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MODERN CONCEPTS OF ARNOLD-CHIARI MALFORMATION

professor Shodiyev Amirqul Shodiyevich

Samarkand State Medical University, Neurosurgery Department

The review is devoted to the problem of Chiari malformation. The issues of etiology, pathogenesis and epidemiology are considered. Particular attention is paid to modern methods of instrumental examination of the disease, their comparison with each other with a description of the advantages and disadvantages.

Key words: Chiari malformation, magnetic resonance imaging, craniovertebral region, ectopia cerebellum.

Chiari malformation (CMA) is one of the many congenital pathologies (developmental defects) of the craniovertebral region, caused by asynchronous growth of the bone and brain structures of the posterior cranial fossa, manifested by the descent of the caudal parts of the cerebellum and medulla oblongata through the foramen magnum.

The first to describe this pathology was T. C. Liam in 1883, noticing the elongation of the brainstem and the descent of the cerebellar tonsils into the spinal canal in 9 deceased infants [1, 2]. In 1891, Jaans-younchiai described a congenital anomaly consisting of a hernia-like protrusion of the cerebellar tonsils below the level of the foramen magnum [3, 4].

Neurological diagnostics of MC is associated with certain difficulties, since not all cases of cerebellar tonsil descent below the foramen magnum are accompanied by clinical manifestations. On the other hand, the clinical signs of MC themselves are polymorphic [5, 6, 7].

The prevalence of MC ranges from 3.3 to 8.2 per 100,000 population. The ratio of men and women, according to a number of authors, is approximately the same, however, women suffer from this pathology somewhat more often.

[8, 9, 10]. Since the active use of neuroimaging methods (MRI and MRA), the number of registered patients diagnosed with MC has increased more than twofold [11]. The incidence of the disease among examined patients with neurological symptoms, according to some authors, was 140%. A study conducted by T.A. Akhadov using MRI made it possible to identify MC in 26% of patients among 2286 patients with suspected various pathologies of the cervical spine.

spinal cord diseases and craniospinal tumors [12, 13].

Many open questions remain regarding the pathogenesis of bone anomalies of the vault and base of the skull, malformations of the central nervous system (CNS), pathology of the cerebrospinal fluid spaces of the brain and spinal cord (syringomyelia, hydrocephalus) associated with CM. At present, the structural features of the Willis circle, cerebral hemodynamics, and functional disorders of the structures of the brainstem and spinal cord in patients with CM have not yet been sufficiently studied. Also, despite the described familial cases of CM, genetic disorders that contribute to the development of this pathology have not yet been identified, which does not allow us to determine the associated genetic risk [13, 14].

The main method of treating MC is surgical, aimed at equalizing the hydrodynamic pressure of the cerebrospinal fluid at the level of the craniospinal junction, creating a large occipital cistern and eliminating compression of the brainstem [15, 16].

To date, no sufficiently reliable and universal method for diagnosing MC has been developed that would allow for the selection of patients at the early stages of the disease for their referral for a more in-depth and specialized examination. In this regard, further comprehensive study of MC seems relevant, which will allow for a deeper understanding of the mechanisms of its development and the development of clearer tactics for the management and treatment of patients depending on the varying degrees of severity of the pathology, the presence of concomitant anomalies and clinical manifestations of the disease.

Etiology and pathogenesis

Despite the widespread use of modern diagnostic methods for MC, its etiology and pathogenesis still remain insufficiently studied. There are several well-founded theories of the origin of MC. Most authors believe that the main role in the development of the anomaly is played by pathology of intrauterine development of bone and soft tissue structures of the posterior cranial fossa. Thus, numerous studies have established the important role of heredity in the determination of MC, there is information on the accumulation of familial cases of MC, including in monozygotic twins [17, 18]. When analyzing them, the most likely are autosomal dominant or autosomal recessive types of inheritance. In addition, there is a hypothesis about the genetic nature of the disease, possibly associated with the expression of Pax-1 and Pax-2 genes, which determine the development of axial formations [19, 20].

