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Back Pain with Leg Pain

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Abstract
Purpose of Review The clinical diagnostic dilemma of low back pain that is associated with lower limb pain is very common. In relation to back pain that radiates to the leg, the International Association for the Study of Pain (IASP) states: "Pain in the lower limb should be described specifically as either referred pain or radicular pain. In cases of doubt no implication should be made and the pain should be described as pain in the lower limb."
Recent Findings Bogduks' editorial in the journal *PAIN* (2009) helps us to differentiate and define the terms somatic referred pain, radicular pain, and radiculopathy. In addition, there are other pathologies distal to the nerve root that could be relevant to patients with back pain and leg pain such as plexus and peripheral nerve involvement. Hence, the diagnosis of back pain with leg pain can still be challenging.
Summary In this article, we present a patient with back and leg pain. The patient appears to have a radicular pain syndrome, but has no neurological impairment and shows signs of myofascial involvement. Is there a single diagnosis or indeed two overlapping syndromes? The scope of our article encompasses the common diagnostic possibilities for this type of patient. A discussion of treatment is beyond the scope of this article and depends on the final diagnosis/diagnoses made.

Keywords Sciatica · Somatic referred pain · Radicular pain · Neuralgia · Back pain · Leg pain

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Introduction

Mr. A.B. is a 45-year-old healthy white male. He is a heavy smoker, not physically active, and works in a sedentary job. He presents to his primary family physician, due to 2 weeks of mild low back pain that started after lifting a heavy weight. He reports that for the last 2 days, his low back pain has become more severe and has developed right leg pain (both are 8/10 on Visual Analogue Scale for pain—VAS). He describes the back pain as dull, mainly in the mid-line at the level of L5. Walking and coughing increase this pain and bed rest alleviates it. He also describes the leg pain as lancinating, radiating along the posterior thigh and calf (above the ankle) as well as a sensation of numbness in a typical L5 dermatomal distribution. On physical examination: The patient is obese (BMI = 32), walks with an antalgic gait, and has reduced range of motion of the lumbar flexion but no muscle weakness. He has normal tendon reflexes. The "straight leg raising" (SLR) is limited at 40° on the right and 60° on the left. The "jump sign" (sensitive trigger points) is positive above the gluteus muscle. No red or yellow flags were identified in a systematic history and physical exam overview.

What is the most likely diagnosis? What should be the next step taken by the primary physician (GP or the orthopedic surgeon) to evaluate the patient's pain? What should be the first-line treatment to help this patient?

The clinical dilemma of low back pain that is associated with lower limb pain is very common. In this article, we will try to answer these questions and to outline a rational algorithm for primary physicians to approach this very common clinical scenario.

But first, we must go back to the International Association for the Study of Pain (IASP) classification of pain, Second Edition (Revised), published in 2011. In the chapter about back pain that radiates to the leg it is stated: "Pain in the lower limb should be described specifically as either referred pain or radicular pain. In cases of doubt no implication should be made and the pain should be described as pain in the lower limb" [1].

Bogduk helps us to differentiate and define the different terms: *Somatic referred pain* is a nociceptive pain caused by a noxious stimulation of structures in the lumbar spine (muscles, ligaments, and joints) that produces referred pain to the lower limb in addition to the back pain [2••]. *Radicular pain* is a neuropathic pain evoked by ectopic discharges emanating from a dorsal root or its ganglion. *Radiculopathy* is a neuropathy manifested by conduction block. It may be painful or non-painful. If the sensory fibers are involved, numbness is the main clinical feature. If the motor fibers are involved, muscle weakness is more prominent. Diminished reflexes occur as a result of either sensory or motor involvement. Radicular pain and radiculopathy often co-exist, but either of them can stand alone without the other component.

Another type of back pain not mentioned in Bogduks' article that will be discussed here is neuropathic pain with neural involvement distal to the nerve root—the plexus and peripheral nerves.

Somatic Referred Pain and Trigger Points

Somatic referred pain is by far the most common reason for low back pain with lower limb pain [3]. As discussed above, noxious stimuli of muscles, joints, or ligaments in the lumbar region will often produce local nociceptive pain as well as referred pain to the lower limb [2••].

