Increasing use of diagnostic intracranial imaging has led to more frequent diagnosis of arachnoid cysts. Although arachnoid cysts are occasionally symptomatic, most agree that a majority of arachnoid cysts are found incidentally. There are reports of cysts arising de novo as well as cysts becoming larger or smaller over time, and even spontaneously resolving. We recently reported on the natural history of these lesions in children and found a generally benign natural history.

Many surgeons have suggested that clearly symptomatic arachnoid cysts should be treated. In some cases, previously asymptomatic arachnoid cysts may rupture, causing a symptomatic hygroma in the subdural space. Most prior reports have advocated surgical treatment for arachnoid cyst–associated subdural hygromas. We have observed that patients with a symptomatic subdural hygroma associated with an arachnoid cyst will frequently experience complete and lasting spontaneous resolution of their neurological symptoms if managed without surgery. We report our recent experience with managing arachnoid cyst–associated subdural hygromas without surgery and suggest that the often benign natural history of this condition should be considered when making treatment decisions.
Subdural hygromas associated with cysts

3-T imaging unit. All patients who had a cyst-associated hygroma during this interval were included. During this time period, 1 patient with an associated subdural hematoma was excluded. We found patients who presented with arachnoid cysts and hygromas that were managed without surgery at the University of Michigan (Cases 1–6). To these we added a single case that was managed without surgery at Nationwide Children’s Hospital (Case 7). Finally, we found 1 patient with an arachnoid cyst–associated hygroma who was surgically treated at our institution during this time period (Case 8). For each of these patients, we collected information including age, sex, presenting symptoms, trauma history, surgical history, and clinical course. The images were examined in every case to confirm the diagnosis of arachnoid cyst and subdural hygroma as well as to assess for any changes over time. Patients with subdural hematomas were not included in this analysis.

**Summary of Cases**

**Case 1**

A 10-year-old boy presented 3 weeks after a minor head injury. He experienced mild headaches from the time of injury, but at 3 weeks after injury, he began to experience worsening headaches. Computed tomography scanning showed a large left middle fossa arachnoid cyst (Fig. 1). Findings during the neurological examination were normal. After a discussion with the child and his parents, we decided to manage the hygroma without surgery. In the days after this decision, the patient’s symptoms initially worsened. He began to have nausea and vomiting and developed diplopia. He was seen by an ophthalmologist who documented bilateral papilledema and a left cranial nerve (CN) VI palsy. A new MR image showed that the subdural collection had expanded. Initial plans were made for surgical treatment within several days, but the diplopia markedly improved within 2 days, and plans for surgery were cancelled. Two weeks later, all symptoms including the diplopia had completely resolved and the papilledema had improved. Magnetic resonance imaging confirmed resolution of the subdural hygroma and persistence of the arachnoid cyst.

**Case 2**

A 12-year-old girl presented to the emergency department 2.5 weeks after a minor head injury that occurred during a soccer game. She had immediate onset of a headache after the injury. Her headache progressively worsened over the next several days, and she began to experience nausea and vomiting, but she had no visual complaints. A right middle fossa arachnoid cyst with associated hygroma was found on MRI. Four weeks later, her headaches were much better and the hygroma was smaller. Three months after the injury, the headaches had resolved and the hygroma had disappeared. The arachnoid cyst appeared unchanged.

**Case 3**

A 16-year-old boy began to experience headaches 1 day after sustaining a minor head injury during a football game. The headaches lasted for approximately 2 weeks and then resolved. At the time of his presentation to the neurosurgery clinic, he was asymptomatic and had normal findings on examination. Magnetic resonance imaging demonstrated a right middle fossa arachnoid cyst and bilateral subdural hygromas (Fig. 2). No treatment was recommended. Follow-up imaging performed 3 months after the injury showed a decrease in the size of the hygroma and the arachnoid cyst. Further decrease in size was noted at the 1-year follow-up.
Case 4

An 8-year-old boy presented to his primary care physician with a gradually worsening headache 2 weeks after a minor head injury. He had vomited once several days earlier but had no other complaints. His neurological examination was normal. Three weeks later, he presented to the emergency department with a headache and transient subjective diplopia that resolved within 20 minutes. Findings during neurological examination remained normal. Brain MRI demonstrated a right middle cranial fossa arachnoid cyst and a thin subdural hygroma over the cerebral convexities bilaterally. During the following week, the patient had 4 episodes of vomiting and complained of a headache on several occasions. Repeat MRI was performed 3 weeks later because of persistent headaches and showed a subtle interval increase in the hygroma with interval increase in the mass effect on the right cerebral hemisphere. Two months later the patient was asymptomatic, and MRI showed slight reduction in size of the hygroma. The hygroma had completely resolved on MRI performed 7 months later. The arachnoid cyst was unchanged.

