Piriformis muscle hypertrophy as an etiology for posterior femoral cutaneous nerve entrapment neuropathy.

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Clinical History

• 60-year-old gentleman presented with a 12-month duration of symptoms in the posterior aspect of the left thigh exacerbated by sitting down or walking.

• The patient reported symptoms of numbness and reported that the pain would radiate to his calf muscles.

• The symptoms were gradually increasing and was subsequently taking narcotics on a regular basis.
Neurological examination

- Awake alert oriented ×3.
- Cranial nerve examination was within normal limits.
- Motor examination showed normal strength in both upper extremities.
- Right lower extremity strength examination showed a strength of 5/5 both distally and proximally.
- Left lower extremity:
  - strength at the ankle was 5/5 in the dorsiflexors
  - ankle flexors, inverters, everters hamstrings 4/5
  - hip flexors 5/5
  - hip extensors 5/5.
Neurological examination

• **Sensory exam** showed a clear cut *dermatomal* loss of *decrease in sensation* to pinprick touch and temperature sense in the *left posterior femoral cutaneous nerve distribution*.

• Knee reflexes were 2/4.

• Ankle jerks were absent on both sides.

• Plantar reflexes were downgoing on both sides. Coordination was intact.

• He had significant difficulty with sitting and showed mild antalgic gait with walking.

• **NCV/ EMGs in 2016 of the bilateral lower extremities were unremarkable.**
Imaging

- Initial lumbar radiographs demonstrated mild multilevel degenerative changes with mild facet arthropathy at the distal levels.
- Successive MRI of the lumbar spine exhibited mild multilevel disc bulges, mild endplate degenerative changes and mild facet arthropathy at L4/5 and L5/S1 with no significant central canal or neuroforaminal narrowing at any level.
Initial lumbar radiographs demonstrated mild multilevel degenerative changes with mild facet arthropathy at the distal levels.
T2 right parasagittal, midline, and left parasagittal images.
Mild multilevel disc bulges, mild endplate degenerative changes and mild facet arthropathy at L4/5 and L5/S1 with no significant central canal or neuroforaminal narrowing at any level.
Imaging

• However, the neurologist was persistent in pursuing further imaging given the patient’s significant symptoms and positive neurological examination.

• Subsequent MRI of the pelvis revealed hypertrophy of the left piriformis muscle with muscular slips of the piriformis entrapping the left S1 and S2 nerve roots as they exited their respective neuroforamina.
Unenhanced T1 axial image of the pelvis demonstrate a hypertrophied left piriformis muscle (→).
Axial STIR image of the pelvis demonstrate a hypertrophied left piriformis muscle (→).
Unenhanced T1 axial image of the pelvis (at a level more cephalad to previous figure) demonstrate slips of the hypertrophied left piriformis muscle circumferentially entrapping the S1 (←) and S2 (←) Nerve roots as they exit their respective neuroforamina.
Unenhanced T1 coronal image of the pelvis demonstrate a hypertrophied left piriformis muscle (←) and exiting S1 nerve roots (→).

Unenhanced T1 coronal image of the pelvis demonstrates the S1 nerve traversing through the left piriformis muscle.
Hypertrophy of the left piriformis (←) causing encroachment of the left S2 neuroforamen and contacting the left S2 nerve (→) as it exits the neuroforamen.

Coronal T1

Coronal T2 Fat Saturated image
Etiologies that can incite piriformis syndrome symptoms and PCFN neuropathy

• Trauma to the gluteal region
  – Impact injury
  – Repetitive trauma from cycling
  – Injection injury

• Masses along the expected course of these nerves such as tumor, hematoma, aneurysm formation, myositis ossificans, anomalies of the piriformis muscle

• Variant/anomalous course of the sciatic nerve.
Treatment options

Noninvasive/conservative

- Nonsteroidal anti-inflammatory drugs (NSAIDs)
- Physical therapy
- Osteopathic manipulative treatment
- Muscle relaxants
- Ice and rest

Invasive

- Point injection with lidocaine hydrochloride, steroids, or botulinum toxin.
- Surgical intervention has been curative in cases where imaging clearly exhibits a resectable cause of nerve entrapment or muscle compromise such as tumor, aneurysm formation, myositis ossificans, or anomalies of the piriformis muscle.
Discussion

• Etiologies or factors that impinge upon or involve the PFCN can contribute to the symptoms of “sitting pain.”

• Classically, the PFCN is arises from the ventral rami of S1, S2, and S3 spinal nerves from the sacral plexus. It exits the sciatic foramen and the pelvis below the piriformis muscle, and is located posteromedial to the sciatic nerve. The PCFN superficially gives rise to the perineal and inferior clunial branches which provide sensory innervation to the inferior posterior buttock. The main trunk traverses distally in the midline to provided innervation to the posterior thigh.

• There is expected overlap with symptoms of piriformis syndrome as the sciatic nerve exhibits a similar course through the greater sciatic foramen and given its close proximity.
Discussion

• Isolated cases of PCFN neuropathy are rarely encountered.

• However, reports have been described secondary to inguinal hematoma formation after gluteal intramuscular injection. $^{2,3}$
Discussion

- In this particular case, the presentation and the clinical/neurological examination suggested a PFCN compression by a hypertrophied left piriformis muscle.
- These findings were more conspicuous on the MRI of the pelvis as opposed to the MRI of the lumbar spine.
- This clinical scenario further underscores the need for possible further MRI imaging of the pelvis, should the clinical presentation warrants further interrogation, despite a relatively unremarkable MRI of the lumbar spine.
- The entrapment of the S1 and S2 nerve by the piriformis muscle also should be included in the causative factors for the PFCN neuralgia.
References