Somatic Referred Pain from Muscles

Myofascial pain (MFP) is a subtype of nociceptive pain, caused by an active trigger point (TrP) located in a taut band within a muscle. The muscle has a unique and well-recognized referred pain pattern. The five main clinical characteristics of active TrPs causing MFP are spot tenderness, pain recognition, referred pain, palpable taut band, and twitch response [4].

Several muscles in the lumbar region have referred pain patterns involving the lower limb: the gluteus minimus, medius, and maximus, the piriformis muscle, and the muscles of the posterior thigh (semitendinosus and semimembranosus).

MFP from active TrPs in the gluteus minimus muscle is one of the main reasons for low back pain with lower limb pain. The referred pain pattern of the gluteus minimus muscle may mimic radicular pain and therefore has been named the "pseudo-sciatica muscle" [5] (Fig. 1). Myofascial pain from active TrP in the piriformis muscle is another common reason for back pain with lower limb pain [5]. As mentioned above, this muscle may also cause true neuropathic pain due to entrapment and has therefore been named the "double devil," due to the anatomical course of the sciatic nerve that lies adjacent to and sometimes within the muscle. Pain syndromes of this muscle may therefore include a neurogenic referral pattern in addition to the piriformis somatic referred pain pattern.

Somatic Referred Pain from Joints

The lumbar facet joints can produce referred pain patterns involving the low back and the lower limb. Gellhorn described the pain distribution from the lumbosacral facet joints [6]. Irritation of the facet joints in normal volunteers produces a referred pain pattern that mimics sciatic pain. Anesthetic facet block or facet denervation can produce relief of this pain [7].

The role of the sacroiliac joint as a sole source of referred pain to the lower limb is less clear, but it might be part of the piriformis muscle referred pain, as described by Freiberg [8]. A more recent study has described sacroiliac joints as causes of mainly back and buttock pain, but lower extremity pain was reported in 50% of the patients, pain distal to the knee in 28%, and pain reaching the ankle in 14% [9].

Somatic Referred Pain from Intervertebral Discs

Noxious stimulation of the intervertebral disc itself may result in a referred pain pattern that extends into the lower extremity above and below the knee [10]. The distal extent of pain produced depends on the intensity of the noxious stimulation.

Somatic Referred Pain from Ligaments

Ligaments are well recognized as sources of somatic referred pain [11•, 12]. Kellgren, in his novel work in 1939, injected 6% hypertonic saline into the interspinous ligament from the level of cervical spine to the lumbosacral area of healthy volunteers.

The exact incidence or prevalence of somatic referred pain and MFP that causes low back pain with lower limb pain is not known but the articles that specifically distinguished somatic referred pain from radicular pain found that somatic referred pain was responsible to 79–88% of all cases of low back pain with lower limb pain [2••, 3, 13].

Despite its potential severity, most somatic referred pain and MFP causing acute low back pain with lower limb pain