However, there are publications that prove the role of birth trauma in the development of CM. As a result of morphometric studies of bone and neural structures of the posterior cranial fossa in patients with CM, disproportions were noted between the length of the Blumenbach's clivus and the brainstem, which are supposedly caused by birth trauma [21,

22]. In trauma, damage to the sphenoid-ethmoid and sphenoid-occipital synchodrosis initially occurs. Bone fragments cannot grow together due to their constant mobility caused by brain pulsation. Under such unfavorable healing conditions, calcification and formation of bone structures occur slowly and incorrectly, which leads to shortening and deformation of the clivus and, as a consequence, disrupts the formation of the posterior cranial fossa in the postnatal period [23, 24].

The described pathophysiological mechanisms can lead to a disproportion between the volume of neural formations and the capacity of the posterior cranial fossa, causing morphological and clinical manifestations of MC [25]. One of the most severe clinical forms is a combination of MC and syringomyelia, which is observed in 48-76% of MC cases [26, 27]. In most cases, the cavities in the spinal cord are hydromyelia, that is, an expansion of the central spinal canal under the influence of increased cerebrospinal fluid pressure. Complications of MC also include hydrocephalus, which occurs

resulting from long-term increasing disturbances in the dynamics of the cerebrospinal fluid. Depending on the cause of disturbances in the circulation of cerebrospinal fluid in MC, their classification has been proposed, with the allocation of an isolated form of MC, malformations in combination with syringomyelia, malformations with hydrocephalus, and malformations with a pathological volume of cerebrospinal fluid in the posterior cranial fossa [28, 29].

The mechanisms of occurrence and progression of syringomyelia accompanying CM are controversial. Several hypotheses have been proposed to explain the pathophysiology of syringomyelia in patients with CM. Thus, the accumulation of structures in the foramen magnum may act as an external "plug" compressing the lower parts of the brainstem and upper cervical spinal cord, which may interfere with the transmission of water pulse waves of the cerebrospinal fluid (hydrodynamic hypothesis of V. Gardner) [30]. According to other authors, the occurrence of syringomyelia in combination with CM is associated with stretching of the spinal cord and low location of its conus in the "rigid terminal thread" syndrome, leading to secondary damage to the brain structures of the craniovertebral region [31].

The use of magnetic resonance imaging allows us to accurately determine the degree of cerebellar displacement, determine the form of malformation and the degree of disease progression. In this regard, a number of new classifications of MC have emerged, based on the neurovisual picture. The most common is the classification proposed by DoynDetal. [32, 33]:

• I - descent of the cerebellar tonsils below the level of the foramen magnum;

• II - descent of the cerebellar tonsils to level C2 with the location of the pons and medulla oblongata below the Twining line (between the tubercle of the sella turcica and the internal occipital protuberance);

• III - prolapse of the cerebellar tonsils in combination with hypertensive-hydrocephalic syndrome;

• IV - hypoplasia of the cerebellum and descent of the medulla oblongata.

It has been established that MC types II-IV occur predominantly in childhood and are combined with a wide range of neurological pathologies. Recently, a zero type of MC (type 0) has also been identified, which has some clinical symptoms of type I (frequent headaches and other neurological

manifestations), but the ectopia of the cerebellar tonsils in such patients does not reach 5 mm, which does not allow us to classify such cases as classical types of the disease [34, 35]. Clinical and neurological manifestations

The clinical manifestations of Chiari malformation are varied and, as a rule, are represented by various combinations of symptoms of hypertensive-hydrocephalic,

cerebellar, spinal, bulbar and syringomyelic syndromes. Clinical signs appear gradually, increase, and may differ in individual patients [36, 37,]. Often the first symptoms appear in childhood or adolescence, less often the manifestation of the disease is observed in adulthood. As a rule, patients with craniovertebral anomalies, including Chiari malformation, have a dysraphic status: short neck, cervical ribs, low hairline in the occipital region, asymmetry of the face and skull, gothic palate, adhesion of the earlobes, kyphoscoliosis of the spine, costal hump, uneven position of the scapulae, funnel chest, etc. [38, 39].

