Title:

The Deep Gluteal Syndrome, an underestimated etiological factor for Sciatica, Low Back Pain and other chronic pain conditions? A case series report, review of the literature and a proposal for diagnostic criteria.

Author Name

Mikael Nordfors M.D.

GP

Kildegårdsvej 12 A

2900 Hellerup

Denmark

ph +45 58101782

e-mail: mikael@kunlabori.org

Affiliations: None

Domains:

Orthopedics, Orthopedic Medicine, Physiotherapy, Ostheopathy, Chiropractics, Reumatology, Neurology

Type of publication = Case Study

Acknowledgements:

Thanks to Dr. Breck Mckay, Dr. Michael Yelland in Australia and Dr Olle Myrin in Sweden and Chris Cook in the UK for reviewing, correcting language and advising me regarding this paper.

Abstract

Objective:

This article proposes a new diagnostic entity called the Deep Gluteal Syndrome and makes a very rough estimation of its minimum prevalence in a primary care environment.

Design

It is based on a collection of case reports on retrospectively collected data from an unselected patient material in a Norwegian primary care setting.

Results

Deep Gluteal Syndrome (DGS) was diagnosed in 187 out of 986 patients, giving a prevalence of 19.9%. Only patients with anamnestic signs of Deep Gluteal Syndrome were investigated. This means that the real prevalence of Deep Gluteal syndrome may be higher. Deep Gluteal Syndrome was found in association with a range of conditions including lumbago, diffuse neck/shoulder/arm problems, hip and buttock pain, sciatica and diffuse lower abdomen/pelvic pain, fibromyalgia and tension headache.

The syndrome is defined as tenderness and abnormal pain sensitivity in the Parasacrococcygeal area. The pain disappears together with the above mentioned associated conditions after appropriate stretching, or injection with local anaesthetics. The syndrome can be treated with a probable success rate of more than 80%. The main treatments are exercise, manipulation, stretching exercises, manual manipulation of the deep gluteal muscles, ultrasound treatment, steroid and/or botulinum toxin injections and surgery.

Conclusions

These data suggests that the so-called Deep Gluteal Syndrome(DGS) is severely underdiagnosed. It also postulates DGS as a possible etilogical factor for Low Back Pain, Sciatica and several other chronic pain conditions. With more research, and education about appropriate diagnosis and treatment, there might be a huge potential for relief of suffering and financial savings to the health system.

Synopsis

Data from a primary care setting in Norway and large case series from other parts of the world suggests that the so-called Deep Gluteal Syndrome(DGS) is severely underdiagnosed. The present article makes a literature review, present a case series and proposes diagnostic criteria for the syndrom. It also postulates DGS as a possible etilogical factor for Low Back Pain, Sciatica and several other chronic pain conditions. With more research, and education about appropriate diagnosis and treatment, there might be a huge potential for relief of suffering and financial savings to the health system.

Main Text

Introduction

The Piriformis Syndrome(PS)

In order to better understand the concept of DGS, the author will start with an introduction about the Piriformis Syndrome(PS). PS can be classified as a subgroup of DGS, meaning that all PS are DGS, but not all DGS are PS.

PS was already described in 1936 by the American surgeon Thiele[3]. Medical reports relating pain in the parasacrocoocygeal region to back pain, Sciatica and other conditions date back as early as 1859[3].

PS occurs when the Piriformis muscle, which originates on the front of the sacral bone, and inserts on the Major Trochanter of the Femur, compresses the sciatic nerve as it leaves the buttock just below the greater sciatic foramen. Clinical signs for PS include positive straight leg raise, weakened abduction of the flexed thigh, and tenderness at the intersection of the muscle and nerve. Normally, the Sciatic Nerve passes through the greater sciatic foramen below the Piriformis Muscle. However, it may divide into its common fibular and tibial nerve components within the pelvis and its relationship with Piriformis is variable. Anatomical variations may contribute to Piriformis syndrome, coccygodynia and muscle atrophies[4,5].