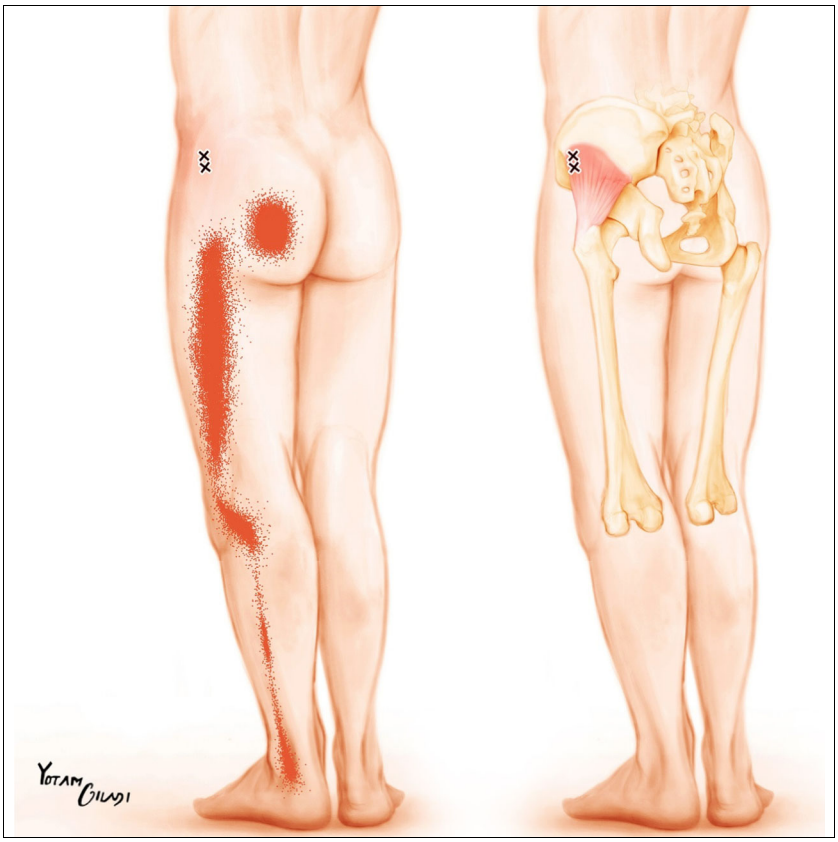

Fig. 1 Trigger points in the anterior gluteus minimus muscle

is self-limited. Dry needling, TrP injections with or without adjuvant physical therapy can dramatically improve the patient symptoms [14], but even without any treatment, most patients will significantly improve with time. Nevertheless, a minority of the patient will proceed to chronic pain (above 3 months). Risk factors for developing chronic pain will include perpetuating factors such as mechanical causes (such as structural, scoliosis, postural problems, and ergonomics), psychological conditions (such as anxiety, depression, and litigation), and nutritional and metabolic disorders (such as vitamin and iron deficiencies and hypothyroidism) [4].

The Natural History of Radicular Pain

Lumbar intervertebral disc herniation (LIDH) is a common occurrence usually resulting from damage or degeneration of the annular fibrosus of the intervertebral disc leading to bulging and protrusion of the annulus or extrusion or sequestration of the nucleus pulposus (Fig. 2). The evolution of radicular pain is usually caused by a combination of pressure exerted on the dorsal root ganglion, together with nerve root irritation [15]. Irritation of neurological structures is manifested as motor, reflex, or sensory dysfunction in the lower extremities and (rarely) as bowel or bladder dysfunction [16]. The clinical manifestations can include some or all of the following: sharp or burning pain radiating down the leg, often associated with paresthesia or numbness. Additional findings, dependent on the degree of nerve root compression and compromise, may include motor signs such as weakness and decreased tendon reflexes [17]. It is important to note that although most cases of radicular pain are due to LIDH, there exists an important list of differential diagnoses such as extra-spinal bone and soft tissue tumors [18], radiculitis, piriformis syndrome, and somatic referred pain [2••, 19]. Although acute radicular pain may be severe, the important question arises as to the natural history of this syndrome, whether conservative measures suffice and what are the indications for surgery.

Excellent studies have been published concerning the clinical course of conservatively and surgically treated patients suffering from radicular pain. Hakelius published a monumental study of 583 patients referred to a tertiary center orthopedic department for treatment of acute sciatica [20]. One hundred sixty-six patients were treated surgically and 417 conservatively. At 6 months, the results were comparable with 99 and 93% of surgical and conservative patients, respectively, reporting a favorable effect of the treatment at 6 months. Since this report, other studies have shown similar excellent results for conservative treatment of acute radicular pain [21–23]. In general, it is fair to state that the long-term results of conservative therapy for acute radicular pain are comparable to surgical therapy although it would appear that the early

