DIFFERENCES BETWEEN SERUM TOTAL TESTOSTERONE LEVELS IN OBESE AND NON-OBESE MALE HEALTH WORKERS

Putu Ayu Indah Saraswati[1*], Ketut Suastika[2], I Made Pande Dwipayana[3]

[1-3]Medical Faculty of Udayana, University/Prof. Dr. I.G.N.G Ngoerah General Hospital

Correspondence Email: indahsaraswati11@gmail.com

Disubmit: 27 Mei 2025 Diterima: 20 Februari 2026 Diterbitkan: 01 Maret 2026
Doi: https://doi.org/10.33024/mahesa.v6i3.20851

ABSTRACT

Obesity is a metabolic condition that poses a global health challenge and is associated with various complications, including reduced testosterone levels. Low testosterone is a critical component in metabolic syndrome and hormonal dysfunction in men. This study aims to analyze the differences in total serum testosterone levels between obese and non-obese male healthcare workers and identify factors influencing low testosterone levels at Prof. dr. I.G.N.G. Ngoerah General Hospital, Denpasar. This cross-sectional study involved 89 male healthcare workers grouped based on body mass index (BMI): obese (BMI ≥ 25 kg/m^2) and non-obese (BMI < 25 kg/m^2). Total serum testosterone levels were measured using the Enzyme-Linked Immunosorbent Assay (ELISA) method. Statistical analysis included bivariate tests using Chi-Square and Fisher-Exact tests, followed by multivariate logistic regression to determine the relationship between obesity and low testosterone levels, adjusting for confounding factors such as age, type 2 diabetes mellitus, hypertension, and heart disease. The mean total serum testosterone level was 528.74 ± 265.13 ng/dL. The prevalence of low testosterone was significantly higher in the obese group (82.6%) compared to the non-obese group (17.4%), with an odds ratio (OR) of 13.691 ($p < 0.001$). Multivariate analysis identified obesity as an independent predictor of low testosterone, with an adjusted odds ratio (AOR) of 6.101 (95% CI: 1.531–24.307; $p = 0.010$). Age was also a significant factor, with participants aged ≥30 years having a higher risk of low testosterone compared to younger participants (AOR: 4.961; 95% CI: 1.118–22.004; $p = 0.035$). Type 2 diabetes mellitus was strongly associated with low testosterone levels (OR: 22.909; $p < 0.001$) but was not significant in the multivariate analysis ($p = 0.060$). Obesity is a significant risk factor for low testosterone levels in male healthcare workers, with age being another key determinant. These findings highlight the importance of early detection and management of obesity to prevent hormonal dysfunction.

Keywords: Obesity, Total Serum Testosterone, Male Healthcare Workers.

INTRODUCTION

Obesity is a global problem that is increasing worldwide. In the last 40 years, it is estimated that obesity has approximately tripled and is predicted to continue to increase. By 2030, it is estimated that 1 in 5 women and 1 in 7 men will be living with obesity. In Indonesia, within 10 years there has been a significant increase in obesity, from

10.5% in 2007 to 21.8% in 2018 (Bluher, 2020).

According to the World Health Organization (WHO) International, obesity is defined as a BMI greater than or equal to 30 kg/m2. However, the definition of obesity in Asian populations is different. According to the Asian Obesity criteria, BMI is defined as greater than or equal to 25 kg/m2. Obesity is closely associated with metabolic diseases such as glucose intolerance, type 2 diabetes mellitus, dyslipidemia, hypertension, non-alcoholic liver disease (NFLD), increased inflammation, and metabolic syndrome, increasing the risk of cardiovascular disease as well as reproductive system disorders in men. Obesity is defined as a condition with abnormal or excessive accumulation of fat in adipose tissue that can interfere with health. One of the pathogenesis of obesity is the occurrence of a proinflammatory state that causes an increase in adipokines, free fatty acids and estrogen from adipose tissue. This increase in pro-inflammatory cytokines is an important risk factor that can contribute to the development of metabolic syndrome and deficiency in testosterone levels (Bluher, 2020).

