A Novel Condition Impacting Female Physical and Mental Health: Research Status and Expert Consensus Interpretation of the Postprint on Persistent Genital Arousal Disorder/Genito-Pelvic Dysesthesia Syndrome

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Abstract

Persistent genital arousal disorder (PGAD) and genito-pelvic dysesthesia (GPD), collectively referred to as persistent genital arousal disorder/genito-pelvic dysesthesia syndrome (PGAD/GPD), constitute a recently identified condition characterized by spontaneous or secondary persistent states of sexual arousal and/or sensory abnormalities in the genitalia and other pelvic regions. This disorder predominantly affects women, and due to the distinctive nature of its symptoms, often exerts severe impacts on patients' physical and mental well-being, with the majority experiencing anxiety and depression, and some exhibiting suicidal tendencies. PGAD/GPD has attracted considerable attention from the international medical community and has been incorporated into the International Classification of Diseases-11 (ICD-11); however, domestic awareness of this newly recognized condition remains insufficient. Herein, we provide an overview of the current international research status and latest expert consensus, aiming to raise awareness among domestic colleagues regarding this disorder.

Full Text

A Newly Discovered Condition Affecting Women' s Physical and Mental Health: Current Research Status and Expert Consensus Interpretation of Persistent Genital Arousal Disorder/Genito-Pelvic Dysesthesia Syndrome

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Abstract

Persistent genital arousal disorder (PGAD) and genito-pelvic dysesthesia (GPD) are collectively known as persistent genital arousal disorder/genito-pelvic dysesthesia syndrome (PGAD/GPD). This recently identified condition is characterized by spontaneous or secondary persistent genital arousal and/or sensory abnormalities in the genital and pelvic regions. Primarily affecting women, its distinctive symptoms can seriously impact patients' physical and mental health, with most experiencing anxiety, depression, and even suicidal ideation. While PGAD/GPD has gained recognition in international medical communities and was included in the International Classification of Diseases-11 (ICD-11), awareness remains limited among healthcare providers in China. This review examines current international research and the latest expert consensus to raise clinical awareness of this condition.

Keywords: persistent genital arousal disorder/genito-pelvic dysesthesia; female physical and mental health; pathophysiological mechanisms; diagnosis and treatment; expert consensus

Introduction

In 2001, Leiblum and Nathan first proposed the diagnosis of persistent sexual arousal syndrome (PSAS) after analyzing five female patients with persistent sexual arousal symptoms [1]. Recognizing that "genital arousal" more accurately described the symptoms than "sexual arousal," Leiblum renamed the condition persistent genital arousal disorder (PGAD) in 2006 [2]. Subsequent international reports on PGAD gradually increased, though most consisted of case reports or case series with varying analyses of etiology, treatment approaches, and prognosis [3]. In 2016, the International Society for the Study of Women' s Sexual Health (ISSWSH) provided the first definition of PGAD and its associated risk

factors, describing it as "a distressing, persistent or recurrent condition of genital arousal or being on the verge of orgasm (genital dysesthesia) occurring in the absence of sexual interest, lasting for more than six months" [4].

In 2019, the ISSWSH expert panel reached a consensus [5] to rename PGAD as PGAD/GPD (Persistent Genital Arousal Disorder/Genito-Pelvic Dysesthesia). The new consensus defined PGAD/GPD as "a distressing, persistent or recurrent state of genital arousal (such as feeling on the verge of orgasm, genital lubrication or swelling, genital tingling, throbbing, and contractions) occurring spontaneously and lasting for more than three months, which may be accompanied by other genito-pelvic dysesthesias (such as burning, throbbing, itching, and pain)."

1 Epidemiological Research on PGAD/GPD

PGAD/GPD primarily affects women. Due to the unique nature of symptoms and privacy concerns, patients often experience shame or guilt that prevents them from disclosing their condition, while the medical community' s limited awareness further complicates understanding of its epidemiology. Current prevalence data derive from three main studies: Garvey and colleagues reported a 1% prevalence among women at a London sexual health clinic after surveying 96 patients [6]; Dettore et al. found a 1.62% prevalence among 679 Italian university students [7]; and the largest study by Robyn' s team analyzed two North American samples (n=1,634 and n=1,026), reporting a prevalence of 0.6-2.7% [8]. These studies share the limitation of small sample sizes and limited representativeness. Consequently, the ISSWSH expert panel provisionally estimated the prevalence of PGAD/GPD among women at approximately 0.6-3% [5].

