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Risk of obesity on adult male fertility in Gaza Strip, the Palestinian Authority

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Abstract

Background/aim: Globesity and/ or fertility are intricate multifactorial phenomena, environmentally, genetically, physiologically and behaviorally determined, thus an interdisciplinary approach is required to understand. This study assessed the risk of obesity on Palestinian male fertility in terms of reproductive hormonal profile and semen parameters, and their associations with body mass index (BMI).

Materials and methods: This study was a case-control design and comprised 80 obese and 80 non-obese adult males matched for age. The BMI was measured. Lipid profile, testosterone, gonadotropins and semen parameters were determined.

Results: Significant increase in the BMI of obese males compared to non-obese male controls (33.4 ± 2.5 vs. 22.8 ± 1.5 kg/m^2, P<0.001). Cholesterol, triglycerides and low-density lipoprotein cholesterol (LDL-C) were also significantly increased in obese males (P<0.001). Testosterone showed significant decrease in obese males than non-obese group (3.6 ± 1.5 vs. 6.3 ± 0.8 ng/ml, P<0.001) whereas luteinizing hormone (LH) and follicle stimulating hormone (FSH) showed significant increase in obese males (9.7 ± 5.3 and 12.3 ± 6.4 vs. 4.7 ± 2.6 and 4.6 ± 1.3 mIU/ml, P<0.001, respectively). Sperm concentration, motility and normal form were significantly lower in obese males compared to non-obese controls whereas immotile and abnormal sperm form were significantly higher in obese males. The BMI showed significant positive correlation with cholesterol, triglycerides, LDL-C, LH, FSH, immotile and abnormal sperm form, while significant negative correlation was found with testosterone, sperm concentration, motility and normal form.

Conclusion: The relationship of obesity with lipid profile, reproductive hormones and semen parameters suggests that improvement of fertility may be targeted throughout weight-loss health programs for obese males.

Introduction

Obesity is a condition of abnormal or excess fat accumulation in adipose tissue, to the extent that health may be impaired [1]. Many diseases are associated with obesity that include cardiovascular diseases, diabetes mellitus and malignancies [2-4]. Obesity is not a single disorder but a heterogeneous group of conditions with multiple etiologies of an intricate interplay of environmental and genetic factors each of which is ultimately expressed as obese phenotype [5]. The BMI is a simple index that commonly used to classify underweight, overweight and obese in adults. It is defined as the weight in kilograms divided by the square of the height in meters [6]. An adult person with a BMI of 18.5-24.9 kg/m^2 is considered by World Health Organization (WHO) to have a normal weight, while obesity is defined as having a BMI of 30 kg/m^2 or more [7].

The epidemiology of obesity has been reached alarming levels, affecting virtually both developed and developing countries. In the majority of European countries, the prevalence of obesity was increased from 10% to 40% in the last decade, and specifically in England, it increased more than three-folds [8]. In USA, the prevalence of obesity in adults was 42.4% in 2017-2018 [9]. The Arab countries showed adult obesity trends of 23.0% to 46.0% [10,11]. In the Palestinian territories including Gaza Strip, the prevalence of obesity in adults ranged from 18% to 30% [12].

Fertility is the natural capability to produce offspring. As a measure, the total fertility rate identifies the average number of children per woman if a group of women were to experience the age-specific fertility rates for a given year [13]. The primary reproductive organs of the male are the testes, which have both an exocrine (sperm producing) and an endocrine (testosterone producing) functions [14]. Impeccable coordination of the hypothalamic-pituitary-gonadal axis is required for normal testicular function. Pulsatile secretion of gonadotropin-releasing hormone (GnRH) by the hypothalamus stimulates the biosynthesis of pituitary gonadotropins, LH and FSH that, in turn, sustain intragonadal testosterone production and spermatogenesis [15].

Over the last two decades, obesity is linked to human fertility. Although a growing body of research has indicated the potential role of obesity in the deterioration of male fertility, yet the underlying mechanisms of obesity-induced male infertility have not been

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fully understood. However, obesity may act through alteration in the hypothalamic-pituitary-gonadal axis and its cross talks with other hormones, thus impairing the intricate orchestration of prime endocrine regulators (LH and FSH) of male reproduction [16]. Importantly, obesity and its underlying mediators result in a negative impact on semen parameters, including sperm concentration, motility, viability and normal morphology [17]. In addition, obesity is combined with general metabolic derangements including lipid profile abnormalities that affect negatively on testicular function [18].

