Updated assessment and current classification of spinal meningeal cysts

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The classification of spinal meningeal cysts (MC's) in the literature is indistinct, confusing, and in certain categories histologically misleading. Based on a series of 22 cases, the authors propose a classification comprising three categories: spinal extradural MC's without spinal nerve root fibers (Type I); spinal extradural MC's with spinal nerve root fibers (Type II); and spinal intradural MC's (Type III). Although water-soluble myelography may disclose a filling defect for all three categories, computerized tomographic myelography (CTM) is essential to reveal communication between the cyst and the subarachnoid space. Communication demonstrated by CTM allows accurate diagnosis of a spinal MC and rules out other mass lesions. Magnetic resonance imaging appears useful as an initial study to identify an intraspinal cystic mass. Final characterization is based on operative inspection and histological examination for all three categories.

KEY WORDS: spinal meningeal cyst, magnetic resonance imaging, myelography, computerized tomography

The classification of spinal meningeal cysts (MC's) in the literature is indistinct, confusing, and in certain categories histologically misleading. In reports on the thoracolumbar region, previous investigators have referred to extradural MC's as extradural "cysts," "pouches," or "diverticula." The classification of extradural MC's of the sacral region has been divided into "perineurial cysts," "meningeal diverticula," and "occult intrasacral meningoceles." Various other terms have been used to designate each of these categories. Some categories of extradural MC's were determined by the existence of communication with the subarachnoid space. Tarlov's "perineurial cysts" were distinguished from "spinal nerve root diverticula" because the "perineurial cyst" did not communicate with the subarachnoid space, whereas the "spinal nerve root diverticula" had free communication. Some authors have reported that delayed myelography with Pantopaque (iophendylate) occasionally revealed communication of "perineurial cysts" with the subarachnoid space. In our current experience, computerized tomographic myelography (CTM) usually demonstrates that both categories of cysts communicate with the subarachnoid space, and that they are actually the same lesion. Although extradural cysts are often termed "arachnoid cysts," the inner arachnoid lining with a single-cell layer is frequently absent on histological examination. Intradural MC's have also been designated "arachnoid cysts" or "arachnoid diverticula." Based on a series of 22 cases, of which all except three were collected within the past 2 years, we are proposing a concise and current approach to examine and categorize these lesions. We recommend magnetic resonance (MR) imaging to identify an intraspinal cystic mass, then CTM to demonstrate communication between the cyst and the subarachnoid space. By operative and histological examination, spinal MC's should be classified into three major categories (Table 1): extradural cysts without spinal nerve root fibers (Type I); extradural cysts with spinal nerve root fibers (Type II); and intradural cysts (Type III). Detailed case reports are presented to illustrate these three types.

Illustrative Cases

Table 2 summarizes the cases of Type I spinal MC's and Figs. 1 to 7 illustrate the abnormalities. Table 3...
Spinal meningeal cysts

TABLE 1
Classification of spinal meningeal cysts

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>extradural meningeal cysts without spinal nerve root fibers</td>
</tr>
<tr>
<td>IA</td>
<td>&quot;extradural meningeal cyst&quot; (&quot;extradural arachnoid cyst&quot;)</td>
</tr>
<tr>
<td>IB</td>
<td>&quot;sacral meningocele&quot; (&quot;occult sacral meningocele&quot;)</td>
</tr>
<tr>
<td>II</td>
<td>extradural meningeal cysts with spinal nerve root fibers</td>
</tr>
<tr>
<td></td>
<td>(&quot;Tarlov's perineurial cyst,&quot; &quot;spinal nerve root diverticulums&quot;)</td>
</tr>
<tr>
<td>III</td>
<td>spinal intradural meningeal cysts (&quot;intradural arachnoid cyst&quot;)</td>
</tr>
</tbody>
</table>

and Figs. 8 to 11 present the pathology in Type II spinal MC's, and Table 4 and Figs. 12 and 13 summarize Type III cases.

