

Piriformis Muscle Syndrome, Post Discectomy: a Comprehensive Review of its Diagnostic Process and Management, Illustrated with the Aid of a Case Report

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Abstract

This review, with the help of a case study, provides a detailed account of the possible biopsychosocial risk factors underlying piriformis muscle syndrome, post discectomy. The diagnostic process including imaging, nerve conduction studies, musculoskeletal examination coupled with provocation maneuvers, palpation, and the musculoskeletal differential diagnosis of buttock pain is illustrated. Reference is made to the aberrant sciatic nerve anatomy and to the myofascial component which is frequently reported to underlie piriformis muscle syndrome. Additionally, the use of validated neuropathic pain assessment tools in grading the possibility of neuropathic pain due to piriformis muscle syndrome is discussed. The prevalence and mechanisms of piriformis muscle syndrome, especially post spinal surgery, are reviewed. Evidence-based multidisciplinary management underlying this condition is discussed. In refractory cases, treatment escalation, using intramuscular botulinum neurotoxin injection, usually yields very positive results and long-term pain resolution, as evidenced by the case study.

Keywords Piriformis muscle syndrome · Piriformis syndrome · Botulinum toxin · Failed back surgery syndrome · Discectomy

Introduction

This review, with an integrated case report of a patient suffering predominantly from piriformis muscle syndrome (PMS) post discectomy, will detail the latest evidence-based risk factors, differential diagnosis, and management of PMS.

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Case Study Assessment (Supplementary Material)

Patient History

The patient is a 37-year-old male, who works as an office clerk on a full-time basis. The patient's low back pain (LBP) started insidiously in 2005. He was prescribed celecoxib 400 mg daily, to no avail. Soon afterwards, a conventional supine magnetic resonance imaging (MRI) scan without myelography showed a moderately sized posterior disc herniation at L5/S1, impinging on the left S1 root. Three years later, the patient underwent an L5/S1 discectomy, due to severe LBP with radiculopathy. The patient recovered relatively well but still suffered from frequent episodes of LBP.

Following a fall, he fractured the left tibia, increasing his LBP. A repeat spine and pelvis conventional supine MRI without myelography did not show any recurrence of disc herniation. Following the recovery from the fracture, he started to attend Pilates classes which decreased his LBP but caused moderate to severe neck pain. Consequently, he soon stopped attending classes as he felt that this was increasing his pain, fearing that the exercises could jeopardize his spine.

Subjective Assessment

Currently, his main complaint is left-sided, deep-seated, severe buttock pain, aching in nature, of relatively stable intensity with frequent pain attacks lasting for about half an hour. He complained of moderate intensity, non-specific, mechanical LBP too. The short-form McGill pain questionnaire was used and the following descriptors were chosen for his buttock pain:

- Aching—severe
- Tender—severe
- Tiring/exhausting—severe
- Punishing—severe

The visual analog scale (VAS) for the hip pain was 9.3 out of 10. Although he did not mention any associated physical features, his buttock pain is described as “mood altering pain” portrayed in his Hospital Anxiety and Depression score (HADS) ([Supplementary Material](#), page 3).

The pain is eased temporarily by heat and bed rest. Paracetamol 1 g daily in the morning and pregabalin 150 mg/day decreased his hip pain slightly. His left buttock pain is aggravated by sitting for more than 10–20 min, especially on hard surfaces and during cross-legged sitting. His LBP increased with prolonged standing and spasms after 15-min walking. He complains of daily morning stiffness lasting for 30 min. He spends much time in bed, and he avoids lifting heavy objects or doing house maintenance works. He underwent intra-articular steroid facet joint infiltrations and a medial branch block which decreased his LBP slightly.

The patient is a clerk and currently employed within the family run business. Thus, he felt safe and understood at work. However, he feels isolated from his friends, as he cannot continue to play the occasional football game with them due to the pain. He acknowledged his wife for her great support and the more significant burden of the housework was relying on her.

