Persistent Genital Arousal Disorder

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INTRODUCTION:

Persistent Genital Arousal Disorder was only first described in 2001 and is often erroneously diagnosed as hypersexuality. PGAD requires careful history gathering and empathetic healthcare providers to correctly diagnose, due to the variable nature of the disorder.[1] PGAD is defined by unwarranted physiological sexual stimulation unaccompanied by psychological or subjective arousal.[2] This contradiction often preludes a great amount of patient distress, which may manifest in variable ways, including stress, anxiety, feelings of shame, and suicidal ideation.[3]

Research efforts since have worked to narrow the spectrum of this disease but have faced great difficulty due to a low case population, variable nature of the disorder, and no clear standard of treatment. Today still, the disease remains much of a mystery. Here we present the case study of a 47-year-old female afflicted with PGAD. Through this unique case study, with hallmark manifestations of the disorder, we seek to further elucidate the true nature of PGAD and standards of treatment.

CASE PRESENTATION:

Mrs. M is a married 47 year old female who presented to clinic due to crying spells, agitation, paranoia, and sexual arousals. Her past medical history was significant for depression and hypothyroidism, with the former treated ineffectively in the past with sertraline and hydroxyzine. The initial interview focused on symptomology and signs regarding affective, psychotic, and bipolar disorders. The patient appeared hesitant to discuss her sexual arousals, which were reported as unwanted orgasms with non-intimate physical stimulation, such as driving down the road, being touched on her head, or being hugged by her children; she was awaiting results of CT abdomen/pelvis and testosterone level, as ordered by her ob/gyn. At the initial encounter, she was found to meet criteria for Other Specified Depressive Disorder due to dysphoria, frequent crying spells, and decreased sleep; started on low-dose escitalopram; and referred for psychotherapy.

Upon further evaluation during psychotherapy, the patient endorsed previous sexual trauma, and this was explored additionally during her next medication management appointment. Her subjective history of irritability and frequent angry outbursts, difficulty trusting others and subsequent paranoia, and feelings of worthlessness appeared most consistent with a diagnosis of Post-Traumatic Stress Disorder (PTSD). The patient's unwanted sexual arousals were unchanged, and in the context of a diagnosis of PTSD, there was concern that the etiology could be due to her experiencing physiological reactivity after exposure to a triggering event that resembled some aspect of the trauma event, which can be seen in individuals with highly somatic presentations. She reported previously ordered CT abdomen/pelvis was unremarkable, and testosterone was elevated, with concern by her ob/gyn for genital hyper-stimulation, so spironolactone was initiated by her endocrinologist. To better target the patient's symptoms of PTSD and depression, escitalopram was titrated, and trazodone 50mg nightly as needed for insomnia was initiated.

While titration in escitalopram appeared to somewhat improve her affective symptoms, the patient's unwanted sexual arousals persisted, causing her extreme distress, especially in regard to not being able to hug her children without experiencing unwanted stimulation. Because of this, escitalopram 15mg daily was discontinued, and fluoxetine was initiated due to its known side effect of anorgasmia and to continue targeting affective symptoms.

After a literature review and further evaluations, the subjective history appeared to be most consistent with a diagnosis of Persistent Genital Arousal Disorder (PGAD), given that unwanted hyperarousal sensations and worsening of irritable moods started in the context of a stressful situation involving her children; the sensations were only intermittently relieved with orgasm; she experienced clitoral pain; and she experienced deep shame regarding her symptoms. Fluoxetine, which had been titrated to 40mg daily, was moderately efficacious for affective symptoms, but did not improve severity or frequency of sexual arousals, so duloxetine was initiated, and the patient was advised to follow-up with a neurologist due to concern for a multi-factorial etiology of PGAD, given her history of traumatic back injury and multiple pelvic surgeries; at this time, the patient has not undergone a neurological evaluation. After titrating duloxetine from 30mg daily to 60mg daily over the course of several months, the patient reported tremendous improvement in frequency and severity of hyperarousal to the point of no longer orgasming with non-intimate physical stimulation and resolution of clitoral pain; she denied functional impairment. In addition, her affective symptoms appeared well-controlled with this dosage of duloxetine.

DISCUSSION:

There is evidence that Persistent Genital Arousal Disorder has afflicted patient populations long before its conceptualization. Case accounts date as far back as second century AD. However, Goldmeier and Leiblum only fully defined the condition as PGAD in 2006.[4] The features they highlight detail six criteria for the diagnosis of PGAD. These include: 1. Symptoms characteristic of sexual arousal that persist and do not subside on their own. 2. Physiological arousal that does not resolve with standard orgasmic experiences. 3. Symptoms of arousal are unrelated to subjective feelings. 4. The persistent sexual arousal may be triggered by sexual arousal or innocuous stimuli. 5. Symptoms are experienced as intrusive and unwanted. 6. Symptoms correlate with a moderate to high degree of patient distress.

