Spontaneous Resolution of a Meckel’s Cave Arachnoid Cyst Causing Sixth Cranial Nerve Palsy

Maud Jacob, MD, Sachin Gujar, MD, Jonathan Trobe, MD, and Dheeraj Gandhi, MD

Abstract: A 32-year-old pregnant woman developed a progressive right sixth cranial nerve palsy as an isolated finding. Brain MRI disclosed a discrete lobulated lesion centered in the right Meckel’s cave with intermediate signal on T1, high signal on T2, and diffusion characteristics similar to those of cerebrospinal fluid on apparent diffusion coefficient mapping. The initial radiologic diagnosis was schwannoma or meningioma. No intervention occurred. Shortly after cesarean delivery, the abduction deficit began to lessen spontaneously. One month later, the abduction deficit had further improved; 7 months later it had completely resolved. Repeat MRI after delivery failed to disclose the lesion, which was now interpreted as consistent with an arachnoid cyst arising within Meckel’s cave. Twenty-one similar cases of Meckel’s cave arachnoid cyst or meningocele have been reported, 7 found incidentally and 14 causing symptoms, 2 of which produced ipsilateral sixth cranial nerve palsies. All previously reported symptomatic patients were treated surgically. This is the first report of an arachnoid cyst arising from Meckel’s cave in pregnancy and having spontaneous resolution.

CASE REPORT

A 32-year-old woman in the 28th week of a normal pregnancy complained of binocular horizontal diplopia of 2 weeks’ duration. On her initial examination, abduction of the right eye was reduced to 60%. Measurements of alignment showed a 10 prism-diopter esotropia in primary gaze, worsening to 20 on right gaze and improving to 4 on left gaze. Results for the rest of the neuro-ophthalmologic examination were normal.

Examination 1 month later disclosed a complete lack of abduction in the right eye. At that time, she reported having experienced a 3-week episode of intense right periocular pain that resolved spontaneously. Otherwise, there were no neurologic manifestations.

Brain MRI performed without contrast injection because of her pregnancy showed a well-defined, lobulated, extra-axial lesion with its epicenter in the right Meckel’s cave. The lesion measured 11 mm anteroposteriorly and 6 mm transversely. It demonstrated intermediate signal intensity on T1 and high (fluid-like) signal on T2. On the ADC map, the lesion showed diffusion coefficient values similar to those of CSF (Fig. 1). The initial radiologic diagnosis was nerve sheath tumor (schwannoma) or, less likely, meningioma.

Ten weeks after the onset of diplopia, she underwent cesarean section delivery for fetal distress. The birth was otherwise normal. On the day after delivery, she noticed slight improvement in her diplopia. By 1 month after delivery, abduction of the right eye had improved to 70%, and there were no other findings. Seven months after delivery, she had had completely recovered normal ocular motility. A second MRI, performed with contrast medium at 1 month after delivery and 3 months after the beginning of her symptoms, failed to disclose the lesion (Fig. 2).
Head CT performed 3 months after delivery disclosed no erosion of the petrous apex. Based on the typical location of the lesion and its imaging characteristics, a diagnosis of an arachnoid cyst arising from Meckel’s cave was made in retrospect.

**DISCUSSION**

Our patient developed a right sixth cranial palsy during pregnancy that was associated with a cystic lesion in the right Meckel’s cave. After delivery, the sixth nerve palsy regressed, and at 1 month after delivery the lesion had vanished on MRI. Cystic lesions in that region have been given various names: Meckel’s cave arachnoid cyst (1,2), petrous apex cephaloceles (3–5), petrous apex arachnoid cyst (6–9), and arachnoid cyst involving the Gasserian ganglion (10). They are all cystic lesions centered in the posterior portion of Meckel’s cave that may expand into the petrous apex when they grow and erode the bone in this case. If the surgeon discovers a herniation of dura and arachnoid, the term “meningocele” is applied; if the cyst lining consists only of arachnoid, the term “arachnoid cyst” is applied (9). The arachnoid cyst may have herniated through a hole in the adjacent dura (10).

