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Konovalova N.V., Zborovska O.V.*, Buşuyeva N.M.*, Guzun O.V.*, Kovtun O.V.

OFTALMOLOGİYADA ARNOLD-KİARİ SİNDROMU (KLİNİK HALLAR)

Odessa Milli Tibb Universiteti, Ukrayna Səhiyyə Nazirliyi, Odessa, Ukrayna

Korrespondensiya üçün:
Konovalova Natalya Valeryevna, t.e.d., professor, "Ukrayna Milli Tibb Elmləri Akademiyasının V.P.Filatov adına Göz Xəstəlikləri və Toxuma Terapiyası İnstitutu" Dövlət Müəssisəsinin "Gözün iltihabi xəstəlikləri" şöbəsinin baş elmi işçisi
E-mail: kvnkonovalova@gmail.com
https://orcid.org/0009-0001-8164-4654

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XÜLASƏ

Arnold-Kiari sindromu kəllənin arxa çuxurunun (posterior kranial fossa) ölçüləri ilə beyin strukturlarının yerləşməsi arasındakı uyğunsuzluq nəticəsində yaranan, beyinciyin (serebellum) və beyin kötüyünün (serebrum) onurğa kanalının (spinal kanal) böyük dəliyindən (foramen) aşağıya doğru yerdəyişməsi ilə xarakterizə olunan patologiyadır.

Məqsəd – Arnold-Kiari malformasiyasının oftalmologiyada diferensial diaqnostikasına kompleks yanaşmanın nəticələrini iki xəstənin nümunəsi əsasında qiymətləndirmək.

Material və metodlar
Arnold-Kiari malformasiyası ilə 2 pasiyent (4 göz) əsas oftalmoloji metodlar ilə yanaşı optik koherens tomoqrafiya (OKT) üsulu vasitəsilə müayinə edilmişdir.

Nəticələr
Arnold-Kiari sindromunun heç bir patoqnomonik simptomu yoxdur; diaqnostika yalnız neyrovizualizasiya üsulları ilə mümkündür. Görmə sinirin diskinin iltihab, durğunluq əlamətləri və ya atrofiyası hallarında oftalmoloq ilk növbədə xəstəni maqnit-rezonans tomoqrafiya (MRT) və ya kompüter tomoqrafiya (KT) müayinələrinə yönləndirməlidir. Müvafiq oftalmoloji dəyişikliklərin erkən mərhələdə müəyyən edilməsi və neyrovizualizasiya müayinə üsullarının tətbiqi bu anomaliyanın vaxtında aşkar edilməsinə imkan verir.

Yekun
Müasir diaqnostika və müalicə yanaşmaları müxtəlif ixtisas sahələrinin – oftalmoloqların, radioloqların və nevroloqların – sıx əməkdaşlığını tələb edir və həkimlər qarşısında yeni vəzifələr müəyyənləşdirir. Təqdim olunan klinik hallar Arnold-Kiari sindromuna məxsus əlamətlərini, o cümlədən çəpgözlük və görmə sinirinin durğunluğunu nümayiş etdirmiş, bu da MRT müayinəsi ilə təsdiq edilmişdir.

Açar sözlər: *Arnold-Kiari sindromu, optik koherent tomoqrafiya, optik sinir, maqnit rezonans tomoqrafiya*

**V.P. Filatov adına Göz Xəstəlikləri və Toxuma Terapiyası İnstitutu, Ukrayna Milli Tibb Elmləri Akademiyası, Odessa, Ukrayna*

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Konovalova N.V., Zborovska O.V.*, Bushueva N.M.*, Guzun O.V.*, Kovtun O.V.

ARNOLD-CHIARI SYNDROME IN OPHTHALMOLOGY (CLINICAL CASES)

Odessa National Medical University, Ministry of Health of Ukraine, Odesa, Ukraine

For correspondence:
Konovalova Natalia Valerievna, DMSc, Professor, Senior Researcher of the Department of Inflammatory Eye Diseases of the State Institution "V. P. Filatov Institute of Eye Diseases and Tissue Therapy of the National Academy of Medical Sciences of Ukraine"
E-mail: kvnkonovalova@gmail.com
https://orcid.org/0009-0001-8164-4654

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Authors participation:
Concept and design of investigation: Konovalova N.V., Zborovska O.V.
Material collection and processing: Bushuyeva N.M., Guzun O.V., Kovtun O.V.
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SUMMARY

Arnold-Chiari syndrome is a pathology manifested by displacement of the cerebellum and cerebrum through the foramen of the spinal canal, inherited from the inconsistency of the dimensions of the posterior cranial fossa and placement brain structures.

