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Persistent genital arousal disorder: a special sense neuropathy

Anne Louise Oaklander[a,b], Saurabh Sharma[c,*], Katie Kessler[c], Bruce H. Price[a,c]

Abstract

Introduction: Persistent genital arousal (PGAD) is a syndrome of unprovoked sexual arousal/orgasm of uncertain cause primarily reported in female patients. Most patients are referred for mental-health treatment, but as research suggests associations with neurological symptoms and conditions, there is need to analyze cases comprehensively evaluated by neurologists.

Methods: The IRB waived consent requirements for this retrospective university-hospital study. We extracted and analyzed neurological symptoms, test, and treatment results from all qualifying participants' records and recontacted some for details.

Results: All 10 participants were female; their PGAD symptoms began between ages 11 to 70 years. Two patterns emerged: 80% reported daily out-of-context sexual arousal episodes (≤30/day) that usually included orgasm and 40% reported lesser, often longer-lasting, nonorgasmic arousals. Most also had symptoms consistent with sacral neuropathy—70% had urologic complaints and 60% had neuropathic perineal or buttock pain. In 90% of patients, diagnostic testing identified anatomically appropriate and plausibly causal neurological lesions. Sacral dorsal-root Tarlov cysts were most common (in 4), then sensory polyneuropathy (2). One had spina bifida occulta and another drug-withdrawal effect as apparently causal; lumbosacral disc herniation was suspected in another. Neurological treatments cured or significantly improved PGAD symptoms in 4/5 patients, including 2 cures.

Conclusions: Although limited by small size and referral bias to neurologists, this series strengthens associations with Tarlov cysts and sensory polyneuropathy and suggests new ones. We hypothesize that many cases of PGAD are caused by unprovoked firing of C-fibers in the regional special sensory neurons that subserve sexual arousal. Some PGAD symptoms may share pathophysiologic mechanisms with neuropathic pain and itch.

Keywords: Neuropathic pain, Pelvic pain, Tarlov cysts, Peripheral neuropathy, Spinal cord, C-fibers

1. Introduction

The anatomy and physiology—and thus the innervation—of sexual arousal are dimorphic, but it has been studied almost exclusively in male patients, and the peripheral and spinal pathways and neurotransmitters mapped primarily in rodents.[15,16,27] Studies mapping human arousal are rare and mostly conducted in spinal cord–injured or multiple sclerosis patients.[1,20] Veterans Administration and other investigators have studied effects of myelopathies, radiculopathies, neuropathies, and various medications on male arousal,[20] but research in female patients is nearly nonexistent. Women's complaints of inappropriate arousal are typically attributed (by predominantly male evaluators) to psychopathology or misinterpreted as beneficial.[4]

Here, we begin neurological investigation of persistent genital arousal disorder (PGAD), a largely female-reported syndrome of out-of-context sexual arousal and/or orgasm. PGAD has been mostly investigated by psychologists. With physicians and neuroscientists largely unaware of it, medical causality has not been systematically investigated.[14] Feigenbaum and Komisaruk established the firmest association to date, with sacral Tarlov cysts. These form exclusively on and can damage sensory ganglia and roots.[2,11] Some cases are attributed to brain effects of serotonergic and dopaminergic drugs,[5,22] and sexologists have hypothesized that other neurological problems may be associated, mentioning restless leg syndrome, fibromyalgia, genital sensory hyperesthesia, neuropathic pain, and sensory neuropathy, but we are unaware of previous neurologically focused investigations.[7,8,17,19,22–24,26]

Sponsorships or competing interests that may be relevant to content are disclosed at the end of this article.

[a] Department of Neurology, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA, [b] Department of Pathology (Neuropathology), Massachusetts General Hospital, Boston, MA, USA, [c] Department of Neurology, McLean Hospital, Harvard Medical School, Belmont, MA, USA

**Corresponding author. Address: 99 Beauvoir Ave, Summit, NJ 07901. Tel: 908-522-2829; fax: 214-702-0473. E-mail address: emailsaurabhsharma@gmail.com (S. Sharma).*

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2. Methods

A lack of standardized nomenclature (synonyms include persistent sexual arousal syndrome and restless genital syndrome) and billing codes precluded systematic case ascertainment, so we reviewed records from our university–hospital neurology practices for PGAD mentions and solicited additional referrals regardless of whether neurological symptoms were present. The review board waived consent, although we obtained verbal consent to anonymous publication. All genders and ages were eligible; inclusion required neurological evaluation of diagnosed or suspected PGAD, and some patients were reinterviewed. We analyzed demographics, medical histories and examinations, results about localization, etiology, and treatment.

