

Hip-spine Syndrome

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Abstract

The incidence of symptomatic osteoarthritis of the hip and degenerative lumbar spinal stenosis is increasing in our aging population. Because the subjective complaints can be similar, it is often difficult to differentiate intra- and extra-articular hip pathology from degenerative lumbar spinal stenosis. These conditions can present concurrently, which makes it challenging to determine the predominant underlying pain generator. A thorough history and physical examination, coupled with selective diagnostic testing, can be performed to differentiate between these clinical entities and help prioritize management. Determining the potential benefit from surgical intervention and the order in which to address these conditions are of utmost importance for patient satisfaction and adequate relief of symptoms.

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Subjective reports of pain in the buttock, thigh, and/or knee, with or without a limp, are common in patients with degenerative changes of the hip and spine.¹⁻³ Failure to appropriately diagnose the primary source of pain can result in delayed relief of symptoms and patient frustration. A structured history and physical examination, along with the use of specific diagnostic modalities, can help differentiate between symptomatic osteoarthritis (OA) of the hip and degenerative lumbar spinal stenosis (DLSS).

Degenerative pathology of the hip and lumbar spine is common in the aging patient. OA is the most common musculoskeletal disease of aging and the most frequent cause of musculoskeletal disability.⁴ It is second only to heart disease as the predominant cause of functional decline among the elderly.⁵ Hip OA is typically characterized as either primary (ie, idiopathic) or secondary, caused by entities such as gout, chondrocalcinosis, and hemochromatosis. Primary hip OA accounts for most

cases. The prevalence of radiographic hip OA is 27% in adults aged ≥ 45 years.⁶ However, not all patients with radiographic hip OA are symptomatic. Symptomatic hip OA is reported in 9.2% of adults aged ≥ 45 years.⁶ Thus, the treating physician must correlate the radiographic findings with subjective symptoms and physical examination findings consistent with hip arthritis.

DLSS can also present with extremity pain and limitations in walking. DLSS is the most frequent indication for spinal surgery in persons aged >65 years.^{7,8} In the aging population, approximately 1.2 million physician office visits per year in the United States are believed to be related to symptoms of lumbar spinal stenosis.⁷ Many types of lumbar stenosis exist, including congenital, iatrogenic, degenerative, and post-traumatic. The degenerative type is the one most frequently observed in this patient population.

The clinical scenario of concurrent hip OA and DLSS, or hip-spine syndrome, was first described by Offier-

ski and MacNab³ in a retrospective review published in 1983. They categorized patients as having simple, complex, or secondary hip-spine syndrome. In simple hip-spine syndrome, pathologic changes exist in the hip and lumbar spine, but only one clear source of disability is present. Persons with complex hip-spine syndrome present with coexisting pathologic changes but with no clear source of disability. Ancillary investigations are required to further differentiate between the two. In secondary hip-spine syndrome, the pathologic processes are interrelated, with each exacerbating the other. For example, a patient who stoops forward may do so because of a positive sagittal balance deformity and concurrent hip arthritis with a flexion contracture.

Diagnosis

History

Obtaining a thorough history is essential to understand the pathology. Radiating pain involving the lower extremity is common secondary to hip and spine pathology. Hip OA is often associated with groin and buttock pain, a limp, referred knee pain, and pain with hip range of motion. Khan et al⁹ reported 84.3% sensitivity and 70.3% specificity for groin pain in patients with hip OA. Patients with groin pain have been shown to be seven times more likely to have a hip disorder only or a hip-plus-spine disorder than a spine-only disorder.¹⁰ Leshner et al¹¹ evaluated pain referral patterns in patients undergoing fluoroscopically guided intra-articular injections for known hip pathology. They assessed visual analog pain score and the location of the pain before and after anesthetic injection. In contrast to previous reports, Leshner et al¹¹ demonstrated the buttock region to be the most

common anatomic location of referred pain in patients with isolated hip pathology (71%), followed by combined thigh and groin pain (55%). No patient with hip pathology had pain referred to the lumbar spine. Although pain referred distal to the knee joint is classically believed to be a result of lumbar stenosis, Khan et al⁹ found that 47% of patients with isolated hip arthritis reported pain radiating below the knee.

