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Original Article

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Epidemiology of childhood cataracts

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ABSTRACT

Aims: The aim of the study was to investigate the epidemiologic characteristics of pediatric patients with cataracts and to make a statistical comparison.

Methods: The study included 25 eyes of 19 children aged 16 years and younger who presented to Kırıkkale University, Faculty of Medicine, Department of Ophthalmology with visual complaints between April 2018 and November 2023. The information about the patients was obtained and recorded by scanning the patient files. The patient data included age, gender, cataract type, trauma history, medical history, family history, and surgical intervention.

Results: In the study, 25 eyes of 19 children were examined. Eleven (57.9%) of the patients were girls, and eight (42.1%) were boys, with a mean age of 4.74±4.22 years (0.2-14). When all age groups were compared, there was no statistically significant difference between boys and girls in the incidence of childhood cataracts (p=0.532). Both eyes were affected in 5 children (26.3%), the right eye in 8 (42.1%), and the left eye in 6 (31.6%). Regarding etiology, 5 were idiopathic, 4 were genetic, 2 were metabolic syndrome, 2 were steroid-induced, and 6 were traumatic.

Conclusion: Congenital cataracts are the most common type of cataract seen in early childhood. Etiological causes in childhood cataracts differ from adult cataracts.

Keywords: Congenital cataracts, epidemiologic characteristics, statistical comparison

INTRODUCTION

Cataract is a condition in which the transparent lens becomes opaque. Pediatric cataracts are cataracts seen between the ages of 0-16 years, of which congenital cataracts seen in early childhood are the most common.[1,2] The prevalence of pediatric cataracts is estimated to be between 2.2 and 13.6 per 10,000 children worldwide.[3]

The etiology of pediatric cataracts is variable. The most common etiology is idiopathic, followed by genetic and infectious causes.[4] Globe trauma plays an important role in the etiology of cataracts in advanced childhood, and this rate is higher in developing countries.[5]

Cataract-related conditions in childhood pose a huge problem in terms of morbidity, economic loss, and social isolation. Due to the life expectancy of the pediatric population, restoring the vision of a child blinded by cataract is equivalent to restoring it in 10 adult patients.[6] Especially since early childhood is the period of visual development, delays in diagnosis and treatment may lead to amblyopia, unlike adults. This means that, unlike adults, treatment does not end with surgery and requires a long treatment and follow-up period for visual rehabilitation.

The aim of this study is to describe the epidemiology of cataract in children aged 16 years and younger evaluated at Kırıkkale University Faculty of Medicine, Department of Ophthalmology, which is located in Central Anatolia and provides tertiary care for the surrounding provinces, over a period of 5 years. This study also examines the frequency, causes, and treatment outcomes of cataracts in children.

METHODS

Ethics

The study was initiated with the approval of the Kırıkkale University Medical Faculty Clinical Researches Ethics Committee (Date: 17.04.2024, Decision No: 2024/06). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

Patients and Methods

The study included 25 eyes of 19 children aged 16 years and younger who presented to Kırıkkale University, Faculty of Medicine, Department of Ophthalmology with visual complaints between April 2018 and November 2023. Patient information was recorded by scanning patient files. The extracted patient data included age, gender, cataract type, trauma history, medical history, family history, and surgical intervention. Biomicroscopic examinations, including dilated fundus examinations, were noted in detail. Medical and surgical histories were noted if additional surgery was required. Children were divided into three age groups: 0-5 years (infancy and preschool), 6-10 years (primary education), and 11-14 years (secondary education).

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics 20. Frequency distributions were generated for cataract type and cause. A statistical analysis of quantitative data was performed for all variables. Frequency analysis was performed with the chi-square test. P-values of 0.05 and lower were considered statistically significant.

RESULTS

The study included 25 eyes from 19 children. Eleven (57.9%) were girls and 8 (42.1%) were boys, with a mean age of 4.74±4.22 years (0.2-14). The most common age at diagnosis was in the 0-5 age group, with 63.2% (n=12). This was followed by children aged 6-10 years (26.3%, n=5) and 11-16 years (10.5%, n=2) (Table). When all age groups were compared, there was no statistically significant difference between boys and girls in the incidence of childhood cataracts (p=0.532). Both eyes were affected in 5 children (26.3%), the right eye in 8 (42.1%), and the left eye in 6 (31.6%).

Table. Demographic characteristics of childhood cataracts

Characteristics	Number (%)
Age	
0-5	12 (63.2%)
6-10	5 (26.3%)
11-16	2 (10.5%)
Gender	
Male	8 (42.1%)
Female	11 (57.9%)

Regarding etiology, 5 were idiopathic, 4 were genetic, 2 had metabolic syndrome, 2 were steroid-induced, and 6 were trauma-induced (Figure). All idiopathic cases were consulted to the pediatric department for underlying metabolic or genetic factors and a systemic investigation was performed.

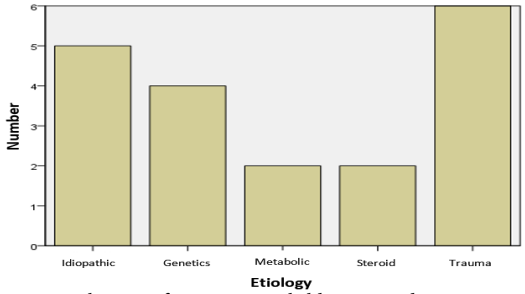

Figure. Distribution of cataracts in children according to causes

Of the traumatic patients, 2 developed cataracts after blunt trauma, and 4 had penetrating trauma. Two patients had Down syndrome, and two patients had cataracts due to nephrotic syndrome caused by long-term steroid use. Both of the patients with metabolic syndrome were followed up with diet because of galactosemia, and all other patients underwent surgery. Due to the age of the patients or corneal perforation, 9 patients were left aphakic, and secondary IOL implantation was performed in a second session. The average age of patients who had lenses placed in the second session was 3.77±2.45 years.

The most common accompanying ocular findings were strabismus (42.1%) and nystagmus (26.3%). 3 (15.7%) patients had microphthalmia.

DISCUSSION

There is a critical period of visual development during which any damage that causes a change in the normal visual stimulus (unilateral or bilateral) leads to the emergence of amblyopia. The most vulnerable period is observed in the first 3 years of life.[7,8] Therefore, the main aim of early diagnosis of cataracts is to prevent irreversible complications, especially amblyopia, which directly affects the visual prognosis of the child. In our study, amblyopia was present in all patients whose visual acuity was evaluated.

The percentage of blindness due to lens problems is around 7%,[9] making it a very important cause of preventable blindness. The detection of cataracts requires some basic parental awareness. If parents lack this education, cataracts may go unrecognized and lead to delays in diagnosis. In four of the patients in our study, the diagnosis was made during routine ophthalmologic examinations, independent of visual complaints.

Children with acquired cataracts have very good vision and are therefore often recognized late. Teachers play an important role in the diagnosis of acquired cataracts.[10] Therefore, school vision screening programs and teacher training are very important for the detection of visual pathologies in children.

The most important cause of acquired cataracts in children is childhood ocular trauma.[11] Similarly, trauma was the cause of 6 of 8 patients with acquired cataracts in our study. Although studies have shown that cataract development due to childhood trauma is 10 times more common in males,[12,13] it was found to be equal in our study. However, this may be

due to the small sample size. Traumatic cataracts also tend to occur at older ages [mean age (78.5 months)].[14] In our study, the mean age of children with cataracts due to trauma was 6.33±4.08 years.

