Editorials

Deep gluteal syndrome:

an overlooked cause of sciatica

Mention sciatica and most clinicians will first think of pressure on the lumbosacral nerve roots from a herniated intervertebral disc or stenosis of the spinal canal. Magnetic resonance imaging (MRI) raised hopes of making more precise anatomical diagnoses, but MRI, as routinely practised, fails to demonstrate the cause in as many as 49% of patients[1] while disc lesions are present in many asymptomatic people.

A less well known cause of sciatica is impingement of the nerve trunk from a range of musculoskeletal conditions that compress the sciatic nerve as it passes through the confines of the deep gluteal space, termed the deep gluteal syndrome (DGS). This editorial seeks to answer four questions. First, what is the evidence that deep gluteal syndrome exists? Second, how common is it? Third, are there accurate diagnostic tests? Fourth, are there effective treatments? MEDLINE, EMBASE, CINAHL and PEDro were searched using the term 'deep gluteal syndrome' without filters. Systematic reviews and primary research were assessed for bias and the best available evidence is reported. Non-systematic reviews were read for background and secondary references.

Compression of the nerve by the piriformis muscle was the first of such conditions to be described and led to the term 'piriformis syndrome'.[2] This term is being replaced by DGS as other musculoskeletal structures have been shown to cause sciatica, and in acknowledgement that we often do not understand the precise pathophysiology of a patient's symptoms and signs. It is estimated that between 6%[3] and 17%[4] of patients seen in secondary care with sciatica meet the diagnostic criteria for DGS, though difficulties with case definition may have led these two studies, which are the best of the prevalence studies, to underestimate the problem.

The best evidence that nerve impingement can cause sciatica comes from case reports linking the clinical picture, imaging, and perioperative findings. Surgical division of contracted or hypertrophied piriformis muscles seen on MRI has been shown to relieve symptoms.[2] While this might be dismissed as a placebo effect, successful outcomes have still been reported where previous, conventional lumbar surgery failed to have an effect.[5]

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ANATOMY AND DIAGNOSIS

The deep gluteal space lies between the gluteus maximus muscle posteriorly and the posterior surface of the femoral neck anteriorly (see https://www.angliangp.org/piriformis-syndrome for further anatomical information). The upper limit of the space is the inferior margin of the greater sciatic notch. It contains the lumbosacral plexus and several muscles, notably the piriformis, under which the sciatic nerve passes. The close relationship between nerve and muscle makes the nerve liable to impingement by changes in the piriformis muscle (such as hypertrophy or contracture) or as a result of congenital anomalies in the course of the sciatic nerve. At operation, changes in other muscles (such as the obturators or gemelli), and fibrous bands compressing the sciatic nerve, have more recently also been implicated as potential causes of DGS.[6] Space occupying structures have also been reported to mimic the syndrome: haematomas, abscesses, endometriotic deposits (causing cyclical sciatica), aneurysms, sarcomas, and metastases.

Four clinical features of DGS have been reported most frequently in the literature:[2]

- buttock pain;
- aggravation of pain on prolonged sitting;
- tenderness on deep palpation of the buttock; and,
- passive stretching or resisted contraction tests.

Deep palpation should be at the greater sciatic notch, found at a point one-third along an imaginary line from the greater trochanter towards the sacral hiatus.

Several diagnostic tests have been described[2] that increase tension in the muscles, and thereby put pressure on the sciatic nerve, so that a positive result reproduces or augments the pain. These include the seated piriformis test (forced passive stretching of a flexed, adducted, and internally rotated hip) and the active piriformis test (resistance to active abduction and external rotation of the hip). Links to descriptions and videos of these tests can be found at https://www.angliangp.org/copy-of-clinical-features.

A well conducted cross-sectional study, using endoscopic visualisation of actual compression as the reference standard, found that a combination of the seated and active piriformis tests had a sensitivity of 91% and specificity of 80%.[7] However, the accuracy may be an overestimate as the study was set in a tertiary centre.

