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EARLY EJACULATION

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Abstract

Information on the epidemiology, etiology and treatment of premature ejaculation is reviewed. Evidence of the prevalence of premature ejaculation indicates that subjective concern about rapid ejaculation is a common concern worldwide. The hypotheses regarding the pathogenesis of premature ejaculation include: 1.) that it is a learned model of ejaculation maintained by interpersonal anxiety and 2.) that it is a dysfunctional result of the central or peripheral mechanisms that regulate ejaculatory thresholds and 3.) that it is a normal variant in latency ejaculation. Current evidence-based treatment interventions include behavioral psychotherapy and the use of pharmacological agents, including topical anesthetics and selective serotonin reuptake inhibitors.

The purpose of this paper is to review the existing knowledge base on the definition, prevalence, etiology and treatment of premature ejaculation.

Key words: sexual impulse, sexual behavior, ejaculation disorders, premature ejaculation sexual dysfunction, delayed ejaculation.

INTRODUCTION

The definition and treatment of premature ejaculation has evolved considerably in recent decades.

It was initially considered a learned behavior that could have been treated with behavioral therapy. Then, once it was recognized that serotonergic drugs could delay ejaculation, clinicians began to assume that physiological mechanisms rather than psychological ones could be primary in the etiology and maintenance of rapid ejaculation. To date, there is no definitive evidence on the etiology, and there is minimal evidence to dictate whether behavioral treatment or a combination of these should be used in treatment.

Premature ejaculation is considered one of the most common male sexual dysfunctions. Some doctors have even suggested that the term premature ejaculation involves pathology and that it should be replaced with the term rapid ejaculation, which is simply descriptive.

Officially accepted definitions are inaccurate. There is a lack of agreement on the operational definitions used in clinical research and there is also a lack of agreement on the threshold value of ejaculatory latency, which delimits a pathological condition of normality.

Masters and Johnson defined premature ejaculation as the man's inability to delay ejaculation long enough for his partner to reach orgasm in 50% of coital encounters. This definition has a major defect, namely, it is

conditioned by the partner's orgasm. Because, if a woman was anorgasmic, then her partner would have been diagnosed with premature ejaculation. Helen Singer Kaplan defined ejaculation as the absence of voluntary control over ejaculation. However, many men would not consider their ejaculatory latency to be under voluntary control. There are two official definitions. Manual of Diagnosis and Statistics of Mental Disorders (DSM V) and International Classification of Diseases and Related Health Problems (ICD – 10). DSM V defined premature ejaculation as persistent or recurrent ejaculation with minimal stimulation before or immediately after penetration and before the person desires it. ICD-10 has a definition that requires an inability to delay ejaculation, enough to enjoy sexual activity. Ejaculation should occur before or very soon after penetration.

DSM-V makes a distinction between lifelong and acquired premature ejaculation, as well as worldwide premature ejaculation and the premature situation. There is minimal evidence that such distinctions have clinical correlations or treatment implications. In general, it is assumed that global premature ejaculation may also show a constitutional predisposition to rapid ejaculation, while premature ejaculation in one sexual situation and not another would be more likely to be related to psychological problems. DSM also recommends distinguishing between organic and psychological etiologies.

Diagnostic criteria

According to DSM V, the diagnostic criteria refer to a persistent or recurrent pattern of ejaculation during sexual intercourse with a partner, approximately one minute after vaginal penetration and before the individual so desires. This symptom must be present for at least 6 months and manifest in all or almost all (75-100%) of sexual activities. It must cause clinical discomfort. It is also not better explained by a mental disorder without a sexual component or as a consequence of serious problems in the couple, or other stressors, nor can it be considered as a side effect when using certain substances or drugs. Depending on the severity of the symptom, it is: mild, moderate and severe.

The light one occurs 30 seconds to one minute after penetration. In the moderate one, ejaculation occurs 15-30 seconds later vaginal penetration and in severe ejaculation occurs either before, or at the beginning, or in the first 15 seconds after penetration.

Debut and evolution

Permanent premature ejaculation begins in the first sexual experiences and persists throughout life. But there are men who, even if they have problems at the beginning, start to control the duration of ejaculation and there is the other category in which men, after a normal ejaculation in the first period of life, develop this problem and then we talk about acquired premature ejaculation. There is less information about the acquired ejaculation compared to the permanent one, because the acquired ejaculation appears late, usually after the second decade of life, while the permanent one is more stable, appearing, as we mentioned in the first sexual experiences and is maintained on throughout life.