Objective Assessment

The lumbar spine was screened considering its potential role in referring pain distally [1]. There was the absence of the flexion-relaxation phenomenon [2], which was evidenced by an excursion of 1 cm on the Schober test, while the normal range for this test should be at least 5 cm [3]. Lumbar extension increased his LBP. This could suggest a facet joint pathology, but the pain during lumbar extension was partly decreased by placing the patient’s left leg on a stool. This coupled with a reduction in the range of motion (ROM) in both passive hip extension and the femoral stretch test, implicating a shortened left iliopsoas muscle. In addition, the patient had stiff left hip flexor muscle, tightness in both hamstrings, and both piriformis muscles coupled with abdominal muscle weakness, marked gluteal, and erector spinae muscle

atrophy. The hip joint had a marked limitation of passive lateral rotation and a positive Flexion Adduction Internal Rotation (FAIR) maneuver. He had hypoesthesia to brush, vibration, and pinprick in the lateral aspects of both feet, therefore implicating the S1 dermatome bilaterally. The myotomes corresponding to the lumbar levels exhibited weakness in the left hip extension and hip abduction but were normal on the right side. Furthermore, bilateral standing plantarflexion was normal, but single-leg standing plantarflexion was graded as 3 on the Oxford scale in both lower limbs. The remaining myotomes being hip flexion, knee extension and flexion, extensor hallucis longus extension, and ankle dorsiflexion were normal bilaterally. The straight leg raise (SLR) test was limited to 40° on the left-hand side and to 50° on the right-hand side. However, the SLR test did not reproduce radicular pain; on the contrary, there was a pulling sensation in the corresponding hamstring muscle of the tested leg, which was not increased by sensitizing movements. Patellar tendon reflexes were normal bilaterally; however, the Achilles tendon reflexes were absent bilaterally.

The Diagnostic Process

The presence of red flags, despite their debated accuracy, was excluded through history taking and examination [4]. The persistence of pain despite appropriate surgical intervention is the basis of failed back surgery syndrome (FBSS) [5], which becomes the primary focus of the diagnosis [6]. Thus, the International Association for the Study of Pain (IASP) diagnosis [7] is:

Severe Lumbar Spinal Pain after Failed Spinal Surgery of more than 6 month’s duration. 533.91gS.

The reduced passive hip lateral rotation could be caused by shortened hip internal rotators. The piriformis muscle (PM) becomes an internal hip rotator above 60° of hip flexion, while it acts as an external hip rotator below this angle [8]. Also, the PM abducts the femur with hip flexion and additionally tilts anteriorly and rotates the sacrum to the opposite side together with the ipsilateral gluteus maximus [9]. Nonetheless, the action of the PM during hip flexion is debatable [10]. The tight ipsilateral iliopsoas muscle can cause reciprocal inhibition of the weak gluteal muscles [11]. Gluteal muscle weakness affects hip stability by increasing the internal femoral rotation and the valgus stress on the limb, further stretching the hip external rotators, including the PM [12].

The positive FAIR test indicates the possibility of PMS, which can be validated by a local diagnostic injection [13] especially when the imaging and/or electrophysiological modalities are normal [14]. However, the clinical diagnosis of PMS does not have a gold standard test [15], and the

diagnostic process is by exclusion [16]. The PM originates from the anterior surface of the sacrum, and its tendon attaches to the medial aspect of the greater trochanter of the hip [17].

A combination of history, physical examination of the sciatic notch region, eliciting tenderness, coupled with various provocative maneuvers that tension the PM, can be used to support the diagnosis of PMS [18]. If three of the FAIR test, Freiberg test, Lasègue test, and Beatty test, are positive, the diagnosis is PMS [19, 20]. The FAIR test is shown to be the most sensitive test for diagnosing PMS [21]. The presence of the quartet of symptoms being buttock pain, the pain increased by sitting, tenderness near the greater sciatic notch, and any maneuver that increases tension in the PM, make the diagnosis of PMS highly likely [10]. Furthermore, his GP had previously conducted hematology tests including C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR), with normal results.

Differential Diagnosis of PMS

The differential diagnosis included the sacroiliac joint (SIJ) [22]. Although maneuvers that assess the SIJ have moderate evidence [23], their absence in eliciting pain eliminates the SIJ as a source of pain [24]. Obturator internus muscle (OIM) has a close anatomical relationship to PM [25]. Contrary to PM, pain originating from the OIM is aggravated in the lateral recumbent position, while the trigger point of OIM is found more caudal [26].

PMS can be mistaken for a disc herniation, lumbar disc disease, radiculopathy, stenosis, facet joint arthropathy, neuropathic pain (NP), neural sensitization, and referred pain from the lumbar spine [18]. The zygapophyseal joints can only be definitely diagnosed with an intra-articular block [7]. The painful lumbar extension as a provocation test of the facet joints must be interpreted in the light of the poor sensitivity and specificity of these tests [27]. The absence of centralization decreases the likelihood of the discogenic pain [28]. However, discogenic pain lacks precise diagnostic criteria, terminology, and uniform treatment [29]; therefore, according to IASP [7], discogenic LBP can only be diagnosed by provocation discography or by selective anesthetization. In addition, the patient's pain started in his buttock, and it has progressed proximally, which is not the usual presentation of radicular symptoms [1].

