Chapter

DEVELOPMENT OF SYMPTOMS IN ARACHNOID CYSTS

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ABSTRACT

Arachnoid cysts are benign developmental anomalies of the central nervous system and arise in virtually all locations where arachnoid membrane exists. It is reported that they account for approximately 1% of all intracranial lesions and are caused by the splitting of the arachnoid membrane. Many authors have indicated that arachnoid cysts typically remain constant in size or grow very slowly. Therefore, most opportunities to detect arachnoid cysts are incidental. However, recent advent of more advanced imaging techniques has increased the frequencies to find them. The enlargement of arachnoid cysts are reported to occur most frequently in patients under 5 years of age, and the symptoms and signs derived from arachnoid cysts include elevated intracranial hypertension and focal signs due to the effects of compression to the surrounding cerebral parenchyma. Even in cases in which neurological symptoms are present, it is often difficult to properly correlate nonspecific signs and symptoms with the findings of arachnoid cysts. This is especially true in the cases common symptoms, such as headache, that arise frequently in the general population. In most of the cases, arachnoid cysts usually bring with a thin and bulging inner table of the skull. These bone deformities suggest a long process accompanying a gradual increase in intracystic pressure that probably begins in early infancy.

Three mechanisms of the cyst expansion in arachnoid cysts have been described by many authors as follows: First, osmotic gradient between the cyst and the surrounding subarachnoid space induced fluid influx into the cyst cavity; second, fluid is secreted from ependymal cells on the cyst wall; third, a one-way ball-valve mechanism develops on the cyst wall. Almost symptomatic arachnoid cysts belong in children, the progressive growth of the skull and plasticity of the brain in children can buffer the mass effect

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caused by the cysts. Therefore, the arachnoid cysts in children and adolescence are usually asymptomatic or minimally symptomatic. The patient's symptoms develop once the breakdown of compensatory mechanism in the brain surrounding the arachnoid cyst happens. Even in elderly patients with or without cyst expansion, the breakdown of compensatory mechanisms can lead to relatively rapid progression of symptoms. However, the factors that induce the breakdown of compensatory capacity remain unknown.

In addition to those mechanisms, the symptomatic arachnoid cyst infrequently associates with intracranial hematoma, such as chronic subdural hematoma, acute subdural hematoma, acute epidural hematoma, and intra-cyst wall hematoma. Especially, it is well known that arachnoid cysts located in the middle cranial fossa can present with chronic subdural hematoma. The hematomas are frequently induced by minor trauma.

The symptoms in arachnoid cysts are presented according to the various locations arachnoid cysts arise, and the developmental mechanisms are also discussed with the aspects of accompanying hydrocephalus or intracranial hematomas, and patients' ages.

Keywords: arachnoid cyst, development, congenital, symptom

LIST OF ABBREVIATIONS

CPA	cerebellopontine angle
CSDH	chronic subdural hematoma
CSF	cerebrospinal fluid
EEG	electroencephalogram
FIESTA	fast imaging employing steady-state acquisition
MRI	magnetic resonance imaging
WI	weighted image.

INTRODUCTION

Arachnoid cysts are well known as common anomalies of the central nervous system. They are mainly benign congenital origins, and the cysts consist of the wall lined by arachnoid cells and intra-arachnoid collections of cerebrospinal fluid (CSF) within the cyst formed by a splitting or duplication of the arachnoid membrane during the complex folding of the neural tube and formation of the subarachnoid cistern [1-6]. On the other hand, some arachnoid cysts have been documented as being acquired resulting from inflammation, hemorrhage, tumors, trauma, connective tissue disorders, or even from iatrogenic causes [7, 8]. Although, in most cases, arachnoid cysts are clinically asymptomatic and remain static in size, these cysts may cause various neurological findings depending on their locations, mainly because cysts occasionally and slowly expand, and displace the adjacent structures [2, 3, 9]. Arachnoid cysts can occur relatively rare in approximately 1% of all intracranial space-occupying lesions [10-12], and recent reports about magnetic resonance imaging (MRI)-screening examinations found intracranial arachnoid cysts in 1.1-1.7% of healthy and asymptomatic volunteers [13, 14]. They can exist all along the craniospinal axes, but a large majority of cases are located intracranially [11, 15]. Most commonly, arachnoid cysts are located in or adjacent to the Sylvian fissure and in the cerebellopontine angle (CPA) [16].

Regardless of the benign histological nature of arachnoid cysts, the associated expansive effects may occasionally cause the compression of adjacent neural tissue and obstruction of CSF flow, ultimately resulting in a variety of symptoms such as developmental delay, seizures, macrocephaly, insufficiencies of motor and sensory functions, and headaches [17, 18]. Recent advances in neuroimaging techniques and its widespread availability have achieved an increased detection of arachnoid cyst in a neuroradiological screening of outpatient clinic, with the diagnoses made more frequently at earlier stages. Therefore, many authors have been reported that the natural history of arachnoid cysts turns to be quite diverse and unpredictable in each individual [19, 20]. As described above, the arachnoid cysts can occasionally increase in size and intracystic pressure, resulting in producing of symptoms due to mass effect or obstruction. However, the detail mechanism of enlargement of the cysts remains controversial.

SYMPTOMS AND THEIR DEVELOPING MECHANISMS IN EACH LOCATION OF ARACHNOID CYST

Middle Cranial Fossa Arachnoid Cyst

Arachnoid cysts can occur all along the craniospinal axes, but a large majority of cases are located intracranially [16]. Intracranial arachnoid cysts demonstrate deviated preponderance of location for the middle cranial fossa. This finding from literature suggests a possible genetic component in the development of some arachnoid cysts as well as sidedness and sex distribution [21, 22]. It is reported that those cysts located in the middle cranial fossa account for 34% to 50% of all arachnoid cysts [16, 23, 24].

Initially, arachnoid cysts in the middle cranial fossa were believed to arise secondary to primary temporal lobe hypogenesis [25]. Patients with those cysts may present various signs or symptoms, including headache, focal neurological deficits, macrocrania, epilepsy, developmental delay and hydrocephalus [23]. Headaches are the most common symptoms in older children (reported in as many as 70% of symptomatic cases) and usually presented as chronic recurrent attacks. It is speculated that headaches are caused mainly by increased intracranial pressure, which is frequently evidenced by the presence of papilloedema [23]. Meanwhile, macrocrania and a temporal bulge are the most common symptoms in infant [22, 26]. Expansion of arachnoid cysts induces the remodeling process of the bone structure, resulting in deformity of the frontal part of the temporal bone. The remodeling process is that bone tissue undergoes continuous renewal with osteoclasts, which resorbs the calcified matrix and osteoblasts and synthesizes a new bone matrix [3]. It is still controversial whether there is a relationship between epileptic seizures and intracranial arachnoid cysts without obvious intracranial pressure signs. However, arachnoid cysts in the middle cranial fossa can present epileptic seizure most frequently in all arachnoid cysts. One of the possible mechanisms is mass effect derived of arachnoid cysts, such as compression and irritation of the surrounding cortex, and disturbance of local CSF dynamics [27]. Some authors indicated positive results concerning the surgical treatment of arachnoid cysts under the conditions of simultaneous epileptic seizures [28, 29]. In our case, a young male patient with an arachnoid cyst in the middle cranial fossa suffered with syncope attack accompanied with electroencephalogram

(EEG) abnormality on the ipsilateral to the arachnoid cyst. Endoscopic fenestration was successfully performed, and the syncope attack and EEG abnormality were immediately disappeared. However, some authors suggested that arachnoid cysts might not be related to a specific seizure and EEG focus [30, 31]. Although, in some cases, arachnoid cyst is considered as epileptic focus and its surgical treatment is considered to be effective, it remains not to be conclusive. However, seizures and headaches often persist despite adequate surgical treatment of the cyst is performed. Therefore, some have suggested a role for intracranial pressure monitoring or cerebral blood flow measurement to assist with surgical decision making [32, 33].