Electrophysiological diagnosis is made by comparing posterior tibial and peroneal Hreflexes elicited in the anatomical position with those obtained in Flexion, Adduction and Internal Rotation (FAIR-test)[6,7]. The FAIR test also provokes pain in the Piriformis area and sometimes also symptoms of sciatica when PS is present. Filler et al [8] did in year 2005 publish an investigation of 239 patients, with sciatica who had either failed spine surgery (46%) or a failure to determine the exact cause of their sciatica. After performing MR neurography and interventional MR imaging, the final rediagnoses included the following: piriformis syndrome (67.8%), distal foraminal nerve root entrapment (6%), ischial tunnel syndrome (4.7%),discogenic pain with referred leg pain (3.4%), pudendal nerve entrapment with referred pain (3%), distal sciatic entrapment (2.1%), sciatic tumor (1.7%), lumbosacral plexus entrapment (1.3%), unappreciated lateral disc herniation(1.3%), nerve root injury due to spinal surgery (1.3%), inadequate spinal nerve root decompression (0.8%), lumbar stenosis (0.8%), sacroiliac joint inflammation (0.8%), lumbosacral plexus tumor (0.4%), sacral fracture (0.4%), and no diagnosis (4.2%).

This clearly indicates that a vast number of Sciatica patients suffer from a Piriformis Syndrome, and that the diagnosis can be made by MR Neurography. In more than 80% of the patients a good or excellent functional outcome was achieved through treatment with MR-guided Marcaine/Celestone injections or Piriformis surgery. A quality evaluation of MR neuography's diagnostic efficacy revealed that piriformis muscle asymmetry and sciatic nerve hyperintensity at the sciatic notch exhibited a 93% specificity and 64% sensitivity in distinguishing patients with piriformis syndrome from those without who had similar symptoms (p , 0.01). Evaluation of the nerve beyond the proximal foramen provided eight additional diagnostic categories affecting 96% of these patients

The Piriformis Syndrome is in spite of these findings still a debated diagnosis which

is largely unknown to a large number of orthopaedists, neurosurgeons rheumatologists and other back pain specialists. As an example the Swedish medical council (Socialstyrelsen) claimed that there is no evidence that the Piriformis Syndrome exists in a court case in Sweden as late as 2004[9].

The Piriformis syndrome has yet to receive an agreed definition, and it is agreed upon that more reliability studies with respect to tests and syndrome criteria, pathological mechanisms, prevalence, etiology as well as efficacy studies are needed[10,11].

Deep Gluteal Syndrome(DGS)

Many clinicians have experienced that both the prevalence and the clinical signs related to chronic muscle contractions in the Deep Gluteal area are more abundant than previously described. Not only the Piriformis muscle, but also that other deep pelvic muscles like M. Obturator Internus[12], M. Levator Ani, M.Gemelli and m. Coccygeus can be involved in the pathology[4,5]. Some authors have therefore suggested the term "Deep gluteal syndrome" or "external rotator syndrome"[10].

The reason is the above mentioned lack of universally accepted precise diagnostic criteria and understanding of the pathological mechanisms for PS. As it probably refers to many different pathological and ethiological conditions, with a similar treatment, a more well-defined and precise diagnostic term like DGS might in the autor's opinion be of value, for clinical and research purposes.

Symptoms

Australian GP, Breck McKay published in May 2004, data derived from the management of 550 chronic low back pain via his modified Blomberg injection method (McMaropi- Wasubo protocol) and identified many other symptoms associated with low back pain, hip pain and Sciatica that also accompany PS[13].

These symptoms included vertigo, tension headache, Cervicalgia, Thoracalgia, diffuse and localised pain conditions in shoulders and arms, epicondylitis, carpal tunnel syndrome, diffuse lower genital/pelvic/abdominal pain, , which are consistent with McKay and Wall's model of the human body responding as one single organ, rather than many individual parts. [14]

These observations have also been made over many decades of clinical experience in Scandinavia*, and have been observed as disappearing together with other more "classical" Piriformis Syndrome symptoms, when appropriately treated.