3. Results

All participants were female, and on average 53.4 years old on December 31, 2018 (**Table 1**). Ages at PGAD onset ranged from puberty to postmenopausal. We identified 2 patterns of arousal—episodic and sustained. Eighty percent of patients reported daily transient sexual arousals (minutes/few hours) with 40% reporting longer, lesser near-continuous arousals for days-years (2 had both). All PGAD illnesses began as anorgasmic but almost always progressed to include spontaneous orgasms. Patient 4, with ≤30 arousals daily, had 2 unprovoked orgasms in front of a hospital teaching-conference audience. Almost all patients tried masturbation to terminate arousals, and this helped 20%.[13] Patient 10 masturbated 4 to 5 times daily despite the lack of pleasure, to obtain a few hours relief. Patient 3 induced several orgasms each afternoon to quell symptoms until the next morning. Five reported no postorgasm refractory relief, and patient 6 avoided all vulvar contact because of allodynia.

Chronic PGAD always terminated sexual relations. All 6 partnered patients initially sought sex during their arousals, but all of their partners came to perceive their approaches as too frequent and/or "mechanical," and terminated sexual relations, although all marriages continued. Among the 3 patients who were virgins at PGAD onset, 2 remained abstinent and 1 tried intercourse only once, an encounter abrogated by vulvodynia. Every patient reported that PGAD caused new or worse depression and anxiety. Onset in childhood was bewildering, causing confusion, shame, and fear. All patients considered themselves disabled from PGAD and associated symptoms, and most had curtailed daily activities. At presentation, only 20% of patients' physicians recognized their symptoms as PGAD, so most self-diagnosed online. Before onset, all were functioning well and none had major psychiatric diagnoses, yet several reported psychiatric attribution and treatment—for example, with sex therapy and electroconvulsive treatments.

Eighty percent of patients first sought care for their other pelvic symptoms and only mentioned PGAD after establishing trust. Among medical consultations, neurological evaluations were the most productive. They documented colocalizing somatosensory symptoms in 90%, including perineal, buttock, or leg pain and/or sensory loss. Neurological testing was also productive, with 78% (6/8) of sacral magnetic resonance imaging studies revealing radiculopathy, 2/2 nerve-conduction studies diagnostic for sensory polyneuropathy along with 2/5 lower-leg, PGP9.5-immunolabeled skin biopsies. Among 2 composite autonomic function tests, 1 was abnormal, the other borderline. Abnormal urodynamic and anorectal manometry testing confirmed myelopathy in the spina bifida patient. Four electroencephalograms in 2 patients were unremarkable, including one capturing 4 spontaneous orgasms.[9]

Psychiatric treatment was universally ineffective, including 7 psychiatric hospitalizations and 17 electroconvulsive therapy sessions for patient 10. Gynecological and urological treatments, including medications, injections, and electrotherapies, were also ineffective. Local anesthetics and/or corticosteroid injections never had lasting benefit, but a few gave temporary relief, suggesting the potential for diagnostic localization of hyperexcitable sensory nerves as with neuropathic pain conditions. Genitofemoral nerve blocks gave transient relief to 2/3, but pudendal nerve blocks were ineffective or worsened symptoms in 5/5. All epidural corticosteroid injections worsened symptoms. One intravaginal botulinum toxin administration was ineffective.

By contrast, neurological treatment was effective in 80% of patients. Gradual duloxetine taper (patient 5) and Tarlov-cyst resection (patient 2; **Fig. 1**) were curative. Immunoglobulins (2 grams/kg/4 weeks) improved patient 8's PGAD and motor symptoms dramatically. Another Tarlov-cyst patient found intrathecal pressure reduction helpful, whereas surgical resection was ineffective for another.

