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CASE REPORT

Ultrasound-Guided Pudendal Nerve Pulsed Radiofrequency for Refractory Multi-Territory Genital Neuropathic Pain: A Case Report

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Abstract

Background: Chronic neuropathic pain confined to the external genitalia is uncommon in young men, is frequently misattributed to infective or inflammatory urological disease, and often proves refractory to conventional therapy. Pudendal neuralgia is the best-characterised cause, but pain frequently crosses nerve territories, complicating both diagnosis and target selection.

Case Presentation: A 22-year-old man had two years of continuous glans, preputial and right testicular pain (numeric rating scale 10/10) with paraesthesia, allodynia and hyperalgesia, beginning after a suspected fluoroquinolone-associated genital eruption with ulceration. Urological, neurological and dermatological evaluation was unrevealing, and prior intradermal botulinum toxin had failed. A diagnostic pudendal block abolished testicular pain entirely but produced only 30–40% global relief; gabapentin and duloxetine were then started, with pain plateauing before pudendal pulsed radiofrequency (PRF) was performed under ultrasound guidance, producing 70% pain reduction sustained at 11 months after the medications were tapered and discontinued.

Discussion: The patient did not fulfil the Nantes criteria for pudendal neuralgia; the working diagnosis was therefore pudendal-involvement genital neuropathic pain rather than confirmed pudendal neuralgia. The anatomically incongruent block response may be explained by viscero-somatic convergence in the sacral dorsal horn with central sensitisation, presented here as a hypothesis rather than an established mechanism. Failure of intradermal botulinum toxin argued against a peripherally maintained terminal generator and is consistent with, though does not confirm, a more proximal target. PRF was chosen over thermal lesioning to avoid sphincteric and sexual morbidity in a young man, and ultrasound over fluoroscopy to permit direct visualisation of the internal pudendal artery.

Conclusion: Ultrasound-guided pudendal PRF may provide meaningful analgesia in selected patients with refractory genital neuropathic pain when a diagnostic block suggests pudendal involvement, even when pain extends beyond classical pudendal territory and formal Nantes criteria are not met.

Keywords

Pudendal Neuralgia; Pulsed Radiofrequency; Central Sensitization

Introduction

Chronic penile and scrotal pain in a young man is uncommon and diagnostically challenging. Patients may undergo repeated negative investigations and empirical antimicrobial treatment before neuropathic mechanisms are considered.[1,2] Pudendal neuralgia is the best-characterised cause and is defined clinically by the Nantes criteria: pain in the pudendal territory, aggravation by sitting, absence of nocturnal waking, no objective sensory loss, and relief following diagnostic pudendal block.[1] However, genital pain may also arise from the ilioinguinal or genitofemoral nerves, spermatic afferents, or local mucocutaneous disease, and pain crossing territories may not fit a single diagnostic template.[2,3]

Pulsed radiofrequency (PRF) delivers high-voltage electrical fields in short bursts while limiting tissue temperature to 42°C, producing neuromodulatory rather than neurodestructive effects.[4–6] This is attractive for the pudendal nerve, which contains motor fibres to the sphincters and sensory fibres involved in sexual function. We report refractory multi-territory pudendal-involvement genital neuropathic pain not fulfilling the Nantes criteria for pudendal neuralgia in a young man with an anatomically incongruent response to diagnostic pudendal block, treated with ultrasound-guided bilateral pudendal PRF. This manuscript follows CARE reporting guideline. Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Case Presentation

A 22-year-old man presented with a two-year history of continuous burning pain involving the glans, prepuce and right testis, rated 10/10 on the numeric rating scale (NRS), with pins-and-needles paraesthesia. Pain did not radiate to the perineum, buttock or thigh and had no aggravating or relieving factors, including no worsening with sitting. Sleep and daily activity were preserved. Symptoms began after a suspected hypersensitivity reaction to ofloxacin associated with genital pain and ulceration. The ulceration healed, but pain persisted. There were no urinary or ejaculatory symptoms, oral or ocular ulceration, diabetes, trauma, pelvic surgery, cycling or straddle injury. Urological and neurological evaluations were unrevealing, and intradermal botulinum toxin injected into the glans and prepuce elsewhere had produced no benefit.

The external genitalia were structurally normal, without ulceration, sclerosis, phimosis or scarring. Mechanical allodynia and pinprick hyperalgesia were present over the glans, prepuce and right hemiscrotum. The cord and epididymis were normal; cremasteric reflex, perineal sensation, anal tone and lower-limb neurological examination were normal. Tinel's sign was absent over the ischial spine and inguinal canal. DN4 was 5/10, consistent with neuropathic pain.[7]

Scrotal ultrasonography with colour Doppler, urinalysis and culture, expressed prostatic secretions, magnetic resonance imaging of the lumbosacral spine and pelvis, glycated haemoglobin, vitamin B12, thyroid function, antinuclear antibody and viral serology were normal. Dermatological review was also unrevealing.