Pain at night is common for DGS patients. Most people sleep on the side with one leg over the other (The FAIR position, see below), which will stretch the Piriformis muscle, with increased nerve compression and pain as a result. A simple method to diminish this pain is to put a big pillow between the legs while asleep, something that many patients have found out by own experience. It is common to get better during the day, when you get up and move around.

Proposed Diagnostic Criteria

The proposed Diagnosis is very simple and is made by palpating the parasacrococcygeal region from the back, or with more accuracy from the front, per rectum . The first diagnostic criterion for DGS is tense muscles in the DG area that are extremely pain sensitive. In normal cases the DG area is not particularly pain

sensitive, nor tense. A typical sign is that the patient will experience a sharp sensation when you investigate the structures with the palm of your finger, just as if you were hurting him/her with your nail or with a knife. This is because the muscles are tense, in some cases almost like piano strings, compressing the surrounding nerves when you stretch them manually .

In prolonged cases(after >5 years of symptoms), the muscles can get calcified, which can be felt as a hard resistance producing a crunching sound when you try to penetrate the muscle with a needle. These calcifications can also be demonstrated on CT and MR[15].

The second diagnostic criterion is an immediate pain and symptom relief upon massaging the DG structures until they are relaxed and pain-free, or achieving the same result by injecting local anaesthetics.

Below are some short case reports:

- A 69 year old male patient with a 20 year history of low back pain and neck pain, with more recent symptoms of pain in the right wrist and vertigo, gets treatment with an injection of local anaesthetics and steroids in the DG area, followed by stretching per rectum with one finger. He receives complete remission of all the above mentioned symptoms within minutes of the treatment. After a couple of weeks some of the symptoms come back partially. A second injection after one month leaves him completely free of symptoms for more than one year.
- A 47 year old male patient with a history of more than 10 years of Chronic Lower Back Pain and 4 admittances to the hospitals surgery clinic in the last five years due to severe abdominal pains, where the hospital was unable to conclude a final cause of his abdominal pains, comes to the clinic with acute abdominal pain once again. He gets a complete remission from both the abdominal pain and the back pain after treatment with one injection of cortisone and local anaesthetics administered in the DG area, followed by DG stretching per rectum.
- A 38 year old woman with a congenital hip dysplasia and life long accompanying pains in the lower back, neck, shoulders and both arms. She gets an immediate complete relief from all her symptoms within minutes of the first DG injection treatment, but gets a partial recurrence of the symptoms after a couple of weeks. After the second treatment she is well for a couple of months, and now receives one treatment every second or third month, when needed. These findings are consistent with the observation that many patients with underlying conditions like hip arthritis or Mb Bechterew have a big tendency for relapse of DGS, probably due to the irritant action of the underlying condition on the DG muscles.

In the author's opinion, the above mentioned two criteria are enough to conclude and confirm the diagnosis DGS with a great deal of accuracy and safety. Other clinical signs are a positive SLR-test (not very significant in gymnastic patients with loose joints), a positive FAIR-test (Provocation of pain in the Piriformis area by Flexion, Adduction, and Internal Rotation of the affected hip), signs of a stiff and locked Sacro-Iliac joint (the Trendelenburg test, the Derbolowsky test). Piriformis syndrome can also be confirmed by electrophysiological tests[6,16] (Prolonged H-Reflex or caudal equina reflex in the FAIR position), EMG, MR, CT[7,8] and scintigraphy[17]. Another easily visible clinical sign is degeneration of the gluteus muscle on the affected side[3], probably due to lack of motoric nerve stimulation because of

compression of the superior and inferior gluteal nerves by the contracted DG muscles.

