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# Conditions of the Spine and Pelvis Presenting with Posterior Hip Pain

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**Abstract**

Many pathologic conditions of the spine and pelvis have posterior gluteal or "hip" pain as a presenting symptom. It is critical that a thorough history is taken as well as a detailed physical examination of the patient. From this information, pertinent imaging studies may be ordered to better direct the physician towards an accurate diagnosis for the patient's symptoms. Because pain in this region can be from many different pathologic processes, it is very important that the evaluating physician considers and evaluate the patient in a systematic fashion to properly work up the patient.

**Keywords:** Spine; hip pain; Posterior hip pain; Tarlov cyst; Low back pain; Causes of hip pain; Neurogenic back pain

## Introduction

The intersection of several body systems in the posterior pelvic region mandates careful attention to the presenting complaint, the aggravating and alleviating factors. Musculoskeletal conditions such as sacroiliac disease, ischiofemoral impingement, facet degeneration and hamstring injuries can usually be differentiated based on history, radiographic evaluation and physical examination. Neurologic conditions such as piriformis syndrome, spinal stenosis, herniated nucleus pulposus and Tarlov and meningeal cysts require MRI (+/- nerve conduction testing) in addition to the aforementioned diagnostics. Vascular claudication may be differentiated from neurogenic claudication by key components of the history, assessment of risk factors and peripheral vascular examination. Ultrasound blood flow studies, CT Angiography and Magnetic Resonance Angiography studies will ultimately be needed for a definitive diagnosis in such cases.

The confusion that is often caused by posterior gluteal pain and its association with various medical conditions is one that has been present as long as we have been treating disorders of the lower back, pelvis and hip joint. A growing understanding of physiologic sagittal balance and pelvic parameters (Pelvic Incidence, Pelvic Tilt) is increasingly bringing attention on how the lower lumbar region and the hip joint are so intimately related [1,2]. As the total lumbar lordosis is decreased (by degenerative changes of the discs or secondary to surgical fusion) the pelvis tends to assume a retroverted posture as a compensatory mechanism. This then reduces the hip's extension in a normal gait cycle thus accentuating conditions such as ischiofemoral impingement.

The radiographic parameters which may best highlight this condition relates to the patient's sacral slope, pelvic tilt and pelvic incidence [3,4]. It is not uncommon for inexperienced practitioners to comment that they have never seen a symptomatic Sacroiliac joint problem, Piriformis syndrome or Tarlov cyst (to name just a few). However, to the expert examiner in each of these fields, the diagnosis can be made and treatment rendered to resolve the patient's complaint in most instances. If the possibility of a rare or unusual condition afflicting a patient is never considered, an accurate diagnosis and appropriate treatment will certainly evade the physician.

The vast majority of the patients who present with posterior hip pain can be accurately diagnosed into one of the more common diagnoses. Among the spinal conditions to consider, spinal stenosis, herniated nucleus pulposus, degenerative disc disease and lumbar facet degeneration represent the vast majority of these cases [5-7]. When there is difficulty in establishing the diagnosis, it is incumbent on the physician to broaden the scope of conditions being considered and to recognize the considerable variability in presentation amongst the conditions on the differential diagnosis list. In order to make an accurate diagnosis, it is often necessary to perform electrodiagnositcs (EMG/NCV) studies and/or diagnostic injections to more definitively establish a diagnosis. The use of these sophisticated techniques may be the only way to differentiate some of these conditions from one another. In the following sections the process of working up and treating the following conditions will be reviewed: Peripheral vascular disease, Lumbar spinal stenosis, herniated nucleus pulposus, facet degeneration/synovial cyst, degenerative disc disease, Sacroiliac joint disease, Piriformis syndrome, hamstring injuries and Tarlov and meningeal cysts.

## Literature Review

### Herniated nucleus pulposus

Herniated nucleus pulposus (HNP) is common cause of low back pain that can manifest as posterior hip pain. Typically, strain leads to a tear of the outer annulus fibrosis. This is usually associated with significant lower back pain. Either simultaneously or more commonly after the onset of the lower back symptoms, disc material (nucleus pulposus) from the central portion of the disc space may protrude through the opening in the annulus resulting in compression of the local neurologic structures. It is not uncommon for the pain in the lower lumbar region to resolve by the time the radicular complaints become more predominant. Depending on the level of the disc herniation, it can cause compression of the conus medullar is in the upper lumbar levels or spinal nerve roots in the mid to lower lumbar levels. The disc can protrude and create compression at that disc level or extrude through the annulus and migrate superiorly or inferiorly. An understanding of the anatomy of the traversing and exiting nerve roots at each level is crucial in understanding the symptomatology.

A foraminal or extraforaminal herniation will compress the exiting

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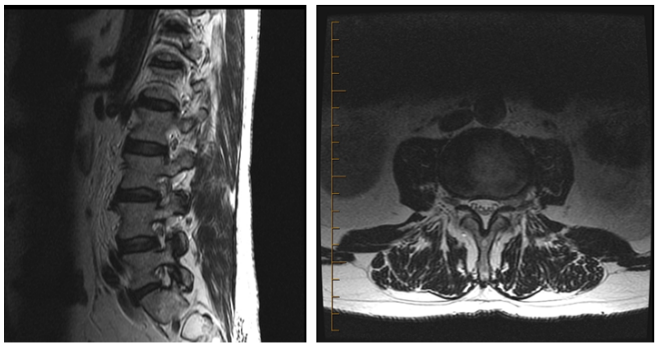
nerve root, so at L3/L4 foraminal herniation would cause compression of the L3 nerve root (Figures 1a and 1b). The more common subarticular or lateral recess herniation typically causes compression of the traversing nerve root, or the nerve root below the corresponding disc level. So at L5/S1 a posterolateral disc herniation would compress the S1 nerve root on the affected side (Figures 2a and 2b). Central disc herniations are uncommon causes of neurologic compression. Typically, they are more associated with axial lower back pain than radicular symptoms. Rarely, a very large central disc herniation may cause compression of the entire cauda equina and cause cauda equina syndrome-a surgical emergency.

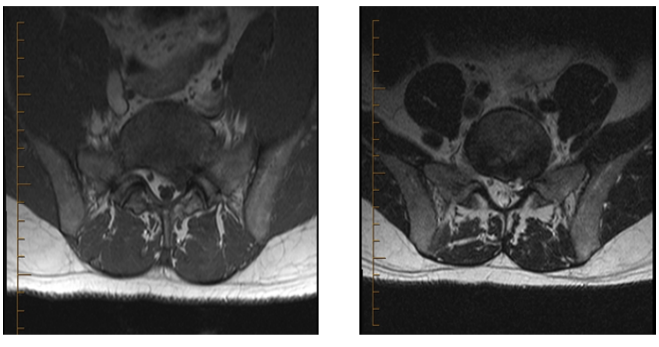
The most common levels to exhibit symptomatic disc herniations with associated radiculopathy are at the L4/L5 and L5/S1 levels however they can occur any level in the spine from C2 to the sacrum. Subarticular disc herniations are the most common location for neurologic compression to occur.

**History:** Patients with a symptomatic herniated lumbar disc will often describe pain after an activity that required a higher level of physical exertion. However, a patient may experience symptoms without a definite preceding event. The presenting symptoms can vary from low back pain to buttock or leg pain that is often exacerbated by sitting or leaning forward, and relieved by standing or back extension. Other symptoms include lower extremity weakness, numbness, or paresthesias. Rarely, cauda equina syndrome can manifest secondary to a herniated disc and includes lower extremity weakness, saddle anesthesia, and/or bowel/bladder incontinence. Most patients with herniated discs will have improvement of symptoms over time as the herniated disc has the capacity to spontaneously resorb. Typically, the larger and more acute the herniation is the more likely it is to resorb [8]. However, these are also the disc herniations most likely to cause acute radiculopathy. Other conditions with a similar presentation are spinal stenosis or synovial facet cysts which often require MRI to differentiate from herniated nucleus pulposus. All of these conditions tend to produce compression of the traversing nerve root.