The etiology of PGAD remains controversial, with no discrete identifiable causes. Many variations in etiology exist among disease highlighting case reports. The core feature identifiable among cases is the increased sensory perception present in the central or peripheral nervous systems that leads to neuronal excitability or disinhibition.[5] There is also evidence to believe that patient perception regarding the disease, such as infringement on conservative beliefs or moral standards, may be involved in the etiology of the disease.[6] This core etiology elucidates itself in the variable pathophysiology of the disease, with central neurologic, peripheral neurologic, pharmacologic, vascular, dietary, and psychological pathologies. Epileptic foci, AV malformations, and strokes leading to epileptic central nervous system pathology have been correlated to genital arousal symptoms.[7] A correlation to other neurologic disorders, such as Restless Leg Syndrome and Overactive Bladder, have identified a peripheral neurologic branch of the disease that may represent the same pathology, but differ only in the area that is affected.[8] Compression of pelvic nerves, more commonly the Dorsal Branch of the Pudendal Nerve, Median Nerve, and the Sacral Nerve, by Tarlov cysts or pelvic varices, are common findings in PGAD patients.[9] There is a pharmacologic association of PGAD with initiation or withdrawal of SSRI and SNRI medications, including Venlafaxine, Paroxetine, Sertraline, Fluoxetine, and Trazadone, which may influence vascular vasodilation through ANP concentrations.[10] PGAD even has identifiable dietary pathologies, including a case of high soy ingestion that likely induced a hyperestrogenic state, and provocation is even seen with alcohol consumption. Ultimately, it is believed the psychological pathologies may play a major role in case severity as well as provocation of any of the above. The cognitive narrowing induced by focusing on perceived symptoms may contribute to a baseline level of anxiety that feeds a vicious cycle. A correlation to sexual abuse and other psychological pathologies such as major depression, panic attacks, and obsessive symptoms predate many cases of PGAD.[11]

Presentation of PGAD can be elusive and often requires a strong patient-provider relationship, as symptoms detailed and experienced by the patient are used for diagnosis. Onset of PGAD has been shown to be variable, ranging from after puberty onset to postmenopausal.[16] Patients often present with symptoms of hyperesthesia, from tight clothing or prolonged sitting, along the pudendal and ilioinguinal dermatomes. Patients often report experiencing sensations of perceived vasocongestion, wetness, tingling, throbbing, and/or pain, spontaneously or with vaginal penetration. The sensations are most often experienced in the clitoris, vagina, labia, or a combination. Stimuli of these sensations include intercourse, masturbation, psychological stress or anxiety, exacerbated by visual or innocuous stimuli. Most patients experience regular or occasional episodes, and those that meet PGAD diagnostic criteria often report continuous symptoms without relief.[4] Reported relieving factors include masturbation, orgasm, sexual intercourse, distraction, exercise, and cold compresses.[18] However, dysthesia or allodynia is reported with most episodes as levels of genital arousal in PGAD do not show a positive correlation with subjective feelings of arousal or increased desire. PGAD groups were also twice as likely to feel overwhelmed by symptoms, experience greater levels of pain, or lower levels of sexual satisfaction.[13] PGAD symptoms correlate with a high psychological impact, likely due to their intrusive nature. Patients with PGAD report high personal cost in mental health, sexual functioning, and ability to complete daily activities.[14] Self-reported high levels of worry (61.8%), stress (67.7%), and depression (42.7%) showed strongest correlations with PGAD diagnosis.[15]

Treatment of PGAD varies widely based on patient presentation and suspected etiology. This likely explains the sparsity in successful treatment plans. Treatment is focused on eliminating underlying causes of PGAD and often requires a multidisciplinary approach.[17] This approach necessitates thorough diagnostic evaluations to better determine the pathophysiology unique to the patient. Patient's experiencing symptoms of PGAD linked to a small fiber neuropathy have experienced partial or complete relief from PGAD symptoms through various treatments, such as transcutaneous electric nerve stimulation of the pudendal dermatome, bupivacaine block, electroconvulsive therapy, soft tissue mobilization over Alcock's canal, and even clitoridectomy.[3] Coil embolization of pelvic varices and surgical resection of tarlov cysts have been successful in multiple cases with vascular etiologies.[19] Various medications with analgesic effects have been shown to treat symptoms of PGAD, including benzodiazepines, selective serotonin reuptake inhibitors, antipsychotics, and antidepressants.[12] These medications aid with central neurologic gating and reducing levels of anxiety and stress, which are often major components of the disease. Other medications targeted at varying pathologies of PGAD include topiramate and carbamazepine as anti-epileptics as well as gabapentin and pregabalin as peripheral neuropathic agents.[20] Several published case reports detail great success in management of PGAD with Duloxetine, used to target the pathophysiology involving small fiber neuropathy of the pudendal nerve.[21] Treatment also includes cessation of any medications thought to precede symptoms of PGAD, as some medications have been correlated to the disease, such as Tramadol. Addressing midbrain emotions, stigmas, and false cognitions through cognitive behavioral therapy, couples therapy, or mindful meditation has also shown success as a treatment strategy.[22]

CONCLUSION:

The case presented demonstrates the true variable nature of PGAD. With variable etiology, pathology, and concurrent manifestations of the disease. The high physiological and psychological impact on these patients renders the need for increased successful treatment plans and awareness of the disease itself. Randomized control trials for PGAD are likely necessary to establish successful treatment plans. The varied etiology of PGAD requires tailored treatment plans and a flexible physician approach. Case evaluations such as these will likely aid in better treatment plans for those afflicted with PGAD and help to further the understanding and awareness of the disorder.

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