Treatment

The purpose of the treatment is to relax the chronic muscle contractions, through exercise, stretching and massage of the muscles in the DG region. Ultrasound, injections with steroids and local anaesthetics (relaxing the muscles with anaesthetic and minimising the swelling with steroids), local acting muscle relaxants like Botox and Myobloc and as a last resort, operation by splitting the involved muscles (Piriformis and/or Obturator Internus) and freeing the affected nerves[6-8, 19, 21] are other treatment possibilities.

For simple cases, it might be enough with stretching of the tense muscles with the finger either beside the anus, up and beside the sacral bone, or through rectal exploration[1-3]. The stretching simply continues until the sensation of sharp pain is gone. This technique and its successful clinical application was described already by Thiele 1936[3]. The problem with this method is that it can be extremely painful and that the DG area is a very sensitive "private" space for many patients, consequently this part of the treatment is refused by some patients.

Next step in the treatment hierarchy is injections with cortisone and steroids or Botox/Myobloc. Steroids have the advantage of a lower price, but recent research suggests better effects when treated with Botox/Myobloc. As Botox/Myobloc injections are not sponsored by the Norwegian health system, many patients hesitate to spend 500 USD on a treatment largely unknown in conventional medical practice. A combination treatment with injections of steroids, local anaesthetics and Botox has also been used with success*.

As a final resort the condition can be treated by surgery[6,8,19, 21].

Material and Methods

This paper is a result of retrospectively collected treatment data through investigating treatment journals in an unselected patient population at a general practice office in Western Norway, logged in the computerised medical journal system in respect of all patients getting the diagnosis of "Piriformis Syndrome" by the author during a treatment period of 15 months. The term Piriformis syndrome was used together with the diagnostic criteria of DGS, mainly because it is the available diagnostic entity that most closely resembles DGS.

Almost all the patients were from a limited treatment area containing a population of 3000, meaning that the prevalence data are fairly unbiased because of patients search treatment from other areas of the country.

Since there was no initial intention to make a scientific study there has therefore been no systematic collection of treatment results using standardised treatment success criteria after specific follow up intervals, and no control group. It follows that the long term treatment result data cannot be taken too seriously. It has also not been possible to determine how many patients were investigated who did not get a PS diagnosis. A rough estimate is that altogether 200 patients were investigated altogether.

What is more interesting is the minimum prevalence data, using the two above mentioned easily definable diagnostic criteria, which could be obtained with a much greater degree of safety and accuracy.

Results

Associated Symptoms

Among 187 patients with diagnosed DGS, 94 were women, and 93 men. 110 (58,8%) had Lumbago and 58 (31,0%) had hip pain. Another 58 (31.0%) had Sciatica, 24(12,8%) shoulder pain, 22(11,8%) neck pain, 8 (4,3%)epicondylitis or diffuse lower arm pain, 7(3,7%) lower abdominal pain, 3(1,6%) vertigo, 3(1,6%) with tension headache, and 2 (1,1%) pains in the genital area. 44 patients(23,5%) had simultaneous symptoms from different parts of the body, i.e. hip pain and shoulder pain. See table 1

Associated	Lumbago	Hip	Sciatica	Shoulder	Neck	Epi	Ab	Other	Mul
Symptoms		pain		pain	pain	condy litis	domi nal pain	symp toms	tiple symp toms
Number. of patients	110	58	58	24	22	8	7	9	44

One patient(0.5%) had a very pronounced increase of varicose veins[20] in the affected leg, compared with the non-affected side. Many of the patients had multiple symptoms with symptoms from i.e. both neck, shoulders, lower back, hip and one leg. In some cases their condition also fulfilled the criteria's of fibromyalgia.

In all the symptom groups, except for the varicose veins, an immediate positive effect was noted upon all the described symptoms when successfully treated with manual therapy and/or injections of local anaesthetic.