Case 1 (Type I)

This 14-year-old boy complained of difficulty in running. He also experienced lower-thoracic pain radiating to the right anterior area of the chest and bilateral leg numbness. He denied bowel or bladder problems. Physical examination revealed a T-11 sensory level, spastic paraparesis, brisk reflexes in the lower extremities, and a Babinski sign present on the right. Water-soluble myelography disclosed an extradural defect with an incomplete block from T-6 to T-8, and CTM demonstrated a posterior cyst communicating with the subarachnoid space (Fig. 1). A T5–8 laminectomy...
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FIG. 3. Case 1.  Left: Scanning electron photomicrograph of a Type I cyst wall demonstrating fibrocollagenous tissue. ×350.  Right: Another view demonstrating a thin single-cell layer of inner lining. ×350.

FIG. 4. Case 2. Magnetic resonance imaging in the sagittal plane of a Type I cyst disclosing an extradural meningeal cyst at T3–7 (arrow). The cyst has the same low signal intensity as the cerebrospinal fluid on this T1-weighted image.

Fig. 3. Case 1. Left: Scanning electron photomicrograph of a Type I cyst wall demonstrating fibrocollagenous tissue. × 350. Right: Another view demonstrating a thin single-cell layer of inner lining. × 350.

tomy disclosed a glistening cystic mass dorsal to the thecal sac. The cyst was incised, yielding cerebrospinal fluid (CSF). Because of dense fibrous adhesions, the posterior wall was excised. An ostium, which contained vessels from the subarachnoid space leading into the cyst wall, was identified at the T-6 level medial to the left nerve root. The ostium was closed with silk sutures. On light microscopy and scanning electron microscopy (SEM), the cyst wall showed fibrous connective tissue (Fig. 2 left and Fig. 3 left). Only on SEM was a single-cell epithelial layer identified (Fig. 3 right). Postoperatively, the patient’s spasticity gradually improved and his leg strength remained good. An epidural fluid collection present in the immediate postoperative period resolved spontaneously. Six months postoperatively, he was completely normal neurologically.

Case 3 (Type I)

This 18-year-old man complained of right-sided low-back pain radiating to his right flank and abdomen, occasionally simulating acute appendicitis. The patient was normal on neurological examination. Magnetic resonance imaging disclosed an extradural cystic mass which displaced the cord to the right at L1–2 (Fig. 5). Routine water-soluble myelography showed a left extradural defect at L1–2, and CTM revealed communication between the cyst and the subarachnoid space. A T10–L2 hemilaminectomy disclosed a white cystic mass dorsal to the dura. The cyst was totally removed (Fig. 6 upper). A pedicle was identified adjacent to the left L-1 nerve root and was ligated. Histopathological examination disclosed nonspecific fibrous connective tissue (Fig. 6 lower) with an area of calcification. Postoperatively, the patient’s pain resolved and his strength has remained good.

Case 7 (Type I)

This 36-year-old woman underwent a partial L-4 and total L-5 laminectomy for a ruptured lumbar disc in Mexico 2 years before her present admission. The op-
Spinal meningeal cysts

Fig. 5. Case 3. T₁-weighted magnetic resonance images in the coronal plane. An extradural cystic mass at L1–2 is indicated (arrow). The spinal cord is displaced to the right. The cyst’s low-intensity signal on T₁-weighted imaging is similar to that of cerebrospinal fluid.