Another cause of acquired cataracts in our study was steroid-induced cataracts due to chronic drug use. In the prevention of this type of cataract, routine ophthalmologic examinations should be included in the treatment program for chronic use of drugs with known side effects.

While the most common cause of cataract in adulthood is senility, it may vary in childhood, as can be seen in our study. While the timing of surgery does not matter in adults, early intervention is especially important in children as it is an important period of vision development.

CONCLUSION

Although early diagnosis and treatment of childhood cataracts is associated with good visual outcomes, delay in diagnosis and, therefore, delay in surgery is a major public health problem. Awareness-raising among target groups is crucial to improving the prognosis of childhood cataracts.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was initiated with the approval of the Kırıkkale University Medical Faculty Clinical Researches Ethics Committee (Date: 17.04.2024, Decision No: 2024/06).

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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Original Article

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Evaluation of macular vessel density with OCTA in patients undergoing inferior oblique muscle surgery

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ABSTRACT

Aims: To evaluate pre-and postoperative macular vascular density changes in patients undergoing inferior oblique muscle surgery using optical coherence tomography angiography (OCTA).

Methods: 28 eyes of 16 patients who underwent inferior oblique muscle surgery were included in the study. Measurements were taken preoperatively (T0) and one day (PO1), 15 days (PO15) and 6 weeks (PO6w) after surgery. The vessel densities of the superficial capillary plexus (SCP), deep capillary plexus (DCP) and choriocapillaris (CCL) were measured by OCTA.

Results: This study included 28 eyes of 16 patients. The mean age of the patients was 8.07±5.5 years (4-23 years). SCP and DCP mean vascular densities decreased significantly at PO1 and PO15 compared to T0, but increased at PO6w, reaching their initial levels. Mean CCL levels were lower at PO1 compared to all other measurements and reached their preop values at PO6w.

Conclusion: The inferior oblique muscle insertion is known to be close to the macula and surgical interventions for this muscle may cause changes in macular vascular density. In our study, macular vascular density decreased in all parameters at PO1 compared to T0, but almost reached its preop levels at PO6w.

Keywords: Inferior oblique muscle, vessel density, OCTA

INTRODUCTION

Inferior oblique muscle hyperfunction (IOHF) is a common motility disorder associated with many types of strabismus.[1] In case of IOHF, elevation is observed in the adducting eye. If it is caused by paresis or paralysis of the superior oblique or superior recti muscles, it is called secondary IOHF, and if the etiology is unclear, it is called primary IOHF.[2,3] Surgical treatment of strabismus due to IOHF mainly aims to weaken the muscle. The most preferred methods are myectomy and anterior transposition.[4] According to Parks, anterior transposition is the most effective and longest lasting of the weakening procedures.[5]

The inferior oblique muscle insertion is located very close to the macula, one of the most critical regions of the eye. In addition, the inferior oblique muscle is the shortest of the ocular muscles, making it very difficult to manipulate during surgery. Excessive traction and globe compression causes compression of the short posterior arteries and this traction can also affect the macula due to its close proximity. The fusion of the muscle with the sclera varies between individuals and in 50% of cases it fuses in 2-6 separate layers.[6] Some studies have reported that the inferior oblique muscle is more hypertrophic in men than in women.[7,8] Possible changes in the choroidal and retinal circulation after inferior oblique muscle surgery are thought to be related to surgically induced inflammation.[9,10]

Although many studies have been performed using fundus photography, color Doppler ultrasound, fluorescein angiography, indocyanine green angiography, there is no general consensus on hemodynamic changes after strabismus surgery.[11-13] Optical coherence tomography angiography (OCTA) is noninvasive and visualizes microvascular structures of the retina and optic nerve head without pupil dilation. In addition to saving time, it does not require dye injection and shows both thickness and vascular density of certain layers of the retina at the same time compared to fluorescein angiography.[14] When we look at the literature, the effect of surgery on vascular density is not yet clear in OCTA measurements obtained with OCTA in patients undergoing inferior oblique surgery.[10,15] The aim of our study is to show possible macular vascular density changes after inferior oblique surgery in patients with IOFH with OCTA.

METHODS

Twenty-eight eyes of 16 patients who were followed up in the strabismus unit of Fırat University Medical Faculty Hospital and planned to undergo inferior oblique muscle surgery were included. The study was performed with the approval of Fırat University Non-interventional Researches Ethics Committee (Date: 13.02.2024, Decision No: 2024/03-48). Informed consent was obtained from all subjects or their legal representatives and the study was conducted in accordance with the principles of the Declaration of Helsinki. Each patient underwent a complete ophthalmologic examination including visual acuity measurement with Snellen chart with and without correction, cycloplegic refraction, anterior segment examination, dilated fundus examination, detailed strabismus examination (cardinal gaze positions, covering test, prism, Krimsky and head position tests). IOHF grading was performed by the same ophthalmologist specializing in strabismus. IOHF was determined by grading from +1 to +4.[1]

Only IOHF patients with +2 and above were included in surgery. Patients with sensory and restrictive strabismus, history of ocular surgery and corneal opacity, medical history involving retinal and choroidal tissues such as glaucoma, uveitis, maculopathy and amblyopia were excluded. Patients with systemic diseases affecting the vascular tissues such as hypertension and diabetes mellitus, systemic drug use in the last one month affecting the surgery, and patients who could not adapt to OCTA were excluded. Finally, patients with significant growth retardation and neurologic abnormalities were also excluded.

Surgical Method

All surgical procedures were performed under general anesthesia by the same experienced strabismus surgeon. In inferio oblique muscle retraction surgery, the eye was retracted towards the superonasal quadrant after placement of a traction suture superotemporal to the cornealimbus border. An incision was made in the inferotemporal conjunctiva 8 mm from the limbus. Blunt dissection was continued until the sclera was exposed. The inferior oblique muscle was then dissected from the surrounding tissues using two strabismus hooks. The muscle was first clamped with clamped 6/0 vicryl sutures Vicryl near the entry site and then cut. The inferior oblique muscle was sutured with 6/0 Vicryl sutures 2 mm temporal and 2 mm posterior to the insertion site of the inferior rectus muscle in patients without DVD pattern and at the inferior rectus level in patients with DVD pattern. Tenon capsule and conjunctiva were sutured with 8/0 Vicryl and the operation was completed.

Optical Coherence Tomography Angiography

Quantitative measurements were obtained with OCTA (Solix, Visionix/Optovue, USA) preoperatively (T0) and one day (PO1), 15 days (PO15) and 6 weeks (PO6w) after surgery. An eye-tracking system was used during angiographic scans. Pupil dilatation was not performed. Scans were concentrated in the center of the fovea and covered a 3×3 mm area of the macula. Retinal vasculature was assessed in three horizontal segmentations: superficial capillary plexus (SCP), deep capillary plexus (DCP) and choriocapillaris (CCL). According to default settings, the SCP layer was considered to be the ganglion cell layer and the inner plexiform layer (60 µm layer thickness from the inner limiting membrane), the segmentation of the inner plexiform layer and outer plexiform layer for the DCP (30 µm thick layer from the inner plexiform layer), and the CCL segmentation with the outer margin of Bruch's membrane as the upper boundary (30 µm thick layer from the outer edge of Bruch's membrane).[15-17] OCTA examinations were repeated if a decentralized condition was detected. Images showing segmentation errors and/or artifacts were excluded from the analysis.

