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Expanding Cyst of the Septum Pellucidum – Endoscopic Observations on the Mechanism of Development and Results of Treatment

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Abstract

Cysts of the septum pellucidum (CSP) are usually asymptomatic; however, in some cases they can begin expanding and cause neurological deterioration. The mechanism leading to the formation of an expanding cyst of the septum pellucidum (ECSP) is not known. Based on observations made during endoscopic treatment of ECSP we analyzed intraoperative findings in respect to cyst formation mechanism and treatment prognosis. A group of 31 patients was studied. Only cases with bulging cyst walls occupying the frontal horns observed on imaging studies were included. The main symptom was a severe, intermittent headache. In three cases short term memory deficits were diagnosed. In one case papilloedema was observed. All patients underwent endoscopic fenestration of the ECSP. There were no cases of cyst reocclusion during a follow-up period of 1–14 years (mean 6.2 years). In 30 cases headaches resolved completely and in one case its intensity was significantly smaller. There was one case of postoperative hemiparesis. In all but two cases the thin, translucent region in the anterior part of the cyst floor was found. In the region small fissures and in three cases choroid plexus were observed. Endoscopic fenestration is the efficient treatment for ECSP. ECSP is formed on the basis of not completely closed, developmental communication of the cyst with other fluid spaces. The communication is opened by transient elevation of intraventricular pressure, and acts as a valve leading to fluid accumulation among the walls of the previously asymptomatic cavum septum pellucidum.

Key words: **neuroendoscopy, septum pellucidum, cyst**

Introduction

Cavum of septum pellucidum (CSP) appears during the 3rd month of intrauterine growth and subsequently begins to close gradually in caudal to rostral direction at approximately 6 months. It is found in 100% of preemies, 37% of newborns and 10% of adults and is usually an asymptomatic, incidental finding when the central nervous system is imaged.[1)] However, in some cases, it can begin expanding, causing neurological symptoms. The mechanism leading to expansion is poorly understood. Endoscopic methods offers minimally invasive treatment of expanding cyst of septum pellucidum (ECSP) and at the same time permits for *in situ* observation of this pathology.[2–11)] In the following study, we currently present the largest published group of patients with ECSP, who underwent endoscopic fenestration, and analyze the outcome and intraoperative findings with respect to the cyst formation mechanism.

Materials and Methods

Patient population

The study included 31 patients with ECSP (17 females and 14 males) aged 10–37 years (mean 21.4 years) operated between 2000 and 2015 in our

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institution. All patients complained of daily episodes of intermittent severe headache. In one case, the headache returned 3 months after stereotactic ECSP puncture. Additionally, three patients exhibited a short-term memory deficits diagnosed by Stenberg Memory Scan. In two of the patients, preoperative follow-up imaging studies revealed gradual cyst enlargement. In one case, during a preoperative neurological follow-up development of papilloedema was observed. In the other cases, the treatment was undertaken due to increasing headaches intensity. Only the cases with rounded, bulging walls of the cyst, as a sign of increased inner pressure, visible on magnetic resonance imaging (MRI) studies were included. T_2-weighted axial and coronal images with 1 mm slices were used for evaluation. Retrospective study protocol was approved by the Institutional Review Board. All subjects gave informed consent for treatment and additional tests.

Operative method

The patient is positioned supine with the thorax slightly elevated and the head in the neutral position. The burr hole is placed 5 cm from the midline and 1 cm anterior to the coronal suture. The dura is incised in cruciate fashion. A 3.5-mm rigid endoscope is introduced into the frontal horn of the right lateral ventricle. In 25 cases, radionics optical neuronavigation system was used to precisely place the endoscope in the ventricle narrowed by the cyst. Dynamic irrigation with warm lactate Ringer's solution is used. After visualization of the foramen of Monro and identifying the septum pellucidum, place for fenestration anterior to the septal vein and superior to the foramen is localized. Fenestration is performed with a bipolar cautery to obtain an opening not smaller than 1 cm. After fenestration of the right lamina the endoscope is introduced in the lumen of the cyst. Subsequently, the left lamina is fenestrated in the same manner. Fenestration is finished when the endoscope can be advanced to the opposite lateral ventricle to inspect bifrontal cyst communication. After hemostasis the endoscope is withdrawn, the dura opening covered with gelfoam and the skin sutured in a typical manner. Two endoscopes were used: at first GAAB type, then after upgrade pediatric Oi unit.