Operative report indicated that the disc was not removed due to bleeding problems encountered at surgery. At her present admission, she complained of persistent low-back pain and dorsiflexion weakness of the left foot. She denied any bowel or bladder dysfunction. Physical examination revealed decreased sensation to light touch and pinprick on the left posterior calf and foot and 4/5 left dorsiflexion weakness. The deep-tendon reflexes were: 2+ and symmetrical for the biceps and triceps; 3+ for both knee jerks; 1+ for the right ankle jerk; and 0 for the left ankle jerk. Examination of the back disclosed an area of hypertrichosis in the lumbosacral region. Plain x-ray films revealed a widened sacral canal. Magnetic resonance imaging demonstrated a sacral meningeal cyst arising from the most caudal aspect of the dural sac (Fig. 7 right), in addition to a small bulging L4-5 disc on the left. Water-soluble myelography (Fig. 7 left) and CTM confirmed communication between the subarachnoid space and the

Fig. 6. Case 3. Upper: Gross pathology specimen of a Type I cyst. In situ, the cyst was cylindrical and measured 4.5 × 1 × 1 cm. Lower: Photomicrograph of the same cyst. A single-cell layer of inner arachnoid lining is not seen. H & E, × 75.

Fig. 7. Case 7. Water-soluble myelogram (left) and T₁-weighted magnetic resonance image in the sagittal plane (right) of a sacral Type I cyst (arrows).

“meningocele.” An L₅–S₄ laminectomy disclosed a bluish glistening cystic mass originating adjacent to a left sacral nerve root. The cyst was incised and an ostium was identified in direct communication with the subarachnoid space. No nerve roots were present within

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Case 10 (Type II)

This 53-year-old woman complained of a recent exacerbation of low-back pain radiating down the right leg posteriorly into the central three toes, which became worse on coughing and straining. She also had urinary urgency and incontinence. On physical examination, sensory testing disclosed diminished pinprick and light touch sensation for the entire right lower extremity and perianal region. Muscle strength and deep-tendon reflexes were normal. Rectal examination revealed decreased sensation and diminished tone. Plain x-ray films disclosed sacral canal widening. Routine water-soluble myelography showed a ruptured lumbosacral disc on the right side with no communication between the subarachnoid space and what was seen on MR imaging to be a large sacral extradural cyst. Communication with the subarachnoid space was revealed on CTM (Fig. 8). An L5-S2 laminectomy was performed and the ruptured lumbosacral disc on the right was excised. Multiple sacral nerve root-containing cysts were identified, with a particularly large cyst on the S-2 nerve root containing CSF. The cyst was opened and oversewn. The right S-2 nerve root was found in the center of the cyst cavity. Histological examination of the cyst wall disclosed fibrous collagenous tissue. Postoperatively, the patient's back pain and dorsiflexion weakness improved.
Spinal meningeal cysts

Case 11 (Type II)

This 47-year-old man complained of pain in the right leg radiating posteriorly to the ankle, with numbness in the posterior aspect of the right leg and in the sole of the right foot. He denied any weakness or bowel or bladder changes, but there was a history of sexual dysfunction and inability to ejaculate. His physical examination was normal. Magnetic resonance imaging disclosed a meningeal cyst on the right S-2 nerve root (Fig. 9 right). Routine water-soluble myelography (Fig. 9 left) and CTM (Fig. 10) confirmed a communicating sacral cyst on the right side. A total L5–S2 laminectomy was performed. The sacral cyst at the right S-2 nerve root was opened and found to contain CSF. Nerve root fibers were running within the ventromedial wall. After the cyst fluid was drained, no more fluid entered the cavity even when a Valsalva maneuver was induced. Passing a ball dissector cephalad within the cyst at its origin yielded no freely flowing CSF. Therefore, what was originally shown to be a communicating sacral cyst on preoperative diagnostic studies was not freely patent at surgical examination. The cyst wall was shrunk by bipolar cautery and a small piece of fat was placed within the ostium. The cyst wall was then oversewn. Postoperatively, the patient's leg pain was gone, although some numbness persisted on the sole of the right foot. His strength has remained good.

