International Journal of Pharmaceutical research and Applications
Volume 11, Issue 3, May-June 2026, pp:373-378 www.ijprajournal.com ISSN: 2456-4494

Piriformis Syndrome: A Rare Cause of Sciatica - A Case Report

Anamika BS[1] | Chintha Chandran[2] | Jim Litton[3] | Shaiju S dharan[4]

[1] *Pharm D Student , Ezhuthachan college of pharmaceutical sciences, Marayamuttom, Neyyatinkara, Thiruvananthapuram, Kerala, India*

[2]*Assisstant Professor, Department of pharmacy practice, Ezhuthachan college of pharmaceutical sciences, Marayamuttom, Neyyatinkara, Thiruvananthapuram, Kerala, India*

[3]*Consultant physician & diabetologist , General medicine department, NIMS Medicity, Aralumoodu, Neyyatinkara, Thiruvananthapuram, Kerala, India*

[4]*Principal / Head of Department, Pharmacy Practice, Ezhuthachan college of pharmaceutical sciences, Marayamuttom, Neyyatinkara, Thiruvananthapuram, Kerala, India.*

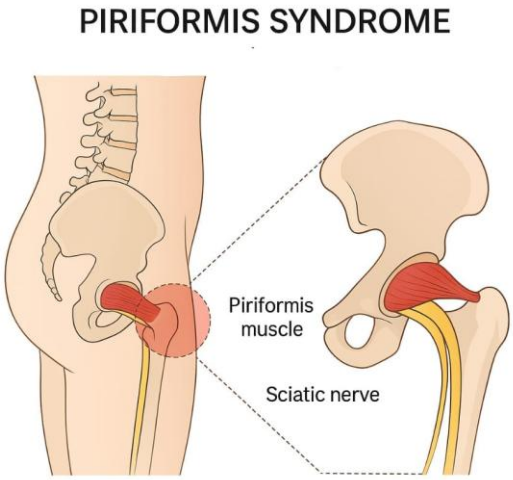
Date of Submission: 28-04-2026 Date of Acceptance: 07-05-2026

ABSTRACT

Piriformis syndrome is a neuromuscular disorder characterized by sciatic nerve entrapment due to piriformis muscle spasm or hypertrophy, often underdiagnosed due to overlapping features with lumbar radiculopathy. It is particularly challenging in geriatric patients with comorbidities like diabetes mellitus. A 70-year-old male with a 25-year history of Type 2 Diabetes Mellitus and recent trauma presented with right-sided gluteal pain and limited mobility. MRI findings, clinical features, and exclusion of differential diagnoses pointed toward piriformis syndrome. The patient exhibited neuropathic pain exacerbated by pressure, accompanied by muscle spasm, and had underlying degenerative spinal changes. Multimodal treatment including physiotherapy, neuropathic agents (pregabalin, duloxetine), muscle relaxants (tizanidine), pain control (tramadol, fentanyl patch), and dry needling significantly improved the condition. He was discharged with a comprehensive medication plan, physiotherapy, and monitoring. This case highlights the complex interplay between diabetes-related neuropathy and musculoskeletal trauma in the development of piriformis syndrome. Early diagnosis, tailored pharmacologic and non-pharmacologic therapy, and interdisciplinary care are essential to optimize outcomes.

