Piriformis Syndrome (PS) is an example of extra-spinal sciatica due to compressive neuropathy and can also be named as ‘deep gluteal syndrome’, ‘pelvic outlet syndrome’ or ‘pseudo-sciatica’. Its prevalence among Low Back Pain (LBP) sufferers ranged between 0.3% and 36%; though the condition is prevalent in women, men also get the disorder [1]. In PS, resultant localized gluteal and radiating LBP is due to a spasmodic Piriformis Muscle (PM) and compressed, irritated and stretched Sciatic Nerve (SN) behind the PM, respectively [1]. In clinical practice, sometimes regional gluteal pain predominates in PS, whereas in some patients, pain alike sciatica can be the presenting complaint confusing treating physician, leading to incorrect treatment of the patients. There are two types of PS – primary and secondary; in case of primary PS, the pathology is intrinsic to PM and or SN, however; in secondary PS, clinical features of associated disorders, like Lumbar Spinal Stenosis (LSS), Leg-Length Discrepancy (LLD), fibromyalgia (FMS), fatty wallet, etc., may complicate the clinical picture [2]. Although 70 years have elapsed after the first clinical description of PS, we are yet to define precisely the PS pathophysiology, etiology, unique clinical features, confirmed diagnostic tests, best treatment modality, natural history of the disease, etc. [3]. Published studies with Lower level of Evidence (LoE) address various aspects of PS and most of the study outcomes are inconclusive. So, we take the privilege of addressing areas of PS that might interest both clinicians and researchers in the field.

Who is prone to get PS?

Piriformis syndrome may affect people irrespective of age, sex and occupation [1]. However, the disorder is apparently more common in women than in men because of the wider pelvis and Q-angle [4]. It appears the condition affects young people less frequently [1]. Since the piriformis muscle is a deep seated structure, overuse of the lower back could pose deep gluteal musculatures including PM under sustained strain evoking clinical manifestations suggestive for PS. In our previous studies, we reported higher PS frequency among Bangladeshi housewives; however day laborers, drivers and bankers also had the disorder [1]. PS may cause problems in dancers [5]. Despite these studies still little is known about the prevalence of PS in men and women and more research is required.

What is the cause of piriformis syndrome?

It is very difficult to make conclusive remarks about PS causation, as few publications are addressing the question what may cause PS to develop. Based on previous LoE articles, overuse of PM may be one of the most important reasons, though most of the published works didn’t precisely define PM overuse, rather hypothesized how it could be [1]. In our previous research, we explained how the PM could get overused; however the way of overusing PM was not the same in all PS cases. So it is important to define exactly the PM overuse pathophysiology causing PS. Anomalous sciatic nerve and / or PM could contribute to PS features, though this idea is not confirmed by a recent large-scale collaborative radiologic study by Bertret and colleagues who didn’t find significant PS prevalence in anomalous sciatic nerves compared to normal [6]. Primary PS features could be due to spreading of myofascial trigger points within the PM. Direct gluteal trauma and a previous fall also may contribute to PS induction [1].

Sometimes PS is synonymously, though incorrectly used with wallet neuritis; in the latter condition patients usually complain about sciatica-like pain upon persistent gluteal compression due to an external fatty wallet and they rarely have deep seated regional gluteal pain and in most cases they are negative for PS maneuvers and most importantly they improve dramatically following radical ‘walletectomy’ [2]. However, both PS and wallet neuritis may simultaneously be present in a single patient [7]. Leg-Length Discrepancy (LLD) may be associated with PS. In LLD, the resultant asymmetry of pelvis poses lower lumbo-sacral segment, pelvis and deep gluteal structures including the PM under intense stretch contributing to PS features. According to the ACR 1990 classification criteria for Fibromyalgia syndrome (FMS), one of the 18 tender areas resides at the insertion of PM tendon to the posterior aspect of greater trochanter of the femur that could get irritated depending on the risk factors causing more gluteal pain or persistent regional gluteal pain. Because of inappropriately treated PS could induce the development of central sensitization which may result in generalized body ache, for example FMS [8,9].