Case 5

A 12-month-old girl was found to have a rapid increase in head circumference on routine screening. Head CT showed a left middle fossa arachnoid cyst and an associated subdural hygroma with mass effect on the ipsilateral hemisphere. Of note, the patient had an unwitnessed fall from her crib 1 month earlier. The patient was neurologically normal after the fall, and the parents did not seek medical care. Since the patient was asymptomatic, surgical treatment was deferred. Magnetic resonance images obtained 1 month, 4 months, and 18 months later confirmed gradual resolution of the hygroma, although the cyst remained unchanged. The rate of head growth slowed, and the child remained neurologically and developmentally normal.

Case 6

A 13-month-old boy presented with a 6-week history of vomiting and a rapid increase in head circumference. Cranial ultrasonography demonstrated a left middle fossa arachnoid cyst and bilateral convexity subdural fluid collections; MRI confirmed these findings. The subdural collections were asymmetric, and minimal sulcal effacement was noted on the side ipsilateral to the larger collection. One month later, the patient was asymptomatic. A follow-up imaging study performed 3 months later showed that the bilateral subdural spaces were much smaller. Ten months after the initial presentation, follow-up imaging showed a further decrease in size of the subdural fluid spaces and a small decrease in the size of the arachnoid cyst. The patient is now neurologically and developmentally normal 12 months after the initial evaluation.

Case 7

A 10-year-old boy sustained a minor head injury without loss of consciousness. He developed headache with photophobia, nausea, and vomiting 1 day after the injury, and these symptoms gradually worsened. Computed tomography scanning performed 2 days after the injury demonstrated a right middle fossa arachnoid cyst. One week later, MRI demonstrated bilateral subdural hygromas in addition to the arachnoid cyst. At the time of his first neurosurgical evaluation 4 weeks after injury, the patient’s symptoms had completely resolved. Repeat brain MRI performed at that time demonstrated a decrease in the subdural hygroma on the left, but a significant increase on the right (Fig. 3). As he was clinically well, the patient was managed without surgery. He remained neurologically intact over a 12-month follow-up period. Seven months after his first visit, MRI demonstrated a decrease in size of both the hygroma and the arachnoid cyst. Three months later, a further decrease in size of both the hygroma and cyst was again noted.
Subdural hygromas associated with cysts

Case 8

A 7-year-old boy presented with a 4-week history of headaches but without other symptoms. There was no known history of trauma. Magnetic resonance imaging showed a right middle fossa arachnoid cyst with an associated subdural hygroma. An ophthalmologist found bilateral papilledema. The patient was treated with a craniotomy for cyst fenestration. Postoperatively, the headaches completely resolved. However, routine follow-up MRI performed 6 months postoperatively demonstrated asymptomatic but significant enlargement of the cyst, and a cystoperitoneal shunt was placed. After this, the hygroma resolved and the cyst became progressively smaller on subsequent follow-up scans at 6-month, 1-year, and 2-year intervals following shunt placement. The patient remains asymptomatic 5 years postoperatively.

Discussion

Posttraumatic subdural hygromas are thought to arise from a tear in the arachnoid membrane resulting in a one-way valve mechanism that leads to the accumulation of CSF under abnormally elevated pressure. This theory, first articulated in 1924 by Naffziger, has remained the best explanation for the phenomenon of the symptomatic hygroma. In some cases, subdural hygromas are found in patients with arachnoid cysts. Cyst-associated hygromas may form when the outer cyst membrane tears, either spontaneously or as a result of trauma or surgical manipulation. Some have speculated that a change in local intracranial pressure (ICP) as a result of a Valsalva maneuver may also lead to cyst rupture with hygroma, although unrecognized or unrecorded trauma may also explain some of these cases. A tear in the cyst lining allowing communication between an arachnoid cyst and an associated subdural hygroma has been documented intraoperatively on at least 4 occasions.