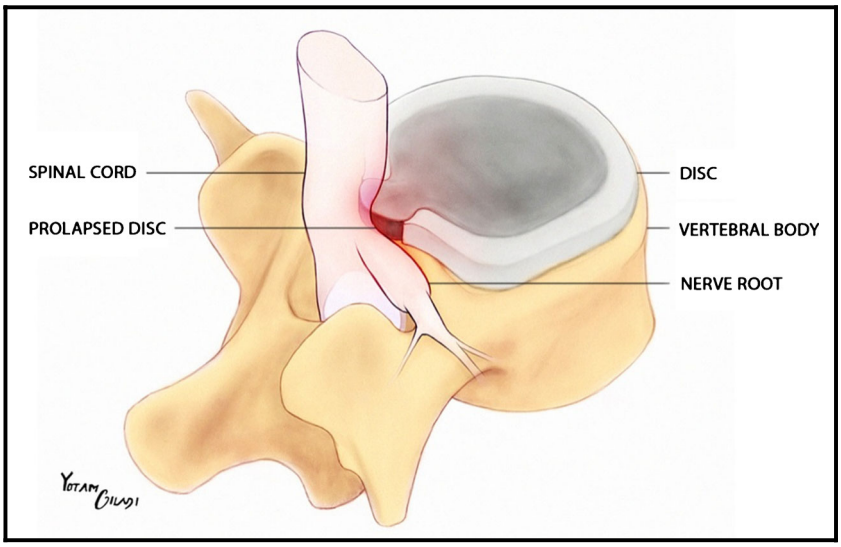

Fig. 2 Prolapsed disc

outcome of pain reduction, especially within the first year, may favor surgical intervention [24].

Interestingly, the examination of the natural history of radicular pain due to LIDH in conservatively treated patents shows a marked resorption of the disc—especially if the initial herniation was large [25–27]. In fact in one study, it was shown that the degree of resorption of the disc correlates well with the outcome of conservatively treated patients. Those whose disc prolapse did not resorb had a worse outcome [28].

Thus, to summarize the natural history of patients suffering from LIDH, we can claim that the approximately 90% of the patients without severe neurological compromise (surgical indicated patients) will do well and show good to excellent recovery over the next 6–12 months, and that the disc, especially if a large prolapse is found, will resorb during this period.

The Role of Compression, Inflammation, and the Immune System on the Nerve Root

Lumbar intervertebral disc herniation may be symptomatic or not as evidenced by incidental findings of LIDH in asymptomatic subjects [29–31]. Thus, we cannot equate LIDH with clinical symptoms in every case and therefore must ask, what are the conditions that manifest as pain and how does this pain express itself. As far back as 1956, Kelly published an important paper on the question of radicular pain and pressure to nervous tissue. He argued admirably that pressure per se, whether from tumors or from herniated intervertebral discs, could not be the sole cause for pain, alluding to other factors, including local inflammation [32].

Bodguk and Merskey defined radicular pain as pain evoked by ectopic discharges emanating from a dorsal root or its ganglion [1, 2••]. According to Bogduk, disc herniation is the most common cause of radicular pain, and inflammation of the affected nerve seems to be the critical pathophysiological process [33, 34]. Lindahl was one of the first to perform increased pressure around suspected nerve root lesions on the painful and the non-painful side on patients suffering from LIDH. Invariably, he found that pressure per se was not a cause of pain and that there was a necessary hyperalgesic component that contributed to the pain with increased pressure [35]. This was further corroborated on histological studies of patients operated for sciatica where 70% of the patients for whom biopsy material was collected from the area of the dorsal root showed inflammatory changes [36]. That many patients suffering from radicular pain have evidence of inflammatory mediators in the perineural tissues, especially in the acute phase, is well established [37]. Studies performed on patients with lumbar disc pathology undergoing surgery have shown pro-inflammatory mediators in the disc and perineural tissue [38–42]. Pro-inflammatory markers have been found in the serum and cerebrospinal fluid of patients suffering from lumbar disc herniation and sciatica [43–45]. An excellent discussion of the various lines of evidence for inflammation as part of the pathogenesis of radicular pain was published by Stafford [34]. It has also been suggested that an immune reaction to nervous tissue may be involved in the pathogenesis of both acute and chronic sciatica [46].

Thus, to summarize, a combination of factors including pressure, inflammation, and an immune response seem to be implicated in the pathogenesis of both acute and chronic radicular pain. Pressure per se would not be a cause of pain, but rather of nerve dysfunction such as weakness and numbness, while the addition of both inflammation and an immune response could explain the severe pain experienced by patients suffering from radicular pain.

Neural Involvement Distal to the Nerve Root

Although lumbar radiculopathy is the most common form of NP [47], several other conditions should be considered in the differential diagnosis of radiculopathy, such as lumbosacral plexopathies and proximal and distal focal peripheral neuropathies. Establishing the right diagnosis might be challenging since many of these conditions (including radiculopathy) have heterogeneous and sometimes overlapping clinical presentations. At the same time, correct diagnosis is essential for proper treatment administration. This section of the article will review some of these conditions.

