Obesity and Male Infertility in The Dukagjin Region in Republic of Kosovo

Afrim Zeqiraj¹, Zafer Gashi¹, Sadi Bexheti³, Shkelzen Elezaj⁴, Sanije Berisha⁵ and Agim Shabani²*

¹Department of Biochemistry, FAMA Colleague, Republic of Kosovo
²Department of Chemistry, University of Tetovo, Republic of Macedonia
³Faculty of Medicine, University of Tetovo, Republic of Macedonia
⁴Clinic of Urology, Regional Hospital of Peja, Republic of Kosovo
⁵Department of Biochemistry, University clinical center, Tetovo, Republic of Macedonia

*Corresponding author: Agim Shabani, University of Tetovo, Tetovo, Republic of Macedonia, Tel: +38944356500; E-mail: agim.shabani@unite.edu.mk

Received date: June 15, 2017; Accepted date: October 25, 2017; Published date: November 06, 2017

Copyright: © 2017 Zeqiraj A, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Citation: Zeqiraj A, Gashi Z, Bexheti S, Elezaj S, Berisha S, et al. (2017) Obesity and Male Infertility in The Dukagjin Region in Republic of Kosovo. Eur Exp Biol. Vol. 7 No. 5:35.

Abstract

In this paper will be studied the causes that can cause overweight in men, and the disorder of hormone production from endocrine glands, as well as the influence of obesity in infertility. Purpose of the work, it is to determine the degree of male infertility by overweight men. The work was done in the region of Peja, in the Republic of Kosovo. Materials and methods, the sampling was done in March 2015-March 2017 in the Biolab-Zafi endocrinology laboratory in Peja, Republic of Kosovo. Our results show a significant increase in male infertility rates with BMI 29.97 ± 3.22, compared with men with BMI 23.52 ± 2.0 (p<0.00001). We also found a significant increase in working group rates with FSH 7.99 ± 8.21, compared to control group FSH 4.72 ± 2.63 (p<0.008). We also found a significant increase in working group rates with LH 5.06 ± 2.74, compared to control group LH 3.79 ± 1.99 (p<0.002). We also found a significant increase in working group rates with Prolactin 17.37 ± 7.66, compared to control group Prolactin 13.05 ± 4.8 (p<0.00004). We also found a significant increase in working group rates with Testosterone 4.16 ± 1.88, compared to control group Testosterone 5.79 ± 1.48 (p<0.005). Conclusion, the results from this study show that obesity in our country, the Republic of Kosovo, is a growing problem in the development of public health, and is an important risk factor for the appearance of male infertility. The effects of BMI growth tend to increase the risk of DNA damage in ejaculates, increase in hormone levels (FSH, LH, prolactin, testosterone), decrease sperm parameters (decrease in number, decrease in movement), oxidative stress, Risk of hypertension, cardiac disorders, diabetes and other chronic disorders.

Keywords: Infertility; BMI; FSH; LH; Prolactin; Testosterone

Introduction

Undoubtedly overweight is a medical condition in which excess body fat, or white fat tissue, accumulates in the body to the extent that such fat collection can adversely affect human reproductive health. An individual can be defined as overweight if their BMI is 25-30 kg/m² and obese if their BMI exceeds 30 kg/m². However, the distribution of body fat specifically in the central abdominal region has also been used to diagnose a patient as obese and currently waist circumference is believed to be a more accurate marker of obesity. However, these definitions should only be considered as guidelines, as the risk of developing chronic diseases increases progressively when the BMI increases above 21 kg/m² [1]. Infertility is a major medical and social problem around the world as regards 15% of couples are infertile and 40% are infertile due to infertility of the male factor and 40% are due to female infertility and the remainder is idiopathic factor [2]. Over the past three decades, many countries in the urbanized world have witnessed a growing epidemic of overweight and obesity. The epidemic is largely fueled by urbanization, economic growth, industrialization, mechanized transport, and the adoption of sedentary lifestyle, coupled with the high availability of foods with high caloric content [3]. Qin et al. (2007) [4] established that the associations between BMI and semen quality were found to be statistically significant even after an adjustment for reproductive hormone levels, suggesting that there are other influencing factors. Some of the elements that may contribute to the effects of obesity on male fertility in infertile patients include increased adipocrine growth from fatty tissue, physical psychiatric problems, apnea, which may adversely affect serum testosterone levels in the morning. (Luboshitzky et al., 2005) [5] and increased scrotal temperature, due to increased fat deposition in the upper thighs and abdomen, which interferes with spermatogenesis (Jung and Schuppe, 2007) [6]. Moreover, overweight (>25 kg/m²) and obese (>30kg/m²) adversely affect clinical pregnancy and live birth rates after treatment with ICSI /