4. Discussion

This report associates PGAD with disorders and lesions of the lower spinal cord, roots, and nerves that control sexual arousal and orgasm. Genital sensory innervation is mostly through the dorsal nerve of the clitoris/penis, a branch of the pudendal nerve that enters the cord through S4 dorsal roots to excite T12-S1 dorsal-horn interneurons (lamina VI and X[27])[1,16] and send axons up the dorsal columns and gracile fasciculus to affect the brain widely.[3,12] In female rats, electrophysiological recordings link pelvic contractions to rhythmic pudendal nerve firing, with L4 spinal cord injury increasing this firing.[1] Autonomic mapping in mice identifies hypogastric sympathetic afferents entering at L2,[16] with efferents exiting the T12-L2 ventral roots and white rami to synapse paravertebrally then send postganglionic fibers through gray rami to the hypogastric nerve. Parasympathetic efferents arise from S2-5, exit as splanchnic nerves through the inferior hypogastric plexus to synapse in ganglia in pelvic organ walls, and increase pelvic blood flow and other arousal responses.

Given our patients' lesion localizations, etiologies, and colocalizing neurological signs and symptoms, we propose that at least some PGAD cases arise from lesions affecting the sacral sensory networks that transmit sexual arousal—that it is a disorder of special sensation akin to neuropathic pain and itch. To reflect this, we propose congruent Greek-derived neurophysiologic nomenclature. For sexual arousal after nonsexual stimulation, we suggest "allodiegersis" (allo/άλλο) for "other" plus diegersis/διέγερσης (sexual arousal), analogous to "allodynia" for pain and "alloknesis" for itch. For spontaneous sexual arousal or orgasm without physical or mental stimulation, we propose "aftodiegersis" from aftomato/αυτομάτο (unprovoked) and diegersis/διέγερσης (sexual arousal). A genito-pelvic nosology has been developed,[19] but adding conventional neurological nomenclature could improve general medical awareness, care, and research.

Our findings will have clinical implications if confirmed because most PGAD patients now linger medically undiagnosed and untreated. Patient-initiated internet sites document thousands of (usually female) questioners. Therefore, this small series, although among the largest of examined patients, cannot fully represent PGAD nor provide accurate prevalences, causes, or treatment outcomes because of referral bias. However, it offers an interim clinical framework. It independently identifies sacral dorsal root Tarlov cysts as causal[2,11] and strengthens associations with

Table 1

Patient characteristics.

