Microsurgical management of vascular malformations of the pineal region

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Title Page

Title
Microsurgical management of vascular malformations of the pineal region

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Abstract

Background: Vascular pineal malformations are rare, and technically demanding lesions. Due to their location, in the last decades, endovascular techniques and radiosurgery have been increasingly used to accomplish a safe occlusion. Nevertheless, sometimes, microsurgical treatment may be required.

Methods: We present a retrospective review of the vascular pineal malformations operated by the senior author. Moreover, we report illustrative cases for the various types of vascular lesions with a careful analysis of the different microsurgical stages.

Results: Eighteen patients with pineal vascular lesions were operated on between 1980 and 2015: 6 patients had vein of Galen malformations, 5 plexiform arteriovenous malformations, 6 cavernous malformations, and one patient had a ruptured medial posterior choroidal artery aneurysm. A complete resection/occlusion was possible in all vascular malformations.

Conclusions: The pineal region is an infrequent but challenging location for vascular lesions. A careful and stepwise operative strategy for the different types of vascular lesion is paramount to accomplish an effective and safe microsurgical treatment, when other alternatives fail or are not available.

Key words:
Arteriovenous malformation, Cavernous malformations, Multidisciplinary management, Microneurosurgery, Pineal region lesions, Vascular malformations, Vein of Galen malformation

Abbreviations and Acronyms
AVM, Arteriovenous malformation, VOGM, Vein of Galen malformation
Introduction
Vascular pineal lesions are placed in a surgically challenging intracranial location. Various surgical routes and microsurgical techniques have been developed along the years for dealing with pineal lesions, all aimed at providing safe and effective surgical access while minimizing neurovascular manipulation.\textsuperscript{1-3}

In the last decades, endovascular techniques as well as radiosurgery have been increasingly used to accomplish a safe occlusion of vascular pineal lesions. Nevertheless, sometimes, when endovascular managements fail and/or if other alternatives are not available, microsurgical treatment may be indicated.\textsuperscript{4-14}

We present a retrospective review of the patients with vascular pineal malformations operated by the senior author (JH). Moreover, we report illustrative cases for the various types of vascular lesions with a careful analysis of the different microsurgical stages.

Our goal is to describe the cornerstones to perform the microsurgical management of these lesions in a simple, safe and effective way, so that it could be easily reproduced in other neurosurgical centers.\textsuperscript{1,2,15,16}

Methods

Study design and ethics
This is a retrospective study on a series of consecutive pineal vascular malformations microsurgically operated by the senior author (JH) between 1980 and 2015. Following the Institutional Review Board approval, data were collected from hospital records. The patients' consent was not required as this is a retrospective study, and the research data analysis had no effect on the participants.

Participants
The senior author along his career operated 18 patients with pineal vascular lesions. We present demonstrative cases for the various types of vascular lesions with a careful analysis of the different microsurgical stages, based on a review of the operative videos.

Pre- and post-intervention considerations
Clinical and microsurgical features of the cases, as well as postoperative long-term clinical and radiological follow up were analyzed.
Results and Illustrative cases

Eighteen patients with pineal vascular lesions were operated on by the senior author (JH) between 1980 and 2015 in two different centers: 6 patients had vein of Galen malformations (VOGMs), 5 plexiform arteriovenous malformations (AVMs), 6 cavernous malformations, and one patient had a ruptured medial posterior choroidal artery aneurysm. Eleven patients with pineal vascular malformations were operated on in our center between 1997 and 2015. Table 1 describes patients’ characteristics.

A complete resection/occlusion was possible in all vascular malformations.

Illustrative cases

Case 1: Vein of Galen malformation

Pre-operative Status: A two-year-old boy harbouring a giant VOGM associated with a left hippocampal plexiform AVM was admitted to our department. During the first year of life, signs of congestive cardiac failure were detected by echocardiography. The patient underwent multiple but minimal endovascular procedures without success (Fig.1).

Finally, it was decided to manage the lesions microsurgically. The patient underwent a two-stage surgical treatment due to the lateral component of the associated plexiform malformation that, in our opinion, would have been extremely difficult to resect by a midline posterior approach. In this regard, the AVM was resected through a subtemporal approach. Then, after few days, an occipital interhemispheric approach, as detailed below, was performed to occlude the VOGM.