2 Pathophysiological Mechanisms of PGAD/GPD

The pathophysiological mechanisms of PGAD/GPD remain unclear but are believed to involve a complex interplay of physiological, psychological, and social factors. Functional magnetic resonance imaging (fMRI) studies show that despite varying triggers, affected neural pathways converge on hyperexcitability of the paracentral lobule in the cerebral cortex [9]. Major contributing factors include:

2.1 Psychological Factors

Leiblum and Chivers proposed a psychological model in 2007 to explain PGAD/GPD development [2]. They suggested that negative cognitions about normal sexual response—such as avoidance, aversion, and fear (common in individuals who are highly introverted, conservative, or have experienced sexual assault or abuse)—increase anxiety and sympathetic nervous system activity, heightening genital arousal and focusing attention on these sensations, thereby creating a vicious cycle [10-14]. Subsequent studies indicate that many PGAD/GPD patients have pre-existing anxiety, depression, or neuroticism,

making these cognitive and behavioral abnormalities both predisposing factors and potential targets for psychological and pharmacological intervention [15-18].

2.2 Pharmacological Factors

Multiple case reports demonstrate that initiation or discontinuation of antidepressants, particularly selective serotonin reuptake inhibitors (SSRIs) and selective serotonin-norepinephrine reuptake inhibitors (SNRIs), can trigger PGAD/GPD [19-24]. The proposed mechanism involves disruption of the serotonergic "pain gating" system in the central nervous system. Serotonergic/noradrenergic pathways between the lower brainstem and spinal cord activate inhibitory interneurons to dampen pain signal transmission [25]; SSRIs/SNRIs may interfere with this pathway, enhancing ascending stimulation and triggering PGAD/GPD. Trazodone is another antidepressant associated with PGAD/GPD, causing abnormal genital engorgement (persistent penile/clitoral erection) through its alpha-1 adrenergic antagonism [26]. Trazodone may also enhance genital arousal by inhibiting histamine transmission and increasing dopamine-mediated signaling in the genito-pelvic sensory system. However, without randomized controlled studies or animal model validation, the mechanisms of drug-induced PGAD/GPD remain uncertain.

2.3 Neurological Factors

Increasing evidence shows that lesions anywhere along the sensory pathway—from nerve endings in pelvic floor organs to branches of the pudendal nerve, the main pudendal nerve trunk, and the cauda equina forming the sacral plexus (primarily S2-4 nerve roots)—can cause PGAD/GPD symptoms through nerve irritation or compression [5,27,28]. Komisaruk and Goldstein' s literature analysis identified sacral Tarlov cysts and cauda equina syndrome from disc herniation as important causes [29]. Feigenbaum and Boone reported significant symptom improvement in 10 of 11 PGAD/GPD patients after Tarlov cyst resection, confirming its pathogenic role [30]. Kim and colleagues validated the role of cauda equina syndrome from disc herniation through surgical treatment of PGAD/GPD secondary to sacral neuropathy [31]. Waldinger et al. suggested that peripheral pudendal nerve branch lesions can also cause PGAD/GPD [32]. Klifto and Dellon followed eight PGAD/GPD patients undergoing dorsal clitoral branch neurolysis, finding significant improvement in seven [33], and subsequently identified peripheral pudendal nerve compression as an important etiology [34-35]. Additionally, central nervous system lesions such as epileptic foci, arteriovenous malformations, demyelination, and tumors can trigger PGAD/GPD.

2.4 Traumatic Factors

Essentially a subset of neurological factors, various traumas involving the brain, spinal cord, lower back, and pelvic structures—including motor vehicle accidents,

falls, childbirth, and sports injuries—can theoretically trigger PGAD/GPD. Anamaria et al. reported a case of PGAD/GPD following a motor vehicle accident, attributing it to lumbar disc herniation compressing the cauda equina, with symptom resolution after treatment [36]. Some Tarlov cysts form after lumbosacral trauma in adolescence, representing another potential trigger [29].