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6	Patient 7	Patient 8	Patient 9	Patient 10
Demographics	Caucasian female age 29.3 years	Arabic female age 35.9 years	Caucasian female age 36.8 years	Caucasian female age 42.1 years	Caucasian female age 50.0 years	Caucasian female age 58.4 years	Caucasian female age 59.0 years	Caucasian female age 60.9 years	Caucasian female age 71.8 years	Caucasian female age 79.8 years
Onset	At menarche (age 12), developed unprovoked arousals without orgasm	At 32, developed unprovoked arousals without orgasm	At 11, near menarche, developed unprovoked arousals without orgasm	At 30, developed unprovoked arousals without orgasm	At 46, developed unprovoked arousals without orgasm for 2 days, then with orgasm	At 50, developed unprovoked arousals without orgasm	At 58, developed continuous strong anorgasmic arousals for 2–3 months, then with orgasm	At 53, developed continuous low-level arousal without orgasm	At 69, developed unprovoked clitoral tingling and arousals, then orgasms early on	At 70, developed unprovoked arousals without orgasm
Temporal characteristics of PGAD episodes	Multiple (≤10) daily episodes of arousal for 5–40 minutes, 20% include orgasms that gave a few hours relief	Multiple (≤5) daily episodes that never caused orgasm	Multiple (≤6) daily episodes often including orgasm, worse in afternoon. Gradual lessening after adolescence	Multiple (≤30) daily brief episodes of almost immediate orgasm even during sleep	Multiple 2 hours episodes daily subsided after duloxetine was resumed at 60 mg then tapered; PGAD ended after 3-week taper, no known recurrence	Near-continuous arousal without orgasms, severe mechanical vulvodynia	40% 1–2 hours of strong arousal per day, 60% 1–2/day episodes of continuous mild or strong arousal, 1 spontaneous orgasm/mo while sleeping	Near-continuous waxing and waning arousal, 1 month-long remission, only 3 spontaneous orgasms	Initially once every few weeks, then more frequent until near-daily episodes	Near constant arousal worse as day progresses, one 3–4 week remission after clozapine stopped
Precipitants	Sitting, vibration travel (sitting + vibration)	Standing, lying flat, menses	Premenses and menses	None	Sitting, squeezing legs together	Sitting, vulvar contact including clothing gives sensation of vaginal distention	Sitting, travel (vibration + sitting) lying flat, twisting, stress, sleeping	Orgasms and genital herpes outbreaks, sudden discontinuation of sertraline	Daily onset at 4–5 AM, standing, walking	Valsalva, defecation
Ameliorants	Avoiding precipitants		Normal aging	None	Restarting duloxetine, cooling vulva, drinking wine	Wearing panty liners to prevent brushing against vulva, avoiding pants with seams	Avoiding some positions	None	Sitting, lying down	Showering
Effects of masturbation	Tried, not helpful	Tried, not helpful	Tried, used daily afternoon masturbation with several orgasms to dampen baseline arousal until next day	Tried, not helpful	Tried, not helpful	Tried, only brief refractory period, so not helpful	Tried, only brief refractory period, so not helpful	Tried, only brief refractory period, so not helpful and premature orgasms were unpleasant	Tried, not helpful	Tried, anhedonic masturbation 4–5× daily gave 1 hour of relief
Urologic symptoms	Neurogenic bladder since infancy, self-catheterizes	Frequency, urgency, mild hesitancy, sense of incomplete voiding	Hesitancy, some incontinence	None	Strong urgency during and between episodes	Urgency, frequency, urinary incontinence	None	Frequency (15× daily), occasional bladder pain labeled interstitial cystitis	None	Frequency
Other neurologic signs and symptoms	Sacral dimple at birth, neurogenic bladder, bowel since birth, uses cecostomy tube to void bowels, perineal sensory loss, pain radiating to upper inner thighs, rarely to toes	Age 23 neuropathic pain in left vulva, rectum, upper thigh, bilateral low back and leg pain, radicular sensory loss left vulva, buttock, thigh, chronic occipital headaches	Tachycardia, gastrointestinal dysmotility with nausea, vomiting, 50 lb weight loss, neurogenic constipation since childhood	None	Restless leg movements at night and while sleeping	Perineal and right buttock burning, itching, pain, severe pain with vaginal insertion	Low back, right leg pain and numbness, occasional clitoral pain, allodynia	Neuropathic pain, numbness, itch in hands and feet, postorgasm headache attributed to CSF leak from cervical TC, chronic fatigue, exertional intolerance, syncope, abnormal GI motility	Pelvic pain soon after onset at right T11-T12, mons-buzzing, poking feelings. Pin examination of right labium majus felt as not sharp, tingling, "sexy"	Bilateral paresthesias in thighs and legs, right leg cramps

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Table 1 (*continued*)

Patient characteristics.