Symptomatic lumbar stenosis typically presents with neurogenic claudication with back and lower extremity pain that begins and worsens with ambulation and is relieved with sitting. Functionally, this presentation can be explained by the dynamics of the spinal column in the sagittal plane. With upright activity, there is a compensatory increase in lumbar lordosis to maintain sagittal alignment and balance, resulting in narrowing of the spinal canal.¹² The pain often resolves or improves on bending forward or sitting. The shopping cart sign is a good clinical indicator of lumbar stenosis. Patients with this sign find comfort ambulating while leaning over a shopping cart. Groin pain is uncommon in patients with lumbar stenosis; however, it can be the presenting complaint with foraminal stenosis at the L1 or L2 level secondary to a far lateral disk herniation or facet arthropathy.¹³ Regardless of the location of the pain, it often worsens on ambulation or standing and improves on leaning forward and/or sitting.

Lateral hip pain poses a unique diagnostic dilemma. Such pain can be a common presenting complaint, with radiation to the buttock and/or lower back region and down the lateral leg. It may be secondary to several different pain generators associated with greater trochanteric pain syndrome, including bursitis and inflammation or tear of the gluteal ten-

don. Lumbar pathology and primary hip OA can also cause referred pain in this region, resulting in an overlay of potential etiologies.

Physical Examination

A thorough physical examination is required to further differentiate the primary pain generator. Reproduction of the pain in the affected extremity on weight bearing is consistent with hip OA. Direct physical examination may elicit pain with manipulation, including internal or external rotation and log roll, antalgic gait, and decreased hip range of motion, which most commonly presents as loss of internal rotation. Brown et al¹⁰ demonstrated that primary hip pathology was routinely predicted by the presence of a limp, groin pain, or limited internal rotation of the hip. Additionally, they found that groin pain elicited by internal rotation of the hip was both sensitive and specific in the diagnosis of hip pathology.

Cam and pincer impingement are evaluated with the anteroposterior and posteroinferior impingement tests.¹⁴ The anteroposterior impingement test (ie, FADIR [flexion adduction internal rotation in extension] test) is performed by first placing the patient supine on the examination table with the hip in 90° of flexion. Symptoms are then elicited with combined adduction and internal rotation of the hip. The posteroinferior impingement test is performed with the patient supine and the hip extended over the edge of the examination table. In this position, pain is caused on external rotation of the hip. Pain on direct palpation over the trochanter is most often associated with local pathology rather than with radicular symptoms.

Physical examination findings are less predictable in persons with spinal stenosis. A minority of patients

Figure 1



Photograph demonstrating patient positioning in the Thomas test, which is used to evaluate for the presence of hip-flexion contracture. The patient lies supine with the pelvis near the edge of the examination table. The hip to be examined is maintained in extension, and the contralateral hip is flexed, bringing the knee up toward the chest. Inability to maintain the hip of the down leg in extension denotes a positive test, as shown here.

may exhibit radicular findings such as a positive straight leg raise or femoral stretch test. Other findings include decreased reflexes; diminished sensation, particularly in a dermatomal distribution; a positive Romberg test; and, less frequently, decreased strength with or without muscle atrophy. A positive femoral tension sign is nearly five times more likely to be noted in persons with lumbar stenosis than in those with hip pathology only.¹⁰ However, the Spine Patient Outcomes Research Trial (SPORT) demonstrated that <20% of persons with lumbar stenosis had either a positive straight leg raise test or femoral tension sign.¹⁵ Persons with hip flexion contracture can have a false-positive femoral tension sign; thus, this test is unreliable in the setting of hip-spine syndrome. The physical examination rarely demonstrates a neurologic deficit, especially in those with mild stenosis.¹⁶

The patient's clinical alignment in the sagittal and coronal planes should be carefully evaluated. Any spine patient who is being considered for a long lumbar fusion or osteotomy to correct a positive sagittal balance should undergo the Thomas test, which evaluates for the presence of a hip flexion contracture (Figure 1).