2.5 Other Factors

Endocrine disorders [37] such as abnormal androgen, estradiol (E2), or thyroid hormone levels; pelvic floor muscle hypertonicity; pelvic arteriovenous malformations; sacroiliac joint dysfunction [38]; sacral foramen compression [39]; and idiopathic PGAD/GPD of unknown cause.

3 Diagnosis of PGAD/GPD

Early studies used Leiblum and Nathan' s 2001 diagnostic criteria [1], which underwent multiple revisions without substantial changes to core content. The 2019 ISSWSH expert consensus first summarized primary and accompanying symptoms, establishing five main symptoms as diagnostic criteria (Table 1).

3.1 Primary Symptoms

(1) Spontaneous, persistent or intermittent, distressing genital arousal lasting $ $3 months; (2) May be accompanied by other genito-pelvic dysesthesias (burning, throbbing, itching, pain); (3) Discomfort most commonly localizes to the clitoris but can occur elsewhere (mons pubis, vulva, vestibule, vagina, urethra, perineum, bladder, rectum); (4) May include feeling on the verge of orgasm, loss of orgasm control, and/or excessive orgasms; (5) Symptoms occur without any sexual thoughts [5].

3.2 Accompanying Symptoms

(1) Sexual activity may partially relieve, not relieve, or worsen symptoms; (2) Impaired orgasm quality (altered timing, intensity, frequency, inadequate or aversive orgasms); (3) Certain situations (sitting, driving, music/sounds, anxiety, stress) may exacerbate genito-pelvic dysesthesia; (4) May include emotional lability, depression, hopelessness, or suicidal ideation; (5) Physical examination may show no signs of genital arousal (lubrication, clitoral or labial swelling) [5].

3.3 Diagnostic Strategy

To accurately identify triggers for targeted treatment, ISSWSH experts proposed a "5-Zone" diagnostic strategy [5]. This divides the neural pathway from genital sensory receptors to cortical arousal centers into: (1) End-organ zone (somatic and visceral sensory nerve peripheral branches); (2) Pelvic/perineal zone (somatic and visceral sensory nerves); (3) Cauda equina zone (cauda equina

and dorsal root ganglia); (4) Spinal cord zone (spinothalamic and spinoreticular tracts); (5) Brain zone (intracerebral fibers to paracentral lobule). Diagnostic evaluation proceeds sequentially through zones 1-5 to localize lesions.

3.4 Auxiliary Examinations

Routine imaging such as ultrasound, plain radiographs, CT, and MRI have limited diagnostic value for PGAD/GPD itself but help identify etiologies: color Doppler ultrasound can detect pelvic vascular malformations; CT and MRI can identify disc herniation and intraspinal masses; MRI can reveal intracranial demyelinating lesions.

3.5 Specific Examinations

Based on accumulated experience, ISSWSH recommends specialized tests [5]: (1) Hormone level measurement (testosterone, free testosterone, sex hormone-binding globulin, estradiol [E2], thyroid hormones); (2) End-organ anesthesia and pudendal nerve block tests using topical infiltration or subcutaneous injection of clitoris, vestibule, urethra, bladder, and pudendal nerve—symptom relief confirms the zone as a trigger; (3) Neurological tests including genital quantitative sensory testing, non-genital sacral skin testing, bulbocavernosus reflex latency, pudendal nerve modulation, abdominal wall nerve block, and transforaminal epidural injection.

Table 1 ISSWSH Expert Consensus Description of Primary and Accompanying Symptoms in PGAD/GPD

Figure 1 [Figure 1: see original paper] ISSWSH Expert Consensus "5-Zone" Diagnostic Strategy and Corresponding Etiologies

4 Treatment of PGAD/GPD

No standardized treatment protocol currently exists; reported approaches are based on clinical experience with varying efficacy. ISSWSH proposes a biopsychosocial comprehensive management model addressing psychological-behavioral, social, cultural, neurological, vascular, and/or endocrine factors to prevent or alleviate symptoms [5,40]. Psychological intervention should be integrated throughout treatment, regardless of medication or surgical approaches.