I. INTRODUCTION

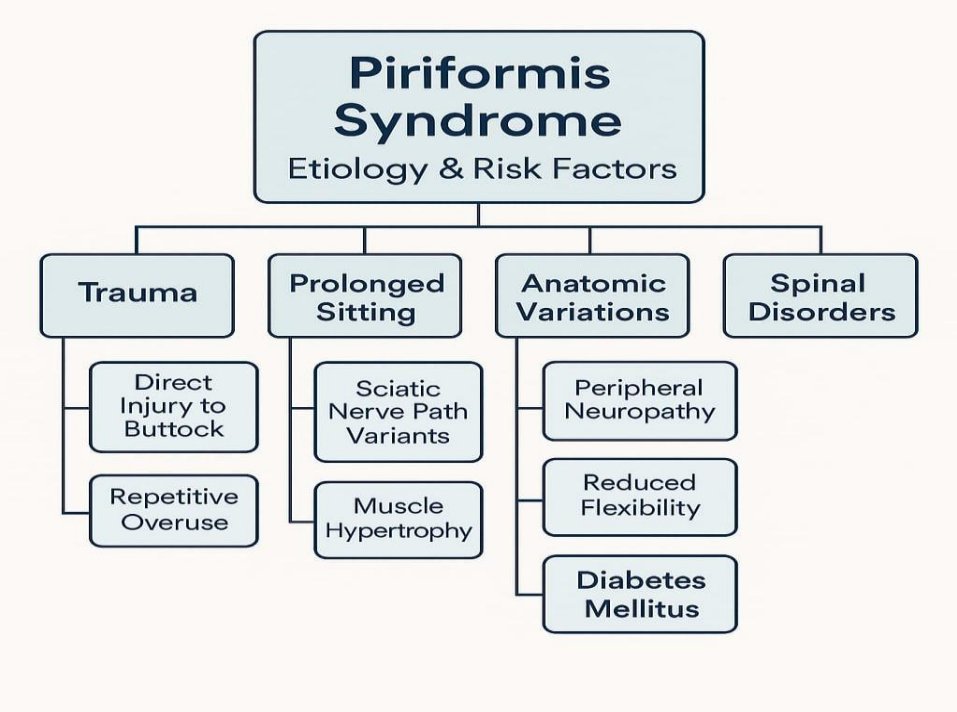
Piriformis syndrome is an uncommon yet significant neuromuscular disorder characterized by compression or irritation of the sciatic nerve by the piriformis muscle, a small, pear-shaped muscle located deep within the gluteal region [1]. First recognized in the early 20th century, the condition remains frequently underdiagnosed, largely due to its clinical overlap with lumbar radiculopathy and other spinal causes of sciatica [1,4].



The reported prevalence of piriformis syndrome is approximately 5–6% among individuals presenting with low back or leg pain [2]. It appears to occur more commonly in females, possibly due to

International Journal of Pharmaceutical research and Applications
Volume 11, Issue 3, May-June 2026, pp:373-378 www.ijprajournal.com ISSN: 2456-4494

differences in pelvic anatomy, and in individuals with sedentary lifestyles or muscle imbalances [3]. Etiologically, the syndrome may result from a variety of factors including direct trauma to the buttock, repetitive overuse, prolonged sitting, anatomical variations in the course of the sciatic nerve, and secondary involvement from spinal disorders [3,6,9]. Systemic conditions such as diabetes mellitus may further predispose individuals to the condition by promoting peripheral neuropathy and reducing muscle elasticity [5,9].



Pathophysiology of piriformis syndrome :

Step 1 — Triggering Factor

- Primary causes: Trauma to buttock region, overuse injury, prolonged sitting, anatomical variations of the piriformis or sciatic nerve [7,8].
- Secondary causes: Space-occupying lesions, post-surgical changes, or inflammatory processes [9].

Step 2 — Piriformis Muscle Response

Injury or irritation → muscle spasm or hypertrophy [4,7].

Persistent spasm → decreased elasticity and increased tone.

Step 3 — Sciatic Nerve Compression or Irritation

Anatomical relationship: In most cases, sciatic nerve passes beneath piriformis, but in variations it may pierce or pass above the muscle [8].

Tightened or inflamed piriformis compresses the nerve.

Step 4 — Local Inflammatory Cascade

Mechanical compression causes local ischemia and perineural inflammation [4,9].

Release of inflammatory mediators sensitizes nociceptors.

Step 5 — Neuropathic Pain Manifestation

Irritation of the sciatic nerve → pain radiating along the posterior thigh, sometimes to the calf/foot [4,5].

May mimic lumbar radiculopathy (overlap in dermatome distribution) [3,11].

Step 6 — Secondary Musculoskeletal Changes

Altered gait and posture from pain → compensatory muscle imbalances in lumbopelvic region [4].

If untreated, chronic changes may persist even after nerve decompression.

Step 7 — Possible Complications

Persistent neuropathic pain, sensory deficits, motor weakness in severe cases [5,14].