A bilateral ultrasound-guided pudendal nerve block was performed at the ischial spine with 4 mL of 0.25% bupivacaine and 4 mg dexamethasone per side. Right testicular pain was completely abolished, but global relief was only 30–40%, with persistent glans and preputial pain.

Gabapentin 200 mg/day was started at the first clinic visit and duloxetine 60 mg/day one week later along with pain neuroscience education and graded activity, reducing pain to NRS 5/10 with approximately 40% patient global improvement. Pain then plateaued at NRS 5/10 for approximately two months after the diagnostic block, remaining above the patient's acceptable state.

Bilateral pudendal PRF was subsequently performed under ultrasound guidance with the patient prone. A low-frequency curvilinear transducer was positioned transgluteally at the ischial spine. The ischial spine and sacrospinous ligament were identified, colour Doppler located the internal pudendal artery, and the pudendal nerve was visualised in the interligamentous plane medial to the artery. A 20G radiofrequency cannula with a 10-mm active tip was advanced in-plane from lateral to medial. Concordant genital paraesthesia occurred at 0.5 V with 50-Hz sensory stimulation, and anal sphincter contraction at 1.5 V with 2-Hz motor stimulation. PRF was delivered at 42°C, 45 V, 20-ms pulse width and 2 Hz for 240 seconds per side. There was no vascular puncture, motor deficit, urinary retention or sexual dysfunction. At four weeks, pain had decreased to NRS 2/10 with 70% patient global improvement, with marked reduction in allodynia and hyperalgesia. Following PRF, gabapentin and duloxetine were continued for 1 month and then progressively tapered and eventually discontinued. Pain remained at NRS 2–3/10 after discontinuation, and the benefit was sustained at 11 months.

Discussion

The principal diagnostic challenge was pain spanning different nerve territories. The glans and prepuce are supplied by the dorsal nerve of the penis, a pudendal branch (S2–S4), whereas testicular afferents travel through the genital branch of the genitofemoral nerve (L1–L2), ilioinguinal nerve (L1) and sympathetic fibres accompanying the spermatic plexus (T10–T11).[3] Thus, no single peripheral nerve anatomically explains the complete presentation.

Local mucocutaneous disease was considered because of the ulcerative onset. Lichen sclerosus was unlikely because examination showed no sclerosis, atrophy or meatal narrowing.[8] Behçet's disease was unsupported by the absence of recurrent oral aphthae, ocular involvement and pathergy.[9] The initial event was most consistent with a fixed drug eruption related to the fluoroquinolone.[10] Normal scrotal imaging and cultures argued against structural scrotal disease and infection, while MRI excluded recognised spinal causes of referred testicular pain and other relevant lumbosacral pathology. Metabolic and infective neuropathies were not identified. The patient lacked the characteristic worsening with sitting and therefore did not fulfil the Nantes criteria for pudendal neuralgia, and the testicular component lay outside the pudendal territory. However, he had neuropathic genital pain with objective allodynia and hyperalgesia and a partial response to diagnostic pudendal block suggesting pudendal involvement. A working diagnosis of pudendal-involvement genital neuropathic pain with possible superimposed central sensitisation was therefore made, rather than a diagnosis of confirmed pudendal neuralgia or pudendal nerve entrapment.

The most informative finding was the mismatch between expected anatomy and block response. A pudendal block would be expected to preferentially relieve glans and preputial pain while leaving testicular pain relatively unaffected; here, testicular pain disappeared whereas glans and preputial pain persisted. Technical factors such as cephalad spread of injectate may account for this; alternatively, viscero-somatic convergence offers one possible hypothetical explanation: visceral and somatic afferents can converge on shared second-order neurons, and central sensitisation may allow interruption of one afferent input to transiently suppress pain arising from a broader convergent network.[11] The persistent glans and preputial component may represent a partly independent post-inflammatory small-fibre generator following mucosal

ulceration. An anatomically incongruent block response may therefore warrant consideration of alternative mechanisms, although technical explanations cannot be excluded.