Lumbosacral Plexopathies

The lumbosacral plexus is located immediately distal to the corresponding nerve roots and is not uncommonly affected by a large number of metabolic, inflammatory, ischemic, autoimmune, oncological, and iatrogenic disorders. Plexopathies are manifested by pain and other sensory, motor, and autonomic symptoms and signs which involve the lower torso, pelvis, and lower extremities and are often asymmetric. They can therefore be easily misdiagnosed as radiculopathy, and vice versa. Asymmetric, painful, proximal weakness, and muscle wasting in one leg in patients with diabetes mellitus is the most common form of lumbosacral plexopathy, termed diabetic amyotrophy or Bruns-Garland syndrome [48]. Direct spread of pelvic tumors, remote metastases, or lymphomas invade the lumbosacral plexus fairly commonly. Cancer treatments including surgery and radiation therapy are well-known causes for painful plexus injuries [49]. Autoimmune conditions, vaccination, and infectious diseases such as herpes zoster or Lyme disease are rare causes of painful lumbosacral plexopathies.

Focal Peripheral Neuropathies

Numerous etiologies have been identified as potential causes of focal peripheral neuropathies, but entrapment injuries along the sciatic nerve from its origin to the terminal ends in the foot are the most common underlying mechanism. Like any other form of neuropathy, entrapment neuropathies can present with pain, paresthesia, sensory loss, and partial or complete motor palsy. According to Lundborg [50], there are three clinical stages in ongoing nerve compression: Stage I is characterized by intermittent paresthesias and sensory deficits occurring primarily at night; Stage II occurs after continued progressive compression, which leads to more severe and consistent symptoms (such as paresthesias and numbness) that fail to resolve during the day; Stage III is defined as pronounced morphologic neural changes, leading to more constant pain that does not disappear, even if the impinging cause is resolved. Generally, stage I conditions can be treated conservatively by physical interventions and pain or inflammation control, but in more advanced pathologies (stage III and occasionally stage II), surgical intervention might be required [51].

Proximal Leg Syndromes

Although rare, the piriformis muscle syndrome (PMS) is a well-recognized cause of non-spinal compressive neuropathy of the sciatic nerve by the piriformis muscle. The initial description of the syndrome was given by Yeoman in 1928 [52], but the entity "piriformis" was coined by Robinson nearly two decades later [53]. A considerable number of clinical and research articles related to the syndrome have been published but its pathophysiology is still called into question [54]. This is mainly due to anatomical variations in the traversing course of the sciatic nerve through the infra-piriformis foramen and in uncertainty about how exactly the piriformis muscle compresses the sciatic nerve (i.e., hypertrophy, contracture) [55].

The clinical symptoms consist of pain spreading from the buttocks through the sciatic territory. Symptoms may be aggravated by intense effort and by "trigger" factors such as distance running and postures involving prolonged periods in a seated position such as cycling or horse riding and professional driving [56, 57]. Physical examination of a patient suspected of PMS is comprised of tenderness on sciatic notch palpation and performing specific maneuvers aimed to provoke the symptoms (buttock pain and sciatic tingling, numbness, or paresthesia) by putting stress on the piriformis muscle. Notably, patients often have to stay for a while in the maneuvered position before symptoms appear. These maneuvers have been recently described in a recent review article, but importantly, the sensitivity and specificity of none of them has not been validated [55].

Imaging studies, mainly pelvic MRI, are aimed primarily to rule out other causes of sciatica although they may occasionally show piriformis abnormalities as well [58, 59]. Electroneuromyography (ENMG) can assist in localizing the injury site along the sciatic nerve [60, 61].

Since not all proximal sciatic nerve pathologies are related to the piriformis, Martin suggested the term "deep gluteal syndrome" (DGS) in relation to chronic gluteal and lower extremity pain, which results from various forms of sciatic nerve entrapment as it courses under the gluteus maximus [62]. This includes entrapment from the piriformis, gluteus maximus, quadratus femoris, obturator or hamstring muscles, ligaments, blood vessels, hematomas, or neoplasms [63]. It also encompasses fibrous bands surrounding the sciatic nerve or posttraumatic scarring in the deep gluteal space [64, 65]. In summary, deep gluteal syndrome should be suspected in patients with posterior hip or leg pain, associated sciatic symptoms, and absence of lumbar pathology.