The elements of cultural or gender diagnosis can be imported into the diagnosis, because the latency time in ejaculation can be different in many cultures, taking into account both religion and genetic variations between populations. Also, the way in which modern society sees a woman's sexual activity has made people have different opinions regarding the latency of ejaculation, women becoming more concerned lately with the couple's sexual activity.

Diagnostic markers

The latency time of ejaculation is usually measured in the centers, by the sexual partner, by using a timing device, although this method is not suitable in real life. In the case of vaginal intercourse, the time from penetration to ejaculation is measured.

Differential diagnosis

If premature ejaculation occurs due to substance use, intoxication or discontinuation

of substance use, the diagnosis of sexual dysfunction induced by substance use or medication should be established.

Ejaculation disorders that do not meet the diagnostic criteria: Men who have normal latency time and who want to increase it and those who have episodic premature ejaculation (during the first sexual intercourse with a new partner, in which case short latency is normal and often encountered) should be identified. Neither of these should lead to the diagnosis of premature ejaculation, even if both situations bring discomfort to men.

Comorbidities

Premature ejaculation may be associated with erectile dysfunctions. It is difficult to establish who preceded the other. Permanent premature ejaculation may be associated with the anxious disorder, while acquired premature ejaculation may be associated with prostate, thyroid disease or substance use.

Epidemiology

The largest study conducted in America was the National Health and Social Survey and included a sample of 18-59 year olds. About 28% reported premature ejaculation problems.

It was the largest complaint reported, in the oldest study group, never married and with a minimum education. Another study conducted in the UK used anonymous questionnaires and the prevalence was 31%. There was a significant association of the presence of premature ejaculation and anxiety, measured on the scale of anxiety and depression. The global study of sexual attitudes and behaviors provided data on 27,500 subjects aged 40-80 in 29 countries, using a standard questionnaire, in-person or telephone interview.

Sampling methods from North America, Australia, South Africa and New Zealand consisted of telephone interviews chosen by numerical dialing. In this population, approximately 28% complained of rapid ejaculation. The prevalence was over 20% in Europe, Asia and South America, while in The Middle East was about 13%. It should be noted that none of these studies assessed ejaculatory latency or the level of interpersonal suffering.

DSM requires the presence of personal or interpersonal distraction to diagnose any sexual disorder. There may be large differences between the prevalence of stress in men compared to their partner. Haavio-Mannila and Kontula reported data on the survey of the population of Sweden, Finland, Estonia, St. Petersburg, Russia and the conclusion was that 2/3 of the men said that their partners have too long to reach orgasm. This is given that 18-20% of women complained of premature ejaculation of their partners, while only 2-3% of men reported having premature ejaculation problems. This proves once again that social change has raised expectations among women.

Given that there is no agreement on the definition, it is not surprising that there are different theories of etiology. These theories are related to different approaches to treatment and there is minimal evidence to support one theory over the others. They fall into two major classes: psychological and biological. The psychological ones fall into two groups based on psychodynamic theory and learning.

The psychodynamic ones they are rarely accepted by clinicians, making unconscious anger towards a partner a major etiological factor and this is because it is assumed that he may have unconscious sadistic feelings towards women, emotional immaturity, denying women's pleasure through this premature ejaculation. The treatment consists of individual psychotherapy. There is minimal evidence, both in support and in rejecting this theory. The most accepted hypothesis is that of Master and Johnson, namely, that it is a learned model of rapid ejaculation, maintained by anxiety. Anxiety regarding sexual insufficiency can interfere with a man's ability to monitor his arousal and ejaculation. This theory has a simplicity of common sense, although there is minimal evidence to support and reject it. Other clinicians argue that including relationship factors, such as the partner not encouraging, or even sabotaging her partner's learning control, and the situation in which the couple would need a "symptom" to draw attention to other

issues. This hypothesis is promoted by a small number of clinics. Laboratory studies have failed to demonstrate a difference between men with premature ejaculation and sexual arousal or sensory sensitivity. Although there is minimal evidence to support a relationship between the laboratory measures of performance anxiety and rapid ejaculation, there is some evidence to support the relationship between premature ejaculation and anxiety as a general trait or psychiatric disorder. Sexual function was examined in patients with panic disorder and social anxiety and it was found that 47% of patients with social phobia suffer from premature ejaculation. Studies that show a shorter latency of ejaculation in penetration activities than masturbating ones in men with premature ejaculation compared to men who have better ejaculation control could be interpreted as claiming that cognitive and biological factors play a role in genesis and maintenance of rapid ejaculation. Biological theories regarding the etiology can be separated into two major groups: those that emphasize peripheral or spinal mechanisms as opposed to those that emphasize brain mechanisms. It is possible that the two are correlated, because the mechanisms of the brain can influence peripheral ejaculatory thresholds and sensory sensitivity. The main theoretical formula for the brain mechanisms that underlie premature ejaculation throughout life was formulated by Dutch psychiatrist Waldinger. He argues that rapid ejaculation patterns are genetically determined and that men with hyposensitivity to the 5HT2c receptor and hypersensitivity to 5HT1a receptor, have ejaculatory thresholds set at a lower point.