Electrodiagnostic tests can be used to rule out radiculopathies [22], but their role in diagnosing PMS is mixed and equivocal [18] since frequently, these are normal in PMS [16]. However, patients with PMS can be easily distinguished from normal subjects by performing electrophysiological tests with the patients in the FAIR position. In patients without PMS, the FAIR position was found to have little or no effect on the H-reflex. On the other hand, patients with PMS having sufficient pressure on the sciatic nerve causes a

functional prolongation of the posterior tibial or fibular nerve H-reflex that was three standard deviations or more beyond the mean during the FAIR test [30].

Imaging is frequently normal in PMS [16], and the patient's previous conventional supine MRI scan without myelography ruled out most of the spinal and hip pathologies [22]. Patients with variant proximal sciatic nerve anatomy are commonly asymptomatic in terms of PMS [31]. A recent radiologic study [32] noted no significant differences in the prevalence of PMS, buttock pain, or sciatica between normal and variant sciatic nerve anatomy. Sciatic nerve anatomy variants are present in about 19% of the population, where 97% of the variant anatomy cases correspond to the type II Beaton and Anson category, where the common peroneal nerve passes through the PM, instead of passing anterior to the muscle along with the tibial nerve [32]. Similarly, a study on 755 consecutive scans found that 87% of the cases had normal anatomy (type I), while in 13%, a type II variant anatomy was present [33].

Nevertheless, the patient's MRI scan did not report any aberrant passage of the sciatic nerve. It is the opinion of many authors that sciatic nerve entrapment is not vital for diagnosing PMS and that this condition is mostly of myofascial origin [14, 34, 35]. Therefore, PMS can exist without radiation to the leg [10].

The limited SLR ROM cannot be used to exclude the PM in favor of a radicular component [10] since tight hamstrings increase the chance of a false-positive result [36]. Buttock pain is significantly and positively associated with NP regardless the presence of leg pain [37], in fact, NP is present in 41.1% of soft tissue syndromes, e.g., PMS [38]. The DN4 due to its high sensitivity and specificity [39] was used to screen for NP [40]. The patient scored 0/10, making the presence of NP unlikely. However, the patient is taking pregabalin which decreased the buttock pain slightly, jeopardizing the DN4 diagnostic value [41].

On the other hand, the updated IASP grading system to diagnose the possibility of NP [42] would categorize this patient as having probable NP, since he has a history of a relevant neurological lesion and pain distribution in a neuroanatomically plausible distribution, yet it is debatable if the altered sensory signs are in the same distribution. This is because certain dermatome charts depict the S1 dermatome originating from the buttock area, while other charts do not. However, in patients with S1 nerve root lesions, most of their pain is located in the buttock area [43]. The patient also had the MRI scan previous to the discectomy showing impingement on the S1 nerve root.

The presence of bilateral hypoesthesia over the lateral toes coupled with the absence of both Achilles deep tendon reflexes indicates maladaptive changes in the S1 nerve root, possibly due to a polyneuropathy, which would necessitate electromyography and nerve conduction studies. The latter

tests apart from confirming or rejecting the diagnosis of a polyneuropathy are helpful in excluding an S1 radiculopathy [44]. In addition, the presence of the left side hip abductor weakness coupled with gluteal atrophy could be due to L5/S1 nerve roots radiculopathy [15], but the presence of a painful radiculopathy was excluded due to good distal myotome power. Nonetheless, the altered distal sensation is of doubtful clinical significance considering the patient never complained of pain radiating down to his toes coupled with the absence of radicular pain in both the crossed SLR and SLR and the DN4 score of 0 decreases the likelihood of radicular pain [45]. Thus, an additional IASP, diagnosis [7] is:

Severe Piriformis Syndrome of more than 6 month's duration. 632.91f.

The Patient's Psychosocial Aspects

His possible preoperative and current psychological state could partly explain the transition to chronic disabling pain [46]. There is a bidirectional association between chronic pain and depression [47] with chronic pain being prevalent in up to 70% of persons, who like our patient have high anxious and depressive disturbances, as evidenced by the HAD score (Supplementary Material, page 3) [48]. The severity of his psychological symptoms portrayed in the phrase "mood altering pain" amplifies and perpetuates his suffering [49]. His poor self-coping and pacing strategies coupled with failed previous biomedical treatment augment his disability [50]. Though he acknowledges his supportive wife, the dyspareunia could stress his marriage. The lack of a proper biopsychosocial (BPS) diagnosis coupled with untreated psychological aspects reduced the effectiveness of his previous treatments. This enhances a catastrophic state [51] which is a crucial step in the fear-avoidance model [52], reducing his social and occupational functioning and further deconditions his muscles [53].

