# The Clinical Relevance of Tarlov Cysts

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**Objective:** The sacral perineural cyst was first described by Tarlov in 1938 as an incidental finding at autopsy. There are very few data in the literature regarding the role of Tarlov cysts in causing symptoms, however. Most studies report low numbers, and consequently, the recommendations for treatment are vague. Our aim, therefore, is to present further detail regarding the clinical relevance of Tarlov cysts and to identify whether or not they are a cause of lumbosacral spinal canal stenosis symptoms.

**Methods:** Over a 5-year period, 3535 patients underwent magnetic resonance imaging (MRI) scan for lumbosacral symptoms. Fifty-four patients were identified as having Tarlov cysts, and their clinical picture was correlated with the findings on MRI.

**Results:** The majority of Tarlov cysts (n = 38) cannot be held responsible for patients' symptoms and are clinically unimportant. However, we encountered several patients in whom Tarlov cysts (n = 9) occurred at the same level as another pathology. In these cases, the cyst itself did not require any specific therapy; treatment was directed at the other pathology, and uneventful symptom resolution occurred. A smaller subgroup of cysts (n = 7) are the main cause of patients' symptoms and may require specific treatment to facilitate local decompression.

**Conclusions:** The majority of Tarlov cysts are incidental findings on MRI. Where confusion exists as to the clinical relevance of a Tarlov cyst, treatment of the primary pathology (ie, non-Tarlov lesion) is usually sufficient. Tarlov cysts may, however, be responsible for a patient's symptoms; possible mechanisms by which this may occur and treatment strategies are discussed.

**Key Words:** Tarlov cyst, spinal stenosis, radiculopathy, low back pain

(*J Spinal Disord Tech* 2005;18:29–33)

The presence of cysts within the sacral spinal canal, so-called "sacral cysts," is well described in the literature. The sacral perineural cyst was first described by Tarlov in 1938 as an incidental finding at autopsy.[1] The cyst walls are composed of perineurium and neural tissue, the cysts occurring on the extradural components of sacral or coccygeal nerve roots.[2] In a series of 500 consecutive magnetic resonance imaging (MRI) scans of the lumbosacral spine, Paulsen et al[3] recorded an incidence of 4.6%, of which 20% were symptomatic.

If these sacral perineural cysts become large, they may cause symptoms related to local compression. The pathophysiology of these large cysts has been described as a "ball-valve" mechanism that allows fluid to enter but not leave, presumably in a gravitational fashion. The cysts communicate with the subarachnoid space and are therefore filled with cerebrospinal fluid (CSF). Occasionally, the narrow communication channel can become partially or completely occluded with proteinaceous material.[4] The pressure in partially occluded cysts can therefore build and cause local symptoms.

Bartels and van Overbeeke[5] inserted a lumbar–peritoneal shunt in two patients, giving relief of symptoms. This was taken as evidence for the fact that CSF drainage plays an important role in the development of symptoms in patients with Tarlov cysts. This may be manifest as pain in a radicular distribution,[3,6] with or without regional neurologic compromise, or as low back, pelvic, or perineal pain.[7] There are also case reports of back pain and sacral insufficiency fractures resulting from local bony erosion as a result of a Tarlov cyst[8] and of disturbance of micturition.[7]

There are very few data in the literature regarding the natural history of Tarlov cysts, however. A classification system for meningeal cysts exists, but this is based purely on operative inspection and histologic findings and is therefore only retrospective in any individual case.[9] Kunz et al[10] randomly allocated 16 patients with Tarlov cysts into two groups—conservative or operative—and compared the results. Operative treatment gave pain relief in three of eight patients, but none was symptom-free. Conservative treatment yielded similar results, and their recommendation was that surgery should be considered only for those with a short history and with neurologic deficit. Paulsen et al[3] recommended percutaneous computed tomography (CT)–guided aspiration as a means of treatment, with relief of symptoms for 3 weeks to 6 months. Their sample size of five symptomatic patients all had return of symptoms coincident with repressurization of the cysts, however. More recently, the instillation of fibrin glue via a percutaneous route has been described in a small series. Although the patients recovered, there was a one-in-four incidence of aseptic meningitis.[7]

Several authors have reported success with operative treatment of Tarlov cysts. Caspar et al[11] reported an 85% success rate for microsurgical excision combined with duraplasty for patients with symptomatic Tarlov cysts. Voyadzis et al[12] performed sacral laminectomy with cyst resection in 10 patients. Those with large cysts and radicular symptoms (7/10)

Received for publication March 10, 2004; accepted May 14, 2004.
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reported better resolution following surgery than those with small cysts (<1.5 cm) and nonradicular pain. Similar results were obtained by Mummaneni et al,[13] who performed microsurgical cyst fenestration and imbrication on eight patients with large Tarlov cysts causing radicular pain refractory to medical therapy. Four reported good relief of symptoms: Three had moderate recovery, and one had a return of pain 9 months post surgery.

