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Proximal Hamstring-Related Sciatic Nerve Entrapment Presenting as Recurrent Hamstring Strain in a Professional Footballer: A Case Report

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Learning Point of the Article:

This case illustrates the diagnostic challenge of persistent posterior thigh pain in elite athletes, demonstrating that symptom persistence despite normal imaging should not exclude proximal sciatic nerve involvement from the differential diagnosis.

Abstract

Introduction: Hamstring injuries are common in elite football and are associated with high recurrence rates. When posterior thigh pain persists or repeatedly recurs despite apparent tissue healing on imaging, mechanisms other than repeat muscle injury should be considered. Proximal hamstring-related sciatic nerve entrapment is an under-recognized cause of chronic posterior thigh pain that can mimic recurrent hamstring strain.

Case Report: This study reported a case of a 33-year-old male professional football midfielder who played for more than 20 years and had multiple clinically diagnosed recurrent hamstring strains over 12 months following an initial Grade II injury. Despite standard rehabilitation and magnetic resonance imaging demonstrating interval tissue healing, he continued to experience deep posterior thigh and buttock pain with high-intensity activity. Clinical reassessment reproduced concordant symptoms with neurodynamic provocation and palpation near the proximal hamstring–sciatic interface. A brief, targeted intervention focusing on sciatic nerve mobility and proximal neuromuscular interface release was administered following clinical suspicion of neural mechanosensitivity. The athlete returned to unrestricted training and competition and remained symptom-free at 14-month follow-up.

Conclusion: Persistent or recurrent posterior thigh pain following an apparently healed hamstring strain may arise from proximal sciatic nerve entrapment or mechanosensitivity rather than repeated muscle injury. Proximal neural involvement should therefore be included in the differential diagnosis, particularly when symptoms persist despite appropriate rehabilitation and normal or improving imaging findings.

Keywords: Chronic pain, football player, hamstring muscles, injuries, sciatic nerve.

Introduction

Proximal hamstring–related sciatic nerve entrapment occurs when fibrous or fibrotendinous scar tissue develops between the sciatic nerve and the ischial tuberosity after a proximal hamstring injury, compressing the nerve as the hamstring muscles contract [1]. Athletes at higher risk for proximal hamstring strain injury, such as sprinters and football players, may develop proximal hamstring–related sciatic nerve entrapment, resulting in recurrent phases of thigh or hemi-posterior pain similar to the pain experienced during the initial injury, even several months or years after the onset [1, 2, 3]. Pain in athletes has traditionally been equated with acute tissue injury, but this view can miss cases where pain persists beyond normal healing [4, 5]. By definition, acute sports injuries have a clear trauma and recovery timeline,

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whereas chronic pain lasts beyond expected healing (often >3 months) [6, 7]. The International Classification of Diseases -11 now recognizes categories such as chronic post-traumatic musculoskeletal pain and chronic neuropathic pain that go beyond structural injury [8, 9, 10]. In elite football, this distinction is consequential: longitudinal surveillance shows hamstring injuries now account for roughly a quarter of all reported injuries and a growing share of absence days, underscoring the cost of misclassification and delayed diagnosis [11]. In soccer, hamstring strains are extremely common (representing roughly 12–25% of time loss injuries) [12] and have a high recurrence rate; for example, Ekstrand et al. found that 18% of male professional football hamstring injuries were recurrences, often within 2 months of return [11]. However, when hamstring pain endures despite standard care, it may reflect central sensitization or neural pathology rather than ongoing muscle damage [13]. Recognizing this distinction, chronic pain versus acute injury, is crucial [14, 15]. Accordingly, contemporary pain science advises that persistent athletic pain may involve neuropathic or nociplastic mechanisms and cannot be fully explained by pathoanatomy alone [12, 16].

Hamstring injuries are notorious for slow and sometimes incomplete recovery, and recurrence estimates after return to play vary widely. Risk factors include large initial injury volume on magnetic resonance imaging (MRI), mild initial strains that may be under-treated, and prior lower limb surgery; persistent flexibility and strength deficits are also implicated [17]. Standard rehabilitation focuses on eccentric strengthening and neuromuscular control, yet even with modern protocols (e.g., Nordic exercises), population-level incidence has not declined [11]. In professional settings, a 25-player squad may accrue ~80 playing days (≈14 matches) lost per season from hamstring strains [11]. Recent league-level analyses further estimate direct monthly salary costs for hamstring strains of ≈ €47,000/club, with additional broadcast-linked losses when performance drops [18]. Clearly, any misclassification (treating chronic pain as acute injury) can multiply this burden [19].