Prevalence of FBSS and PMS

Discectomies generally provide short-term pain relief [54] since approximately 75% of the persons who undergo this type of surgery will suffer from chronic LBP in the following 10–20 years [55]. FBSS is characterized by recurrent or residual back pain after spinal surgery, and it has a reported incidence of 20–40% [56], which increases the likelihood of the patient suffering from PMS as part of the constellation of the painful symptoms. PMS is prevalent in 13.7% of the general population, in 17% of low LBP patients [57], and reaching 40.4% of chronic LBP post discectomy patients [13].

Mechanisms of FBSS and PMS

The sources of FBSS can be attributed to the pre-, intra-, and postoperative period. The presence of preoperative psychosocial aspects increases the incidence of PMS [13]. Going into the mechanisms of FBSS is too complex owing to its multitude of mechanisms. Thus, below are the possible etiological mechanisms for PMS.

PMS can have somatic and neuropathic mechanisms [21]. In the case of type II Beaton and Anson category, there is the possibility of NP in the common peroneal nerve distribution [32], yet this is debatable [10]. The discectomy at L5/S1 level can cause the release of inflammatory mediators leading to segmental sensitization [58] of the proximal L5 and S1 nerve roots, [59] both of which innervate the PM, causing PMS [13]. The myofascial component of PMS which presents as trigger points could be a sign of central sensitization [60].

The overactive and tight ipsilateral hip flexor muscle causes reciprocal inhibition of the inactive gluteal muscles, stretching further the PM [11]. There is substantial evidence that the somatic component underlying PMS represents a myofascial pain disorder [61–63]. Etiology of trigger points is not clear, but the most accepted mechanism is the motor end-plate theory [19]. It is postulated that there is sustained acetylcholine release leading to a constant sarcomere contraction, causing hypoxia and thus releasing inflammatory mediators, causing sensitization [64].

Management

Conservative Multidisciplinary Treatment

Management of chronic pain and FBSS is best tackled with a BPS approach necessitating a multidisciplinary collaboration [6]. The NP component was treated with pregabalin [65]. Once the BPS formulation was discussed with the referring GP, conservative treatment was initially attempted [66].

First line physiotherapy treatment for PMS was conducted within a cognitive behavioral approach [67] utilizing graded exposure [68], and it aimed to reduce his buttock pain through the restoration of the PM length and to address the patient's flexibility and strength deficits. There is no standard gold therapy for PMS [25], but its first-line treatment consists of conservative treatment [66]. Treatment consisted of two physiotherapy sessions per week for six weeks and the provision of explanatory sheets, thus facilitating self-rehabilitation [16].

The session consisted initially of manual therapy including soft tissue therapy in prone lying with the hip in external rotation, thus placing the PM in a shortened position. Afterward stretching was carried out [1]. There is a multitude of studies which provide evidence for stretching as the first line of treatment [11, 16, 21, 22, 64]. PM has different functions at different

hip flexion angles, implying that PM can be stretched using two techniques [8]. Gullledge et al., [11] presented a refined stretching technique for PM, in which the hip is flexed to 90°, adducted and then externally rotated, producing the greatest amount of PM stretch. Neural stretching was given considering its effectiveness in PMS [69].

Any other deficits were corrected, including stretching the shortened iliopsoas, hamstrings and strengthening weak gluteal, and spine muscles [22]. Two studies [70, 71] found that PM EMG activation was high during hip extension, external rotation, and abduction. Recently, Morimoto et al. [72] reported that prone extension in external hip rotation leads to the highest PM EMG activation of all movements and that PM EMG activity is similar to gluteal activity. Therefore, prone hip extension in lateral rotation was given as a strengthening exercise, specifically for PM. An unloading kinesiotape technique was used since a study [19] obtained a significant reduction in VAS score with this technique, which is speculated to stimulate the neuromuscular pathways by increased afferent feedback. Lifestyle modification involving a change in sitting postures and any corrections of abnormal gait and foot mechanics were done [15]. Transcutaneous electrical nerve stimulation provided only short-term analgesia [73].

There is some evidence for passive therapies in myofascial pain, like heat, soft tissue mobilization, ultrasound [31], and needling [74]. However, these were avoided so as not to encourage a passive role by the patient [75]. Likewise, due to the severity of pain, needling the PM was avoided as it could further sensitize the area [76]. At the beginning of every session, a reassessment was carried out via the VAS pain score and the ROM of hip internal rotation in prone lying, passive ROM of lateral hip rotation, muscle power, FAIR, Freiberg, and Pace and Betty maneuvers. Additionally, his GP increased the paracetamol dosing and titrated amitriptyline up to 75 mg/day [77] for which there is more of an evidence based compared to non-steroidal anti-inflammatory drugs and muscle relaxants in myofascial pain syndromes [78].