The mechanism of cyst expansion remains unclear and Williams et al. suggested to be due to CSF pumping through persisting communication pathways between the cyst and CSF space caused by venous and arterial pulsation, which induce not only bony remodeling but also direct bulbar compression [34]. As another possible mechanism of cyst expansion, a slit-valve mechanism was appeared at the point of communication [23, 35]. These two mechanisms, the “water pump mechanism” and the “valve mechanism” force a one-way CSF flow into the lumen of arachnoid cyst leading to cyst expansion [36]. Trauma is considered to be a possible developmental factor of arachnoid cyst, and causes splitting and duplication of the endomeninx in infancy, which is a period of incomplete cistern formation [37]. Representative case is presented as Figure 1.

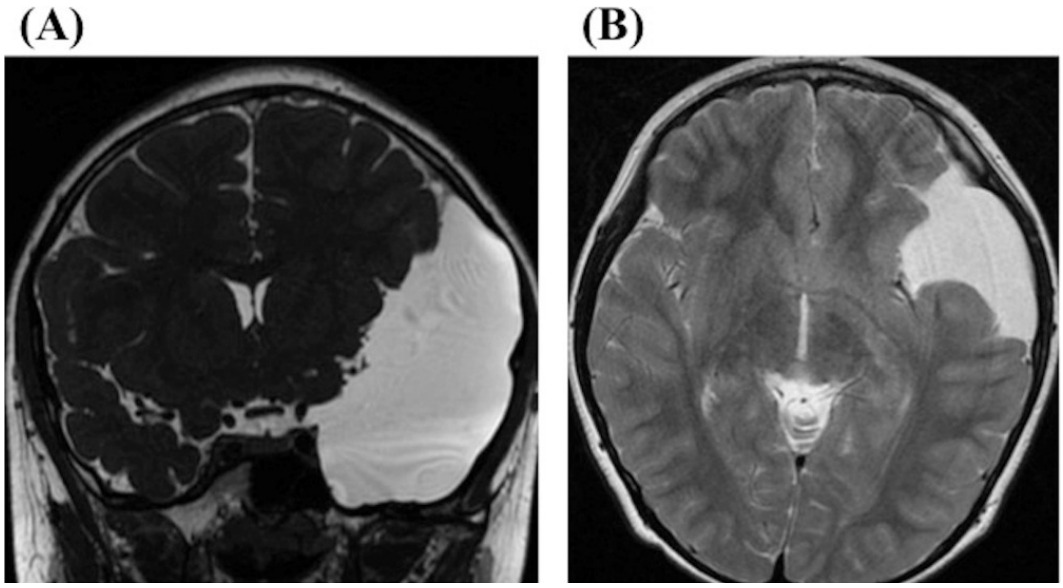

Figure 1. A 9-year-old boy complained of headache presented with a huge arachnoid cyst located in the middle cranial fossa. MRI (A; coronal section on fast imaging employing steady-state acquisition (FIESTA), B; axial section on T2 weighted image (WI)) showed that the arachnoid cyst compressed the surrounding cerebral parenchyma and bulged onto the temporal convexity. Although a light motor retardation had been recognized on preoperative neurological examination, it improved after endoscopic fenestration between the cyst and the carotid cistern.

Cerebellopontine Angle Arachnoid Cyst

The CPA represents the second most common location and accounts for about 11% in all arachnoid cysts [16]. These cysts have been considered to become symptomatic in early childhood. Although there have been only a few small series reporting the CPA arachnoid cyst about the clinical presentation and the natural history, they have been reported to become symptomatic with cranial nerve dysfunctions, ataxia, headache, nystagmus and vertigo [38, 39]. It has been suggested long-term compression of the cranial nerves in the CPA may cause irreversible nerve damage [40]. The most affected cranial nerve is vestibular nerve followed by facial nerve. However, some authors indicated that decompression of vestibular nerve and facial nerve with making fenestration of CPA arachnoid cyst resulted in complete recovery from dysfunctions of both nerves [41, 42]. They addressed that the thick arachnoid wall of the CPA arachnoid cyst transmitted the pulsatile activity of a displaced CPA artery as a mechanism of development and recovery of symptoms [41]. The timing of surgery and the ages are thought to have some role to resolve the symptoms, and children are generally more resilient to neural tissue trauma and have more chances to recovery from the compression with arachnoid cyst compared to an adult [42]. In the cases associated with hydrocephalus, it has been postulated that there is a direct relationship between the development of an arachnoid cyst and abnormal CSF dynamics. However, the development and the expansion of other arachnoid cysts located in the CPA in early childhood remain unknown [43]. Representative case is presented as Figure 2.

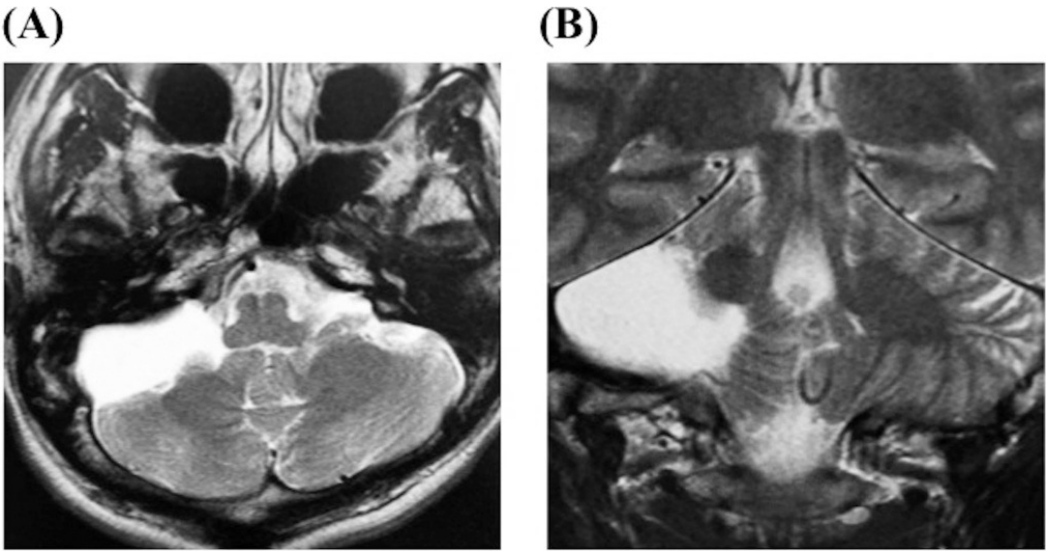

Figure 2. A 35-year-old male suffered from mild headache presented with arachnoid cyst at the cerebellopontine angle on MRI (A; axial section and B; coronal section on T2WI). His symptoms did not include the insufficiencies of vestibular nerve and facial nerve, therefore, no operative indication was considered. And headache disappeared after a little while.