The pathogenesis of a decrease in total testosterone in obese men is caused by excess fat tissue, which increases the activity of the aromatase enzyme that converts testosterone to estradiol (estrogen hormone). This increase in estrogen levels can provide negative feedback on the hypothalamic-pituitary axis, reducing the secretion of luteinizing hormone (LH) necessary for testosterone production in the testes. As a result, there is a decrease in total testosterone levels in the blood circulation (Rosner et al., 2007).

Testosterone has many functions especially in secondary sex development, building body muscles, increasing bone matrix and calcium retention, increasing basal metabolism, triggering spermatogenesis and playing a role in other sexual functions. Low testosterone levels not only affect infertility but are also considered a marker of mortality and morbidity. The decline in testosterone levels begins to occur at the age of 40 years and becomes more pronounced with increasing age. In addition to age, other factors that can cause a decrease in testosterone levels are obesity, a history of smoking and a history of chronic diseases such as type 2 diabetes mellitus, heart disease, and hypertension (Fernandez Miro et al., 2016).

The relationship between obesity and decreased testosterone levels in men is complex and bidirectional. Low testosterone levels predict central obesity and are associated with an increased risk of metabolic syndrome and type 2 diabetes mellitus. Conversely, obesity predicts low total testosterone levels in men. Several mechanisms that disrupt the hypothalamic pituitary gonadal axis are thought to be responsible for low testosterone production in obese men (Wang et al., 2011).

LITERATURE REVIEW

Obesity

Obesity is an excessive and abnormal accumulation of fat in adipose tissue that can be detrimental to health. According to the WHO, obesity is defined as a BMI greater than or equal to 30 kg/m2. According to Asian obesity criteria, BMI is defined as greater than or equal to 25 kg/m2. Obesity occurs due to excess energy stored in the form of fat tissue. This energy

balance disorder can be caused by exogenous factors (primary obesity) as a result of nutrition (90%) and endogenous factors (secondary obesity) due to hormonal abnormalities, syndromes or genetic defects (covering 10%). Regulation of energy balance is played by the hypothalamus through 3 physiological processes, namely: control of hunger and satiety, influencing the rate of energy expenditure, and regulation of hormone secretion (Alexandrova et al., 2020).

Genetic factors can affect many signaling molecules and receptors used by parts of the hypothalamus and gastrointestinal tract to regulate food intake. Genetic factors also regulate energy expenditure including basal metabolic rate, diet-induced thermogenesis and nonvoluntary activity-related thermogenesis. Genetic factors may have a greater effect on body fat distribution, particularly abdominal fat. Obesity during childhood usually persists into adulthood as well. There is an association between being obese during adolescence and being severely obese in adulthood. Studies show that a significant amount of adolescent obesity occurs before the age of 5 years (Mehrzad R, 2020).

In women, there is an association between higher adult BMI and earlier onset of puberty, although this weight gain can be triggered by various factors such as menopause or pregnancy. At menopause, fat distribution in women changes with fat accumulation in the subcutaneous tissue, especially in the gluteal and femoral regions. Estrogen exerts beneficial effects on adipose tissue distribution and glucose metabolism. However, declining estrogen levels and relative hyperandrogeneia are the basis for the development of obesity with fat redistribution during menopause. In men there is an increase in body weight up to the sixth decade. However, after the age of 55-64, weight gain remains largely stable and then begins to decline. This is due to a decrease in serum testosterone levels in men leading to a decrease in muscle mass and an increase in fat mass in older men (Mehrzad R, 2020).