A referral for psychology services was done since these are effective in chronic pain [79] including LBP [80]. The psychologist used a combination of cognitive behavioral therapy (CBT), acceptance and commitment therapy (ACT), and mindfulness therapy [81]. It aims to reconceptualize the patient's pain [82], lessen his catastrophizing [83], and it provided everyday coping skills, e.g., problem-solving, relaxation, breathing techniques, and pacing [84]. In ACT, the patient learned to accept in a non-judgmental way the thoughts and feelings aiming to reduce disability [81] and preventing him from getting entangled in the maladaptive emotions [85]. Mindfulness allowed the patient to respond more wisely to pain by attending his unhelpful thoughts and feelings in an open and accepting way [86]. The patient also learned how to reach his goals despite his pain [87].

Intramuscular Botulinum Neurotoxin Injection

After 2 months of physiotherapy (12 sessions in total) and psychological sessions with moderate pain relief (VAS 9.3 decreased to 6.5), a pain specialist referral was done, with the possibility of providing botulinum neurotoxin (BoNT) injections, which are effective in the long-term [31]. The patient received 50 IU BoNT intramuscular injection in the PM, which produced a drastic reduction in VAS score from 6.5 to 0.3, for 7 months, after which a repeat injection provided further long-term pain relief. The main effect of BoNT is achieved through the muscle weakening effect by inhibiting neurotransmitter release, e.g., acetylcholine, from the presynaptic vesicles and reducing muscle spasm [88]. BoNT also produces analgesia through its significant anti-inflammatory effect [89] while it inhibits sodium channels in central and peripheral neurons [90] thus it decreases sensitization [91]. BoNT injection is superior to placebo [30, 92]. However, it has a synergistic effect when combined with physiotherapy [93]. Seventy-seven percent of patients obtained an excellent or good response rate with BoNT injection [16]. Comparator studies [94, 95] have shown that BoNT is more efficient than local anesthetic and steroids in relieving PMS. Due to the high effectiveness of BoNT, surgical release of the PM is rarely required [22]. Toxin diffusion is rare, and its side effects are mostly related to the injection technique [88]. Nonetheless, BoNT should be reserved for cases refractory to conservative approaches considering the higher cost of BoNT compared with that of local anesthetics coupled with the risk of muscular atrophy and fibrosis following repeated BoNT injections [92].

Flare-up Plan

The fluctuating nature of chronic pain necessitates a flare-up plan which was developed in a joint session between the physiotherapist, the psychologist together with the patient so that the long-term benefits can be maintained [96]. The flare-up plan included [97]:

1. The adherence to the home exercise program beyond the treatment period.
2. The identification of high-risk situations.
3. Criteria for visiting the physician.
4. Performing relaxation techniques.
5. Activity management through pacing [98].
6. Using cognitive behavioral techniques when confronted with maladaptive thoughts [99].
7. Reassuring the patient while simultaneously discouraging any passive treatment.

If necessary booster sessions were made available to prevent or deal with relapses [100]. Therefore, this case was well

managed through a multidisciplinary collaboration [6]. Treatment in its entirety was effective, and it followed an evidence-based procedure with an escalation of procedural interventions to gain better analgesia. The chronic nature of the patient's pain, his psychological aspects, and the neural sensitization could justify the reason why initial conservative treatment only provided moderate pain relief. Despite the possible sensitization, gentle needling could have been attempted to deactivate the PM trigger point [101].

Conclusion

Therefore, PMS is highly prevalent in post discectomy patients. Differential diagnosis of other nearby structures and medical causes that refer pain to the buttock needs to be ruled out, since the diagnosis of PMS does not have a gold standard and on the contrary, it is driven by exclusion. The quartet of symptoms being buttock pain, pain increased by sitting, tenderness near the greater sciatic notch, and maneuvers that tension, the PM help in diagnosing PMS. There is an abundance of literature stating that the first line of treatment for PMS is conservative therapy, which is based on PM stretching, correction of any muscular strength and flexibility deficits, psychological assistance, and lifestyle modifications. If conservative treatment fails, interventional procedures are advocated, with the best long-term results obtained with intramuscular BoNT injections.

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Compliance with Ethical Standards

Conflict of Interest The author declares that he has no conflict of interest.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent Informed consent was obtained from all individual participants included in the study.

Statement of Animal Welfare This article does not contain any studies with animals performed by any of the authors.

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