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The Impact of Body Mass on Male Fertility in a Cohort of 127 Patients

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Abstract

Background: Aromatase and leptin are two adipose tissue cytokines. The former converts androgens into estrogens and stimulates adipogenesis. The latter cannot fully stimulate GnRH release as its hypothalamic receptors are reduced in obese men. Thus, obesity which is associated with an adipose tissue increment can interfere with male fertility. Objective: We aim to study the correlation between the body mass index (BMI) of an individual and the quality of semen he produces. Patients and Method: By means of the software R 4.2.1 we performed a retrospective analysis of the relationship between the BMI and the semen alterations in the patients managed at the former Military Teaching Hospital of Cotonou from October 1, 2017, to September 30, 2022: a bi-varied analysis and Fischer's exact test (significance threshold 5%, confidence interval 95%) followed by a logistic regression when a non-significant p-value is below 0.20. Results: 127 males managed for infertility (mean age = 36.2 years) were recorded, including 11.1% obese (BMI > 30 kg/m^2) and 36.5% overweighted (25 kg/m^2 < BMI ≤ 30 kg/m^2). The most frequent semen alterations were: oligoasthenospermia (27.8%), asthenospermia (22.2%), oligoasthenoteratospermia (14.3%), azoospermia (13.5%) and asthenoteratospermia (9.5%). Bi-varied analysis showed no correlation between the BMI and the semen alterations (p-value ranged from 0.086 to 0.9) and no difference in semen alterations between patients with BMI below and above 25 kg/m^2 (p-value ranged from 0.12 to 0.9). Logistic regression demonstrated that asthenoteratospermia were correlated with BMI ≥ 25 kg/m^2 [OR = 2.1, 95% CI (1.50 - 2.70), p = 0.021]. Conclusion: Male obesity and overweight can trigger asthenoteratospermia.

Keywords

Body Mass Index, Male Infertility, Semen Alterations

1. Introduction

Infertility is a public health issue which involves 15% of couples worldwide [1]. In Africa, 43% of couples' infertility is linked to a male factor [2]. Etiologies of male infertility are variable. Obesity can possibly alter the spermatogenesis [3]. In fact, obesity is associated with an increment of fatty tissue [4]. The latter produces aromatase and leptin, two cytokines that interfere with spermatogenesis and testicular steroidogenesis. The aromatase converts testosterone into estrogens. The estrogens exert negative feedback onto the hypothalamus-hypophysis axis. Besides, estrogens stimulate adipogenesis [5] while testosterone inhibits adipogenesis and stimulates myogenesis [6]. Clearly, obesity induces in males a vicious cycle of estrogen excess and androgen deficit. The leptin stimulates the hypothalamus to release GnRH thereby making the hypophysis to release FSH and LH and stimulates testicular spermatogenesis and steroidogenesis [7]. The leptin's effect is reduced in obese males as they do experience a reduction in hypothalamic leptin receptors [8]. In our institution, the prevalence of male infertility is 14.1% among patients aged 15 through 40 years [9]. Elucidating the obesity's fertility-altering effect can insert anti-obesity measures into the therapeutic armamentarium against male infertility.

2. Objective

This study aims to elucidate the correlation between the body mass and male infertility in males whose couples are managed or followed up for infertility. Specifically, we will study the relationship that links a male's body mass index (BMI) to the quality of the semen that he produces.

3. Patients and Method

The study was conducted at the former Military Teaching Hospital of Cotonou from October 1, 2017, to September 30, 2022. It included all patients that were managed in the urological department for infertility, who were living a heterosexual couple life for at least 12 months and who had performed at least one semen analysis. Those patients who performed a premarital medical examination or any form of self-evaluation of fertility status and those patients who had undergone any treatment (inguinal surgery, chemotherapy, etc.) or event that could alter their semen were excluded. Smoker and alcoholic patients were also excluded. We conducted an exhaustive medical-records-based census of all patients who met the above-mentioned criteria. The data collected in each patient were age, weight, height, and semen analysis report. We calculated the body mass index (BMI in kg/m^2) by dividing the weight (kg) by the square of the height (m). The patients were categorized into classes of BMI: less than 18.5, 18.5 to 25, 25 to 30 and more than 30. We used the R 4.2.1. software to analyze the data. By means of a bi-varied analysis and Fischer's exact test, we studied the link between the BMI and the semen alterations that the patients presented on semen analysis. The significance threshold was 5% and the interval of confidence

was 95%. We used logistic regression to further analyze the data when the p-value was non-significant but less than 0.20.