Results

The postoperative follow-up period was 1–14 years (mean 6.2 years). In 30 patients, headache resolved completely and in one was of significantly smaller intensity and frequency. In all but one patient with preoperative short-term memory deficits, the improvement occurred during the first few postoperative days. In one case, the deficit got worse postoperatively. During a 2-year follow-up, the patient showed a significant improvement compared with the preoperative period; however, slight deficits of short-term memory still persisted.

In one case, during placement of the endoscope shaft into the narrow ventricle, the head of the caudate nucleus was accidentally penetrated. Postoperatively, the patient presented a moderate hemiparesis which improved to a slight weakness 6 months after the surgery and remained stable during follow-up. In four cases, due to a direct contact of the extended walls of the cyst with the lateral walls of the frontal horns, the endoscope was introduced directly through a narrow space of the ventricle into the lumen of the cyst. In these cases, bilateral fenestration was performed without difficulties from this position. During the follow-up period, there were no cases of cyst reocclusion.

Intraoperative findings

In all cases, the walls of the cyst were under tension, bulging into the frontal horns. It caused significant narrowing of the anterior aspect of the lateral ventricles to such a degree, that in some cases, navigation in the ventricle was performed by pushing away the wall of the cyst from the lateral wall of the ventricle with the endoscope. Also, the floor of the frontal horns was elevated compromising lower part of the foramen of Monro, with the septal and the thalamostriate veins markedly dilated (Fig. 1). Even after the cyst was fenestrated and its walls relaxed this configuration of the foramen persisted, however the veins became less prominent. The inner walls of the cyst had the same endoscopic appearance as the outer walls. In 29 cases, the anterior part of the cyst floor was thinner, becoming a pulsating, translucent membrane. Among undulating layers of the membrane, small fissure-like openings could be observed (Fig. 2). During pulsations the membrane was pushed into the cyst, what caused distention of the openings and then out of the cyst with collapse of the openings. In three cases, elements of the choroid plexus protruding through the opening were visible. The membrane was in direct contact with the leptomeningeal and subarachnoid interhemispheric space and/or third ventricle. Occasionally, the medial surface of the hemispheres and branches of the anterior cerebral arteries, in the region of lamina terminalis cistern, could be seen through it. In five cases such thinning of the floor could be observed in two places; one behind the other. The posterior thinning was located over the third ventricle. In these cases, the endoscopic fenestration was performed in the thinned area and anterior part of the third ventricle visualized through it (Fig. 3).

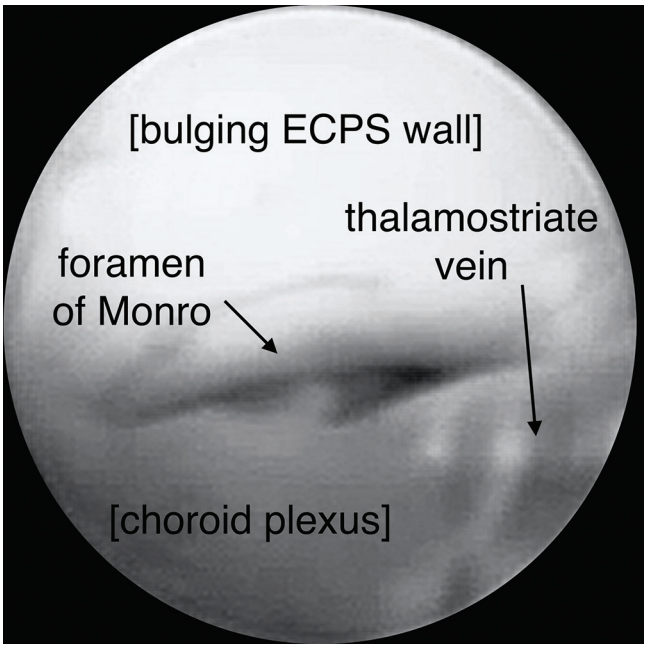

Fig. 1 Endoscopic view of the frontal horn of the right lateral ventricle before ECSP fenestration. The frontal horn of the lateral ventricle and lumen of the foramen of Monro are narrowed by bulging wall of the ECSP. ECSP, expanding cyst of septum pellucidum.