None of the studies in the literature reports significant numbers, and there seem to be no clearly defined criteria for surgical intervention. Our aim, therefore, is to present further detail regarding the presentation of Tarlov cysts and, in particular, to clarify their role in the origin of the symptoms of lumbosacral spinal canal stenosis.

## PATIENTS AND METHODS

Over a 5-year period, data were gathered prospectively from patients who were referred for a specialist opinion for lower back pain, sciatica, or spinal stenosis and who had a Tarlov cyst identified by MRI scan in our institution. A total of 54 patients were identified from a total of 3535 patients who underwent lumbosacral MRI scanning. The prevalence within these patients was therefore 1.5%. Thirty-eight of these (70%) were female. The age range was from 27 to 83 years, with a mean age of 54.4 years. The duration of symptoms prior to consultation varied from a few months to several years.

A record was made of the patients' symptoms, in particular, low back pain, and the distribution of radicular pain or neurologic dysfunction, if present. The MRI scans were reviewed to ascertain the level at which the Tarlov cysts were present. An assessment was made as to whether or not the patient's symptoms could be attributed to the presence of a Tarlov cyst or if they were due to other lumbosacral pathology, for example, intervertebral disc prolapse or degeneration. This follows the usual method of correlating patient symptoms with the specific findings on MRI familiar to spinal surgeons worldwide. Treatment was based accordingly and the response to treatment recorded.

## RESULTS

Figure 1 shows the patients' main presenting complaints. Essentially, these were the typical case mix seen by a spinal surgeon. Several patients had a combination of symptoms (eg, low back pain and leg pain). Treatment modalities included specialist physiotherapy classes with back exercises, lumbar epidural, nerve root block or facet joint injections, and surgical treatment of the major pathology, whether this was the cyst or otherwise. Operative treatment was either stabilization or local decompression of a degenerate spinal segment, simple decompression of a herniated intervertebral disc, or decompression of the cysts themselves. All patients had improved as a result of treatment. Adverse events are described below.

All cysts occurred within the sacral spinal canal. The cysts were often multiple and occurred in a variety of locations (unilateral, central, bilateral, or a combination). The majority of Tarlov cysts (n = 38) are those identified in patients who have lumbosacral symptoms that are not attributable to the presence of a Tarlov cyst. These cysts are relatively small, are often multiple, and may occur bilaterally (Fig. 2). They form the majority of the Tarlov cysts identified within the sample population and are not clinically relevant. Treatment should be directed at the causative lesion, whatever that may be; the Tarlov cysts themselves can effectively be ignored.

In addition, a further subgroup of Tarlov cysts (n = 9) were identified. These could be thought to contribute to patient symptoms by virtue of their anatomic location but are not thought to be the primary cause. These patients have other pathology identified by MRI that is responsible for their symptoms. In essence, these cysts occur at the lumbosacral junction and may occur with, for example, an L5–S1 disc prolapse that compresses the S1 nerve root or facet joint hypertrophy causing lateral recess stenosis. In this setting, the Tarlov cysts could act as an additional compressive agent within an already narrowed spinal canal, for example, due to disc prolapse or acquired stenosis, a sort of "double-crush" lesion. An example of this is shown in Figure 3. Three of these patients underwent local decompression of the affected level, without surgery to the cysts themselves, and made a good recovery; two more recovered with conservative measures (epidural injection and physiotherapy). Subsequent MRI of those who underwent discectomy has shown that the disc itself has been adequately removed, but that there has been no change in the Tarlov cysts themselves, suggesting that the cysts per se were not the cause of symptoms and play no part in the development of compressive symptoms. One patient presented with long-standing low back pain and had marked multilevel degenerative change with a degenerative