Distal Leg Syndromes

Injuries to the sciatic nerve or to one or more of its branches may occur more distally. Peripheral nerve entrapments of the lower extremity are relatively rare and consist of a heterogeneous group of nerve disorders. Peroneal nerve palsy is the most common entrapment neuropathy of the lower extremity and typically results from compressive pathology at the level of the fibular head [66]. A number of causes for peroneal nerve compression have been reported including external factors such as habitual leg crossing, prolonged squatting, and wearing tall boots or braces or internal factors such as exercise- or trauma-induced compartment syndromes, vascular abnormalities, and traumatic and surgical nerve injuries [51]. Depending on the etiology, the clinical presentation may consist of partial or complete foot drop, with or without associated pain, sensory abnormalities, and Tinel's sign. Early recognition of this condition is important since acute drop foot may also be the presenting sign of acute disc herniation which sometimes requires immediate surgical decompression of the affected nerve root. Other cases may have more gradual onset of symptoms. Careful medical and neurological examinations and imaging studies (MRI or ultrasound) can help identifying the underlying pathology. In contrast, nerve conduction studies can be normal and fail to show pathology in the early stages of acute nerve injury.

Entrapment or impingement of the superficial peroneal nerve (SPN) may occur in response to localized trauma to the nerve. It most commonly accompanies ankle sprains, mostly during inversion injuries which result in abnormal nerve traction. The typical clinical presentation is localized pain in the anterior part of the distal leg and dorsal foot, aggravated by activity. Motor weakness is typically absent. The course of the nerve may be sensitive to palpation and a positive Tinel sign can help establishing the diagnosis [67].

Deep peroneal nerve entrapment also known as anterior tarsal tunnel syndrome is another distinct pain syndrome of the distal extremity, whereby the nerve is impinged by osteophytes, ligamentous laxity, muscle hypertrophy trauma, and other factors while passing through the tarsal tunnel [51]. Pain and paresthesia in the dorsum of the foot during activity are typical, although night pain can also occur. Examination typically reveals pain exacerbation during plantarflexion of the foot, Tinel sign, and possible weakness of the extensor digitorum brevis muscle, depending on the site of injury. CT scan may show osteophytes or bone fractures and MRI may detect additional pathologies, but importantly, negative studies do not rule out the diagnosis. The same is true for EMNG although electro-physiologic evidence for abnormal EDB innervation supports the diagnosis.

Before closure of this section, it is important to note again that painful focal neuropathies mimicking radiculopathy are not caused exclusively by entrapments of isolated peripheral nerves. Hereditary, metabolic, infectious, autoimmune, and neoplastic diseases can underlie focal peripheral neuropathies, can all cause similar symptoms and signs, and should therefore be considered in the differential diagnosis of sciatica when no spinal abnormalities are found.

An Algorithm for the Diagnosis and Treatment of Patients with Back Pain with Leg Pain

The main challenge facing the clinician with a patient presenting with back pain and leg pain is to relate the patients' complaints to a nociceptive/somatic referred (NSR) syndrome without direct neural involvement versus a neural syndrome such as a radicular pain and peripheral nerve entrapment pain. It is interesting to note that Kellgren as far back as 1940 suggested a diagnostic algorithm quite similar to the one presented here [3] (Table 1).

The history may be suggestive of either somatic referred or neural origin pain but the physical examination is vital for refining the diagnosis. In general, we look for signs of neural involvement and deficit in motor, sensory, and reflex distributions. In addition, the use of straight leg raising (SLR) test, also known as the test of Lasègue, and the femoral stretch test is considered important for diagnosing radicular involvement,

Table 1 History taking—searching for clues as to NSR pain or neural origin pain

Symptom	Nociceptive/somatic referred (NSR) syndrome	Neural origin pain
Onset	Gradual or sudden	Gradual or sudden
Pain provocation	Mechanical challenge of the somatic structures	Pressure on the neural components such as standing, bending to involved side
Quality of pain	Dull, aching gnawing	Electric, tingling, lancinating, shocking, vice like
Location of pain	Back pain generally worse than leg pain	Leg pain generally worse than back pain
Pain referral	Referral with non-dermatomal distribution Often segmental distribution	Narrow band dermatomal distribution
Severity	Can be up to 10/10	Can be up to 10/10

NSR nociceptive/somatic referred pain

Table 2 Physical examination signs for differentiating between NSR pain or neural origin pain