Testosterone

Testosterone is a male steroid sex hormone (androgen) formed from cholesterol. The three androgens important for male reproductive function are testosterone, dehydrotestosterone and estradiol. In terms of quantity, testosterone is the most important androgen. Almost 95% of testosterone is produced by Leydig cells (interstitial cells) in the testes and the rest comes from the adrenals (Weinbauer et al., 2010). Testosterone is responsible for primary sexual development, which includes testicular desensus, spermatogenesis, penile and testicular enlargement, and increased libido. In addition, testosterone causes the development of male secondary sexual characteristics, beginning at puberty and ending at adulthood. These secondary sex characteristics include male hair patterns, voice changes and deepening, and increased sebaceous gland secretion (McBride et al., 2015). In general, the male body produces 40-60 times more testosterone than women. Testosterone also exists in the form of dehydrotestosterone and androstenedione. Dehydro testosterone and androstenedione are weak forms of androgens. Both testicular and adernal androgens can be formed from cholesterol or directly from acetyl coenzyme A (Guyton and Hall, 2011).

Testosterone can be measured through several laboratory techniques, including imonoassay, high performance gas or liquid chromatography. Each technique has advantages and disadvantages. Which technique is chosen depends on the laboratory instruments, cost, ability and skills of the laboratory personnel. Immunoassay is a technique that is often used both clinically and in research, although the gold standard is chromatography. Testosterone in the body is known in 3 forms, namely those that bind to SHBG, those that bind to albumin, and those that circulate freely as free testosterone, which is the most active testosterone to reach target organs. Measurement of testosterone in the body can be done by measuring all three in the form of (1) total testosterone, (2) free testosterone only, or (3) measurement of calculated free testosterone, namely by using the formula from Vermeulen based on total testosterone, SHBG, and albumin. It is also possible to estimate free testosterone from total testosterone and SHBG (also known as Free Testosterone Index/FTI or Free Androgen Index/FAI). Measurement of free testosterone can be done by ammoniumsulfate precipitation and dialysis techniques which are non-automated, time-consuming, and expensive examinations, therefore not routinely carried out in the laboratory (Pangkahila, 2006). Examination of total testosterone levels can be done by ELISA (Enzyme Linked Immunosorbent Assay) method. Blood is taken from the vein then put into a tube and centrifuged at 3500 rpm for 10 minutes. The blood serum was then used for analysis of testosterone hormone levels using a testosterone hormone kit. Measurement of testosterone hormone levels is usually done in the morning because testosterone hormone increases in the morning by 30% and shows the lowest point at 18.00 to 22.00. Normal levels of free testosterone average 700 ng/dl with a range of 300-1100 ng/dl, while the Free Androgen Index (FAI) ranges from 70-100%. The Endocrine Society USA consensus (2005) recommends normal total testosterone levels are ≥ 200-300 ng/dL. The American Urological Association (AUA) in 2018 recommends low serum total testosterone levels when < 300 ng/dl (Shores et al., 2012).

Low testosterone levels are known as hypotestosterone. This will cause clinical manifestations not only in the reproductive field, but also in the metabolic and endocrine fields. Based on the cause, decreased testosterone levels are divided into primary or testicular hypofunction caused by obesity and diabetes mellitus. Secondary causes or pituitary hypofunction, caused by the decay of two organs and can affect each other such as infection, trauma to the pituitary gland, bleeding and brain tumors (Hisasue, 2015).

Relationship Between Low Testosterone Levels And Obesity

The fact that obese men have lower testosterone than those of normal weight has been known for over 30 years. The relationship between obesity and low testosterone levels is thought to be bidirectional. This occurs because of age-related testicular dysfunction, which is at least partially compensated by increased pituitary LH secretion. Obesity causes hypothalamic-pituitary suppression regardless of age that cannot be compensated by physiological mechanisms (Fui et al., 2014).

Obesity is also associated with an increased release of proinflammatory cytokines

especially macrophages that proceed from adipose tissue such as tumor necrosis factor α (TNF α), IL-6, and IL-8. In a study, it was shown that TNF α and IL-6 are able to suppress the secretion of GnRH and LH, thereby reducing peripheral testosterone secretion (Kurniawan, 2021). So the research is interested in examining whether there are differences in serum total testosterone levels in obese and non-obese male health workers. This study aim to determine the relationship between serum total testosterone levels in obese and non-obese male health workers.

RESEARCH METHOD

This study was an analytical observational study with a cross sectional study approach conducted at Prof. Dr. I.G.N.G Ngoerah Denpasar Hospital from August to September 2024. Sampling was done with non probability sampling technique with consecutive sampling technique.