Eighteen cases of petrous apex arachnoid cysts have been studied previously by MRI (1–10,13). In two cases, imaging descriptions included only T2 imaging characteristics (2,5). Low T1 and high T2 signals have been found in all but one reported case (3,4,6–9), in which intermediate to high T2 signal was reported (7) (Fig. 3). Mild rim enhancement on postcontrast scans has been noted in

![FIG. 1. MRI performed during pregnancy. A. Precontrast T1 axial MRI shows a lesion in the right Meckel’s cave with intermediate signal intensity. B–C. T2 axial MRI shows a fluid-like high signal on T2. Note the prominent septations within the lesion (black arrows). D. Apparent diffusion coefficient map shows cerebrospinal fluid–like diffusion characteristics (arrow).](image)

![FIG. 2. MRI performed 1 month after delivery. Precontrast T1 axial MRI (A), postcontrast T1 axial MRI (B), and T2 axial MRI (C) show that the Meckel’s cave lesion has vanished and that the cave now appears normal on both sides.](image)
two cases (3,6) and no enhancement in five cases (3,4,7–9). Lobulation was described once (9). Similar to previous descriptions, the presumed Meckel’s cave arachnoid cyst in our patient had high T2 signal paralleling that of CSF. The only atypical feature in our patient was intermediate (rather than low) T1 signal. In all likelihood, the high T1 signal intensity in our patient derived from averaging of the septae within the lesion with the low T1 signal from CSF (Fig. 1C).

The diffusion characteristics of Meckel’s cave arachnoid cysts have not been reported previously, although they may be expected to be helpful. Arachnoid cysts, like the lesion shown in our patient, demonstrate a CSF-like signal, a clear distinction from epidermoid cysts, which also arise in this region and which demonstrate a diffusion signal of brain parenchymal intensity (11,12). In our patient, the initial imaging diagnosis was schwannoma or meningioma, although such a high T2 signal would not be consistent with either diagnosis. Lack of familiarity with this lesion, which has been infrequently reported, probably led to the error. Larger lesions have disclosed bone erosion (Fig. 4A), which was found on high-resolution temporal bone CT in all of the 10 patients in whom such a study was performed (3–10). Our patient lacked these findings, perhaps because the lesion was short-lived and relatively small (Fig. 4B).