Case 22 (Type III)

This 61-year-old woman complained of low-back pain, numbness from the waist down, bilateral leg weakness, and urinary incontinence. Physical examination was remarkable for a L-7 sensory level to light touch and pinprick, no vibratory sense in the right leg, 4/5 muscle strength in all muscle groups in both legs, and decreased sphincter tone. Magnetic resonance imaging disclosed an extra-axial mass, with the same signal intensity as CSF on T1- and T2-weighted images, which flattened the spinal cord posteriorly at T-7 (Fig. 13). Routine water-soluble myelography disclosed an intradural filling defect at T-7, and CTM demonstrated flattening of the mid-thoracic cord with homogeneous contrast enhancement at this site. A T6–8 laminectomy was performed. When the dura was opened the cyst was also entered, yielding a gush of CSF. The cyst was completely dissected from the surrounding tissue except...
TABLE 2
Summary of cases of Type I spinal extradural meningeal cysts*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Symptoms</th>
<th>Cyst Location</th>
<th>Location of Cyst Pedicle</th>
<th>Communication†</th>
<th>Arachnoid Layer‡</th>
<th>Comments</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>14, M</td>
<td>rt thoracic pain, bilat leg weakness &amp; numbness</td>
<td>T6-8</td>
<td>lt T-6 n root</td>
<td>yes</td>
<td>no</td>
<td>yes</td>
</tr>
<tr>
<td>2</td>
<td>16, M</td>
<td>bilat leg weakness, numbness both legs to mid-chest</td>
<td>T3-7</td>
<td>rt T-3</td>
<td>---</td>
<td>---</td>
<td>yes</td>
</tr>
<tr>
<td>3</td>
<td>18, M</td>
<td>rt flank &amp; abdominal pain</td>
<td>L1-2</td>
<td>lt L-2 n root</td>
<td>yes</td>
<td>no</td>
<td>yes</td>
</tr>
<tr>
<td>4</td>
<td>20, F</td>
<td>LBP</td>
<td>L2-3</td>
<td>lt L-2 n root</td>
<td>no</td>
<td>no</td>
<td>yes</td>
</tr>
<tr>
<td>5</td>
<td>11, F</td>
<td>bilat leg pain, inversion of both feet</td>
<td>T12-L3</td>
<td>lt L-2 n root</td>
<td>---</td>
<td>no</td>
<td>yes</td>
</tr>
<tr>
<td>6</td>
<td>42, F</td>
<td>urinary retention</td>
<td>dumbbell lesion: L-1 intraspinal &amp; massive intrapelvic extension</td>
<td>between two coccygeal n roots</td>
<td>---</td>
<td>yes</td>
<td>yes</td>
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<tr>
<td>7</td>
<td>36, F</td>
<td>LBP, dorsiflexion, weakness</td>
<td>L5-S4</td>
<td>lt S-2 n root</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
</tr>
<tr>
<td>8</td>
<td>77, F</td>
<td>LBP, abdominal pain</td>
<td>S1-3</td>
<td>caudal tip of dural sac, ? n root</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>9</td>
<td>32, F</td>
<td>LBP</td>
<td>S1-2</td>
<td>caudal tip of dural sac, ? n root</td>
<td>---</td>
<td>---</td>
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</tr>
</tbody>
</table>

* LBP = low-back pain; n = nerve; MRI = magnetic resonance imaging; SEM = scanning electron microscopy; VP = ventriculoperitoneal; --- = study not performed.
† Communication with the subarachnoid space detected by computerized tomographic myelography (CTM), or routine myelography, or at surgery.
‡ Arachnoid layer found in histological examination.

for dense adhesions attached to the left dorsal T-7 nerve root. This nerve root was sacrificed and the cyst was completely removed. Histological examination of the cyst disclosed areas of opaque delicate fibrocollagenous tissue with a single-cell cuboidal epithelial lining, and areas of dense fibrocollagenous tissue. Postoperatively, the pain and numbness resolved. The patient's strength improved and she regained bladder control.