Interhemispheric Arachnoid Cyst

Interhemispheric arachnoid cysts arise from arachnoid membrane of the interhemispheric fissure or parasagittal region, and they account for 5-6% of all intracranial arachnoid cysts [12]. They usually accompany agenesis or hypogenesis of corpus callosum [44], and exist both sides on the same extent manner. Neuroradiological evaluation showed remarkable compression onto the surrounding cerebral parenchyma, and the anterior horn of the lateral ventricle present characteristic configuration, which is called “bat wing appearance.” The interhemispheric arachnoid cysts frequently present multi-lobular, different from other types of intracranial arachnoid cysts [45].

They are made a diagnosis until 2 months old in half of the cases and present various symptoms, including intracranial hypertension syndrome (headache, vomiting, and papilloedema), macrocrania, seizure, psychomotor delay, motor insufficiencies, gait disturbances, urinary incontinence mainly due to compression onto the surrounding cereberum [27,46]. Therefore, if the cysts are so large that they are close to the ventricle or the cistern, it is necessary to build a stable communication between the cyst and the normal CSF pathway. The procedures of endoscope are reported to be effective with few complications [47]. Representative case is presented as Figure 3.

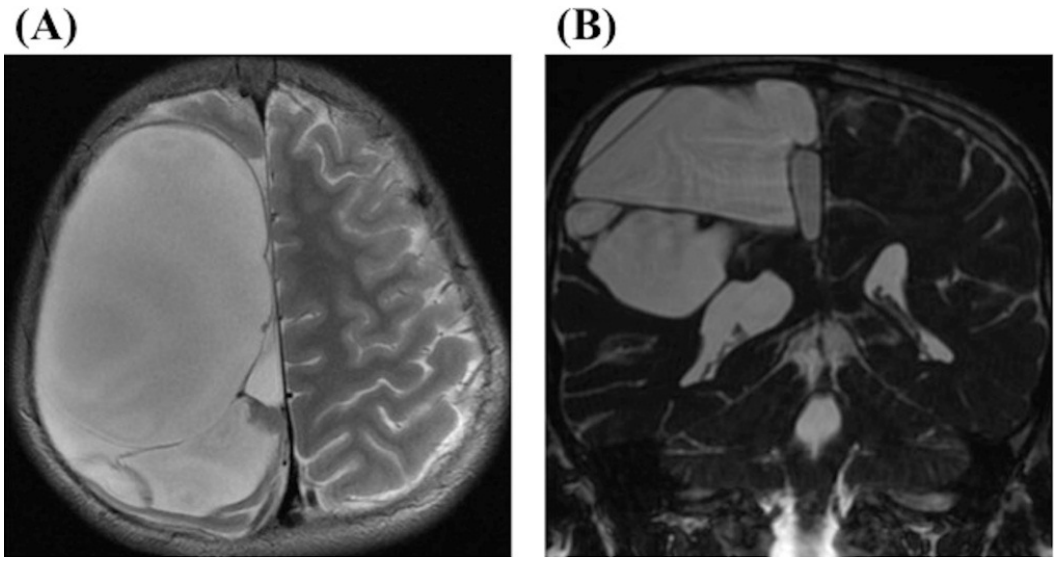

Figure 3. An 11-year-old boy manifested mild hemiparesis presented with interhemispheric arachnoid cyst with multiple cystic components on MRI (A; axial section on T2 WI and B; coronal section on FIESTA). His symptoms improved after endoscopic fenestration between multi-cyst components.

Cerebral Convexity Arachnoid Cyst

Cerebral convexity arachnoid cysts arise from arachnoid membrane of the cerebral convexity, and they account for 4% of all intracranial arachnoid cysts. Their symptoms are different depending on the patients’ group of ages. In childhood, they are progressing in size

and characteristically present an asymmetrical macrocrania. In adulthood, their symptoms are seizure, headache, and progressive hemiparesis due to compression of elevated intracystic pressure. Among the symptoms the cysts can manifest, seizure is the most popular and usually associated with headache. Their neuroradiological evaluation revealed convexity of the skull asymmetrically enlarged with bony thinness or erosion, and suture split [16, 48]. Representative case is presented as Figure 4.

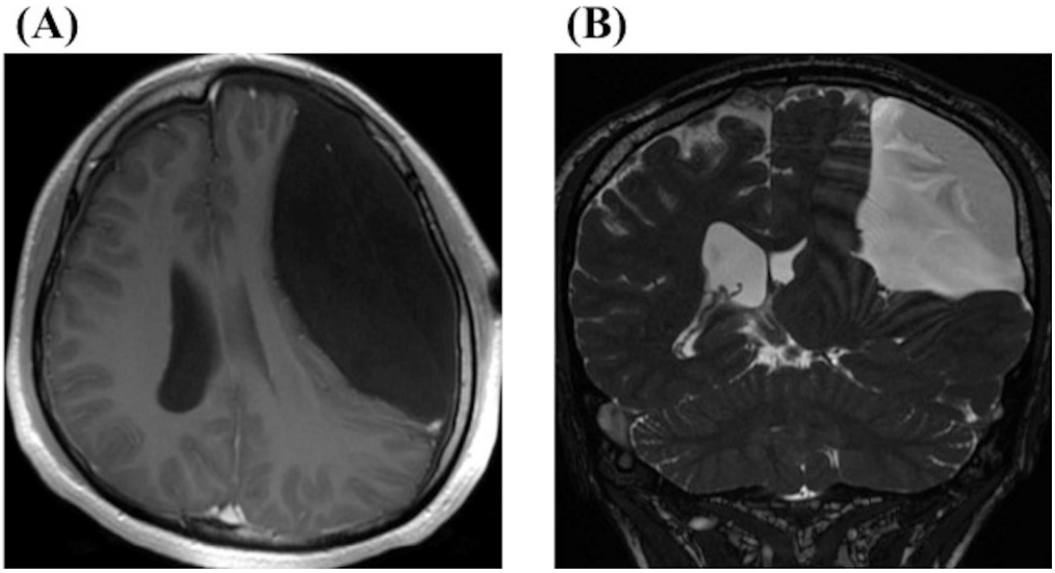

Figure 4. A 22-year-old male complained of headache and unsteadiness presented with a huge convexity arachnoid cyst at the parietal region. MRI (A; axial section on T1WI, B; coronal section on FIESTA) showed that the arachnoid cyst revealed marked compression on the adjacent cerebral parenchyma and ipsilateral lateral ventricle. His symptoms were completely disappeared after endoscopic fenestration between the cyst and the surrounding subarachnoid space.