Purpose – to evaluate the results of a comprehensive approach to the differential diagnosis of Arnold-Chiari malformation in ophthalmology using two patients as an example.

Materials and methods
2 patients (4 eyes) were examined for Arnold-Chiari malformation by generally accepted methods, and optical coherence tomography (OCT).

Results
There is no pathognomonic symptom or group of symptoms that allow for the clinical diagnosis of Arnold Chiari syndrome, and only neuroimaging diagnostics are possible. Whenever there is inflammation, congestive disc, or optic nerve atrophy, the first action of the ophthalmologist is to refer the patient for magnetic resonance imaging (MRI) or computed tomography (CT) imaging. Identification of relevant ophthalmological disorders at an early stage allows for timely suspicion and detection of this anomaly. Neuroimaging diagnostics justifies the doctor's alertness to this pathology.

Conclusion
Current diagnostic and treatment advances bring together specialists from various fields – ophthalmologists, radiologists, and neurologists – and present new challenges for physicians. The presented series of clinical cases demonstrated possible manifestations of Arnold-Chiari syndrome, including strabismus and congestion in the optic nerve, which was confirmed by MRI.

Key words: *Arnold-Chiari syndrome, optical coherence tomography, optic nerve, magnetic resonance imaging*

**V.P. Filatov Institute of Eye Diseases and Tissue Therapy, National Academy of Medical Sciences of Ukraine, Odesa, Ukraine*

Against the background of rapid brain growth and relatively slow growth of the skull bones, some anomalies are congenital in nature, including Arnold-Chiari malformation. This malformations of type I are the most common malformations of the cranio-vertebral junction in adults, often combined with each other and manifesting synchronously, are very important anomalies that lead to instability and compression in the occipital-cervical junction and have complex clinical characteristics [1, 2]. The frequency ratio is estimated from 2.4 per 100,000 in children and from 9.6 to 19.7 per 100,000 in adults [3].

Arnold-Chiari syndrome is a pathology characterized by displacement of the cerebellum and medulla oblongata through the occipital foramen into the spinal canal due to a mismatch in the size of the posterior cranial fossa and the brain structures located in it [4, 5].

Chiari malformation was first described by Cleland in 1883. Then the syndrome was described in 1891 by pathologist G.Chiari (Austria) and in 1894 by professor of pathology Y.Arnold (Germany). The frequency of this disease is from 3.3 to 8.2 cases per 100 thousand population. The pathogenesis of the disease has not been definitively established, most often considered are hereditary congenital osteoneuropathies, traumatic injuries of the sphenoid-latitudinal and sphenoid-occipital slope due to birth trauma, hydrodynamic impact of cerebrospinal fluid on the walls of the central canal of the spinal cord. But most scientists believe that the disease has a genetic basis. In the syndrome, the position of structures located in the posterior cranial fossa may change.

In Chiari malformation, the cerebellar structures are displaced, and in some cases, sections of the brainstem (medulla oblongata, pons) are below the level of intracranial pressure (ICP). As a result, they are compressed. The upper cervical segments of the spinal cord, lower cranial nerves, and cerebellar arteries are also compressed. The outflow of cerebrospinal fluid from the 4th ventricle is disrupted, which leads to a general disorder of the circulation of cerebrospinal fluid [4]. Impaired free circulation of cerebrospinal fluid leads to its accumulation and increased pressure in the brain with the development of both hypertensive cerebrospinal fluid syndrome and hydrocephalus. Hydrocephalus is a serious medical condition characterized by the accumulation of excess fluid in the cerebral ventricles. This can lead to increased intracranial pressure, which can cause damage to brain tissue and other serious complications. Timely diagnosis and treatment of hydrocephalus are extremely important to improve the prognosis and quality of life of patients. Diagnosis of pathological conditions allows us to suspect Arnold-Chiari malformation in the patient. Chiari malformation is a collection of interrelated developmental anomalies of the posterior cranial fossa that range from asymptomatic to fatal. Cranial and spinal decompression can help relieve symptoms of increased cerebrospinal fluid (CSF) pressure and correct spinal deformity [6].

