



Case Report

Cavum veli interpositi: Why this anatomical variant exists?

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ABSTRACT

The cavum veli interpositi (CVI) represents a cerebrospinal fluid (CSF) filled space formed by the corpus callosum and fornix above, the roof of the third ventricle and thalamus below and the crus of the fornix on each side laterally. It is frequent in infants but infrequent in children above 2 years of age and adults. We describe the case of a 20-year-old woman, which revealed a helmet-shaped CSF space at the anatomical location of the CVI on brain MRI performed because of recurrent headaches. We present this case in order to review the literature and to discuss the possible cause of the existence of such an anatomical variation. © IJAV. 2010; 3: 2–5.

Key words [cavum veli interpositi] [magnetic resonance imaging] [venous pressure]

Introduction

The cavum veli interpositi (CVI) is a space formed by the fold of pia mater (tela choroidea) during the interhemispheric cleavage in relation to the development of the corpus callosum, which occupies the transverse cerebral fissure (choroidal fissure). It represents a potential cisternal space containing cerebrospinal fluid (CSF) between the internal cerebral veins and posterior medial choroidal artery.

CVI is triangular in shape with a wide base dorsally. Its boundaries are the hippocampal commissure and corpus callosum superiorly, the tela choroidea inferiorly and the crus of the fornix on each side laterally. It may extend anteriorly in the roof of the third ventricle as far as the interventricular foramina, which opens caudally into the cisterna venae magnae Galeni [1].

Embryologically, CVI is an anatomic variation originating from the roof plate of the diencephalon by a process of the pia mater, which protrudes into the primitive neural tube during the third fetal month [2]. It is a part of the normal developing structures of the cerebral midline, which decreases in size after full-term birth [3]. It may appear as a cyst in the pineal region on neonatal ultrasonograms, is frequent in infants but it is much less frequent in children above 2 years of age and adults [4].

We present this case intending to associate the existence of this structure with venous pressure equilibrium in brain, while reviewing thoroughly the literature.

Case Report

A 20-year-old woman underwent a brain MRI examination in our outpatient department because of recurrent headaches. Neurological examination was free of any pathologic findings.

The brain MRI examination, which included T1-, T2-weighted and flair sequences before contrast agent administration as well as T1-weighted post-contrast sequences in all 3 planes (sagittal, axial, coronal) revealed a helmet-shaped CSF containing space at the anatomical location of the CVI. The boundaries of this space were the hippocampal commissure and corpus callosum superiorly, the tela choroidea and thalamus inferiorly, the foramen of Monro anteriorly, the pineal region posteriorly and the crus of the fornix bilaterally. The internal cerebral veins were visible at the inferolateral borders of the cistern (Figures 1,2,3,4).

Discussion

A variety of CSF-containing midline cysts of the anterior brain exist and may be found, from anterior to posterior, in four variants as absence septum pelucidum (ASP), cavum septi pellucidi (CSP), cavum vergae (CV) and cavum veli interpositi (CVI). They are all cystic anomalies of septum pellucidum, which were first described in the 1600's and are noted in the 110 mm crown-rump human embryo [1]. The precise genetics of these intracranial cysts are unknown in humans. However, in another study it had been shown that corpus callosum dysmorphology is the



Figure 1. Sagittal T1-weighted MRI image showing a low CSF signal-intensity anatomical structure occupying the space between the splenium of the corpus callosum and massa intermedia. (*Arrow: CVI*)

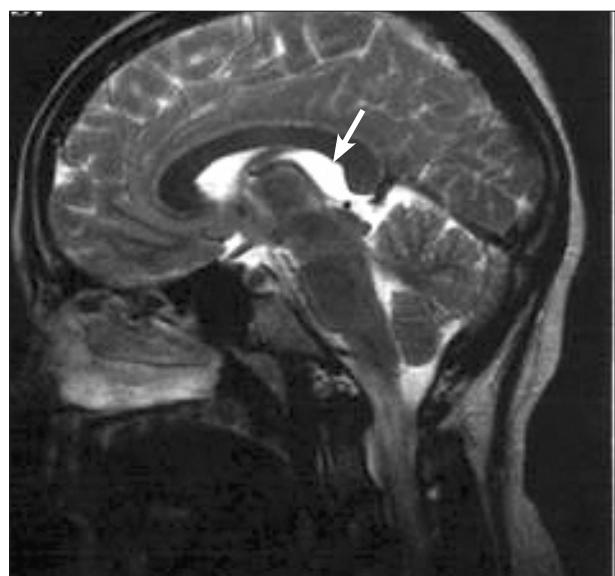


Figure 2. Sagittal T2-weighted image showing the CVI. (*Arrow: CVI*)

result of more than one major genetic locus in inbred mouse strains [5].

These cysts occur during the developmental process of the brain in the embryonic life and are actually subarachnoid spaces, which regress between the seventh month of intrauterine life and the second year of postnatal life. They are normally present during fetal life and in certain proportion of the adult population [6], thus they are called “persistent primitive structures” [7].