4. Results

Among the 2506 patients managed during the study period, 293 males (11.7%) consulted for infertility. But semen analysis data were fully available in only 127 of them (Table 1). Their age ranged from 23 to 60 years; their mean age was 36.3 years. Their height ranged from 1.62 to 1.90 meters; their mean height was 1.71 meters. Their BMI ranged from 17.9 to 35.9 kg/m^2. The BMI in 62 (48.8%), 47 (37.0%), 14 (11.0%) and 4 (3.1%) of the 127 patients was respectively less than 18.5, between 18.5 and 25, between 25 and 30 and more than 30 kg/m^2.

Table 2 summarizes the semen analysis data. The main semen alterations observed were oligoteratospermia (27.6%), asthenospermia (22%), oligoasthenoteratospermia (14.2%), azoospermia (13.3%), asthenoteratospermia (9.4%), oligospermia (4%) and teratospermia (3.2%). The semen analysis was normal in 2, *i.e.*, 1.6% of patients.

The BMI in most of the patients, *i.e.*, 109 out of 127 patients, ranged from 18.5 to 30 kg/m^2 (Table 3). The bi-varied analysis demonstrated no correlation between the BMI and the semen alterations: the p-value ranged from 0.086 for asthenoteratospermia to 0.9 or more for alterations such as oligoasthenoteratospermia. Resuming the analysis with the 127 patients categorized into two subsets (Table 4), one with BMI < 25 kg/m^2 and the other with BMI ≥ 25 kg/m^2, showed no significant correlation between the BMI and the semen analysis data. Here, the p-value varied from 0.12 for teratospermia to 0.9 or more for alterations such as asthenospermia.

Table 1. Demographic characteristics of the patients.

	Age (years)	Weight (kg)	Height (m)	BMI (kg/m^2)
Minimum	23	54	1.62	17.9
Maximum	60	108	1,9	35.9
Mean	***36.3***	***74.3***	***1.71***	***25.4***

Table 2. Semen alterations.

ALTERATIONS	SIZE	%	ALTERATIONS	SIZE	%
Asthenospermia	28	22	OAS	35	27.6
Oligospermia	5	4	OATS	18	14.2
Teratospermia	4	3.2	OTS	1	0.8
Necrospermia	1	0.8	ONS	1	0.8
Hypospermia	1	0.8	OANS	1	0.8
Azoospermia	17	13.4	ATS	12	9.4
Leucospermia	1	0.8	Normal	2	1.6

Table 3. Correlation between BMI and semen alterations.

SEMEN ALTERATIONS	BMI < 18.5 (*n* = 4)	18.5 ≤ BMI < 25 (*n* = 62)	25≤ BMI < 30 (*n* = 47)	BMI ≥ 30 (*n* = 14)	p-value
Asthenospermia	0 (0.0%)	15 (24.2%)	11 (23.9%)	2 (14.3%)	0.8
Oligospermia	1 (25.0%)	2 (3.2%)	2 (4.3%)	0 (0.0%)	0.3
Teratospermia	0 (0.0%)	4 (6.5%)	0 (0.0%)	0 (0.0%)	0.3
Necrospermia	0 (0.0%)	1 (1.6%)	0 (0.0%)	0 (0.0%)	> 0.9
Hypospermia	0 (0.0%)	1 (1.6%)	0 (0.0%)	0 (0.0%)	> 0.9
Azoospermia	0 (0.0%)	10 (16.1%)	6 (13.0%)	1 (7.1%)	0.9
Leucospermia	0 (0.0%)	0 (0.0%)	1 (2.2%)	0 (0.0%)	0.5
OAS*	1 (25.0%)	15 (24.2%)	12 (26.1%)	7 (50.0%)	0.3
OATS*	0 (0.0%)	9 (14.5%)	8 (17.4%)	1 (7.1%)	0.9
OTS*	***0 (0.0%)***	***0 (0.0%)***	***0 (0.0%)***	***1 (7.1%)***	***0.14***
ONS*	0 (0.0%)	0 (0.0%)	1 (2.2%)	0 (0.0%)	0.5
OANS*	0 (0.0%)	1 (1.6%)	0 (0.0%)	0 (0.0%)	>0.9
ATS*	***2 (50.0%)***	***4 (6.5%)***	***5 (10.9%)***	***1 (7.1%)***	***0.086***
Normal	0 (0.0%)	0 (0.0%)	1 (2.2%)	1 (7.1%)	0.2

*OAS = oligoasthenospermia, OATS = oligoasthenoteratospermia, OTS = oligoteratospermia, ONS = oligonecrospermia, OANS = oligoasthenonecrospermia, ATS = asthanoteratospermia.

Table 4. Correlation between BMI and semen alterations.