Hygromas undoubtedly result in elevated ICP in many cases. Evidence for this is found in the common symptoms that are seen with hygromas, as well as the physical findings that are sometimes seen, such as papilledema and even transient CN VI palsy. The pattern of presentation is remarkably consistent for these patients (Tables 1 and 2). Symptoms usually include headache, nausea, vomiting, and rarely diplopia from CN VI palsy. Physical findings are usually limited to papilledema. Most reported cases have a history of minor trauma without loss of consciousness. Symptoms usually become increasingly severe for days or weeks after onset, but they eventually resolve.

Some authors have suggested that surgical treatment of subdural hygromas associated with arachnoid cysts is always or almost always necessary, with some going so far as to call nonsurgical management inappropriate. Others have even suggested that hygromas, since they have symptoms of elevated ICP, should be considered potentially life threatening. Although this is no doubt theoretically or even actually possible, we have never encountered a report of a fatality or permanent neurological deficit resulting from nonsurgical management of an arachnoid cyst–associated hygroma. The case series described above identifies the possibility of a benign natural history for arachnoid cyst–associated subdural hygroma. Every patient in this series who was managed nonoperatively experienced a complete symptomatic resolution. Therefore, we suggest that the decision to surgically treat symptomatic hygromas associated with arachnoid cysts should be made carefully and only after the generally benign natural history of this condition is considered. Half of the patients in this series with cyst-associated hygroma had objective findings strongly correlated with elevated ICP including CN VI palsy, papilledema, and progressive macrocephaly, yet all these patients had spontaneous resolution of these physical findings; therefore, symptoms of elevated ICP are not an absolute indication for surgical treatment in our opinion.
headaches and vomiting are added to the list of ICP-related symptoms, then in 7 of our cases, symptoms of elevated ICP resolved without treatment. Furthermore, the one patient who was treated surgically had a very large arachnoid cyst; this was not typical of our recent experience. Because of what appears to be a high potential for spontaneous resolution, in patients presenting with new-onset symptoms of elevated ICP and an arachnoid cyst, the possible presence of an associated hygroma should be carefully investigated before pursuing any surgical cyst treatment. If a hygroma is found in a patient with new-onset symptoms, we believe that this should suggest that an initial decision to manage without surgery is likely to result in a good outcome with symptom resolution in many cases. Close clinical follow-up is required for symptomatic patients who are managed without surgery. We would still prefer surgical treatment as an initial therapy for patients with severe symptoms or an especially concerning neurological examination. Furthermore, surgical treatment is indicated for those patients whose condition is refractory to initial conservative management. In our practice, we would generally attempt to treat with surgical fenestration and would reserve shunt placement for those with treatment failures after fenestration.

The suggestion that arachnoid cysts should be treated if they demonstrate evidence of local mass effect on imaging is potentially problematic in the context of subdural hygroma. Any moderately sized hygroma will have the appearance of mass effect on imaging. The appearance of local mass effect, therefore, is too inclusive to be used as a reliable criterion for selecting patients who require treatment. Furthermore, most asymptomatic arachnoid cysts even in the absence of hygroma will produce the appearance of local mass effect on adjacent tissues.

Including all reported cases of arachnoid cyst with associated subdural hygroma in which the treatment and outcome has been specified, there are 28 reported cases in which this treatment was tried, 2 patients had symptom resolution without surgery, and 1 patient went on to have surgical decompression of the hygroma. In addition, Tamburrini et al. reported on 2 patients who had a short trial of acetazolamide for symptomatic postoperative hygromas after arachnoid cyst fenestration, but this treatment did not improve symptoms in either case. Given the tendency for symptoms to resolve over time in many cases, it is impossible to make any claims about the efficacy of acetazolamide treatment for this condition based on the existing literature.

Hygroma resolution with the return of prior arach-
TABLE 2: Reported cases of arachnoid cysts associated with subdural hygroma treated with surgery*