There are four anatomical variants of Chiari malformation, which were identified by Chiari in 1891. Type I is characterized by an abnormal shape of the cerebellar tonsils, which are displaced below the level of the foramen magnum. Hydrocephalus is rare in this variant [7]. Typically, type I Arnold-Chiari malformation is caused by a disorder of mesoderm development, but neuroectodermal and acquired forms have also been described. This is a group of congenital diseases in which there is a downward displacement of the posterior part of the cerebellum or a combination of prolapse of the cerebellum and the lower part of the medulla oblongata through the large occipital opening (foramen magnum) into the spinal canal. Such a pathology is often accompanied by increased cerebrospinal and intracranial pressure, and therefore visual impairment. With Chiari I, hoarseness, vocal cord paresis, soft palate dysfunction, pharyngeal achalasia,

slurred speech, ataxia, nystagmus, central and obstructive sleep apnea, headache, sensorineural deafness, sinus bradycardia, hiccups, general weakness, hyperreflexia, Babinski sign, as well as sensory and motor neurological deficiency caused by syringomyelia, which often accompanies this disease.

Type II Arnold-Chiari malformation is the most common and occurs with more severe changes. There is a prolapse of the cerebellum through the foramen magnum with dislocation of the brainstem. This variant is associated with spina bifida and other developmental anomalies of the brain, spinal cord, and meninges. Hydrocephalus is present in 70% of cases and is obstructive in nature.In Type II Arnold–Chiari malformation, fundus changes, decreased visual acuity, visual field defects, diplopia, strabismus, paralysis of upward gaze, downward displacement of the eyeballs, and other ocular manifestations are most common.

Type III Arnold-Chiari malformation is rare and combines a small posterior cranial fossa with a high cervical or occipital encephalic herniation, into which the cerebellar structures are usually displaced. Also, displacement of the brainstem downward into the spinal canal is often observed. Hydrocephalus is observed in 50% of cases and is obstructive in nature due to stenosis of the cerebral aqueduct or the presence of Dandy-Walker malformation [5]. Patients with type III Arnold-Chiari malformation in most cases die in the neonatal period due to respiratory disorders. Those who survive develop severe neurological disorders – mental retardation, epilepsy, paresis and paralysis of the extremities, cranial nerve lesions, cerebellar disorders, as well as various visual acuity disorders and oculomotor disorders.

Type IV describes cerebellar hypoplasia that is not associated with other types of Arnold-Chiari malformation [8, 9]. Unfortunately, there is no specific pathognomonic criterion for confirming Arnold-Chiari malformation other than imaging. Some patients wait years for a correct diagnosis.

Purpose – to evaluate the results of a comprehensive approach to the differential diagnosis of Arnold-Chiari malformation in ophthalmology.

Materials and methods

A limited number of patients were examined; two patients (4 eyes) with Arnold-Chiari malformation using conventional methods, in particular OCT. The study was presented as a case series.

Clinical case 1

Patient Z., 6 years old (Medical card No. 595698), born at 37 weeks by cesarean section with a weight of 2550 g and a body length of 49

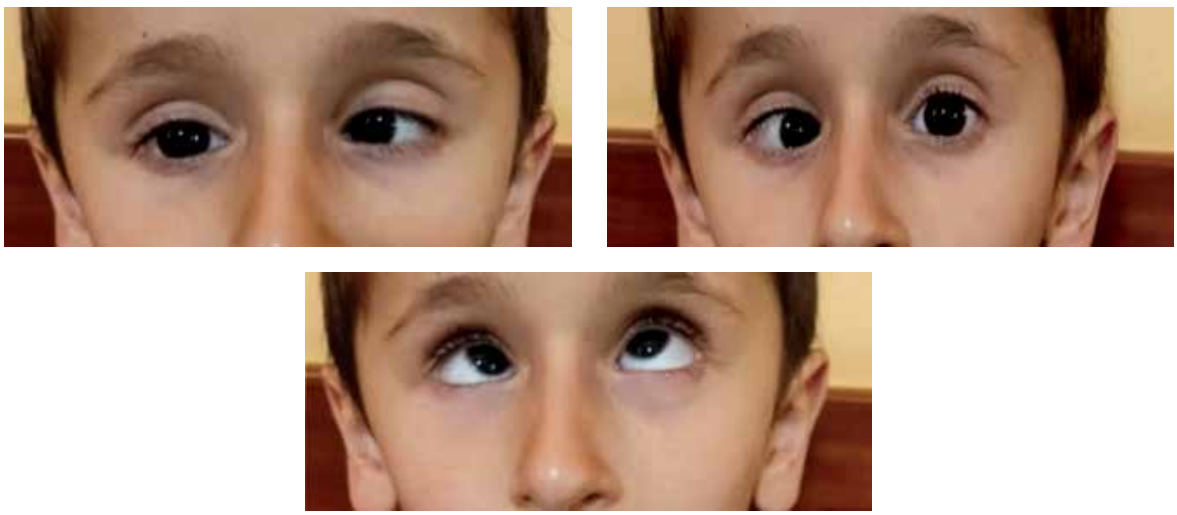

Figure 1. *Photo of eye movements of patient Z., 6 years old (ambulance card No. 595698)*

