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ORIGINAL ARTICLE

Insight on pathogenesis of lifelong premature ejaculation: inverse relationship between lifelong premature ejaculation and obesity

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Although both biological and psychological factors are important in the etiology, the exact pathogenesis of lifelong premature ejaculation (PE) remains to be clarified. Obesity is a worldwide epidemic that contributes to many chronic diseases. Obesity is associated with erectile dysfunction, but the relationship between obesity and PE has not yet been specifically investigated. The aim of this study was to evaluate the relationships of these two conditions. Between January 2008 and December 2009, we evaluated consecutive patients with lifelong PE in the urology outpatient clinic. Control cases without lifelong PE were selected randomly among cases attending the department of internal medicine for a checkup procedure. The age and sex of control group were matched with that of the study group. Body mass index (BMI) of each case was calculated using the World Health Organization criteria by the measurements of the physician instead of relying on verbal expressions. The mean (±s.d.) age of the premature ejaculators was 31.7 ± 5.7 (range 21–51) years and in the control cases it was 32.3 ± 6.7 (range 22–54) years. The comparison of the mean (±s.d.) weight between the study (74.1 ± 11.2 kg) and control groups (81.9 ± 6.4 kg) revealed a significant difference ($P < 0.001$). The mean BMI of premature ejaculators (24.9 ± 3.4 kg m^{-2}) was lower than the mean BMI of control (27.5 ± 3.6 kg m^{-2}; $P < 0.001$). As the BMI increased, the number of patients decreased in the PE group. The number of the obese cases in the control group ($n = 26$, 24.1%) was three times greater than the obese premature ejaculators ($P < 0.005$), and the number of PE patients were approximately two times greater than the control cases in the normal-weight class ($P < 0.001$). This is the first prospective study that investigated the relationship between lifelong PE and obesity, and we found that patients with lifelong PE were leaner than the healthy control cases.
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Introduction

Obesity is a worldwide epidemic that contributes to many chronic diseases, and it will steadily increase in the near future due to substantial lifestyle changes.[1,2] It is a disorder characterized by an increased mass of adipose tissue. Especially, an increase in visceral adiposity is considered the key feature of metabolic abnormalities, known as metabolic syndrome. This syndrome includes glucose intolerance, insulin resistance, dyslipidaemia, hypertension, with an increased risk of cardiovascular disease and diabetes melitus.[3,4] Premature ejaculation (PE) is the most frequently reported bothersome male sexual problem.[5,6] The ISSM (International Society for Sexual Medicine) *Ad Hoc* Committee for the Definition of PE defines lifelong PE as a male sexual dysfunction characterized by:

- ejaculation that always or nearly always occurs before or within approximately 1 min of vaginal penetration;
- the inability to delay ejaculation on all or nearly all vaginal penetrations;
- negative personal consequences such as distress, bother, frustration and/or the avoidance of sexual intimacy.[7]

Premature ejaculation can be classified as either a lifelong condition (present since the onset of sexual maturity) or an acquired condition that develops after an interval of normal sexual function.[8] Although both biological and psychological factors

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are important in the etiology, the underlying physiopathology of this condition is not well understood. Prevalence of PE is higher than erectile dysfunction, ranging from 9 to 31% in the general male population.[7] Obesity and the MS are associated with erectile dysfunction.[9,10] However, the relationship between obesity and PE has not yet been specifically investigated. The aim of this study was to investigate the relationships of these two conditions.