4.1 Psychological Intervention

Cognitive-behavioral therapy (CBT), developed by A.T. Beck in the 1960s, is the primary psychosocial intervention targeting mental health conditions like depression and anxiety by modifying maladaptive cognitions [11]. For patients struggling to accept their diagnosis or experiencing suicidal ideation, mindfulness practices focusing on "acceptance" and "self-compassion" help develop a rational approach to managing PGAD/GPD [12].

4.2 Pharmacotherapy

Given the nascent understanding of PGAD/GPD, no specific medications exist; treatment relies on empirical drug use [5,24,41]. ISSWSH experts, analyzing case reports and expert opinions, suggest that rational use of existing medications can effectively treat PGAD/GPD [5]. Recommended approaches include: GABA receptor agonists and/or ion channel inhibitors (clonazepam, gabapentin, pregabalin, lamotrigine, oxcarbazepine, topiramate) for genito-pelvic arousal; varenicline for arousal with restless legs syndrome; opioid agonists (tramadol, hydrocodone) for genito-pelvic pain; SNRIs (duloxetine, paroxetine) or tricyclic antidepressants (nortriptyline, amitriptyline, clomipramine) for pain with mood symptoms; combined SNRIs and NSAIDs (duloxetine and ibuprofen) for genito-pelvic symptoms with low back pain; muscle relaxants (baclofen) and botulinum toxin type A for symptoms with pelvic floor dysfunction; and methimazole for symptoms with hyperthyroidism.

4.3 Physical Therapy

Common modalities include pelvic floor manual therapy and exercise, transcutaneous electrical nerve stimulation (TENS), transcranial magnetic stimulation (TMS), neuromodulation, and electroconvulsive therapy [42].

4.4 Surgical Treatment

Surgical intervention may be considered when conservative treatment fails. ISSWSH recommendations include [5]: consultation with neurosurgery for intracranial lesions (traumatic foci, epileptic foci, AVMs, aneurysms, tumors); orthopedic consultation for sacral Tarlov cysts, disc herniation, and other intraspinal lesions; orthopedic consultation for pudendal nerve compression requiring neurolysis; general surgery consultation for iliohypogastric, ilioinguinal, or genitofemoral nerve compression; vascular surgery consultation for pelvic varices or AVMs; and urology/gynecology consultation for external genital abnormalities (penile/clitoral adhesions, proliferative neuralgia).

5 Summary and Outlook

PGAD/GPD' s distinctive symptoms cause significant physical discomfort, psychological distress, and may lead to anxiety, depression, and suicide [43,44]. Epidemiological data suggest approximately 0.6-3% of women worldwide may be affected [5]. However, PGAD/GPD remains unfamiliar to most healthcare providers and the public, with limited awareness representing the greatest challenge to diagnosis and treatment. International organizations like ISSWSH have addressed this by including PGAD in ICD-11 under "other specific sexual arousal dysfunctions." The 2019 ISSWSH consensus clarified definitions and diagnostic criteria and introduced the "5-Zone" diagnostic model. The primary limitation is the scarcity of research data, with most evidence comprising case reports and

expert opinions lacking large-scale randomized controlled trials or animal model validation, limiting understanding of pathogenesis and treatment efficacy.

ISSWSH has identified future research directions: (1) large-scale epidemiological surveys across diverse cultures; (2) development of validated instruments for diagnosing subtypes and assessing outcomes; (3) longitudinal studies examining natural history and predictive factors; (4) neuroimaging studies exploring why pleasurable sexual sensations become distressing; (5) clinical and laboratory research on neurophysiological mechanisms and animal models; (6) investigation of fear-avoidance models and development of cost-effective, low-invasive treatments; (7) systematic evaluation of biopsychosocial treatment efficacy and safety; and (8) extension of research and treatment to diverse gender populations.