Diagnostic Tests

Plain radiography is the initial ancillary study obtained in the workup of hip OA. Radiographic findings consistent with hip OA include femoral and/or acetabular osteophytes, subchondral cysts, and joint space narrowing on weight-bearing views.¹⁷ Osteonecrosis and cam or pincer impingement are painful precursors to OA. These may be seen on radiographic studies obtained prior to presentation with hip-spine syndrome.

Subchondral lucency in the femoral head, which has the potential to progress to collapse and deformation, is indicative of more advanced osteonecrosis. However, early osteonecrosis can be visualized only on MRI. Radiographic findings of cam and pincer impingement include a bony prominence near the anterolateral head and neck junction, anterior overcoverage, acetabular retroversion, coxa profunda, and protrusio acetabuli. Minimal joint space narrowing may be evident in the early stages. The labrum, which is often the first structure to fail, is best visualized on MRI arthrogram. Labral tears are often asymptomatic, so it is important to ensure that the patient's complaints correlate with the labral tear visualized on MRI. MRI can also be helpful in ruling out an occult femoral neck or pelvis fracture, infection, or tumor as the cause of pain.

Fluoroscopically guided hip anesthetic injections can help further elucidate the primary pain generator. Given the potential toxicity of anesthetics on chondrocytes, these injections should be reserved for persons with radiographic evidence of hip OA.¹⁸ This test should be performed in persons with a history and physical examination that implicate the hip as the primary pain generator. Many studies have demonstrated that patients who experience $\geq 50\%$ pain relief following an intra-articular hip injection are likely to have a successful outcome following total hip arthroplasty (THA).

Crawford et al¹⁹ followed 42 patients who were being considered for primary THA and in whom it was unclear whether the hip was the source of their pain. Of the 33 patients who experienced pain relief following intra-articular injection of bupivacaine, 32 went on to a successful THA (sensitivity, 96%). In a study of 18 patients with radio-

graphic evidence of hip-spine syndrome, Kleiner et al²⁰ reported that relief of symptoms following an intra-articular hip bupivacaine injection had a sensitivity of 87% and specificity of 100% in diagnosing hip OA as the primary pain generator. For pain that is primarily lateral, an injection of the trochanteric bursa can be diagnostic and frequently serves as definitive therapy. If injection and/or other empiric interventions (eg, therapy, phonophoresis) do not provide pain relief, imaging of the spine should be considered.

In persons with suspected DLSS, imaging typically begins with upright plain radiographs, including AP, lateral, flexion, and extension views. These views allow assessment of spinal alignment, signs of radiographic instability, and identification of degenerative changes at the disk space and posterior elements. If the clinical examination findings or lumbar radiographs are suggestive of spinal deformity, an upright 36-inch radiograph should be obtained to accurately assess the deformity and evaluate for compensatory curves along the length of the spine. MRI or CT myelography is used to identify neural impingement. MRI is the study of choice in those without a contraindication given that it provides superior detail of the soft tissues. CT myelography is invasive and exposes the patient to radiation; therefore, it should be reserved for patients with preexisting spinal hardware that would distort the MRI quality and in patients with implants for which MRI is contraindicated (eg, pacemaker). Positive findings on MRI or CT myelography in asymptomatic patients increase with age; thus, it is important to correlate the history and physical examination findings with findings on ancillary studies.^{21,22}

Electrophysiologic studies are used when the diagnosis remains unclear.

Normal findings on electrophysiologic studies do not rule out DLSS, whereas findings of bilateral polyradiculopathy at multiple levels can be suggestive of this process.²³ Electrophysiologic studies are especially helpful in distinguishing the neurologic changes of spinal stenosis from either peripheral nerve compression or diabetic peripheral neuropathy.²⁴ The treadmill test provides a functional assessment of lumbar stenosis. Similar to treadmill testing for coronary artery disease, treadmill testing for DLSS is performed by having the patient walk on a treadmill for a set time period or distance, or until the onset of neurogenic claudication symptoms. However, because both conditions can result in limited ambulation, this test is not useful in differentiating lumbar stenosis from hip OA.²⁵

Fluoroscopically guided epidural steroid injections (ESIs) may be diagnostic or confirmatory. Improvement in the primary symptoms following ESI can help confirm stenosis as the primary pain generator. However, lack of improvement following ESI does not definitively rule out lumbar stenosis as the primary pain generator. Persons with isolated lumbar stenosis can have a minimal response to an injection yet have significant improvement following decompressive surgery.¹⁵ Although injection is useful to manage neurogenic pain that is secondary to an inflammatory process, it is not helpful in managing ischemic processes. Many studies have demonstrated the efficacy of fluoroscopically guided injections in managing radicular leg pain secondary to lumbar stenosis; however, the improvement is often temporary.