cm. Two weeks after birth, the child underwent ventricular peritoneal shunting due to Arnold-Chiari syndrome. During the examination, MRI of the brain was performed, which revealed Arnold-Chiari malformation type I. The child underwent brain bypass surgery 2 weeks after birth. In consultation with a neurosurgeon, a detailed examination revealed no motor neurological deficit caused by syringomyelia or specific symptoms of the disease (**Fig. 1**).

Both eyes – Anisometropia. Mixed astigmatism. Divergent, concomitant strabismus. Left eye – Dysbinocular amblyopia of high degree. Optic neuropathy. Visual acuity: right eye 1.0 with cor. Sph + 1.0D = 1.0, left eye 0.04 with cor. sph + 3.0 D cyl + 2.0 D ax 168° = 0.04. Deviation - 10°-15° divergent, conjunctival (Fig. 1). Color test - monocular vision. Fixation OD foveal OS paramacular.

Ultrasound biometry of the PZV: right eye - 23.15 mm; left eye - 22.25 mm. Pachymetry: right eye - 0.565 mm; left eye - 0.555 mm. Phosphene electrical sensitivity threshold (PEChf): right eye 77/45; left eye 80/45. Maculotest right eye - IV group, threshold 5.0; left eye - III group, threshold 9.0. Field of vision: right eye - narrowed peripherally by 15-200; left eye - depression in the nasal half. Ultrasound scan - the retina is attached in both eyes.

Clinical examination. Both eyes. The cornea is transparent. The anterior chamber is of medium depth, the pupil is round, 3 mm, direct and cooperative pupillary reaction without pathology. The lenses and vitreous body are transparent. The fundus of both eyes - the optic nerve discs are paler on the temporal side with clear boundaries, on the right eye there are optic nerve drusen. The arteries are narrowed, the veins are tense, but without congestive phenomena.

The operation consisted of bilateral 7 mm recession of the lateral rectus muscles for cosmetic purposes. After the operation, the deviation according to Hirschberg = 0. The operation was performed due to the fact that other methods used (prismatic lenses, pleoptic treatment) did not give the desired result. As a result of the operation, the oculomotor muscles were strengthened, the position of the eyes was aligned.

Due to the fact that, there is a potential risk of unintentional puncture of the dura mater, theoretically, if the patient has unrecognized intracranial hypertension, a decrease in cerebrospinal fluid pressure can cause dislocation of the brainstem through the foramen magnum and, as a result, lead to fatal consequences. The decision on the choice of anesthesia method was made collectively by the anesthesiologist and the neurosurgeon. Brain shunting at 2 weeks after birth in the patient in our case was justified and lifesaving. Surgery for the treatment of convergent strabismus with hyperfunction of the inferior oblique muscles can be both cosmetic purposes and increase visual acuity. Given that Arnold-Chiari anomaly is quite rare and there is not enough data in the literature on the treatment of these patients, long-term observation by an ophthalmologist and neurosurgeon is necessary. In children with Arnold-Chiari I syndrome decompressive surgery with duraplasty before puberty may prevent progression of scoliosis.

Cranial vault expansion may be recommended in pediatric Chiari malformations associated with craniosynostosis. Children with Chiari type I malformations can participate in sports due to the low risk of worsening of the condition. Early diagnosis and adequate decompressive surgery with duraplasty before puberty are crucial to mitigate the impact of this disease on the child's well-being [10 - 13].

Based on the results of the studies by Goel A., Joaquim A.F., it can be concluded that the pathogenesis of cavernous bodies of the vertebrae with or without concomitant basilar invagination and/or syringomyelia is primarily associated with atlantoaxial instability. The data obtained indicate that surgical treatment in these cases should be aimed at atlantoaxial stabilization and segmental arthrodesis. Except in cases of atlas assimilation, occipital bone involvement is not indicated and does not provide optimal stability [10, 14, 15].