In Chinese culture, women tend to be more conservative about sexual issues, with shame and social stigma creating barriers to seeking care. Combined with clinicians' near-complete lack of awareness, underdiagnosis and misdiagnosis are likely common. We propose that proactive education (for healthcare providers and the public) combined with clinical research should guide China' s response: (1) professional training through lectures and case discussions; (2) public education to help patients seek appropriate care; and (3) designing clinical and laboratory studies based on international findings to further explore pathophysiology and treatment.

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References

[1] LEIBLUM S, NATHAN S G. Persistent sexual arousal syndrome: a newly discovered pattern of female sexuality [J]. J Sex Marital Ther, 2001, 27(4): 365-380. DOI: 10.1080/009262301317081115.

[2] LEIBLUM S, SEEHUUS M, BROWN C. Persistent genital arousal: disordered or normative aspect of female sexual response? [J]. J Sex Med, 2007, 4(3): 680-689. DOI: 10.1111/j.1743-6109.2007.00495.x.

[3] PFAUS J G. Persistent genital arousal disorder-fact or fiction? [J]. J Sex Med, 2017, 14(13): 318-319. DOI: 10.1016/j.jsxm.2017.01.

[4] PARISH S J, GOLDSTEIN A T, GOLDSTEIN S W, et al. Toward a more evidence-based nosology and nomenclature for female sexual dysfunctions–Part II [J]. J Sex Med, 2016, 13(12): 1888-1906. DOI: 10.1016/j.jsxm.2016.09.020.

[5] GOLDSTEIN I, KOMISARUK B R, PUKALL C F, et al. International society for the study of women' s sexual health (ISSWSH) review of epidemi-

ology and pathophysiology, and a consensus nomenclature and process of care for the management of persistent genital arousal disorder/genito-pelvic dysesthesia (PGAD/GPD) [J]. J Sex Med, 2021, 18(4): 665-697. DOI: 10.1016/j.jsxm.2021.01.172.

[6] GARVEY L J, WEST C, LATCH N, et al. Report of spontaneous and persistent genital arousal in women attending a sexual health clinic [J]. Int J STD AIDS, 2009, 20(8): 519-521. DOI: 10.1258/ijsa.2008.008492.

[7] DÉTTORE D, PAGNINI G. Persistent genital arousal disorder: a study on an Italian group of female university students [J]. J Sex Marital Ther, 2021, 47(1): 60-79. DOI: 10.1080/0092623X.2020.1804022.

[8] JACKOWICH R A, PUKALL C F. Prevalence of persistent genital arousal disorder in 2 north American samples [J]. J Sex Med, 2020, 17(12): 2408-2416. DOI: 10.1016/j.jsxm.2020.09.004.

[9] KOMISARUK B, WISE N, FRANGOS E, et al. Women' s clitoris, vagina, and cervix mapped on the sensory cortex: fMRI evidence [J]. J Sex Med, 2011, 8(10): 2822-2830. DOI: 10.1111/j.1743-6109.2011.02388.x.

[10] LEIBLUM S R, CHIVERS M L. Normal and persistent genital arousal in women: new perspectives [J]. J Sex Marital Ther, 2007, 33(4): 357-373. DOI: 10.1080/00926230701385605.

[11] CARVALHO J, VERISSIMO A, NOBRE P J. Psychological factors predicting the distress to female persistent genital arousal symptoms [J]. J Sex Marital Ther, 2015, 41(1): 11-24. DOI: 10.1080/0092623X.2013.869776.

[12] LEIBLUM S, SEEHUUS M, GOLDMEIER D, et al. Psychological, medical, and pharmacological correlates of persistent genital arousal disorder [J]. J Sex Med, 2007, 4(5): 1358-1366. DOI: 10.1111/j.1743-6109.2007.00575.x.

[13] LAMPE A, SOLDER E, ENNEMOSER A, et al. Chronic pelvic pain and previous sexual abuse [J]. Obstet Gynecol, 2000, 96(6): 929-933. DOI: 10.1016/s0029-7844(00)01072-3.

[14] PUKALL C F, GOLDSTEIN A T, BERGERSON S, et al. Vulvodynia: definition, prevalence, impact, and pathophysiological factors [J]. J Sex Med, 2016, 13(3): 291-304. DOI: 10.1016/j.jsxm.2015.12.021.