(A) (B)

**Figure 1:** Foraminal disc hernation (A) A patient with left hip pain demonstrating a left foraminal extrusion at L3/L4 compressing the exiting L3 nerve root with severe left foraminal stenosis. (B) Axial image at L3/L4 demonstrating the foraminal location of the left disc herniation-compressing the left L3 nerve root.



(A) (B)

**Figure 2:** Disc herniation with left hip pain. Images show a L5/S1 left subarticular extrusion compressing the let S1 nerve root. (A) Demonstrates the disc extrusion within the left sub-articular recess at L5/S1 extending inferiorly to the left S1 lateral recess with compression and posterior displacement of the left S1 nerve root. (B) Axial t2 images demonstrating the same with profound t2 hypertensity of the extrusion (high intenzity zone). Note how the extrusion on T2 images can blend in with the epidural fat and how much easier on T1 weighted images in this case the extrusion is seen.

**Physical exam findings:** A detailed physical examination is essential to accurately diagnose a herniated nucleus pulposus as the source of the patient's symptoms. It is very common to identify asymptomatic disc herniations on MRI or CT myelogram. Therefore, a thorough neurological examination is important in order to corroborate the level and side of nerve root compression with the identified pathology on the imaging studies. With a herniated disc, motor exam may yield weakness in a specific muscle group corresponding to compression of a specific level such as weakness of the tibialis anterior with dorsiflexion of the ankle indicating problems with the L5 nerve. Upper motor neuron signs such as hyperreflexia or clonus indicate compression at the level of the spinal cord rather than at the root level. Sensory examination in a dermatomal pattern is very helpful to further delineate the involved nerve root.

Although the classic finding of sciatica with pain radiating all the way down a particular dermatome with associated weakness of the corresponding motor unit is often seen with disc herniations, the clinical presentation can be quite varied [9]. Often times there will be occasional sciatica pain with intervening periods of more "centralized" pain in the lower back and gluteal region. Therefore, posterior hip or gluteal pain may be the chief presenting complaint for many of these patients.

**Imaging:** The easiest and least expensive imaging modality to evaluate back pain is a plain x-ray. The x-ray might reveal loss of disc height in a patient with a degenerative or herniated disc. Standing radiographs will also help to identify subtle instability on flexion/extension or the presence or absence of spinal deformities such as spondylolisthesis, kyphosis or scoliosis. The preferred imaging modality for identification of an herniated nucleus pulposus is an MRI. Intravenous contrast with gadolinium is often helpful in situations where prior surgery has been performed to differentiate epidural scar tissue from recurrent disc herniations. In the lumbar spine having a combination of T1 and T2 axial and sagittal images are necessary. STIR images acquired routinely at most facilities are also very useful and should be acquired on a routine basis [10]. This sequence may be the only sequence to demonstrate signs of peridiscal inflammation or osseous edema. An MRI will show the location and size of the herniated disc, as well as identify other possible causes for pain/radiculopathy such as spinal stenosis or facet degeneration/cyst. However, many asymptomatic patients will have abnormal findings on the MRI as well, so treatment decisions on imaging alone is fraught with error. Any disc herniation's significance must be corroborated with the clinical exam findings [11]. The use of contrast is also helpful when infection or tumor is suspected.

**Diagnostic tests:** Imaging studies such as MRI or CT are the primary diagnostic modality for herniated nucleus pulposus. CT myelogram is

useful in those patients not able to obtain MRI. In general, there is little added information with myelography in patients who can obtain a high-quality MRI and non-contrast CT. Also unlike the cervical spine where the neural compression is most commonly osseous, in the lumbar spine that is not the case, hence making CT myelography far less necessary in the lumbar spine. EMG is often useful in evaluating which nerve root level is affected especially in a patient with a contraindication to MRI such as a pacemaker. Steroid injections can aid the diagnosis of the specific pain generator by relief with injection of a specific nerve root.

**Treatment:** Initial treatment is typically conservative management with the use of NSAIDs, physical therapy, muscle relaxants, or a short course of oral steroids as the symptoms from most herniated discs will resolve with a combination of conservative treatments. If a patient fails these conservative treatments, consideration for an epidural steroid injection or surgical treatment may be warranted. Surgical treatment is reserved for patients with symptoms recalcitrant to conservative management, and surgery has shown improvement in pain and function compared to non-operative treatment. The operative treatment typically performed is a microdiscectomy which includes a laminotomy with discectomy.

## SI joint problems

The sacroiliac joint is a source of mechanical back pain that has potential to be missed by practitioners. SI joint pain is typically a chronic pain in the lower back or gluteal region of the pelvis. Problems with the SI joint can arise from a trauma, pregnancy, or may also be seen in syndromes such as infection, ankylosing spondylitis or Reiter's syndrome. It is believed that long spinal fusions to the sacrum may also result in sacroiliac pain. The imaging findings in such cases may be quite variable.

**History:** Patients with the SI joint as a pain generator typically present with chronic, low back pain without radiculopathy that often has an inciting event such as a fall on the buttocks or motor vehicle collision. A thorough history is important to help aid in the diagnosis of associated syndromes. Ankylosing spondylitis is an auto-immune condition with positive HLA-B27 and may have uveitis associated with the SI joint pain. Also, Reiter's syndrome is a triad of oligoarticular arthritis, conjunctivitis, and urethritis. Other conditions with similar presenting symptoms are facet disease, piriformis syndrome, and degenerative disc disease [12].

**Physical exam:** The physical exam findings in SI joint problems will be pain with typically 3 or more provocative maneuvers. Many maneuvers exist and include palpating the SI joint and also the FABER test by flexing, abducting, and externally rotating the leg which should recreate the pain. Another maneuver is Gaenslen's test done by stressing both SI joints at once by having the patient lie flat on the examination table, flexing one knee to the chest and extending the other leg while it hangs off the table [13].

**Imaging:** An x-ray may show some non-specific changes such as sclerosis, erosions, osteophytes or ankylosis of the SI joint. Because of MRI's sensitivity to bone marrow edema and inflammatory changes, MRI with gadolinium is the preferred method of imaging. MRI will show edema, as well synovial/joint space enhancement and fluid earlier than other methods [14]. CT is useful when looking for abnormalities seen later in the course of the disease, such as sclerosis and ankylosis or when evaluating for subtle erosions (Figures 3a and 3b)

**Diagnostic tests:** SI joint injections with fluoroscopic guidance can not only be therapeutic but diagnostic in cases of SI joint pain. Relief of pain after steroid or local anesthetics into the joint may be helpful in confirming the SI joint as the primary pathologic process. Lab tests can also help with diagnosis such as ESR, CRP, WBC for sacroiliitis, HLA-B27 for ankylosing spondylitis, and RF for the spondyloarthropathies. Patients with ankylosing spondylitis may ultimately autofuse this joint and surgical treatment is not typically indicated.