Clinical case 2

Patient M. was under our supervision for 15 years. He first came to us at the age of 38, in 2009. After a viral infection, he noticed metamorphopsia, deterioration of visual acuity in both eyes. During the examination, the maximum corrected visual acuity (MCVA) of the right eye was 0.3, the left eye was 0.2. During the initial examination: the cornea is transparent, spherical. Single floating opacities in the vitreous body. Swelling and hyperemia of the optic disc, single hemorrhages in the marginal zone and around the optic disc due to venous stasis, compression of the veins and impaired blood supply in the capillaries. The disc radiates above the level of the retina into the vitreous body to 3.5 - 4.5 D. The borders and vascular funnel are not visible due to edema, which spreads to the posterior pole of the eye and the macular area, where pathological reflexes, plasmorrhagia in the form of whitish-yellow spots, foci of transudation from the area of tissue edema are observed. The retinal veins are sharply dilated, tortuous, of uneven caliber, the arteries are narrowed (**Fig. 2, a, b**). The visual field of both eyes is enlarged by 3-4 mm, with the blind spot increasing in size by 3-4 mm. The optic nerve electrical sensitivity threshold for phosphene is 120 μA in the right eye and 110 μA in the left eye. For the purpose of differential diagnosis of optic neuritis and congestive disc, the patient was prescribed an MRI of the brain. During MRI examination of the brain: the patient had an increase in the occipital foramen (80 mm fronto-occipital and 50 mm temporal). Consultation of a neurologist: Arnold-Chiari syndrome in the stage of decompensation. Treatment course: corticosteroids according to the scheme; antiviral therapy, endonasal electrophoresis