The independent variables in this study were weight, height, BMI, type 2 diabetes mellitus, hypertension, and heart disease. The dependent variable in this study was serum total testosterone level. The confounding variables in this study were men with cholesterol-lowering treatment, men with testosterone replacement treatment, malignancy or history of chemotherapy.

The final results of this analysis are presented in tabular form, where the results of the normality test, Chi-Square or Fisher Exact test, and Binary Logistic Regression are reported systematically. Results that had a p value <0.05 were considered statistically significant, indicating a meaningful association or influence between the variables analyzed.

RESEARCH RESULTS

A total of 89 patients were obtained as study participants. The mean age of the participants was 30.29 ± 4.918 years, with 43.8% (39 participants) aged more than 30 years and 56.2% (50 participants) aged less than 30 years. The mean body weight was 75.95 ± 14.980 kg, while the mean height was 172.39 ± 6.295 cm. Overall, the mean body mass index (BMI) was 25.53 ± 4.760, with 40.4% (36 participants) classified as obese (BMI ≥25) and 59.6% (53 participants) non-obese (BMI <25). The overall mean waist circumference was 96.16 ± 8.375 cm, while the mean hip circumference was 38.53 ± 2.869 cm. The mean waist-to- hip ratio (WHR) was 2.50 ± 0.133, with a mean testosterone level of 528.74 ± 265.130 ng/dL, of which 25.8% (23 participants) had low testosterone levels and 74.2% (66 participants) had normal levels. In addition, 16.9% (15 participants) had type 2 diabetes mellitus, 9% (8 participants) had hypertension, and 3.4% (3 participants) had heart disease, while the majority of participants had no history of related diseases or comorbidities.

Tabel 1. Demographic Characteristics Of The Study Sample (N=89)

Variable	N (%)	Mean ± SD
Mean (Mean ± SD) (year)		30.29 ± 4.918
≥30 years	39 (43,8%)	
<30 years	50 (56.2%)	
BW (Mean ± SD) (kg)		75.95 ± 14.980
BH (Mean ± SD) (cm)		172.39 ± 6.295
BMI (Mean ± SD)		25.53 ± 4.760
Obese (BMI ≥25)	36 (40,4%)	
Non obese (BMI <25)	53 (59,6%)	
Testosterone (Mean ± SD) (ug/mL)		528.74 ± 265.130
Low	23 (25,8%)	
Normal	66 (74,2%)	
Diabetes Mellitus		
Yes	15 (16,19%)	
No	74 (83,41%)	
Hipertension		
Yes	8 (9%)	
No	81 (91%)	
Heart disease		
Yes	3 (3,4%)	
No	86 (96,6%)	
Total	**89 (100%)**	

Bivariate comparison analysis to determine the relationship between variables found that participants with obesity had a prevalence of low testosterone of 82.6% compared to 17.4% in non-obese participants, with an odds ratio (OR) of 13.691 and p value <0.001. By age group, 87.0% of participants aged more than equal to 30 years had low testosterone compared to 13.0% of those aged below 30 years, with an OR of 16.491 and p value <0.001. Type 2 diabetes mellitus also showed a significant association, where 52.2% of participants with type 2 diabetes mellitus had low testosterone compared to 47.8% of those without type 2 diabetes mellitus, with OR 22.909 and p value <0.001. In addition, hypertensive disease showed a prevalence of low testosterone of 21.7% in participants with hypertension, compared to 4.5% in those without, with an OR of 5.833 and a p value of 0.025. In contrast, the association between heart disease and low testosterone was not statistically significant with OR 6.190 and p value 0.163. The results of the overall analysis are outlined in **(Table 2)**