The straight leg raising test (flexion of the hip with the straight leg reproducing the sciatic pain) may or may not be positive, and therefore is not useful in differentiating DGS from spinal sciatica.

The standard investigation for sciatica (MRI of the lumbosacral spine) does not visualise the deep gluteal space, and will not therefore identify DGS. Case series of pelvic MRI have demonstrated space occupying lesions and abnormalities of the piriformis muscle,[8] but pelvic MRI is not routinely performed in the UK. An enhanced form of MRI (MRI neurography) that can identify lesions of the sciatic nerve has also been proposed,[9] and electromyography may also show changes specific to sciatic nerve impingement.[10] However, good studies of diagnostic accuracy are lacking for both of these claims.

"The straight leg raising test ... is not useful in differentiating deep gluteal syndrome from spinal sciatica"

TREATMENT

Conservative treatments for DGS parallel those recommended for musculoskeletal conditions in general, but no controlled trial of physiotherapy (stretching muscles or mobilisation exercises) was found. Most reports for injection therapies (anaesthetic, corticosteroid, or botulinum toxin) and acupuncture come from case series or poorly reported trials, and even the few, better designed randomised controlled trials have small samples or significant flaws in their design.[11] A study by Fishman *et al* found a statistically significant difference between botulinum toxin and placebo, but it is not clear that this was clinically significant.[12]

Physiotherapy is the least invasive treatment and motivated patients can be directed to a video demonstration of home exercises at https://www.nhs.uk/video/Pages/sciatica-piriformis-syndrome.aspx.

Surgical nerve decompression has a place in selected cases of DGS. In one series of 60 patients, not only did pain improve, but so did the special tension tests, suggesting that release of nerve compression plays its part.[13] Unfortunately, the methods were incompletely reported, lacking details of how patients were recruited and whether assessments were made by independent observers, bringing the reliability of the results into question.

DIRECTION OF FUTURE RESEARCH

Although research concerning DGS is growing and improving in quality, study design remains problematic. The ideal study for prevalence and diagnosis would be a cross-sectional study in primary care, comparing clinical features against findings on both spinal and pelvic MRI scans. The resulting improvement in case definition could lead to well designed, large-scale trials of all modalities of treatment. Sadly, without any profit incentive from pharmaceutical sales, there is unlikely to be funding for such a multifaceted approach.

IMPLICATIONS FOR GPS

There is enough evidence to prompt greater attention to DGS as a possible explanation for some of the patients with sciatica for whom no cause is found on conventional testing. Given the current state of knowledge, there are two important things that GPs can do: diagnose the syndrome and refer the patient for physiotherapy.

GPs should suspect DGS when patients present with sciatica that is accompanied by pain in the buttock, and when the pain is aggravated by sitting. Another occasion to consider the diagnosis is when routine MRI has failed to find a cause for the sciatica. Clinical tests are simple: palpation, and one or two of the special tests described above. The paucity of evidence for treatment is not unique to DGS, shared as it is (albeit to a lesser degree) with disc lesions and spinal stenosis. Referral of suspected cases to physiotherapy is justified as it concurs with current NICE guidance for persistent sciatica.[14]

As a diagnostic term, DGS includes several pathologies which cannot be distinguished clinically. Its value lies in shifting the focus away from radiculopathy with two important consequences. First, it opens up treatment in the form of an alternative approach to physiotherapy and DGS-specific surgical options. Second, regardless of treatment, it answers patients' need for an explanation. Patients who do not receive an explanation for chronic back pain or sciatica suffer not only the direct consequences of their condition, but also the despair arising from the lack of a legitimising diagnosis.[15] Correctly identifying DGS as a specific entity adds precision to the diagnosis, helps the clinician to explain more clearly their patient's symptoms, and gives patients the legitimation many desperately need. Pending further research, GPs should consider diagnosing DGS to help patients understand their pain and to offer some hope from physiotherapy.

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