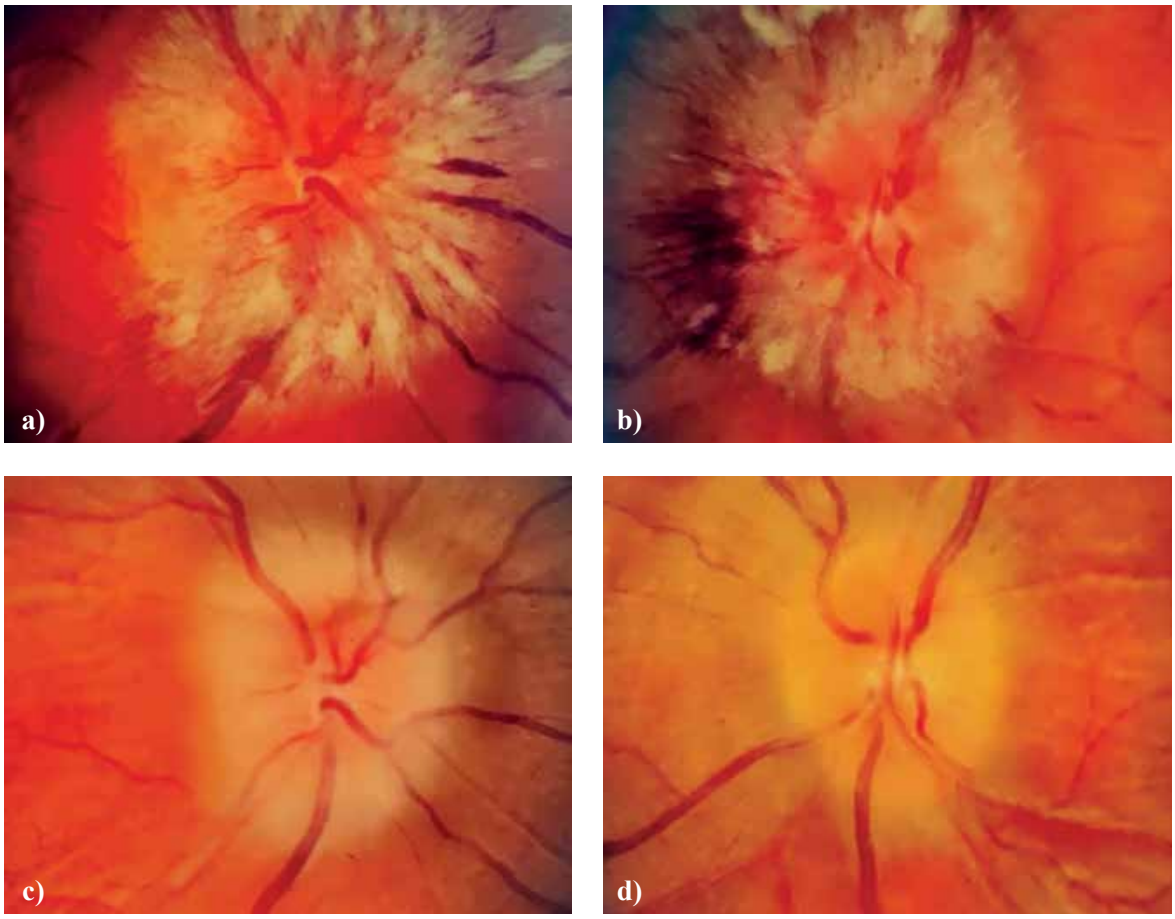

Figure 2. *Patient M., born in 1971: a) photo of the fundus of the right eye before treatment (2009); b) photo of the fundus of the left eye before treatment (2009); c) photo of the fundus of the right eye a year after treatment (2010); d) photo of the fundus of the left eye a year after treatment (2010).*

of nonsteroidal anti-inflammatory drugs in combination with corticosteroids; dehydrating, antihistamine drugs, phonophoresis of resorbable drugs (**Fig. 2**).

Against the background of treatment, after a year, indocyanine green (ICG) of the right eye was 0.6, of the left eye 0.5. The disc radiates above the level of the retina into the vitreous body to 2.0-2.5 D. panretinal photocoagulation (PRP) of both eyes: the size of the blind spot increased by 1.5-2 cm, pattern electroretinogram (PECHf) of the right eye 100 μA, left eye 96 μA. The edema near the optic nerve disc decreased. Plasmarrhagia and hemorrhages partially resolved (**Fig. 2, c, d**).

Over the past fifteen years, for the purpose of neuroprotection, the patient has received courses of laser diode stimulation of the optic nerve [16], citicoline 1000 mg per day, oral form for 1 month, meldonium 500 mg per day for 1 month.

Examination 2024: ICG of both eyes 0.4. The fundus condition is stabilized. The optic disc is pale, the borders are clear, the vessels are narrowed (**Fig. 3, a**). PECHf right eye 100 μA, left eye 96 μA. According to OCT, there is a slight thinning of the retinal ganglion cell complex (GCL + IPL) in both eyes (96 and 97 nm, respectively, in the right and left eyes) (**Fig. 3, b**). According to OCT, thinning of almost all parameters in both eyes, which concerns the DZN, however, the peripapillary nerve fiber indices retinal nerve fiber layer (RNFL) are determined within normal values (**Fig. 3, c**). Diagnosis: Partial optic nerve atrophy. Arnold-Chiari syndrome.