Discussion

Spinal meningeal cysts (MC's) are actually diverticula of the spinal meningeal sac, nerve root sheath, or arachnoid. For the sake of simplicity, and to avoid further confusion in the nomenclature, we continue to refer to these diverticula as "cysts." The spinal MC's examined in this study are considered congenital. Although some authors have described hemosiderin in the cyst wall in at least two categories, attributing their origin to trauma, we believe the presence of pigmented cells to be an epiphenomenon unrelated to the etiology. Trauma did not play a role in the pathogenesis of our 22 cases, despite histopathological findings in one case; histological examination of the intradural MC specimen obtained from Case 19 (a 3-year-old boy with multiple congenital anomalies who had no prior history of trauma) revealed hemosiderin-laden cells within the cyst wall. Further discussion of the congenital etiology of these lesions is presented later.
Spinal meningeal cysts

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Symptoms</th>
<th>Cyst Location</th>
<th>Communication†</th>
<th>Arachnoid Layer‡</th>
<th>Comments</th>
</tr>
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<tbody>
<tr>
<td>10</td>
<td>53, F</td>
<td>LBP, rt leg pain, urinary incontinence</td>
<td>rt S-2 n root</td>
<td>yes</td>
<td>no</td>
<td>yes</td>
</tr>
<tr>
<td>11</td>
<td>47, M</td>
<td>rt leg pain</td>
<td>rt S-2 n root</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
</tr>
<tr>
<td>12</td>
<td>59, F</td>
<td>lt buttock pain, lt leg pain</td>
<td>lt S-2 n root</td>
<td>yes</td>
<td>yes</td>
<td>no</td>
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<tr>
<td>13</td>
<td>70, F</td>
<td>LBP</td>
<td>lt S-2 n root</td>
<td>yes</td>
<td>no</td>
<td>no</td>
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<tr>
<td>14</td>
<td>23, M</td>
<td>coccygodynia</td>
<td>lt S-2 n root</td>
<td>yes</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>15</td>
<td>61, M</td>
<td>urinary retention, sepsis</td>
<td>rt S-3 n root</td>
<td>yes</td>
<td>no</td>
<td>no</td>
</tr>
<tr>
<td>16</td>
<td>15, F</td>
<td>pain &amp; numbness of lt leg &amp; lt labia</td>
<td>multiple sacral n root cysts, largest cyst at lt S-3 n root</td>
<td>yes (except for lt S-3 cyst)</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>17</td>
<td>26, F</td>
<td>LBP, lt leg pain</td>
<td>lt S-2 n root</td>
<td>--</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>18</td>
<td>14, F</td>
<td>headache, abdominal pain, LBP</td>
<td>bilat S-2</td>
<td>--</td>
<td>yes</td>
<td>--</td>
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</table>

* LBP = low-back pain; n = nerve; MRI = magnetic resonance imaging; -- = study not performed.
† Communication with the subarachnoid space detected by computerized tomographic myelography (CTM), or routine myelography, or at surgery.
‡ Arachnoid layer found on histological examination.

Our classification divides spinal MC's into three groups: extradural MC's without spinal nerve root fibers (Type I); extradural MC's with spinal nerve root fibers (Type II); and intradural MC's (Type III). Extradural MC's without spinal nerve root fibers (Type I) are dural diverticula that occur anywhere along the thecal sac, and include "extradural meningeal cysts" and "sacral meningoceles." Type I extradural MC's arising above the sacrum contain a pedicle that is usually adjacent to the entrance of the dorsal nerve root. "Sacral meningoceles" are Type I extradural MC's of the sacrum which contain a pedicle at the caudal tip of the dural sac, usually adjacent to dorsal sacral or coccygeal nerve roots.

Thoracic Type I extradural MC's occur more frequently in adolescents, and sacral Type I cysts are found more often in adults. The duration of symptoms is shorter for thoracic MC's than for sacral lesions; the different diameters of the thoracic and sacral spinal canal may account for this. Symptom presentations are likewise different, and are dependent upon the proximity of the cyst to the spinal cord and nerve roots. Thoracic and cervical Type I cysts may present with spasticity and sensory levels. Lumbar Type I cysts may present with low-back pain and radiculopathy. Sacral Type I cysts may present with radicular symptoms with bowel or bladder dysfunction.