Karppinen et al²⁶ randomized 160 patients with lumbar radicular pain to saline control transforaminal injection or methylprednisolone bupivacaine injection. At 2-week follow-up, a statistically significant

improvement was found in the steroid group with regard to leg pain ($P = 0.02$), the straight leg raise test ($P = 0.03$), lumbar flexion ($P = 0.05$), and overall satisfaction ($P = 0.03$). However, this difference in therapeutic efficacy between groups was lost after 4 weeks. Botwin et al²⁷ followed elderly patients with spinal stenosis after an average of 1.9 transforaminal injections and demonstrated statistically significant improvements in pain ($P < 0.0004$) and function ($P < 0.0004$) at 2 and 12 months following injection. In a study of 140 patients with lumbar spinal stenosis, 32% had >2 months of relief from their symptoms following ESI.²⁸

Because ESI has the potential for complications and lacks long-term efficacy, it should be done as a diagnostic or confirmatory test only in patients with a history and physical examination that indicate lumbar stenosis as the primary pain generator. In the patient who does not experience relief following lumbar ESI, an intra-articular hip injection can be considered before deciding which pathology to manage first.

Differential Diagnosis

In the patient with hip-spine syndrome, it is essential to rule out other causes of lower extremity pain. These include clinical entities such as peripheral vascular disease, diabetic peripheral neuropathy, and pelvic pathology. Sources of pain about the pelvis are numerous. Labral tears of the hip are an underappreciated source of pain and can be difficult to diagnose; they often present with normal or osteoarthritic radiographs. Painful osseous pathology includes metastases, Paget disease, occult hip fractures, insufficiency fractures of the sacrum, and osteonecrosis. Neurologic etiologies include

meralgia paresthetica and shingles. More lateral pain can be secondary to greater trochanteric bursitis or gluteal tendinitis/tendon ruptures.

Vascular claudication should be evaluated in persons with skin discoloration, skin ulcers, lower extremity alopecia, and diminished or absent pulses. The ankle brachial index is the most reliable and least invasive objective assessment of peripheral arterial disease. A value of <0.90 has been reported to be 89% sensitive for isolated femoropopliteal disease and 97% sensitive for isolated aortoiliac disease.²⁹ Claudication of the internal iliac artery can result in Leriche syndrome, of which one symptom is buttock pain. If confusion persists, further vascular studies can be obtained, including duplex ultrasonography.

Knee OA is a common cause of lower extremity pain, especially in the aging population. DLSS and hip OA can both present with referred knee pain. History, physical examination, and knee radiographs, including standing AP, standing lateral, and patellofemoral joint views, are routinely used in the clinical diagnosis. Further imaging modalities are rarely necessary. As with hip OA, intra-articular injections can be diagnostic and therapeutic for pain symptoms that are primarily related to knee OA and can help identify the primary pain generator.

Management

After the predominant pain-generating pathology has been determined, nonsurgical management is attempted (eg, ESI, fluoroscopic guided hip injection). Surgical management is considered when nonsurgical measures are unsuccessful (Figure 2). History, physical examination, and radiographs are used to confirm the diagnosis of concurrent

hip OA and DLSS. Patients with progressive neurologic deficits require urgent consultation with a spine specialist. In the absence of a progressive neurologic deficit, the predominant patient complaint guides treatment.

A subjective report of primarily groin pain warrants further evaluation of intra-articular hip pathology. Radiographic changes that are consistent with mild OA warrant further workup with an MRI arthrogram. Intra-articular injection of local anesthetic at the time of the arthrogram should be strongly considered as a therapeutic and a diagnostic tool to elucidate the primary pain generator. Most positive intra-articular findings on MRI require referral to a hip specialist to address labral tears and the cam and/or pincer deformities that can contribute to these injuries. Patients with obvious radiographic findings of hip OA should undergo fluoroscopically guided intra-articular hip injection. THA may be considered if the patient has relief of the primary pain generator following injection. Conversely, if the patient does not have pain relief following intra-articular hip injection, she or he should proceed to workup of spine pathology.