Statistical Analysis

The study data were computerized and evaluated using "SPSS (Statistical Package for Social Sciences) for Windows 25.0 (SPSS Inc., Chicago, IL)". The conformity of continuous numerical variables to normal distribution was evaluated by Shapiro Wilk test. Paired Samples T test was used to compare the data between the times of measurements. Bonferroni correction was used in pairwise comparisons to determine the significant group. 95% confidence interval was used. Data were summarized as mean±standard deviation values. P<0.05 was accepted as statistically significant for all analyses.

RESULTS

The study included 28 eyes of 16 patients. Ten of the patients were female and six were male. The mean age of the patients was 8.07±5.5 years (4-23 years). There was no significant difference in best corrected visual acuity before and after surgery (p>0.05). There were no complications during and after surgery. Inferior oblique muscle function improved significantly in all patients.

Table shows the vascular density values in the SCP, DCP and CCL in the macular region before and after surgery. Accordingly, SCP and DCP mean vascular densities decreased significantly at PO1 and PO15 compared to T0, but increased at PO6 weeks and reached their initial levels (p<0.05, p<0.05, p<0.05, p<0.05, respectively). Mean CCL levels were lower at PO1 compared to all other measurements and reached their preop values at PO6 weeks (p<0.05).

Table. Vascular density in the macula by OCTA before and after inferior oblique muscle surgery

OCT and OCTA measurements	T0	PO1	PO15	PO6w
SCP	48.464±1.426[a]	45.721±1.278	47.386±1.910[a, b]	48.550±1.702[a, c]
DCP	50.986±2.243[a]	48.443±2.279	49.286±2.574[b]	51.043±2.138[a, c]
CCL	55.357±2.822[a]	52.779±2.520	54.150±2.630[a]	56.500±2.308[a, c]

CCL, choriocapillaris layer; DCP, deep capillary plexus; SCP, superficial capillary plexus. Results are given as mean±standard deviation. [a]p<0.05 compared with PO1, [b]p<0.05 compared with T0, [c]p<0.05 compared with PO15

DISCUSSION

In this study, we evaluated the vascular densities in the capillary plexuses (SCP, DCP, CCL) in the macular region before and after inferior oblique muscle surgery with OCTA in patients with IOHF who underwent surgery. Although vascular density decreased in the early postoperative period (postoperative day 1 and day 15), it reached the preoperative values at postoperative week 6.

OCTA can objectively measure the retinal microvasculature in a noninvasive and reproducible manner.[18] It provides a three-dimensional representation of the retinal vascular circulation in the macula in different layers: SCP, DCP and CCL. Numerous studies using OCTA have demonstrated vascular changes in the posterior pole in different diseases.[16,19] Strabismus surgery is especially performed in the pediatric population. Therefore, the fact that OCTA has an eye tracking system and provides high quality photography without pupil dilatation provides an advantage in the field compared to many other examinations.[18] Studies using OCTA in patients undergoing strabismus surgery are available in the literatüre.[10,15,17] However, there is no clear consensus on the effect of surgery on retinal vascular circulation.

In our study in which we performed inferior oblique muscle surgery in 28 eyes of 16 patients and examined the vascular density in the macula with OCTA one day, 15 days and 6 weeks after preop surgery; SCP and DCP mean vascular densities decreased at PO1 and PO15 compared to T0 and increased at PO6w and reached their values at T0, CCL mean levels were lower at PO1 compared to all other measurements and reached their preop values at PO6w. In the retrospective OCTA study by Çelik et al.,[10] which was similar to our study and was conducted with a relatively larger number of patients and eyes, the vascular density in the SCP, DCP, CCL and foveal avascular zone (FAZ) were examined preop, at 1, 7 and 30 days after surgery. In this study, there was no difference in SCP, DCP and FAZ after surgery, and although there was an increase in CCL after one week, it reached its initial levels after one month.

When we look at other strabismus studies performed with OCTA, the study of Vage et al.[15] and the study of Inal et al.[17] are found in the literature. Vage et al.included 92 eyes of 56 patients and performed surgery mostly on the medial retctus and lateral rectus muscles. In this study, DCP and CCL measurements obtained by OCTA increased significantly on the first postoperative day compared to the preoperative values, but again reached the preoperative values on the 30th postoperative day. Inal et al. performed surgery unilaterally on two horizontal muscles in their OCTA study involving 32 eyes of 16 patients. In this study, they found a significant increase in SCP and DCP and a decrease in the foveal avascular area 3 months after surgery. The medial rectus and lateral rectus muscles are also supplied by the anterior ciliary artery.[20] The inferior oblique muscle is supplied by branches from the ophthalmic artery and infraorbital artery.[21] The vascular supply between the horizontal muscles and the inferior oblique muscle may explain the differences between the studies. Previous studies have also shown that retinal blood supply increases in the first days in some studies, but the general opinion is in favor of returning to baseline levels by day 7.[11,20,22]

Atalay et al.[23] in their study including 41 patients in whom they underwent rectus muscle surgery and inferior oblique muscle surgery, they found a greater increase in choroid thickness in patients in whom they underwent rectus surgery than in inferior oblique muscle surgery. They attributed this result to less inflammation in inferior oblique muscle surgery because they made a fornix incision and did not use sutures. They also argued that inferior oblique hyperfunction may have created a traction effect in the macular region and that this traction was reduced by surgery. Celik et al.[10] they found an increase in CCP only in the first postoperative week after inferior oblique muscle surgery. And they attributed this result to inflammation and the possibility that compensatory mechanisms may have developed to prevent anterior segment ischemia. Again, in a recent study involving 18 patients, they concluded that inferior oblique myectomy surgery may increase choroidal hemodynamics in the early period and that this effect is temporary.[25]

There are 3 phases of wound healing: The hypodynamic phase (1), characterized by limited blood loss; the hyperdynamic phase (2), characterized by increased blood flow; and the healing phase (3), which lasts for months and attempts to return the human body to its pre-injury state.24 In our study, our finding of low avacular density in all layers in the early postoperative period can be explained by hypodynamia in the early phase of wound healing. In the 6th week, it returned to its previous levels, which can be explained by the completion of wound healing. When we look at the literature, the effect of surgery on vascular density in OCTA measurements taken in patients undergoing inferior oblique surgery is not yet clear. Differences in study results may be attributed to differences in individual healing response.

Limitations

The small number of cases included in our study is our biggest limitation. Further studies with a larger patient population are needed.

CONCLUSION

Our results demonstrate that transient hemodynamic changes may occur in the SCP, DCP, and CCL following inferior oblique muscle anterior transposition surgery and emphasize the utility of OCTA in assessing vascular changes after strabismus surgery.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was performed with the approval of Fırat University Non-interventional Researches Ethics Committee (Date: 13.02.2024, Decision No: 2024/03-48).

Informed Consent

Written consent was obtained from the patient participating in this study.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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Archives of
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Case Report

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Left posterior cerebral artery occlusion following minor head trauma: a case report

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ABSTRACT

A 17-year-old male patient who received a punch behind the left ear 2 days ago, was admitted to the emergency room with a headache and dizziness. His neurological examination was normal. Brain computed tomography (CT) images demonstrated diffuse hypodense areas and sulcus deletion in the left occipital lobe. On magnetic resonances (MR) images, diffusion restriction for acute ischemia in the left posterior cerebral artery (PCA) watershed area was determined, and hospitalization was recommended. However, he and his relatives left the hospital. Through the electronic information system called "e-nabız", it was learned that the patient was hospitalized in an external center and treated with antiplatelet and anticoagulant regimens for ten days due to an acute thrombus at the left PCA P1 distal-P2 level observed on the brain CT and MR angiography images. In our clinic, his neurological examination was completely normal thirty days later. Still, a visual field examination revealed a total quadrianopsy in the upper temporal pole on the right and left eyes. An encephalomalacia in the left PCA watershed area was observed on CT images. The findings of this case showed that minor head trauma in children can cause large vessel occlusion and therefore close follow-up of these children for a certain time would be appropriate.