In Gaza Strip, most of the published research on obesity is limited and has been focused on epidemiological surveys and few related biochemical aspects. To the best of our knowledge, no previous study linked obesity with adult male fertility. Therefore, this study is the first to assess the risk of obesity on male fertility in terms of hormonal profile and semen quantity and quality, and their associations to obesity in Gaza Strip, Palestine.

Materials and methods

Study population and experimental design

This study used a case-control design. The study population included 160 individuals based on evaluation of obesity among adult males aged 20-40 years old in Gaza Strip. Cases comprised 80 obese males with BMI ≥ 30 kg/m^2 and equal number non-obese males with BMI 18.5-24.9 kg/m^2 apparently healthy individuals as a control group. Cases and controls were age matched. Smoker men and men with diabetes mellitus, testicular disease or testicular surgery and family history of infertility were excluded.

Sampling and sample size

The case group was married obese men visiting obesity clinics in Gaza Strip seeking for weight control and the control group included married normal weight men. The sample size calculations were based on the formula for case-control studies. EPI-INFO statistical package version 3.5.1 was used with 95% CI, 80% power and 50% proportion as conservative and OR > 2. The sample size in case of 1:1 ratio of case control was found to be 71:71. For a no-response expectation, the sample size was increased to 80 obese men. The control group was also consisted of 80 non-obese men.

Ethical consideration

The necessary approval to conduct this study was obtained from Helsinki committee for ethical approval number PHRC/HC/27/12 in the Gaza Strip and all study participants signed an informed consent form.

Questionnaire interview

A face to face interview was used for filling in a mini questionnaire. The questions were one of two types: the yes/no question, which offers a dichotomous choice; and the multiple choice question, which offers several fixed alternatives [19]. The questionnaire was validated and piloted, and included questions on age, have children, education, employment and family income/month.

Body mass index

The body weight and height of each individual dressed in light clothing without shoes were measured using a carefully calibrated balance (Detecto, CAP-180 Kg, USA) for weight and a vertical measuring rod for height, and the BMI was calculated as Kilogram (kg) body mass/height in meter squared [7].

Blood sampling and processing

Twelve hours overnight fasting venous blood samples were collected in vacutainer tubes. Blood was left for a while without anticoagulant to allow blood to clot. Then, serum samples were obtained by centrifugation at 3000 rpm for 10 minutes using a Rotina 46 Hettich Centrifuge, Japan.

Biochemical analysis

Serum cholesterol and triglycerides were measured by the cholesterol oxidase/POD method and by the glycerol phosphate oxidase/POD method, respectively, using the BioSystems kit, Spain [20,21]. High-density lipoprotein cholesterol was determined by the precipitating method using Labkit kit, Spain [22]. Low-density lipoprotein cholesterol was calculated using the empirical relationship of Friedewald [23]. Serum testosterone, LH and FSH were determined according to the methods of Tietz, Lenton *et al.* and Vitt *et al.* [24-26], respectively using enzyme-linked immunosorbent assay (ELISA) TECO kits.

Semen collection and analysis

Semen samples were collected by masturbation in sterile polypropylene containers after sexual abstinence of 2-7 days. Semen volume was measured. Routine semen analysis was carried out by light microscopy. The concentration, motility and morphology of spermatozoa were assessed according to WHO criteria [27].

Statistical analysis

Data were analyzed using the IBM SPSS software version 23.0 for windows (Statistical Package for the Social Sciences Inc, Chicago, Illinois). A Simple distribution of the study variables and cross tabulation was applied. Chi-square (χ^2) was used to identify the difference between variables. Yates's continuity correction test, $\chi^2_{(corrected)}$, was used when not more than 20% of the cells had an expected frequency of less than five and when the expected numbers were small. The independent sample t-test procedure was used to compare means of quantitative variables by the separated cases into two qualitative groups such as the relationship between testosterone levels of non-obese and obese males. Pearson's correlation test was applied. The results were accepted as statistically significant when P<0.05. The percentage difference was calculated according to the formula: Percentage difference equals the absolute value of the change in value, divided by the average of the 2 numbers, all multiplied by 100.