Suprasellar Arachnoid Cyst

Suprasellar arachnoid cysts arise from an anomaly of the diencephalic membrane of Liliequist, either as a diverticulum or from splitting of the membrane and secretion of CSF within the cyst, and account for 1-10% of all intracranial arachnoid cysts [49]. They can cause obstructive hydrocephalus as a result of compression or direct occlusion of a cerebral aqueduct or the foramen Monro in the majority of them, and progressively enlarges and become symptomatic in early childhood, and 60-90% of the patients are reported to be children [50]. Macrocrania and psychomotor retardation are the most common symptoms in infants, whereas intracranial hypertension syndrome, psychomotor retardation, seizure, and precocious puberty are the most common symptoms in older children [23, 51]. Stretching of the optic nerve and chiasm over the cyst wall causes the visual function insufficiencies, such as visual field defect, optic atrophy, and papilloedema. In addition, the compression to the proximity of the hypothalamus and pituitary induces endocrinological abnormality, such as amenorrhea, short stature, and obesity as well as precocious puberty [50].

Two types of suprasellar arachnoid cyst can be demonstrated with neuroradiological evaluation and neurosurgical observation. First, the most frequent type, a communicating cyst as a result of dilation of the interpeduncular cistern. It has been understood as the Liliequist membrane, which is probably congenitally imperforate, expands upward to form a communicating cyst with the prepontine cistern by CSF pulsations and elevated intracystic pressure below [52]. Second, less frequent type is a non-communicating cyst of the diencephalic membrane of the Liliequist membrane to form a suprasellar cyst [53]. Caemaert et al. addressed another possible mechanism of cyst expansion, which is a slit-valve mechanism at the point of communication [35]. He reported the first endoscopic observation of a slit-valve created when the basilar artery pierces the prepontine arachnoid membrane, and the valve action was found to be synchronous with arterial pulsation. Crimmis et al. described the intraoperative endoscopic finding about the slit-valve mechanism as “duck-beak” and recognized the same findings in 80% of their study [54]. El-Ghandour reported that this suspected valve mechanism was verified with endoscopic observation in 68% of patients of their series. These cysts are thought to be originated in the prepontine cistern, rather than in the suprasellar cistern, and can extend upward into the suprasellar cistern [23]. Bobble-head doll syndrome is a rare movement disorder associated with suprasellar arachnoid cyst, and characterized by a 2- or 3-Hz back and forth movement of head, and sometimes also of the neck, trunk, and upper extremities. The pathophysiological mechanism of this bobble-head is speculated as the rhythmic movement caused by the intracystic pulsation pressure on the surrounding structures, such as thalamus and on its connections to the basal ganglia and the motor cortex by the dilated third ventricle [55].

Intrasellar Arachnoid Cyst

A major difference between intrasellar arachnoid cysts and other arachnoid cysts is that these arachnoid cysts are not associated with normal CSF cistern because leptomeninges are considered to be absent in the sellar turcica [56]. On the other hand, cystic lesions within the sella turcica extending into the suprasellar cistern are frequently detected. These lesions include pituitary adenoma, Rathke cleft cyst, arachnoid cyst, pituitary cyst, and other rarer cysts, however, intrasellar arachnoid cysts are very rare [57, 58]. Although their pathogenesis remains unclear, Beneditti et al. presented an ectiopathogenetic correlation between primary empty sella syndrome and intrasellar arachnoid cysts. Initially, largely communicating subarachnoid spaces extending into the sella turcica, and the subarachnoid spaces are secondarily isolated by closure of the communication because of meningitis, hemorrhage, and inflammation. Subsequently, intrasellar arachnoid cysts are formed. Dubuisson et al. suggested a developing mechanism of these arachnoid cysts as follows. There is no arachnoid membrane below the diaphragm sellae under the normal condition. A large diaphragmatic aperture and pulsatile CSF force would let the suprasellar cistern enter into the sella turcica. The pituitary stalk and gland could work as a ball-valve mechanism to reocclude the dural defect after CSF entrance. Apposition of arachnoid membrane at some point could result in a communicating cyst in the sella turcica [57].

They become symptomatic mostly in adulthood, and this is quite different from suprasellar arachnoid cysts, which become symptomatic mostly in childhood [59]. Visual function disturbances and headache are common presenting symptoms associated with these

intrasellar arachnoid cysts (55-60%). Endocrine symptoms are less common, however, mostly suffered axis of the hypothalamus and pituitary is gonadotropin, followed by thyrotropin and adrenocorticotropin. Their symptoms are menstrual irregularities, infertility, decreased libido, or impotence [56, 58, 60]. Representative case is presented as Figure 5.

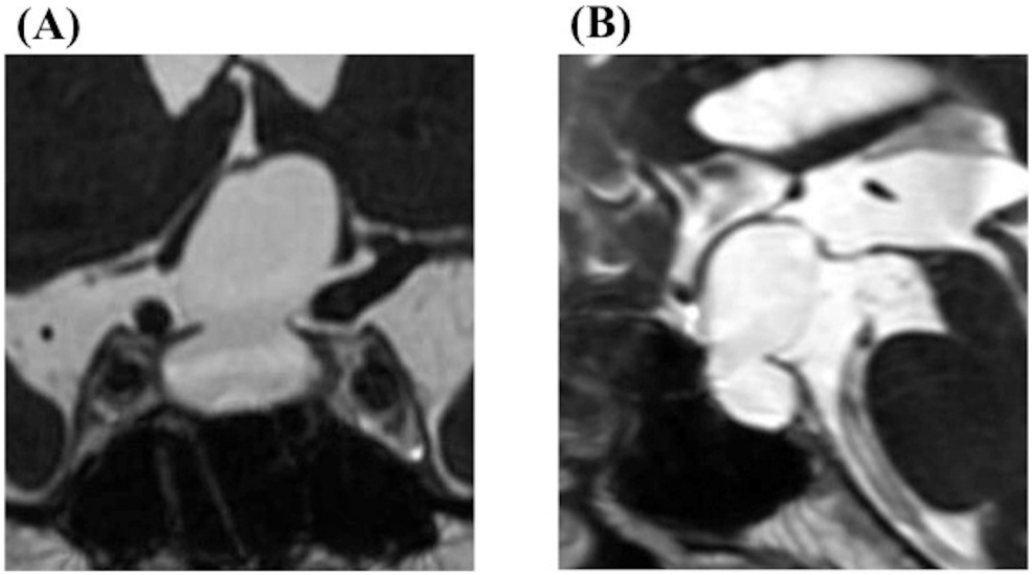

Figure 5. A 62-year-old female manifested with visual function impairment showed a large intrasellar arachnoid cyst extending into the suprasellar cistern. MRI (A; coronal section and B; sagittal section on FIESTA) revealed the arachnoid cyst compressed the optic chiasm remarkably. Her symptom completely diminished after endoscopic cyst removal with endonasal approach.

Quadrigeminal Cistern Arachnoid Cyst

In 1993, a quadrigeminal cistern arachnoid cyst was first classified as “quadrigeminal plate cyst,” originating in the quadrigeminal cistern region by Di Rocco et al. It has been reported that quadrigeminal arachnoid cysts are rare, and account for 5-10% of all intracranial arachnoid cysts [16, 61]. These cysts may have different extension toward surrounding regions, according to the presence of “loci minoris resistentiae,” such as the region of the trigone cranially, the supracerebellar cistern caudally, the third ventricle anteriorly, and the ambient cistern laterally. Because of their intimate relationship with dorsal midbrain, the quadrigeminal cistern arachnoid cysts make the cerebral aqueduct distort or compress at an early stage, resulting in symptomatic with obstructive hydrocephalus in almost all the cases [62]. Symptoms are commonly caused by associated hydrocephalus, such as macrocrania, intracranial hypertension syndrome (headache, vomiting, lethargy, and papilloedema), or compression of the dorsal midbrain, such as visual impairment (convergence spasms, abnormality of the pupils, upper lid retraction), and Parinaud syndrome. Seizure, developmental delay, gait ataxia are other uncommon symptoms. The duration of symptoms are reported as a range from 1 month to 1.3 years (mean 9.5 months). Because symptomatic quadrigeminal cistern arachnoid cysts are almost inevitably associated with hydrocephalus, it

has been increasingly reported that endoscopic treatment for these cysts can be performed safely and provide a high rate of success [63]. Representative case is presented as Figure 6.