[15] CARVALHO J, VERISSIMO A, NOBRE P J. Cognitive and emotional determinants characterizing women with persistent genital arousal disorder [J]. J Sex Med, 2013, 10(6): 1549-1558. DOI: 10.1111/jsm.12122.

[16] JACKOWICH R A, POIRIER E, PUKALL C F. A comparison of medical comorbidities, psychosocial, and sexual well-being in an online cross-sectional sample of women experiencing persistent genital arousal symptoms and a control group [J]. J Sex Med, 2020, 17(1): 69-82. DOI: 10.1016/j.jsxm.2019.09.016.

[17] LEIBLUM S, BROWN C, WAN J, et al. Persistent sexual arousal syndrome:

a descriptive study [J]. J Sex Med, 2005, 2(3): 331-337. DOI: 10.1111/j.1743-6109.2005.20357.x.

[18] FACELLE T M, SADEGHI-NEJAD H, GOLDMEIER D. Persistent sexual arousal disorder: characterization, etiology, and management [J]. J Sex Med, 2013, 10(2): 439-450. DOI: 10.1111/j.1743-6109.2012.02990.x.

[19] LEIBLUM S R, GOLDMEIER D. Persistent genital arousal disorder in women: case reports of association with anti-depressant usage and withdrawal [J]. J Sex Marital Ther, 2008, 34(2): 150-159. DOI: 10.1080/00926230701636205.

[20] HARTMANN U H. Re: Persistent genital arousal disorder in women: case reports of association with anti-depressant usage and withdrawal [J]. Eur Urol, 2009, 55(5): 1233-1235. DOI: 10.1016/j.eururo.2009.01.054.

[21] JACKOWICH R, PINK L, GORDON A, et al. Symptom characteristics and medical history of an online sample of women who experience symptoms of persistent genital arousal [J]. J Sex Marital Ther, 2018, 44(2): 111-126. DOI: 10.1080/0092623X.2017.1321598.

[22] KRUGER T H C, SCHIPPERT C, MEYER B. The pharmacotherapy of persistent genital arousal disorder [J]. Curr Sex Heal Reports, 2020, 12(1): 34-39. DOI: 10.1007/s11930-020-00240-0.

[23] CALABRÓ R S. Lamotrigine-induced persistent genital arousal disorder: an unusual side effect [J]. Epilepsy Behav, 2017, 68(1): 234-235. DOI: 10.1016/j.yebeh.2017.01.012.

[24] MIYAKE K, TAKAKI M, SAKAMOTO S, et al. Restless genital syndrome induced by milnacipran [J]. Clin Neuropharmacol, 2018, 41(3): 109-110. DOI: 10.1097/WNF.0000000000000279.

[25] HEALY D, LE NOURY J, MANGIN D. Enduring sexual dysfunction after treatment with antidepressants, 5a-reductase inhibitors and isotretinoin: 300 cases [J]. Int J Risk Saf Med, 2018, 29(3): 125-134. DOI: 10.3233/JRS-180744.

[26] BATTAGLIA C, VENTUROLI S. Persistent genital arousal disorder and trazodone. Morphometric and vascular modifications of the clitoris. A case report [J]. J Sex Med, 2009, 6(10): 2896-2900. DOI: 10.1111/j.1743-6109.2009.01418.x.

[27] PEREIRA A, PÉREZ-MEDINA T, RODRÍGUEZ-TAPIA A, et al. Correlation between anatomical segments of the pudendal nerve and clinical findings of the patient with pudendal neuralgia [J]. Gynecol Obstet Invest, 2018, 83(6): 593-599. DOI: 10.1159/000489497.

[28] KOMISARUK B R, GOLDSTEIN I. Persistent genital arousal disorder: current conceptualizations and etiologic mechanisms [J]. Curr Sex Heal Reports, 2017, 9(4): 177-182.

[29] KOMISARUK B R, LEE H. Prevalence of sacral spinal (Tarlov) cysts in persistent genital arousal disorder [J]. J Sex Med, 2012, 9(8): 2047-2056. DOI: 10.1111/j.1743-6109.2012.02765.x.