Signs	Nociceptive/somatic referred (NSR) syndrome	Neural origin pain
Motor strength	Generally normal but can be mildly reduced (part of myofascial pain syndrome)	May be normal but also markedly reduced (quadriceps weakness, drop foot, drop ankle)
Tendon reflexes	Usually normal (knee jerk occasionally reduced with quadriceps trigger points)	Tendon reflexes reduced in involved segment (L3–4—knee, L5–S1—ankle)
Sensory deficit	Absent	May see hypoesthesia, dysesthesia in involved segment with dermatomal distribution
Neural stretch tests	SLR for sciatic stretch usually negative (more pain in back than leg). Femoral stretch usually negative—except for psoas involvement	SLR for sciatic stretch hallmark of radicular pain. Femoral stretch may be positive—upper lumbar segments
Taut bands	Very common in MFP	May co-exist
Trigger points	The hallmark of MFP	May co-exist

NSR nociceptive/somatic referred pain, *MFP* myofascial pain syndrome, *SLR* straight leg raising

despite the low specificity for these tests [68]. For somatic referred pain, the main findings are of tenderness in appropriate structures and evidence of pain referral under palpatory challenge; thus, we search for taut bands and trigger points (Table 2).

Refining the history and physical examination will lead us to a proposed diagnosis and differential diagnosis. We will then turn to the accessory tests as needed—imaging studies, nerve conduction, and electromyography. These studies are not essential but depend on the clinical setting—helping settle diagnostic dilemmas or guidance for treatment procedures.

Treatment will be allocated depending on the diagnosis—if somatic referred pain is the primary diagnosis, treatment should be directed towards increasing mobility and a quick return to the previous level of function. If neural involvement and deficit is suspected, treatment would depend on the degree and severity of the deficit and can range from conservative measures such as medication and mobilization and up to surgery if deemed necessary.

Discussion

The common complaint of back pain with leg pain poses a diagnostic dilemma for the clinician. In the days prior to advanced radiology such as computerized tomography scanning and magnetic resonance imaging, in most cases of back pain with leg pain, the referred component was considered to be of somatic origin. Thus, Kellgren, Steindler, and Good have all recorded high incidences of back pain with leg pain as being of somatic origins [11•, 13, 69, 70]. Later studies demonstrated the referral of pain down the leg from interspinous ligaments in a manner most reminiscent of radicular pain [12, 71]. In patients with back pain and leg pain, we clearly need to entertain a large possibility for the diagnosis to be one of somatic referred pain [72] (Fig. 3).

With the advent of advanced spinal imaging, starting in the 1970s with computer tomography (CT), and the first report documenting the ability to recognize herniated lumber discs in 1979 [73], thought has turned to radicular syndrome as the main cause of pain referring from the back to the leg. We can see this as early as 1988 when Frymoyer wrote on sciatica and mentioned only radicular causes relevant to this clinical entity. He quoted from Smyth that "Sciatica requires mechanical and inflammatory stimuli to the anterior primary rami of lumbar nerve roots" [33, 74]. More recent articles have also named the nerve root almost exclusively as the cause for back pain and leg pain—under the name of sciatica [17, 75].

The case vignette presented at the beginning of this article has signs and symptoms that could easily belong to both a radicular syndrome such as LIDH of the lumber fifth nerve root as well as signs of somatic referred pain from the gluteus medius and minimus muscles. Since these diagnostic entities may coexist, and since there is no evidence of imminent danger such as acute radiculopathy, the patient can be followed and treated conservatively. If the condition deteriorates, the plan for the patient should include a diagnostic arm such as imaging, according to the recommendations of the American College of Physicians [76]. The therapeutic arm of this patient's plan can include reassurance, medications, physical therapy, and even an epidural steroid injection, but a detailed discussion of treatment options is beyond the scope of this article.