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Age (yrs), Sex</th>
<th>Cyst Location</th>
<th>Trauma History</th>
<th>Injury to Presentation</th>
<th>Time Interval</th>
<th>Nausea &amp;/or Vomiting</th>
<th>Papilledema</th>
<th>Diplopia</th>
<th>Treatment</th>
<th>Clinical Outcome</th>
<th>Cyst Imaging Outcome</th>
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<tbody>
<tr>
<td>Albuquerque &amp; Giannotta, 1997</td>
<td>6, M</td>
<td>lt MF, QC</td>
<td>yes (minor)</td>
<td>1 day</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>unk</td>
<td>no</td>
<td>CCF, SD drain</td>
<td>good (w/ SD shunt)</td>
</tr>
<tr>
<td></td>
<td>25, M</td>
<td>bilat MF</td>
<td>yes (minor)</td>
<td>1 day</td>
<td>no</td>
<td>unk</td>
<td>unk</td>
<td>unk</td>
<td>unk</td>
<td>SD drain</td>
<td>good</td>
</tr>
<tr>
<td></td>
<td>9, unk</td>
<td>MF</td>
<td>yes</td>
<td>unk</td>
<td>no</td>
<td>yes</td>
<td>unk</td>
<td>yes</td>
<td>unk</td>
<td>SD drain</td>
<td>good</td>
</tr>
<tr>
<td></td>
<td>25, unk</td>
<td>MF</td>
<td>yes</td>
<td>unk</td>
<td>no</td>
<td>yes</td>
<td>unk</td>
<td>yes</td>
<td>unk</td>
<td>SD shunt</td>
<td>good</td>
</tr>
<tr>
<td></td>
<td>10, unk</td>
<td>lt MF</td>
<td>no</td>
<td>unk</td>
<td>no</td>
<td>yes</td>
<td>unk</td>
<td>yes</td>
<td>unk</td>
<td>SD shunt</td>
<td>good</td>
</tr>
<tr>
<td>Bristol et al., 2007</td>
<td>17, M</td>
<td>rt MF</td>
<td>yes (brief LOC)</td>
<td>3 days</td>
<td>no</td>
<td>yes</td>
<td>no</td>
<td>unk</td>
<td>unk</td>
<td>CCF</td>
<td>good</td>
</tr>
<tr>
<td>Cakir et al., 2004</td>
<td>9, M</td>
<td>rt MF</td>
<td>no</td>
<td>unk</td>
<td>no</td>
<td>yes</td>
<td>no</td>
<td>no</td>
<td>bur hole &amp; drain</td>
<td>good</td>
<td>improved</td>
</tr>
<tr>
<td>Cayli, 2000</td>
<td>12, F</td>
<td>lt MF</td>
<td>no</td>
<td>3 wks</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>no</td>
<td>unk</td>
<td>unk</td>
<td>unk</td>
</tr>
<tr>
<td>Cakir et al., 2004</td>
<td>11, M</td>
<td>lt MF</td>
<td>no (swimming)</td>
<td>1 mo</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>no</td>
<td>unk</td>
<td>CCF</td>
<td>unk</td>
</tr>
<tr>
<td>Dharmarajan et al., 1988</td>
<td>14, M</td>
<td>lt MF</td>
<td>yes (minor)</td>
<td>4 wks</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>no</td>
<td>unk</td>
<td>CCF</td>
<td>good</td>
</tr>
<tr>
<td></td>
<td>5, M</td>
<td>rt MF</td>
<td>yes (minor)</td>
<td>10 days</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>no</td>
<td>unk</td>
<td>CCF</td>
<td>2nd craniotomy 2 mos later; good</td>
</tr>
<tr>
<td>Ergün et al., 1997</td>
<td>14, M</td>
<td>rt MF</td>
<td>no</td>
<td>20 days</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>no</td>
<td>no</td>
<td>CCF</td>
<td>good</td>
</tr>
<tr>
<td>Gelabert-González et al., 2002</td>
<td>13, M</td>
<td>lt MF</td>
<td>yes (minor)</td>
<td>4 wks</td>
<td>no</td>
<td>yes</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>CCF</td>
<td>good</td>
</tr>
<tr>
<td></td>
<td>12, M</td>
<td>lt MF</td>
<td>yes (minor)</td>
<td>unk</td>
<td>yes</td>
<td>yes</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>CCF</td>
<td>good</td>
</tr>
<tr>
<td></td>
<td>6, M</td>
<td>lt MF</td>
<td>yes (minor)</td>
<td>1 mo</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>unk</td>
<td>uns</td>
<td>SD drain</td>
<td>req’d SD shunt, then OK</td>
</tr>
<tr>
<td>Goswami et al., 2008</td>
<td>8, M</td>
<td>rt MF</td>
<td>yes (minor)</td>
<td>1 wk</td>
<td>no</td>
<td>yes</td>
<td>no</td>
<td>unk</td>
<td>no</td>
<td>CCF</td>
<td>good</td>
</tr>
<tr>
<td>Gupta et al., 2004</td>
<td>22, M</td>
<td>lt MF</td>
<td>yes (minor)</td>
<td>3 days</td>
<td>unk</td>
<td>yes</td>
<td>yes</td>
<td>unk</td>
<td>no</td>
<td>acetazolamide</td>
<td>repeat drainage; unk</td>
</tr>
<tr>
<td>Herman &amp; Siegel, 2010</td>
<td>0, unk</td>
<td>retrocerebellar</td>
<td>unk</td>
<td>unk</td>
<td>no</td>
<td>unk</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>SD shunt</td>
<td>good</td>
</tr>
<tr>
<td>Klein et al., 2006</td>
<td>14, M</td>
<td>rt MF</td>
<td>yes (minor)</td>
<td>2 wks</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>unk</td>
<td>yes</td>
<td>SD drains</td>
<td>new CN palsy &amp; SD shunt req’d</td>
</tr>
<tr>
<td>Kulali &amp; von Wild, 1989</td>
<td>15, M</td>
<td>rt MF</td>
<td>no (dancing)</td>
<td>3 wks</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>no</td>
<td>CCF</td>
<td>good</td>
</tr>
<tr>
<td></td>
<td>6, M</td>
<td>rt MF</td>
<td>yes (minor)</td>
<td>4 wks</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>unk</td>
<td>no</td>
<td>CCF</td>
<td>good</td>
</tr>
<tr>
<td>Longatti et al., 2005</td>
<td>7, M</td>
<td>rt MF</td>
<td>yes</td>
<td>1 mo</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>unk</td>
<td>steroid &amp; acetazolamide</td>
<td>good</td>
</tr>
<tr>
<td></td>
<td>present series (Case 8)</td>
<td>7, M</td>
<td>rt MF</td>
<td>no</td>
<td>4 wks</td>
<td>no</td>
<td>yes</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>CCF</td>
</tr>
<tr>
<td>Offiah et al., 2006</td>
<td>8, M</td>
<td>bilat MF</td>
<td>yes (minor)</td>
<td>? wks</td>
<td>no</td>
<td>yes</td>
<td>no</td>
<td>no</td>
<td>bur holes</td>
<td>recurrent symptoms, pseudomeningocele, SD shunt</td>
<td>enlarged</td>
</tr>
</tbody>
</table>