**Treatment:** Conservative treatment is the first step such as NSAIDs and physical therapy. Continued pain may be improved with corticosteroid injections into the SI joint. The spondyloarthropathies may also require TNF inhibitors. For infection, IV antibiotics are warranted with a transition to oral antibiotics once symptoms improve. Pharmacologic treatment for the various spondyloarthropathies is beyond the scope of treatment covered in this segment. Many new medications which modulate the immune response are being used in this capacity. Rheumatologic evaluation should be advised in such cases to direct treatment. If surgery is needed for anyone on these immune modulating medications—careful attention must be given to stopping and restarting these medications in a timely fashion before and after surgery to minimize the risk of postsurgical infection.

## Facet disease/Synovial facet cyst

Facet disease is another generator of low back pain. Facet arthritis is a very common cause of low back pain as the cartilage within the joints may degenerate (Figures 4a and 4b) Also, facet cysts filled with synovial fluid from the facet joint can form and cause some compression of the traversing neurologic structures. Lumbar instability on flexion and extension is commonly identified amongst patients with a synovial cyst. Standing x-rays and flexion/extension views should routinely be performed as it may have implications on the ultimate treatment.

**History:** Patients with facet arthritis will most likely have some component of low back pain and will typically present in the 5th or 6th decade of life or later. The pain typically has a mechanical component: worse with more vigorous activity and alleviated by rest [15]. Synovial cysts occur in a broader age range and can have both axial lower back pain and radicular pain as a presenting complaint. Facet cysts may wax and wane with respect to their size based on the level of the patient's activity. Thus the symptoms associated with synovial cysts may be better or worse at times as the cyst changes in size/shape.

**Physical exam:** Neurologic exam may reveal tenderness to palpation of the low back near the specific arthritic facet joints. Facet syndrome typically exhibits pain with extension of the lumbar spine as the spine preferentially loads the posterior elements in extension. Pain may also be seen with lateral bending towards the affected facet joint for the same reason. Careful neurologic examination should be performed to identify the precise nerve root which is compressed.

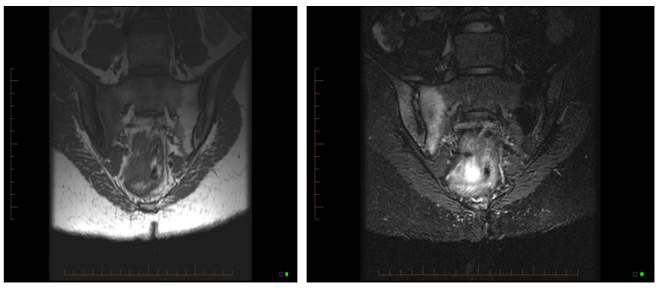



**Figure 3:** T1 coronal image showing areas of diminished signal within the sacrum and iliac bone along the right sacroiliac joint. (A) T1 hyperintensity of the left sacrum likely is secondary to postinflammatory fatty infiltration. (B) STIR coronal image showing the increased signal on the right about the sacroiliac joint typical of sacroilitis.

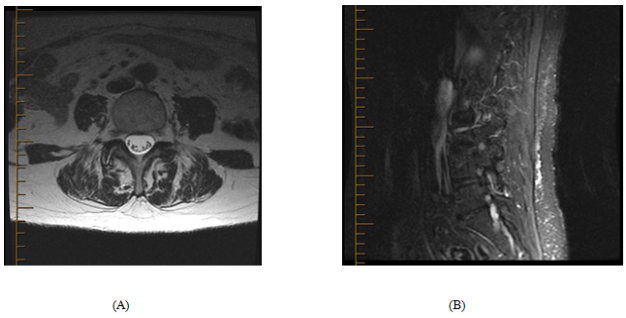


**Figure 4:** Facet degeneration (A) T2 axial image showing right facet effusion with some facet hypetrophic changes. (B) Sagittal STIR showing facet effusion with mild subjacent osseous edema.

**Imaging/diagnostic tests:** X-rays can aid in the diagnosis of facet arthritis with the facet joints appearing sclerotic and hypetrophied on both PA and lateral radiographs. MRI is the most useful imaging study of the facet joints. Differentiating disc herniation from facet synovial cyst can only be accurately accomplished with MRI. Also correctly identifying facet synovitis by visualizing facet joint edema, synovial fluid/thickening, and perifacet inflammation is frequently only conspicuous on MRI rather than CT or XRAY. (Figures 5a and 5b) MRI with contrast, in this scenario can improve the sensitivity to facet joint inflammation demonstrating enhancement within and around the facet joint.

**Treatment:** Conservative management is the mainstay of treatment for both facet degeneration and synovial cysts [16]. Arthritis may be improved with NSAIDs and physical therapy, and possibly ultimately with corticosteroid injections. Patients who experience substantial pain relief with corticosteroid facet injections may find better and longer lasting relief with radiofrequency ablation of the nerve which innervates the facet joint complex. Facet cysts may resolve over time, but if they do not regress with time and continue to cause symptoms, then surgical excision may be warranted to remove the cyst. A synovial cyst without associated spondylolisthesis may be treated with just a laminotomy to gain access to the cyst and cyst removal [16]. Larger cysts and cysts found in conjunction with spondylolisthesis may require more expansile decompression and fusion to prevent postsurgical instability or cyst recurrence.

## Hamstring injury with avulsion

Hamstring injuries when associated with avulsion can cause posterior hip pain. Most hamstring injuries occur at the myotendinous junction; however, the hamstring can avulse the ischial tuberosity. The hamstring includes the semitendinosus and semimembranosus, and biceps femoris muscles.

**History:** The patient may give a history of participating in physical activity such as sprinting or water skiing during the time of injury. They may describe feelings of a "pop" during exercise with bruising and swelling afterwards.

**Physical exam:** Upon physical exam, there may be extensive ecchymosis and edema as well as a palpable mass in the posterior thigh. Often times the patient will have difficulty walking or running immediately after the injury.

**Imaging:** Plain x-rays of the pelvis may show an avulsion at the ischial tuberosity with avulsed bone fragment. However, this is less common in adults as opposed to adolescents where the apophysis will avulse due to lack of complete ossification. MRI is the exam of choice. Not only will it show the avulsed tendon but it will better show the degree of tendon retraction (Figure 6).

**Treatment:** A hamstring injury with rupture at the myotendinous juncture warrants limited weightbearing status for a few weeks with stretching and strengthening to follow. An avulsion injury may warrant operative repair of the hamstring tendon to the ischial tuberosity with suture anchors. The decision to operate depends on patient demands, extent of injury and disability.

## Lumbar spinal stenosis

Spinal stenosis of the lumbar spine is typically a multifactorial condition that results in narrowing of the entire spinal canal. Impingement of the spinal canal is typically caused by a combination of facet hypertrophy, disc bulge and thickening of the ligamentum flavum. It is a common cause of posterior hip pain. The incidence of spinal stenosis increases significantly after age 60. Spinal stenosis is nearly always present in the face of a degenerative spondylolisthesis. When a spondylolisthesis is present the stenosis will usually affect the subarticular recess preferentially. Many authors support a multifactorial etiology in the progression of lumbar stenosis. Disk degeneration alters mechanics and loading of facet joints. The stress on the facet joint leads to hypertrophy of the facets, joint capsule, and ligamentum flavum, all of which result in decreased space in the spinal canal. Placing the lumbar spine in extension causes the ligamentum flavum to buckle and further decrease the spinal canal diameter. Thus, patients have increased symptoms when in extension.