Prevalence Data

During 15 months in a general practice in Western Norway 187(19, 9%), out of a total of an estimated 200 investigated, and 739 non-investigated patients, fulfilled the first criterion of DGS (Tenderness over the DG area or the Piriformis muscle). The second most common diagnosis was high blood pressure (62 patients=6, 6%). There was no significant sex difference (84 women and 83 men) of these 172 patients received treatment, while 16 for different reasons did not want or get an opportunity for treatment. For 6 patients the immediate treatment effect was not evaluated. From the remaining 165 patients 139 received treatment with injections and digital stretching, while 27 were treated with only stretching of the DG region. 161 of the 165 patients (97, 6%) got an immediate symptom relief, thus confirming the second diagnostic criterion. Three out of the four immediate non-responders did receive steroid/cortisone injections, while one received a very modest manual treatment.

Prognosis

DGS and PS are highly treatable conditions with a success rate of > 80%, when giving optimal treatment conditions, which has been shown in several controlled studies, and from decades of clinical experience[1,2, 6-8,18,19, 21].

Data from the general practice in Western Norway clearly indicates there is a relationship between symptom duration and success of treatment, so that young people with a short symptom history are much easier to treat, than more chronic cases, where anatomical findings like gluteus degeneration and calcification of the DG muscles have occurred.

In the Norwegian material (where the treatment results should not be taken too

seriously because of lack of consequent collection of follow up data at similar time intervals, lack of control group, lack of independent and blind rating strategies etc.) 34 (20,6%) of a total 165 treated patients where verified symptom free after one year of follow up, 68 (41,1%) free from symptoms after a shorter follow up time, or symptom free without any signs of relapse noted in the medical journals, 40 (24,2%) were partially better, and 19(11,5%) had an initially good effect, only to relapse later, 4 (2,4%) no effect whatsoever, and no-one(0%) got worse.

Among the side effects, a few (<10) cases of temporary incontinence and walking problems were noted due to the effect of the local anaesthetic. A few patients with diabetes also suffered from a mild elevation of their blood sugar levels due to the cortisone injections. More common side effects were a temporary sense of numbness in the hip, genital, buttocks and legs, also due to the effect of the local anaesthetic. No side effects lasting more than a couple of days were noted.

Discussion

Aetiology

There are as far as the author is aware, no hard scientific data on the aetiology of DGS, but repeated clinical observations show DGS patients often have a history of trauma towards the sacro-coccygeal region[6,11,21,]. Another common starting point is pregnancy and childbirth. Chronic pain and inflammation affecting the DG area from i.e. disc hernias, Mb Bechterew and hip arthrosis[22] might be another ethiological factor. In these cases, it is very likely that the DGS symptoms will recur after treatment, unless you remove the primary disease.

The trauma or inflammation leads to pain, which in turn triggers a contraction reflex in order to inimise painful movements. The contraction in turn leads to even more pain through compression of nerves and lack of circulation in the chronically contracted muscle and the vicious circle is completed.

Not only sensory nerves become compressed, but also motoric nerves such as the inferior and superior gluteal nerve, which innervates the gluteal muscles. This leads to a relative weakness in the strongest extensors of the hip, which in turn gets compensated by an increased tension in m. Erector Spinae and Quadratus Lumborum in order to keep equilibrium between the extension and tension part of the body. This increased tension can also be demonstrated by a positive erector Spinae reflex, (tickling the erector Spinae gives a kicking extension movement in legs).

The increased tension in the spine compresses the vertebraes and the intervertebral discs, with back pain and rhizopathias as a consequence.

When the stabilizing functions of the gluteal muscles disappear, the body pulls the "emergency brake" = locks the muscles in the spine in order to diminish movability in order to protect the vital spinal cord. This also causes contraction in the iliopsoas muscle, with possible disturbances in the autonomic nerves passing through it on the front of the spine as a consequence, which can be a possible part-explanation of the abdominal symptoms described above. The scalene muscles in the neck respond in a similar manner completing a total body response as afferents pass to the mid brain and higher centres and autonomic and motor efferents prepare the body for flight/fight or fear/freeze response. [14]

Another possible explanation is that the area around the coccyx for phylogenetic reasons still plays an important role for our proprioception, as our animal forefathers

used the tail in order to maintain their balance. If our tail muscles suffer from chronic contractions, they will send false proprioceptive nerve impulses to the brain, suggesting that we are falling forwards (Falling forwards will lead to increased tension in the muscles that lowers the tail), thus activating both the erector Spinae, the Quadratus Lumborum, the gluteal muscles and the Piriformis).