Keywords: Posterior cerebral artery, stroke, occlusion, minor head trauma

INTRODUCTION

It has been reported in the literature that dissection-occlusion may occur in intracranial vessels after minor head trauma.[1] Different publications show that 12% of children followed up with acute ischemic stroke (AIS) have a history of trauma, which is independent of energy, up to an average of 12 weeks ago.[2,3] While intracranial hemorrhages may be seen in high-energy traumas, it has been noticed that vascular occlusions may occur after low-energy traumas. Due to this ischemia, a wide variety of permanent or temporary sequelae may appear depending on the affected area of the brain.[1]

In this case report, the patient who developed posterior cerebral artery (PCA) occlusion after minor head trauma was discussed.

CASE

A 17-year-old male patient presented to the emergency department of our hospital with a headache and dizziness. It was learned from his anamnesis that he was punched behind the left ear two days ago during a fight with his friend and was discharged from the hospital after no abnormal findings were detected in computed tomography (CT) images of the brain taken at an external center. During the examination performed at our center, he was conscious, cooperative, and oriented, and had a Glasgow Coma Scale (GCS) score of 15/15. Cranial nerve and cerebellar examination findings were found to be normal. No deficit or pathological reflex was detected in the motor examination. In addition, it was learned from the patient's medical history that he had no known comorbidities and no medication use. Based on the patient's clinical findings, it was suspected that there might be an intracranial lesion and a brain CT was performed. CT images showed diffuse hypodense areas and sulcus effacement in the left occipital lobe (Figure 1). Brain magnetic resonances (MR) images revealed diffusion restriction in the anterior part of the left thalamus and the left PCA watershed area, and hospitalization was recommended; However, patients and their relatives left the hospital voluntarily (Figure 2). Through the electronic

information system (called "e-nabiz"), it was learned that the patient was admitted to an external center and his treatment started on the same day. Brain CT-angiography and MR-angiography images taken in this external center revealed an acute thrombus in the lumen of the left PCA P1 distal-P2 level and there was no filling in the distal part (Figure 2). It was learned that the patient was discharged with full recovery under "acetylsalicylic acid" and "enoxaparin sodium" regimens after a ten-day treatment period at the relevant external center.

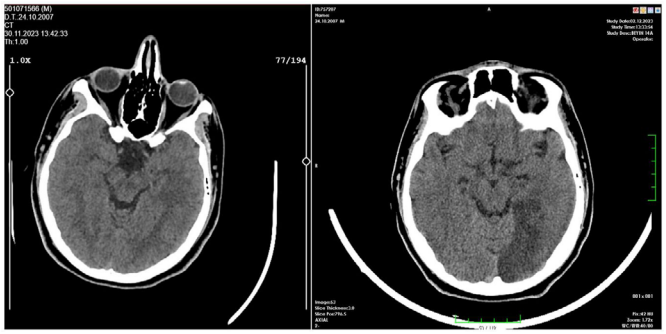

Figure 1. The patient's early CT scans at an external center were normal (left). A hypodense area (ischemia?) located in the left occipital lobe was detected in CT images taken in our hospital two days after the trauma (right)

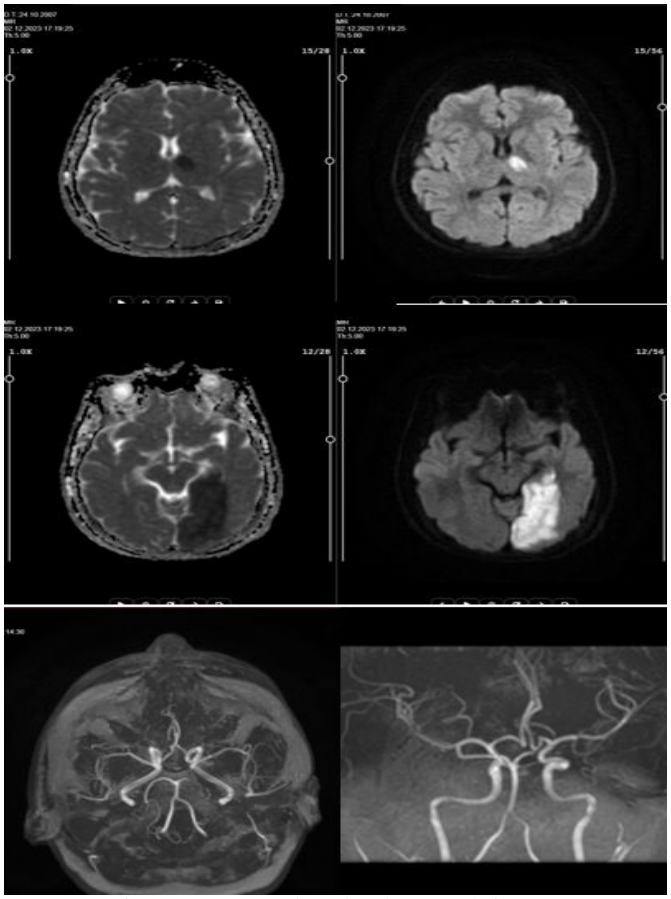

Figure 2. Diffusion MR images show that there was diffusion restriction in favor of acute ischemia in the anterior part of the left thalamus and the left PCA irrigation area. Brain MR-angiography images showed that there was no filling in the patient's left posterior cerebral artery after the P1 segment

One month later, the patient applied to our outpatient clinic for a check-up. The neurological examination was found to be normal. A loss of density consistent with chronic ischemia was observed in the control brain CT images taken of the patient (Figure 3). Following the eye examination, the direct and indirect light reflexes of the right and left eyes were normal, and the visual acuity value was found 20/20, the biomicroscope examination of the right and left eyes showed natural findings, the fundus, and optic disc, and macula findings of the right and left eyes were normal, the pupils of the eyes were normal. It was learned that there was no tear, hole, or detachment in the retina after dilatation with the applied pharmacological agent. In the visual field examination, total quadrianopsia was detected in the upper pole of the temporal field on the right, and partial hemianopia in the upper pole of the temporal field on the left. Examination performed by Pediatric Hematology revealed no coagulation disorder/bleeding diathesis. Blood biochemistry and coagulation parameters were performed. In the results, there were seen that D-dimer was under 0.19 mg/L (reference range < 0.55mg/L), fibrinogen was 1.97g/L (reference range 1.7-4.2 g/L), activated partial thromboplastin time (aPTT) was 26.2 seconds (reference range 21-32 seconds), prothrombin time (PT) was 12.6 seconds (reference range 9.8-14 seconds), prothrombin activity was 68.7% (reference range 70-130%), international normalized ratio (INR) was 1.14 INR (reference range 0.8-1.2 INR), c-reactive protein (CRP) was 0.9 mg/L (reference range 0-5 mg/L), collagen and epinephrine induced bleeding time was more than 300 seconds (reference range 82-150 seconds), collagen and adenosine diphosphate (ADP) induced bleeding time was 80 seconds (reference range 60-100 seconds), troponin-I H was under 0.0025 ng/mL (reference range <0.045ng/mL). In addition, no thrombus or anomaly was detected in the heart cavities, heart valves, and main heart vessels by examination of Pediatric Cardiology. Finally, the carotid artery and vertebral artery Doppler ultrasonography revealed no thrombus, dissection, plaque, aneurysm, or filling defect in the relevant vessels.