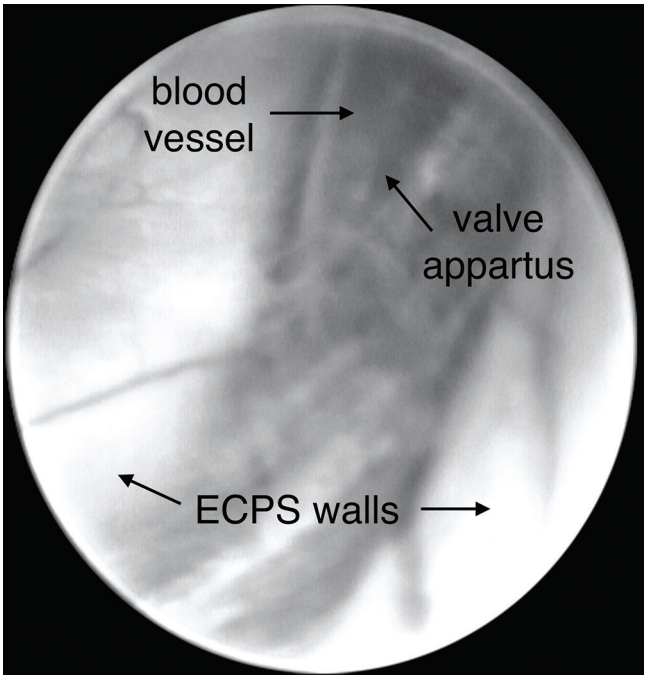

Fig. 2 Endoscopic view of the membranous area at the anterior part of the ECSP floor. Slit among layers of the membrane with the uncovered blood vessel coming through the opening-valve apparatus. ECSP, expanding cyst of septum pellucidum.

Imaging studies

Analysis of preoperative MR images revealed that there can exist possible communication of the ECSP through the anterior part of the floor with region of the lamina terminalis cistern or with the supraoptic recess of the third ventricle (Fig. 4). The pericallosal cistern seemed to be separated from the cyst. The cyst's bulging walls hung down over the foramen of

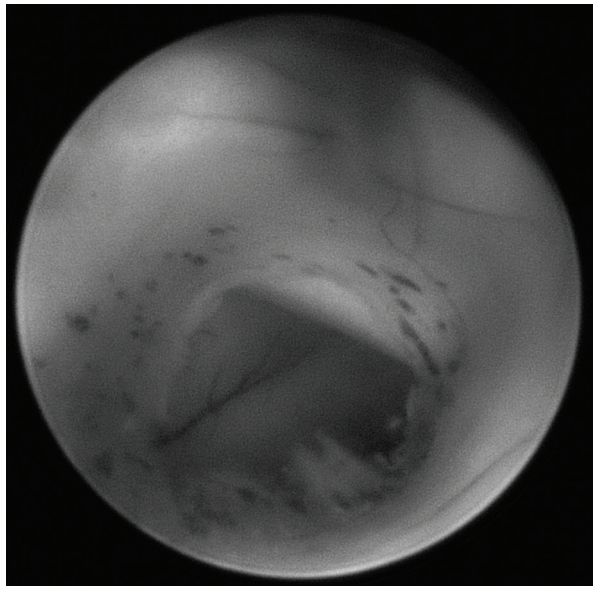

Fig. 3 View of the third ventricle through the fenesration of the membrane in the cyst floor.

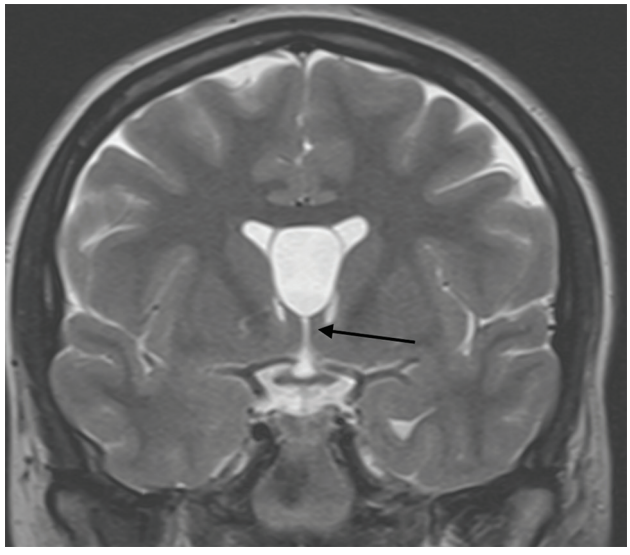

Fig. 4 Preoperative MR image showing distended walls of the ECSP narrowing the lower aspect of the frontal horns and CSF space continuation between the ECSP and the region of lamina terminals cistern (*arrow*). ECSP, expanding cyst of septum pellucidum.