(continued)
noid cyst dimensions may result from healing of the tear in the outer arachnoid cyst lining. Persistent tears in the lining might lead to cyst resolution in rare cases. Supporting this theory, the development of a subdural hygroma has occasionally been associated with concurrent “spontaneous” cyst size reduction or resolution. Including our own 3 cases (Cases 3, 6, and 7), this phenomenon has now been reported 6 times. Furthermore, in a recent review of all reported cases of documented “spontaneous” arachnoid cyst resolution, Thomas et al. found that 6 of 19 cases had a recent history of head injury, suggesting that an unrecognized tear in the cyst lining may explain at least some of these cases. Spontaneous cyst resolution has also been documented in patients with no history of trauma or subdural hygroma, although it is difficult to exclude minor trauma in this group of children. We may also speculate that rupture not only of the outer cyst lining in the subdural space, but also of the inner lining adjacent to the basal cisterns, may be especially likely to lead to cyst resolution. In numerous reports, surgeons have encountered fluid under pressure while treating these lesions surgically. These pressure elevations sometimes persist for long durations postoperatively, occasionally resulting in permanent shunt placement, even if that was not the original surgical plan. The new onset of subdural hygroma is a recognized side effect of arachnoid cyst fenestration. In reported series, surgical fenestration of arachnoid cysts has resulted in iatrogenic postoperative subdural hygroma in approximately 4% to 29% of cases. The incidence of asymptomatic subdural collections following surgical fenestration may be even higher. As with idiopathic or posttraumatic hygromas, these postoperative hygromas may cause symptoms of elevated ICP leading Maixner et al. to term this phenomenon “pseudotumor syndrome following surgery for arachnoid cysts.” The higher than expected rate of postoperative pseudomeningoceles and CSF leaks after fenestration probably results from a transient ICP elevation after cyst fenestration. The tendency for postoperative subdural hygromas to become symptomatic days or weeks after the initial cyst fenestration procedure may be analogous to the posttraumatic variety presenting days or weeks after the traumatic event. In many previously reported cases, the development of a new symptomatic hygroma after cyst fenestration was treated with further surgery including permanent shunting of the subdural space.