© Under License of Creative Commons Attribution 3.0 License | This article is available from: http://www.imedpub.com/european-journal-of-experimental-biology/
IVF [7,8]. BMI is used as the chief indicator of obesity, with stratified BMI categories as follows: 18.5–24.9 kg/m² (normal), 25 kg/m² and above (overweight) and 30 kg/m² and above (obese) [9]. Luboshitzky et al. (2005) Reducing the concentration of testosterone in serum in infertile males is related to the fact that sleep apnea in obese people is associated with decreased pituitary function for gonadal stimulation. [5]. Although the impact of obesity on male fertility is only now being evaluated in depth, Hammoud et al. (2008) [10] show that Avicena's research on excessive weight on males and on health disadvantages in which he describes the stupid man as infertile and unable to carry women. This is based on multiple research, with fatigued men who have demonstrated erectile disorder and low sperm values (Hammoud et al., 2012) [11]. Hormones FSH, LH and testosterone are the main regulators of the development of male reproductive organs and useful in the management of male infertility. For the production of spermatozooids, the presence of hormones FSH, LH and testosterone is required. Spermatogenesis stimulates the binding of sertol cells to FSH, while LH stimulates the production of testosterone in Leydig cells. The failure of pituitary to secret FSH and LH will result in disruption of testicular function leading to infertility [12].

Purpose of the Work

It is to determine the degree of male infertility by overweight men. The work was done in the region of Peja, in the Republic of Kosovo.

Materials and Methods

In order to determine the hormonal parameters, infertile blood patients have been received after being introduced to the doctor due to the inability to conceive after a period of more than a year of regular, unprotected relationships. The sampling was done in March 2015 - March 2017 in the Biolab-Zafi endocrinology laboratory in Peja, Republic of Kosovo. All patients were taken: name, surname, year of birth, BMI (body mass index), infertility periods (primary or secondary infertility), and is blood collection for analysis. Measurement of hormone parameters was done with the Biomerieux Mini Vidas Automated Immunoassay Analyzer. Finding is to calculate the statistical program used Anova and t-test (Student TEST). The standard deviation is calculated, the arithmetic average. The importance of the presentation is at p<0.05. Blood samples were collected from 207 patients, with 137 infertility patients and 70 patients in the controls. The age of men in the study was between 20-45 years.

Results

Our results show a significant increase in male infertility rates with BMI 29.97 ± 3.22, compared with men with BMI 23.52 ± 2.0 (p<0.00001).

We also found a significant increase in working group rates with FSH 7.99 ± 8.21, compared to control group FSH 4.72 ± 2.63 (p<0.0008).

We also found a significant increase in working group rates with LH 5.06 ± 2.74, compared to control group LH 3.79 ± 1.99 (p<0.002).

We also found a significant increase in working group rates with Prolactin 17.37 ± 7.66, compared to control group Prolactin 13.05 ± 4.8 (p<0.00004).

We also found a significant increase in working group rates with Testosterone 4.16 ± 1.88, compared to control group Testosterone 5.79 ± 1.48 (p<0.005) (Table 1, Figures 1 and 2).

Table 1: BMI, FSH, LH, prolactin, testosterone levels in working group and control group.

Figure 1: Frequency distribution for BMI, FSH, LH, prolactin, testosterone in working group.

Figure 2: Frequency distribution for BMI, FSH, LH, prolactin, testosterone in control group.

This article is available from: http://www.imedpub.com/european-journal-of-experimental-biology/
levels in proven treatment with exogenous testosterone is likely to adversely affect sperm quality and fertility. FSH plays a key role in initiation of spermatogenesis and maturation of spermatozoa, FSH is necessary. In the infertility men, higher concentration of FSH is considered to be a reliable indicator of germinal epithelial damage, and was shown to be associated with azoospermia and severe oligozoospermia [14] de Kretser et al. [15] reported elevated levels of serum FSH with increasing severity of seminiferous epithelial destruction. However, a recent study by Najafi et al. (2011) showed a decrease in fertility parameters like sperm count and motility among overweight and obese men [16]. In the present study, gonadotropin (FSH and LH) levels were significantly elevated in infertile males when compared with the levels in proven fertile controls. These results are in accordance with the studies of Sulthan et al. [17], Zabul et al. [18] Weinbauer and Nieschlag [19], and Subhan et al. [20] who showed elevated levels of both follicle stimulating hormone and luteinizing hormones in infertile males. Although obesity is associated with low serum testosterone concentrations, treatment with exogenous testosterone is likely to adversely affect fertility as a result of the feedback mechanism influencing gonadotrophin release. After the inhibition of aromatase inhibitory activity in overweight patients, resulted in decreased estradiol levels but in some cases helped to normalize spermatozoa [21]. In the present study, gonadotropin (FSH and LH) levels were significantly elevated in infertile males when compared with the levels in proven fertile controls. These results are in accordance with the studies of Sulthan et al. [22]. Zabul et al. [18], Weinbauer and Nieschlag [23] and Subhan et al. [24] who showed elevated levels of both follicle stimulating hormone and luteinizing hormones in infertile males. FSH, LH and testosterone are prime regulators of germ cell development. The quantitative production of spermatozoa generally requires the presence of FSH, LH and testosterone. FSH acts directly on the seminiferous tubules whereas luteinizing hormone stimulates spermatogenesis indirectly via testosterone. FSH plays a key role in stimulating mitotic and meiotic DNA synthesis in spermatogonia [25]. FSH, LH and testosterone evaluation is useful in the management of male infertility.

