Tarlov or perineurial cysts are pathological formations located in the space between the peri- and endoneurium of the spinal posterior nerve root sheath at the DRG.[17,39,41] These lesions have been estimated to affect between 4.6 and 9% of the adult population.[24,34] Although originally believed by Tarlov[39] to be asymptomatic lesions, these cysts, when present in the sacral neural canal and foramina, have since been found to cause a variety of symptoms, including radicular pain, paresthesias, and urinary or bowel dysfunction.[4,5,7,15,16,18,24,31,32,38,46] The development of CT myelography has led to an improvement in our ability to diagnose “Tarlov cysts” as a cause of sacral radiculopathy.[16,17,20,21,42] Although the term Tarlov cyst has often been erroneously applied to other cystic spinal lesions,[9,13,17,19,26,27,29,35] the distinctive feature of the Tarlov perineurial cyst is the presence of spinal nerve root fibers within the cyst wall, or the cyst cavity itself.[9,17,37–41]

Despite advancements in diagnosis, there remains a great deal of controversy regarding the optimal treatment of symptomatic Tarlov cysts. Nonsurgical therapies include lumbar CSF drainage[2,4] and CT scanning–guided cyst aspiration,[23,24] neither of which prevents symptomatic cyst recurrence. Neurosurgical techniques for symptomatic perineurial cysts include simple decompressive laminectomy,[33] cyst and/or nerve root excision,[20,37,38,41,42] and microsurgical cyst fenestration and imbrication.[16] Although no consensus exists on the definitive treatment of symptomatic Tarlov cysts, we believe surgical methods have yielded the best long-term results to date. We describe the case of one patient with a symptomatic Tarlov cyst to illustrate the surgical treatment involving cyst fenestration, partial cyst wall resection, and myofascial–cutaneous flap repair and closure in a case of a symptomatic sacral Tarlov cyst. They review the medical literature, describe various theories on the origin and pathogenesis of Tarlov cysts, and assess alternative treatment strategies.

**CASE REPORT**

**Presentation.** This 47-year-old woman presented with a 1-year history of progressive, intractable sacrococcygeal pain and numbness as well as dysesthesias of both feet. At the time, she was becoming increasingly incapacitated, although she was still able to work as a flight attendant. She rated her pain as 6 of 10 possible points on a visual analog scale. Her symptoms were aggravated by standing,
walking, lifting, and climbing stairs as well as by coughing. Pain was rapidly relieved by recumbency. She had a history of renal calculi. She had no bowel or bladder dysfunction, and sensation for urination and defecation was normal.

**Examination.** On physical examination, a grade of 5/5 strength was demonstrated throughout all muscle groups. Heel and toe walking and knee bends were well performed. Straight leg raising was negative. Jugular compression test induced sacrococcygeal paresthesias without pain. Sensory examination showed diminished sensory perception to pinprick on the soles of her feet and in the S1–2 distribution. There was no sensory deficit over the perineum. Anal sphincter tone and constriction were normal. Knee jerks were hyperreflexive with a Grade 4+ response bilaterally, and ankle jerks were Grade 2+ bilaterally. Lumbar flexion seemed to relieve the pain, whereas extension made it worse.

Preoperative MR imaging demonstrated a large sacral cyst arising within the thecal sac at S-2, with expansion of the osseous sacral central canal and enlargement of S-1 and S-2 neural foramina causing compression of all adjacent nerve roots. Postmyelography CT scanning revealed evidence of cauda equina compression. The cyst did not fill with contrast material and appeared to have no communication with the spinal subarachnoid space (Fig. 1).