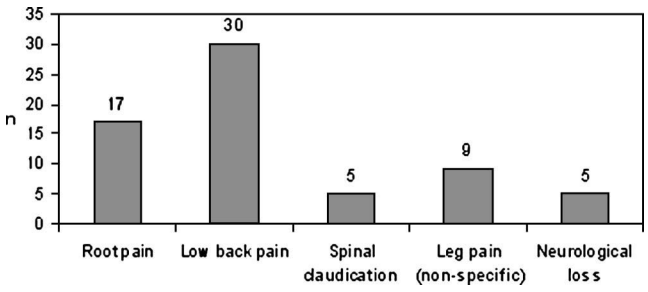


**FIGURE 1.** Patients' symptoms.

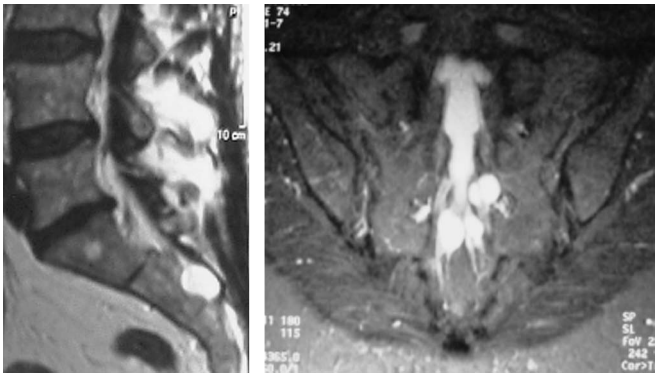

**FIGURE 2.** Examples of small Tarlov cysts.

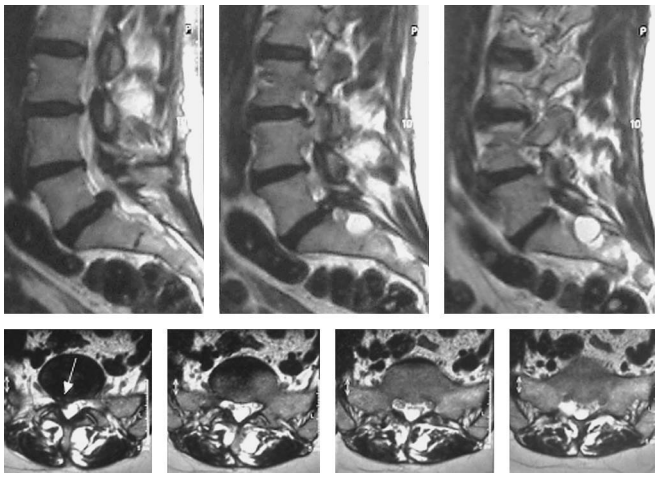

**FIGURE 3.** Sagittal and axial T1 images of a Tarlov cyst with nearby large right-sided paracentral disc prolapse at L5–S1 (arrow).

spondylolisthesis at L4–L5 and a large Tarlov cyst causing sacral ectasia on the left side at S1. Her MRI is depicted in Figure 4. Another patient presented with low back pain, with MRI showing a lytic spondylolisthesis at L5–S1 and large Tarlov cysts causing sacral erosion. Neither of these patients had radicular symptoms, and both reported improved symptoms with physiotherapy alone, suggesting that it was the degenerative disease, not the cyst, that was the cause of symptoms. Two patients presented with radicular symptoms due to acquired spinal stenosis at L4–L5 and L5–S1; MRI also showed cysts at the lumbosacral junction. In these cases, the cysts themselves needed no treatment, but spinal decompression of the affected levels was undertaken in routine fashion with good relief of symptoms. Repeat MRI has again shown that decompression was adequate but that the Tarlov cysts have remained unchanged, confirming that they were not causative.

We also identified several Tarlov cysts (n = 7) that were, by our interpretation, directly responsible for the patient's symptoms. This implies that there was a Tarlov cyst causing pain, either locally or in a radicular distribution, or neurologic dysfunction consistent with the anatomy of the cysts, and no other identifiable pathology recognizable on the MRI scan. This is illustrated in Figures 5–7. In practice, these cysts can be

