial pathologies were present in 3% to 8% of AACE cases in previous studies with different sample sizes (Table 3) [1,2,4,7–9,13]. In most cases of neurological AACE reported in the literature, the esotropia was accompanied by other ophthalmic or neurological abnormalities, such as headache, cerebellar signs, nystagmus, papilledema, or pallor of the optic disc.

The onset of comitant esotropia in a child may be the first sign of a cerebellar tumor without any other neurological signs and symptoms. However, papilledema or pallor of the optic disc can be observed on fundus examination [14,15]. A fundus examination should be carefully repeated when treating children with the first presentation of acute nonaccommodative concomitant esotropia.

Although isolated AACE has been considered a benign form of strabismus that did not require further evaluation, several reports have mentioned that neurologically associated AACE without ophthalmic or neurological abnormalities can occur in up to 6% of cases [1,2,4,7]. It is important to note that a normal ophthalmic and neurological evaluation does not rule out the possibility of an intracranial pathology.

Table 3. A summary of studies that describe an association between AACE and intracranial disease

Study	Study period	Age group (yr)	Intracranial disease/ total AACE (%)	Diagnosis (no. of cases)	Neurological or ophthalmological abnormalities (no. of cases)
Buch and Vinding [4]	2000–2013	<18	3/48 (6.3)	Increased intracranial pressure due to cyclosporine (1) Pontine glioma (1) Thalamic glioma (1)	Headache and papilledema (1) Headache and nystagmus (1) None (1)
Schorkhuber et al. [13]	2000–2014	1–18	3/53 (5.7)	Sinus vein thrombosis (1) Congenital mega cisterna magna (1) Viral meningitis (1)	Papilledema and nystagmus (1) Known cause (2)
Chen et al. [1]	2010–2014	5–59	3/47 (6.4)	Cerebellopontine angle tumor (2) Spinocerebellar ataxia (1)	Bilateral nystagmus (1) Nystagmus and poor coordination of gait (1) None (1)
Neena and Giridhar [9]	2013–2016	4–17	1/12 (8.3)	Central nervous system glioma (1)	Temporal pallor (1)
Cai et al. [2]	2011–2017	5–47	2/45 (4.4)	Pituitary tumor (1) Demyelination (1)	Headache and papilledema (1) None (1)
Lekskul et al. [8]	2017–2019	6–39	1/30 (3.3)	Arnold–Chiari malformation (1)	Gaze-evoked nystagmus (1)
Meng et al. [7]	2018–2019	8–55	3/51 (5.9)	Pituitary tumor (1) Brainstem tumor (1) Arnold–Chiari malformation (1)	None (3)

AACE = acute acquired comitant esotropia.

In addition, the presence of a normal neurological evaluation at first examination does not rule out the possibility of developing an intracranial disease at a later stage in life. Zweifach [16] reported the case of a 10-year-old boy with normal neurological and neuroradiologic evaluations who had undergone strabismus surgery after the sudden development of esotropia. However, he developed clinical signs that led to a diagnosis of cerebellar medulloblastoma 18 months postoperatively. Schreuders et al. [17] reported the case of a 5-year-old boy with a diffuse pontine glioma who had presented with acute onset comitant esotropia with diplopia on the first examination. Neurological symptoms appeared 10 weeks after the first visit.

AACE has also been described in non-neoplastic neurological disorders, such as demyelinating disease, idiopathic intracranial hypertension, cerebellar ataxia, Chiari malformation type I, and myasthenia gravis [1,9,11]. Neena and Giridhar [9] reported that one patient with AACE and diplopia for 2 years was found to have ocular myasthenia. His esotropia and diplopia disappeared with oral pyridostigmine therapy. Studies have also been reported on the associations between AACE and human growth hormone and cyclosporin, and have suggested that esotropia development may be related to idiopathic intracranial hypertension [4,18].