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6	Patient 7	Patient 8	Patient 9	Patient 10
Obstetric-gynecology history	Nulliparous, severe pelvic pain worse on right, severe vaginal insertional pain, polycystic ovarian syndrome	Nulliparous, bilateral ovarian cysts	Nulliparous, extrauterine endometriomas and adhesions, managed hormonally, surgically	Nulliparous, menorrhagia-treated hormonally and surgically; previously anorgasmic despite attempts with stimulator	Three uncomplicated vaginal childbirths; no gynecological concerns	One pregnancy terminated, premenopausal menorrhagia	Two pregnancies, one preterm, one twin	One pregnancy; Cesarian delivery; endometriosis treated with lysis of adhesions; uterine fibroid; genital herpes since youth	Tubal ligation, breast biopsy, abdominal surgery for ovarian cyst and appendectomy	Two uncomplicated vaginal childbirths
Sexual sequelae	No sexual experience at PGAD onset, one failed intercourse attempt, then abstinent	No sexual experience at PGAD onset, remained abstinent	No sexual experience at PGAD onset, remained abstinent	Sexual relations with fiancé ended within a year, then abstinent	Remained abstinent until PGAD resolution	Sexual relations with husband ended; then abstinent	Sexual relations with husband ended; then abstinent	New near-immediate orgasm during sex, sexual relations with husband ended; then abstinent	Clitoral stimulation painful, sexual relations with husband ended; then abstinent	Sexual relations with husband ended; then abstinent
Psychiatric symptoms	Depression, anxiety	Depression	Depression, anxiety	Depression	Depression, anxiety	Depression, anxiety	Pre-existing depression	Depression	Depression	Depression, anxiety tardive dyskinesia, benzodiazepine dependence
Initial PGAD attribution	Attributed to STD despite no previous partners	Depression, stress, myofascial leg pain plus occipital neuralgia	Uncertain	Hypersexuality, seizures, endocrine dysfunction	Sudden duloxetine discontinuation	Sexologist-diagnosed PGAD, uncertain of cause	Prolonged sitting position during medical procedure	No explanation offered by gynecologist	Pudendal neuropathy	"Psychosexual mania" prompting 7 psychiatric hospitalizations
Lumbosacral MRI (Fig. 1)	Malfusion of S1 arch, L4-S1 facet arthropathy, L5-S1 HNP with mild left root compression	Sacral TC, 6.3 cm at S1, into pelvis compressing iliopsoas, bilateral S2 cysts with foraminal erosion; mild lumbar DJD	No full sacral MRI, lumbar noncontributory, mild lumbar DJD.	L5-S1 HNP with moderate foraminal stenosis, right L5 root contact	Not performed	Sacral TC; small right S1, large bilateral S2, S3 small S4	Noncontributory, sacral MRI with only mild sacroiliac DJD lumbar mild DJD, L45, L5-S1 facet arthropathy,	Lumbar TC, bilateral L5 1 cm on right, two 0.4-cm cysts on left	Sacral TC; 5-mm right S3, L4-L5, L5-S1 DJD, disc bulges, bilateral mild–moderate foraminal stenosis	Sacral TC; large bilateral S3, S4
Peripheral nerve testing	Urodynamics with outlet obstruction, postvoid residual, anorectal manometry with high resting anal pressure with outlet obstruction, low squeeze pressure, poor rectal sensation	Not performed	Abnormal lower leg skin biopsy (103 ENF/mm^2 skin surface area, at <1st centile of predicted), borderline composite autonomic function testing	Normal lower leg skin biopsy (398 ENF/mm^2 of skin surface area, at 84th centile of predicted)	Not performed	NCS with right-only slow pudendal nerve motor latency, EMG normal	Normal lower leg skin biopsy (278 ENF/mm^2 skin surface area, at 86th centile of predicted), EEG noncontributory	In 2015 had 28 ENF/mm^2 skin surface area; <1st centile of predicted, EMG/NCS with demyelinating + axonal motor sensory changes, normal AFT. 2018 skin biopsies diagnostic for SFN at distal leg and thigh, reduced sweat-gland innervation at distal leg, AFT diagnostic for SFN	Normal distal leg skin biopsy (235 ENF/mm^2 skin surface area, at 88th centile of predicted)	Not performed

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Table 1 (*continued*)

Patient characteristics.