Microneurosurgical management: The patient was placed in the “sitting-praying position”, and a bilateral occipital interhemispheric approach was carried out under the operating microscope. After a single layer musculocutaneous incision, a single burr hole craniotomy was performed. A high-vascularized dura mater was detached using a long blunt flexible dissector, and swabs soaked in peroxide as well as bipolar coagulation were used for the hemostasis. The superior sagittal sinus was covered with Tachosil® (Takeda Austria GmbH: Linz, Austria) and cottonoids. “Tack up” stitches were used to prevent epidural collections.

Initially, we opened the dura mater on the right side of the superior sagittal sinus. A running suture and a strong retraction controlled the bleeding coming from its border. Few bridging veins were coagulated, and a big cottonoid was placed on the occipital lobe surface to avoid injuries in case of retraction. Feeding vessels of the vein of Galen aneurysm along the falx cerebri were coagulated and cut, and the falx itself was divided just
proximal to the aneurysm. Once the splenium of the corpus callosum was reached, the aneurysm was identified by Doppler ultrasound and intraoperative angiography. At this point, the splenium was partially divided in order to approach the superior and anterior walls of the vascular lesion. Then, multiple feeders coming from the posterior cerebral artery were coagulated and cut. After the occlusion of the feeders, the aneurysm was shrunk by bipolar coagulation along its superior and anterior surface. Intraoperative angiography showed still some filling into the vein of Galen aneurysm.

Hence, the dura mater of the left side of the superior sagittal sinus was opened, all the residual feeders were occluded, and the remaining part of the aneurysm was shrunk by continuous bipolar coagulation as well. A small residual portion of the previously operated AVM was completely resected. Finally, we repeated an intraoperative angiography that showed a complete occlusion of the VOGM. After a careful hemostasis, the closure, skin included, was performed under the microscope. The surgical time was 6.4 hours (Fig.1).

Post-operative follow-up: The patient developed meningitis, and received medical treatment. After two months, he underwent ventriculoperitoneal shunt for hydrocephalus. At four years follow up, the patient is still very dependent (modified Rankin Scale, mRS, 4) without any cardiovascular dysfunction (Fig.1).

Case 2: Cavernous malformation

Pre-operative Status: A 33-year-old man was admitted to our department for recurrent diplopia, headache and Parinaud's syndrome. His pathological anamnesis was unremarkable. Cerebral CT scan showed hydrocephalus, and was suggestive of a pineal region lesion. Brain MRI was evocative of a pineal cavernoma (Fig.2).

Microneurosurgical management: With the patient placed in the “sitting-praying position”, a conventional right supracerebellar infratentorial approach was carried out. After the craniotomy, the dura was opened under the microscope. Once the arachnoid membranes of the dorsal mesencephalic cisterns were reached, a yellowish pineal discolored area was noted. Arachnoid membranes were opened with microscissors and the lateral borders of the cavernoma were dissected with the help of a cottonoid. Then, the hidden inferior border of the cavernoma was progressively detached using the thumb controlled suction tube in the left hand, and the bipolar coagulation forceps as well as small curved microdissectors in the right hand. Thin non-functional vascular structures running on the cavernoma’s wall were coagulated and cut. The vascular lesion was taken out in one piece. Hemosiderin borders were not aspirated. A careful coagulation, as well as Surgicel® (Ethicon Inc, Johnsons & Johnson: Switzerland) and Tachosil® were used for the hemostasis. The closure was performed under the microscope. The microsurgical time was 63 minutes (Fig.2).
Post-operative follow up: Post-operatively the patient presented some vertigo associated with balance problems, Parinaud’s syndrome, and mild neuropsychological symptoms. At 2 years follow up, the symptoms resolved almost completely with just some rare visual disturbance, and the patient could recover his driver license.

Case 3: Arteriovenous malformation

Pre-operative Status: A 47-year-old man presented continuous headache and diplopia. The radiological studies revealed a plexiform AVM of the quadrigeminal plate feeded by distal branches of the posterior cerebral arteries (Fig.3).