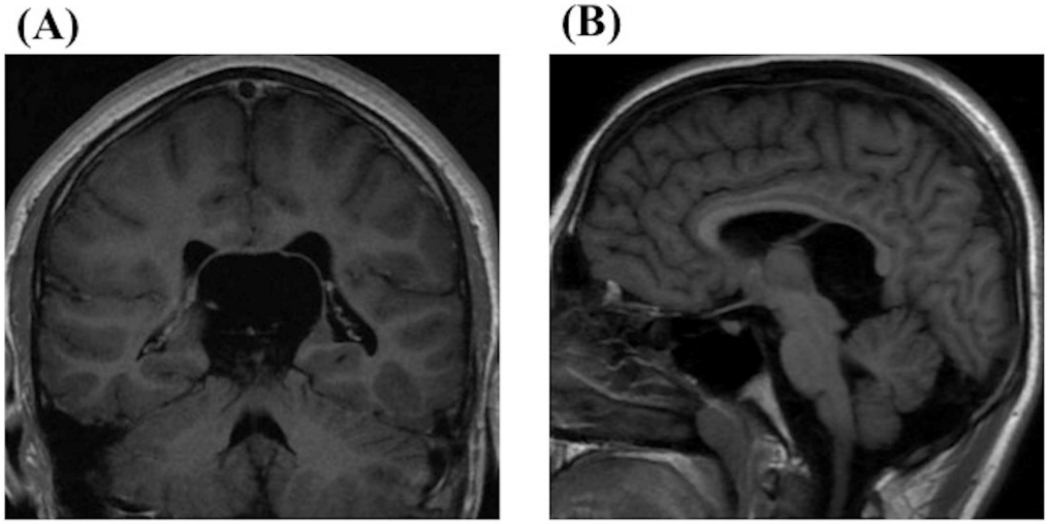

Figure 6. A 13-year-old boy associated with diplopia presented with a large quadrigeminal cistern arachnoid cyst on MRI (A; coronal section and B; sagittal section on T1WI). His symptom improved immediately after endoscopic fenestration between the lateral ventricle and the cyst.

Intraventricular Arachnoid Cyst

The origin of solely intraventricular arachnoid cyst is not fully clarified. There are isolated case reports in the literature discussing the presence of arachnoid cyst located in the lateral ventricle or the fourth ventricle [64]. They are frequently associated with obstructive hydrocephalus. Therefore, intraventricular arachnoid cysts can present with intracranial hypertension syndrome (headache, vomiting, lethargy, and papilloedema), macrocephaly and hydrocephalus in infants, and with position-related headaches in older patients [65, 66]. Ependymal, choroid plexus, epidermoid and parasitic cysts in the fourth ventricle were considered to be differential diagnosis [67]. Other manifestations of the lateral ventricle arachnoid cysts are seizure, psychomotor delay, visual function insufficiencies (disturbance at the visual tracts), and those of a fourth ventricle arachnoid cyst are progressive vertigo and ataxia [68]. Representative case is presented as Figure 7.

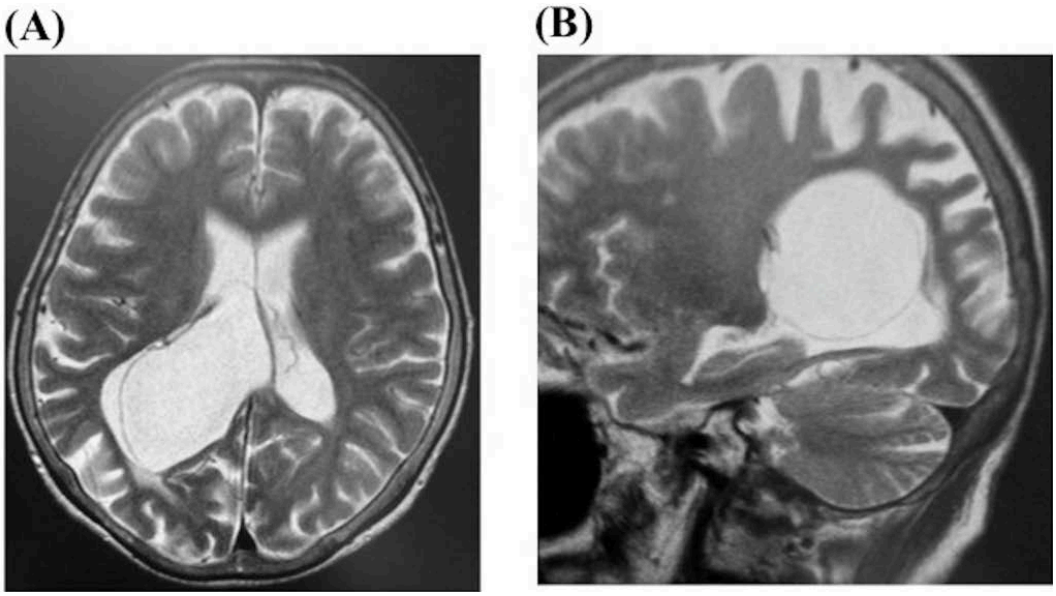

Figure 7. A 54-year-old female associated with headache and mild burred vision on her left showed a large arachnoid cyst in the right lateral ventricle on MRI (A; coronal section and B; sagittal section on T2WI). Her symptom improved after endoscopic fenestration between the lateral ventricle and the cyst.

Posterior Fossa Arachnoid Cyst

Posterior fossa arachnoid cysts are divided according to their location, the vermis-cisterna magna, the cerebellar hemisphere, the cerebellopontine angle, the quadrigeminal cistern, and the cerebellocervical junction. These cysts are characteristically associated with obstructive hydrocephalus by obstructing the openings of the fourth ventricle or by trapping of these orifices at the foramen magnum, and may evolve with cerebellar tonsillar descent (acquired Chiari malformation) and syringomyelia. There exists a pressure gradient across the cranial and spinal compartment, due either to the increased intracranial pressure. Oldfield et al. proposed that the descended cerebellar tonsils could obstruct the CSF flow to and from the spinal compartment at the foramen magnum, leading to the development of syringomyelia from the findings of dynamic MRI study [69]. In addition to these findings, these cysts frequently accompanied with other congenital anomalies at the skull base, including achondroplasia and platybasia [65, 70]. Their symptoms are intracranial hypertension syndrome (headache, vomiting, lethargy, blurred vision, 6^{th} cranial nerve palsy, and papilloedema), followed by macrocrania, split sutures, irritability, and developmental delay in infants and young children, and neuralgia, ataxia, dizziness, and vertigo in older children [46, 70]. Among them, headache is a clinical relevant factor for these patients. In the series by Helland and Wester [24], 11 out of 13 surgically treated posterior fossa arachnoid cysts presented with headache, and only one of them had a poor clinical outcome after surgical decompression.