## Discussion

It is very important to note that some obese males have normal sperm quality and fertility, giving inferences that obesity may not be consistent, as suggested in a study by Pauli et al. (2008) in which there was no association between Obesity and sperm quality. [9]. Male obesity assessment methods showed a significant correlation of obesity with male infertility, for our study we have chosen BMI as an indicator of obesity over the percentage of body fat, for the reason that BMI measurement is appropriate in the venation of a is developing, which can be most easily determined by the health personnel. [13]. For initiation of spermatogenesis and maturation of spermatozoa, FSH is necessary. In the infertile men, higher concentration of FSH is considered to be a reliable indicator of germinal epithelial damage, and was shown to be associated with azoospermia and severe oligozoospermia [14] de Kretser et al. [15] reported elevated levels of serum FSH with increasing severity of seminiferous epithelial destruction. However, a recent study by Najafi et al. (2011) showed a decrease in fertility parameters like sperm count and motility among overweight and obese men [16]. In the present study, gonadotropin (FSH and LH) levels were significantly elevated in infertile males when compared with the levels in proven fertile controls. These results are in accordance with the studies of Sulthan et al. [17], Zabul et al. [18] Weinbauer and Nieschlag [19], and Subhan et al. [20] who showed elevated levels of both follicle stimulating hormone and luteinizing hormones in infertile males. Although obesity is associated with low serum testosterone concentrations, treatment with exogenous testosterone is likely to adversely affect fertility as a result of the feedback mechanism influencing gonadotrophin release. After the inhibition of aromatase inhibitory activity in overweight patients, resulted in decreased estradiol levels but in some cases helped to normalize spermatogenesis and fertility [21]. In the present study, gonadotropin (FSH and LH) levels were significantly elevated in infertile males when compared with the levels in proven fertile controls. These results are in accordance with the studies of Sulthan et al. [22]. Zabul et al. [18], Weinbauer and Nieschlag [23] and Subhan et al. [24] who showed elevated levels of both follicle stimulating hormone and luteinizing hormones in infertile males. FSH, LH and testosterone are prime regulators of germ cell development. The quantitative production of spermatozoa generally requires the presence of FSH, LH and testosterone. FSH acts directly on the seminiferous tubules whereas luteinizing hormone stimulates spermatogenesis indirectly via testosterone. FSH plays a key role in stimulating mitotic and meiotic DNA synthesis in spermatogonia [25]. FSH, LH and testosterone evaluation is useful in the management of male infertility.

### Table 1

| Working groups (137 patients) Average/Std | Control groups (70 patients) Average/Std | t-test | Significant p<0.05 | S-significant N-no significant |
|----------------------------------------|------------------------------------------|--------|--------------------|-------------------------------|
| BMI (body mass index)                  | 29.97 ± 3.22                            | 23.52 ± 2.0 | 6.026 | p<0.00001 | S |
| FSH                                    | 7.99 ± 8.21                             | 4.72 ± 2.63 | 3.182 | p<0.008 | S |
| LH                                     | 5.06 ± 2.74                             | 3.79 ± 1.99 | 2.884 | p<0.002 | S |
| Prolactin                              | 17.37 ± 7.66                            | 13.05 ± 4.8 | 4.017 | p<0.00004 | S |
| Testosterone                           | 4.16 ± 1.88                             | 5.79 ± 1.48 | -2.555 | p<0.005 | S |

## Conclusion

The results from this study show that obesity in our country, the Republic of Kosovo, is a growing problem in the development of public health, and is an important risk factor for the appearance of male infertility. The effects of BMI growth tend to increase the risk of DNA damage in ejaculates, increase in hormone levels (FSH, LH, prolactin, testosterone), decrease sperm parameters (decrease in number, decrease in movement), oxidative stress, risk of hypertension, cardiac disorders, diabetes and other chronic disorders. Therefore, our suggestions are to increase the level of health education policies that should be recommended by health professionals in educating the population to maintain adequate body weight, obtaining healthy foods, regular physical exercise, walking, running, recreation, etc. So this is a prerequisite that at minimum costs and adequate health education we achieve the right intention to have a healthy body, while at the same time reducing the degree of male infertility. Our study found that overweight affects the appearance of male infertility at significant levels compared to the control group taken in the study.