The clinical features of similar cystic lesions arising from Meckel’s cave have been adequately documented in 21 cases (Table 1), including 15 women and 6 men with a mean age of 46 years (range 1.5–82 years). Seven of these 21 patients were asymptomatic (3,4,7–9), with brain imaging having been performed for symptoms unrelated to the lesion. Among the 14 symptomatic patients, 10 had manifestations related to cyst mass effect on adjacent structures (1–3,6–10) and 4 had manifestations related to a CSF fistula (3,5,7,13). Cyst mass effect was responsible for binocular diplopia in four patients [1 with third cranial nerve palsy (3), 2 with sixth cranial nerve palsy (1,3), and 1
<table>
<thead>
<tr>
<th>Reference and case no.</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Ocular motor manifestation</th>
<th>Fifth cranial nerve involvement</th>
<th>Effects of CSF fistula</th>
<th>Other manifestations</th>
<th>Postoperative outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moore et al (3) (Case 1)</td>
<td>25</td>
<td>F</td>
<td>Sixth cranial nerve palsy</td>
<td>Numbness</td>
<td></td>
<td></td>
<td>Diplopia improved, but not trigeminal numbness</td>
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<tr>
<td>Beck et al (1) (Case 12)</td>
<td>1.5</td>
<td>M</td>
<td>Sixth cranial nerve palsy</td>
<td></td>
<td></td>
<td>Ipsilateral exophthalmos</td>
<td>Normal examination 6 years later</td>
</tr>
<tr>
<td>Moore et al (3) (Case 3)</td>
<td>48</td>
<td>F</td>
<td>Third cranial nerve palsy</td>
<td>Present</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Woerner et al (2)</td>
<td>44</td>
<td>M</td>
<td>Transient diplopia</td>
<td>Dysesthesia</td>
<td></td>
<td>Transient vertigo</td>
<td>Lesion recurred 3 months after surgery; asymptomatic 15 months after second surgery. Trigeminal pain disappeared almost immediately; total right conductive hearing loss developed; headache and facial paresthesias improved but persisted; no lesion present on MRI 1 year after surgery</td>
</tr>
<tr>
<td>Achilli et al (6)</td>
<td>40</td>
<td>F</td>
<td>Pain, dysesthesia, and numbness (V1, V2)</td>
<td>Dizziness without vestibular dysfunction</td>
<td></td>
<td></td>
<td>Trigeminal anesthesia (V3) persisted, but dysesthesia subsided</td>
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<tr>
<td>Moore et al (3) (Case 4)</td>
<td>59</td>
<td>F</td>
<td>Pain (V1), dysesthesia, and numbness (V1, V3)</td>
<td></td>
<td></td>
<td></td>
<td>Carbamazepine eventually failed to control facial pain preoperatively; postoperatively, pain controlled with low-dose carbamazepine during 6-month follow-up</td>
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<tr>
<td>Jelsma et al (10)</td>
<td>58</td>
<td>F</td>
<td>Pain (V1), dysesthesia, and numbness (V1, V3)</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Cheung et al (7) (Case 1)</td>
<td>46</td>
<td>F</td>
<td>Pain (V1, V2)</td>
<td></td>
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<td></td>
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<tr>
<td>Chang et al (8) (Case 12)</td>
<td>9</td>
<td>M</td>
<td>Numbness (V1, V2, V3)</td>
<td>Headache</td>
<td></td>
<td></td>
<td>Trigeminal dysesthesia resolved after 6 weeks</td>
</tr>
<tr>
<td>Batra et al (9)</td>
<td>55</td>
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<td>Pain (V3), dysesthesia (V3), and numbness</td>
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<td>Moore et al (3) (Case 2)</td>
<td>5</td>
<td>M</td>
<td>Pain (V3), dysesthesia (V3), and numbness</td>
<td>Otorrhea into the middle ear</td>
<td></td>
<td></td>
<td>CSF otorrhea resolved</td>
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<tr>
<td>Motojima et al (5)</td>
<td>6</td>
<td>F</td>
<td></td>
<td>Rhinorrhea and recurrent meningitis</td>
<td></td>
<td></td>
<td>Headache, vomiting, and rhinorrhea resolved</td>
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<tr>
<td>Hall et al (13)</td>
<td>33</td>
<td>M</td>
<td></td>
<td>Otorrhea</td>
<td></td>
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<tr>
<td>Chang et al (8) (Case 13)</td>
<td>70</td>
<td>F</td>
<td></td>
<td>Chronic suppurative otitis media</td>
<td></td>
<td></td>
<td>CSF otorrhea resolved</td>
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<tr>
<td>Moore et al (3) (Case 5)</td>
<td>45</td>
<td>F</td>
<td></td>
<td></td>
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<tr>
<td>Moore et al (3) (Case 6)</td>
<td>82</td>
<td>F</td>
<td></td>
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<tr>
<td>Moore et al (3) (Case 7)</td>
<td>46</td>
<td>F</td>
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<tr>
<td>Moore et al (3) (Case 8)</td>
<td>72</td>
<td>F</td>
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<tr>
<td>Moore et al (3) (Case 9)</td>
<td>66</td>
<td>F</td>
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<tr>
<td>Moore et al (3) (Case 10)</td>
<td>36</td>
<td>M</td>
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<tr>
<td>Srinivasan et al (4)</td>
<td>65</td>
<td>F</td>
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CSF, cerebrospinal fluid; M, male; F, female.
with transient diplopia without any further details (2)], fifth cranial nerve dysfunction in 9 patients (2,3,6–10), exophthalmos in 1 patient (1), and vertigo in 2 patients (2,6)). The lesion triggered a CSF fistula in four patients (3,5,7,13) when a tear occurred in the cyst wall, allowing CSF to leak into the petrous apex. Because the aerated petrous bone communicates with the middle ear, CSF otorrhea (2 patients) (3,13) or rhinorrhea (1 patient) (5) resulted. CSF fistula was also responsible for recurrent meningitis (1 patient) (5) and chronic otitis media (1 patient) (8). Five patients had headache (3,8), but whether it was caused by that particular lesion is uncertain.