A common predisposing factor for the development of neurological symptoms in patients with MC is minor trauma, less commonly infection [40]. Cases of neurological disorders have also been described after lumbar punctures, lumboperitoneal shunting, or prolonged stay in a flexed neck position in patients with MC [41]. In most cases, neurological symptoms in patients with MC are progressive [42,], especially with constant physical stress [30].

According to a number of researchers, up to 50% of patients with CM have pain in the neck, and 25%-80% suffer from suboccipital-occipital headaches due to increased intracranial pressure [43]. Such a variety of neurological symptoms is pathogenetically due to the fact that with this pathology, the cerebellar tonsils and the lower parts of the medulla oblongata descend downwards, which leads to compression of the caudal parts of the brainstem, cerebellum, upper cervical segments of the spinal cord, as well as to compression and bending of the lower cranial nerves. Increased suboccipital pain when

coughing or physical exertion in patients with CM is explained by a further increase in the herniated protrusion of the cerebellar tonsils [44, 45]. In addition, at the level of

the foramen magnum, there is compression of the vertebral and posterior inferior cerebellar arteries with the development of ischemic disorders in the vertebral-basilar basin. In addition, cerebrospinal fluid-dynamic disorders occur with the formation of hypertensive-hydrocephalic syndrome and syringomyelia [46, 47]. Also, in the pathogenesis of neurological symptoms in CM, a major role is played by concomitant anomalies of the craniovertebral region, which cause additional trauma to the descended tonsils of the cerebellum and medulla oblongata [48].

Other common neurological manifestations of CM include vestibular-cerebellar disorders [49], caused by impaired blood supply due to compression of the vertebral artery or posterior inferior cerebellar artery at the level of the foramen magnum [50]. Vestibular dysfunction in patients with CM is characterized by short-term attacks of non-systemic dizziness and increased sensitivity to vestibular loads. Vestibulovegetative paroxysms have been described, which are accompanied by nausea and vomiting, general weakness and disorientation, as well as respiratory failure. During examination of patients with CM, various types of nystagmus (horizontal, rotatory, vertical "beating down and up") are often detected, which, according to some data, occur in almost half of patients with CM [51]. The most characteristic disorder is nystagmus, "beating down", which occurs as a result of imbalance in the central vestibulo-ocular pathways [52]. A number of authors consider this type of central vestibular nystagmus to be a diagnostic sign of damage to the craniovertebral region.

[53]. Gait disorders are often accompanied by subjective sensations in the form of staggering. At the same time, it was noted that staggering when walking was noticed by strangers, and the patient himself did not feel such a deviation. Gait disorders were observed in 21.5% of patients with CM. In some patients, cerebellar disorders are manifested only in special tests [54]. The most indicative was the detection of Babinski asynergy in the horizontal and vertical positions of the large. A number of studies have established that a deficit in coordination tests was observed in all groups of patients with ectopia of the tonsils [55]. In general, despite the presence of cerebellar pathology in any

degrees of MC, there is a tendency for it to be most pronounced in patients with cerebellar ectopia of more than 10 mm, with a predominance of symptoms of dynamic ataxia.

In addition, patients with MC have motor disorders characterized by gait disturbance of isolated spastic or mixed spastic-ataxic type. However, pyramidal insufficiency is detected more often [56, 57]. It has been shown that progressive paresis of the lower extremities can both prevail over upper paraparesis and occur less frequently [58]. The paresis of the

upper extremities that occurs is of spastic and spastic-atrophic nature. The prevalence of proximal paresis over distal paresis is also noted [59].

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