Serotonin is a neurotransmitter with the function of a neuromodulator synthesized from the amino acid tryptophan. Tryptophan is an amino acid found in the proteins we take from our diet. Once in the body, proteins turn into 5-HTP, which in turn turns into serotonin. . 5-HT1 receptors have been implicated in producing the antidepressant effect of new antidepressant drugs, which selectively inhibit serotonin reuptake. 5-HT2 receptors are represented in the cortex, in the extrapyramidal system and have been involved in the mechanism of hallucinations, by some hallucinogenic substances, as well as in anxiety phenomena.

The hypothesis of different effects on ejaculation by stimulating serotonergic receptors is mainly based on data that serotonergic drugs that activate the 5HT2 receptor (eg paroxetine) delay ejaculation and that this can be reversed by drugs that stimulate the 5HT1 receptor (eg buspirone). Although this theory seems appealing, there is little evidence to support it. Waldinger reported a higher family incidence of premature ejaculation based on a small sample of men with fast ejaculation. Intrinsic to Waldinger's theory is that rapid ejaculation is a normal variation of ejaculatory speed and as such is not a psychiatric disorder. This is similar to the hypothesis that rapid ejaculation can probably have adaptive value. We can note that the hypothesis of the genetic difference in the ejaculatory threshold does not exclude the fact that men with a tendency to ejaculate quickly have the opportunity to learn ways to compensate for their hereditary tendencies. In conclusion, there is minimal evidence to support any of the current theories regarding the etiology of premature ejaculation throughout life. It would seem reasonable to assume that there are inherited differences in the ejaculatory threshold, so that the tendency to rapid ejaculation can be compensated to some extent by social learning and that interpersonal anxiety could interfere with this learning. Although, this statement is compatible with the available data, there is minimal evidence to support it.

There is isolated evidence of possible factors contributing to premature ejaculation. Several clinical series have reported a high prevalence of premature ejaculation in men with chronic prostate and there is a case report of normalization of ejaculatory time with prostate treatment. There are also reports regarding the high incidence in patients who suffered traumatic brain injuries, spinal cord injury, in men with diabetes, in hemodialysis patients. None of these studies had a comparison group.

Due to the fact that in the case of the studies, the sample was small and also the absence of a uniform definition, these findings should be considered only generators of hypotheses.

Treatment

Three major treatment approaches are known: behavioral therapy procedures, topical anesthetic ointments, and oral agents, especially those with serotonergic effects. All three approaches have been shown to be effective.

The technique of manual stimulation of the penis by the partner, which was stopped when he signaled orgasm can also be useful. Repeating this technique at least twice a week for 5-6 weeks while giving up intercourse reported an increase in ejaculatory control, probably because the man became more aware of the level of arousal. This technique was modified by adding brake squeezing, when the man signaled to his partner that ejaculation was imminent. Numerous clinicians have reported high success rates with these techniques, and better combined with couple therapy.

The use of local anesthetic creams and sprays has been reported to be effective in delaying ejaculation. The side effect is penis hypoanesthesia. Vaginal absorption may occur if a condom is not used.

Pharmacological treatments originate in the fact that some psychiatric drugs have been found to have a side effect, delayed ejaculation. For example, clomipramide, used in the treatment of obsessive-compulsive disorder in 1987, found that 96% of patients had an inability to ejaculate or severe delayed ejaculation. Studies have shown that chronic doses of clomipramide, paroxetine, sertraline, fluoxetine and citalopram delayed ejaculation in men with rapid ejaculation.

CONCLUSIONS

It is obvious that complaints about premature ejaculation are quite common, globally. Absence of an accepted and precise definition limits the conclusions we can draw on the epidemiology of this disorder.

There is evidence that men with premature ejaculation are more likely to take questionnaires that indicate anxiety and there is also evidence that may indicate certain changes in social norms that have made women have other expectations about sexual activity.

The available data suggest that both behavioral therapy and pharmacotherapy may be effective. Among the pharmacotherapeutic approaches, evidence supports the efficacy of antidepressants (paroxetine and clomipramide) and the use of topical anesthetics agents. There is no evidence as to when we should use psychotherapy as opposed to pharmacological products, or when they both should be used at the same time.

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