**Operation.** To relieve progressively incapacitating symptoms, surgery was recommended. After sacral laminectomy, microsurgical cyst fenestration was performed with the assistance of intraoperative electromyographic monitoring. Muscle flap closure reinforcement was conducted as described previously. Briefly, after exposure of the S2–S4 sacral nerve roots, a large meningeal cyst was identified arising from the S-2 nerve root. The thin transparent cyst wall membrane was widely fenestrated with a scalpel and microscissors. Clear fluid contents of the cyst drained spontaneously. The posterolateral wall of the cyst was resected after electrical stimulation verified that no motor nerve fibers were present. Anal sphincter electromyography was activated by low intensity 0.4 mA electrical stimulation applied to the anterior and medial surfaces of the cyst wall. Although rapid high-volume drainage of CSF from the rostral subarachnoid space was not observed, some seepage did occur, indicating that microscopic communication was present.

Fibrin glue was then applied to fill the cyst cavity. To prevent cyst recurrence or CSF leakage, a sacral spinalis muscle flap was then rotated into the epidural space created by evacuation of the S2–S4 cyst. Cyst closure was also supported using a lumbar subarachnoid drain for postoperative CSF diversion (Fig. 2). Although the cyst wall specimen was sent to the laboratory for pathological examination, its volume was inadequate to determine whether nerve root fibers were present.

**Postoperative Course.** The patient’s postoperative course was uneventful. The lumbar drain was removed on postoperative Day 3, and a CT myelography revealed a very small amount of contrast exiting from the thecal sac to surround the myocutaneous flap. There was only mild indentation of the posterior thecal sac (Fig. 3). She noted marked improvement in her pain symptoms prior to discharge home on Day 4 postoperatively. During the follow-up visit 5 weeks later, she reported that the burning in her
Diagnosis and management of sacral Tarlov cysts

Fig. 3. A and B: Postoperative CT myelograms demonstrating obliteration of the cyst cavity with a muscle graft occupying the space formerly filled by the cyst.

feet had resolved; however, she experienced some intermittent residual sacral coccygeal and retrorectal pain, which was much less severe than it had been preoperatively. There were no focal sensory or motor deficits. Subsequent communication from the patient indicated that further resolution of residual pain occurred slowly over the ensuing 3 months.

DISCUSSION

Definition and Classification of Tarlov Cysts

Tarlov’s original description of “perineurial cysts” was reported in 1938 and based on observations made during dissection of 30 cadaveric terminal filum specimens at the Montreal Neurological Institute.39 He noted extradural cysts, often multiple, on portions of the posterior sacral and coccygeal nerve roots in five of these specimens. On histological examination, he found that these cysts were located in the perineurial space, between the endo- and perineurium at the junction of the posterior nerve root and its ganglion. These lesions either surrounded the entire nerve root or invaded it and became surrounded by compressed nerve fibers. Based on this and later studies, Tarlov38,39,41 distinguished perineurial cysts from meningeal diverticula on the basis of three main findings (Table 1). First, Tarlov cysts have a potential, but not an actual, communication with the spinal subarachnoid space and thus usually fill initially during myelography. Meningeal diverticula, on the other hand, are in free communication with the spinal subarachnoid space and thus may exhibit delayed filling or lack of filling on myelographic examination. Meningeal diverticula, on the other hand, are in free communication with the spinal subarachnoid space and thus usually fill initially during myelography. Second, Tarlov cysts occur at or distal to the junction of the posterior nerve root and the DRG, usually in the

TABLE 1

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Communication W/ SS</th>
<th>Filling Pattern on Myelography</th>
<th>Location Along Nerve Root</th>
<th>Nerve Fibers W/ Nerve Root</th>
</tr>
</thead>
<tbody>
<tr>
<td>perineural cysts (Tarlov cysts)</td>
<td>no</td>
<td>delayed filling</td>
<td>at or distal to junction of pst nerve root &amp; DRG</td>
<td>yes</td>
</tr>
<tr>
<td>meningeal diverticula</td>
<td>yes</td>
<td>rapid filling</td>
<td>proximal to DRG</td>
<td>no</td>
</tr>
<tr>
<td>long arachnoidal prolongations</td>
<td>yes</td>
<td>rapid filling</td>
<td>continuous prolongation of SS over nerve root</td>
<td>NA†</td>
</tr>
</tbody>
</table>