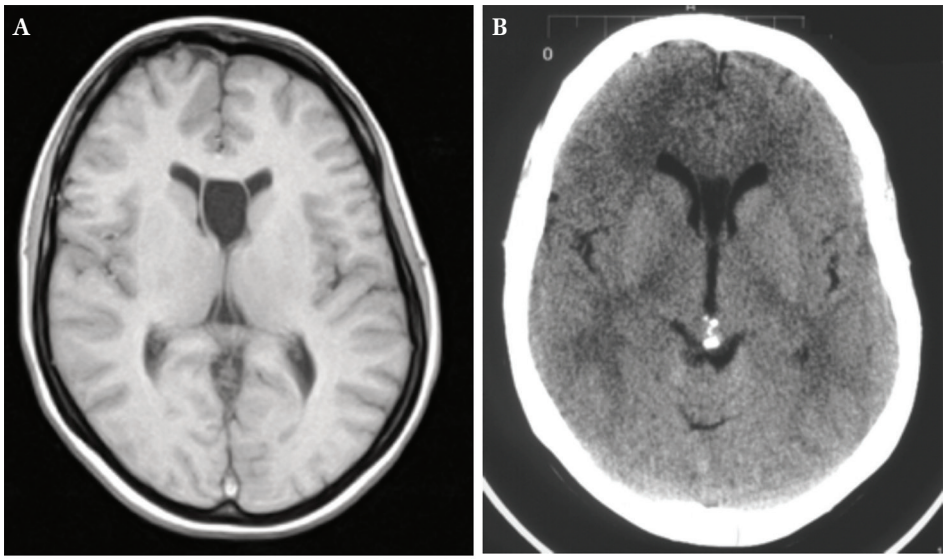

Fig. 5 Before endoscopic fenestration the cyst's bulging walls hung down over the foramen of Monro and rest against the head of the caudate nuclei as a sign of increased inner pressure significantly narrowing the posterior part of the frontal horns (A). Postoperative CT image demonstrating relaxed, parallel walls of the cyst (B).

Monro and rested against the head of the caudate nuclei, significantly narrowing the posterior part of frontal horns (Fig. 5A). Immediate postoperative imaging revealed deflation of the cysts with parallel walls in all cases (Fig. 5B). Preoperative cine MR, which was performed in three cases, revealed turbulent cerebrospinal fluid (CSF) flow inside the ECSP without signs of fluid jets.

Discussion

Treatment

Cavum of septum pellucidum are generally asymptomatic and usually incidental findings on brain imaging studies performed for other reasons. In these cases, the walls delineating the cavity have a straight course, without bulging into the frontal horns of the lateral ventricles.[12] However, in a small percentage of cases the cysts begin to expand. It is thought that in these instances at least part of the neurological symptoms are caused by the compression of adjacent structures by the cyst walls which expand due to increased intracystic pressure.[13–21]

In cases of ECSP, an operative treatment is undertaken with good results since the report by Meyer.[13] Several techniques such as craniotomy, internal cystoventricular and cystoperitoneal shunting and stereotactic fenestration have been used for decompression.[17,20,22–24] Endoscopic surgery makes it possible to communicate these lesions with the lateral ventricles in a minimally invasive way and still under visual control. This method allows for direct inspection of performed fenestration quality with preservation of the conditions in the ventricular system close to the physiological one. It obviates the need to place a shunt and makes it possible to observe the anatomy and behavior of the ECSP *in vivo*. Published studies on the efficacy of this treatment consists mainly of case reports and a small series reported recently.[1–10] The series presented here is, to the best of our knowledge, the largest one analyzed to date.