[30] FEIGENBAUM F, BOONE K. Persistent genital arousal disorder caused by spinal meningeal cysts in the sacrum: successful neurosurgical treatment [J]. Obstet Gynecol, 2015, 126(4): 839-843. DOI: 10.1097/AOG.0000000000001060.

[31] KIM C, BLEVINS J, HANLEY J, et al. 019 neurogenic persistent genital arousal disorder (PGAD) secondary to radiculopathy of sacral spinal nerve roots (SSNR): treatment outcome following spine surgery [J]. J Sex Med, 2019, 16(6): S9. DOI: 10.1016/j.jsxm.2019.03.476.

[32] WALDINGER M D, SCHWEITER D H. Restless genital syndrome (ReGS) should be distinguished from spontaneous orgasms: a case report of cannabis-induced spontaneous orgasm [J]. J Sex Marital Ther, 2018, 44(3): 231-235. DOI: 10.1080/0092623X.2017.1377130.

[33] KLIFTO K, DELLON A L. Persistent genital arousal disorder: treatment by neurolysis of dorsal branch of pudendal nerve [J]. Microsurgery, 2019, 40(2): 160-166. DOI: 10.1002/micr.30464.

[34] KLIFTO K, DELLON A L. Persistent genital arousal disorder: review of pertinent peripheral nerves [J]. Sex Med Rev, 2020, 8(2): 265-273. DOI: 10.1016/j.sxmr.2019.10.001.

[35] OAKLANDER A L, SHARMA S, KESSLER K, et al. Persistent genital arousal disorder: a special sense neuropathy [J]. Pain Rep, 2020, 5(1): 801. DOI: 10.1097/PR9.0000000000000801.

[36] ANAMARIA P, MEENA K, JOHN M T. Persistent genital arousal disorder after motor vehicle accident: a case report [J]. Women' s Heal Rep, 2020, 1(1): 341-344. DOI: 10.1089/whr.2020.0043.

[37] TANNENBAUM J, GREENBERG J, RABEEM O, et al. Hormonal imbalances association to persistent genital arousal disorder in females [J]. J Sex Med, 2022, 19: S1-45.

[38] YUNXU Z, LI S, GE H, et al. Persistent Genital Arousal Disorder/Genito-Pelvic Dysesthesia (PGAD/GPD) caused by Sacroiliac Joint Dysfunction [J]. J Sex Med, 2022, 10(5): 53-70. DOI: 10.1016/j.esxm.2022.100544.

[39] GOLDSTEIN I, KOMISARUK B, YEE A, et al. Sacral foraminal (S2-3) radiculopathy syndrome (SFRS): Identification of a new PGAD/GPD trigger in the cauda equina [J]. J Sex Med, 2022, 19: S102-122.

[40] PEASE E R, ZIEGELMANN M, VENCILL J A, et al. Persistent genital arousal disorder (PGAD): a clinical review and case series in support of multidisciplinary management [J]. Sex Med Rev, 2022, 10(1): 53-70. DOI: 10.1016/j.sxmr.2021.05.001.

[41] KRUGER T H C. Can pharmacotherapy help persistent genital arousal disorder? [J]. Expert Opin Pharmacother, 2018, 19(15): 1705-1709. DOI: 10.1080/14656566.2018.1525359.

[42] MARTIN-VIVAR M, VILLENA-MOYA A, MESTRE-BACH G, et al. Treatments for persistent genital arousal disorder in women: a scoping review [J]. J Sex Med, 2022, 19(6): 961-974. DOI: 10.1016/j.jsxm.2022.03.220.

[43] COHEN A B, HELLSTROM W J, HODGES S J. Persistent genital arousal and major depressive disorder in an adolescent male: case report and discussion [J]. Urology, 2021, 157(1): 239-241. DOI: 10.1016/j.urology.2021.08.012.

[44] JACKOWICH R A, POIRIER E, PUKALL C F. A comparison of medical comorbidities, psychosocial, and sexual well-being in an online cross-sectional sample of women [J]. J Sex Med, 2020, 17(1): 69-82. DOI: 10.1016/j.jsxm.2019.09.016.

Note: Figure translations are in progress. See original paper for figures.