Researchers have long recognized that the posterior thigh region can be a site of nerve-related pain. Adverse neural tension tests (e.g., slump test) are more often positive in athletes with repetitive hamstring symptoms than in healthy controls, supporting a neural contribution [20]. Proximal hamstring injury/tendinopathy may generate fibrosis near the ischial tuberosity, tethering or irritating the sciatic nerve, proximal hamstring tendinopathy-related sciatic nerve entrapment, with deep buttock/posterior thigh pain, sitting intolerance, and stretch-provoked symptoms that mimic recurrent strain [21]. Case reports confirm that tears or avulsions can secondarily compress the sciatic nerve and produce persistent radicular-type symptoms; in some instances, compression from a large hematoma has resolved under conservative care [18, 22]. Together, these observations indicate that persistent or rapidly recurrent symptoms after apparent tissue healing warrant targeted neural assessment [1].Clinically, neuropathic or nociplastic pain may be mislabeled as simple muscle strain. Piriformis-related sciatic entrapment in elite soccer illustrates how peripheral neural pathology can masquerade as hamstring injury; even when MRI suggests healing, symptoms may persist due to denervation or neural sensitivity. International pain experts therefore advocate a multidimensional view: Chronic pain cannot be fully explained by pathoanatomy alone [23]. Consistent with classic pseudoradicular descriptions, peripheral entrapments can mimic lumbosacral radiculopathy, so peripheral sources should be excluded before spine-directed or purely muscle-centric plans are pursued [12, 24].

Against this background, the present case report documents a professional midfielder with multiple clinically labeled (recurrent hamstring strains) whose symptoms resolved only after a brief, targeted neural-focused intervention at the proximal hamstring–sciatic interface. By integrating the case narrative with succinct prior evidence, we aim to illustrate practical indicators of proximal sciatic entrapment (e.g., sitting intolerance, stretch-provoked pain, positive neurodynamic testing, and imaging–symptom discordance), clarify how misclassification sustains relapse, and highlight a focused assessment pathway that can redirect management when muscle-centric rehabilitation fails.

Case Report

This retrospective case description is presented to illustrate the suspected mechanism of persistent posterior thigh pain in an elite footballer; no protocolized outcomes or statistical analyses were performed, and the intent is explanatory rather than to claim treatment efficacy. Informed consent for publication was obtained. To preserve anonymity, all identifying personal, professional, and imaging information has been generalized. The patient is a 33 year old male professional footballer with more than 20 years of competitive experience. He sustained an acute Grade II hamstring strain during a professional match. After conventional physiotherapy, he returned to play in 8 weeks but soon experienced persistent deep posterior thigh pain. MRI demonstrated interval resolution of the previously identified hamstring injury, with restoration of tendon continuity and no evidence of residual fiber disruption, re-tear, or edema. There were no features suggestive of acute muscle injury or associated structural pathology. He underwent extended rest and further rehabilitation, but each return cycle saw recurrence of symptoms. Over 12 months, he had three

documented "hamstring injuries," each halting play for several weeks. Team physicians considered him prone to strains, but treatments (strengthening, massage, injections) yielded only temporary relief. Across this period, there were no red flag features (e.g., progressive neurological deficit, systemic illness), and no new acute tearing events were identified on follow-up

Table 1: Distinguishing features between acute hamstring injury and chronic post-strain hamstring pain