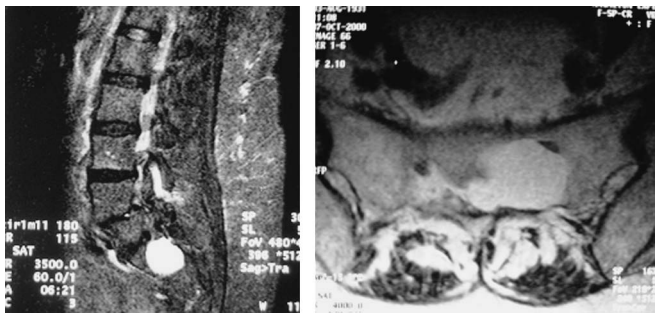

**FIGURE 4.** Sagittal and axial scans showing Tarlov cyst with marked sacral ectasia.

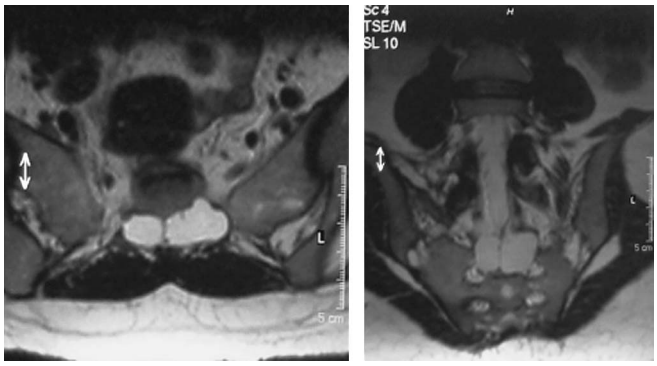


**FIGURE 5.** Multiple large Tarlov cysts.

massive, causing sacral ectasia and/or nerve root compression. We did not encounter any sacral insufficiency fractures. In our series, four of the seven patients in this group declined surgical intervention. Of these, one patient had fluctuating radicular symptoms and was prepared to tolerate them. Her symptoms remain similar after 3 years of follow-up. Another had definite right-sided S1 pain, with a cyst at the right S1 nerve root foramen, but was not interested in surgery (repeat MRI 5 years after initial presentation has shown no change in the cyst). The third had marked sacral ectasia with multiple Tarlov cysts. He also had dural ectasia throughout his lumbar spine. His symptoms were low lumbar/sacral pain. The fourth patient had a right-sided S2 Tarlov cyst with intermittent right S2 nerve root signs but no sphincter disturbance. She has not required

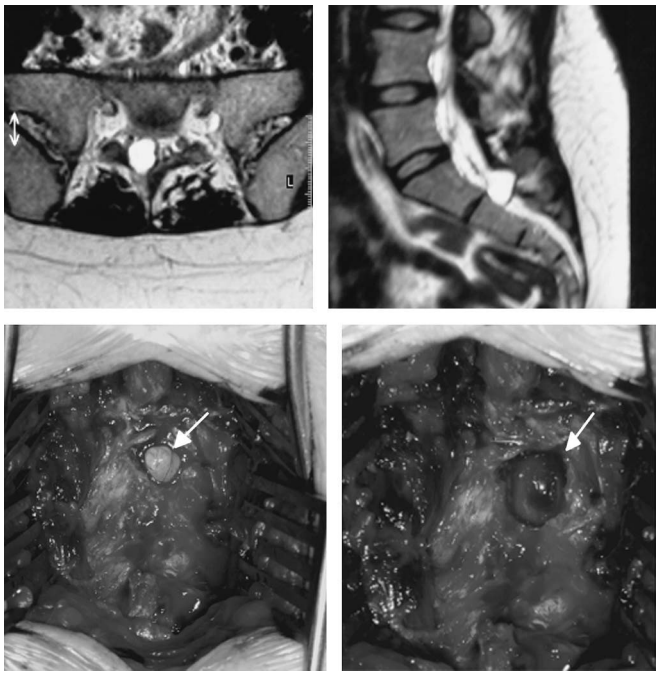

**FIGURE 6.** MRI and intraoperative images of a solitary Tarlov cyst at the right S2 level (arrows). The first intraoperative picture shows the local sacral erosion, the second the cyst once fully exposed.

