

# Degenerative lumbar spinal stenosis and its imposters: three case studies

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*Degenerative lumbar spinal stenosis causing neurogenic claudication is a common condition impacting walking ability in older adults. There are other highly prevalent conditions in this patient population that have similar signs and symptoms and cause limited walking ability. The purpose of this study is to highlight the diagnostic challenges using three case studies of older adults who present with limited walking ability who have imaging evidence of degenerative lumbar spinal stenosis.*

*La sténose lombaire dégénérative à la base d'une claudication d'origine neuronale est une condition fréquente affectant la faculté de la marche chez les adultes âgés. Les patients de cette catégorie peuvent être affectés par d'autres maladies très courantes qui présentent des signes et des symptômes similaires, et qui restreignent leur capacité de marche. Le but de cette étude est de souligner les difficultés en diagnostic au moyen de trois études de cas de personnes âgées qui présentent une capacité de marche réduite et qui souffrent de sténose lombaire dégénérative mise en évidence par imagerie médicale.*

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## Introduction

Degenerative lumbar spinal stenosis (DLSS) is a leading cause of pain, disability, and loss of independence in older adults.<sup>1</sup> The prevalence and economic burden of DLSS is growing exponentially due to the aging population. It is a chronic disease caused by age related degenerative

narrowing (stenosis) of the spinal canal that can lead to compression and ischemia of the spinal nerves (neuro-ischemia).<sup>2</sup> The clinical syndrome of DLSS is known as neurogenic claudication. It is characterized by bilateral or unilateral buttock, lower extremity pain, heaviness, numbness, tingling or weakness, precipitated by walk-

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ing and standing<sup>3</sup> and relieved by sitting and bending forward<sup>4,5</sup>. Lower back pain is not necessarily associated with neurogenic claudication. Limited walking ability is the dominant functional impairment caused by DLSS.<sup>4</sup> There are many other common conditions in the elderly that also give rise to lower extremity symptoms and limited walking ability.<sup>4,5</sup> Often more than one condition can be present at the same time which makes diagnosis even more challenging. The ability to accurately diagnose DLSS and the various other conditions that give rise to similar symptoms and limitations is paramount for appropriate treatment. The purpose of this study is to demonstrate using three case studies the challenges when presented with an elderly patient who presents with back pain, lower extremity symptoms, and limited walking ability. Signed informed consent was obtained from three patients whose cases are presented in this study.

### Case 1

Ms. AK is a 73 year old retired public health nurse who presents with chronic episodic lower back pain and a two year history of increasing bilateral calf pain. The calf pain comes on after a few minutes of walking and intensifies as she continues to walk. The calf pain is immediately relieved with sitting or lying down. She reports three bouts of severe sciatica over the past three years and otherwise experiences recurrent low grade backache. She was being treated for a recurrent infection of the right toe. She has had previous chiropractic treatment that included manual therapy, acupuncture, and flexion exercises with no help. She is normally a very active person but frustrated with her limited ability to walk. She takes high blood pressure and cholesterol lowering medication. Ms. AK had a previous history of Raynaud's Syndrome involving the upper extremities, and was under the care of a rheumatologist for Sjogrens Disease which was in remission. She had a previous arterial Doppler ultrasound test of the lower extremities, the results were equivocal. She claims to be healthy otherwise.

On examination, she is slim built, stand with a flattened lumbar lordosis and has difficulty with balance testing. She can toe-heel walk and squat without difficulty. Range of motion (ROM) of the lumbar spine is full and painless during forward flexion. Lumbar extension is moderately limited and reproduces lumbo-sacral back pain but not lower extremity symptoms. Sitting straight leg rais-

ing (SLR)<sup>6</sup> is full and painless bilaterally. Supine SLR<sup>6</sup> is mildly limited by tight hamstring muscles bilaterally. End range SLR with dorsiflexion of the foot<sup>6</sup> reproducing moderate calf pain bilaterally. There is an absent left Achilles reflex with no evidence of lower extremity sensory or motor deficits. No atrophy of the calf muscles are noted and during palpation of the lower extremities pulses appear present and bilaterally equal and her feet appeared warm. Hip examination reveals full and painless ROM. Moderate tenderness is noted during palpation of the L4-5 and L5-S1 spinal segments. An MRI performed two year earlier revealed severe multi-level degenerative joint and disc disease with severe lateral recess stenosis at L4-5 and L5-S1.