Table 2. Relationship between Subject Characteristics and Low Testosterone Level

Variable		Testosterone (N (%)		OR	P value
		Low	Normal		
Obesity	Yes	19 (82.6)	17 (25.8)	13.691	<0.001*[a]
	No	4 (17.4)	49 (74.2)		
Age	≥30 years	20 (87.0)	19 (28.8)	16.491	<0.001*[a]
	<30 years	3 (13.0)	47 (71.2)		
Type 2 Diabetes Melitus	Yes	12 (52.2)	3 (4.5)	22.909	<0.001*[b]
	No	11 (47.8)	63 (95.5)		
Hipertension	Yes	5 (21.7)	3 (4.5)	5.833	0.025*[b]
	No	18 (78.3)	63 (95.5)		
Heart disease	Yes	2 (8.7)	1 (1.5)	6.190	0.163[b]
	No	21 (91.3)	65 (98.5)		

*Statistically significant (p<0.05); aChi-Square test; bFisher-Exact test

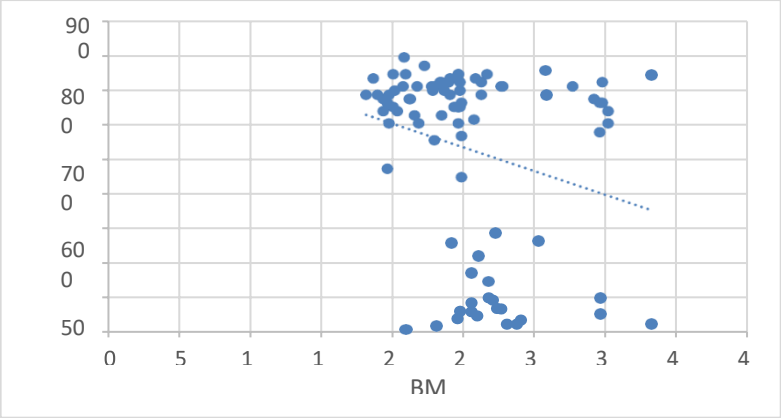

Figure 1. Scatter Plot Between Serum Total Testosterone Levels And Obese And Non-Obese Men.

Scatter Plot shows a negative relationship between BMI and total testosterone levels. The trend line (regression) with a negative slope indicates that the higher a person's BMI, the lower the testosterone levels tend to be.

Multivariate analysis was conducted to determine the association of obesity with low testosterone levels after adjusting for other variables. Based on the analysis, obesity was a significant predictor of low testosterone levels, with an adjusted odds ratio (AOR) of 6.101 (CI95%: 1.531-24.307) and a p value of 0.010. In addition, age greater than equal to 30 years also showed a significant association, with an AOR of 4.961 (CI95%: 1.118-22.004) and a p value of 0.035. In contrast, type 2 diabetes mellitus had an AOR of 6.798 (95% CI: 0.924-50.025), but this association was not statistically significant (p value 0.060). Similarly, hypertension (AOR: 0.269; 95% CI: 0.023-3.168; p value 0.297) and heart disease (AOR: 0.288; 95% CI: 0.012-6.940; p value 0.443), both of which showed no

significant association. The overall analysis results are shown in **(Table 3).**

Table 3. The Effect of Obesity and Other Risk Factors on Low Testosterone Levels

Variable	B	Wald	*Adjusted* OR	CI95%	P value
Obesity	1.808	6.576	6.101	1.531 – 24.307	0.010*
Age ≥30 years	1.602	4.441	4.961	1.118 – 22.004	0.035*
Type 2 Diabetes Mellitus	1.917	3.542	6.798	0.924 – 50.025	0.060
Hipertension	-1.314	1.090	0.269	0.023 – 3.168	0.297
Heart disease	-1.246	0.588	0.288	0.012 – 6.940	0.443

*Statistically significant (p<0.05) by Binary-logistic regression test

DISCUSSION

Total Testosterone Levels in Obese and Non-Obesity Men

This study showed a significant association between obesity and serum total testosterone levels in male health workers. Based on the analysis, the prevalence of low testosterone levels was substantially higher in the obese male group compared to the non-obese male group. A total of 82.6% of obese men had low testosterone levels, while only 17.4% of non-obese men experienced the same. This difference was statistically significant, with a p value of <0.001 and an odds ratio (OR) of 13.691. Thus, obese men had a 13.7 times greater risk of having low testosterone levels compared to non-obese men. This finding confirms the role of obesity as a major risk factor for hormonal dysfunction in men (Kruljac et al., 2020).