SEMEN ALTERATIONS	BMI < 25 kg/m^2 (*n* = 66)	BMI ≥ 25 kg/m^2 (*n* = 61)	p-value
Asthenospermia	15 (22.7%)	13 (21.3%)	0.9
Oligospermia	3 (4.5%)	2 (3.3%)	>0.9
Teratospermia	4 (6.1%)	0 (0.0%)	0.12
Necrospermia	1 (1.5%)	0 (0.0%)	>0.9
Hypospermia	1 (1.5%)	0 (0.0%)	>0.9
Azoospermia	10 (15.2%)	7 (11.5%)	0.6
Leucospermia	0 (0.0%)	1 (1.6%)	0.5
OAS*	16 (24.2%)	19 (31.1%)	0.4
OATS*	9 (13.6%)	9 (14.8%)	0.8
OTS*	0 (0.0%)	1 (1.6%)	0.5
ONS*	0 (0.0%)	1 (1.6%)	0.5
OANS*	1 (1.5%)	0 (0.0%)	>0.9
ATS*	6 (9.1%)	6 (9.8%)	0.9
Normal	0 (0.0%)	2 (3.3%)	0.2

*OAS = oligoasthenospermia, OATS = oligoasthenoteratospermia, OTS = oligoteratospermia, ONS = oligonecrospermia, OANS = oligoasthenonecrospermia, ATS = asthanoteratospermia.

Table 5. (a) Correlation between BMI and asthenoteratospermia; (b) Correlation between BMI and oligoteratospermia.

(a)

	OR	95% IC	p-value
BMI			***0.021***
<25	1		
≥25	2.1	1.50 - 2.70	0.019

(b)

	OR	95% IC	p-value
BMI			***0.071***
<25	1		
≥25	1.9	0.20 - 3.60	0.08

The asthenoteratospermia ($p = 0.086 < 0.20$) and the oligoteratospermia ($p = 0.14 < 0.20$) seemed to be associated to the BMI. Nevertheless, submitting those two semen parameters to a logistic regression analysis (**Table 5(a)**, **Table 5(b)**) demonstrated that only the asthenoteratospermia was significantly correlated to the BMI (OR = 2.1, 95% CI = 1.50 - 2.70, $p = 0.021$). The correlation between the oligoteratospermia and the BMI was not significant (OR = 1.9, 95% CI = 0.20 - 3.60, $p = 0.071$).

5. Discussion

The effects of the leptin and the aromatase portend that the fatty tissue increment and leptin resistance associated with obesity can alter the testicular production of spermatozoids. In our population, eleven subsets of patients presented either single or combined alterations on semen analysis. Only 1 case of hypospermia and 2 cases of normal semen analysis were observed.

There was no correlation between the BMI and most of the observed semen parameters' alterations. Only the asthenoteratospermia and the oligoteratospermia were associated with BMI ≥ 25 kg/m^2 (*i.e.*, overweight and obesity): although their risk was respectively 2.1 and 1.9-fold increased, only the asthenoteratospermia was significantly correlated with BMI ≥ 25 kg/m^2.

Studies of the effect of obesity on fertility have so far led to variable and even contradictory results. According to McDonald, there is no link between obesity and sperm volume alteration [10]. Some authors have found that obesity triggers hypospermia [11]. Other authors have shown that BMI is negatively correlated the sperm motility and concentration [12]. Some study has demonstrated a positive correlation between BMI and sperm morphologic anomalies [13]. According to Dubeux, there is no association between obesity and semen alterations [14]. Further, Iliceto has demonstrated that semen parameters' alterations are not linked to obesity [15]. All those divergent results suggest that the relationship between the adipose tissue and fertility may be more complex than the ef-

fects of leptin and aromatase do suggest.

Our study has demonstrated that male obesity or overweight can lead to asthenoteratospermia thereby stressing that men should maintain a BMI below 25 kg/m^2. However, we think that studying the effect of counter-obesity therapeutic measures on asthenoteratospermia in males with no other cause of semen alteration than obesity will further test the link between the body mass index and the asthenoteratospermia.

6. Conclusion

The risk of asthenoteratospermia was 2.1-fold higher in obese and overweighted men. The other semen alterations were not correlated with the body mass.

Limitations of This Study

It is a retrospective study. The population's size is relatively limited. Ideally, the correlation between the body mass index and the sperm count should be studied by comparing two demographically similar populations of men, one set of fertile males and one set of infertile males.

Conflicts of Interest

We have no conflict of interest to declare.

Ethical Issues

The study has been submitted to and approved by our "Comité d'Ethique et de Recherche en Sciences de la Santé" before being carried out.

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