We present with a rare case of quadrigeminal cistern arachnoid cyst [71]. A 28-year-old woman complained of chronic and progressively worsening headache following the delivery of her first child. MRI revealed marked triventriculomegaly, arachnoid cyst located in the quadrigeminal cistern, and cerebellar tonsillar descent (Figure 8). Papilloedema suggested a long-standing elevation of intracranial pressure. Endoscopic third ventriculostomy was performed successfully and resulted in complete recovery from her headaches and papilloedema. Postoperative MRI revealed resolution of ventriculomegaly and cerebellar tonsillar descent, suggesting that the forth ventricle outlet obstruction was associated with the development of the hydrocephalus.

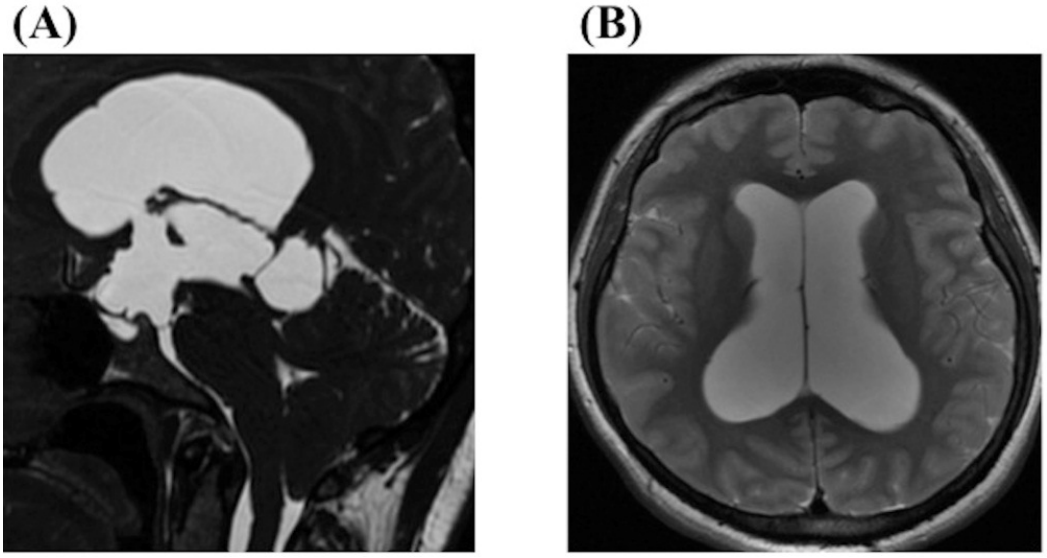

Figure 8. A 28-year-old female complained of chronic and progressively worsening headache after delivery of her first child. MRI (A; coronal section and B; sagittal section on T1WI) revealed an arachnoid cyst located in the quadrigeminal cistern, and cerebellar tonsillar descent with marked ventricular dilatation. Endoscopic third ventriculostomy was performed successfully and resulted in complete relief from her headaches.

SYMPTOMS MANIFESTATION ACCORDING TO DIFFERENT CONDITIONS OF ARACHNOID CYSTS

Pathogenesis of Arachnoid Cyst Associated with Hydrocephalus

Although, in most cases, arachnoid cysts are clinically asymptomatic and remain static in size, these cysts may cause various neurological signs once hydrocephalus appears in association with arachnoid cysts depending on their locations. There are several explanations for the occurrence of hydrocephalus as follows; First, hydrocephalus may be produced by mechanical obstruction of CSF pathway by arachnoid cysts (obstructive hydrocephalus) [52, 70]. Second, CSF dynamics aberration caused by arachnoid cysts make derangement in CSF flow and absorption [72]. Third, intraventricle arachnoid cysts are thought to arise from displacement of arachnoid cells by the vascular mesenchyme, through the choroidal fissure,

during the process of choroid plexus development [66]. Fourth, arachnoid cysts of the interhemispheric fissure may represent an extension of preexisting hydrocephalus, especially in cases that associate with corpus callosum dysgenesis. Fifth, in cases of multiple cysts, the origin of both the hydrocephalus and the arachnoid cysts seems to be closely related to a diffuse faulty development of the subarachnoid space and/or to abnormal deposits on the arachnoid surface and/or the convexities of the brain [65, 72]. Arachnoid cysts may be pathologically related with hydrocephalus, and conversely, arachnoid cysts may cause hydrocephalus. Aberrant CSF dynamics seems to play a major role in the development of both entities.

Combination with Intracranial Hemorrhage in Arachnoid Cysts

Many authors addressed intracranial hematoma as one of the important complications of arachnoid cysts. A small percentage present with hemorrhage, with annual risk estimated at 0.1% per year [73]. As intracranial hemorrhages co-existed with arachnoid cysts, CSDH were reported most frequently, followed by subarachnoid hemorrhage, acute subdural hemorrhage, acute epidural hemorrhage, and intracystic hemorrhage. In a study of 658 patients with CSDHs, Parsh et al. reported a 2.43% incidence of arachnoid cysts [73]. Among them, since Davidhoff and Dyke first reported the co-existence of arachnoid cysts and chronic subdural hematomas (CSDHs) [74], it has been well known that the arachnoid cysts located in the middle fossa occasionally associated with the development of CSDH [75-77]. Most of the CSDHs associated with arachnoid cysts exist on the same side of the cysts. Cress et al. performed a case-control study evaluated the association of arachnoid rupture (intracystic hemorrhage, adjacent subdural hematoma, or adjacent subdural hygroma) in pediatric patients with previous asymptomatic arachnoid cysts [78]. They suggested larger arachnoid cyst size (larger than 5cm in maximal diameter) and recent head trauma (within the previous 30 days) are risk factors for symptomatic arachnoid rupture and hemorrhage. However, the exact developmental mechanism of the co-existence of these two entities remains unknown. As described above, in most cases, arachnoid cysts are clinically asymptomatic and remain static in size, they can become symptomatic if once CSDH occur.

CSDH associated with arachnoid cyst is considered to emerge from the vessels surrounding the cyst wall or floating inside the cyst [79, 80]. Several authors have emphasized the structures between the dura mater and the arachnoid membrane. However, it is very difficult to detect the bleeding point during CSDH surgery associated with arachnoid cyst because normal anatomical structures are already distorted by hematoma. This CSDH associated with arachnoid cyst has some characteristics in their clinical features as follows; First, they usually have history of minor trauma, and sport is predominant as etiology of trauma. CSDH manifested typically within a few weeks after a minor head injury and their neurological symptoms are due to intracranial hypertension [81,82]. Second, the arachnoid cysts locate in the middle cranial fossa in most of cases, and Galassi type II account for about 50% among them. Third, CSDHs occurs mainly in older children whose ages are from 10 to 15, which is much younger that usual cases with CSDH [83].