Percentage difference=(|(V1 - V2)|/((V1+V2)/2))×100.

Results

Socio-demographic aspects and anthropometric measurements

Table 1 showed no significant differences between obese and non-obese males in terms of age and education (P > 0.05). However, significant differences were found between obese and non-obese males, with less number of children ($\chi^2_{(corrected)}$ test=4.329, P=0.037), lower number of employee (χ^2=5.479, P=0.019) and less family income/month (χ^2=20.486, P=<0.001) among obese group. As indicated in Table 2, BMI in obese male was significantly higher compared to non-obese males (33.4 ± 2.5 vs. 22.8 ± 1.5 kg/m^2, % difference=37.7, P<0.001).

Biochemical parameters

Lipid profile of obese compared to non-obese males is illustrated in Table 3. The mean levels of cholesterol (212.6 ± 42.4 vs. 127.9 ± 38.4

mg/dl), triglycerides (230.9 ± 30.7 vs. 113.8 ± 35.6 mg/dl) and LDL-C (121.7 ± 32.8 vs. 93.9 ± 22.9 mg/dl) were significantly higher in obese males compared to non-obese controls (P<0.001), with % differences of 49.8, 67.9 and 25.8, respectively. On the other hand, no significant difference was found in HDL-C among obese and non-obese males. Hormonal analysis presented in Table 4 revealed significant decrease in the mean level of testosterone in obese males than non-obese group (3.6 ± 1.5 vs. 6.3 ± 0.8 ng/ml, % difference=54.5 and P<0.001). Conversely, the mean levels of LH and FSH were significantly increased in obese males (9.7 ± 5.3 and 12.3 ± 6.4 vs. 4.7 ± 2.6 and 4.6 ± 1.3 mIU/ml, % differnces=69.4 and 91.1, P<0.001, respectively).

Semen parameters

Table 5 summarizes semen parameters in obese and non-obese males. Sperm concentration including sperm volume (2.2 ± 0.9 vs. 3.1 ± 0.6 ml), sperm count (7.8 ± 5.2 vs. 43.7 ± 16.0 million/ml) and total sperm count (17.6 ± 21.6 vs. 135.3 ± 55.8 million) were significantly lower in obese males compared to non-obese controls (P<0.001), with % differences 34.0, 139.5 and 15.4, respectively). Sperm motility showed significant decrease in total motility (28.7 ± 14.8 vs. 57.7 ± 11.2%, P<0.001), Fast progressive (0.23 ± 1.27 vs. 18.9 ± 5.4%, P<0.001) and slow progressive (18.7 ± 11.6 vs. 28.7 ± 7.5%, P<0.001) in obese males than non-obese controls, with % differences of 67.0, 195.3 and

Table 1. Socio-demographic aspects of the study population

Character	Non-obese male (n=80)	Obese male (n=80)	Test	P-value
Age (year)	31.7 ± 5.6	31.7 ± 6.1	t=0.000	1.000
Have children Yes No	 80 (100.0) 0 (0.0)	 74 (92.5) 6 (7.5)	χ^2=4.329	0.037*
Education University Secondary school Preparatory school Primary school	 18 (22.5) 54 (67.5) 8 (10.0) 0 (0.0)	 22 (27.5) 42 (52.5) 10 (12.5) 6 (7.5)	χ^2=5.708	0.127*
Employment Yes No	 34 (42.5) 46 (57.5)	 20 (25.0) 60 (75.0)	χ^2=5.479	0.019
Family income/month (NIS) < 1000 1000-2000 > 2000	 8 (10.0) 36 (45.0) 36 (45.0)	 30 (37.5) 34 (42.5) 16 (20.0)	χ^2=20.486	< 0.001

NIS: new Israeli Shekel (~ 0.27 $US).
Values are n (%) except age where values are expressed as means ± SD.
*P-value of $\chi^2_{(corrected)}$ test; P<0.05: Significant; P>0.05: not significant.