Extradural MC's without spinal nerve root fibers (Type I) are treated by closing the ostium between the cyst and the subarachnoid space. Usually Type I cysts above the sacrum can be completely dissected and elevated off the dura as seen in Cases 3, 4, and 5. In some instances, dense fibrous adhesions prevent this and, as in Cases 1 and 2, the posterior wall must be resected. For Type I cysts of the sacrum, the ostium is sufficient. Postoperatively, seven patients with Type I cysts improved with resolution of pain and return of neurological function. In Case 2, epidural fluid reaccumulated after the operation. This was the only patient who did not improve after simple closure of the ostium; he required placement of a ventriculoperitoneal shunt. This patient had ventriculomegaly preoperatively. In Case 5, symptoms recurred after several months due to severe progressive scoliosis unrelated to cyst or fluid reaccumulation.

Extradural MC's with nerve root fibers (Type II) appear grossly as dilatations of spinal nerve root sleeves and include what was previously termed Tarlov's "perineurial cysts" and "spinal nerve root diverticula." In most situations, CTM shows that "perineurial cysts" and "spinal nerve root diverticula" communicate with the subarachnoid space, and are therefore the same lesion. Six of the Type II cysts in our series were shown by CTM to communicate with the subarachnoid space. Although these cysts are often multiple and occur on dorsal nerve roots anywhere along the spinal cord, they are most prominent and symptomatic in the sacrum. The spinal nerve root fibers for these cysts either lie within the cyst wall or exist freely, centrally, within the cyst bathed in CSF. Type II extradural MC's occur in adults and are often asymptomatic; however, when they do cause symptoms, the patient may present...
with “sciatica” or a complaint of bowel or bladder problems.

Since Type II cysts do not have a pedicle, the aim of surgery is to obliterate the cyst by partial resection and oversewing the cyst wall (that is, the dilated nerve root sheath) or by total excision of the cyst with the nerve root. We prefer to preserve as much function as possible using the former technique. One of the authors (E.B.B.) incises the nerve root sheath, then Oversews and reconstructs it under the operative microscope using a Silastic graft. Simple aspiration of the cysts is not recommended and has produced poor results with fluid reaccumulation.65 Postoperatively, all of our patients with Type II cysts improved, with resolution of their pain and return of neurological function including bladder control.

In nine of the patients with Type I or II cysts, MR imaging revealed an extrapinal cystic mass. On MR imaging, extradural MC’s are depicted on sagittal, coronal, and axial planes as masses with low intensity on T₁-weighted images and with high intensity on T₂-weighted images, similar to CSF. Because MR studies in Case 2 provided such detailed images, no additional preoperative radiodiagnostic studies were obtained (Fig. 4). Routine water-soluble myelography of extradural MC’s demonstrates an extradural defect, but filling of the cyst itself may not be visualized. If filling is not present, it is essential to perform CTM, which usually demonstrates communication with the subarachnoid space. This allows radiographical confirmation of the diagnosis of a spinal extradural MC and rules out numerous other mass lesions. Only in two of the 11 examples from both categories of extradural MC’s was patency not apparent on CTM. In these two cases, delayed CTM may have disclosed patency.

Bone erosion, demonstrated by canal widening, pedicle erosion, foraminal enlargement, or scalloping of the vertebral bodies or sacrum, was present to some degree in all 18 cases of extradural MC’s. Gortvai23 described how an opening into the cyst could not by itself explain cyst expansion or compression, by either static, dynamic, or pulsatile features of CSF pressure. McCrum and Williams38 discussed CSF dynamics and, like Gortvai, proposed a valve-like mechanism with intermittent surges in CSF pressure to explain cyst expansion and bone erosion. Like these authors, we believe that bone erosion implies the presence of a valve mechanism that is responsible for producing forces of CSF pressure within the cyst greater than normal hydrostatic forces.