Microneurosurgical management: the patient was placed in the “sitting-praying position”, and a conventional right supracerebellar infratentorial approach was performed. The dura mater was incised under the operating microscope. The cisterna magna was opened in order to release cerebrospinal fluid. Once the arachnoid membranes of the quadrigeminal cistern were opened, the right medial posterior choroidal artery was disclosed. Then, we followed the course of this vessel into the pineal region up to expose the AVM, as confirmed by intraoperative angiography. The AVM was totally resected using standard microsurgical technique with the aid of the bipolar coagulation and microscissors. Intraoperative angiography was repeated to confirm the complete removal of the vascular lesion. Glue, Surgicel® and Tachosil® were used for the hemostasis.

Post-operative status: The immediate postoperative course was uneventful; diplopia still persisted as preoperatively without any other neurological complication. The radiological studies did not show any residual lesion.

Discussion

The pineal region is an infrequent location for vascular lesions. In this paper we aimed to present a detailed microsurgical strategy for the different types of vascular lesion, based on a careful surgical videos analysis from patients operated by a neurosurgeon with enormous experience in the management of such lesions.

Vein of Galen malformation

VOGMs are AVMs of the choroidal system with direct and/or indirect arterial feeders flowing into the median prosencephalic vein of Markowski.\(^17\) They represent 30% of vascular malformations in the pediatric age group.\(^18,19\)
Some anatomical aspects of this lesion include: 1) two different types of angioarchitecture are usually encountered: choroidal and mural type. The mural form presents few direct feeders to the aneurysm, and is better tolerated; 2) the presence of a limbic arch formed thanks to a communication between the anterior and posterior cerebral arteries through a pericallosal artery; 3) a classic “epsilon shape” configuration in the venous phase of the angiography, which represents the drainage of the thalamostriate veins into a subtemporal or a lateral mesencephalic vein. This is due to the absence of the sinus rectus, and to an underdevelopment of the sigmoid sinus and the jugular bulbs; 4) falcal dural channels drain the pouch of the malformation into the posterior third of the superior sagittal sinus, and later, with the maturation, into the cavernous sinus. The venous drainage includes also the facial veins and the inferior petrosal sinus.

The clinical presentation varies according to the age. The neonatal presentation (0-2 months) usually comprises a severe cardiopulmonary distress associated with pulmonary, neurological, hepatic, and renal dysfunction. Infants present with increasing head circumference and hydrocephalus; toddlers present with developmental delay, hydrocephalus, and/or seizures; older children may have subarachnoid hemorrhage, headache and/or seizures. The Bicêtre-score is helpful to evaluate the therapeutical management of neonates with VOGM. The primary goal is the stabilization of the life-threatening congestive heart failure, and the next step is the treatment of the VOGM. In infants and children, the immediate goal is to preserve the hydrovenous equilibrium, as it will be outlined later, to preserve the normal brain development, and to control the epilepsy. The decision to do not treat asymptomatic lesions may be extremely dangerous, as a delayed management of a VOGM may determine various cerebral sequelae such as calcifications, subependymal atrophy (pseudoventriculomegaly), and the stigmata of previous acute accidents with cortical and subcortical atrophy.

The endovascular procedures have improved the outcome of VOGMs. Before the advent of endovascular interventions, the mortality in the neonatal group was nearly 90%. The introduction of endovascular techniques and advanced neonatal critical care for severe cardiopulmonary illness have reduced the mortality to around 50%.

Regarding to the hydrocephalus, the aqueduct is patent in almost all patients and the abnormal accumulation of cerebrospinal fluid mainly results from an abnormal posterior convergence of the venous drainage of the brain and from the immaturity of the granulation system. The venous pressure at the venous sinus confluence is over 50 ml H_2O, and with a 1:5 ratio between the intraventricular pressure and the superior sagittal sinus pressure. The cerebrospinal fluid shunting in the setting of a persistent VOGM exacerbates the low vascular resistance and
can lead to intracerebral hemorrhage or worsening of the flow through the lesion. Any considered interventions (shunt or endoscopic ventriculostomy) should be performed at a later time, if needed.\textsuperscript{7,20}