Reduced mobility leading to deconditioning and functional decline [16].

Diagnosis is primarily clinical and is one of exclusion, given the absence of pathognomonic tests. Physical examination findings often include buttock tenderness over the greater sciatic notch and reproduction of symptoms with provocative maneuvers such as the Freiberg, Pace, or FAIR tests

International Journal of Pharmaceutical research and Applications
Volume 11, Issue 3, May-June 2026, pp:373-378 www.ijprajournal.com ISSN: 2456-4494

[4,10]. Imaging studies, including MRI and MR neurography, can help exclude spinal pathologies and may occasionally reveal hypertrophy, edema, or displacement of the sciatic nerve, although such findings are often nonspecific [13]. In diabetic patients, differentiation from conditions such as diabetic amyotrophy or lumbosacral radiculoplexus neuropathy can be particularly challenging [12].

If left untreated, piriformis syndrome may lead to chronic neuropathic pain, impaired mobility, and functional disability. In patients with comorbidities such as diabetes, delayed diagnosis can further worsen prognosis by compounding neuropathic damage and impairing rehabilitation potential [14,15].

Management typically begins with conservative measures. Pharmacologic options include non-steroidal anti-inflammatory drugs (NSAIDs), neuropathic pain medications such as gabapentin or pregabalin, muscle relaxants like tizanidine, and opioids for severe pain [16,17]. Non-pharmacologic strategies—particularly physical therapy with stretching and strengthening programs, as well as techniques like Transcutaneous Electrical Nerve Stimulation (TENS) and dry needling—play a central role in recovery [18]. For refractory cases, interventional procedures including local anesthetic or corticosteroid injections, botulinum toxin administration, or surgical decompression may be considered [19].

This report presents a complex case of piriformis syndrome in a geriatric male with longstanding type 2 diabetes mellitus and recent trauma, illustrating the interplay between metabolic, neurologic, and musculoskeletal factors, and highlighting the importance of early recognition and multidisciplinary management to optimize patient outcomes.

II. CASE REPORT

A 70 year old male patient was admitted to general medicine department with complaints of severe pain over right gluteal region since 10 days with alleged history of fall on 14/2/25 and right foot diabetic ulcer (healed). He has a history of Type II Diabetes Mellitus for 25 years and Dyslipidemia for 1 year. His medication history includes TAB.DIETHYLCARBAMAZINE CITRATE 100 mg P/O TDS, TAB.ROSUVASTATIN 20 mg P/O HS, TAB.GLICLAZIDE 80 mg BD, TAB.GABAPENTIN 100 mg & NORTRIPTYLINE 10mg HS, CAP.OMEPRAZOLE 10 mg& DOMPERIDONE 10 mg, TAB.TENELIGLIPTIN 20 mg BD.

He was conscious, afebrile, chest was clear, moving all limbs. During admission, he had a pulse rate of 80 beats/min, respiratory rate of 18 breaths/min, blood pressure of 120/70 mmHg. His laboratory investigation showed an elevation in Eosinophils (9.8 %), ESR (45 mm/hr), Absolute Neutrophil Count (6450 cells/cumm), HbA1C (9.4 %), AST (34 U/L), Serum Globulin (3.5 g/dL), CRP (10.2 md/dL), D-Dimer (3.12 ng/ml), Pus cells (1-2/ HPF) and showed decline in Lymphocytes (23.8 %), Serum Sodium (133 mmol/L), Vitamin D (20.7 ng/ml), HDL (26 mg/dL). MRI Lumbar Spine with Whole Spine Screening done and showed heterogenous marrow signals – possibly secondary to osteoporosis, multilevel minimal endplate signal changes and small marginal osteophytes, straightening of cervical and lumbar lordosis, desiccated L1 – 2, L2 – 3 and L5 – S1 inter vertebral discs, disc height loss, moderated diffuse disc bulge and left foraminal broad based disc protrusion at L5 – S1 level causing grade II narrowing of ipsilateral neural foramen impinging the traversing roots., posterior osteophyte – disc complex obliterates ventral thecal space and narrow bilateral neural formina from C3 through C6 – 7 levels, smoothly intends the ventral cord surface at C3 – 4 and C4 – 5 levels, short segment intramedullary cord signals at C4 upper body level – suggestive of myelomalacia.