As these three cavities in the cerebrum are not lined by ependyma or choroid plexus cells, they do not produce

cerebrospinal fluid and therefore they are not considered part of the ventricular system [1]. They are in direct contact with the subarachnoid space but not with the ventricular system [2,8].

Most of these fluid collections are benign and disappear with time, usually soon after birth, with no consequence; however, some patients may have associations or potentially become symptomatic mostly due to the mass effect of these cavities [9,10].

A persistent septal cyst enclosed by the leaves of the septum pellucidum is called cavum septi pellucidi (CSP) and is located between the frontal horns of the lateral ventricles. The extension of a CSP posteriorly, beyond the columns of the fornix and foramina of Monro has been called a cavum vergae (CV). CV always coexists with CSP.

Cisterna interventricularis, ventriculi tertii, transverse fissure and sub-trigonal fissure are also terms used instead of cavum velum interpositum [1], which was first described in the 1930's by Kruse and Schaetz. Cavum veli interpositi, although a normal structure in the fetus, usually regresses over time and is not a common finding in adults. Persistence in 1-10 years old children is approximately 30% [1].

There have been a few studies concerning the incidence of CVI in the general adult population. This incidence is variable and depends on the population studied.

Cheng et al. in an ultrasonographic study of preterm infants found that the incidence of CVI was 21% [2].

Picard et al. by studying pneumoencephalographic examinations of infants with (n=53) and without (n=105) CVI, stated that the incidence of CVI decreased with age and had no gender predilection. CVI was

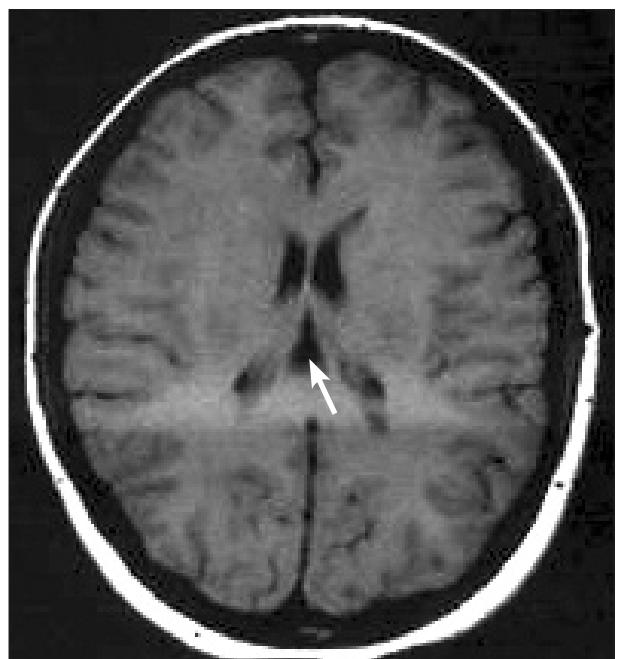


Figure 3. Axial T1-weighted image showing the cyst-like space as a triangular structure between the columns of the fornix. (*Arrow: CVI*)

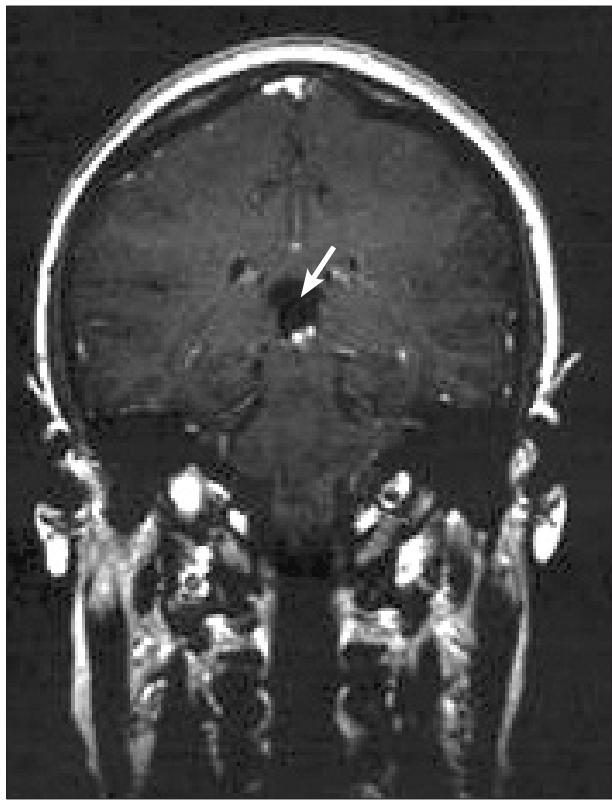


Figure 4. Coronal T1-weighted image following intravenous contrast material injection. The CVI is rectangular in shape in this plane and the internal cerebral veins can be identified at its inferior border. (Arrow: CVI)

detected in the 34% of the infants who underwent pneumoencephalography [3].