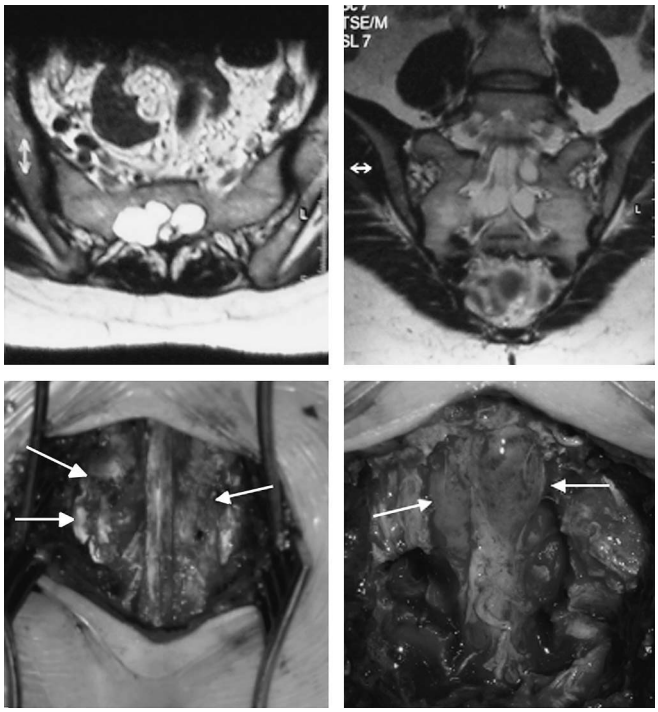

**FIGURE 7.** Axial and oblique scans showing multiple Tarlov cysts at S2 and S3 with corresponding intraoperative images. The image on the left shows sacral thinning (arrows) due to pressure from the cysts; the one on the right shows the cysts fully exposed by sacral laminectomy (arrows). These cysts were noted to be ''valved'' at the time of surgery.

surgery as of yet, and subsequent MRI (2 years post presentation) has shown no further enlargement of her cyst.

Three patients underwent surgery for symptoms solely attributable to their Tarlov cysts. The first presented with low back pain, bilateral sciatica, and intermittent urinary incontinence. Her MRI showed multiple large sacral Tarlov cysts (see Fig. 5), and she underwent surgical decompression in the form of deroofing of the cysts with aspiration of their contents (CSF). Her symptoms improved dramatically postoperatively, but she developed a dural leak on the 6th day post surgery. This was treated with a muscle patch. She has made a good recovery and has achieved full return of normal bladder function and now has no sciatica. The second presented with discrete right-sided S2 distribution pain with a large Tarlov cyst at this level on MRI (see Fig. 6). She underwent surgical decompression (sacral laminectomy and cyst aspiration with muscle patching). Immediately post surgery, she reported a marked improvement in her symptoms but on the second postoperative day developed cauda equina compression due to dislodgement of the muscle patch. She had further surgery to remove the muscle patch from the sacral canal and a new patch fashioned. She has made a good recovery and has noted complete resolution of her symptoms compared with before surgery. Her cauda equina symptoms fully recovered within 6 months of the surgery. The third patient presented with intermittent cauda equina syndrome and marked numbness of both feet. Her MRI showed multiple large sacral cysts (see Fig. 7); these were decompressed surgically, muscle patched, and she made a good recovery with marked resolution of symptoms. She remains symptom-free 1 year post surgery.

Both of the patients with multiple cysts had aspiration of all the visible cysts after sacral laminectomy. It became clear after aspiration that not all the cysts could have a "ball-valve" mechanism as several refilled with CSF within a few minutes. Those that remained deflated by the end of the operation were therefore described as "valved" cysts and those that refilled were described as "nonvalved" cysts. In the patient with the solitary S2 cyst, aspiration proved this to be a valved cyst.

Postoperative MRI scanning at 6 months and 1 year after surgery in all three patients has shown that the valved cysts all remained deflated, whereas the nonvalved cysts had regained their preoperative size. However, review of the MRI films before surgery showed that the valved cysts were more likely to be associated with bony erosion than nonvalved cysts. This implies that it is the increased static pressure within the valved cysts that erodes bone, as a direct application of Wolff law, and not pulsed pressure. There were insufficient patients and operated cysts in this study to come to firm conclusions regarding the likelihood of symptoms arising only from valved and nonvalved cysts, although we think it is likely as the two patients with multiple cysts had good clinical outcomes despite the reaccumulation of fluid in the nonvalved cysts. The patients who have not required or wanted surgical intervention as of yet may be inferred, using the same logic, to have nonvalved cysts. The precise mechanism of their cyst development and the reason for intermittent symptoms remain elusive, however.

## DISCUSSION

Tarlov cysts are a relatively common finding on lumbosacral MRI, with a prevalence of 1–2% in this study. This is slightly lower than that reported in previous studies. Most (70%) are unrelated to the patients' symptoms and require no specific intervention; treatment should be aimed at the underlying pathology, whatever it may be. In some cases, because of the anatomic location of the cysts near an additional pathology (eg, prolapsed intervertebral disc), there may be confusion as to whether or not the cyst is responsible for symptoms. In our series, where none of these cysts was treated, symptom relief was obtained in all cases by treatment of the other pathology. Subsequent MRI showing no change in the cysts despite eradication of symptoms would confirm that these cysts can also be regarded as clinically insignificant.