If the presenting symptoms and imaging appearance are similar between idiopathic, posttraumatic, and postsurgical cyst-associated hygromas, it is possible that postoperative cyst-associated hygromas are likely to share a common natural history as well. In our own practice, we have treated 2 patients for arachnoid cysts in the last 6 years who have developed new symptomatic subdural hygromas after cyst fenestration. In both cases, the patients initially did well after surgery and were dismissed from the hospital in good condition. They then returned with symptoms of elevated ICP including persistent nausea and vomiting in the weeks after surgery. In both cases, we may also speculate that rupture not only of the outer cyst lining in the subdural space, but also of the inner lining adjacent to the basal cisterns, may be especially likely to lead to cyst resolution.
Subdural hygromas associated with cysts

no further treatment other than an admission for intravenous fluid resuscitation was needed, and the symptoms and scans improved over the course of the following month. Based on our own experience with postoperative hygromas, as well as the often benign natural course of idiopathic and posttraumatic cyst-associated hygromas, it has become our own practice to initially recommend no further surgical treatment for postoperative subdural hygromas after cyst fenestration, even when they are symptomatic. As with spontaneous or posttraumatic hygromas, we only recommend surgical treatment in cases that are refractory to conservative measures.

The prevalence of hygroma in patients with arachnoid cysts is not known and, due to detection bias, this case series of patients who presented with neurological symptoms is not able to shed light on that issue. Patients with a subdural hygroma are more likely to be symptomatic and, therefore, more likely to come to medical attention. In prior reports, we analyzed all patients undergoing MRI rather than those who presented for neurosurgical attention.1,2 We would expect that analysis of a general imaging database would have less detection bias for subdural hygroma compared with a clinical case series. In our prior analysis of 309 arachnoid cysts in children who were identified using an imaging database that included consecutive patients who underwent MRI of the brain for any reason at our institution between January 1997 and June 2008, we found only 2 children who presented with a hygroma.2 In a subsequent analysis of 661 adult patients with arachnoid cysts using our imaging database, we found 1 symptomatic patient and 1 asymptomatic patient who presented with both hygroma and an arachnoid cyst on initial imaging.1 Furthermore, in prior reports, we observed 111 children with arachnoid cysts for a mean duration of 3.5 years and 213 adults for a mean duration of 3.8 years and found no patient in those groups who developed hygroma during the follow-up intervals.1,2 We conclude that hygroma appears to be a rare complication of an untreated arachnoid cyst, but the exact prevalence cannot be determined based on existing data.

There are several limitations to our report. We did not include patients with arachnoid cysts and associated subdural hematomas, and we do not believe that this case series should be applied to that very different patient population. In our review of the literature, it is possible that some patients in prior reports who were diagnosed as having a cyst with associated hematoma, in fact, had a hygroma. We made an effort to exclude patients with subdural hemorrhage rather than hygroma.4,5,6,7 This distinction is sometimes difficult when interpreting early reports, since there may still be some confusion regarding the classification of subdural collections.7 There are intriguing reports from the era preceding modern CT and MRI that describe patients with delayed onset of headaches and nausea days after a minor head injury who were later discovered to harbor arachnoid cysts. Unfortunately, the anatomical description is not sufficient to determine if hygromas were present as a determining factor in these cases.4,5,6 Similarly, very early reports of arachnoid cysts occasionally considered the cyst itself as a “hygroma in the subdural space.”10,14,16,32,64 Finally, our own case se-

Conclusions

Subdural hygroma may lead to symptomatic presentation for otherwise asymptomatic arachnoid cysts. The natural course of cyst-associated subdural hygromas, even when symptomatic, is generally benign, and symptom resolution can be expected in most cases. We suggest that symptomatic hygroma is not an absolute indication for surgical treatment and that expectant management can result in good outcomes in many cases.

Acknowledgment

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Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: Maher, Jackson. Acquisition of data: Maher, Jackson, Al-Holou, Garton, Trobe. Analysis and interpretation: Maher, Jackson, Garton. Drafting the article: Maher, Jackson, Garton, Al-Holou, Trobe, Muraszko. Critically revising: all authors. Reviewed submitted: all authors. Approved final version: Maher. Study supervision: Maher.

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