At tentative diagnosis of neurogenic claudication due to degenerative lumbar spinal stenosis was given and a treatment program of flexion-distraction/ side posture spinal mobilization/ manipulation, neural mobilization, flexion based home exercises including a progressive stationary cycling program was prescribed. After 6 weeks of treatment at a frequency of 2-3 treatments per week she was re-evaluated. Using a self-report improvement scale (completely better, much improved, slightly improved, no change, slightly worse, much worse and worse than ever)<sup>7</sup> she reported no significant change in her calf pain or walking ability. She stated she was compliant with her exercises except the stationary cycling program. She was referred to her family doctor for another arterial Doppler ultrasound test which revealed moderate bilateral tibial obstructive artery disease. She was subsequently referred to a vascular surgeon who confirmed the diagnosis and initiated a trial therapy with cilostazol 100 mg twice a day to improve blood flow. Response to the medication was excellent with little lower extremity symptoms or limitations during walking.

### Case 2

Mr. JP is a 62 year old consultant who presents with a 30 year history of episodic lower back pain and nine month history of progressive right lateral thigh and leg pain. Over the past 6 months both lower back and leg pain intensifies with walking. He starts to limp after several metres and now his walking is limited to about 20 metres. The pain is described as burning and achy and appeared to travel from the back along the lateral hip and occasionally into the knee, groin and into the right foot. Stretching and

swimming provide temporary relief. Associated symptoms included urinary hesitancy which he has had on and off for 15 years. He had tried physiotherapy, chiropractic, and acupuncture without success. He takes neuropathic medication and narcotics for pain control. He states he is otherwise healthy.

On examination, he stands with a flexed posture and walks with a left leaning gait. ROM of the lumbar spine is full (finger tips reaching toes) and painless during flexion. Lumbar extension is moderately limited and reproduces moderate lower back pain not leg pain. Supine SLR<sup>6</sup> and prone femoral nerve stretch<sup>8</sup> are full and painless with no evidence of nerve tension signs bilaterally. Moderate muscle hypertonicity is noted over the right piriformis muscle. There is mild restriction in internal rotation and flexion of the right hip with minimal pain at the end range. Quadriceps reflexes are 2+ and bilaterally equal. Achilles reflexes could not be elicited bilaterally. No lower extremity sensory deficits are noted. There is mild atrophy of the right calf and hamstring muscles. A recent MRI of the lumbar spine revealed congenitally narrowed pedicles and severe multilevel spondylosis of lumbar spine with severe central spinal stenosis at L2-3 with associated neural compression and moderate foraminal stenosis at right L4-5 and bilaterally at L5-S1. According to Mr. JP a recent right hip x-ray revealed no significant abnormalities, (his wife is a radiologist). However this was not confirmed.

A tentative diagnosis of neurogenic claudication due to degenerative lumbar spinal stenosis with underlying congenitally narrowed pedicles was given and a six week (twice per week) treatment program consisting of flexion-distraction and side posture spinal mobilization/manipulation, neural mobilization of the femoral and sciatic nerves and, flexion based home exercises was started. After several weeks of treatment no improvement was noted. The patient was re-evaluated and a moderate deterioration in ROM (subjectively assessed) of the right hip was noted especially during internal rotation and flexion with moderate pain elicited at the end range. Combined flexion, abduction, external rotation also was moderate restricted and reproduced moderate pain. An x-ray of the right hip was performed which indicated moderate degenerative joint disease. Several months later Mr. JP received a successful total right hip replacement which significantly improved his lower extremity symptoms and walking ability.

### Case 3

Ms. NK is a 71 year old retired physical therapist who presents with an 18 months history of left lateral thigh and chronic low back pain. The thigh pain is described as a dull nagging ache made worse with prolonged walking, stair climbing, getting out of a car, and lying in bed. The pain occasionally radiates to the lateral left knee and limits her walking ability. The lower back pain is described as a steady dull nagging ache worse with physical activity, twisting actions, and prolonged sitting. She has tried a lumbar epidural steroid injection, massage therapy, anti-inflammatory medication, and acupuncture with no significant improvement in symptoms. She is otherwise healthy.