The failure of intradermal botulinum toxin may also be relevant. Its peripheral actions include inhibition of nociceptive transmitter release and modulation of TRPV1 trafficking.[12] Its complete failure might suggest a more proximal pain generator, although non-response has several possible explanations, including dose, injection depth and patient selection. Conventional thermal radiofrequency was avoided because coagulative lesioning of the pudendal nerve could jeopardise sphincteric and sexual function in a 22-year-old. PRF instead delivers short pulses separated by silent phases, limiting tissue heating and producing neuromodulatory effects with preservation of motor conduction.[4–6,13]

Published evidence for pudendal PRF remains limited to case reports and small series, but reports describe meaningful relief lasting approximately three to twelve months, with repeat procedures generally effective and no reported sphincteric or sexual morbidity.[14–16] This patient differs from most published cases, which describe pudendal neuralgia, because he did not fulfil the Nantes criteria, was younger, and had pain extending beyond the pudendal territory. PRF was therefore undertaken with the expectation of substantial but potentially incomplete relief. The sustained 70% improvement at 11 months, maintained after gabapentin and duloxetine were discontinued, is consistent with a contribution from the pudendal component even when central or overlapping mechanisms may be present, although the preceding pharmacotherapy and education may also have contributed.

Several features favour PRF as the main contributor to the additional improvement, although they cannot establish causality. First, pain had plateaued at NRS 5/10 on gabapentin and duloxetine for approximately two months before PRF, by which time the analgesic effect of both drugs would generally be expected to be established; a delayed pharmacological effect is therefore unlikely to explain the further fall to NRS 2/10 within four weeks of the procedure. Second, pain remained at NRS 2–3/10 after both medications were tapered and discontinued, whereas a predominantly drug-mediated benefit would be expected to diminish on withdrawal. Third, pain had been continuous and severe for two years before presentation, making spontaneous improvement coinciding with PRF less likely. Nevertheless, a delayed or cumulative effect of pain neuroscience education and graded activity, regression to the mean, or natural fluctuation cannot be excluded in a single uncontrolled case.

Ultrasound was selected instead of fluoroscopy. Fluoroscopy identifies the ischial spine but does not directly demonstrate the nerve, sacrospinous ligament or internal pudendal artery. Ultrasound with colour Doppler permits direct visualisation, in-plane needle tracking and identification of the artery, while avoiding ionising radiation in a young patient who may require future procedures.[17,18] A randomised comparison found ultrasound- and fluoroscopy-guided pudendal blocks comparable in analgesic efficacy and procedural time, supporting ultrasound where appropriate expertise is available.[19]

Several learning points emerge. First, unexpected relief of a non-pudendal territory after pudendal block raises the possibility of convergence and central sensitisation, although technical explanations should also be considered. Second, failure of a well-targeted peripheral treatment may prompt consideration of a more proximal pain generator. Third, severe reported pain with preserved sleep and function should not be interpreted as exaggeration; possible nociplastic mechanisms warrant pain neuroscience education and appropriate psychometric assessment alongside intervention.[11,20]

This report is limited by its single-patient, uncontrolled design. The patient did not meet the classic Nantes criteria for pudendal neuralgia, particularly because there was no aggravation

with sitting; therefore, the diagnosis should be considered pudendal involvement rather than confirmed pudendal neuralgia or entrapment. The diagnostic pudendal block was neither repeated nor placebo-controlled. Gabapentin, duloxetine, pain neuroscience education, and graded activity were introduced before PRF and could independently have contributed to symptom improvement. The further reduction in pain after PRF and its persistence after both medications were tapered and discontinued make a purely pharmacological explanation less likely, but contribution from these co-interventions cannot be excluded, limiting attribution of the response to PRF. Outcome assessment relied primarily on NRS and patient global impression; inclusion of a validated neuropathic pain measure and/or quality-of-life or functional outcome measure would have strengthened the assessment. The mechanistic explanations involving viscero-somatic convergence, central sensitisation, and interpretation of botulinum toxin failure as evidence of a proximal pain generator remain hypotheses and cannot be established from a single case. Longer follow-up is required because PRF effects may attenuate. If analgesia wanes, dorsal root ganglion stimulation at S2–S4, or L1 for isolated recurrent testicular pain, may provide more selective targeting in these territories.[21–23]

Conclusion

Refractory genital pain in a young man warrants structured, mechanism-based evaluation rather than repeated empirical antimicrobial or surgical treatment. When diagnostic testing suggests pudendal involvement, ultrasound-guided pudendal PRF may provide meaningful analgesia without the functional risks of thermal lesioning or the radiation exposure of fluoroscopy. An anatomically incongruent block response may be informative and may prompt consideration of convergence and central sensitisation alongside technical explanations.

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Declarations

Ethics Approval

Not applicable. As a single de-identified case report, formal institutional ethics committee approval was not required per journal policy.

Consent for Publication

Written informed consent was obtained from the patient for publication of this case report and accompanying images. The patient was informed that identifying information would not be disclosed.

Conflict of Interest

The authors declare that they have no conflict of interest related to this case report.

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Author Contributions

Dr Swati Verma: conceptualization, clinical management, and manuscript writing. Dr Sachin Kumar: supervision and critical review. Dr Meghna Kahlon: data collection and manuscript review. Dr Bhavana D R: data collection and manuscript review. All authors approved the final manuscript and agree to be accountable for the work.