## References

1. Haslam DW, James WP (2005) Obesity. Lancet 366: 1197-1209.
2. Weinbauer GF, Nieschlag E (1995) Gonadotrophin control of testicular germ cell development. Adv Exp Med Biol 377: 55-65.
3. Hruby A, Hu FB (2015) The Epidemiology of Obesity: A Big Picture. Pharmacoeconomics 33: 673-689.
4. Qin DD, Yuan W, Zhou WJ, Cui YQ, Wu JQ, et al. (2007) Do obesity, overweight and high body mass index affect semen quality in humans. Andrologia 39: 203-215.
5. Luboshitzky R, Lavie L, Shen-Orr Z, Herer P (2005) Altered luteinizing hormone and testosterone secretion in middle-aged obese men with obstructive sleep apnea. Obes Res 13: 780-786.
6. Jung A, Schuppe HC (2007) Influence of genital heat stress on semen quality in humans. Andrologia 39: 203-215.
7. Keltz J, Zapantis A, Jindal SK, Lieman HJ, Santoro N, et al. (2010) Overweight men: clinical pregnancy after ART is decreased in IVF but not in ICSI cycles. J Assist Reprod Genet 27: 539-544.
8. Petersen GL, Schmidt L, Pinborg A, Kamper-Jørgensen M (2013) The influence of female and male body mass index on live births.
after assisted reproductive technology treatment: a nationwide register-based cohort study. Fertil Steril 99: 1654-1662.

9. Pauli EM, Legro RS, Demers LM, Kunselman AR, Dodson WC, et al. (2008) Diminished paternity and gonadal function with increasing obesity in men. Fertil Steril 90: 346-351.

10. Hammoud AO, Gibson M, Peterson CM, Meikle AW, Carrell DT (2008) Impact of male obesity on infertility: a critical review of the current literature. Fertil Steril 90: 897-904.

11. Hammoud AO, Meikle AW, Reis LO, Gibson M, Peterson CM, et al. (2012) Obesity and male infertility: a practical approach. Semin Reprod Med 30: 486-495.

12. Sultan C, Craste de Paulet B, Audran F, Iqbal Y, Ville C (1985) Hormonal evaluation in male infertility. Ann Biol Clin (Paris) 43: 63-66.

13. MacDonald AA, Herbison GP, Showell M, Farquhar CM (2010) The Impact of Body Mass Index on Semen Parameters and Reproductive Hormones in Human Males: A Systematic Review with Meta-Analysis. Human Reproduction Update 16: 293-311.

14. Bergmann M, Behre HM, Nieschlag E (1994) Serum FSH and testicular morphology in male infertility. Clin Endocrinol (Oxf) 40: 133-136.

15. de Kretser DM (1974) The management of the infertile male. Clin Obstet Gynecol 1: 409-427.

16. Najafi M, kavitha P, Sreenivasa G, Chaithra PT, Vineeth VS, et al. (2011) Overweight and obese men are more prone to infertility-Myth or fact. Journal of paramedical sciences 2: 1.

17. Sultan C, Craste de Paulet B, Audran F, Iqbal Y, Ville C (1985) Hormonal evaluation in male infertility. Ann Biol Clin (Paris) 43: 63-66.

18. Zabul J, Mierzejewski W, Rogoza A (1994) Usefulness of examining gonadotropin hormones and testosterone in men with abnormal semen. Ginekol-pol 65: 71-74.

19. Weinbauer GF, Nieschlag E (1995) Gonadotrophin control of testicular germ cell development. Adv Exp Med Biol 377: 55-65.

20. Subhan F, Tahir F, Ahmad R, Khan ZD (1995) Oligospermia and its relation with hormonal profile. J Pak Med Assoc 45: 246-247.

21. Roth MY, Amory JK, Page ST (2008) Treatment of male infertility secondary to morbid obesity. Nat Clin Pract Endocrinol Metab 4: 415-419.

22. Sultan C, Craste de Paulet B, Audran F, Iqbal Y, Ville C (1985) [Hormonal evaluation in male infertility]. Ann Biol Clin (Paris) 43: 63-66.

23. Weinbauer GF, Nieschlag E (1995) Gonadotrophin control of testicular germ cell development. Adv Exp Med Biol 377: 55-65.

24. Subhan F, Tahir F, Ahmad R, Khan ZD (1995) Oligospermia and its relation with hormonal profile. J Pak Med Assoc 45: 246-247.

25. Anderson RA, Wallace EM, Groome NP, Bellis AJ, Wu FCW (1997) Physiological relationships between inhibin B, follicle stimulating hormone secretion and spermatogenesis in normal men and response to gonadotrophin suppression by exogenous testosterone. Hum Reprod 12: 746-747.