Recent studies have reported that AACE may occur in the absence of an associated neurological pathology in children and adults (Table 4) [3,10,19–25]. This is consistent with the present study, which reported a series of patients presenting with AACE who had no other ocular or neurological abnormalities and whose evaluation did not identify any intracranial pathology that would explain acute onset strabismus. None of the patients had medical ailments. All patients were followed up for a mean of 1.5 ± 0.8 years (range, 0.1 to 3.8 years), and repeated examinations showed that they did not develop any neurological symptoms. The mean follow-up time for patients with no imaging data was over 2 years. The data reflects a common clinical scenario: when isolated AACE is present for a long duration, its etiology is usually benign.

Recently, many studies have suggested that an excessive use of smartphones may also be the root cause of AACE [7,23–25]. In the present study, chronic use of near vision, namely using smartphones and other electronic devices, was not evaluated in relation to sudden onset esotropia; however, the duration of near work was not related to the degree of angle deviation. This result is consistent with that of a study conducted by Meng et al. [7].

It is difficult to ascertain which patients presenting with AACE have underlying neurological diseases. Buch and Vinding [4] suggested that clinical risk factors of intracranial disease associated with AACE were onset at age >6 years, greater deviation at distance, late recurrence deviation, and neurological abnormalities. However, the present study shows that the mean onset was 10 years and that none of the patients had an underlying intracranial disease. This is consistent with the results of studies by Erkan Turan and Kansu [19] and Dotan et al. [21] who reported that patients presenting with isolated AACE with an onset at age >6 years did not have abnormal neuroimaging. A sin-

Table 4. A summary of studies that describe no association between acute acquired comitant esotropia and intracranial disease

Study	Study period	Age group (yr)	No. of patients	Etiology
Fu et al. [3]	2008–2012	4–62	69	Idiopathic
Erkan Turan and Kansu [19]	1993–2014	20–43	9	Idiopathic
Lee et al. [23]	2009–2014	7–16	12	Excessive smartphone use
Li and Sharan [20]	2009–2015	9–17	7	Idiopathic
Lee and Kim [10]	2008–2016	17–64	16	Idiopathic
Wu et al. [24]	2016–2017	6–46	26	Excessive smartphone use
Dotan et al. [21]	2014–2018	4–15	20	Idiopathic
Topcu et al. [25]	2006–2019	6–44	27	Near work with digital displays
Tong et al. [22]	2018–2019	14–38	22	Idiopathic
This study	2017–2021	3–44	41	Idiopathic

gle clinical symptom or sign cannot reliably reveal whether AACE is secondary to a brain tumor. AACE can be the first symptom of a serious intracranial pathology. In most cases, other ophthalmic and/or neurological abnormalities exist when AACE is related to intracranial disease. Other studies recommend that patients who have an unclear precipitating history, abnormal ocular examination findings, or neurological symptoms, such as headache, cerebellar imbalance, weakness, or nystagmus, undergo neuroimaging [1,2,4,13,26]. The decision of when neuroimaging should be performed remains controversial, and there is an ongoing debate among clinicians about both children and adults presenting with AACE [9,19,21,26]. In particular, it is practically impossible to immediately perform a radiologic examination on all children. However, isolated AACE is more often benign than not. When patients present with isolated AACE, it is important to discuss the need for immediate neuroimaging with the patient or the patient's caregivers.

This study had a few limitations, the first of which is its retrospective design. Second, not all patients were subjected to neuroimaging studies or neurological examinations. Five patients were under observation and were followed up and subjected to repeated examinations for over a year, during which they did not develop any neurological symptoms. Third, the mean follow-up duration was short; the onset of ophthalmic or neurological abnormalities may occur after this period. Finally, this study cannot fully eliminate the possibility of a prior sixth cranial nerve paresis that has partially recovered with time. However, no patients had any history of head trauma or underlying systemic disease.

In conclusion, the possibility of neurological etiologies causing chronic AACE in the absence of other ophthalmologic or neurological abnormalities was found to be rare. The most important point to remember when treating AACE is that AACE may be the first sign of a neurological disease. Patients who do not immediately undergo imaging must be monitored and evaluated for signs of intracranial disease.

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