Table 2. Anthropometric measurements of the study population

Anthropometric measurement	Non-obese male (n=80)	Obese male (n=80)	% difference	t-value	*P*-value
Weight (kg) (min-max)	76.8 ± 7.8 59.1-93.7	112.6 ± 11.1 90.0-132.1	37.8	16.595	< 0.001
Height (m) (min-max)	1.83 ± 0.06 1.68-1.94	1.82 ± 0.08 1.67-1.96	0.5	0.235	0.815
BMI (kg/m^2) (min-max)	22.8 ± 1.5 18.9-24.8	33.4 ± 2.5 30.0-40.6	37.7	22.443	< 0.001

kg: Kilogram; m: Meter; BMI: Body mass index: People with BMI=18.5-24.9 were considered to have normal weight and people with BMI ≥ 30.0 were classified obese (WHO, 2020).
Values are expressed as means ± standard deviation (SD).
P<0.05: Significant; P>0.05: Not significant.

Table 3. Lipid profile of the study population

Parameter (mg/dl)	Non-obese male (n=80)	Obese male (n=80)	% difference	t-value	*P*-value
Cholesterol (min-max)	127.9 ± 38.4 65-195	212.6 ± 42.4 94-303	49.8	9.320	< 0.001
Triglycerides (min-max)	113.8 ± 35.6 53-184	230.9 ± 30.7 144-292	67.9	15.750	< 0.001
HDL-C (min-max)	52.7 ± 5.4 45-63	51.9 ± 4.0 45-59	1.5	0.918	0.360
LDL-C (min-max)	93.9 ± 22.9 53-143	121.7 ± 32.8 49-178	25.8	4.390	< 0.001

HDL-C: High-density lipoprotein cholesterol; LDL-C: Low-density lipoprotein cholesterol.
Values are expressed as means ± standard deviation (SD).
P<0.05: Significant; P>0.05: Not significant.

Table 4. Hormonal level of the study population

Hormone	Non-obese male (n=80)	Obese male (n=80)	% difference	t-value	*P*-value
Testosterone (ng/ml) (min-max)	6.3 ± 0.8 4.3-7.0	3.6 ± 1.5 1.0-8.0	54.5	10.190	< 0.001
LH (mlU/ml) (min-max)	4.7 ± 2.6 2.0-13.0	9.7 ± 5.3 1.2-26.8	69.4	5.290	< 0.001
FSH (mlU/ml) (min-max)	4.6 ± 1.3 2.6-9.7	12.3 ± 6.4 1.8-37.0	91.1	7.117	< 0.001

LH: Luteinizing hormone; FSH: Follicle stimulating hormone.
Values are expressed as means ± standard deviation (SD).
P<0.05: Significant.

Table 5. Semen parameters of the study population

Parameter	Non-obese male (n=80)	Obese male (n=80)	% Difference	t-value	*P*-value
Sperm concentration Volume (ml) (min-max)	3.1 ± 0.6 2.0-4.5	2.2 ± 0.9 0.8-5.5	34.0	5.353	< 0.001
Count (million/ml) (min-max)	43.7 ± 16.0 23.0-83.0	7.8 ± 5.2 1.5-25.0	139.5	13.550	< 0.001
Total count (million) (min-max)	135.3 ± 55.8 9.0-294.0	17.6 ± 21.6 3.2-137.5	15.4	12.440	< 0.001
Sperm motility					
Total motility (%) (min-max)	57.7 ± 11.2 40-80	28.7 ± 14.8 4-66	67.0	9.861	< 0.001
Fast progressive (%) (min-max)	18.9 ± 5.4 10-40	0.23 ± 1.27 0-8	195.3	21.150	< 0.001
Slow progressive (%) (min-max)	28.7 ± 7.5 15-40	18.7 ± 11.6 0-50	42.1	4.560	< 0.001
Non-progressive (%) (min-max)	10.1 ± 4.5 2-22	9.8 ± 4.7 2-28	2.8	0.269	0.789
Immotile (%) (min-max)	42.4 ± 11.2 20-60	71.3 ± 14.8 34-96	50.9	9.860	< 0.001
Sperm morphology					
Normal form (%) (min-max)	39.0 ± 4.6 30-51	25.9 ± 4.7 12-38	40.5	12.680	< 0.001
Abnormal form (%) (min-max)	61.0 ± 4.6 49-70	74.1 ± 4.7 62-88	19.5	12.630	< 0.001

Values are expressed as means ± standard deviation (SD).
P<0.05: Significant; P>0.05: Not significant.