Most children with macrocrania present some degree of mental retardation. In view of the poor prognosis of the disease, specialists and parents tend to accept as normal a child with mild retardation (up to 20\% of normal for the chronological age). Such delay allows the child to attend a normal school, although with support.\textsuperscript{20}

About the spontaneous thrombosis of the lesions, we should mention that the probability of preoperative death (6\%) is higher than the probability of spontaneous thrombosis of the lesion (1\%).\textsuperscript{7} According to a recent metanalysis the outcome for a patient with an early spontaneous thrombosis is good. However, once the disease develops, the outcome for an adult with a spontaneous thrombosis is poor.\textsuperscript{21}

As described by Li et al., the following 3 factors were associated with poor prognosis: major comorbidities (encephalomalacia, complex congenital heart disease requiring surgery, and portocaval shunt) with or without embolization, failure of the embolization procedure, and the lack of the procedure’s long-term effect (persistent congestive heart failure, progressive head enlargement).\textsuperscript{4,18,23} Others have reported that congestive heart failure, perinatal presentation, and choroidal angioarchitecture showed the worst outcomes.\textsuperscript{24} However, mortality is related with the age of treatment, worst in early stages and with cardiac failure and hydrocephalus.\textsuperscript{25}

The management of those complex lesions involves different approaches: open microsurgery, a direct transtoricular approach to ligation, transarterial embolization, or transvenous embolization.\textsuperscript{25} A structured literature review made by Khullar in 2010 found that 72\% of the patients receiving endovascular therapy had a favorable outcome, and the mortality rate was 15\%. Microsurgery was found to have an 84.6\% mortality rate. Furthermore, 76.7\% of untreated patients died. They confirmed that endovascular embolization has become the mainstay of VOGM treatment and has considerably improved outcomes in patients with VOGM. Transtoricular and transvenous approaches have not been successful.\textsuperscript{25–27}

However, in case of failure of those procedures, critical status of the kidneys and liver to use a iodinated contrast, or in centers without availability of those technologies, a skillful microneurosurgery is still an option to manage this complex pathology.\textsuperscript{28} The senior author, along his career, operated 6 cases. A total occlusion of the malformation was always reached without any operative mortality. As reported in previous publications, young patients had in all cases some neurological developmental delay.\textsuperscript{4,29}

\textit{Arteriovenous malformation}

Pineal region AVMs are rare, small vascular abnormalities mainly located into the tectal region.\textsuperscript{8} Currently there
is a consensus that AVMs are acquired lesions caused by endothelial cell disorders, and the postnatal maturation of the intracranial venous system plays an important role in their development.\textsuperscript{30–32} According to Hernesniemi et al., the prognosis of those lesions is related to the rupture status (worst in ruptured AVMs), the location (worst in deep and infratentorial location), and to the size (worst in AVMs larger than 5 cm in diameter).\textsuperscript{6,9,33,34}

Anatomic features of pineal region AVMs involve tectal and circumferential feeding arteries arising from the vertebrobasilar system which form a nidus with dilated tectal and superior cerebellar draining veins into the straight sinus.\textsuperscript{8}

A peculiar association between tectal AVMs with a probable acquired and reversible Chiari malformation type 1 and syringomielia, which may be produced by a venous congestion of the posterior fossa, was reported.\textsuperscript{8}

Feeding arteries of tentorial dural fistulas are usually branches of the meningohypophyseal trunk, middle meningeal artery, and occipital artery draining into pial veins. Treatment protocols combine transarterial embolization with stereotactic radiation or microsurgery. Microneurosurgery is indicated when the feeding arteries are too small to be embolized, if the patient does not improve after transarterial embolization, or if the risk of radiation injury of the surrounding structures is too high.\textsuperscript{35}

On the other hand, pial arteriovenous fistulas have their arterial supply from pial or cortical arteries directly draining into some cortical veins. Pial arteriovenous fistulas in the pineal region are suspected when the neuroradiologic imaging reveals bilateral thalamic hyperintensities, which are produced by venous congestion, and bilateral thalamic edema.\textsuperscript{36}

The microsurgical management of these lesions include a challenging complete marginal resection of the AVM due to the presence of functional nervous tissue closely related with the AVM itself. Then, after controlled the major feeding arteries and occluded the draining veins, a final retrograde coagulation of the nidus by bipolar coagulation would be an option. Somatosensory evoked potential and cranial nerve monitoring during the surgery would also be helpful to prevent some structural lesion.\textsuperscript{5}