Frontal approach seems to secure a good entry point for bilateral cyst fenestration and inspection of the foramen of Monro. However, in cases when the large ECSP fills the anterior horns of the ventricle, the endoscope can be mistakenly introduced directly into the cyst. It does not affect the fenestration technique; however, one has to be aware of such a possibility in order to not get lost, mistaking the interior of the cyst for the ventricle. It happened four times in our series in cases with significantly dilated cysts. In such cases, the lumen of the frontal horn is almost hypothetical and attempting to place the endoscope into the frontal horn jeopardizes periventricular structures. This complication occurred in one of the presented cases. Therefore, a purposeful, direct entry into the cyst with the use of a navigating system seems to be safer in this situation. The other option is a posterior approach through the right occipital burr hole and the occipital horn of the ventricle.[5] This approach obviates the risk of damage to vascular and neural structures surrounding the foramen of Monro. However, in our opinion, the trajectory makes bilateral fenestration difficult. Authors reporting this approach were able to perform bilateral fenestration in one of the two presented cases.

It is hard to explain postoperative worsening of preexisting memory deficits in one of our patients, since surgery was uneventful and fenestration was performed in the part of cyst walls significantly anterior to the fornix. A possible explanation is that due to the changed anatomy of the region, fibers of

the fornicis also spread to the front in a distended walls of the septum. Therefore, the structure was more prone to injury during fenestration. An atypical arrangement of the fornix in ECSP and cavum vergae was already reported.[1,25,26]

The technique of fenestration is a basic endoscopic skill. However, orientation at the frontal horn after introduction of the endoscope can be difficult. The bulging walls of the cyst usually are in direct contact with the head of the nucleus caudatus and obscure such orientation points as the foramen of Monro and choroid plexus. These structures are localized by pushing aside the wall of the cyst while advancing the endoscope. During the follow-up period, we did not observe the recurrence of the preoperative symptoms. Nevertheless, our endoscopic observations revealed that despite fenestration of the cyst, deformation in the region of inferior walls of the frontal horn still persisted. The floor of the frontal horns was elevated and narrowed the lower part of the foramen of Monro posing potential resistance to CSF outflow. Therefore, follow-up of the patients should be continued. Ventriculomegaly was not observed in the presented series. Performed endoscopic communication of ECSP with the ventricles was sufficient to secure appropriate CSF circulation. Therefore, placement of ventriculoperitoneal shunt seems to be necessary, if at all, in sporadic cases.[1]

Mechanism of expansion

The mechanism of ECSP formation is not fully understood. In our material and the reports of other authors this malformation is found in the pediatric as well as in the adult group. In some cases, there is a documented triggering event, such as head trauma, accidental placement of the ventricular catheter in the CSP or venous sinus occlusion.[27–31] There are also reports of concomitant enlargement of the ventricles.[19,32,33] These findings point to the role of two factors; preexisting structural anomaly and episodes of increased intracranial pressure. In all but two cases, we observed a thin, translucent membrane at the bottom of the cyst. Small slits among the layers of the pulsating membrane were visible. During CSF, pulsations cyclic movements of the membrane could be observed. It protruded into the cyst causing distention of the slits and subsequently relaxed leading to collapse of the openings. These observations imply that the membrane creates a slit valve which is the entrance of the CSF and causes its entrapment in the ECSP. In three cases, the choroid plexus protruded through the membrane. The origin of the plexus is hard to determine. It was not visible on preoperative MR images. However, it was probably the plexus from the roof of the third ventricle since part of the ECSP was located directly over the ventricle what was confirmed by us during endoscopy. These changes at the floor of the ECSP were always located in its anterior part, as if there were anatomical or developmental basis for it. Rakic and Yakovlev[34] showed, in the human foetus, that growing fibers of the rostrum close the CSP which is originally opened to the interhemispheric fissure. The incomplete closure may lead to the formation of a persistent point of lower resistance between the CSP and the interhemispheric fissure or the third ventricle. Communication at the region of the anterior part of floor of the ECSP was found in cases studied post mortem and during preoperative computed tomography (CT) cisternography.[1,30] Our endoscopic *in situ* observations confirm these indirect findings. Presumably, there exists such an originally incomplete closure persisted from developmental stages. As a point of lower resistance, it creates a precursor for formation of the valve mechanism. Transient elevation of the intracranial pressure leads to tearing of this thin part of the CSP and changes it into one-way flow structure. It would explain cases of transformation of CSP into ECSP documented in the literature.[29,32,35] Formation of ECSP in an iatrogenic cases also speaks for the role of one way valve mechanism.[28,31] In these cases, the ECSP was formed after accidental placement of a ventricular catheter into the CSP or septum pellucidum. The perforation of the CSP lead to an inflow of CSF around the catheter. The outflow was compromised by a higher resistance from the hydrocephalic ventricle, not properly drained by the misplaced catheter, what finally resulted in a distention of the cavity. Diffusion through the cyst wall or production of fluid by the tissue lining of the cyst are two other alternative hypothetical mechanisms considered to be possible explanations for the ECSP formation. The diffusion hypothesis postulates that the CSF travels passively from the ventricle into the septum through the septal lamina by a pressure gradient and is absorbed by septal capillaries and veins. In this case, ECSP forms as a result of problems with veinous CSF absorption.[29,36] However, if this hypothesis were true then we would observe ECSP in a majority of patients with veinous obstruction. Various authors proposed that the cystic fluid could originate from the cells lining the cavum wall.[3,22,37] However, the nature of the cells, lacking typical features of ependymocytse, has been a controversial issue and histologic findings did not clearly reveal cellular apparatus typical of CSF production.[3,12,25] Additionally, it is difficult to explain the cyst's sudden expansion at a random point in the patient's life with this theory. Ronsin et al.[1] found that cellular structure of the inner surface of the cyst walls is similar in asymptomatic and symptomatic CSP. Therefore, it does not seem that fluid secretion by cyst walls