The patient was treated with INJ. IBUPROFEN 400mg/100ml for sever pain, INJ.PARACETAMOL 1g for muscle pain, TAB. ETORICOXIB + THIOCOLCHICOSIDE 5mg+4mg for pain relief with muscle relaxation, TAB. GABAPENTIN 300 mg for neuropathic pain and stopped on 26/2/25, TAB. PREGABALIN + DULOXETINE 75mg+20mg for shooting leg pain and reducing pain related anxiety , TAB. DIETHYLCARBAMAZINE 100mg for eosinophilia, TAB.ROSUVASTATIN 2mg for Dyslipidemia, TAB. GLICLAZIDE 80 mg for Type 2 Diabetes Mellitus, TAB. GABAPENTIN + NORTRYPTILINE 300mg + 10 mg for neuropathic pain, CAP. OMEPRAZOLE + DOMPERIDONE 20 mg + 10 mg for gastric irritation, TAB . TENELIGLIPTINE 40mg for Type 2 Diabetes Mellitus, INJ. TRAMADOL HYDROCHLORIDE 50mg for severe pain, INJ ONDANSETRON 4mg for preventing side effects of medications (like tramadol, pregabalin, NSAIDs), CAP. GAMMA LINOLENIC ACID + MECOBALAMIN + VITAMIN C 100 mg + 100 mcg + 100 mg for peripheral neuropathy, TAB. DIOSMIN + HESPERIDIN 450 mg + 50mg for improving venous tone, lymphatic drainage and capillary

International Journal of Pharmaceutical research and Applications
Volume 11, Issue 3, May-June 2026, pp:373-378 www.ijprajournal.com ISSN: 2456-4494

permeability, TAB. TIZANIDINE 2 mg for relaxing the piriformis muscle and reducing nerve compression, DICLOFENAC GEL for local application for reducing muscle pain and inflammation over the gluteal area, TAB. FOXOFENADINE HYDROCHLORIDE + MONTELUKAST 120mg + 10mg for correcting sleep disturbance and neuropathy, DERMADEW CALOE LOTION for local application for moisturizing dry diabetic skin, TAB. CALCIUM CARBONATE + VITAMIN D3 500mg + 250 IU for bone loss prevention, CAP. CHOLECALCIFEROL 60000 IU for Vitamin D insufficiency, TAB. FLUPIRTINE + PARACETAMOL 100mg + 325mg for chronic back pain. On 2/3/25 INJ.TRAMADOL HYDROCHLORIDE was changed to TAB. TRAMADOL HYDROCHLORIDE+ ACETAMINOPHEN 37.5mg + 325mg. On 5/3/25 applied FENTANYL TRANSDERMAL PATCH 2.5mg (each patch delivers 25mcg/hr fentanyl) for severe musculoskeltal pain.The patient was managed with TDP physiotherapy and othe supportive measures like piriform dry needling.

The patient got clinically better and discharged with medications like TAB. GAMMA LINOLENIC ACID + MECOBALAMIN + VITAMIN C 100mg + 100 mcg + 100 mg, P/O, 1-0-1 for 5 days, TAB.PREGABALIN + DULOXETINE 75mg + 20 mg, P/O, 1-0-1 for 5 days, TAB. DIOSMIN + HESPERIDIN 450mg + 5mg, P/O, 1-1-1 for 5 days, TAB.TIZANIDINE 2mg, P/O, 1-1-1 for 10 days, TAB. CALCIUM CARBONATE + VITAMIN D3 500mg + 250IU, P/O, 1-0-0 for 5 days, TAB.FLUPIRTINE + PARACETAMOL 100mg +325mg, P/O, 1-0-1 or 5 days, CAP.CHOLECALCIFEROL 60k, P/O, once weekly for 5 more weeks, TAB. FOXOFENADINE HYDROCHLORIDE + MONTELUKAST 120mg + 10mg, P/O, 1-0-1 for 5 days, DERMADEW CALOE for L/A for 5 days, DICLOFENAC GEL for L/A for 5 days, TAB.GABAPENTIN 300mg, P/O, 0-0-1 for 5 days, BUPRENORPHINE TRANSDERMAL PATCH , 5mg, Q7 days – 2, CAP.OMEPRAZOLE + DOMPERIDONE, 20mg + 10mg, P/O, 1-0-0 to continue, TAB.GABAPENTIN + NORTRIPTYLINE, 300mg + 10mg, P/O, 0-0-1 to continue, TAB.ROSUVASTATIN, 20mg, P/O, 0-0-1 to continue, TAB.GLICLAZIDE, 80mg, 1-0-1 to continue, TAB.TENSTAR 40mg, 1-0-1 to continue, INJ. HUMAN MIXTARD, 50/50, 26units – 24units – 22units.