The double crush hypothesis postulates that axons compressed at one site could also get compressed at another site and first came into
light by Upton and McComas in 1973. Based on this hypothesis higher prevalence of cervical radiculopathy is observed in patients with Carpal Tunnel Syndrome (CTS) than that of non-CTS [10]. Piriformis syndrome especially in association with LSS could be an example of ‘double crush syndrome’, as SN nerve roots and SN get compressed at lumbar spine and extra-spinal levels (behind PM), respectively; however requires further exploration.

Piriformis pyomyositis due to an invading infectious agent, such as Staphylococcus aureus, group A. Streptococcus, B. melitensis could cause severe gluteal pain with fever and require different treatment approach than usual for PS [11,12]. As of today, published papers regarding etiology and association of PS are scarce and mostly based on poor study samples, warranting more research.

What are the common diagnostic features of PS?

Regional and or radiating gluteal pain according to the sciatic nerve distribution is the mostly seen pattern in PS, often mimicking true lumbar spinal sciatica. In PS, gluteal pain may aggravate during longstanding sitting, especially on hard surface and is associated with tingling, numbness or burning sensations on the outer aspect of the ipsilateral leg. Pain also aggravates with walking, lying on the affected side, during attempted standing from sitting position or squatting, however, in chronic cases ambulation may provide some pain relief [1]. The most important peculiarity of PS diagnosis are its ‘inconsistent clinical manifestations’ and one of the most important reasons of why PS can be over-diagnosed is because of the ‘double crush syndrome’. Moreover, nerve conduction velocity studies and electromyogram of the affected gluteal region and ipsilateral lower limb can differentiate between myopathy and neuropathy - true spinal sciatica from extra-spinal sciatica [1,7]. Taheri et al. describe that the musculoskeletal ultrasonography has higher sensitivity in diagnosing hypoechoic myofascial trigger points and appears to be useful in diagnosing piriformis myofascial pain syndrome, however its reliability should be tested in a longitudinal study with large number study participants [15].

So in terms of confirmed diagnostic test for PS we are yet to have the most appropriate one.

Is PS life threatening?

It is very difficult to provide a straightforward answer of this question as studies addressing the fact are lacking. Most published works did not highlight this part of PS and rather mentioned it as a benign neuromuscular condition without any significant comorbid consequences, although it may be very painful and incapacitating. As of now, no published report document mortality issues associated with PS. However, recently, piriformis pyomyositis is being considered as a life threatening cause of PS and may be fatal when proper attention has not been given in terms of timely diagnosis and treatment [11,12]. In a
published case report Gaughan and colleagues hypothesized that transient intravenous cannula induced septicemia could complicate piriformis pyomyositis due to seeding of the infectious agent into the PM and generated gluteal pain with fever, treated successfully with antibiotics [11]. Jeon et al. documented PS features, gluteal atrophy and ipsilateral lower limb weakness due to radiotherapy induced scarring of the respective PM compressing SN vicinity in cervical cancer [16]. Lower limb weakness is reportedly possible in cases of bilateral PS [13]. However, all these study outcomes based on lower LoE and further research with large number of PS could provide further valuable information.

What are the most effective treatments for PS?

Before treating PS, it is of paramount importance to classify it. In case of primary PS, treatment should focus on intrinsic PM pathology only, however secondary PS deserves treatment for associated conditions. Commonly used pharmacological and non-pharmacological modalities that are found to be useful in PS: Non-Steroidal Anti-Inflammatory Drugs (NSAIDs), analgesics (including adjuvants), piriformis stretching exercise, activities of daily living modification; however, intra-lesional steroid-lidocaine combination and botulinum toxin-A injections are reportedly most effective in the disorder, but we are yet in the dark to know exactly how frequent and how long patients with PM should receive these sort of interventions, in order to experience complete pain relief [1]. PS secondary to PM infection doesn’t suit above modalities, judicial selection of antibiotics is indicated a [11,12]. Sometimes exploration of PM for surgical drainage, PM tendon resection or sciatic nerve neurolysis appears appropriate. So, best treatment approach for PS depends on myriad factors and there is no rule of thumb, for each individual a personal approach should be applied [1].

In conclusion, piriformis syndrome is an example of pseudo-sciatica with varying clinical manifestations. Our current knowledge about its pathophysiology, epidemiology, causation, risks association, clinical presentations, diagnosis, treatment, and consequences is insufficient and most importantly they are based on inconclusive study outcomes. So, future researchers could explore them and could provide more consistent information concerning piriformis syndrome.

References