**History:** Patients with lumbar spinal stenosis often complain of lower back pain, buttock and leg pain, pseudoclaudication, standing discomfort, numbness and lower extremity weakness. These symptoms are usually exacerbated with walking, standing, descending hills and going down stairs. Their pain will start proximally and proceed distally. Patients usually report relief of symptoms with changes in position (sitting down, leaning forward). Patients' buttock and leg pain is usually unilateral neurogenic pain that can radiate down the leg. Pain is not commonly located in the groin, as is common with hip pathology. Other symptoms can include bladder disturbances and cauda equina syndrome. Neurogenic claudication can be distinguished from vascular claudication in that it is typically worse with extension of the back and relieved with flexion of the back whereas vascular claudication is exacerbated by activity not position. See the section on vascular claudication for further distinguishing features.

**Physical exam:** Patients with lumbar stenosis will tend to have a normal physical exam. They will usually exhibit focal tenderness to palpation in the lumbar spine that correlates with the location of impingement. Patients may also exhibit lower extremity weakness

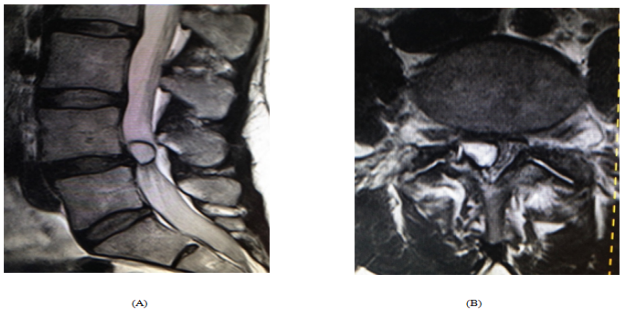


**Figure 5:** Syonvial Cyst causing spinal stenosis. (A) Sagittal T2 image showing the synovial cyst at the L4/L5 disc level. (B) Axial T2 MRI image at L4/L5 disc level. Right sided synovial cyst from the right L4/L5 facet joint. This is typically found in the setting of subtle spondylolisthesis such as in this case.

that follows a segmental pattern in accordance with which nerve root is being impinged. Patients generally will not have tenderness to palpation in the buttock or groin. Symptoms will not be reproduced with passive hip ROM. If vascular claudication is suspected, ABIs are appropriate. Similar Presenting Conditions As highlighted in the background section, lumbar stenosis may be caused by a wide variety of conditions. The symptoms that are common to these conditions can also be present in other conditions such as vascular claudication, hip osteoarthritis, avascular necrosis of the femoral head, piriformis syndrome, trochanteric bursitis, Tarlov cysts, facet cysts, sacroiliac joint disease, and peripheral neuropathy.

**Imaging:** Proper imaging is critical to the diagnosis of lumbar spinal stenosis. Imaging studies should include AP, lateral and oblique radiographs, flexion-extension radiographs, MRI and/or CT myelogram. It is very important to obtain standing lumbar radiographs. On the standing x-rays the following issues should be specifically reviewed: standing posture, presence or absence of spondylolisthesis or instability on flexion and extension. It is not unusual that MRI or CT will miss a subtle spondylolisthesis due to the recumbent position that the images are obtained. Oblique radiographs may be helpful to identify pars lesions or spondylolysis. From cross sectional images MRI is again the diagnosis of choice. The stenosis will be visible, but the dominant cause of the stenosis (disc, facet, ligamentum flavum, epidural lipomatosis) and where the stenosis is located will be visible. (Figure 7) CT myelography is beneficial in those patients in whom MRI cannot be obtained, but myelography is not a simple or straightforward procedure in patients with severe spinal stenosis.

## Special diagnostic testing

EMG is a useful tool to differentiate lumbar spinal stenosis from peripheral neuropathy or piriformis syndrome. Ankle Brachial Index should be part of the assessment if vascular claudication in suspected.

**Treatment:** Non-surgical interventions for lumbar spinal stenosis have marginal long term efficacy [17,18]. They include drug therapy, physical therapy and epidural steroid injections. Drug therapy should consist of acetaminophen and NSAIDs initially. Narcotics and muscle relaxants should be used sparingly and only on a short term basis. Physical therapy should focus on core strengthening, lumbar stabilization, flexibility and aerobic conditioning. Epidural steroid injections can provide pain relief for patients on a short term basis, but they have not been shown to change the natural progression of disease or decrease the need for future surgical intervention [19].

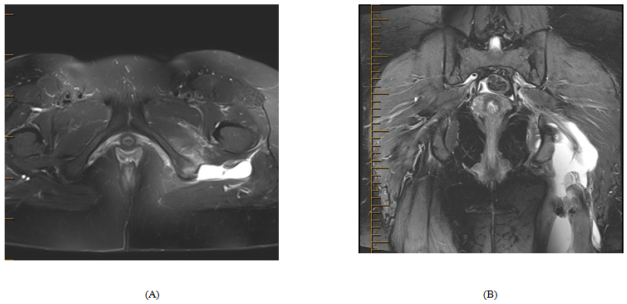


**Figure 6:** Hamstring avulsion injury. **(**A) Axial STIR image showing absent left hamstring tendon at the level of the ischial tuberosity with edema and hemorrhage within the subjacent soft tissues. Edema is also seen in the left ischial tuberosity. (B) Coronal STIR image shwong the complete avulsion on the left with distal retraction of the tendon (arrow) and extensive surrounding hematoma.

Patients whose symptoms are recalcitrant to non-surgical treatment regimens are candidates for surgical intervention. The mainstay of surgical intervention for lumbar spinal stenosis is direct nerve root decompression via laminectomy or laminotomy. Spinal fusion surgery is generally not indicated for patient with spinal stenosis without instability or other spinal deformity (i.e., scoliosis). Indirect nerve root decompression via distraction of the disc space or other interspinous process device (Coflex, X-stop) may be reasonable in some selected cases. However, the results of the SPORT (Spine Patient Outcomes Research Trial) study demonstrate the very good and reproducible outcomes that may be achieved via lumbar laminectomy [20-26].

## Degenerative disc disease

As patients age, the composition of vertebral discs is altered. The number of viable cells decreases, as does the amount of proteoglycans within the nucleus of the disc space. The decrease is proteoglycans leads to a loss of hydration within the disk. The resultant structural changes include an expansion of the inner layers of the annulus and decreased disk height and ultimately lead to altered biomechanics (as previously discussed in the lumbar spinal stenosis section). These degenerative changes are termed degenerative disc disease (DDD). DDD causes an increase in mechanical load and stress on the ligaments and facet joints around the degenerative disc. This leads to abnormal motion and hypertrophy of the affected facets. Common comorbidities associated with DDD include diabetes mellitus, vascular insufficiency, and smoking; however no causal link has been established.

**History:** The primary manifestation of DDD is back pain without radiculopathy. Patients will complain of midline pain in the lumbar spine that can usually be well-localized. This pain may be exacerbated by axial loading, sitting and bending. It is also important to differentiate whether the pain is acute or chronic. Acute low back pain is described as lower back pain of less than 3 months duration that is functionally limiting. It is not usually accompanied by neurologic symptoms, is usually self-limiting and typically resolves within a short time frame. EMG and other nerve conduction studies generally are not warranted. While most causes of acute low back pain resolve over time, some acute pathology requires immediate attention. These include metastatic disease, infection, vertebral fracture, cauda equina syndrome, herniated nucleus pulposus. As such, it is imperative that a thorough history include questions of cancer history, unexplained weight loss, fever, recent sickness or infection, changes in mental status, recent trauma or fall, urinary retention, saddle anesthesia, numbness, tingling and neurologic deficit. Positive findings to any of these lines of question should alert the provider to investigate further.