Materials and methods

Between January 2008 and December 2009, we evaluated consecutive patients with lifelong PE in the urology outpatient clinic. Age- and sex-matched control cases without lifelong PE were selected randomly among cases attending the Department of Internal Medicine for a checkup procedure. If the patient declared that he ejaculated in <1 min during more than half of his intercourse attempts, he was enrolled in the study. In all, 104 premature ejaculators and a cohort of 108 control cases were enrolled in the study. All participants provided informed consent according to the Helsinki guidelines. Clinical research ethics committee of Mustafa Kemal University approved this study. The following criteria were used for the inclusion of subjects in this study:

The subjects were eligible if they:

- were between the ages of 18 and 60;
- were in a stable, monogamous, heterosexual relationship with the same, nonpregnant, sexually active partner for at least 6 months;
- had the complaint of PE since their first sexual encounters and were seeking medical treatment for that;
- were not suffering from any sexual disorders other than lifelong PE;
- did not have a history of any significant psychiatric disorder and/or drug treatment;
- did not complain of any organic cause of PE (evaluated by urinalysis, biochemical, hematological and endocrine testing), including anatomical abnormalities;
- did not have any other malignancy or chronic diseases that might interfere with mental and sexual health;
- did not have previous urogenital surgery (except cystoscopy);
- had a normal IIEF (International Index of Erectile Function) erectile function domain score (IIEF-EF $\geqslant 26$).

Subjects with devastating illnesses, including type 1 diabetes mellitus, malignancies, acute and chronic renal failure, chronic liver diseases, hyper- or hypothyroidism and heart failure, were excluded to avoid their possible effects on weight. Body mass index (BMI) of each case was calculated using the World Health Organization criteria by the measurements of the physician instead of relying on verbal expressions.[11] Weight in kg was divided by height in meters squared, and normal weight was considered as $\leqslant 24.9$, overweight as 25–29.9 and obesity as a BMI of $\geqslant 30.0\,kg\,m^{-2}$. All patients and subjects in the control group who met the inclusion criteria had undergone the same physical examination.

A commercially available statistics software package (SPSS for Windows v. 12.0, Chicago, IL, USA) was used to perform all statistical calculations. Student's *t*-test was used to compare mean values. The χ^2 test was used to compare the frequency of different BMI classes in the study and the control group.

Results

The somatometric parameters of the premature ejaculators and the control group are shown in Table 1.

The mean ($\pm$s.d.) age of the premature ejaculators was 31.7 ± 5.7 (range 21–51) years and in the control cases it was 32.3 ± 6.7 (range 22–54) years. A statistical comparison showed that the patients in the study group were compared with the age-matched controls. There was no difference between groups regarding marital status, household income or education level. The mean ($\pm$s.d.) height of the patients in the study group was 1.72 m (± 0.1) and 1.73 m (± 0.1) for subjects in control groups ($P > 0.05$). The comparison of the mean ($\pm$s.d.) weight between the study (74.1 ± 11.2 kg) and the control groups (81.9 ± 6.4 kg) revealed a significant difference ($P < 0.001$). The mean BMI of premature ejaculators ($24.9 \pm 3.4\,kg\,m^{-2}$) was lower than the mean BMI of the control group ($27.5 \pm 3.6\,kg/m^{-2}$; $P < 0.001$).

The BMI classes of subjects are presented in Table 2. The number of the obese cases in the control group and in the PE group was 26 (24.1%) and 9 (8.7%), respectively ($P < 0.005$). The number of the cases with normal weight in the PE group and in the control group was 59 (56.7%) and 33 (30.5%),

Table 1 The somatometric parameters in the premature ejaculators and the control cases

	Premature ejaculators (n = *104*)	*Controls* (n = *108*)	P-*value*[a]
Age (years)	31.7 ± 5.7	32.3 ± 6.7	>0.05
Height (m)	1.72 ± 0.07	1.73 ± 0.06	>0.05
Weight (kg)	74.1 ± 11.2	81.9 ± 6.4	<0.001
Body mass index ($kg\,m^{-2}$)	24.9 ± 3.4	27.5 ± 3.6	<0.001

[a]Student's *t*-test. The values are presented as mean $\pm$ s.d.