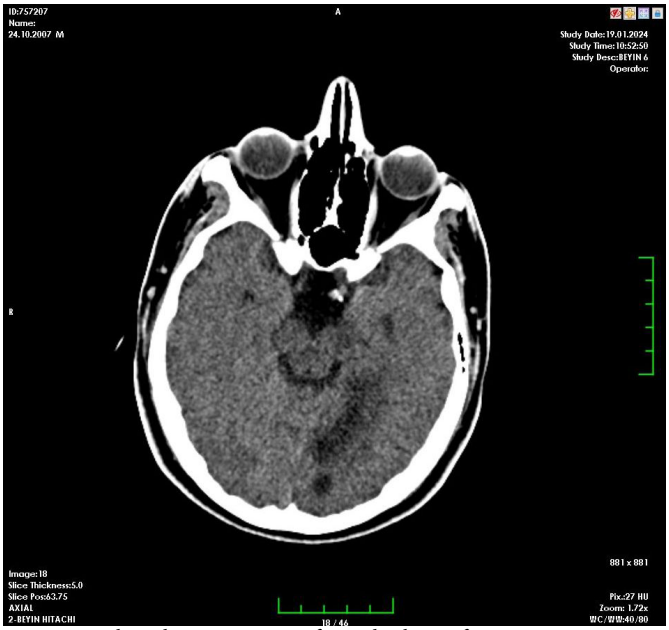

Figure 3. When the patient came for a check-up after receiving inpatient treatment, areas of chronic ischemia were seen on the CT images taken in our hospital

DISCUSSION

Headache is the third most common reason why pediatric patients visit the emergency department.[4] It is known that headaches are mostly caused by upper respiratory tract infections or sinusitis.[5] It was learned that the patient

received a fist blow behind the left ear two days ago and had no complaints other than headache and dizziness, which continued to increase within forty-eight hours. At the time of admission, it was observed that blood pressure was within the normal range, there was no fever, and respiratory and pulse rates were normal and rhythmic. The physical examination was normal. According to the anamnesis, it was learned that there was no acquired or hereditary disease. Considering that the patient presented with complaints that increased after trauma, CT scans were taken due to the possibility of intracranial disorders (subarachnoid hemorrhage, epidural hemorrhage, etc.), and an infarct area in the PCA watershed on CT images was seen. Therefore, it was thought that the patient might have a pediatric ischemic stroke.

It has been reported in the literature that pediatric acute ischemic stroke (pAIS) syndrome occurs mostly in male children (60%) and that even if treated, two-thirds of the patients may have sequelae of various degrees.[2] One of the most common causes of pAIS is cardiac problems (heart valve diseases, large vessel diseases, cardiac anomalies, rheumatic diseases, etc.).[6,7] Another important factor is arteriopathies, and the most common arteriopathy is a focal occlusive disease of the terminal internal carotid or proximal middle/anterior cerebral artery, and these arteriopathies may be triggered by infection and inflammation.[1,2,6-8] In our patient, infection was not considered based on his history, clinical findings, and blood biochemistry test results at the time of admission. In addition, echocardiography revealed neither thrombus nor anomalies in the heart cavities, heart valves, and main heart vessels. Finally, the carotid and vertebral artery Doppler ultrasonography examinations showed no filling defect in the carotid and vertebral arteries. With these findings, it was thought that the patient's PCA occlusion could not be of cardiac or main vasculature origin.

Additionally, it is known that hematological disorders (bleeding diatheses, coagulation disorders, etc.) may cause pAIS. Although it has been reported that factors such as protein S, antithrombin III, plasminogen and plasminogen activator inhibitor (PAI) deficiencies, lipoprotein (a) elevation, antiphospholipid syndrome, and hyperlipidemia may also cause pAIS, in terms of prothrombic factors, Factor V (Leiden) mutation, protein C deficiency, and prothrombin mutation are the most common causes.[1]

In the present case, the Pediatric Hematology consultation revealed no hematological disease. On the other hand, no visual field defect could be detected in the patient with confrontation, the Ophthalmology Department revealed no pathological findings originating from the orbit. However, in the automatic perimetry examination, total quadrianopsy was detected in the upper pole of the temporal field on the right eye, and partial hemianopsia in the upper pole of the temporal field on the left eye. With these findings, although the neurological examination findings were found to be normal, it was concluded that objective examinations should support the findings.

Additionally, it has been reported that pAIS can occur as a result of minor or major head trauma.1 This is thought to be due to the anatomical location of the brain vessels of trauma in pediatric patients. It is known that brain parenchymal vessels in children are present at a steeper angle than in adults. For this reason, the vessels can be stretched and ruptured due to the tension force and high inertia that occur after trauma. If the damage occurs with high energy, it causes bleeding due to complete dissection. On the other hand, trauma with lower energy causes damage to the vascular endothelium due to partial dissection. The inflammatory process secondary to this damage to the vascular endothelium leads to traumatic endothelial intimal lesions, followed by fibrin accumulation, leukocyte reaction, and white thrombus formation, resulting in lumen obstruction. This occlusion causes cerebral parenchymal ischemia with clinical findings after a symptom-free latent period.[1,3] After the patient's history, consultations, radiological imaging, and biochemical research results, it was thought that the acute-subacute infarct area in the patient's left occipital lobe might be caused by minor trauma behind the patient's left ear.

Moreover, it has been reported that the time to emergence of neurological disorders (such as headache, dizziness, nausea, hemiparesis, drowsiness, and confusion) may be shorter in children with pAIS. For this reason, it has been reported that waiting for a focal deficit that continues for 24 hours, as in adults, may cause a delay in the correct diagnosis and treatment process.[6] In the literature, recombinant tissue plasminogen activator (r-tPA) and/or heparin infusion are recommended in adults if PCA or other main vessel occlusions are detected within the first four hours of their occurrence. If this period is exceeded, heparin, anticoagulant, and antiplatelet treatments can be applied. However, considering the severity of hemorrhagic complications in pediatric patients, r-tPA or heparin infusion is not recommended, instead acetylsalicylic acid (ASA), low molecular weight heparin, or coumadin is preferred. While very long-term treatment is recommended in pAIS of cardiac origin, it is recommended to keep the treatment duration shorter in post-traumatic pAIS.[6,9,10] Since the patient's vital signs were stable, there was no significant neurological loss, and the diagnosis of PCA occlusion could be made forty-eight hours after the minor trauma, it was observed that the patient was treated at an external center with acetylsalicylic acid as an antiplatelet and low molecular weight heparin (enoxaparin sodium) as an anticoagulant. The fact that no progression was detected in the patient's radiological imaging after 30 days suggested that the treatment given was effective.

In conclusion, it is recommended that pediatric patients with asymptomatic minor head trauma and no abnormal findings detected in the first brain CT images taken should be followed closely and that patients who are symptomatic in the future should be subjected to detailed radiological, hematological, and cardiological and laboratory examinations in terms of complications that may occur secondary to trauma and it was argued that families should be informed in detail in advance about possible symptoms and complications.

ETHICAL DECLARATIONS

Informed Consent

The signed written informed consent form was obtained from the patient's parents.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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Case Report

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A Kabuki syndrome presenting with strabismus: case report

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ABSTRACT

Kabuki syndrome affects many systems and is characterized by a typical facial appearance and is a genetically inherited disease. Ophthalmological involvement is quite common and can be diagnostic. In this article, we aimed to present the ophthalmological findings of our patient with Kabuki syndrome, who was diagnosed and treated based on ophthalmological findings.