Intradural MC’s (Type III cysts) also contain a valve mechanism that regulates communication with the subarachnoid space. Intradural MC’s can also erode bone. On routine water-soluble myelography these lesions appear as intradural defects, but on CTM they enhance homogeneously when they communicate with the subarachnoid space. In the one case where there was no communication, delayed CTM might have disclosed patency. In Case 22, MR imaging was useful as an initial study, demonstrating an extraaxial lesion with the same signal intensity as CSF that compressed the spinal cord posteriorly (Fig. 13).

Type III cysts usually occur anywhere along the posterior spinal subarachnoid space. Anterior cysts are less common.61 Like Type II cysts, they are often multiple and asymptomatic.65,67 When these cysts do become symptomatic, they produce signs and symptoms like any intradural mass lesion, depending on their anatomical relationship to the spinal cord and spinal nerve roots. The etiology of these lesions is attributed to the proliferation of arachnoid trabeculae. Some authors believed that intradural cysts developed from the arachnoid “faults” in the septum posticum,67 but this explanation does not explain anterior lesions.

### TABLE 4

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Symptoms</th>
<th>Cyst Location</th>
<th>Communication†</th>
<th>Arachnoid Layer‡</th>
<th>Comments</th>
</tr>
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<tbody>
<tr>
<td>19</td>
<td>3, M</td>
<td></td>
<td>LBP, urinary incontinence</td>
<td>T10–T12</td>
<td>no</td>
<td>no</td>
<td>yes (ostium not identified)</td>
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<tr>
<td>20</td>
<td>72, F</td>
<td></td>
<td>LBP</td>
<td>rt L-5 &amp; rt S-1</td>
<td>yes</td>
<td>no</td>
<td>no</td>
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<tr>
<td>21</td>
<td>61, F</td>
<td></td>
<td>LBP, bilateral leg pain</td>
<td>L1–3</td>
<td>—</td>
<td>no</td>
<td>no</td>
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<tr>
<td>22</td>
<td>61, F</td>
<td></td>
<td>LBP, bilateral leg weakness, numbness from waist down, urinary incontinence</td>
<td>T6–8</td>
<td>yes</td>
<td>no</td>
<td>no</td>
</tr>
</tbody>
</table>

* LBP = low-back pain; MRI = magnetic resonance imaging; — = study not performed.
† Communication with the subarachnoid space detected by computerized tomographic myelography (CTM), or routine myelography, or at surgery.
‡ Arachnoid layer found on histological examination.
Spinal meningeal cysts

Two of our examples of Type II cysts (Cases 17 and 18) were completely excised with adhesive attachments that originated laterally on the spinal nerve roots before they exited the dura. In cases where the cyst cannot be resected completely due to dense fibrous adhesions attached to the spinal cord or nerve roots, marsupialization should be performed as widely as possible. Intradural MC's have been reported to recur after incomplete excision. Surgery with complete excision in our four patients with Type III cysts produced an excellent return of neurological function in each case.

Several authors have proposed various etiologies for each category of spinal MC. In 1934, Elsberg, et al., attributed the origin of Type I cysts to either congenital diverticula of the dura or a herniation of the arachnoid through a congenital dural defect. Although an arachnoid layer may be present, light microscopy examination revealed a fibrous wall without a single-cell inner lining in six of our Type I cases. This finding favors the former etiology proposed by Elsberg, et al. A very thin single-cell layer was found in one of our two cases examined by SEM (Case 1, Fig. 3 right), even though no such layer was revealed by light microscopy. The arachnoid may be eroded or fused to the herniated dural layer over time and, when present, may require magnification greater than the light microscopic level to be visualized. A congenital origin for all three categories of cysts is further supported by the fact that a number of previous investigators have described a familial tendency or associated congenital anomalies. In our series, one patient with a Type I cyst and one with a Type III cyst had stigmata of spinal dysraphism. The fact that Type I cysts usually arise at a consistent location near the origin of the dorsal spinal nerve root also tends to favor a developmental etiology.