Evaluation of chronic low back pain can be multifaceted and

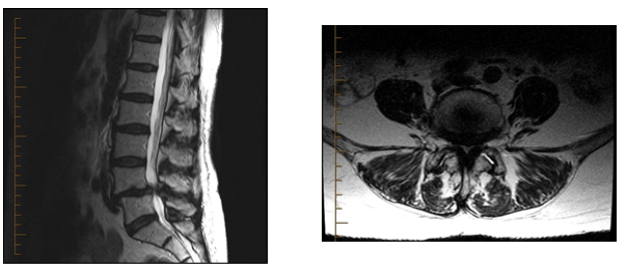


**Figure 7:** (A) and (B) Spinal stenosis: Sagittal and axial T2 images showing spondylolisthesis and severe spinal and sub-articular stenosis at L4/L5 from a combination of spondylolisthesis, disc bulge, facet degeneration and ligamentum flavum buckling.

nuanced. In general, pain should be localized to a specific region of the spine and the patient should be asked to distinguish the nature of the pain. As with acute low back pain, chronic low back pain must be evaluated for pathology that requires immediate attention, such as fracture, neoplasm or infection. It is also critical to screen for underlying psychiatric disease, secondary gain and inconsistencies in history and exam findings.

**Physical exam:** On physical exam, patients with DDD will demonstrate midline tenderness to palpation over specific areas of the lower back that correspond to the levels where DDD changes are occurring. It is important to differentiate this mechanical pain from myofascial pain. They may also have a decreased or painful range of motion. Tension signs such as straight leg testing and bowstring testing will usually be normal.

**Similar presenting conditions:** As discussed in the history section, metastatic disease, infection, vertebral fracture, cauda equina syndrome, and herniated nucleus pulposus can all mimic some of the symptoms of DDD. In addition, there are many abdominal conditions that can present with low back pain as a primary symptom. These include abdominal aortic aneurysm, renal colic, pelvic inflammatory disease, urinary tract infection, and retrocecal appendicitis.

**Imaging:** Radiographs may show disc height loss or may be normal. If DDD is suspected, MR is again the imaging study of choice. Characteristic findings of DDD on MRI would include disk height loss and a low signal (black disc) on T2 weighted image without significant herniation or stenosis. As progressive pathologic disc degeneration ensues, osteophyte formation, intradiscal/nuclear gas, annular tears/high intensity zones, and Modic changes in the subjacent vertebral bodies can be seen though not necessarily in that order. At this point the findings are starting to move beyond the realm of normal (aging related) degeneration and towards pathologic degeneration (internal disc disruption).

## Special diagnostic testing

Provocative discography has been used as a means of confirming DDD as the source of low back pain. A positive test shows concordant pain response, shows abnormal disc morphology on fluoroscopy, and has negative lumbar spine control levels. While diagnostically accurate, discography is somewhat controversial. Possible injury to the disc at control levels and the subjective nature of how the results are interpreted by the examiner have been some of the shortcomings of this technique.

**Treatment:** Non-operative management includes rest, activity modification, NSAIDs, muscle relaxants, bracing and physical therapy focusing on stretching, strengthening and weight control. Other interventions that have shown benefit include behavioral modification, smoking cessation and activity modification. Immediate surgical intervention is necessary for cases of neoplasia, infection, fracture, herniated nucleus pulposus, or in patients who develop neurological deficits including those with cauda equine syndrome. In such cases, surgical intervention should be tailored to treat the specific cause and will not be covered in detail in this section. Surgical management of confirmed DDD should only be pursued if the patient has had persistent symptoms that do not resolve with a six-month regimen of non-operative treatment as previously described. Options for surgical intervention include lumbar discectomy and fusion, transforaminal interbody fusion or lumbar total disc replacement.

## Piriformis syndrome

Compression of the sciatic nerve after it leaves the pelvis can cause posterior hip and leg pain known as piriformis syndrome. This entrapment can occur anterior to the piriformis and posterior to the gemelli and obturator internus at the level of the ischial tuberosity [27,28].

**History:** Patients with piriformis syndrome will complain of posterior gluteal pain that migrates down the back of the leg that is often described as burning or aching in nature.

**Physical exam:** Patients with piriformis syndrome can demonstrate tenderness to palpation lateral to the ischial tuberosity and/or weakness or diminished sensation in the lower leg. The definitive test for piriformis syndrome is conducted by passively placing stress on the piriformis and short external rotators. This is known as the FAIR test due to the proper positioning (Flexion, Adduction, Internal Rotation). The FAIR test is positive when placing the patient in this position leads to a reproduction of symptoms [29,30].

**Imaging**: In patients with piriformis syndrome radiographs are usually negative. Initially MRI of the pelvis was thought to also bear little additional information but has now proven to be a useful test. Findings frequently seen include piriformis enlargement (of multiple causes), muscle inflammation and scaring, and congenital muscular variants of size and attachment (Figure 8). Lumbar MRI is helpful in ruling out spinal causes of nerve compression that could mimic a lower entrapment neuropathy.

## Special diagnostic testing

Electrodiagnostic studies such as EMG and nerve conduction

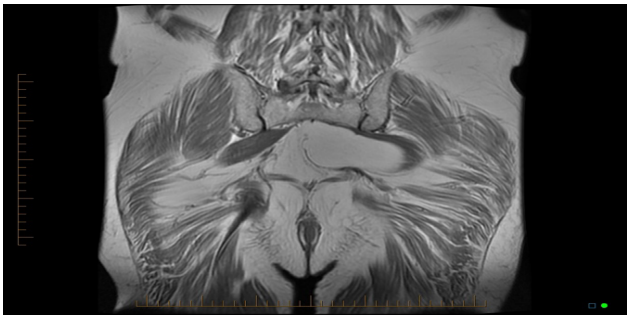


**Figure 8:** T1 coronal image showing a left piriformis lipoma (arrow) in some one presenting with left piriformis syndrome.

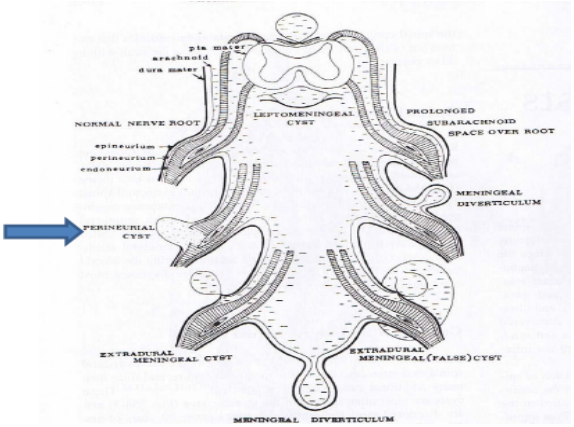



**Figure 9:** Image from the original publication by IM Tarlov describing different types of meningeal cysts in the sacrum arising from the spinal sac, including perineurial cysts (arrow), which were later named after him [37].

studies are useful in the diagnosis of piriformis syndrome and may demonstrate functional impairment of sciatic nerve [31]. However, negative results do not exclude piriformis syndrome as the diagnosis.

**Treatment:** Non-operative management includes rest, NSAIDs, muscle relaxants, physical therapy focusing on stretching the piriformis muscles and short external rotators, and corticosteroid injections directed near the piriformis muscle. Surgical management is indicated only in refractory cases without response to conservative treatment and consists of piriformis muscle release or external sciatic neurolysis.