Keywords: Kabuki syndrome, strabismus, KMT2D gene defect

INTRODUCTION

Kabuki syndrome was first described in Japan and is a genetically inherited syndrome, also known as Kabuki make-up syndrome or Niikawa-Kuroki syndrome, characterized by a typical facial appearance.[1] Although its inheritance pattern and etiology have not been elucidated yet, it is thought to be inherited in an autosomal dominant manner. Mutations are frequently found in the KMT2D, KDM6A, and MLL2 genes. In addition to the typical dysmorphic facial appearance, bone and skeletal deformities, cardiac anomalies, urinary system anomalies, gastrointestinal system anomalies, growth retardation, and psychomotor retardation may be observed. Ophthalmologic involvement is observed quite frequently and may be diagnostic.[2,3]

In this article, we aim to present the ophthalmologic findings of our patient with Kabuki syndrome, who was diagnosed with ophthalmologic findings and treated.

CASE

A 13-year-old male patient was admitted to our outpatient clinic with ocular misalignment. The best corrected visual acuity (BCVA) was 0.8 (1.50 axis 135) in the right eye and 0.9 (+0.50-1.25 axis 30) in the left eye using Snellen's threshold. Stereopsis was evaluated as an 800-second arc. Distance and near shift were evaluated as 40 prism diopters (PD) esotropia with an alternating on-off test. There was a-1 restriction on outward gaze in bilateral eyes. The patient also had bilateral ptosis. The right eyelid gap was 8 mm, the left eyelid gap was 8 mm, and the and the margin-reflex distance (MRD) was 1 mm right and 2 mm left. Levator function was measured at 11 mm bilaterally. There was a long palpebral fissure, and the laterals of the lower eyelids were turned outward. Lateral parts of the eyebrows were sparse. When we evaluated the anterior segment, the corneas were transparent, with a horizontal diameter of 10 mm and a vertical diameter of 9.5 mm bilaterally. The lens was clear, and no pathology was found in the fundus examination. The axial length measurements of the patient were 22.31 mm in the right eye and 22.28 mm in the left eye.

The patient had strabismus since the age of 2 years, but this condition was initially evaluated as pseudoesotropia, and no treatment was recommended. She used to wear glasses sometimes. The degree of strabismus has increased over the years.

Attentional hyperactivity and mild mental retardation were present. The patient with dysmorphic facial appearance, large prominent ears, and a single kidney was referred to the pediatrics and genetics unit for a multisystemic evaluation. Genetic analysis revealed a pathogenic KMT2D gene defect.

After consecutive follow-up with the patient, a 6 mm retraction operation was performed on the bilateral medial rectus due to esotropia. During the operation, gray-black pigmentation was observed in the sclera, 8mm behind the insertion in the medial rectus trace. The patient's shifts were orthophoric at the 2nd week and 3rd month postoperatively. No intervention was performed for ptosis or valve anomalies (Figure).

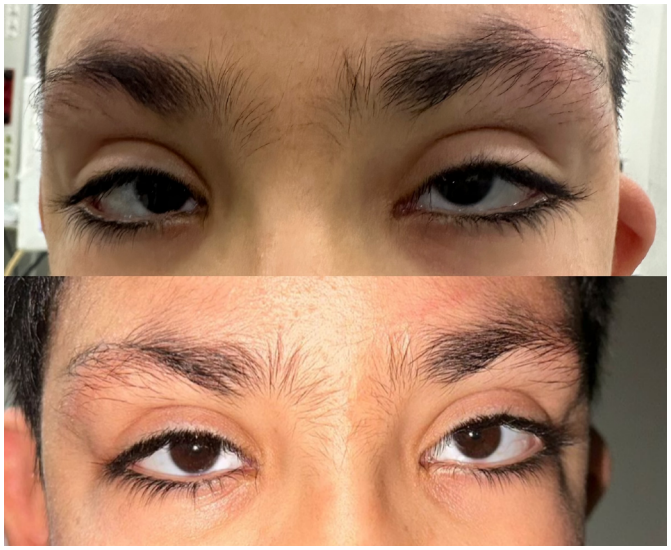

Figure. Picture of the patient before and after the operation

DISCUSSION

Kabuki syndrome is a multi-systemic, genetic disease characterized by mental retardation, developmental delay, musculoskeletal anomalies, and a typical facial appearance. The incidence in the community is estimated to be 1 in 32 thousand.[1] The characteristic facial appearance observed in Kabuki syndrome has been defined as long palpebral fissures, eversion of the lower eyelids, prominent eyelashes, a more prominent brow arch, sparseness of the eyebrows, flattening of the nasal tip, and remarkably large ears.[4] In ophthalmologic involvement, ptosis, esotropia, microcornea, corneal opacity, cataract, nystagmus, blue sclera, iris, retina, choroid, and optic disc coloboma, retinal telangiectasia, and pigmentation may be observed.[5] Other findings include cardiovascular system anomalies, gastrointestinal system and renal anomalies, autistic findings, and behavioral disorders.[6] Although the first striking feature in the diagnosis is the characteristic facial appearance, not all patients with this facial appearance are diagnosed with Kabuki syndrome. An important part of the components constituting this facial appearance is ophthalmologic. Şenel et al.[7] reported a case in which the diagnosis was based on nystagmus, coloboma, and microphthalmia findings. In our case, strabismus, microcornea, long palpebral fissures, ptosis, and eyelid disorders were present.

De novo or inherited pathogenic variants in the KMT2D gene are the most common cause of Kabuki syndrome, accounting for approximately 75% of patients. Mutations in the KDM6A gene account for approximately 5% of patients. Kabuki syndrome is very rare in Turkiye, and in a recent study by Usluer et al.,[8] a KMT2D mutation was found in 21 of 32 patients (65%) who were diagnosed with Kabuki syndrome.[2,3] Our patient's typical facial appearance, ophthalmologic findings, existing renal anomaly, and KMT2D gene mutation found in genetic analysis supported the diagnosis of Kabuki syndrome.

Strabismus is the most common ophthalmologic finding in children with Kabuki syndrome, and its frequency was found to be approximately 20% in a study by E. Ming et al.[9] in our patient, esotropia and limitations of outward gaze movement in both eyes were observed on examination at presentation. It was thought that the limitation in outward gaze was due to esotropia, which was present for a long time.

CONCLUSION

After strabismus surgery due to esotropia, we achieved an orthophoric appearance in the eyes, although we could not achieve a significant improvement in stereopsis due to the advanced age of the patient. In conclusion, ophthalmologists may assume the initiating role in the diagnosis of Kabuki syndrome, and the ophthalmologist's treatment approach to the existing findings plays an important role in improving the quality of life of patients with Kabuki syndrome. However, patients with typical examination findings should be referred to other related branches, and a multidisciplinary approach should be organized.

ETHICAL DECLARATIONS

Informed Consent

The patient's parents signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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Case Report

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Isolated medial rectus hematoma following blunt trauma-a novel case report

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ABSTRACT

This report presents a rare case of isolated medial rectus hematoma following blunt trauma in a 16-year-old male. Typically, traumatic eye injuries lead to orbital fractures or hemorrhages, but isolated rectus muscle injury and hematoma are exceptionally uncommon. Our patient exhibited unilateral total outward gaze limitation, esotropia, and horizontal diplopia without any associated cranial or orbital fractures. Orbital computed tomography and magnetic resonance imaging played pivotal roles in diagnosing the isolated hematoma, which was managed conservatively with oral corticosteroids, leading to complete resolution of symptoms and radiologic findings within a month. This case underscores the importance of thorough clinical examination and careful evaluation of imaging in the differential diagnosis of posttraumatic diplopia, highlighting the diagnostic challenges and the clinical utility of orbital imaging in such rare presentations.