42.1, respectively. In contrast, immotile sperms were significantly higher in obese males (71.3 ± 14.8 vs. 42.4 ± 11.2%, % difference=50.9 and P<0.001). However, no significant difference was found in non-progressive motility among obese and non-obese males. Sperm morphology showed significantly lower normal forms (25.9 ± 4.7 vs. 39.0 ± 4.6%, P<0.001) whereas abnormal forms were significantly higher (74.1 ± 4.7 vs. 61.0 ± 4.6%, P<0.001) in obese males with respect to controls, with % differences of 40.5 and 19.5, respectively.

Body mass index in relation to the study parameters

As indicated in Table 6, Pearson correlation test displayed significant positive correlations of BMI with cholesterol (r=0.737, P=<0.001), triglycerides (r=0.836, P<0.001), LDL-C (r=0.493, P<0.001), LH (r=0.445, P<0.001), FSH (r=0.492, P<0.001), immotile sperm (r=0.738, P<0.001) and abnormal sperm form (r=0.759, P<0.001), while significant negative correlation was found with testosterone (r=- 0.755, P<0.001), sperm concentration in terms of volume (r=- 0.465, P<0.001), count (r=- 0.774, P<0.001) and total count (r=- 0.742, P<0.001), sperm motility in terms of total motility (r=- 0.738, P<0.001), fast progressive (r=- 0.856, P<0.001) and slow progressive (r=- 0.494, P<0.001), and normal sperm form (r=- 0.759, P<0.001).

Discussion

Obesity is a modern day epidemic complex multifactorial disease, with genetic, behavioral, socioeconomic, and environmental origins, that greatly raises risk of chronic disease morbidity; namely disability,

Table 6. The correlation of BMI with the study parameters

Parameter	BMI (kg/m²) Pearson's correlation (r)	*P*-value
Lipid profile		
Cholesterol (mg/dl)	0.737	< 0.001
Triglycerides (mg/dl)	0.836	< 0.001
HDL-C (mg/dl)	-0.034	0.693
LDL-C (mg/dl)	0.493	< 0.001
Hormone		
Testosterone (ng/ml)	-0.755	< 0.001
LH (mlU/ml)	0.445	< 0.001
FSH (mlU/ml)	0.492	< 0.001
Sperm cocentration		
Volume (ml)	-0.465	< 0.001
Count (million/ml)	-0.774	< 0.001
Total count (million)	-0.742	< 0.001
Sperm motility		
Total motility (%)	-0.738	< 0.001
Fast progressive (%)	-0.856	< 0.001
Slow progressive (%)	-0.494	< 0.001
Non-progressive (%)	-0.064	0.573
Immotile (%)	0.738	< 0.001
Sperm morphology		
Normal form (%)	-0.759	< 0.001
Abnormal form (%)	0.759	< 0.001

HDL-C: High-density lipoprotein cholesterol; LDL-C: Low-density lipoprotein cholesterol; LH: Luteinizing hormone; FSH: Follicle stimulating hormone.
P<0.05: Significant; P>0.05: not significant.

depression, diabetes mellitus, cardiovascular disease, certain cancers, and mortality. The worldwide prevalence of overweight and obesity (Globesity) has doubled since 1980 to an extent that nearly a third of the world's population is now classified as overweight or obese [28]. In the last twenty years, particular attention has been paid to the effect of obesity on male fertility. However, the mechanisms underlying this effect still need to be clarified. Reproductive hormones and semen parameters may vary in obese compared to non-obese males resulting in impairment of fertility. This research is aimed to evaluate testosterone, LH, FSH, semen parameters in terms of quantity and quality as well as lipid profile, and their correlations with BMI in obese and non-obese Palestinian adult males.

The present study showed significant differences between non-obese and obese males, with less number of children, lower number of employee and less family income/month among obese group. The negative impact of obesity on male fertility is expected to be reflected in having a lower number of children [17]. Regarding employment, Tunceli and his colleagues found that obesity is associated with reduced employment [29]. In other words, a significant negative association between obesity and employment was reported [30]. The more frequent obesity with less family income was previously documented [31]. The inability of low income families to afford healthy food may be a major contributor to the increase prevalence of obesity [32].