The results of the multivariate analysis reinforced these findings, with obesity remaining an independent predictor of low testosterone levels even after adjusting for other variables such as age, type 2 diabetes mellitus, hypertension and heart disease. The adjusted odds ratio (AOR) of 6.101 (95% CI: 1.531- 24.307; p = 0.010) indicated that obesity significantly increased the risk of testosterone deficiency. Age was also found to have a significant association, with men aged more than equal to 30 years having a higher risk of testosterone deficiency.

Additionally, systemic inflammation commonly associated with obesity plays a significant role. Adipose tissue in obese individuals produces pro-inflammatory adipokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), which are known to inhibit GnRH (gonadotropin-releasing hormone) production in the hypothalamus, thereby reducing LH and testosterone secretion. This inflammation can also affect insulin sensitivity, which in turn suppresses testosterone synthesis through the

insulin resistance pathway (Dandona et al., 2020; Fernandez et al., 2019).

In this study, results showed that obese men had a prevalence of low testosterone of 82.6% compared to only 17.4% in non-obese men. Statistical analysis revealed a significant association between obesity and low testosterone levels with an OR of 13.691 ($p < 0.001$). This association remained significant after multivariate adjustment, with an adjusted OR (AOR) of 6.101 (95% CI: 1.531–24.307; $p = 0.010$), reinforcing that obesity is an independent predictor of hypogonadism (Fernandez et al., 2019).

Clinically, the implications of low testosterone in obese men are extensive, including increased risk of metabolic syndrome, type 2 diabetes mellitus, and cardiovascular disease. This condition is also associated with reduced quality of life, sexual performance, and muscle mass (Chooi et al., 2018; Erenpreiss et al., 2019; Dandona et al., 2020). Therefore, early detection of low testosterone levels in obese men, as conducted in this study, is crucial for determining appropriate interventions to reduce the risk of these comorbidities (Diaz-Arjonilla et al., 2008; Dandona et al., 2020).

Factors Affecting Testosterone Levels

Age

The physiological decline in testosterone levels that occurs with age is a consequence of changes in the hypothalamic-pituitary-testicular axis. Testosterone production by Leydig cells in the testes decreases due to a decrease in sensitivity to LH stimulation, which is influenced by a decrease in GnRH secretion in the hypothalamus. Additionally, increased SHBG levels in older age reduce the amount of free testosterone available for use by target tissues (Barone et al., 2022).

Decreased testosterone is also associated with changes in the diurnal rhythm of hormone secretion, which decreases in intensity with age. This condition leads to late-onset hypogonadism characterized by symptoms such as decreased libido, muscle weakness, decreased bone mass, and chronic fatigue (Masliukov & Nozdrachev, 2021).

Diabetes Mellitus

Insulin resistance is a key characteristic of type 2 diabetes mellitus (DM2) that directly and indirectly affects testosterone production. This study found that men with a history of diabetes mellitus had a prevalence of low testosterone of 52.2% compared to only 4.5% in men without a history of diabetes (OR 22.909; $p < 0.001$). This indicates that men with diabetes mellitus have over a 22-fold increased risk of having low testosterone levels. Insulin resistance increases the production of pro-inflammatory cytokines such as TNF-α and IL-6, which can inhibit the hypothalamic-pituitary-testicular axis by reducing GnRH secretion. As a result, stimulation of testosterone production by Leydig cells is significantly reduced. In a longitudinal study, low baseline testosterone levels were found to be associated with a significant increase in insulin resistance after 10 years, with $\beta = -0.096$ ($p = 0.006$), supporting a bidirectional relationship between insulin resistance and testosterone levels (Ottarsdottir et al., 2018).