In a CT study of 442 adults over the age of 18, which were receiving medical treatment in a neurosurgery department without clinical findings of midline cystic abnormalities, it was found that CVI had the incidence by 7.24% [11].

In an MRI study of 505 non-psychotic patients at the age of 2 months to 79 years with various prediagnoses, an incidence of CVI of 5.54%, was determined [12]. There was no statistically significant difference in the frequency of CVI between the genders and between age groups. It was shown that CVI is twice as frequent as the cavum vergae. CVI may be confused with CV. However, a CV always coexists with a CSP and the location of the internal cerebral veins, as seen on MRI, can help differentiating them. CV lies above these veins, whereas CVI encloses them.

It is also important to differentiate CVI from a pathologic pineal cyst or an arachnoid cyst of the quadrigeminal cistern. Again, the location of the internal cerebral veins may provide useful information regarding the origin of the cyst, since the pineal gland lies inferiorly to the internal cerebral veins, whereas CVI encloses the veins at its lateral and inferior borders [2].

Few associations have been reported with the presence of this cavity. The clinical significance of CVI is unclear.

Interestingly, in another study [6] it was reported of monozygotic twins, the one with cavum velum interpositum and psychosis and the other without such anomaly. For the authors that finding may indicate a dysgenic process in early brain development and thus, play a role in the etiology of psychosis. In their opinion a large CVI may have clinical significance. According to this study one possibility is that individuals with developmental anomalies are more prone to psychosis if they also carry additional genetic risk factors.

By studying sonographies of two fetuses with CVI and postnatal MRIs of them, there were found no abnormalities regarding the growth and development [13]. It was concluded that further evaluation is needed to find whether there is any association between the presence of CVI and neuropsychiatric disorders.

A syndrome has been proposed which is characterized by a progressive increase in head size, moderate ventricular dilation without intracranial hypertension and CVI [14]. Children with dilated CVI may become symptomatic and present with a large head, hydrocephalus, mental retardation and seizures [14,15].

Endoscopic ventricular fenestration is the treatment of choice for these large CSF cysts when they become symptomatic [2,16], because it provides communication between the cyst and the ventricular system and prevents from ventriculoperitoneal shunting of the cyst [16].

In a study of identical twin brothers with midline intracranial cysts, mild ventricular enlargement and macrocephaly; it was shown that the cysts were obliterated in serial imaging and the ventricles decreased in size, followed by the fall of each child's head circumference onto normal [17]. It was suggested that some infants with midline intracranial cysts and macrocephaly might represent transient episodes of raised intracranial pressure from mass effect that if merely observed, will dissipate with the collapse of the midline cavity. Shunting or fenestration of midline cysts in infants with macrocephaly and mildly dilated ventricles should be carefully decided. Such a phenomenon does not occur normally when CVI exists, since CVI communicates with adjacent cisterns and typically does not cause mass effect or hydrocephalus.

In the literature it had been described a transformation of the cisterna CVI into a cyst [14]. The intermittency of symptoms was explained by a ball-valve type of connection between the cisterna CVI and the cisterna venae magnae Galeni.

Other persistent midline cavities such as cavum septi pellucidi and cavum vergae were not recognized in our patient. CVI was probably an incidental finding, which encountered because of the tendency to perform brain MRIs in patients with headache, and did not cause symptoms.

In all studies concerning these anatomical variants (CSP, CV, CVI) it can be observed that for their differentiation the localization of internal cerebral veins is of great importance. In other words that all this CSF containing spaces develop around internal cerebral veins. Sagittal

sinus, sigmoid sinus and internal cerebral veins represent the most common venous structures, which can be found in every individual. The very important clinical significance of internal cerebral veins patency is shown by the fact that in their thrombosis severe thalamic edema follows that in turn can lead to death [18].

The consistence of appearance of internal cerebral veins and the knowledge of the relationship between venous pressure and CSF pressure [19] lead us to the hypothesis that all these CSF spaces, if they exist, regulate in a certain functional way the CSF and venous pressure equilibrium of the brain.

Ventricle size depends upon brain turgor and lateral ventricle pressure. If impaired venous drainage occurs rapidly, and the lateral ventricles can empty, the ventricles will be small. In cases of primary cerebral infections

the brain becomes edematous and the ventricles tend to empty as CSF passes into the spine. The patients' condition will become critical when the intracerebral subarachnoid spaces are obliterated [20].

A possible mechanism in our case may be the increased flow of CSF in these supplementary spaces (CSP, CV, CVI) from intracerebral subarachnoid spaces and cisterns, in cases of high intraventricular CSF pressure that could result in a decrease in the pressure of cortical veins. In the MRI images of our patient we also observed that the subarachnoid spaces of the whole brain were not dilated and were occupied by brain parenchyma. May be these CSF cavities are observed in the absence of CSF compensation via dilated subarachnoid spaces. Further clinical studies need to be done in order to support this hypothesis.

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