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6	Patient 7	Patient 8	Patient 9	Patient 10
Other neurologic testing	Thoracic MRI with T11-12 HNP and mild cord signal changes	None performed	Cervical MRI with mild DJD, 56-panel whole exome sequencing with no pathogenic variants	3 normal EEGs including 1 during 4 spontaneous orgasms, brain MRI noncontributory	Brain MRI noncontributory	None	None performed	Cervical MRI with C6, C8 Tarlov cysts, multilevel DJD Brain MRI, PET noncontributory	None performed	Brain MRI, CTA normal; thoracic and lumbar MRI with mild DJD
Current PGAD attribution	Symptomatic sacral spina bifida occulta	Sacral radiculopathy from multiple Tarlov cysts	Small-fiber neuropathy, possible plexus irritation by endometriomas	L5S1 HNP with L5 radiculopathy	Sudden duloxetine discontinuation	Sacral radiculopathy from multiple Tarlov cysts	Unknown, sacral MRI recommended	Atypical CIDP with small-fiber involvement plus lumbar Tarlov cysts	Sacral radiculopathy from right S3 Tarlov cyst	Sacral radiculopathy from multiple Tarlov cysts
Ineffective symptom treatments	Pudendal nerve block, empiric anti-infectives for STDs, vaginal Botox injections to bladder neck, clitoris, pelvic floor, topical lidocaine	None offered	Nortriptyline offered but declined	Sex therapy, levetiracetam	Unknown	Pudendal nerve blocks, caudal epidural steroids, testosterone, pelvic PT, TENS, tibial nerve stimulator	Pudendal nerve blocks, epidural steroids, trigger point injections, gabapentin, topical amitriptyline, baclofen, local anesthetics	Unknown	Bilateral pudendal, genitofemoral, ilioinguinal, nerve blocks, gabapentin, pregabalin, sertraline, acupuncture, topical lidocaine, amitriptyline/ gabapentin/baclofen	Pudendal nerve blocks, TENS, electroconvulsive therapy ×17, citalopram leuprolide, escitalopram, amitriptyline, ovarian vein embolization
Effective symptom treatments	Bilateral genitofemoral nerve block gave 80% relief for 3 days only	Mild improvement with duloxetine, acetazolamide	Gradual improvement with aging	Mild improvement with risperidone	None	None	Mild help from gabapentin cream, genitofemoral nerve blocks gave few weeks of 85% relief	None	None	None
Definitive neurologic treatments	None tried	Complete remission after surgical Tarlov-cyst resection 1.5 years ago	None tried; trial of IVIg recommended but declined	None tried; surgical consultation for HNP decompression recommended	5 year total remission after duloxetine 60 mg resumed on day 3; tapered over 3 weeks	Resection of left and right S2 and right S3 Tarlov cysts was ineffective	None recommended or tried	IVIg reduced PGAD days from maximum of 30 days/month to 4 days/month and greatly improved other neuropathy symptoms eg, weakness	None tried; acetazolamide trial recommended	None tried; Tarlov-cyst resection recommended but declined

AFT, autonomic function testing; CIDP, chronic inflammatory demyelinating polyneuropathy; CSF, cerebrospinal fluid; DJD, degenerative disk disease; ECT, electroconvulsive therapy; EEG, electroencephalogram; EMG, electromyography; ENF, epidermal nerve fibers; GI, gastrointestinal; HNP, herniated nucleus pulposus; LS, lumbosacral; MRI, magnetic resonance imaging; NCS, nerve conduction study; PGAD, persistent genital arousal disorder; PT, physical therapy; TC, tarlov cyst; TENS, transcutaneous electrical nerve stimulation.