plays any important role in ECSP formation. Intermittent and recurrent character of headaches speaks for the temporary elevation of the intraventricular and intracystic pressure which can be subsequently diminished only by a relatively fast outflow of CSF. Such dynamic changes could not be explained on the basis of secretion/absorption mechanisms. The more possible explanation is that enlarged ECSP obstructs CSF outflow through the foramen of Monro as it has been postulated.[16,19,38] This obstruction causes a temporary rise in intraventricular pressure which forces the fluid out of the cyst. The excess fluid from the cyst is pushed out through the previously described opening, changing it momentarily from a one way to two way flow apparatus. Subsequently, relaxed walls of the cyst facilitates outflow through the foramen of Monro. The process is cyclic and leads to chronic intermittent headaches. There are several reports on ECSP leading to an obstruction of the foramen of Monro and hydrocephalus.[16,17,19,24] We did not observe enlargement of the lateral ventricles in the presented series. However, in all cases, cyst walls were bulging and covered the head of the caudate nucleus. Such displacement of septal walls creates a partial obstruction to CSF flow through the foramen of Monro.[39] The floor of the frontal horns was also elevated and occluded the lower aspect of the foramen which was clearly seen during endoscopy. These changes made the foramen of Monro much more prone to occlusion by the expanding cyst and subsequently led to intermittent accumulation of the fluid in the lateral ventricles. Shaw and Alvord[25] distinguished two forms of CSP: incidental (asymptomatic) and symptomatic with increased internal pressure. Our observations and critical review of the literature indicate that ECSP may develop from asymptomatic CSP, lying dormant for a long time. It is understandable that the majority of the reports present the cases examined after the symptoms had started. Therefore, it is hard to define morphology of septum pellucidum before the formation of the symptomatic cyst. However, there are few reports when brain imaging was performed for other reasons, before ECSP developed.[29,30,32,33] In all these cases asymptomatic CSP was present. Thus, classifying CSP as asymptomatic and symptomatic, one should be aware that these two terms can describe temporary forms of a dynamic process. Transition from the asymptomatic form to the symptomatic one can occur on the basis of developmental anomaly of the region and intraventricular pressure changes. The major limitation of the present study is the difficulty in observation of the CSF flow. The endoscopy offers possibility of the direct observation of the cyst in almost natural conditions. However, direction of the CSF flow can be estimated only indirectly by observation of the movement of surrounding structures. The CSF flow dynamic in the intracerebral cysts is usually too small to capture the location of the inflow by flow sensitive MRI. Therefore, further studies of symptomatic patients with ECSP are necessary to elucidate the nature of formation and natural history of this pathology.[38]

Conclusion

Endoscopic fenestration is an efficient treatment for ECSP. Additionally, endoscopic inspection of the *in vivo* morphology of ECSP allows for observation of entities not previously noticed. ECSP forms in cases of incomplete development of the septum pellucidum. The prerequisites for its formation are non-fused lives of the septum and incompletely closed embryonic communication between them and the lamina terminalis cistern or the supraoptic recess of the third ventricle. In situations of transient increase of the intracranial pressure this potential communication is forced open by CSF and activated as a one-way valve. It leads to conversion of the asymptomatic CSP into the symptomatic ECSP.

Conflicts of Interest Disclosure

The authors declare that they have no conflicts of interest.

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