Tarlov cysts may, however, be the main cause of symptoms. This may be in the form of local pain due to bony erosion, which may cause an insufficiency fracture to develop,[8] or by local nerve root compression. The reasons why some large Tarlov cysts cause symptoms that progress whereas others cause only vague and intermittent symptoms are uncertain, but the "ball-valve" theory has been postulated previously.[4] Those cysts that have free flow of CSF both in and out (nonvalved) are unlikely to cause progressive symptoms, whereas if CSF accumulates under gravitational pressure within the cyst (due to a valve-like phenomenon), the cysts could become larger with time and cause local nerve compression and bone resorption. In our series, three of seven such patients underwent

surgical decompression of their Tarlov cysts, in the form of sacral laminectomy, cyst decompression, and muscle patching. Although this does not eradicate the cysts themselves, it appears to offer symptomatic relief from the effects of nerve root compression. The effect on back pain appears to be less predictable. In addition, our experience would suggest that this surgery is technically demanding and carries a risk of dural leak and dislodgement of the muscle patch. Other authors have suggested extirpation of the cyst and closure of the communicating channel,[11,13] whereas Voyadzis et al[12] suggested that the indication for surgery is size of $>1.5$ cm with radicular symptoms. In our opinion, surgery to excise the cyst carries additional risks of iatrogenic nerve root damage, and if the cyst is valved and therefore a cause of symptoms, simple decompression by aspiration would seem to be sufficient. Our experience would suggest that it is not the size of the cyst per se but its proximity to the nerve root and the presence of a valve mechanism within the cyst itself that predict symptom development and progression.

Our intraoperative experience of cyst decompression by aspiration has shown that the valve theory is a likely mechanism of symptom production and as a result can offer some guidance in terms of treatment. If cysts can be identified as being valved (most probably by CT radiculography), then simple cyst aspiration should be sufficient to treat them. The CT scan would have to be performed immediately after injection of contrast medium, however, as with time, the contrast medium will leak across the arachnoid and at least faintly opacify even valved cysts. Nonvalved cysts, being less likely to cause symptoms, should need no invasive treatment. There should be no need for fibrin glue instillation in valved cysts as these would remain deflated after simple aspiration, and the occurrence of aseptic meningitis after fibrin glue treatment[7] can be understood if it is assumed that the glue was injected into nonvalved cysts and therefore was able to spread into the general intradural space. Since the original study period, we have treated one additional patient with CT-guided aspiration. This patient presented with a symptomatic S1–S2 cyst causing radicular pain. As the fluid was evacuated from the cyst, the patient reported provocation of his symptoms, which abated when the cyst had emptied. The cyst immediately filled with air, and on repeat scan several minutes later, the cyst remained air-filled. From this, we can infer not only that the cyst had not refilled with CSF, but also that no air had leaked into the spinal canal, and thus the presence of a valve mechanism is confirmed. To date, he has had no recurrence of his symptoms.

We did not identify any cases of pathologic fracture in our series, but these have been previously reported.[8] Several patients with large cysts causing subtotal sacral erosion were identified, however. These are currently being monitored with regular clinical review and serial MRI scanning. Currently, there are no guidelines for surgical intervention in these cases. Surgical access to the sacrum involves a wide exposure and carries with it the risk of iatrogenic neurologic injury. In addition, large areas of sacral ectasia would require bone grafting to reduce the risk of insufficiency fracture. This would carry additional risks, both from morbidity from donor sites and from the risk of graft dislodgement and subsequent nerve compression.

In summary, we have identified a series of patients who have had Tarlov cysts identified by MRI scanning. The majority of Tarlov cysts are clinically irrelevant, and in cases where an additional pathology exists, appropriate treatment of this pathology alone is usually sufficient. Our experience of surgical decompression indicates that it is both technically demanding and high risk, but our intraoperative findings support the valved/nonvalved theory for Tarlov cysts. If these can be distinguished by contrast radiography, we would advocate simple percutaneous aspiration as this may well be sufficient treatment and would obviate the need for invasive surgery. At the very least, CT-guided aspiration should be used prior to considering open surgical decompression.

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