Feature	Acute hamstring injury (sports injury model)	Chronic post-strain hamstring pain/chronic pain mechanism
Typical onset	Clear, identifiable incident (sprint, acceleration, stretch), often described as a sudden "pull" or "pop."	May follow an initial strain but persists beyond expected healing; can recur across training/match cycles without a clear new tearing event.
Expected healing timeline	Symptoms generally improve along a predictable trajectory with rehabilitation within weeks (severity dependent)	Symptoms persist beyond the expected time for tissue healing (often >3–6 months) or recur despite appropriate rehabilitation and rest.
Pain location	Posterior thigh at the muscle–tendon unit; localized tenderness at the injury site	Often deep proximal pain (gluteal/ischial region) and/or posterior thigh discomfort; may feel "deeper" and less clearly localized
Pain quality	More mechanically linked to load; sharp pain with high-speed running or stretching	Can be disproportionate to load; may include burning, aching, sharp episodes; pain can feel "stuck" despite improved strength/conditioning.
Aggravating factors	Sprinting, acceleration, eccentric loading, and stretching reproduce local pain.	Sitting intolerance, stretch-provoked buttock/thigh pain, neural tension positions (e.g., slump); symptoms may flare unpredictably with return-to-play demands.
Neurological features	Usually absent	May include radiating symptoms, neural tension signs, "tightness" that behaves like nerve sensitivity rather than muscle stiffness
Clinical tests	Local palpation tenderness; pain on resisted knee flexion/hip extension; deficits in eccentric strength are common	Neural tension tests may reproduce posterior thigh pain (e.g., slump-related provocation); pain may reproduce with compression/irritation near the ischial region.
Imaging correlation	MRI/ultrasound is more likely to show acute strain features consistent with symptoms	Imaging may show healed tissue or tendinopathy that does not fully explain symptom severity; discordance between imaging and pain/function can occur.
Response to rest	Often improves reliably with rest and graded rehab	Rest may reduce symptoms temporarily, but recurrence occurs with performance demands; repeated "return–flare–rest" cycles are common
Response to standard rehab	Typically improves with progressive loading, eccentric strengthening, and neuromuscular control.	Often incomplete or short-lived response; repeated rehab cycles may not produce durable resolution if neural/chronic pain drivers persist.
Conceptual driver (as framed in this paper)	Primary tissue injury and local healing/rehabilitation	Persistent posterior thigh pain maintained by neural entrapment/adverse neural tension and/or pain sensitization mechanisms following the initial injury
Practical implication	Treat as acute injury: tissue healing + graded return-to-play	Consider reclassification and broader assessment (neural, lumbopelvic contributors, chronic pain features) to prevent repeated mislabeling as reinjury

MRI: Magnetic resonance imaging

imaging until the episode described below.

MRI assessment of the posterior thigh and proximal hamstring region demonstrated no signs of acute or recurrent musculotendinous injury. The proximal hamstring tendon appeared structurally intact, with preserved attachment at the ischial tuberosity and no evidence of avulsion or retraction. Signal characteristics were consistent with resolved prior injury, with no active edema or hemorrhagic changes. No space-occupying lesion, hematoma, or structural abnormality compressing the sciatic nerve was identified on standard imaging sequences. Notably, the absence of imaging abnormalities did not correlate with the severity and persistence of the patient's symptoms, raising suspicion of a non-structural or neural pain driver.

At presentation to our clinic, the player described persistent hamstring pain and was initially unconvinced that the origin was neural rather than muscular. Clinical examination reproduced concordant posterior thigh pain with neurodynamic provocation (slump-type tensioning), and palpation near the ischial origin elicited deep buttock discomfort; routine myotomal strength and reflexes were otherwise unremarkable, and the prior MRI documented interval tissue healing characterized by normalization of signal intensity and preserved structural integrity of the proximal hamstring complex, without evidence of recurrent tearing, intramuscular edema, or surrounding soft tissue abnormality. At the time of presentation to our clinic, the athlete was not undergoing any concurrent standard or conventional treatment, as he had completed previous rehabilitation attempts without sustained benefit. No physiotherapy, medication, or procedural interventions were provided before, during, or after the complementary session. Following a single session targeting sciatic nerve mobility and neuromuscular release, he experienced immediate and complete resolution of symptoms. Although pain-free, he continued treatment for five weeks as a preventive strategy. At 14-month follow-up, he remained entirely symptom-free, with no recurrence of tightness or discomfort. During this interval, he trained and competed without restriction, tolerating sprinting and other high-speed activities without posterior thigh symptoms.

After this period of sustained symptom resolution, the athlete later developed a new episode of lower back pain while maintaining full training and match demands. During this time, he reported emerging posterior thigh discomfort and subsequently sustained an acute Grade II hamstring tear. A focused clinical review and treatment session addressing both lumbopelvic contributors and the hamstring region was provided approximately 1 week after the injury, after which he returned to running within ~12 days. This follow-up development is presented to complete the clinical course and does not imply treatment efficacy; rather, the sequence is consistent with the broader clinical framework discussed in this report, in which recurrent posterior thigh presentations in elite football may involve lumbopelvic and neuromuscular contributors rather than isolated local tissue pathology alone.