Keywords: Blunt eye trauma, pediatric ocular trauma, post-traumatic diplopia, post-traumatic rectus hematoma.

INTRODUCTION

In the spectrum of trauma affecting the orbit and its proximate structures, occurrences of orbital fracture or hemorrhage are relatively common outcomes. However, the manifestation of isolated rectus muscle injury and hematoma following blunt trauma is an exceptionally rare complication, adding intricacy to the clinical landscape.[1] Within this context, we present the case of a 16-year-old male patient who experienced an isolated medial rectus hematoma subsequent to blunt trauma. While the literature acknowledges spontaneous rectus hematomas, the body of evidence is limited, with only a few documented cases of isolated rectus hematoma.[1-3] Notably, our case report contributes a unique dimension to this rarity, as, to the best of our knowledge, no prior instances of isolated medial rectus hematoma have been documented in the existing literature.

Informed consent form was obtained from the child and his parents.

CASE

A 16-year-old male presented to the emergency department with complaints of a red eye and diplopia following an injury involving a screwdriver to his right eye. The patient reported no loss of consciousness, dizziness, nausea, or vomiting. Notably, visual acuity was 20/20 in both eyes, and intraocular pressure was within normal limits. Clinical examination revealed an absence of edema and ecchymosis in the periocular structures. Anterior and posterior segment examination through biomicroscopy showed no abnormalities, except for a significant subconjunctival hemorrhage in the lower nasal quadrant of the right eye. Light reflexes were normal, and relative afferent pupillary defect was not observed. However, the patient exhibited a-4 limitation in lateral gaze in the right eye, along with esotropia measuring 10 prism diopters at near and 20 prism diopters at a distance. Furthermore, hypertropia of 4 prism diopters on the right was noted, accompanied by horizontal diplopia exacerbated in right gaze (Figure 1).

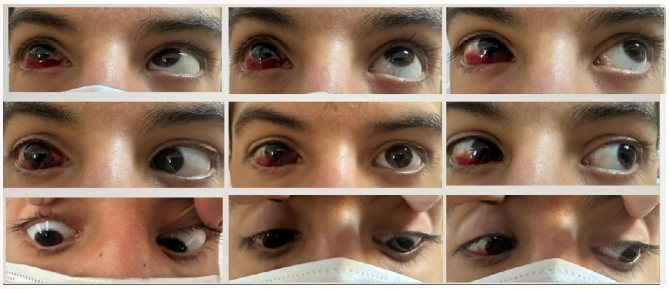

Figure 1. The patient exhibits nine cardinal gaze positions: A notable limitation of -4 in the right gaze accompanied by esotropia, which persists even in the primary position. Other than the identified subconjunctival hemorrhage in the right inferonasal quadrant, the ocular structures and their appendages appear grossly normal upon examination

The neurological examination conducted in the emergency department yielded unremarkable findings. In response to a

suspected traumatic sixth nerve palsy, an urgent cranial computed tomography (CT) scan was conducted, revealing no discernible pathology. Subsequently, orbital magnetic resonance imaging (MRI), prompted by the suspicion of an orbital hematoma, identified an area situated slightly lateral to the proximal 1/3 of the optic nerve, positioned 13 mm posterior to the disc level in the right orbit. However, the accurate assessment of its size and intensity was hindered by a magnetic susceptibility artifact, as illustrated in Figure 2A. Following the suspicion of a potential metallic foreign body, radiology was asked to carefully re-evaluate the patient's orbital CT, which was performed 1 day before under emergency conditions. Contrary to expectations, the CT scan elucidated "asymmetric thickening and coarse densities in the central part of the right medial rectus muscle." The findings were subsequently interpreted as indicative of an isolated medial rectus hematoma, as illustrated in Figure 2B.

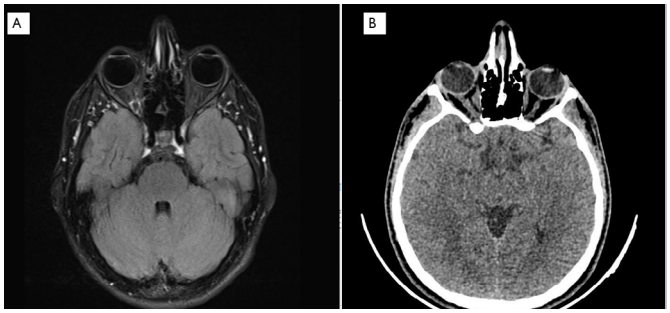

Figure 2A. Contrast-enhanced orbital MRI, axial T1-weighted with fat suppression. The image reveals an anomalous region situated slightly lateral to the proximal 1/3 of the optic nerve, positioned 13 mm posterior to the disc level in the right orbit. Unfortunately, the precise assessment of its size and intensity is impeded by a magnetic susceptibility artifact, as indicated in the description
Figure 2B. Axial CT scan: asymmetric thickening and coarse densities in the central right medial rectus

Oral methylprednisolone at a dosage of 30 mg/day was administered to the and regular weekly follow-ups were conducted. The corticosteroid dose was systematically tapered and eventually discontinued after three weeks of treatment. Notably, at the first-month follow-up, a remarkable restoration of ocular movements was evident, accompanied by a complete reversal of the previously observed findings on radiologic images, as depicted in Figure 3.

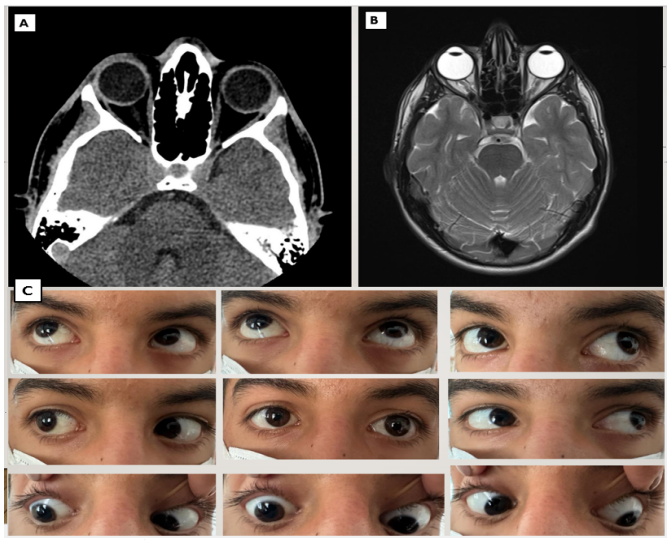

Figure 3A. Axial CT scan
Figure 3B. Contrast-enhanced MRI axial T2 weighted. In this follow-up radiologic assessment conducted one month after the trauma, both images exhibit the complete resolution of the medial rectus hematoma without any lingering sequelae
Figure 3C. One month after trauma, as seen in nine cardinal gaze positions, unimpeded ocular movements in all directions, and orthophoria was achieved

DISCUSSION

Traumatic eye injuries cover a wide variety of conditions and can adversely affect the appearance and function of the eyeball and orbit. Additionally, orbital hemorrhages and subperiosteal hematomas are prevalent complications.[4] The rigid nature of orbital walls renders them susceptible to substantial soft tissue damage, even with minor injuries.[5] Orbital fractures and hemorrhages are common sequelae of blunt trauma and the clinical presentation is almost never limited to an isolated single muscle. While medial rectus injuries themselves are not exceedingly rare, the specific presentation of an isolated medial rectus hematoma without associated orbital fractures or other structural injuries is particularly unusual.