A subjective report that primarily consists of paresthesias or radiculopathy warrants further workup of spine pathology. Hip OA with a flexion contracture and concurrent symptoms of lumbar spinal stenosis is a potential confounding variable. In secondary hip-spine syndrome, the processes are interrelated and exacerbate one another. For example, in the patient with positive sagittal balance deformity and concurrent hip OA with a flexion contracture, we recommend addressing the hip flexion contracture first with preoperative physical therapy or surgical intervention, even if lumbar stenosis is thought to be the primary pain gen-

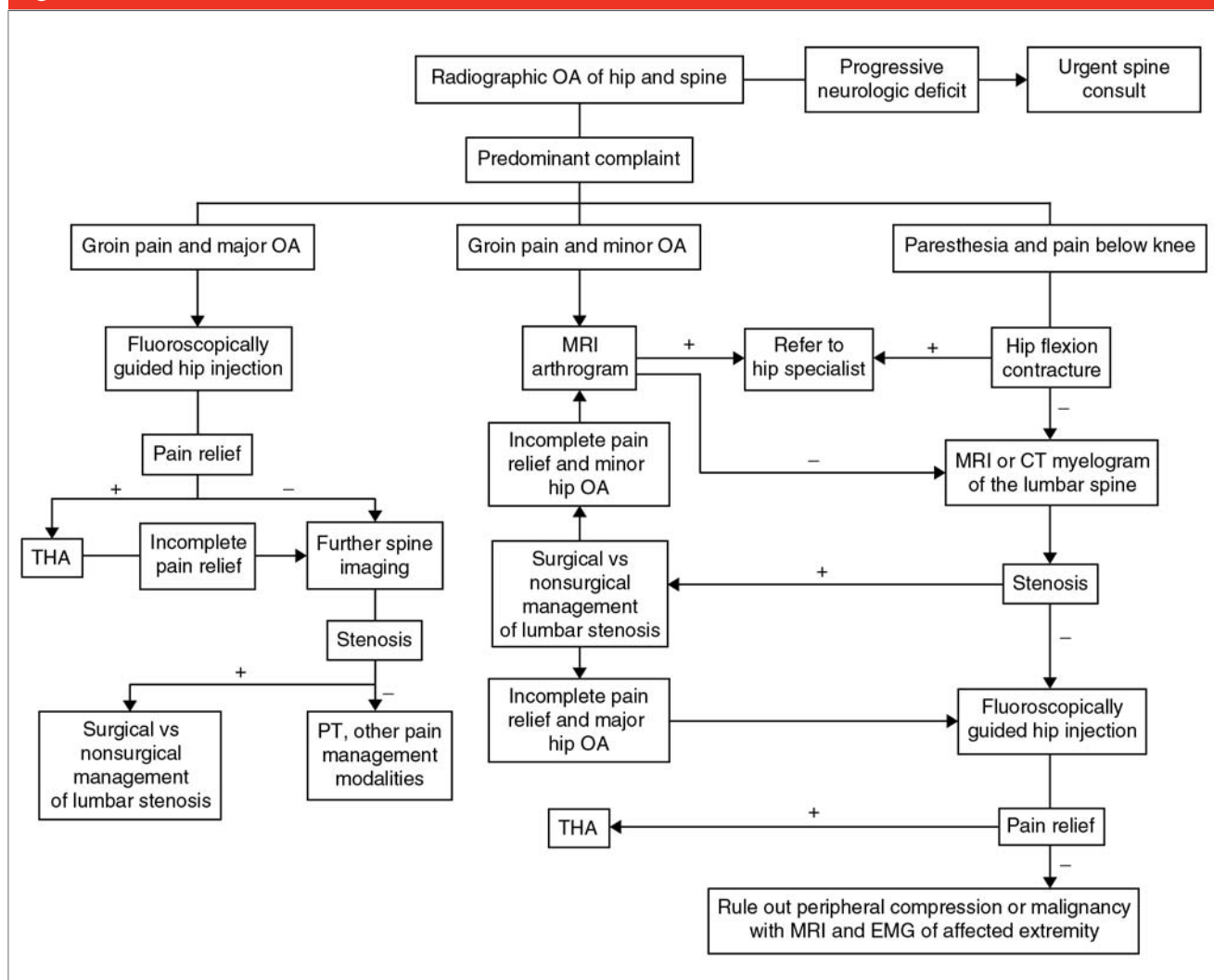
erator. This is important because failure to restore appropriate sagittal balance during instrumented lumbar fusion predicts a poor outcome.³⁰ The hip surgeon must be cognizant of the patient's pelvic tilt while performing the corrective THA because a hip flexion contracture with inadequate corrective lordosis to compensate for upper sagittal deformity can contribute to pelvic extension and relative acetabular retroversion.³¹ Pelvic tilt also has been shown to be dynamic and can change following THA, thereby confounding attempts to appropriately place the acetabular component.³²