Nugent, et al., proposed that the arachnoidal proliferations within the nerve root sleeve, as described by Rexed and Wennström, led to the obstruction of normal CSF pathways and the development of spinal MC's. Certainly, this explanation is plausible for Type II cysts, but Fortuna, et al., believed that the arachnoid proliferations were responsible for the origin of all three categories. The developmental differentiation between Type I and Type II cysts may be due to CSF obstruction proximal to the arachnoid proliferations within the nerve root sleeves for Type I cysts and distal to these proliferations for Type II cysts: Type I cysts (diverticula) would develop at congenital meningeal defects proximal to the arachnoid proliferations at the origin of the spinal nerve root, and Type II cysts (nerve sheath dilations) would develop within the confines of spinal nerve root sheaths distal to the arachnoid proliferations at the origin of the spinal nerve root.

At surgery, there were no arachnoid proliferations within the ostium of Type I cysts. Examination of the ostium of Type II cysts did reveal arachnoid proliferations obstructing the flow of CSF. This obstruction to CSF was more complicated than a simple one-way valve because CSF was impeded from not only exiting but also entering the cyst. At surgery, five cases of Type II cysts did not exhibit free flow of CSF into the cyst. This complex valve mechanism is probably what has misled previous investigators to believe that some cysts did not communicate with the subarachnoid space, hence the separate categorization between "perineurial cysts" and "spinal nerve root diverticula." As demonstrated by our Cases 16 and 18, the development of the complex valve mechanism theoretically may be directly responsible for the degree of communication with the subarachnoid space both into and out of the cyst, and also for cyst size and growth. In Case 16, CTM showed multiple Type II cysts communicating with the subarachnoid space; however, the largest cyst did not (Fig. 11). In Case 18, the largest of all the Type II cysts in this series, the cyst also did not communicate with the subarachnoid space on CTM. Although an ostium was not evident on gross surgical inspection of our four examples of Type III cysts, one probably existed microscopically. The issue of cyst patency, which was the primary distinguishing feature between Type II "perineurial cysts" and "nerve root meningoceles," has been proven invalid on CTM studies. Similarly, whether or not Type I or III cysts communicate with the subarachnoid space is no longer a matter of debate. It appears that all spinal MC's (Types I to III) do or did at one time communicate with the subarachnoid space.

Upon surgical inspection and histopathological examination, the presence or absence of spinal nerve root fibers was the distinguishing feature between the two classes of extradural lesions. Although an arachnoid layer has been described in cases of extradural cysts, all seven specimens examined from both categories of extradural lesions in our study lacked an inner single-cell layer on light microscopy histological examination. Although all four specimens of Type III cysts in our series exhibited a single-cell arachnoid layer, reports have also described nonspecific connective tissue. The presence of a single-cell arachnoid layer for any category is an inconsistent finding. The diagnosis of spinal MC's is not made solely by histological examination. Rather, these lesions are pathoanatomic entities of congenital origin, possibly derived directly from, or secondary to, spinal arachnoid proliferations.

Conclusions

In the preoperative assessment of spinal MC's, MR imaging was performed in 10 examples and delineated an intraspinal cystic mass in each instance. Although routine water-soluble myelography disclosed filling defects, CTM characterized these lesions by demonstrating a communication with the subarachnoid space in 12 of 14 cases. Spinal MC's were classified into three major categories: extradural MC's without spinal nerve root fibers (Type I); extradural MC's with spinal nerve fibers (Type II); and intradural MC's (Type III). This classification was based upon findings from surgical
examination and histopathological review. The authors believe that previous classifications based on the presence of an “arachnoid” layer or communication with the subarachnoid space were misleading or inaccurate, and that the classification of these cysts described here, based on current radiographic, histological, and operative findings, provides a better understanding of the preoperative assessment, anatomic development, and surgical management of these lesions.

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