

Perspectives on the Chiari Malformation's Hydrodynamics

Abstract

The clinical course of Chiari 1 malformation, which is a radiologic finding of caudal cerebellar tonsillar displacement, can range from benign to complications involving potentially fatal hydrocephalus. The clinical realities and dilemmas these patients present necessitate a coherent approach to this entity, despite the fact that the pathophysiologic processes underlying this variation in outcome remain a subject of scientific debate. We hope to shed light on the various processes that lead to hydrocephalus in patients with Chiari 1 malformations in this review. Understanding the etiology of such hydrocephalus is essential for the treatment of Chiari 1 malformations with associated hydrocephalus. Hydrocephalus can occur simultaneously, as a result, or as a cause of the Chiari 1 malformation.

Keywords: Chiari • Chiari decompression • Chiari malformation • Foramen magnum • Hindbrain • Hydrocephalus • Posterior fossa

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Introduction

From a strictly morphological perspective, the radiological definition of Chiari Malformations (CMI) is used. They comprise of ptosis of the cerebellar tonsils through the foramen magnum of more prominent than 3 or 5 mm. The MacRae line defines the foramen magnum. Although not all patients exhibit symptoms, the radiological incidence of CMI is 0.77 percent of the global population. The clinical incidence was determined to be approximately 3.08:100,000. There is more than one clinical presentation, primarily consisting of headaches, as well as visual (objective or subjective) and otoneurological symptoms, symptoms of the brainstem or cranial nerve, and medullary symptoms. The majority of clinical symptoms are directly related to ptosis of the tonsils or the complications that result from it, like syringomyelia. Because symptoms are frequently vague, it can be challenging to determine whether surgery is necessary. Idiopathic intracranial hypertension, for instance, is one of the differential diagnoses that can radiologically mimic a CMI. In fact, we observed ptosis of the cerebellar tonsils in 20.9% of patients with nonspecific headaches who had idiopathic intracranial hypertension [1].

Discussion

Hydrodynamics and hemodynamics prior to surgery

By definition, CMI reduce or block the local flow of CSF by reducing the surface of subarachnoid spaces at the cranio cervical junction. The flush of CSF toward the spinal subarachnoid spaces during systole plays a crucial role, as can be seen from our current understanding of the physiology of intracranial flow. Indeed, each cardiac systole is accompanied by an increase in intracranial vascular volume. A “perfect fluid” is not blood; the vascular tree has a high resistance to flow and a viscosity that is approximately three times higher than that of water. During the cardiac cycle, this causes an increase in the SV_{vasc} (stroke volume of arterio venous in the cranium) blood volume [2].

The cranial box, on the other hand, is rigid and inflexible. The “mobile compliance” of the CSF flush from the intracranial spaces to the compliant spinal subarachnoid spaces is caused by this increase in vascular volume. However, it has been demonstrated that this is not entirely accurate throughout the cardiac cycle, despite the fact that the Monro Kellie doctrine asserts that

intracranial volume remains constant. The stroke volume of CSF in the spinal canal is smaller than the stroke volume of arteriovenous blood, and the flow of CSF throughout the cardiac cycle does not completely balance the flow of arteriovenous blood. Consequently, ICP fluctuates throughout the cardiac cycle and exhibits P1, P2, and P3 amplitude peaks [3].

Interestingly, we did not observe a decrease in CSF dynamics measured at the upper cervical level in our group when compared to a control group from the literature. This is explained by the fact that abnormal additional pulsatility of the medulla oblongata or cerebellar tonsils through the foramen magnum is also the source of pulsatility at the cervical level. This is in addition to the restricted CSF flow at the level of the foramen magnum. The pulsatility amplitude of CSF measured at the cervical level was mirrored by the synchronized movement of these parenchymal structures. ICP regulation is mediated by the “mobile compliance” necessary for the homeostasis of vascular intracranial flow through the medulla oblongata and cerebellar tonsils. Mechanical stress could result from these abnormal, non-physiological movements of parenchymal tissues, which could manifest as symptoms.

Surgery, changes in Hydrodynamic and Hydrodynamic Parameters

The ventricular CSF flow measured in the aqueduct was normal and unaffected by CMI, as demonstrated by the comparison of the Craniospinal Hemohydrodynamics of CMI before and after the procedure. Hydrocephalus, which is uncommon in CMI, was not seen in any patients.

Using the same technique as the CSF, the semi-automatic segmentation of the bulbar medullary junction and the amygdala that was used to analyze the stroke volume of the tonsils made it possible to quantify the tonsil's pulsatility over the course of a single cardiac cycle. The number of tonsils displaced during a single cardiac cycle is known as the stroke volume of the tonsils or pulsatility of the tonsils. During the systolic phase, this displacement is in the craniocaudal direction, while during the diastolic phase, it is in the caudocranial direction. Low CSF oscillations at the cranio-cervical junction and tonsil pulsatility, which decreased following surgery in tandem with an increase in CSF volume pulsatility, are demonstrated here [4, 5].

CSF velocities at the cranio-cervical junction have been found to decrease after surgery and to be higher in Chiari populations than in a control group. This is easily explained by the fact that the velocities and resistance to flow increase as CSF spaces decrease, resulting in a decrease in volume displacement for the same pressure. We believe that velocity alone is not the most appropriate parameter for assessing the impact of CMI. It depends on the segmented region and the foramen magnum's anatomy, which may contain multiple compartments. Since the intracranial blood volume expansion varies from person to person, we prefer to take into account the total CSF volume displaced through the foramen magnum in the spinal canal [6].

Additionally, it has been reported that duraplasty permits a greater reduction in the tonsil's pulsatility. Nevertheless, the time between the control MRI and this result may have an impact. In fact, at three, six, and twelve months, two of our patients underwent additional control MRI immediately following surgery. One patient underwent surgery with only bone decompression, while the other received a duraplasty in addition. The intradural patient experienced an immediate hydrodynamic effect from the surgery. The CSF dynamics of the patient who received only bone decompression treatment improved gradually. There was a rapid improvement in symptoms in both instances. As a result, the large cistern appears to gradually expand after extradural surgery, making it possible to achieve the same outcomes as intradural surgery over a longer period of time [7, 8].

Conclusion

In clinical practice, phase-contrast MRI is simple to use, but complete interpretation necessitates quantitative post processing. Tonsil pulsatility was accompanied by an exclusive change in CSF dynamics at the foramen magnum plane that we observed in CMI. We noticed a decrease in the sufficiency of jugular blood surge and change of the cerebral venous seepage pathway. We also noticed that surgery had a quantitative effect on both the hydrodynamics and hemodynamics. In addition to morphological imaging, phase-contrast MRI could be a useful tool for postoperative evaluation and follow-up. By taking into account information about the dynamics of neurofluids in addition to morphology, these findings could be used to reevaluate the classification of CMI [9, 10].

Acknowledgement

None

Conflict of Interest

None

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