In the absence of a hip flexion contracture, lumbar MRI is recommended to confirm the diagnosis of lumbar spinal stenosis. CT myelography may be done in the patient for whom MRI is contraindicated. A fluoroscopically guided ESI can be administered, with the patient carefully documenting pain relief. If an ESI does provide relief, then the surgeon can proceed with decompression, with or without fusion, as warranted.

THA is considered to be a benchmark for a surgical treatment that achieves a statistically and clinically relevant improvement in health-related quality of life. In a recent prospective cohort study, Mokhtar et al³³ demonstrated that decompression and fusion for lumbar stenosis and acquired degenerative spondylolisthesis results in significant improvement in quality of life and yields health-related quality of life levels comparable to those of THA.

In some cases, surgical management of one complaint alleviates the symptoms caused by another pathology. This was well demonstrated recently by Parvizi et al,³⁴ who indicated that 170 of 344 patients slated to undergo THA reported low back pain preoperatively (49%). Of these 170 patients, 113 (66%) experienced resolution of low

Figure 2



Treatment algorithm for hip-spine syndrome for patients in whom appropriate history and physical examination have been performed and in whom radiographs of the hip and spine demonstrate concurrent degenerative findings. + = findings on imaging studies or a response to treatment, - = imaging studies were normal or there was no response to treatment, EMG = electromyography, OA = osteoarthritis, PT = physical therapy, THA = total hip arthroplasty

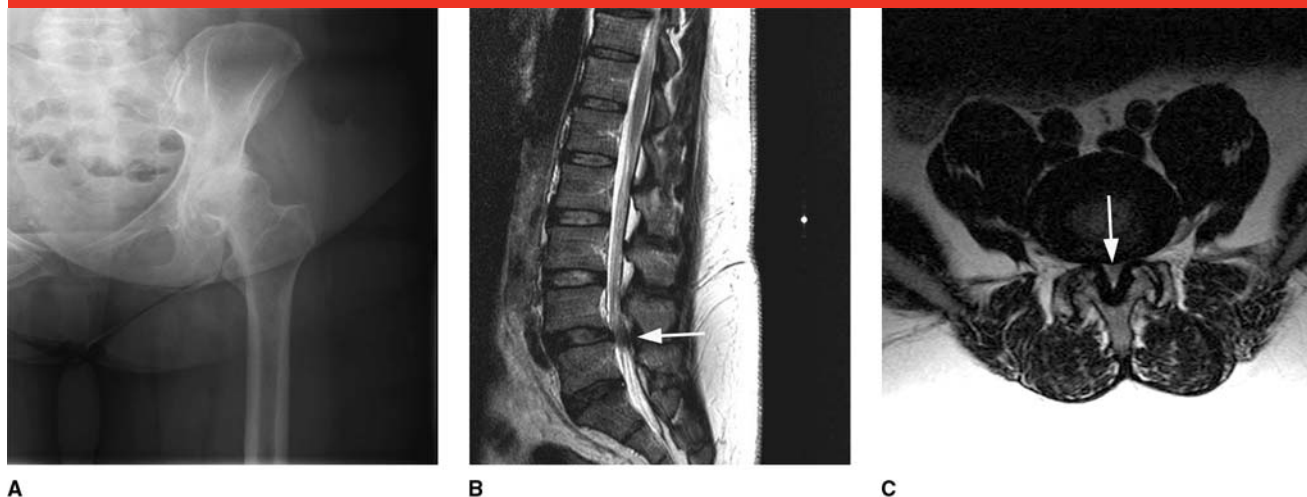
back pain following THA.