## Peripheral vascular disease

Arterial Insufficiency is due to narrowing of arteries in the lower extremities. Atherosclerosis leads to a narrowing of the arterial blood supply, such that when a patient has an increased oxygen demand (during exercise or walking for example) the diminished blood supply is not able to keep up with oxygen demand from the muscle tissue, resulting in intense pain and cramping of the legs. The popliteal artery is the most common location of arterial insufficiency because it is the smallest diameter artery that is still a common location for atherosclerotic build-up. Incidence of arterial insufficiency increases with age. Most patients will be over fifty at first onset of symptoms. Hypertension, diabetes, smoking, and obesity are strongly correlated with arterial insufficiency [32].

**History:** The hallmark presenting symptom of arterial insufficiency is "vascular claudication." Claudication is defined as cramping activity-related leg pain. It has insidious onset over many years and leads to decreased activity levels. Patients relate that they are no longer able to walk for as long as they used to due to cramping pain in their feet, calves and thighs. Patients report relief of symptoms with rest. Patients can also present with numbness and tingling in their feet. Symptoms can be unilateral or bilateral. In earlier stages, the symptoms are unaffected by changes in position. However, as disease advances, even standing can cause symptoms.

**Physical exam:** Patients with arterial insufficiency can present with diminished or absent distal pulses, cold extremities, cyanosis, or atrophy in the affected limb. Atrophic changes can include loss of hair, atrophied muscles and shiny skin. ABIs will be diminished in the affected limb.

**Similar presenting conditions:** It is important to distinguish between neurogenic claudication and vascular insufficiency as the source of pain in patients who present with claudication as their chief complaint. In general, vascular claudication is worsened with activity while neurogenic claudication is worsened with position. Thus, a patient who relates relief of symptoms with decreased activity and notices no difference with postural changes is more likely to have vascular insufficiency. Conversely, extension of the lumbar spine closes down the area available for the spinal cord and increases neurogenic claudication. Thus, it is worsened by standing, walking upright, and walking downhill, but activity that places the back in flexion, such as walking downhill, riding a bicycle, or leaning over while pushing a shopping cart will improve symptoms. Another determining factor in differentiation is the peripheral exam. Vascular claudication is more likely to present with abnormal pulses, skin changes, cyanosis and cold extremities.

**Imaging:** Doppler ultrasound is the gold standard for initial non-invasive evaluation and readily identifies stenosis and occlusion of the vasculature. CT Angiography and MR Angiography is now routinely used for characterization of the location and degree of stenosis. Direct catheter angiography and arteriography can be useful for patients requiring more detailed surgical evaluation. Treatment Non-operative management includes lifestyle modification and control of chronic medication conditions such as hypercholesterolemia, hypertension, and diabetes mellitus. Operative management includes angioplasty, stenting and arterial bypass grafting. Amputation often becomes necessary as a salvaging procedure in patients with uncontrolled diabetes.

## Other musculoskeletal conditions/considerations

This chapter will not specifically address other conditions such as complications secondary to total hip arthroplasty or hip resurfacing. Ischiofemoral impingement and other intra-articular conditions of the hip joint are discussed elsewhere and are beyond the scope of this chapter.

## Tarlov cysts

Tarlov (perineurial) cysts are one of several distinct types of spinal meningeal cyst commonly found in the lumbosacral region [33] (Figure 9). They share the potential to cause symptomatic spinal nerve root compression and bone remodeling presenting as sacral or posterior hip pain. Unfortunately, knowledge concerning these cysts has historically been limited, and past teachings have been to avoid them at all costs. This most likely stems from their reputation for poor surgical outcomes and a high rate of complications, particularly cerebrospinal fluid (CSF) leakage. Though not a prerequisite for causing symptoms, Tarlov cysts can sometimes be radiologically impressive in size and extent (Figure 10). These factors, along with the complexity of the presenting symptoms, can combine to pose a daunting treatment quandary, even for the most seasoned spine surgeon or pain management practitioner alike. Unfortunately, when the diagnosis of symptomatic Tarlov cysts is missed and standard evaluations for hip and SI joint pathology are unsurprisingly normal, patients are often relegated to endless pain clinic and physical therapy, or simply told that there is nothing wrong and that their problem is psychological. Still worse, patients can be misdiagnosed with other orthopedic, spinal, gynecological, urological or gastroenterological disorders and subjected to an endless array of unneeded interventions. Topping this list are misguided SI joint fusion, an assortment of spinal surgeries, hysterectomy (often while still of child-bearing age), exploratory laparotomy, and bladder procedures (Figure 11). This section is therefore intended to give the reader an understanding of sacral Tarlov cysts and their presentation so that misdiagnosis can be prevented and patients can receive appropriate care.

**Pathophysiology:** As defined by IM Tarlov himself, a Tarlov (perineurial) cyst is a dilation of a spinal nerve root arising proximal to the dorsal nerve root ganglion [5] (Figure 9). Although the term was originally intended to describe perineurial cysts alone, in current

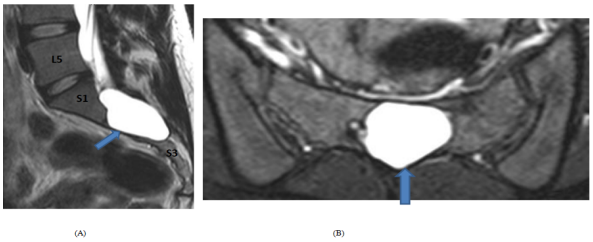



**Figure 10:** A large sacral Tarlov cyst is seen on sagittal. (A) Axial (B) T2-weighted MRI. The cyst fills the entire spinal canal from S1 to S3, compressing the nerve roots of the sacral cauda equina. The bone remodeling caused by the cyst is extensive, with the S2 vertebra almost no longer visible on the sagittal image. In fact, the cyst appears to have penetrated completely through the back of the sacral lamina dorsally on the axial view.

practice it is often misapplied to other types of spinal meningeal cysts due to their similar radiographic appearance. The exact mechanism leading to Tarlov cyst formation has not yet been elucidated. By some mechanism, spinal fluid enters and accumulates within a nerve root, either as a result of being trapped there, or due to laxity in the dura of the nerve root sleeve. Fluid accumulation in the nerve root then causes it to balloon, sometimes to the point of producing symptoms by compressing adjacent structures. Although they can form at any spinal level, symptomatic Tarlov cysts are most commonly found affecting the sacral nerve roots [33]. They most often involve the portion of the nerve root in the spinal canal, but can also develop anywhere along the course of a spinal nerve, including in the neural foramen and the retroperitoneal pelvis (Figure 12).

**Symptoms:** Tarlov cysts can cause symptoms by producing mechanical compression of adjacent spinal nerve roots in the spinal canal. In the sacrum, this can result in a corresponding pattern of sacral radiculopathy symptoms (Table 1). The variety of symptoms displayed and their laterality depends on the location of the cyst/s and the extent to which they are compressing adjacent nerves. Compression of the S1 and S2 nerve roots typically produces sacral pain radiating to the buttock, posterior hip, and down the back of the leg to the bottom or lateral aspect of the foot. Numbness in a similar distribution and weakness in plantar flexion and the intrinsic muscles of the foot are also often present.

Sacral and posterior hip pain almost always limits the ability to sit, with patients constantly squirming in their chair and avoiding seated activities. Interestingly, patients often adopt curious seated postures, constantly leaning far to one side in order to decrease direct pressure on the sacrum or hip. They also tend carry special cushions with them to sit on for similar reasons despite the awkward nature of carrying a cushion or pillow in public.