The BMI was significantly higher in obese males compared to non-obese control group. This finding coincides with that obtained by other authors [33,34]. The BMI is the metric measure currently in use for defining anthropometric height/weight characteristics in adults and for categorizing them into groups [35]. Obesity is defined as abnormal or excessive fat accumulation that may impair health and the presence of a BMI ≥ 30 kg/m^2 [7]. Therefore, the BMI is widely adopted as an index of obesity. In general, obesity results from an energy imbalance caused by eating too many calories and not getting enough exercise [36]. It has been accepted that exercise is essential for the management of obesity [37].

The mean levels of cholesterol, triglyceride and LDL-C were significantly increased in obese compared to non-obese males. Significant positive correlations were also found between BMI with total cholesterol, triglyceride, and LDL-C. Similar results were achieved in the literature [34,38]. Dyslipidemia in obese males is mainly attributed to insulin resistance that develops in obese individuals. In such circumstances, there is an increase in the mobilization of free fatty acids from fat depots, since insulin inhibits the hormone sensitive lipase. Excess serum fatty acids are converted into triglycerides, phospholipids and cholesterol in liver which may be discharged into blood [39,40].

Hormonal analysis revealed significant decrease in the mean level of testosterone in obese males than non-obese group. Conversely, the mean levels of LH and FSH were significantly increased in obese males. A significant negative correlation was found between BMI with testosterone whereas significant positive correlations with LH and FSH. This means that obesity is associated with the disturbance of reproductive hormones and subsequently impose a deleterious impact on male fertility. Such findings are in agreement with that reported earlier [41,42]. Increase levels of LH and FSH in obese men is more likely to be due to negative feedback control excreted by low testosterone level on hypothalamic-pituitary-gonadal axis [43,44]. Significant alterations and correlations of testosterone, LH and FSH with BMI may explain the role of such tri-hormonal interplay in subfertility of obese men. However, fertility is a complex phenomenon, partly physiologically and partly behaviorally determined, thus an interdisciplinary approach is required to understand it.

Semen parameters in terms of quantity and quality were altered in obese compared to non-obese control males. Sperm concentration, motility and normal sperm form were significantly lower while immotile and abnormal sperm form were significantly higher in obese males. The BMI showed a significant positive correlation with immotile and abnormal sperm form and a significant negative correlation with sperm concentration, motility and normal sperm form. These results in combination with reproductive hormonal disturbance do confirm the potential risk of obesity on male fertility. Many authors showed such alterations of semen parameters in obese men [42,45,46]. Obesity may negatively affect sperm count and sperm characteristics through several interfered and intricate possible mechanisms including: 1) impaired Sertoli cells function and spermatogenesis [47], 2) increased scrotal temperature due to the increased scrotal adiposity, which may damage spermatogenesis and impair semen parameters [42], 3) increased production of reactive oxygen species and inflammatory mediators impairing testicular and epididymal tissues [48], 4) accumulation of toxic substances and liposoluble endocrine disruptors in fat tissue [47], 5) epigenetic changes including methylation of sperm DNA, nuclear protein composition and modification of non-coding RNA [49], and 6) elevation of leptin levels which have negative associations with testosterone levels and sperm parameters and positive associations with FSH and LH levels and abnormal sperm morphology [50]. The last speculation is supported by decreased levels of testosterone concurrent with increased levels of FSH and LH in obese males that presented in this study. Finally, this research accumulates further evidence on obesity-induced male subfertility and/or infertility in terms of having a deleterious effect on reproductive hormones as well as on semen parameters.

In Conclusion Obesity is associated with lipid profile, testosterone, gonadotropins, semen parameters and BMI in Palestinian adult males. Serum cholesterol, triglycerides, LDL-C, LH, FSH, immotile and abnormal sperm form were significantly increased whereas testosterone, sperm concentration, motility and normal form were significantly decreased with BMI increase. Frequent monitoring of lipid profile is necessary for obesity, and testosterone, LH, FSH and semen parameters are very important test for male fertility. Therefore, obese males with high lipid profile, LH and FSH as well as with low testosterone levels and altered semen parameters are highly encouraged for weight-loss diet and/or exercise to improve their fertility.

Declaration of conflict of interest

The authors report no conflicts of interest.

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