* NA = not applicable; pst = posterior; SS = subarachnoid space.
† Although these can be confused with meningeal diverticula and perineurial cysts, arachnoidal prolongations are not actual cystic lesions.
Signs and Symptoms of Tarlov Cysts

In his original description, Tarlov noted that perineurial cysts developed in asymptomatic patients, although he later speculated that these cysts could cause sacral radiculopathy when he noted that resection of a cyst relieved one woman of sciatic pain. Although they most often affect S-2 and S-3 nerve roots, Tarlov cysts have been found to occur anywhere in the cervical, thoracic, and lumbar spine. Most are diagnosed as incidental findings on CT or MR imaging. Approximately one fifth of Tarlov cysts are symptomatic and can produce, according to their anatomical location, a variety of symptoms of nerve root compression (Table 3). Sacral cysts have been noted to cause sacral radiculopathy, hip, leg, or foot pain, perineal pain, paresthesias, and bowel or bladder dysfunction. Symptoms can be exacerbated by standing, coughing, or other Valsalva maneuvers, because elevated subarachnoid pressure forces CSF from the spinal subarachnoid space through a small valve-like communication into the perineurial cyst cavity. Tarlov cysts may also cause diffuse poorly localized sacral pain due to pressure on adjacent periosteum and joint capsules and can also result in sacral insufficiency fractures from erosion of the sacrum.

Radiological Features

Tarlov cysts are associated with a variety of radiological findings. Initial plain radiographic examination may reveal Tarlov cysts causing erosion of the sacrum, bone scalloping, or a rounded paravertebral shadow. Schriever and Haddad were the first to demonstrate that Tarlov cysts exhibited a characteristic delayed filling pattern on oil-based pantopaque contrast-enhanced myelography. This pattern, in which Tarlov cysts do not immediately fill with oil-based contrast but can later be visualized hours, days, or weeks later, has since been described by several authors, and was one of the criteria used by Tarlov to distinguish perineurial

<table>
<thead>
<tr>
<th>Classification, Authors &amp; Year</th>
<th>Nomenclature</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Goyal, et al., 1987</td>
<td>perineurial cyst/Tarlov cyst root sleeve dilation extradural arachnoid cyst traumatic root cyst</td>
<td>cyst formation w/in nerve root sheet at DRG enlargement of SS around nerve root proximal to DRG arachnoid outpouching through dural defect traumatic tear in leptomeninges causing CSF collection extradural meningeal cyst w/out nerve root fibers extradural meningeal cyst w/ nerve fibers intradural spinal meningeal cyst</td>
</tr>
<tr>
<td>Nabors, et al., 1988</td>
<td>Type I</td>
<td>Type II (Tarlov cyst) Type III</td>
</tr>
</tbody>
</table>

## TABLE 3

Clinical and radiological characteristics of Tarlov cysts*

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>No. of Cases</th>
<th>Location of Cysts</th>
<th>Presenting Symptoms</th>
<th>Radiological Exam</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nabors, et al., 1988</td>
<td>9</td>
<td>sacral (S2–3)</td>
<td>sacral radicular pain in 5; low-back pain in 4; bladder dysfunction in 2; coccydynia in 1; abdominal pain in 1; headache in 1 sacral radicular pain in 8; motor deficits in 4; bladder dysfunction in 3; impotence in 1</td>
<td>CTM alone in 5; CTM + MRI in 2; MRI alone in 1; MRI + conventional myelography in 1</td>
<td>MRI showed spinal cyst in 4 of 4 patients; CTM showed communication w/ SS in 7 of 7†</td>
</tr>
<tr>
<td>Mummaneni, et al., 2000</td>
<td>8</td>
<td>sacral (S1–3)</td>
<td></td>
<td>MRI in all; CTM in 6</td>
<td>MRI confirmed spinal cyst in all; CTM showed communication w/ SS in 5 of 6</td>
</tr>
<tr>
<td>Voyadzis, et al., 2002</td>
<td>10</td>
<td>sacral (S1–3) in all; L-5 meningeal diverticulum in 1</td>
<td></td>
<td>CTM in all; MRI in some‡</td>
<td>CTM showed communication with SS in all; MRI demonstrated spinal cyst in at least 1‡</td>
</tr>
</tbody>
</table>

* CTM = CT myelography.
† In one patient with multiple sacral nerve root cysts, CT myelography showed all but one cyst to communicate with the SS.
‡ The number of patients who underwent preoperative MR imaging is not reported.