III. DISCUSSION

Piriformis syndrome, while well-documented in anatomical and clinical literature, continues to be a subject of diagnostic ambiguity. As Papadopoulos and Khan [1] note, the lack of universally accepted diagnostic criteria has led to significant variability in reported incidence and management approaches. Our case mirrors this challenge—despite MRI evidence of multilevel degenerative spine changes, the patient's focal tenderness over the piriformis muscle and symptom resolution with targeted therapy supported the diagnosis, reinforcing the assertion by Boyajian-O'Neill et al. [4] that clinical assessment remains paramount.

The estimated prevalence of 5–6% among individuals with low back or leg pain [2] is often debated. Hopayian and Danielyan [3] argue that the syndrome may be underreported due to misclassification as lumbar radiculopathy, particularly in cases where imaging demonstrates spinal abnormalities. Our patient's spinal MRI revealed foraminal narrowing and myelomalacia, yet these findings were incidental to the true pain generator—underscoring Hicks et al.'s [11] warning that imaging alone can mislead clinicians.

The role of anatomical variation is well recognized [7,8]. In Fishman et al.'s [9] long-term series, anatomical anomalies correlated with more severe presentations and poorer conservative treatment outcomes. While no variant anatomy was confirmed in our case due to imaging limitations, the presence of trauma and prolonged sitting (due to reduced mobility) likely contributed to the mechanical irritation of the sciatic nerve.

In diabetic patients, symptom interpretation becomes more complex. Vinik et al. [5] and Tesfaye et al. [14] highlight that peripheral neuropathy from long-standing diabetes may mimic or exacerbate symptoms of piriformis syndrome, leading to delayed or missed diagnoses. Our patient's elevated HbA1c (9.4%) suggested poor glycemic control, which likely worsened neuropathic pain, in line with the American Diabetes Association's [15] emphasis on strict glycemic optimization to improve nerve health.

Regarding diagnostic strategies, Filler et al. [13] advocate for MR neurography as a tool to visualize sciatic nerve irritation in suspected cases, although its limited availability and cost remain barriers in routine practice. Our diagnosis relied on clinical features and response to targeted therapy, which is consistent with Michel et al.'s [10] findings that physical examination remains a reliable and

International Journal of Pharmaceutical research and Applications
Volume 11, Issue 3, May-June 2026, pp:373-378 www.ijprajournal.com ISSN: 2456-4494

cost-effective diagnostic approach when conducted systematically.

Therapeutically, our management strategy aligns with recommendations from Attal et al. [16] and Baron et al. [17], employing a combination of neuropathic agents, muscle relaxants, NSAIDs, and opioids for breakthrough pain. The addition of dry needling—a technique supported by Akınoğlu and Köse [18]—appeared to accelerate recovery in our patient. Interventional procedures, as described by Jeong et al. [19], were not required given the positive response to conservative therapy.

Finally, the complexity of treating piriformis syndrome in older adults with multiple comorbidities cannot be overstated. Maher et al. [20] caution that polypharmacy increases the risk of adverse events and drug interactions, a concern we addressed by tailoring the regimen to balance pain relief, glycemic control, and cardiovascular risk management.

Overall, our findings reinforce the need for a multidisciplinary, individualized approach, particularly in patients where musculoskeletal, neurologic, and metabolic pathologies intersect. Early recognition, careful diagnostic exclusion, and targeted therapy remain the cornerstones of favorable outcomes in piriformis syndrome.