Compression of S2, S3 or S4 can produce perineal pain and numbness. Neurogenic bladder symptoms are not uncommon, with the patient experiencing urinary urgency and frequency, as well as urinary retention requiring the patient to Valsalsva, or press on their abdomen (Crede maneuver) in order to empty their bladder completely. They can also have neurogenic bowel symptoms, with constipation requiring extensive laxative use or manual assistance to have a bowel movement. Some also describe the loss of sensation to know when to empty their bladder or bowel. Patients frequently describe dyspareunia, or painful intercourse, due to their perineal pain, or sexual dysfunction due to the loss of perineal sensation. The presence of sacral or posterior hip pain in combination with the above sacral radiculopathy symptoms should prompt an evaluation for Tarlov cyst or other pathology affecting the sacral nerve roots.

**Imaging:** MRI is currently the best modality for diagnosing spinal meningeal cysts, such as Tarlov cysts. The water content in the cerebrospinal fluid makes them stand out, particularly on T2-weighted imaging. Since Tarlov cysts arise from spinal nerve roots, they tend to be found laterally in the spinal canal, as opposed to centrally, which is more typical of other cyst types. Since each Tarlov cyst is a nerve root, it contains neural elements and a careful search can sometimes reveal the fascicle bundle within a Tarlov cyst, particularly on axial images (Figure 13).

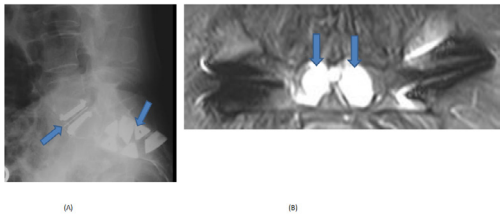


**Figure 11:** The diagnosis of symptomatic Tarlov cysts was missed in this patient with posterior hip and sacral symptoms. Instead, she underwent artificial disc placement at L5/S1 (left arrow) and bilateral sacroiliac joint fusion procedures (right arrow), whose hardware can be seen on lateral x-ray of the pelvis. (A) Despite the flanking presence of metal artifact from the patient's sacroiliac fusion procedures, an axial MRI of the sacrum (B) reveals two large Tarlov cysts filling the spinal canal (arrows), which turned out to be the true source of her symptoms.

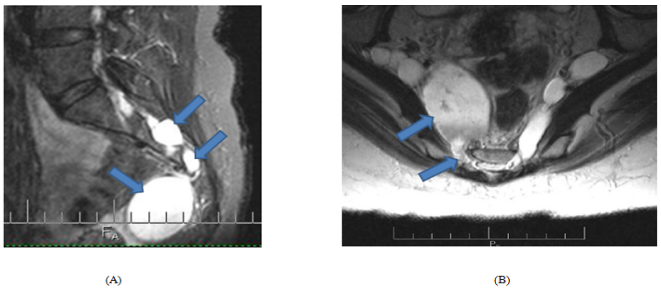


**Figure 12:** A Tarlov cyst can develop along any portion of a spinal nerve root throughout its course, as seen in this patient with multiple cysts. On sagittal MRI (A) Tarlov cysts are seen in the spinal canal (upper arrow), in the foramina exiting the sacrum (middle arrow), and in the retroperitoneal pelvis (lower arrow). On axial MRI (B) both the intra-foraminal (lower arrow) and retroperitoneal (upper arrow) portion of the same S3 nerve root have become cystic. The contralateral S3 nerve root is also cystic in appearance.

| Common symptoms due to sacral tarlov cysts |
|---|
| Sacral pain radiating to the hip, buttock, and down the back of the leg to the bottom of the foot |
| Numbness or tingling in a similar distribution |
| Weakness in plantar flexion and the intrinsic muscles of the foot |
| Perineal pain and numbness |
| **Neurogenic bladder symptoms** |
| Urinary urgency, frequency, and retention with the need to Valsalva or perform the Crede maneuver in order to empty completely |
| **Neurogenic bowel symptoms** |
| Constipation requiring the extensive use of laxatives or manual facilitation |
| Dyspareunia (painful intercourse) |
| Sexual dysfunction |
| Inability to tolerate sitting |

**Table 1:** Common symptoms due to sacral Tarlov cysts.

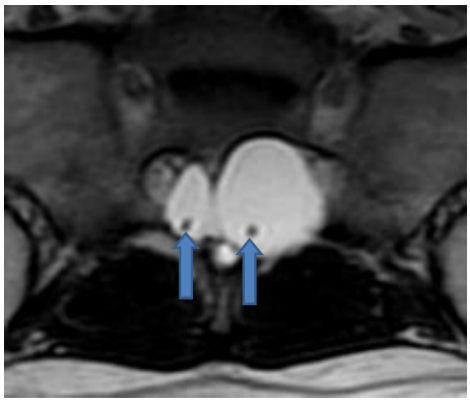


**Figure 13:** Two Tarlov cysts are seen side-by-side filling the spinal canal in the sacrum on axial MRI. The spinal fluid filling the cysts appears bright. Each cyst is a spinal nerve root containing a nerve fascicle bundle that is clearly seen (arrows).

Tarlov cysts can be single, or multiple can be present in the same patient (Figure 14). The likelihood of causing symptoms increases with the number of cysts, as their cumulative effect is to occupy a greater volume of the spinal canal typically reserved for the spinal nerve roots alone. In other words, multiple small cysts in the same area of the spinal canal have the same compressive effect as one large cyst of the same overall volume.

MRI also allows optimal visualization of the surrounding neural structures, such as adjacent spinal nerve roots under compression (Figure 15). These relationships are critical for diagnosis, particularly when the laterality and distribution of a patient's symptoms can be correlated with a specific nerve root that is observed on MRI to be blatantly compressed by a Tarlov cyst. These relationships are also obviously important for surgical planning purposes. The addition of contrast to an MRI is useful for identifying other potential pathologies affecting the sacral nerve roots, such as tumors, including spinal schwannomas, neurofibromas, and meningiomas.

Although patients with larger cysts tend to have better surgical outcomes [34], it is not necessary for a cyst to be "big" to cause symptoms. Instead, it is more important to know its location and which nerves it is compressing. If the radiographic appearance of a Tarlov cyst can be correlated with the laterality and distribution of symptoms, then the cyst is suspect, regardless of its size. Although one's attention when reviewing a lumbar MRI is easily distracted by a radiographically impressive sacral cyst, it is critical to review the entire imaging study for other potential sources of sacral and hip symptoms. A search for alternate pathology that might explain symptoms should also be made higher up in the lumbar spine, towards the sacroiliac joints, and in the pelvis, particularly as it relates to the lumbosacral plexus. Further clues can sometimes be found suggesting the presence of sacral nerve root dysfunction. For example, an abnormally distended bladder may reflect neurogenic urinary retention caused by a Tarlov cyst compressing the sacral nerve roots (Figure 16).

CT scanning is of limited value in diagnosing Tarlov cysts since neural structures are not visualized. In certain instances, it can be useful for delineating the extent of bone remodeling/erosion caused by a cyst, or for identifying painful sacral insufficiency fractures (Figure 17). CT myelography is a lesser alternative to MRI, but may be necessary when patients harbor non-MRI compatible hardware. However, the appearance of spinal meningeal cysts is variable and unreliable on CT myelography when compared to MRI. This is particularly true when the penetration of spinal fluid into a cyst is very slow and the dye does not have time to enter and fill the cyst. For this reason, additional cysts can sometimes be identified when delayed CT myelography images are obtained. Regardless, the absence of a spinal meningeal cyst cannot reliably be determined with CT myelography.