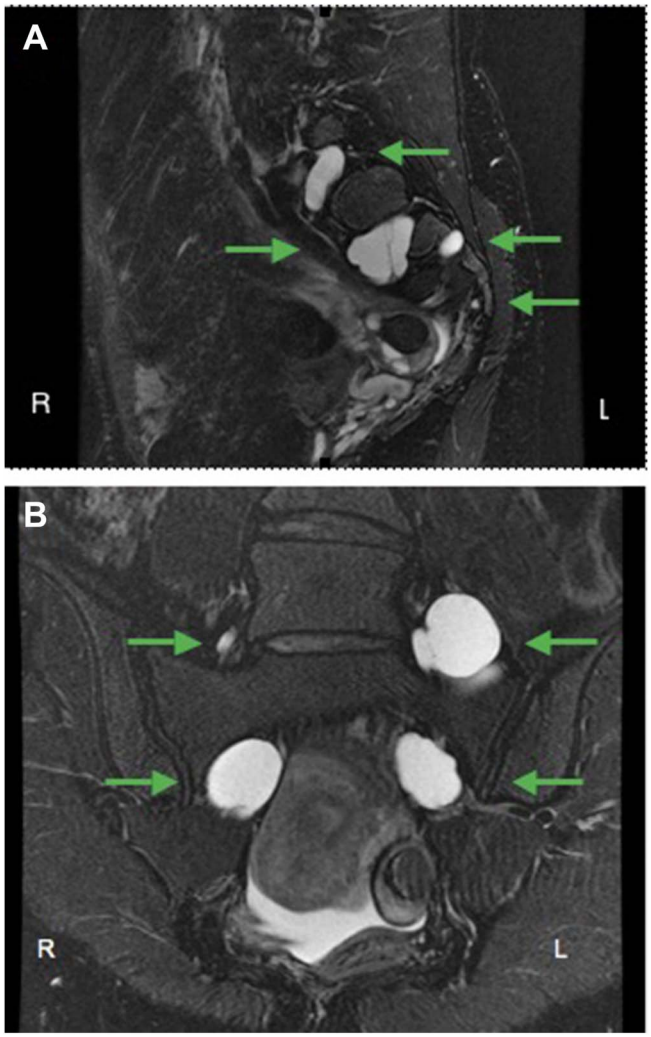

Figure 1. Magnetic resonance imaging identifying sacral Tarlov cysts (patient 1). (A) Sagittal view. Green arrows depict trilobed, multiseptate T2 hyperintense, T1 hypointense nonenhancing Tarlov perineurial cysts in the left L5-S1, left S1-S2, and left S2-S3 neural foramina (right S1-S2 cysts were also present and seen on other images). (B) Coronal view. Green arrows depict multiseptated T2 hyperintense, T1 hypointense perineurial cysts in the same patient. The largest cyst, at left L5-S1, tracks along the course of the L5 nerve root and measures 6 cm in greatest dimension.

sensory polyneuropathy. It adds another case associated with lumbosacral disc herniation and proposes cauda equina malformation and sensory CIDP as potential new causes. It associates PGAD with abrupt duloxetine withdrawal, given that duloxetine resumption was curative, extending reported associations beyond initiation of libido-promoting drugs (eg, dopaminergics) and abrupt discontinuation of libido-inhibitors (eg, serotonergic antidepressants).[5,6,8,22] It is unknown whether the synapses involved in drug-associated PGAD are central or peripheral. In patient 8, the fact that immunoglobulin treatment resolved not only motor CIDP symptoms but also reduced PGAD-symptomatic days from 30 to 4/month and stopped spontaneous orgasms suggests potentially broader associations between PGAD and sensory polyneuropathy, and potential effectiveness of standard neuropathy treatment for PGAD. Patient 4 developed isolated PGAD with only L5-S1 disc herniation identified. Given her L5 foraminal but not central stenosis, if this contributed, impingement of entering clitoral afferents was a more likely source than the conus medullaris.[16] Given the high prevalence of disc herniations, her PGAD may be unrelated, but the associations of PGAD with sacral radiculopathy from Tarlov cysts convey plausibility.

Female over-representation of published PGAD is high—we know of only 5 to 6 unique male cases (including 1 with L5-S1 disc herniation and 2 with small-fiber polyneuropathy).[8,10,21,24] Conceivably, male patients merely seek treatment less often, but we propose 3 biological contributors. A total of 90% to 95% of symptomatic Tarlov-cyst patients are women, because of their thinner meninges and tilted pelvis containing more-vertical nerve roots more exposed to CSF pressure waves. In addition, female patients represent 3/4 of many small-fiber neuropathy cohorts,[13] and 2/3 of US antidepressant users.[18] Female patients thus have a higher risk of associated neurologic conditions.

For lesion localization, 3-mm-cut sacral MRI and tests for neuropathy were highly useful. Pudendal nerve conduction should be measured more often, particularly with glove electrodes now standard. Brain MRI and EEG were futile.[23] Regarding treatment, skilled neurosurgeons report good outcomes for Tarlov-cyst resection.[2] Medical management of PGAD symptom should include gradual tapering of causal medications, and perhaps considering libido-dampening drugs. For nerve and nerve-root lesions, tricyclics, ion-channel blockers, and anti-epileptics—effective for neuropathic pain and itch—deserve consideration. Neurological evaluation and treatment should precede psychotherapy, electroconvulsive therapy, or clitoridectomy.[25]

Disclosures

The authors have no conflict of interest to declare.

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