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cysts from meningeal diverticula. Because Tarlov cysts have only a potential, or very small communication with the subarachnoid space, the visualization of the cyst cavity on myelography occurs as a result of a much slower process in which contrast is forced, via the hydrostatic pressure of CSF, from the subarachnoid space through this small aperture or by transudation across the cyst wall membrane, into the perineurial cyst cavity. Meningeal diverticula, on the other hand, having a relatively greater communication with the spinal subarachnoid space, can be seen to fill with oil-based contrast immediately after injection during myelography. Tarlov cysts can, however, often be seen to fill more rapidly on myelography with lower viscosity water-soluble contrast materials (metrizamide). The authors of other studies have found Tarlov cysts to have no communication with the spinal subarachnoid space, thus only producing an extraarachnoid filling defect on myelography.

Computerized tomography scanning, both with and without intrathecal contrast material, has enhanced our ability to detect perineurial cysts. These lesions are iso- dense with CSF on noncontrast CT scans and can often be seen to cause various osseous abnormalities and erosions. Postmyelography CT scanning is effective in demonstrating the presence of a communication of the cyst with the spinal subarachnoid space, and it can also demonstrate surrounding sacral bone scalloping. Because of the absence of interference from bone, enhanced resolution of tissue density, and its multiplanar imaging capabilities, MR imaging is particularly useful in studying sacral Tarlov cysts, thus, it is considered to be the preferred initial imaging modality when a sacral perineurial cyst is suspected. These cysts have CSF-like characteristics on MR imaging—that is, a low signal on T₂-weighted images and a high signal on T₁-weighted images. Magnetic resonance imaging can also be used to delineate the exact relationship of the cyst to the thecal sac, as well as the total volume of fluid within the cyst. It may also demonstrate bone and pedicle erosion, sacral canal widening, and neural foramina enlargement. The senior author (P.R.W.) and others have recommended MR imaging for initial identification of a spinal cystic mass, followed by CT myelography for further delineation of cyst characteristics (Table 3).

Origin and Pathogenesis of Tarlov Cysts

In his original description, Tarlov discovered inflammatory cells in some cyst walls and adjacent tissues, leading him to postulate that these cysts were formed by a process of inflammation within the nerve root sheath, followed by inoculation of fluid. He later noted that pannus-like contrast could initiate an inflammatory process that sealed off communication between an asymptomatic meningeal diverticulum and the subarachnoid space, creating a symptomatic perineurial cyst. He also described changes consistent with ischemic degeneration in some cysts. A hemorrhagic origin was later described by Tarlov and others, in which cyst formation occurs as a result of infiltrating subarachnoid hemorrhage, or traumatic intraneural hemorrhage, followed by cystic degeneration of red blood cells and destroyed neural tissue. Nishiura, et al., described a history of antecedent trauma in 40% of their patients with Tarlov cysts. Schreiber and Haddad noted that cysts can form as a result of dural lacerations during spinal surgery, leading to pseudomeningocele formation. Some authors favor a congenital origin and hypothesize that these cysts can arise from a congenital dural diverticulum or weakness, or from persistent embryonic fissures. Tarlov cysts have been associated with other congenital abnormalities, connective tissue disorders, and nerve root sheath duplications. It may also demonstrate bone and pedicle erosion, sacral canal widening, and neural foramina enlargement. The senior author (P.R.W.) and others have recommended MR imaging for initial identification of a spinal cystic mass, followed by CT myelography for further delineation of cyst characteristics (Table 3).