On examination she stands with a flat lumbar lordosis and mild scoliosis. She is able to heel-toe walk and squat without difficulty. ROM of the lumbar spine is mildly restricted and painful during forward flexion. Moderate pain is elicited during lumbar extension without reproducing her left leg pain. There is moderate tenderness over the L4-L5 and L5-S1 articulations during deep palpation. There is moderate-severe pain elicited during palpation over the left trochanteric bursa and tensor fascia lata. Ms. NK indicated that this pain is similar to the leg pain experienced during walking. Hip examination revealed mild pain at the end range of external rotation of the left hip. Neurological examination is unremarkable. Supine SLR with dorsi-flexion of the foot<sup>6</sup> did not reproduce any leg symptoms bilaterally. An MRI revealed degenerative joint and disc disease throughout the lumbar spine with a partial sequestered right L4-L5 para-central disc herniation giving rise to moderate lateral recess stenosis on the right with potential compression of the descending L5 nerve root. Similar findings were noted at the L5-S1 level with mild compression of the existing right L5 nerve.

A working diagnosis was moderate left trochanteric bursitis and chronic mechanical low back pain with moderate degenerative joint and disc disease. Her leg symptoms and limited walking ability appeared to be primarily due to the trochanteric bursitis and not due to the lumbar spinal stenosis. She began a treatment program of twice per week for six weeks consisting of deep cross-fiber massage over the left trochanteric bursa and tensor fascia lata, home stretches and icing, manual therapy directed to the lumbar spine and a home based lumbar spine exercise program. After six weeks of therapy there was slight

improvement, using a self report improvement scale<sup>7</sup> in her symptoms and walking ability.

## Discussion

This study highlighted three common conditions that present with similar symptoms that are often misdiagnosed as caused by degenerative lumbar spinal stenosis. All three cases involved older adults who had lower back pain, lower extremity symptoms that limited walking ability (claudication) and moderate-severe degenerative lumbar stenosis on imaging.

A common reason for misdiagnosis is the interpretation of findings on imaging, particularly MRI and CT scan. Degenerative changes seen on imaging of the lumbar spine including degenerative central canal and lateral recess narrowing are common in older adults and often do not correlate with patient symptoms. Moderate lumbar spinal stenosis is noted in up to 30% of asymptomatic individuals over the age of 55.<sup>9</sup> Therefore imaging is not a reliable modality for the diagnosis of lumbar spinal stenosis causing neurogenic claudication. A diagnosis of neurogenic claudication is made clinically from a thorough history and physical examination and not solely by imaging evidence of spinal stenosis.<sup>5</sup> Important clinical features include age over 70, bilateral buttock or leg pain, no pain when seated, symptoms worse standing/walking, symptoms improve when bending forward, wide stance gait and urinary disturbances.<sup>5</sup>

An understanding of the dynamic nature of neurogenic claudication and a comprehensive evaluation of other potential sources of symptoms and limited walking ability is paramount to appropriate diagnosis. The dynamic nature of neurogenic claudication refers to the reduction or elimination of lower extremity symptoms with sitting or leaning forward during walking or standing.<sup>4</sup> This is a result of an increase in the cross sectional area of the lumbar spine with lumbar flexion which reduces compression to the spinal nerves.<sup>10,11</sup> This phenomenon is also demonstrated with the *shopping cart sign* which refers to the reduced symptoms and increased walking ability when leaning forward on a shopping cart.<sup>4,5,11</sup> Supine and sitting straight leg raising tests<sup>6</sup> is usually negative in neurogenic claudication because these maneuvers introduces flexion to the lumbar spine and reduces neuro-ischemic compression.<sup>4,5</sup> Results of lower extremity sensation and strength testing is variable in neurogenic claudication but deficits

are usually seen in more severe or long standing cases and usually correlates to the involved nerve roots. The same holds true when assessing deep tendon reflexes of the lower extremities which tend to be difficult to elicit in older individuals in general. Another important feature associated with neurogenic claudication is loss of balance<sup>5</sup> which is due to impaired proprioception secondary to neuro-ischemia of spinal nerve roots<sup>12</sup>.

Other common conditions can have similar symptoms and walking impairments.