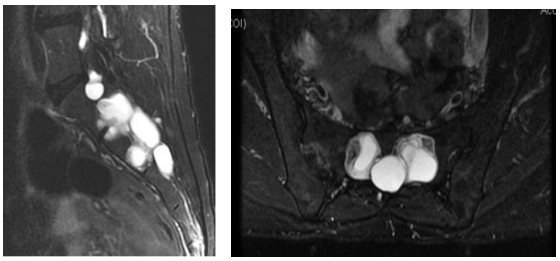


**Figure 14:** This symptomatic patient had multiple Tarlov cysts in the spinal canal, as seen on sagittal. (A) Axial (B) pelvic MRI. Interestingly, she was also known to have a connective tissue disorder, suggesting a causal relationship.

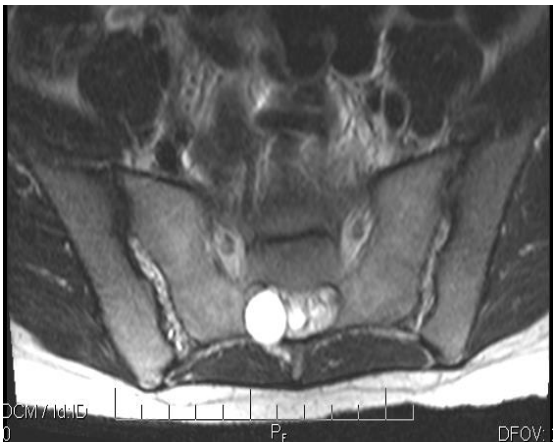


**Figure 15:** In this patient with right S2 distribution symptoms, axial sacral MRI reveals a compressed right S2 nerve root (arrow tip) by a Tarlov cyst affecting the right S3 nerve root (obscured by arrow).

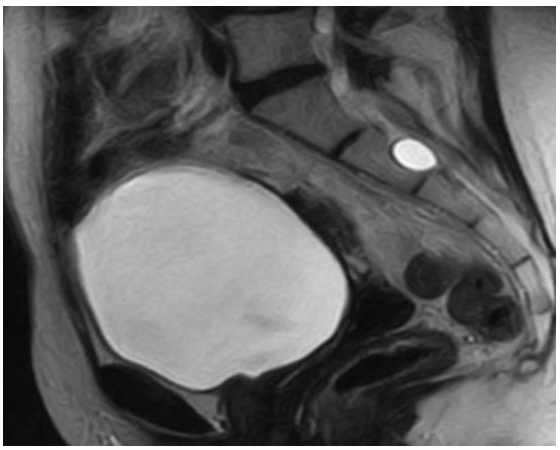


**Figure 16:** Evidence of urinary retention caused by a Tarlov cyst (right arrow) compressing the sacral nerve roots is seen in the form of a severely distended bladder (left arrow).

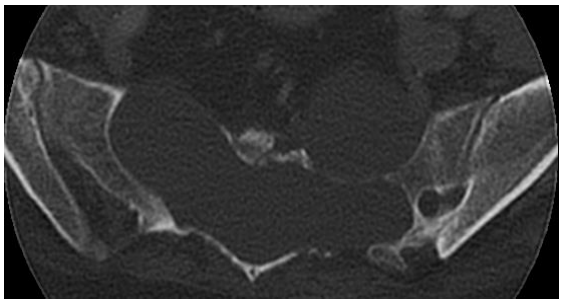


**Figure 17:** The extent to which Tarlov cysts can produce sacral osseous destruction is evident on this axial CT of the sacrum where spinal canal (star) and foramina are dramatically enlarged and there is extensive bone loss. The patient presented after a fall with pelvic-spinal dislocation due to sacral insufficiency fractures.

In antiquated theories, CT myelography was thought to be useful for distinguishing Tarlov cysts from other types of spinal meningeal cysts based on how fast they filled. However, these theories are illogical and have fallen by the wayside, primarily since the extent to which spinal meningeal cysts communicate with the spinal sac is not cyst

specific. In other words, the size of the opening between the spinal sac and a meningeal cyst is not constant based on the cyst type. Some spine surgeons erroneously use CT myelography to decide which Tarlov cysts to treat surgically, thinking that if the cyst is not seen on a CT myelogram, then the risk of postoperative cerebrospinal fluid leakage is eliminated. This assumption is essentially a gamble, because it can be safely said that, with rare exception, all Tarlov cysts are in communication with the spinal sac and have the potential to leak spinal fluid following surgery, regardless of how fast they filled with dye on a preoperative CT myelogram.

**Electrodiagnostics:** The use of EMG and nerve conduction studies in the evaluation of sacral Tarlov cysts is usually of little benefit since they routinely do not assess nerve function below the level of S1. Dysfunction of the S2-4 nerve roots, which are the most likely to be impacted by symptomatic sacral Tarlov cysts, is therefore missed. For this reason, it is not unusual to encounter patients with blatant sacral nerve root dysfunction and a completely normal electrodiagnostic study. Some centers perform EMG studies which do evaluate the lower sacral nerve roots, but patient discomfort is significant since it requires placement of multiple needles in the perineum.

**Treatment:** In cases where a Tarlov cyst is the suspected etiology, the presence of neurological deficit, or progressively worsening symptoms should prompt referral to a clinician experienced in their diagnosis and treatment. An attempt at conservative management can be made in patients without neurological deficit provided a careful evaluation for lumbosacral spinal nerve dysfunction has been made. Conservative management efforts typically involve physical therapy and pain management focused on lumbar spine, hip, and SI joint, but are usually ineffective, or make symptoms worse. In some centers pelvic floor therapy is an option for perineal and pelvic symptoms, although data on its effectiveness is sparse.

For the most part, needle procedures near a spinal meningeal cyst should be limited to selective nerve blocking in experienced hands for the purposes of diagnosis. For example, if a patient with left S2 distribution symptoms has a Tarlov cyst compressing the left S2 nerve root, then a diagnostic left S2 nerve root block followed by temporary symptomatic relief can help confirm the diagnosis. Practitioners should resist the urge to order percutaneous drainage procedures. Tarlov cysts are in direct communication with the spinal fluid of the spinal sac. Drainage alone is therefore pointless, since the cyst simply refills again, with the patient having been exposed to risks such as cerebrospinal fluid infection, hemorrhage or nerve injury. Some centers inject materials into Tarlov cysts, such as fibrin glue, simplistically thinking that it will somehow prevent further entry of spinal fluid into the cyst. However, this technique has multiple short-comings, with prior publications describing postinjection meningitis and adhesive arachnoiditis, since any substance introduced into a Tarlov cyst is also introduced into the CSF of the central nervous system [35,36].

Additionally, fibrin glue injection complicates subsequent definitive surgical treatment by making nerve fascicles within a Tarlov cyst more difficult to identify intraoperatively and protect. Tarlov cysts cannot simply be resected since each cyst is a spinal nerve root. This is particularly true in the sacrum; nerve root sectioning can result in unacceptable deficits, such as the loss of bowel and bladder function. Alternate surgical approaches, such as performing a laminectomy to "give the nerves space" without treating the Tarlov cyst/s has been tried and failed, as might be expected [37].

Attempting Tarlov cyst surgery without specific experience in their treatment often leads to a long, punishing misadventure, even among those with extensive skills in other areas of spine surgery. To complicate matters further, Tarlov cysts often appear similar to other types of spinal meningeal cysts on preoperative imaging, and the cyst type is sometimes not definitely known until it is evaluated intraoperatively.

## Conclusion

There is significant variability in the surgical strategy required to treat different types of meningeal cysts and the ability recognize and treat each cyst type is needed. Therefore, reliance on intraoperative improvisation is ill advised and a strong argument can be made that surgery for these complex cysts is best accomplished by those with specific experience in their treatment.

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