Surgical management should be directed at the primary pain generator. It is important, however, to make sure that the patient understands that treatment of one condition can improve activity level and make the untreated condition more symptomatic. Thus, the patient must be counseled that he or she may not get full relief of symptoms despite the identification and management of the primary pain generator.

Bohl and Steffee³⁵ first detailed this in 1979 in a series of eight patients with persistent pain following THA. These patients had resolution of groin and anteromedial thigh pain, but posterior thigh pain became more symptomatic postoperatively. The symptoms resolved in six of the patients who underwent decompressive lumbar laminectomy. These findings were further substantiated in a study by McNamara et al,¹ in which five patients with concurrent

hip OA and lumbar stenosis initially underwent THA. Following THA, two of the five patients who initially presented with hip OA and DLSS required subsequent lumbar decompression. The second group in the study consisted of nine patients whose symptoms of DLSS were masked by hip pain and were subsequently exacerbated following THA. Seven of these nine patients underwent lumbar decompression, and 85% had good or excellent results.

Figure 3



A, Hip AP radiograph demonstrating left hip osteoarthritis in the setting of degenerative lumbar spinal stenosis in a 52-year-old woman. Sagittal (**B**) and axial (**C**) magnetic resonance images demonstrating degenerative lumbar spinal stenosis at L4-5 (arrows) with an associated acquired degenerative spondylolisthesis.

In the setting of severe lumbar stenosis, evidence exists that decompression should be performed before addressing the hip. Pritchett¹² reported on 21 patients with severe spinal stenosis who developed foot drop after THA. Of the 16 patients who underwent lumbar decompression, only 6 had complete recovery of extensor function. The five patients who were treated nonsurgically did not demonstrate neurologic improvement.

In our own practice, a 52-year-old woman presented with left hip OA and DLSS. Evidence of left hip OA was noted radiographically (Figure 3, A), along with osteophyte formation and joint space narrowing. Evidence of lumbar spinal stenosis and degenerative spondylolisthesis are noted, as well (Figure 3, B and C). The patient presented to the spine clinic following a lumbar MRI that demonstrated findings of lumbar spinal stenosis and an acquired degenerative spondylolisthesis. Although history and physical examination revealed that neurogenic claudication was the primary subjective com-

plaint. Additionally, the patient had received an ESI, which relieved a significant portion of her symptoms. After failing nonsurgical care, the patient underwent L4-5 laminectomy and instrumented posterior spinal fusion. The patient became more ambulatory postoperatively, and the hip OA became more symptomatic, with resultant groin pain. The patient was referred to a total joint surgeon for THA, after which her symptoms improved significantly.