Lawton et al. described a multimodality treatment of deep-seated AVMs using microneurosurgery, embolization and radiosurgery. Only one of two brainstem lesions required a transfemoral embolization and none of them radiosurgery. Both patients presented a 1-point improvement of the Glasgow Outcome Score in the early postoperative period (the preoperative values were respectively 2 and 4).\textsuperscript{10}

Salomon et al. also reported 4 cases with subarachnoid hemorrhage due to AVMs located in the quadrigeminal cistern or tectal plate. A case with devastating subarachnoid hemorrhage underwent conventional radiotherapy and was severely disabled 6 years later with 75% reduction of the AVM. Another patient went to embolization,
which resulted in 85% reduction of the AVM and developed visual problems and ataxia. Another two underwent complete microsurgical resection of the AVMs and one of them developed a new Parinaud’s syndrome. 37

Cavernous malformations

The pineal region is one of the uncommon locations for cavernous malformations, with only 21 cases reported so far according to a recent review. 12 However, we have to recognize that some big surgical series of pineal lesions include no mentioned cavernous malformations. 11,13,14,29,38–43 Those vascular lesions are often misdiagnosed with germ cell tumors, choriocarcinomas (recurrent bleeding) or hemorrhagic pineal cysts, particularly when the clinical manifestation is related with intracranial hemorrhage. In those cases, MRI studies show mixed signals without hemosiderin rim on the T1- and T2-weighted MRI, differently from a non-hemorrhagic cavernoma. 11,38 Although endoscopic or stereotactic biopsy for pineal lesions could be useful for the confirmation of the histologic diagnosis, if the suspected diagnosis is a cavernous malformation, there is a potential risk of fatal bleeding. In this regards a direct microsurgical excision of the vascular malformation is the standard treatment. 13,14

The approaches that are generally used to access the pineal region include: the supracerebellar infratentorial approach, and the occipital transtentorial approach. 11,13,14,29,38–42 In the posterior routes to the pineal regions, the sitting position may offer several advantages over the horizontal position, and several protocols may be effectively used in the clinical setting in order to prevent its major complications. 1,2,29,44–50 Whatever approach is selected, skillful and clean microneurosurgery preserving the normal anatomy is imperative during pineal region operations. 15,16

Differently from a plexiform AVM, cavernomas offer a relatively well-defined plane of dissection. While the bipolar forceps or the ring forceps pull out the lesion, soft thumb controlled aspiration separates the cavernoma from the surroundings. The endoscope assistance may be useful to recognize some residual part of the lesion in the inferior portion of the surgical field. 13,39

Glue and Tachosil ® are important tools for the hemostasis in a VOGM surgery. Glue may also be very helpful to control small bleedings in cavernous malformation surgeries.

Conclusion

Vascular malformations of the pineal region are rare lesions currently treated safely by endovascular techniques as well as radiosurgery. However, when endovascular managements fail and/or if other alternatives are not
available, microsurgical treatment is a valid option. By a careful and skillful microsurgical technique, vascular malformations of the pineal region could be managed in a simple, safe and effective way. In this regard, the described microsurgical keystones might be reproduced in other microneurosurgical centers.

Disclosure

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References


Figure Legends

**Fig.1** Pre- (A, B, C, D) and post-operative (E, F) images of the vein of Galen malformation. Preoperative MRI few days after the patient birth (A), and at 3 months (B). Digital subtraction angiography after multiple endovascular procedures (C). Preoperative 3D computed tomography angiography (D). Postoperative MRI (E) and 3D computed tomography angiography (F) showing the complete occlusion of the vein of Galen malformation.

