REVIEW

Persistent genital arousal in women – a new syndrome entity

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Summary: The persistent sexual arousal syndrome (PSAS) is a newly described entity where women become involuntarily aroused genitally for extended periods in time in the absence of sexual desire. Genital vasoengorgement and oedema have been observed. These women are found to be usually very distressed. The cause of the syndrome in the majority of cases is unknown, although a number of women report symptoms after withdrawal from selective serotonin reuptake inhibitors (SSRI) antidepressants. There is no specific therapy at present, although electroconvulsive therapy (ECT) has resulted in clinical improvement in cases where there was concomitant severe depression.

Keywords: persistent arousal, women

Sexual dysfunction and its management have received much attention over the last few years, in large part because there are now effective and safe treatments for men with erectile dysfunction. However, in terms of prevalence, female sexual dysfunction is more common than male sexual dysfunction. Low sexual desire and arousal account for a majority of complaints. Female arousal problems may be classified into those related to low or no mental (subjective) arousal, low or no genital arousal, or combined subjective and genital arousal problems.

Recently, a new clinical syndrome has been described, where the complaint is one of high levels of genital arousal occurring in the absence of subjective interest or arousal. Women with this clinical experience find the symptoms unwelcome since the genital arousal is usually persistent, unprovoked and unrelieved by orgasm. Indeed, women with persistent genital arousal report a high degree of psychological distress and even suicidal thoughts. A recent Internet survey suggests that women with persistent sexual arousal syndrome (PSAS) come from a wide variety of ages and backgrounds. When the genital area had been objectively viewed in a clinical setting, there was evidence of engorgement and oedema of the labia, vulva and clitoris. As yet, there is no clear idea of what the aetiology of the condition might be. Cases have been reported in association with ingestion of large amounts of phyto-oestrogens, local arteriovenous fistulas, and local nerve entrapment syndromes (Goldstein I, personal communications).

In a recent small study group consisting of physicians and of women with PSAS, several sufferers reported that they had been on selective serotonin reuptake inhibitors (SSRIs) in the past, and that their problems with unprovoked genital arousal began when these agents were discontinued. This has also been reported in the literature. The SSRI discontinuation syndrome indeed might be a clue as to one possible causation of PSAS. The syndrome may be particularly related to those SSRI agents with a short half-life, such as paroxetine. The discontinuation syndrome consists of physical and psychological features, which typically last a few days to a few weeks. However, there have been case reports showing the discontinuation syndrome can last for more than 18 months. It has been suggested this discontinuation syndrome is caused by 5-hydroxytryptamine (5-HT) synaptic downregulation. It is, therefore, possible that this neuronal involvement, whether at central, peripheral or autonomic level, has a direct effect on genital vasculature. It has also been suggested that serotonin receptor downregulation causes peripheral release of atrial natriuretic peptide (ANP). ANP is known to be a profound vasodilator as well as to be causing vascular leakage, making it a biologically plausible agent in the cause of the local symptoms and signs of PSAS.

At present, there is no agreed-upon or proven therapy for PSAS. Improvement on reintroduction of SSRIs has not been reported. Vasopressin might be helpful in that it causes vasoconstriction. Recently, a case was described where a woman with PSAS along with features of major depression received electroconvulsive therapy (ECT). This resulted in relief of the suicidal depression and,
unexpectedly, temporary relief from the PSAS symptoms. It has been speculated that ECT causes upregulation of a number of 5-HT receptors in cortical and limbic areas, in particular 5-HT 1, 2 and 3 receptors, as well as causing release of dopamine and noradrenaline.\textsuperscript{13} Research in primates suggests that these changes last 4-6 weeks after one episode of ECT.\textsuperscript{14} Interestingly, the patient with PSAS, who responded to ECT needed this treatment to be repeated every four weeks in order to remain asymptomatic.

Much remains to be elucidated in regard to PSAS, including its prevalence, aetiology and management. It deserves continued research not only since it is such a distressing and perplexing condition, but also because understanding its aetiology and treatment may lead to greater understanding of other aspects of female sexual response.

References
6 Sawa T, Goldmeier D, Richardson D. Persistent sexual arousal in a woman with an atrial septal defect— is increased atrial natriuretic peptide the cause. In: \textit{Poster 54 ISSWSH Conference}, Las Vegas, October 2005
10 Green B. Persistent adverse neurological effects following SSRI discontinuation (PANES) Psychiatry online [www.itascapsych.com/pharm_arch7.html]
12 Kellner CH, Patrides G, Fredrickse M, Yero S, Goldstein I. Relief of expressed suicidal intent by electroconvulsive therapy (ECT) in a woman with persistent sexual arousal syndrome (PSAS). ISSWSH Conference, Las Vegas Podium 15, October 2005
13 Ishihara K, Sasa M. Mechanism underlying the therapeutic effects of electroconvulsive therapy (ECT) on depression. \textit{Japan J Pharmacol} 1999;80:185-9
14 Strome EM, Clark CM, Zis AF, Doudet DJ. Electroconvulsive hock decreases binding to 5 HT2 receptors in non human primates: an in vivo positron emission tomography study with [18F] setoperone. \textit{Biol Psychiatry} 2005;57:1004-1010

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