The possible pathogenesis of the CSDHs associated with arachnoid cysts has been described as follows. First, arachnoid cysts have less compliance than a normal brain, resulting in reduced intracalvarial cushioning following trauma. The fluctuating movement of the cyst transfers shearing movements to the outer membrane thus tearing the small vessels

between the outer membrane and the dura mater, and causing small bleeding into the subdural space [79, 84]. Second, injury of the bridging veins or vessels running in the cyst wall due to the easy transfer of pressure through the cyst facilitates the influx of the blood into the subdural space [80, 85]. Third, laceration of the cyst wall due to trauma leads to the influx of CSF and blood within the membrane, and formation of granulation tissue with an inflammatory reaction. Subsequent repeated hemorrhages from the organized granulation tissue lead the development of hematoma within, inside, or outside the cyst wall [20, 86]. Regarding the granulation formation, it was assumed that the vessels' ingress occurs around the cyst wall after its formation, and the injury of vessels can cause an inflammatory response to lead to the formation of the granulation tissue and subsequent hematoma [74].

We have already presented a rare case of a 9-year-old girl suffered with a headache, nausea, and diplopia. MRI showed an arachnoid cyst in the left-side middle fossa and ipsilateral CSDH with a remarkable mass effect. Irrigation of the CSDH, partial removal of the outer membrane of the cyst and CSDH, and endoscopic cystocisternostomy were performed to relieve her symptoms (Figure 9). Postoperative clinical course was excellent. Histopathological examination of the outer membrane of the CSDH demonstrated an arachnoid cell layer and hemorrhage from the granulation inside the membrane, and collagen fibers outside the membrane. These findings strongly suggested that the membrane and the content of the CSDH were derived from the outer membrane of the arachnoid cyst, and the CSF including the hemorrhage within the membrane, respectively. Laceration and hemorrhage from granulation tissue within the outer membrane was considered as one of the developmental mechanisms of the CSDH associated with arachnoid cyst in the middle fossa [87].

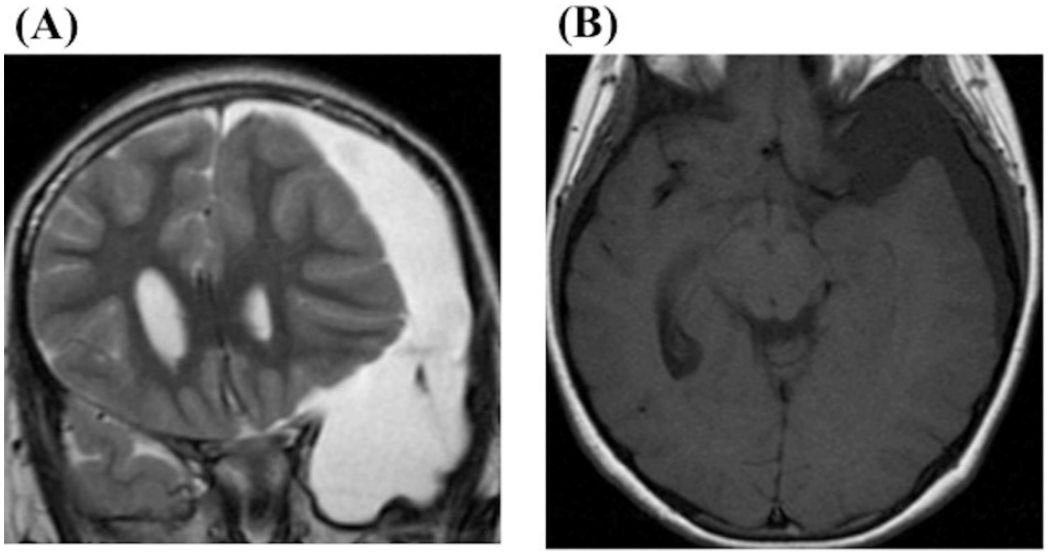

Figure 9. A 9-year-old girl suffered with a headache, nausea, and diplopia showed an arachnoid cyst in the left-side middle fossa and ipsilateral CSDH with a remarkable mass effect on MRI (A; coronal section on T2WI, B; axial section on T1WI). Irrigation of the CSDH, partial removal of the outer membrane of the cyst and CSDH, and endoscopic cystocisternostomy were performed to relieve her symptoms. Postoperative clinical course was excellent.

Symptoms Manifestation in Adult and Elderly Patients with Arachnoid Cysts

Arachnoid cysts can present with various symptoms and signs during early childhood, and after that most of arachnoid cysts remain clinically stable. However, arachnoid cysts in adult and elderly patients occasionally have the symptoms, which can show good recovery after proper surgical treatment [88]. Their symptoms are seizure, headache, and hemiparesis in adult, and headache, recognition impairment, hemiparesis, gait disturbance, seizure and urinary incontinence in elderly. It is unlike in children, intracranial hypertension syndrome is reported to be rare. Because arachnoid cysts in the elderly are found in the almost same locations as arachnoid cysts in children, the developmental mechanisms leading to this condition are considered to be similar between the two aged groups. While the progressive growth of the skull and plasticity of the brain in children can buffer the mass effect caused by a cyst, the breakdown of compensatory mechanisms over the surrounding cerebral parenchyma in elderly patients with or without cyst expansion can lead to relatively rapid progression of symptoms, once they appear. However, factors inducing the breakdown of compensatory capacity remain unknown.

We encountered a very rare case of 76-year-old woman who presented with progressive gait disturbance. MRI of the head showed a large arachnoid cyst in the foramen of Magendie that was compressing the inferior vermis and medial aspects of the cerebellar hemisphere without causing hydrocephalus (Figure 10). Neurological examination revealed cerebellar ataxia without Romberg's sign. Complete excision of the cyst was successfully performed via a median suboccipital approach. The patient's postoperative course was excellent and her neurological recovery was remarkable. Most cases of the arachnoid cysts located in the foramen of Magendie are reported in children, and it is extremely rare to observe such arachnoid cysts in the elderly. A symptomatic foramen Magendie arachnoid cyst has never been reported previously in an elderly person. Our results indicate that proper surgical intervention can yield highly positive outcomes in such cases [89].

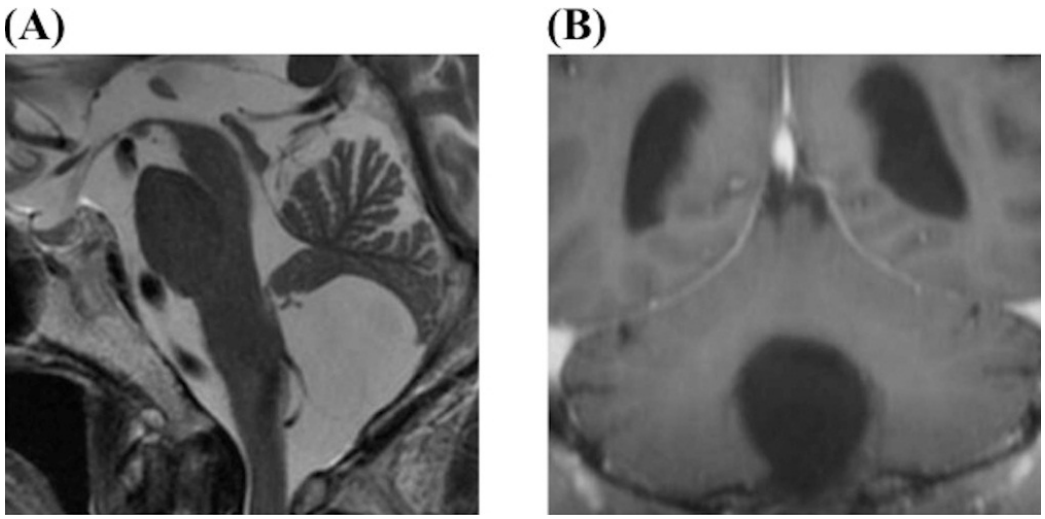

Figure 10. A 76-year-old female presented with unsteadiness and gait disturbance revealed an arachnoid cyst at the foramen of Magendie on MRI (A; sagittal section on T2WI and B; coronal

section on T1WI). Her symptoms improved remarkably after cyst removal with midline suboccipital approach.