Discussion

This case report aimed to demonstrate that persistent or recurrent posterior thigh symptoms after an apparently healed hamstring strain can reflect a proximal neural driver and to describe the clinical course following a brief neural-focused intervention. The main findings were persistent deep buttock or posterior thigh pain despite interval MRI healing, reproduction of concordant pain with neurodynamic provocation and palpation near the ischial origin, immediate and durable symptom resolution after targeted sciatic-focused treatment, and a later, distinct episode associated with lumbopelvic symptoms, underscoring the need to screen for proximal contributors. Although MRI played a role in excluding recurrent structural injury, imaging findings were not presented in this report to maintain strict patient anonymity. Instead, detailed narrative descriptions were provided. This approach aligns with case reporting standards in situations where patient identifiability is a concern, particularly in elite sport.

High-profile careers show the stakes of mismanaged hamstring pain, highlighting how poor load management and rushed return-to-play decisions can have long-term functional and professional consequences [25, 26, 27]. Michael Owen, once England's superstar, attributed the cascade of injuries that shortened his career to an inadequately rehabilitated hamstring rupture at age 19 [28]. In his words, "I think that left me compromised for the rest of my career… it went from hamstrings to groins, to thighs, and it almost took a cycle around my body." His experience underscores how a single hamstring injury can trigger widespread biomechanical and pain issues. More recently, England defender Ben Chilwell's chronic hamstring/gluteal pain has repeatedly sidelined him; clubs have publicly stated his scan-positive "high-grade strain" is proving unusually difficult to rehabilitate. These examples suggest that some elite players suffer not only muscle tears but also complex post-injury pain syndromes [7, 17, 29]. Our case aligns with this perspective by demonstrating features more consistent with a proximal neural pain generator than with a simple repeat tear, particularly the imaging, symptom discordance, and the immediate response to neural-focused care.

Beyond individual careers, the economic and performance burden is immense. As noted, hamstring strains rank among the costliest injuries: Nieto Torrejón et al. reported that each

monthly hamstring-injury-related absence cost LaLiga clubs ~€47,000 on average, and prolonged strains can tip a team's league placement by reducing broadcast revenue [11, 23]. In England, each club loses dozens of matches per season to hamstring injuries. Misclassifying chronic cases as simple reinjury leads to repetitive treatments, imaging, and lost time without resolving the underlying pathology [11, 30]. It inflates medical costs and deprives teams of key players [18]. In practical terms, our case illustrates how a brief neural screen can redirect management when symptoms persist despite healed imaging, helping to avoid serial muscle-centric rehabs that do not address the driver [31, 32].

Compared with prior reports, several features of this patient's presentation echo published signals for proximal sciatic involvement: Deep gluteal/posterior thigh pain aggravated by sitting or stretch; reproduction of concordant symptoms with slump-type provocation in athletes with recurrent hamstring complaints; and improvement when neural mobility or perineural irritability is addressed. Case literature has also documented delayed sciatic neuropathy after hamstring injury (e.g., hematoma or scarring around the nerve) with symptoms that may not map to fresh tearing and can improve with conservative care [33, 34]. A contemporary pain science perspective further supports a multidimensional appraisal, noting that persistent post-injury pain in athletes can involve nociplastic contributions alongside peripheral nociceptive sources [35, 36].

Clinically, when a returning athlete reports disproportionate posterior-thigh symptoms despite adequate strength and a "healed" appearance on imaging, a short neural screen, neurodynamic testing, and palpation at the ischial origin can be incorporated alongside tendon-load progression rather than replacing it. A concise differentiation of clinical features that typically favor an acute strain versus a chronic post-strain neural dominant pattern is summarized in Table 1.

Limitations include the single-case nature, the lack of contemporaneous objective neuromuscular measures, and the absence of imaging specifically targeted at perineural fibrosis in the index episode; therefore, causal inferences about specific interventions cannot be made, and the observations are hypothesis-generating only.

Conclusion

Recurrent posterior thigh pain after an apparently healed hamstring strain may reflect a proximal neural driver rather than repeat tearing. In athletes with imaging–symptom discordance and provocation on neurodynamic testing, embedding a brief neural-focused assessment within standard care may reduce misclassification and support a more durable return to performance.

Clinical Message

Recurrent posterior thigh pain despite apparent tissue healing may reflect a neural rather than muscular origin. A brief neural-focused assessment can aid diagnosis and prevent repeated misclassification as re-injury.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given the consent for his/ her images and other clinical information to be reported in the journal. The patient understands that his/ her names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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