### *Case 1. Peripheral vascular disease*

In Case 1, the patient's main symptoms were a result of peripheral vascular disease (PVD). PVD causing intermittent claudication is common in older adults with the prevalence growing significantly due to the aging population.<sup>13</sup> The risk of PVD is high among patients with diabetes mellitus, hypertension, hyperlipidemia, smoking and vasculitis due to auto-immune disease.<sup>14</sup> Individuals with PVD have higher mortality rates and therefore early diagnosis and treatment is essential.<sup>15</sup> In PVD, claudication symptoms are a result of ischemia to the lower extremities muscles which worsen with walking and alleviated with rest.<sup>16</sup> This symptom pattern is not unlike that of neurogenic claudication.<sup>4</sup> Moreover, the two conditions can often co-exist making diagnosis even more challenging. A recent study demonstrated that 26% of individuals with confirmed neurogenic claudication due to lumbar spinal stenosis have also objective signs of PVD.<sup>14</sup> Although assessment of peripheral lower extremity pulses is recommended, 8% of individuals with no PVD have dorsal pedis pulses that are not palpable and 10% of individuals with normal pulses have PVD.<sup>14,17,18</sup> A more accurate in office method to assess for lower extremity PVD is using the ankle-brachial and toe-brachial indexes. A blood pressure cuff is used to assess the ratio of systolic blood pressure at the two anatomical locations. Ratios less than 0.9 are considered positive for PVD with the toe-brachial index demonstrating more accuracy.<sup>14</sup> Referral for an arterial Doppler test is recommended for confirmation of the diagnosis. Arterial Doppler testing in individuals with at least 50% lower leg vascular occlusion has a sensitivity ranging from 80 to 98% and specificity from 89 to 99% for PVD.<sup>19</sup> Ischemic related skin discoloration and skin infections of the lower extremities, particularly of the feet, as in our Case 1 may help in the diagnosis. Features

of the history may also be useful. Using a shopping cart, stationary bike or walking uphill is not expected to improve symptoms in PVD but tend to reduce symptoms in neurogenic claudication.

### ***Case 2. Hip-Spine Syndrome***

Case 2 has hip-spine syndrome. Hip-spine syndrome refers to the coexistence of radiographic osteoarthritis (OA) of the hip and degenerative stenosis of the lumbar spine. Both degenerative conditions can result in buttock, groin, lateral hip, and leg pain and limited walking ability. The prevalence of radiographic hip OA is 27% in adults 45 years of age or older<sup>20</sup> of which 9.2% are symptomatic<sup>21</sup>. Therefore, like imaging for DLSS, radiographic findings of the hip must be correlated with symptoms and physical examination.<sup>22</sup> 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.<sup>23</sup> A more recent study using fluoroscopic guided intra-articular injections among patients with known hip pathology demonstrated that the buttock region was the most common anatomical location of referred hip pathology (71%) followed by combined thigh and groin pain (55%).<sup>23</sup> In another study, 47% of patients with isolated hip arthritis reported pain radiating below the knee.<sup>24</sup> DLSS rarely refers pain to the groin unless there is involvement of the L1-2 level. Buttock and lateral hip pain however, is a very common area of radicular pain due to DLSS. These findings emphasize the challenges in distinguishing the main source of symptoms by pain distribution alone. Physical examination can be useful in distinguishing the main pain generator. OA of the hip is usually associated with reproduction of symptoms during weight bearing and a limping gait. Passive hip flexion and internal rotation is usually limited in range and reproduces the patients' symptoms. Pain can also be reproduced when turning from a supine to side position on the exam table (or in bed) which often requires internal rotation and flexion of the hip. Patients with DLSS tend to be asymptomatic when lying or turning in bed.<sup>25</sup> Stooped forward posture can be associated with both OA of the hip and DLSS. OA of the hip can lead to contractures of the hip flexors leading to anterior leaning posture.<sup>26</sup> Muscle atrophy of the para-hip musculature can also be seen in both conditions: disuse atrophy in the case of OA of the hip and radiculopathy induced atrophy in the case of neurogenic claudication. When the diagnosis is still

unclear electrophysiological studies can be performed. Normal nerve conduction and electromyographic studies do not rule out neurogenic claudication whereas findings of radiculopathy can be indicative of this process.<sup>27</sup>