Hydrocephalus in Arnold-Chiari malformation is a complication caused by the

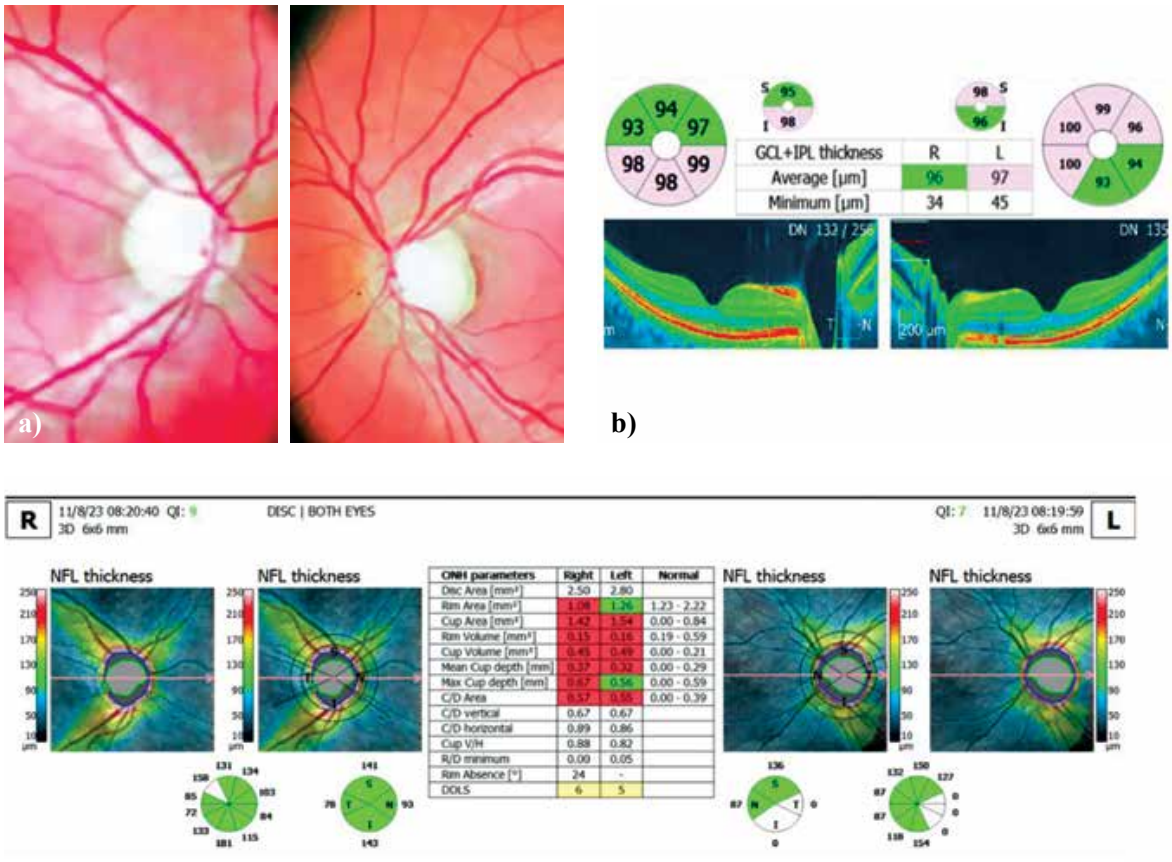

Figure 3. *Patient M., born in 1971, examination data in 2024: a) fundus photo of the right eye and left eye 15 years after the start of treatment; b) OCT of the retinal ganglion cell complex (GCL+IPL) of the right eye and left eye 15 years after the start of treatment; c) RNFL of the right eye and left eye 15 years after the start of treatment.*

displacement of parts of the cerebellum into the spinal canal, which prevents the normal outflow of cerebrospinal fluid (CSF) and leads to its accumulation in the brain. Parts of the cerebellum are displaced downward through the foramen magnum, blocking the normal movement of CSF between the cranial and spinal spaces, which can cause increased intracranial pressure.

The diagnosis of Arnold-Chiari malformation type I is based on the detection on MRI of tonsillar prolapse through the foramen magnum, reflecting the overflow of the immature posterior cranial fossa. However, tonsillar prolapse can be observed in some patients with normal posterior cranial fossa dimensions, and, conversely, even in some patients with small cranial fossa dimensions. The history and course of the disease in the patients presented allow us to assume that the trigger for the clinical manifestations of the syndrome was a viral infection. Most likely, the viral lesion was complicated by inflammation of the meninges - arachnoiditis with localization mainly in the optic-chiasma zone, which led to the development of intracranial hypertension against the background of which, due to the dislocation of brain structures, namely the cerebellum, contributed to the manifestation of Arnold-Chiari syndrome. Thus, both optic chiasm arachnoiditis and the displacement of brain structures contributed to the development of intracranial hypertension, which manifested as congestive optic nerve hypertension. Intensive steroid, anti-inflammatory, and absorbable therapy facilitated regression of the process – the development of optic chiasm arachnoiditis and a reduction in intracranial hypertension. Against the background of treatment, we observed a decrease in the phenomena of stagnation in the optic nerve disc, improvement in visual acuity. The patient's condition and timely therapy allowed preventing surgical intervention.

Conclusion

The modern diagnostic level of medicine unites specialists from various fields: ophthalmologists, radiologists, neurologists and poses new challenges for doctors in both diagnostics and treatment, which were previously unavailable. The presented series of clinical cases demonstrated possible manifestations of Arnold-Chiari syndrome including strabismus and congestion in the optic nerve, which was confirmed by magnetic resonance imaging. Only imaging can make a clinical diagnosis of Arnold-Chiari malformation. Early detection of associated ophthalmologic abnormalities helps to promptly suspect this anomaly. Comprehensive long-term treatment can stabilize the visual functions of patients with Arnold-Chiari malformation.