**Fig.2** Pre- (A, B, C) and post-operative (D, E, F) images of the cavernous malformation of the pineal region.

**Fig.3** Pre- (A, B, C) and post-operative (D, E, F) images of the arteriovenous malformation of the pineal region.
<table>
<thead>
<tr>
<th>Hospital</th>
<th>Date of Surgery</th>
<th>Diagnosis</th>
<th>Age (years), sex</th>
<th>Pre-operative status</th>
<th>Pre-surgical embolization</th>
<th>Positioning</th>
<th>Approach</th>
<th>Radiographic follow up</th>
<th>Immediate clinical follow up</th>
<th>Last clinical Follow up</th>
<th>Last visit to the hospital</th>
</tr>
</thead>
<tbody>
<tr>
<td>HUH</td>
<td>October 2014</td>
<td>Quadrigeminal AVM</td>
<td>47, M</td>
<td>Headache, diplopia</td>
<td>No</td>
<td>Sitting-praying</td>
<td>SCIT</td>
<td>Uneventful</td>
<td>Nystagmus, diplopia, normal gait</td>
<td>Slight III CN deficit</td>
<td>Foreign patient.</td>
</tr>
<tr>
<td>HUH</td>
<td>April 2013</td>
<td>PCA aneurysm.</td>
<td>59, M</td>
<td>Acute perimesencephalic SAH; fourth ventricle hemorrhage; hydrocephalus; EV</td>
<td>No, VPS</td>
<td>Sitting-praying</td>
<td>SCIT</td>
<td>Uneventful</td>
<td>Sent to rehabilitation, disorientated, left sided hemiparesis</td>
<td>Independent gait, slight IV CN deficit.</td>
<td>November 2016</td>
</tr>
<tr>
<td>HUH</td>
<td>April 2013</td>
<td>VOGM (Choroidal type)</td>
<td>2, M</td>
<td>Congestive heart failure, hydrocephalus</td>
<td>Multiple sessions with partial embolization</td>
<td>Sitting-praying</td>
<td>1) ST; 2) OIH (different stage)</td>
<td>Thrombosis of the giant VOGM, cortical atrophy; and ventriculomegaly</td>
<td>Mechanical ventilation, localizes to painful stimuli</td>
<td>Very dependent, epilepsy</td>
<td>March 2017</td>
</tr>
<tr>
<td>HUH</td>
<td>October 2014</td>
<td>Cavernous malformation</td>
<td>33, M</td>
<td>Hydrocephalus, diplopia, headache, Parinaud’s syndrome</td>
<td>No</td>
<td>Sitting-praying</td>
<td>SCIT</td>
<td>Uneventful</td>
<td>Vertigo, balance problems, Parinaud’s syndrome, mild neuropsychological symptoms, sent to rehabilitation</td>
<td>Mild neuropsychological symptoms, diplopia, mild Parinaud’s syndrome</td>
<td>August 2017</td>
</tr>
<tr>
<td>HUH</td>
<td>October 2012</td>
<td>AVM of the roof of the III ventricle</td>
<td>12, M</td>
<td>Incidental finding by CTA after head trauma (CT showing panventricular hemorrhage without SAH)</td>
<td>No</td>
<td>Sitting-praying</td>
<td>1) SCIT+OIH (same stage)</td>
<td>Uneventful</td>
<td>Good, same as preoperative status</td>
<td>Slight concentration deficit</td>
<td>October 2016</td>
</tr>
<tr>
<td>HUH</td>
<td>August 2011</td>
<td>Cavernous malformation</td>
<td>80, M</td>
<td>Incidental finding by CTA-MRI (patient with tinnitus)</td>
<td>No</td>
<td>Park bench</td>
<td>SCIT</td>
<td>Uneventful</td>
<td>Slight diplopia</td>
<td>Normal</td>
<td>April 2014</td>
</tr>
<tr>
<td>HUH</td>
<td>December 2007</td>
<td>Large AVM (posterior Rt paracallosal, roof III ventricle, pineal area) + large pericallosal aneurysm (2 x 68, F)</td>
<td>68, F</td>
<td>Asymptomatic patient with history of SAH; pericallosal aneurysm (20 x 12 mm)</td>
<td>No</td>
<td>Supine</td>
<td>AIH</td>
<td>Infarction of right pericallosal artery territories</td>
<td>Bedridden, opens eyes, dependent, hospitalized</td>
<td>Left sided hemiparesis, dysphasia and neglect syndrome, nutrition by a PEG-tube</td>
<td>August 2017</td>
</tr>
<tr>
<td>Date</td>
<td>Hospital</td>
<td>Diagnosis</td>
<td>Age</td>
<td>Sex</td>
<td>Symptoms</td>
<td>Procedure</td>
<td>Outcome</td>
<td>Comment</td>
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<tr>
<td>Nov 2007</td>
<td>HUH</td>
<td>AVM draining into a VOGM</td>
<td>57</td>