IV. CONCLUSION

Piriformis syndrome remains a diagnostic and therapeutic challenge, especially in the elderly with chronic comorbidities. This case underscores the importance of considering this diagnosis in patients with buttock pain and neuropathic symptoms following trauma. A multidisciplinary approach, addressing not only the musculoskeletal component but also metabolic and neurologic aspects, ensures better outcomes.

Early recognition, judicious use of pharmacologic agents, patient education, and structured physiotherapy can significantly improve quality of life in such complex presentations.

REFERENCES

[1]. Papadopoulos EC, Khan SN. Piriformis syndrome and low back pain: a new classification and review of the literature. Orthop Clin North Am. 2004;35(1):65–71.
[2]. Kirschner JS, Foye PM, Cole JL. Piriformis syndrome, diagnosis and treatment. Muscle Nerve. 2009;40(1):10–18.
[3]. Hopayian K, Danielyan A. Four symptoms define the piriformis syndrome: an updated systematic review of its clinical features. Eur J Orthop Surg Traumatol. 2018;28(2):155–164.
[4]. Boyajian-O'Neill LA, et al. Diagnosis and management of piriformis syndrome: an osteopathic approach. J Am Osteopath Assoc. 2008;108(11):657–664.
[5]. Vinik AI, et al. Diabetic neuropathies: clinical manifestations and current treatment options. Nat Clin Pract Endocrinol Metab. 2006;2(5):269–281.
[6]. Benzon HT, et al. Piriformis syndrome: anatomic considerations, a new injection technique, and a review of the literature. Anesthesiology. 2003;98(6):1442–1448.
[7]. Pećina M, et al. Piriformis muscle: anatomical considerations in relation to pain syndromes. Surg Radiol Anat. 1994;16(4):273–278.
[8]. Smoll NR. Variations of the piriformis and sciatic nerve with clinical consequence: a review. Clin Anat. 2010;23(1):8–17.
[9]. Fishman SM, et al. Piriformis syndrome: diagnosis, treatment, and outcome—a 10-year study. Arch Phys Med Rehabil. 2002;83(3):295–301.
[10]. Michel F, et al. Piriformis muscle syndrome: diagnostic criteria and treatment outcomes. Arch Phys Med Rehabil. 1999;80(7):864–866.
[11]. Hicks BL, et al. The pathophysiology of piriformis syndrome: implications for diagnosis and management. J Am Acad Orthop Surg. 2018;26(6):204–210.
[12]. Dyck PJ, et al. Diabetic lumbosacral radiculoplexus neuropathy: natural history and late responses to immunotherapy. Brain. 2000;123(3):539–550.
[13]. Filler AG, et al. Sciatica of nondisc origin and piriformis syndrome: diagnosis by magnetic resonance neurography and interventional magnetic resonance imaging. Neurosurg Focus. 2005;18(1):E7.
[14]. Tesfaye S, et al. Diabetic neuropathies: update on definitions, diagnostic criteria, estimation of severity, and treatments. Diabetes Care. 2010;33(10):2285–2293.
[15]. American Diabetes Association. Standards of Medical Care in Diabetes—2024. Diabetes Care. 2024;47(Suppl 1):S1–S160.
[16]. Attal N, et al. EFNS guidelines on the pharmacological treatment of neuropathic pain: 2015 revision. Eur J Neurol. 2015;22(9):1150–1168.

[17]. Baron R, et al. Pharmacological therapy of chronic neuropathic pain: the current options. Dtsch Arztebl Int. 2010;107(37):623–631.
[18]. Akınoğlu B, Köse N. Dry needling treatment in patients with piriformis syndrome. J Back Musculoskelet Rehabil. 2018;31(6):1075–1080.
[19]. Jeong HS, et al. Piriformis syndrome: a case of endoscopic sciatic nerve decompression. Skeletal Radiol. 2010;39(6):599–602.
[20]. Maher RL, Hanlon J, Hajjar ER. Clinical consequences of polypharmacy in elderly. Expert Opin Drug Saf. 2014;13(1):57–65.