This also leads to conflicting proprioceptive input from different parts of the body, putting the nervous system under stress and confusion, which might contribute to generalised increase in muscle tension and vertigo, activating the "emergency brake" reflex, contracting the muscles around the spine in order to protect the spinal cord from getting damaged due to too big movements of the spine that cannot be appropriately regulated through the defect neuromuscular system surrounding it[14].

Another contributing factor is prolonged sitting, which lowers the blood circulation and compresses the nerves in the area. Heavy lifting might also be an aetiological factor, maybe because of reflectoric contractions of the lower tail muscles like Piriformis, Obturatur Internus and coccygeus. (Imagine a kangaroo with a long tail extending the back by lowering its tail. Homo Sapiens has a very small tail, and a very small tail muscle, which easily becomes desperately contracted through trying to compensate for the big back on the other side of the lever at the hip) Truck drivers are very likely to have DGS due to the combination of prolonged sitting and heavy lifting.

Although the treatment result data from this data collection should not be considered to be too reliable, there are more treatment data of higher quality from other studies[1,2, 6-8,18,19, 21]. The prevalence data can be considered to be more interesting, as these data are more accurately recorded in the treatment journals this article is based upon.

The prospective case series published by Dr Breck McKay, Queensland, Australia also support these epidemiological findings from the perspective of a different continent[13]. As far as the author knows, there are no other published data on the prevalence of DGS in a general practice environment. Whilst there are limitations to the quality of the data presented in this paper, the results invite more formal studies of these condition findings. A larger prospective series with long-term follow-up and further pragmatic controlled trials of treatment are needed to define the potentially important role of the diagnosis and management of DGS in general practice. With more research and education about appropriate diagnosis and treatment there is a huge potential for relief of suffering and financial savings to the health system and employers of long-term back pain sufferers.

1: Blomberg S

A pragmatic approach to low-back pain including manual therapy and steroid injections in three RCT:s -A Multicentre Study in Primary Health Care Doctoral dissertation at Uppsala University 1993 from the Department of Family Medicine, University Hospital, S- 751 85 Uppsala.

2: Grunnesjo MI, Bogefeldt JP, Svardsudd KF, Blomberg SI. A randomized controlled clinical trial of stay-active care versus manual therapy in addition to stay-active care: functional variables and pain. J Manipulative Physiol Ther. 2004 Sep;27(7):431-41.

3: Thiele GH

Tonic Spasm of the levator ani, coccygeus and piriformis mscles Trans. Am. Pract. Soc. 37 (1936) 145-155

4: Diop M, Parratte B, Tatu L, Vuillier F, Faure A, Monnier G. Anatomical bases of superior gluteal nerve entrapment syndrome in the suprapiriformis foramen. Surg Radiol Anat. 2002 Aug-Sep;24(3-4):155-9. Epub 2002 Sep 11.

5: Babinski MA, Machado FA, Costa WS.

A rare variation in the high division of the sciatic nerve surrounding the superior gemellus muscle.

Eur J Morphol. 2003 Feb;41(1):41-2.

6: Fishman LM, Dombi GW, Michaelsen C, Ringel S, Rozbruch J, Rosner B, Weber C.

Piriformis syndrome: diagnosis, treatment, and outcome--a 10-year study. Arch Phys Med Rehabil. 2002 Mar;83(3):295-301.

7. Fishman LM, Anderson C, Rosner B.

BOTOX and physical therapy in the treatment of piriformis syndrome. Am J Phys Med Rehabil. 2002 Dec;81(12):936-42.