Table 2 The body mass index classes of the premature ejaculators and the control cases

	Premature ejaculators, n *(%)*	*Controls* n *(%)*	P-*value*[a]
Normal	59 (56.7)	33 (30.5)	<0.001
Overweight	36 (34.6)	49 (45.4)	>0.05
Obese	9 (8.7)	26 (24.1)	<0.005
Overweight + obese	45 (43.3)	75 (69.5)	<0.001

[a]The χ^2 test.

respectively ($P<0.001$). When we compared the two groups according to the non-normal (overweight + obese) class of BMI, there were 45 (43.3%) and 75 (69.5%) cases in the PE group and the control group, respectively. The ratio of the overweight and obese patients in the control group was significantly higher than that of the study group ($P<0.001$).

Discussion

We performed a prospective study to determine a possible relationship of lifelong PE with obesity in adult males. For this purpose, we compared the somatometric parameters of premature ejaculators with those of the age-matched controls. We found that the premature ejaculators were leaner than the healthy control cases, and as the BMI increased in the PE group, the number of patients decreased. We also showed in this study that the prevalence of obesity was lower in the PE group than in the control group.

Although the relationship between obesity and impaired sexual health has been known for at least two decades, in this study we investigated whether there was a possible specific association between lifelong PE and obesity. To our knowledge, this is the first prospective study regarding the relationship between obesity and lifelong PE.

Ejaculation is defined as the expulsion of seminal fluid through the urethra and is closely associated with orgasm, extragenital responses and subjective pleasurable feelings in men.[12] Although central and spinal control of ejaculation as the neurotransmitter network involved in the process is available, but the dynamic of this physiological process is not yet fully understood. The reasons for the association between obesity and lifelong PE are not easy to explain. Nevertheless, some hypotheses can be drawn.

Testosterone may have a facilitatory role by both central and peripheral mechanisms in the control of ejaculatory reflex.[13] Keleta *et al.*[14] showed that the chronic treatment with T in rats significantly decreased serotonin (5-hydroxytryptamine (5-HT)) in brain. Serotonin is the most important central monoamine neurotransmitter that is involved in delaying ejaculation. Decreased total and, often, free T levels, decreased gonadotropin levels and increased circulating estrogen levels (which is described as hypogonadotropic hyperestrogenic hypoandrogenemia) are specific to the obese men.[15] The decrease in androgen levels is proportional to the degree of obesity.[16,17] The results of our study are in line with all this evidence because the prevalence of obesity was lower in the PE group than the control group.

The pathophysiology of lifelong PE is unknown in all aspects, although disturbance in the central 5-HT pathway has been advocated by Waldinger[18] as a possible cause. In the central 5-HT pathways the 5-HT_{1A} receptors decrease erection and latency to ejaculation in male rats,[19,20] whereas the 5-HT_{2C} receptors increase both erection and latency of ejaculation.[20,21] Studies investigating the relationship between 5-HT and sexual hormones have shown that oestradiol was reported to induce a desensitization of the 5-HT_{1A} receptors.[22,23] Both estrone and oestradiol are increased in obese men compared with control subjects, a finding attributed to increased peripheral aromatization of androgens.[24] In our study, we found that premature ejaculators were leaner than healthy controls. In light of these scientific data, our results can be interpreted by increased oestradiol levels that lead to increased ejaculation latency time through desensitization of the 5-HT_{1A} receptors in obese men.

We have evaluated the patients and the control cases who have been questioned about lifelong PE. Although the Turkish validation of premature ejaculation diagnostic tool[25] is available nowadays, distress, bother or interpersonal difficulty, which are other components of PE, had been evaluated without using any questionnaire, and this is a limitation of our study. The lack of the measurement of intravaginal ejaculation latency time, and the lack of the measurement of total body fat and sexual hormones are other limitations of this study. Studies with measurements that are mentioned above on larger numbers of participants might evaluate the relation between the lifelong PE and obesity more precisely.

In conclusion, we found that patients with lifelong PE were leaner than the healthy control cases and the number of the patients decreased as their BMI increased. In addition, the prevalence of obesity was lower in the PE group than in the control group. This is the first prospective study that investigated the relationship between lifelong PE and obesity. Although we know that our study may not directly affect patient care, these findings may be useful in understanding the physiopathology of lifelong PE.

Conflict of interest

The authors declare no conflict of interest.

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