<td>M</td>
<td>Aorta dilatation and aortic valve insufficiency</td>
<td>No</td>
<td>Sitting-praying SCIT Uneventful</td>
<td>Awake but disoriented, still hospitalized</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>April 2007</td>
<td>HUH</td>
<td>AVM fistula (ChPLA to sinus rectus)</td>
<td>55</td>
<td>M</td>
<td>Acute panventricular hemorrhage and hydrocephalus (full IV ventricle hemorrhage), poor clinical status</td>
<td>No</td>
<td>Sitting-praying SCIT Uneventful</td>
<td>Same as preoperative status, tracheostomy, extension movement to pain, close to vegetative status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>April 2007</td>
<td>HUH</td>
<td>VOGM (choroidal type)</td>
<td>1</td>
<td>F</td>
<td>Congestive heart failure and hydrocephalus</td>
<td>Sitting-praying</td>
<td>OIH Uneventful</td>
<td>Same as preoperative status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nov 2006</td>
<td>HUH</td>
<td>VOGM (choroidal type)</td>
<td>9</td>
<td>M</td>
<td>Thalamic bleeding and hemiparesis after embolization, developmental delay</td>
<td>Sitting-praying Midline SCIT Uneventful</td>
<td>Same as preoperative status</td>
<td>Slight right hemiparesis, developmental delay; died in an accident in 2008</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>July 1987</td>
<td>KUH</td>
<td>VOGM</td>
<td>46</td>
<td>M</td>
<td>Severe vertigo and hydrocephalus</td>
<td>No</td>
<td>Sitting-praying Rt POIH Uneventful</td>
<td>Confusion and amnesia for 2 weeks, long lasting dyslexia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oct 1983</td>
<td>KUH</td>
<td>VOGM</td>
<td>7</td>
<td>F</td>
<td>Exophthalmos, Parinaud’s syndrome, prominent facial veins, developmental delay</td>
<td>Sitting-praying POIH Uneventful</td>
<td>Good, no complications</td>
<td>Persistence of prominent facial veins and exophthalmos, some developmental delay, but goes to school</td>
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<tr>
<td>July 1982</td>
<td>KUH</td>
<td>VOGM (choroidal type)</td>
<td>4 months</td>
<td>F</td>
<td>Macrocrania, hydrocephalus, VPS</td>
<td>No</td>
<td>Sitting-praying VPS + Rt POIH Uneventful</td>
<td>Good, initially good development.</td>
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<tr>
<td>Before 1997</td>
<td>KUH</td>
<td>Four pineal cavernous malformations completely resected and with no complications</td>
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Table 1. Patients’ characteristics.

ACom, anterior communicating artery; AIH, anterior interhemispheric approach; AVM, arteriovenous malformation; ChPLA, choroidal postero-lateral artery; CN, cranial nerve; CT, computed tomography; CTA, computed tomography angiography; EV, external ventriculostomy; F, female; HUH, Helsinki University Hospital; KUH, Kuopio University Hospital; Lt, left; M, male; MRI, magnetic resonance imaging; OIH, occipital interhemispheric approach; PCA, posterior cerebral artery; PEG, Percutaneous endoscopic gastrostomy; POIH, parieto-occipital interhemispheric approach; Rt, right; SAH, subarachnoid hemorrhage; SCIT, supracerebellar infratentorial approach; ST, subtemporal approach; VOGM, Vein of Galen malformation; VPS, ventriculoperitoneal shunt
Title
Microsurgical management of vascular malformations of the pineal region

Highlights
Vascular pineal malformations are rare, but challenging lesions
We present a retrospective review of 18 microsurgically treated pineal vascular lesions
6 VOGMs, 5 AVMs, 6 cavernomas, one ruptured medial posterior choroidal artery aneurysm
A complete resection/occlusion was possible in all vascular malformations
The main microsurgical stages for the different lesions are illustrated