Orbital injuries frequently implicate the medial and inferior rectus muscles as the most commonly affected extraocular muscles. Extraocular muscle injuries often manifest as traumatic ruptures of the rectus muscles and are commonly associated with orbital fractures, hemorrhages, or globe injuries. While the literature commonly reports spontaneous rectus muscle hematomas, particularly in the inferior rectus muscle, the occurrence of an isolated muscle injury or hematoma following trauma without concurrent structural damage in the orbit is exceptionally rare.[3]

Our report highlights a case of unilateral total outward gaze limitation and esotropia resulting from an isolated medial rectus hematoma following trauma, accompanied by horizontal diplopia in the affected patient. When confronted with a similar clinical presentation, it is imperative to rule out cranial pathologies in the differential diagnosis. Key considerations encompass intracranial hemorrhage, potential brain stem injury, skull base fractures, traumatic cavernous sinus or orbital apex injuries, as well as peripheral abducens (6th) nerve injuries. It is crucial to differentiate our case from trapdoor fracture or orbital retrobulbar hemorrhage, because in these cases conservative treatment may lead to irreversible visual loss. Therefore, a meticulous diagnostic approach is essential to guide appropriate therapeutic interventions and safeguard visual function in affected individuals.[5-7]

The 6th cranial nerve is vulnerable to trauma due to its long intracranial path. Trauma-related 6th nerve palsy, which manifests as limitations in eye abduction, esotropia, and horizontal diplopia.[6] In case of any suspected head trauma, the 6th nerve may be injured anywhere from the dorsal pons to the lateral rectus muscle.[5]

Increased intracranial pressure from intracranial hemorrhage often leads to papilledema and usually bilateral 6th nerve palsy.[7] In 6th nerve palsies, as a characteristic of paralytic incomitant strabismus, the forced duction test is negative, and is therefore one of the leading differential diagnoses in our case, and it should be kept in mind that it can occur in the absence of cranial or orbital fracture. Trauma is the most common cause of isolated 6th nerve palsy in children, with skull base fractures being the most frequent pathology.[8] In our case, skull base or caudal fractures and intracranial hemorrhage were ruled out by neurological examination and cranial imaging upon presentation to the emergency department.

The most common cause of posttraumatic diplopia is mechanical entrapment of the soft tissues due to a blow-out fracture.[5] The medial wall of the orbit, delineated by the fragile lamina papyracea, is particularly vulnerable to trauma from both external injuries and functional endoscopic sinus surgery. This area's susceptibility often results in complications such as diplopia and movement restriction due to muscle compression or damage to its vascular or neural supply.. The medial rectus muscle, adjacent to the thin lamina papyracea, is most often affected during endoscopic sinus surgery due to these anatomical vulnerabilities.[9] Hemorrhage and edema of the orbital fat that causes the tension of the septae may also restrict ocular movement. In both of these conditions, the forced duction test is positive. However, in cases of injury to the ocular muscles, the forced duction test may be negative and the diagnosis may be confused with< paralysis.[2]

Trapdoor fractures in children result from a sudden increase in intraorbital pressure, causing a bone flap to temporarily displace and then return to its original position. Children's bones, with more osteocytes and less calcified tissue, are more flexible and less prone to fragility, contributing to the distinct characteristics of trapdoor fractures in this age group.[7] The weaker bony adhesions of the periosteum in children also make them susceptible to subperiosteal hematoma after trauma, regardless of an orbital fracture.[4,10] In our case, the absence of a positive forced duction test and no orbital fractures on CT ruled out a trapdoor fracture or soft tissue entrapment.[3] Prompt surgical intervention is crucial for entrapment of extraocular muscles or soft tissue in an orbital bone fracture due to the risk of severe complications, including bradycardia, persistent diplopia, and mortality, making it a top priority in differential diagnosis.[7] Subperiosteal orbital hematomas pose a management challenge, as they may require conservative treatment or urgent surgery if there's a risk of compartment syndrome and optic nerve damage. Typically located in the upper orbital wall, these hematomas present with proptosis, hypoglobus, limited upward gaze, and reduced visual acuity. Orbital CT is crucial for accurate diagnosis, guiding treatment, and timely intervention to prevent complications.[10]

In guiding our differential diagnosis, the patient's trauma history and clinical observations are crucial. An isolated rectus muscle hematoma, unlike acute diffuse orbital hemorrhage, suggests a mechanism linked to direct injury to the rectus muscle area rather than changes in intra-orbital pressure seen in blow-out fractures. This type of hematoma is less likely to cause compartment syndrome affecting the optic nerve than high-pressure arterial hemorrhage. Acute orbital hemorrhage typically leads to rapid, painful proptosis, significant eye movement restriction, and optic nerve damage.[11] In our case, the fact that visual acuity and optic nerve functions were not affected, proptosis was not observed and the relatively mild clinical progression leads us away from this diagnosis. However, differentiating isolated rectus hematoma from other posttraumatic diplopia causes is challenging due to the lack of distinct clinical features. Notably, evidence of hemorrhage becomes discernible when the hematoma extends along the muscle sheath, reaching its insertion on the globe.[2] In our case, a subconjunctival hemorrhage was evident along the course of the muscle.

Orbital CT is particularly diagnostic in the diagnosis of isolated rectus hematoma and sections should be carefully examined according to the suspected muscle. Coronal sections for vertical rectus hematomas and axial sections horizontal rectus should be especially evaluated.[1,8] The CT scan revealed distinctly demarcated, irregular coarse densities within the medial rectus muscle, with no pathological findings observed in the adjacent orbital tissues. In patients with isolated rectus muscle hematoma treated conservatively, gradual symptom improvement may confirm the diagnosis, while rapid worsening may necessitate reevaluation.[1] In our case, we initiated oral steroid therapy at a moderate dosage, gradually tapering the dose over time. Existing literature supports the use of oral corticosteroids as a primary medical intervention for managing orbital hematomas and hemorrhages, though conservative approaches have also been proposed. Oral steorides play a crucial role in alleviating edema and spasm within the microcirculation, contributing to a reduction in intra-orbital pressure.[11]

In our case, concerns regarding a potentially metallic foreign body, raised during the orbital MRI examination, were effectively addressed through the use of orbital CT. While MRI is generally superior, particularly in visualizing soft tissues, it can be susceptible to focal field inhomogeneity artifacts. These artifacts, also known as magnetic susceptibility artifacts, arise from localized distortions in the magnetic field. Magnetic susceptibility, indicating a tissue's magnetization capacity, plays a role in these artifacts. Both metallic fragments and blood breakdown products have been identified as culprits for these artifacts. On MRI, these artifacts typically manifest as signal gap areas or concentric patterns of bright and dark circles.[12] The suspicion of a metallic foreign body mentioned on MRI was probably a magnetic susceptibility artifact due to hemorrhage and was eliminated on CT.

CONCLUSION

This report presents a rare instance of an isolated medial rectus muscle hematoma following blunt trauma, a condition not previously documented in the literature. It underscores the importance of a thorough examination of trauma history and clinical presentation in diagnosing this challenging entity. The crucial role of orbital CT in identifying and differentiating this condition from other cranial and orbital causes is highlighted, demonstrating its clinical utility. Our case emphasizes the need for careful evaluation of imaging in emergency conditions and revisiting radiologic images when the clinical picture is unclear, to avoid unnecessary tests and time wastage. Additionally, it reinforces that a negative forced duction test does not rule out muscular pathology in cases of isolated gaze limitation following blunt trauma.

ETHICAL DECLARATIONS

Informed Consent

Informed consent form was obtained from the child and his parents.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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