EXPANSION OF ARACHNOID CYST

Supposed Mechanisms of Cyst Expansion

It is well known that the natural history of most arachnoid cysts remains stable in size and clinically silent in the long term. However, arachnoid cysts have the potential to expand progressively, or on rare occasions, to disappear spontaneously [27, 90]. As the genesis of congenital arachnoid cysts, they have been demonstrated to arise from splitting of arachnoid membrane in the early intrauterine developmental period and secretion of CSF within the cyst. This Starkman's theory has been generally accepted as that arachnoid cysts are developmental anomalies and CSF-like fluid is collected to form the cysts subsequently [6, 16, 91]. Mattel et al. presented a two-hit hypothesis for the development of arachnoid cyst from their findings, in which the combination of an embryological defect in arachnoid development followed by a second event causing impairment of CSF absorption in early childhood could lead to abnormal CSF dynamics and the consequent expansion of fluid collections in the intraarachnoid spaces [20]. Large arachnoid cysts are thought to represent mainly cysts isolated from the subarachnoid space, and these cysts can be increased in size and compress the surrounding cerebral parenchyma [92]. The prevalence of headache and macrocrania have been reported as many as 70% of children with arachnoid cysts [30], on the other hand, definite neurological symptoms that may be correlated with arachnoid cysts, such as psychomotor delay, hemiparesis, or seizure, are found in only 10-20% of the patients, and usually occur along with further radiological enlargement of the cysts [20, 27].

Although arachnoid cysts are inferred that they can enlarge by several mechanism including ones reported previously, the exact mechanism is still a mystery and should be studied in more depth. Arachnoid cysts with symptomatic manifestations have mass effect with elevated intracystic pressure and are clinically important either to treat surgically or follow-up conservatively [19]. Mechanisms proposed so far to explain the enlargement and persistence of cysts include active secretion of fluid produced by identified microvilli on the inner surface of ependymal cells on the cyst wall [93], fluid flux from an osmotic gradient between the cyst content and the CSF [94, 95], and a slit-valve mechanism that functions as one-way valve between the cyst and the subarachnoid space [94, 96]. Simple fenestration of arachnoid cysts will only create a weak point for CSF leakage, and the shrunken cyst will return to its original size or larger. Therefore, it is very important to build a stable CSF communication between the cyst cavity and the surrounding cistern or ventricle.

A minimally higher sodium or protein content could produce an osmotic gradient, which can induce the cyst enlargement [90]. In addition, fluid may be secreted directly into the cyst by the arachnoid cells on the cyst wall or ectopic choroid-like structures. The arachnoid cells have been shown to contain all of the organelles and the enzymes associated with secretory cells. And this mechanism has been supported with the following observation that the walls of arachnoid cysts transport fluid to the center of the lesion [16, 97].

Halani et al. observed endoscopically in the case of suprasellar arachnoid cyst that the slit-valve mechanism of net inflow into the arachnoid cyst. They described a small opening in

the arachnoid wall of the cyst was present directly over the basilar artery, forming a net one-way slit-valve for CSF flow [19]. Caemart et al. observed and reported the presence of the slit-valve mechanism directly. The valve action was found to be synchronous with arterial pulsation, where the basilar artery pierces the prepontine arachnoid membrane [35]. The endoscopic observation revealed an open slit-valve with caudad-to-cephalad CSF flow into the cyst over the caudal basilar artery could be found during the cardiac cycle. On the other hand, a decreased positioned slit-valve with cephalad-to-caudad CSF flow could be detected during the remainder of the cardiac cycle [19]. This slit-valve mechanism would lead to a rather small amount of CSF accumulation during each cardiac cycle, resulting in a slow and gradual increase in size of the arachnoid cyst. Given that the endoscopic observation of this slit-valve phenomenon is presented likely early in life, it would be reasonable that most symptomatic cases of suprasellar arachnoid cysts are reported to occur mainly in childhood with enlargement of the cysts with concomitant obstructive hydrocephalus [6, 36, 90]. In addition, it was also speculated that the location of the slit-valve over the basilar artery is an important key to allow a net influx of fluid with the arterial pulsation and leads to mass effect and clinical manifestation [6, 58, 90].

On the contrary, Halani et al. reported that the communicating arachnoid cyst could be found in the middle cranial fossa arachnoid cyst from endoscopic observation [19]. This arachnoid cyst had a small circular opening through which the cyst communicated with the CSF of the Sylvian fissure, and did not show progressive enlargement and lacked a slit-valve on the cyst wall. The circular opening in the cyst wall allowed bidirectional flow of CSF with equal resistance. According to those findings, they speculated that lack of a slit-valve on the cyst wall strongly suggested that the arachnoid cysts are likely to remain stable in size and silent in clinical condition in a long term.

Hayashi et al. present a rare suggestive case with an arachnoid cyst located in the parietal convexity in the aspect of the expansion of the arachnoid cyst [98]. This arachnoid cyst combined with the subcutaneous tissue mimicking meningocele through a bony and dural defect in the parietal convexity. In the prenatal period, bony and dural defects supposed to be attributable to their maldevelopment due to an expansion of preexisting arachnoid cyst with excessive elevated intracystic pressure or others. They considered the term ‘arachnoid cyst extending into subcutaneous tissue’ might be preferable in this condition.

Spontaneous Regression of Arachnoid Cyst

On the contrary, spontaneous regression or disappearance of the arachnoid cysts was reported to be rare, and this phenomenon was mainly occurred in the arachnoid cysts located in the middle fossa [99-101]. However, Arunkumemar et al. reported one case with the cyst in the posterior fossa, which appeared to lessen in size spontaneously with complete diminishment of symptoms and then enlarged with a worsening in neurological status [102]. Two mechanisms were reported so far as follows; First, elevated intracystic pressure resulting from coughing, crying (Valsalva maneuver), or head trauma leads to the laceration of inner or outer membrane of the cyst, resulting in the communication between the cistern or subdural space and the cyst cavity. If the laceration of the outer membrane of the cyst was produced by head trauma or Valsarva maneuver, the laceration let the cyst fluid enter into the subdural space to make subdural effusion. Then, when subdural effusion is absorbed, the arachnoid

cyst is also disappeared spontaneously [100, 101]. Otherwise, the subdural effusion turns into CSDH, and then surgical irrigation is performed when it expands to be symptomatic. After irrigation of CSDH, the arachnoid cyst is going to be disappeared [99]. Second, arachnoid cyst is coexisted by CSDH, which appeared spontaneously without trauma, and the outflow of the fluid inside the cyst into the CSDH is induced with the osmotic gradient, leading to the disappearance of the arachnoid cyst.

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List of the Representative Publications Last 3 Years

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