Conclusion

In this article, we have shown that the differential diagnosis of patients suffering from back pain with leg pain must include conditions as discussed by Bogduk [2••], which is somatic referred pain, radicular pain, and radiculopathy and indeed other causes of pain involving neural pathways. We must also be aware of overlap syndromes where patients with radicular

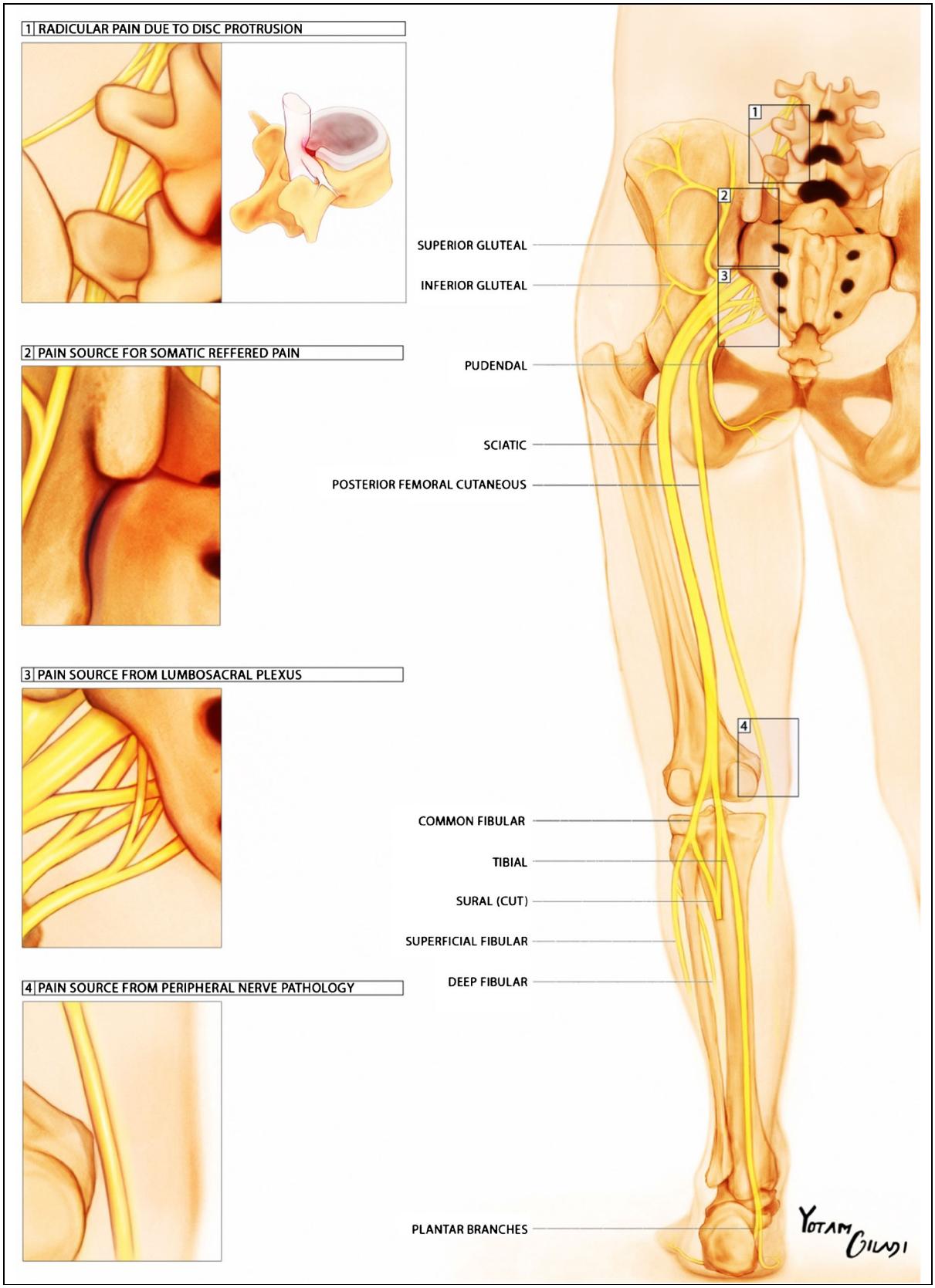

Fig. 3 Sources of back pain and leg pain

pain also exhibit manifestations of myofascial pain such as taut bands and trigger points [77•, 78]. Systematic history taking and physical examination including neurological assessment should be enough to define a working hypothesis and a differential diagnosis. Further studies should be performed as deemed clinically necessary. Often, patients will present with symptoms of both neural and somatic origins, and the clinician will have to decide how to proceed in his treatment.

Compliance with Ethical Standards

Conflict of Interest Simon Vulfsons, Negev Bar, and Elon Eisenberg declare that they have no conflicts of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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