8: Filler AG, Haynes J, Jordan SE, Prager J, Villablanca JP, Farahani K, McBride DQ, Tsuruda JS, Morisoli B, Batzdorf U, Johnson JP

Sciatica of nondisc origin and piriformis syndrome: diagnosis by magnetic resonance neurography and interventional magnetic resonance imaging with outcome study of resulting treatment.

J Neurosurg Spine. 2005 Feb;2(2):99-115

9: Court Decision Länsrätten Stockholm Sweden 5970-02 2004-06-15

10: Broadhurst NA

Low back pain - is it the piriformis muscle? Journal of Orthopaedic Medicine 1991 (13) 8-11

11: Fishman LM, Schaefer MP

The piriformis syndrome is underdiagnosed. Muscle Nerve. Volume 28, Issue 5 (p 646-649)

12: Meknas K, Christensen A, Johansen O.

The internal obturator muscle may cause sciatic pain.

Pain. 2003 Jul;104(1-2):375-80.

13: McKay AB

Pain and Chronic Low Back Pain: A New Model?

Australasian Musculosceletal Medicine 2004 May, 9 (1):14-25

14: McKay AB Wall D

Aust. Musculoskeletal Med Nov 2003, Vol 8 (2): 87-99

The Orienting Response and the Functional Whole Human Body"

15:Beauchesne RP, Schutzer SF.

Myositis ossificans of the piriformis muscle: an unusual cause of piriformis syndrome. A case report.

J Bone Joint Surg Am. 1997 Jun;79(6):906-10.

16: Nakamura H, Seki M, Konishi S, Yamano Y, Takaoka K.

Piriformis syndrome diagnosed by cauda equina action potentials: report of two cases. Spine. 2003 Jan 15;28(2):E37-40.

17: Karl RD Jr, Yedinak MA, Hartshorne MF, Cawthon MA, Bauman JM, Howard WH, Bunker SR.

Scintigraphic appearance of the piriformis muscle syndrome.

Clin Nucl Med. 1985 May;10(5):361-3.

18: Lang AM.

Botulinum toxin type B in piriformis syndrome.

Am J Phys Med Rehabil. 2004 Mar;83(3):198-202.

19: Indrekvam K, Sudmann E.

Int Orthop. 2002;26(2):101-3.

Piriformis muscle syndrome in 19 patients treated by tenotomy--a 1- to 16-year follow-up study.

20: Bustamante S, Houlton PG.

Swelling of the leg, deep venous thrombosis and the piriformis syndrome. Pain Res Manag. 2001 Winter;6(4):200-3.

21: Benson ER, Schutzer SF.

Posttraumatic piriformis syndrome: diagnosis and results of operative treatment. J Bone Joint Surg Am. 1999 Jul;81(7):941-9.

22: Uchio Y, Nishikawa U, Ochi M, Shu N, Takata K. Bilateral piriformis syndrome after total hip arthroplasty. Arch Orthop Trauma Surg. 1998;117(3):177-9.

23: Grant JH.

Leg length inequality in piriformis syndrome.

J Am Osteopath Assoc. 1987 Jul;87(7):456.

24: Porta M. A comparative trial of botulinum toxin type A and methylprednisolone for the treatment of myofascial pain syndrome and pain from chronic muscle spasm. Pain. 2000 Mar;85(1-2):101-5.

25: Childers MK, Wilson DJ, Gnatz SM, Conway RR, Sherman AK Botulinum toxin type A use in piriformis muscle syndrome: a pilot study. Am J Phys Med Rehabil. 2002 Oct;81(10):751-9.

26: Beck PV, Mahajan G, Wilsey BL, Kreis PG, Fishman SM

Fluroscopic and electromyographic guided injection of the piriformis muscle with botulinum toxin type B

Pain Med. 2002 Jun;3(2):182-183.

27: Hanania M, Kitain E.

Perisciatic injection of steroid for the treatment of sciatica due to piriformis syndrome.

Reg Anesth Pain Med. 1998 Mar-Apr;23(2):223-8.

* Blomberg S, Myrin O, Silfverstolpe L Private communications