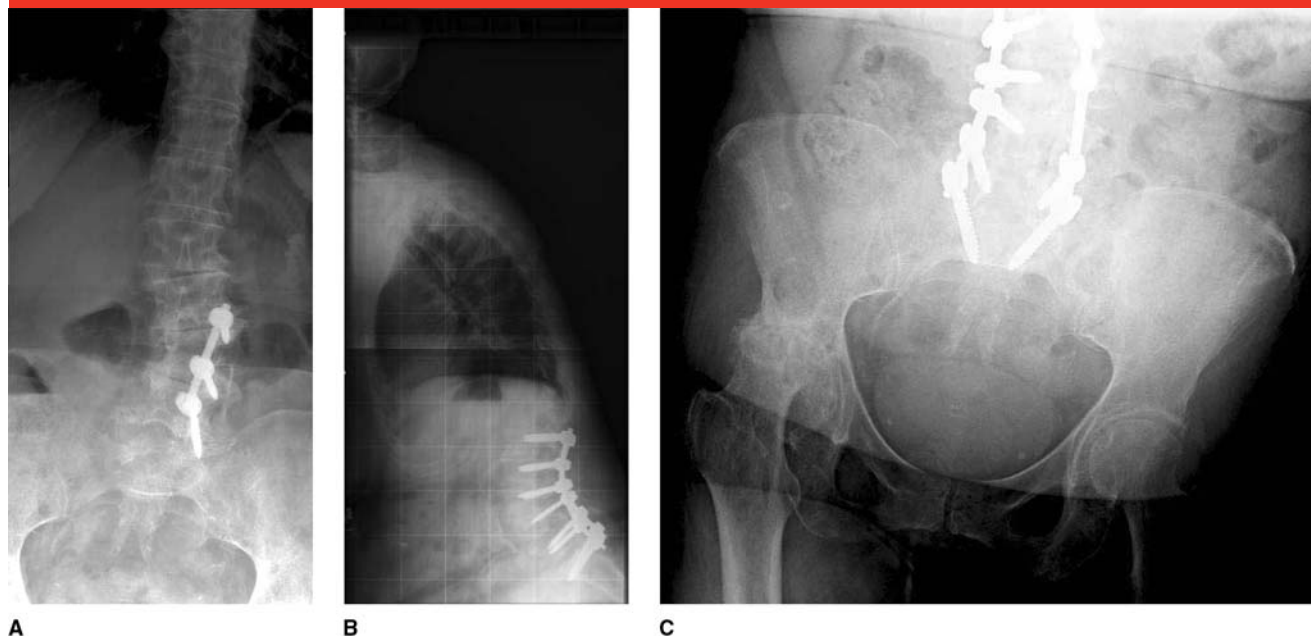
In another case, a 66-year-old presented with complaints of right lower extremity pain and a feeling of stooping forward, with associated back pain and fatigue. She had a history of two previous surgeries (Figure 4, A and B), the first of which was lumbar laminectomy and unilateral L3-5 instrumentation, with minimal relief of her symptoms. She subsequently underwent L1-S1 Smith-Petersen osteotomies to better correct her sagittal plane deformity, which she felt did little to improve her posture. Her examination demonstrated groin pain on hip internal and external rotation and a severe

hip flexion contracture on the Thomas test. Plain radiographs of the pelvis demonstrated severe right hip OA (Figure 4, C). This was felt to be the cause of her sagittal plane deformity. The patient was subsequently referred to a total joint surgeon for right THA, which significantly improved her sagittal balance.

Summary

The patient who presents with lower extremity pain and radiographic evidence of hip-spine syndrome should be managed with a thorough history and physical examination as well as specific diagnostic tests aimed at determining the predominant pain generator. CT and/or MRI of the spine along with other diagnostic modalities can be used to help further delineate the primary pathology and guide the order of surgical management. However, the physician and patient should be aware that a second surgery may be necessary to address the untreated entity should pain persist beyond the normal ex-

Figure 4



A 66-year-old woman presented with persistent back fatigue and positive sagittal balance following two prior spinal surgeries. **A**, AP lumbar spine radiograph demonstrating prior lumbar laminectomy and unilateral instrumented posterior spinal fusion from L3-5 with minimal improvement in her back fatigue or right leg pain. **B**, Lateral spine radiograph demonstrating a positive sagittal balance after a second surgery consisting of multiple Smith-Petersen osteotomies and L1-S1 segmental instrumentation was performed in an attempt to correct her sagittal alignment. **C**, AP pelvis radiograph combined with focused examination of the hip revealed severe right hip OA with an associated hip flexion contracture. Following hip replacement, the patient's sagittal alignment returned to neutral, and her mobility improved.

pected course of postoperative recovery. Identification of the presence and significance of both diseases and management of them in the appropriate order can decrease the likelihood of an inadequate diagnosis and persistent postoperative pain.

References

Evidence-based Medicine: Levels of evidence are described in the table of contents. In this article, reference 25 is a level I study. References 14, 20, and 33 are level II studies. References 4, 5, 9, 12, 15, 18, 19, 23, 24, 26, 28, and 32 are level III studies. References 1, 3, 8, 10, 11, 21, 27, 29-31, and 34 are level IV studies. References 2, 6, 7, 13, 16, 17, and 22 are level V expert opinion.

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