A more invasive diagnostic approach involves fluoroscopic guided hip anesthetic injections. A significant relief of symptoms following an intra-articular hip bupivacaine injection is reported to have a sensitivity of 87% and a specificity of 100% in diagnosing hip OA as the primary pain generator.<sup>28</sup> On the other hand epidural spinal anesthetic injections with or with steroids are less useful in neurogenic claudication since the etiology is primarily due to neuro-ischemia not inflammation.<sup>26</sup>

### ***Case 3. Trochanter bursitis or greater trochanteric syndrome***

In Case 3, the lateral hip pain is most likely due to greater trochanteric pain syndrome (GTPS). GTPS is a term used to describe chronic pain overlying the lateral aspect of the hip. This regional pain syndrome, once described as trochanteric bursitis, often mimics pain generated from spinal pathology including degenerative lumbar spinal stenosis.<sup>29</sup> The term GTPS is suggested to better characterize this condition which is described as reproducible tenderness in the region of the great trochanter in light of the inherent difficulties in localizing the true cause of the pain. Pain generators include any one of the nine bursae, muscles, and tendons that attached to the greater trochanter and surrounding areas.<sup>30</sup> The pain can travel along the lateral hip to the knee in 50% of cases<sup>29</sup> and occasionally below the knee<sup>31</sup>. It is estimated that GTPS affects between 10% and 25% of the population in industrialized societies<sup>32</sup> with significantly higher prevalence in the elderly<sup>33</sup>. GTPS is the second leading cause of hip pain in adults.<sup>33</sup> Risk factors are increasing age, female gender, ipsilateral ilio tibial band pain, knee OA, obesity and low back pain.<sup>32</sup> Mechanisms of GTPS include chronic micro-trauma, regional muscle dysfunction, overuse or acute injury.<sup>32</sup>

In addition to palpation (jump sign), pain can be reproduced by active and resisted abduction, passive abduction of the hip<sup>34</sup> and during combined passive flexion, abduction, external rotation and extension (FABERE)<sup>35</sup>. A Trendelenberg sign (when standing on one leg, the pelvis drops on the side opposite to the stance leg) is often associated with GTPS especially with lateral hip tendon tears.<sup>29</sup>

However, other than point tenderness there are very few diagnostics tests with high specificity for GTPS.<sup>36</sup> A key diagnostics feature that can distinguish GTPS from neurogenic claudication is lateral hip pain with lying on the affective side. Those with GTPS will often complain of night pain and difficulties sleeping because of increased pain where as individuals with neurogenic claudication are usually asymptomatic when lying down. Other distinguishing features include aggravation of GTPS during stair climbing, getting up from a seated position and cycling which causes repetitive rubbing the of iliotibial band over the greater trochanter. These activities generally do not aggravate symptoms in neurogenic claudication due to the flexed posture. A steroid and or anesthetic injections are often used for both diagnosis and therapy but the evidence for their effectiveness generally comes from lower quality evidence.<sup>33,37</sup>

Other conditions that need to be ruled out when assessing the elderly patient with lower extremity symptoms include, diabetic neuropathy, meralgia paresthetica radiculopathy due to lumbar disc herniation, cervical spinal stenosis, knee OA and degenerative facet and sacroiliac joints.

Once the diagnosis of neurogenic claudication is made and other potential conditions ruled out appropriate treatment can be implemented. Treatments for neurogenic claudication include surgical and non surgical. The effectiveness of non surgical treatments including physical therapy, chiropractic, exercise, medication, epidural injections is unknown.<sup>38-42</sup> A rational approach would be to provide instruction on lumbar flexion and core stabilization exercises and overall fitness (using of a stationary forward leaning bike), provide therapy to improve lumbar spine flexibility and instruction on self management strategies to avoid lumbar extension and reduce the lumbar lordosis when standing and walking. Surgical interventions include direct and indirect decompression with and without fusion. Carefully selected patients with leg dominant symptoms usually improve with surgery however; the benefits tend to diminish over time.<sup>4</sup>

## Conclusions

DLSS causing neurogenic claudication is a leading cause of lower extremity symptoms and limited walking ability in the elderly. Other common conditions such as a

PVD, hip osteoarthritis, and GTPS can also give rise to similar symptoms and restricted walking ability which makes identifying the main source of symptoms a challenge in the older population. A careful and thorough history and physical examination, and keen understanding of the underlying pathoanatomy and pathophysiology of the common conditions is paramount for accurate diagnosis and appropriate management.

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