Natural History and Treatment of Symptomatic Tarlov Cysts

Although most remain asymptomatic throughout the patient’s life, the natural history of symptomatic Tarlov cysts is one of progressive enlargement leading to increasing symptoms. The hydrostatic and pulsatile forces of CSF cause Tarlov cysts to grow over time. As the mass enlarges, sensory nerve root filaments are stretched over the periphery of the lesion or are compressed against adjacent bone or other nerve roots, causing pain or other sensory disturbances.

There is no consensus on the optimal management of symptomatic Tarlov cysts. Nonsurgical methods include lumbar CSF drainage to decrease the hydrostatic pressure and therefore the pressure within the cyst cavity. Symptoms do recur, however, after drainage is stopped. In one case the authors found that continuous CSF drainage via a lumbo-peritoneal shunt was an effective therapy for symptomatic Tarlov cysts in each of two patients. Some authors have asserted that by providing temporary symptomatic relief, lumbar CSF drainage can be a useful diagnostic tool when there is a question about the clinical significance of Tarlov cysts.

Another nonsurgical treatment is percutaneous cyst drainage. Symptoms do recur after percutaneous drainage because cysts reform, although the authors of one study asserted that percutaneous cyst aspiration and fibrin glue placement can prevent the recurrence of both the cyst and its associated symptoms. In this study, 75% of the patients developed postprocedural aseptic meningitis. As a result of these findings, some authors have argued that there is no place for percutaneous cyst drainage in the treatment of these lesions.

Surgical treatment of symptomatic perineurial cysts, involving complete cyst removal, and excision of the affected posterior root and ganglion, was advocated by Tarlov and has since been used by others, although...
Indications for Surgery

The authors of one study found that patients with Tarlov cysts greater than 1.5 cm and with associated radicular pain or bowel/bladder dysfunction benefit most from surgery. We have recommended that patients presenting with radicular pain referable to the location of a cyst should initially undergo medical treatment with antiinflammatory drugs and physical therapy. Surgery should be reserved for that subset of patients in whom these conservative therapies include cyst resection at the neck, cyst fenestration and imbrication, and cyst shrinkage by using bipolar cautery. One author described the surgical placement of a cyst–subarachnoid shunt tube to equalize pressures within the spinal subarachnoid space and the cyst cavity. In our experience, good results have been obtained with microsurgical cyst fenestration and imbrication when using electrophysiological monitoring, along with a fat- or muscle graft–reinforced cyst closure. In a study conducted at our institution, seven of eight patients experienced improvement in symptoms. Bladder control improved in two of three patients with bladder dysfunction treated with cyst fenestration and imbrication. There were no postoperative complications. In the present case, cyst imbrication was not performed because CT myelography revealed no direct communication with the spinal subarachnoid space. We instead performed microsurgical cyst fenestration and cyst wall resection with myocutaneous flap closure reinforcement to prevent a reaccumulation of CSF or a cyst recurrence. Cyst wall resection may result in postoperative neurological deficits and should be performed during electrophysiological monitoring to minimize damage to sacral nerve roots.

CONCLUSIONS

Tarlov cysts are a documented cause of sacral radiculopathy and other radicular pain syndromes. These lesions are diagnosed with MR imaging, which should be followed by CT myelography to demonstrate communication with the spinal subarachnoid space. Although first described over 60 years ago, there is still no uniform opinion on the origin of these cysts; they may be caused by inflammation, trauma, or congenital weakness of the dura mater combined with elevated CSF hydrostatic pressure.

Symptomatic Tarlov cysts have been treated with various techniques, including CSF drainage, percutaneous aspiration, and excision. We have described the case of a woman with a symptomatic sacral Tarlov cyst treated successfully with cyst fenestration, cyst wall resection, and closure with myocutaneous flap reinforcement to prevent cyst recurrence or CSF leakage. Electrophysiological monitoring is necessary to minimize damage to sacral nerve roots. Patients with radicular symptoms exacerbated by postural changes and Valsalva maneuvers are the best candidates for surgery.

References

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Manuscript received June 30, 2003. Accepted in final form July 17, 2003.

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