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REFERENCE

1. Liao, C. The Relationship Between Basilar Invagination and Chiari Malformation Type I: A Narrative Review / C.Liao, M.Visocchi, W.Zhang [et al.] // Acta. Neurochir. Suppl., – 2019. 125, – p. 111-118. https://doi.org/10.1007/978-3-319-62515-7_16
2. Brito, J.N.P.O. Basilar invagination associated with chiari malformation type I: A literature review / J.N.P.O.Brito, B.A.D.Santos, I.F.Nascimento [et al.] // Clinics (Sao Paulo), – 2019. 74, – p. 653. https://doi.org/10.6061/clinics/2019/e653
3. Klekamp, J. Incidence and Management of Basilar Invagination With Associated Chiari I Malformation: WFNS Spine Committee Recommendations / J.Klekamp, O.L.Alves, M.Zileli [et al.] // Spine (Phila Pa 1976), – 2025. Jun; 1. 50(11), – p. 786-791. https://doi.org/10.1097/BRS.0000000000005293
4. Wang, J. Acquired Chiari Malformation and Syringomyelia Secondary to Space-Occupying Lesions: A Systematic Review / J.Wang, N.M.Alotaibi, N.Samuel [et al.] // World Neurosurg., – 2017. Feb; 98, – p. 800-808. https://doi.org/10.1016/j.wneu.2016.11.080
5. Ciaramitaro, P. International Experts Jury of the Chiari Syringomyelia Consensus Conference, Milan, November 11-13, 2019. Diagnosis and treatment of Chiari malformation and syringomyelia in adults: international consensus document / P.Ciaramitaro, L.Massimi, A.Bertuccio [et al.] // Neurol. Sci., – 2022. Feb; 43(2), – p. 1327-1342. https://doi.org/10.1007/s10072-021-05347-3
6. Onafowokan, O.O. Predictors of reoperation for spinal disorders in Chiari malformation patients with prior surgical decompression / O.O.Onafowokan, A.Das, J.M.Mir [et al.] // J. Craniovertebr. Junction. Spine, – 2023. Oct-Dec; 14(4), – p. 336-340. https://doi.org/10.4103/jcvjs.jcvjs_140_23
7. Chatterjee, S. Chiari malformation and atlantoaxial instability: problems of co-existence / S.Chatterjee, P.Shivhare, S.G.Verma // Childs Nerv. Syst., – 2019. Oct; 35(10), – p. 1755-1761. https://doi.org/10.1007/s00381-019-04284-z
8. Sun, Y. Clinical analysis of atlanto-occipital decompression in the management of chiari malformation with multi-segmental syringomyelia / Y.Sun, J.Fang, S.Wang [et al.] // Front Pediatr., – 2024. Aug; 29. 12, – p. 1432706. https://doi.org/10.3389/fped.2024.1432706
9. Gepp, R. Pediatric Chiari Malformation Management: WFNS Spine Committee Recommendations / R.Gepp, J.H.Lee, J.Parthiban [et al.] // Spine (Phila Pa 1976), – 2025. Jun; 1. 50(11), – p. 208-212. https://doi.org/10.1097/BRS.0000000000005290
10. Goel, A. Is atlantoaxial instability the cause of Chiari malformation? Outcome analysis of 65 patients treated by atlantoaxial fixation // J. Neurosurg. Spine., – 2015. Feb; 22(2), – p. 116-127. https://doi.org/10.3171/2014.10.SPINE14176
11. Joaquim, A.F. Atlantoaxial instability and Chiari malformation // J. Neurosurg. Spine., – 2015. May; 22(5), – p. 55. https://doi.org/10.3171/2014.12.SPINE141194
12. Guzun, O.V. Reduction of the risk of glaucomatous optic neuropathy progression after complex neuropathy / O.V.Guzun, O.S.Zadorozhnyy, N.V.Konovalova [et al.] // The Odessa Medical Journal, – 2025. №2(193), – p. 31-37. https://doi.org/10.31288/oftalmolzh20253310
13. Visocchi, M. Indications for Surgery and Surgical Options in Chiari Malformation: WFNS Spine Committee Recommendations / M.Visocchi, F.Signorelli, Ó.L.Alves [et al.] // Spine (Phila Pa 1976), – 2025. Jun; 1. 50(11), – p. 760-766. https://doi.org/10.1097/BRS.0000000000005288
14. Chotai, S. Role of Chiari Decompression in Managing Spinal Deformity Associated with Chiari I Malformation and Syringomyelia / S.Chotai, D.J.Aum, J.M.Strahle // Neurosurg. Clin. N. Am., – 2023. Jan; 34(1), – p. 159-166. https://doi.org/10.1016/j.nec.2022.09.007
15. Klekamp, J. Chiari I malformation with and without basilar invagination: a comparative study // Neurosurg. Focus, – 2015. Apr; 38(4), – p. 12. https://doi.org/10.3171/2015.1.FOCUS14783
16. Symeou, S. Applications of Machine Learning in the Diagnosis and Prognosis of Patients with Chiari Malformation Type I: A Scoping Review / S.Symeou, M.Lampros, P.Zagorianakou [et al.] // Children (Basel), – 2025. Feb; 18. 12(2), – p. 244. https://doi.org/10.3390/children12020244