Surgery was performed on all symptomatic patients (except 2, for whom treatment was not described (3,8) and on none of the asymptomatic patients. The surgical procedure was designed to collapse the cyst or to cure the CSF fistula or both. Diplopia improved after surgery in 1 patient (3) (Table 1) and had disappeared in 2 patients after 15 months (2) and after 6 years in another patient (1). For the remaining patient with diplopia, the outcome was not given (3). When the fifth cranial nerve was involved, the pain seemed to resolve postoperatively more often than the numbness (3,6,7,9,10). When described, the pain disappeared shortly after surgery. CSF fistulas finally resolved in all patients for whom an outcome was reported (3,5,13). In none of the seven asymptomatic patients was a follow-up examination reported, so the natural course of the lesion remains unknown. To our knowledge, ours is the first reported instance of the spontaneous resolution of a symptomatic Meckel’s cave arachnoid cyst.

Frequently found on imaging as an incidental finding, arachnoid cysts at any intracranial location may require surgical treatment when they enlarge and become symptomatic. On the basis of endoscopic observation of a suprasellar prepontine arachnoid cyst (14,15), a ball-valve mechanism is suspected to be the basis of enlargement. An alternative hypothesis is that cells lining the cyst wall continuously secrete fluid, which is then trapped (16).

Spontaneous disappearance of arachnoid cysts, as occurred in our patient, is rare in adults, with only one reported case in a 21-year-old patient (17). There are 13 reported cases in patients aged 16 years or younger (18–30). A provocative factor such as head trauma (20,29), meningitis (28), the Valsalva maneuver (31), coughing, sneezing, crying, or sporting activities (24,26,27,32) has sometimes been identified. Arachnoid cysts may progressively shrink without provocation or symptoms, a phenomenon observed in 9 patients (17–25) in whom the putative explanation is formation of a communication between the cyst and the subarachnoid space, allowing the cyst to drain through the normal CSF pathways. Whether there is direct transport through the cyst wall or whether CSF is released through a ball-valve mechanism is uncertain (24).

An alternative explanation for cyst disappearance, described in 5 patients (26–30), is sudden rupture into the subdural space, allowing fluid to spread and subsequently be reabsorbed. A subdural hematoma may sometimes initially accompany the subdural hygroma. In such cases, a tear in the cyst outer wall establishes a communication between the cyst and the subdural space (24,26,29). Supporting this hypothesis is the observation during craniotomy that a Valsalva maneuver caused a tear in the outer cyst wall (31). In our patient, the spontaneous resolution of the lesion occurred without any visible subdural effusion, even though the small size of the initial arachnoid cyst would have made it difficult to observe. The clinical improvement was concomitant with both the delivery and the lesion disappearance on MRI, suggesting the possibility that increased intra-abdominal or thoracic pressure during delivery may have been a triggering factor.

The discovery of a Meckel’s cave arachnoid cyst during pregnancy based on new symptoms raises the question of growth induced by the pregnancy itself (33–36). Three cases of intracranial arachnoid cyst have reportedly been diagnosed during pregnancy or the peripartum period because they had become symptomatic during those periods (33,34,36). The 2 patients who had become symptomatic during pregnancy (right hand tremor associated with headache in one and seizure in the other) underwent cesarean sections under general anesthesia to prevent the pushing efforts in delivery and to avoid increases in intracranial pressure by injection of anesthetic drugs into the epidural spaces (33,34). In the third case (36), the mother had a seizure 9 hours after delivering twins, and brain CT revealed a large medial fossa arachnoid cyst without any sign of rupture or hemorrhage into the subdural space. A fourth patient (35), known before pregnancy to have a posterior fossa arachnoid cyst, underwent cesarean section under epidural anesthesia and showed no change in cyst appearance in the peripartum period. An immunocytochemical study has shown progesterone receptors in the nuclei of cells lining the arachnoid cyst, suggesting an inhibitory influence of progestins on CSF absorption and evoking a similarity with hormone-dependent growth of meningiomas (37). We found no reported cases of Meckel’s cave arachnoid cysts discovered during pregnancy.

This case report describes a Meckel’s cave arachnoid cyst responsible for a sixth cranial nerve palsy during pregnancy and which spontaneously resolved after delivery. Because all of the previously described symptomatic patients had been treated surgically, this is the first reported instance of spontaneous resolution of both clinical and imaging findings. The imaging characteristics may vary
more than previously thought, including an intermediate signal on T1 and the absence of bone erosion in the petrous apex on CT. Pregnancy may have had a role in increasing the cyst size, and the pushing effort during labor may have triggered cyst rupture. Given the experience with this patient, arachnoid cysts should not be surgically treated during pregnancy unless they produce life-threatening manifestations.

REFERENCES