Diabetes mellitus is also associated with increased oxidative stress that damages Leydig cell function in the testes. Leydig cells, which are responsible for testosterone production, are highly

susceptible to oxidative damage caused by reactive oxygen species (ROS). In chronic hyperglycemia, excessive ROS leads to Leydig cell apoptosis and impaired testosterone biosynthesis (Leisegang et al., 2021).

Hypertension and Heart Disease

Hypertension, a major risk factor for cardiovascular disease, has a complex relationship with testosterone levels. Several studies have shown that hypertension can affect the hypothalamic-pituitary-testicular axis through oxidative stress and activation of the renin-angiotensin-aldosterone system (RAAS). RAAS activation can disrupt GnRH release, thereby reducing testosterone production by Leydig cells in the testes. Heart disease has a complex relationship with testosterone levels. Several studies have shown that testosterone deficiency can worsen the metabolic profile, increase insulin resistance, and worsen endothelial dysfunction, all of which can increase the risk of cardiovascular disease (Colafella & Denton, 2018).

Implications of Obesity on Testosterone Levels

This study shows that low testosterone levels in obese men have significant consequences for reproductive and metabolic health. Low testosterone levels are often associated with reproductive disorders such as infertility. This occurs through a mechanism of spermatogenesis inhibition due to changes in the hypothalamic-pituitary-testicular axis and decreased testosterone secretion by Leydig cells. Obesity can increase estradiol production through high aromatase activity in adipose tissue, which ultimately inhibits gonadotropin and testosterone secretion.

Furthermore, low testosterone levels are also associated with an increased risk of metabolic syndrome, which involves hypertension, hyperglycemia, dyslipidemia, and central obesity. Low testosterone levels are known to worsen insulin resistance and endothelial dysfunction, which are key mechanisms in the development of cardiovascular disease. These effects suggest that testosterone deficiency not only compromises reproductive function but also systemically increases the risk of various degenerative diseases. (Elagizi et al., 2018).

The findings of this study highlight the importance of early detection of low testosterone levels, particularly in obese men. In this study population, obese men had a prevalence of low testosterone levels of 82.6%, significantly higher than non-obese men (17.4%). These data underscore the importance of routine hormonal screening to prevent the long-term effects of undiagnosed hypogonadism. Hormonal screening should be part of the examination protocol for high-risk populations such as healthcare workers, who often face irregular work patterns and high stress levels, factors that can worsen their hormonal status (Kruljac et al., 2019).

Obesity, in addition to being an independent risk factor, also interacts with other factors such as age and metabolic comorbidities. Leisegang et al. (2020) emphasize that weight loss combined with pharmacological therapy can significantly increase testosterone levels and reduce the risk of infertility. Therefore, managing obesity through a holistic approach, including lifestyle changes, medical therapy, and nutritional interventions, is highly relevant in this context.

Healthcare workers are a group at high risk of obesity due to irregular work patterns, lack of physical activity, and high-calorie diets under high work stress conditions. This study highlights the need for workplace health interventions targeting weight management and hormonal monitoring in this group. Early intervention can prevent the development of further metabolic complications and improve quality of life.

Given the high prevalence of obesity in this population, preventive measures such as promoting physical fitness, easy access to sports facilities, and nutritional counseling should be integrated into occupational health programs. Early detection and management of obesity among healthcare workers not only contribute to improved individual health but also support their productivity and work effectiveness (Mulhall et al. (2018).

CONCLUSION

There is a difference in total serum testosterone levels between obese and non-obese male health workers. Obesity and age ≥ 30 years are significant predictors of low total serum testosterone levels.

REFERENCES

Aleksandrova, K., Egea Rodrigues, C., Floegel, A., & Ahrens, W. (2020). Omics Biomarkers in Obesity: Novel Etiological Insights and Targets for Precision Prevention. In Current obesity reports (Vol. 9, Issue 3, pp. 219–230). NLM (Medline). https://doi.org/10.1007/s13679-020-00393-y

Barone, B., Napolitano, L., Abate, M., Cirillo, L., Reccia, P., Passaro, F., Turco, C., Morra, S., Mastrangelo, F., Scarpato, A., et al., 2022. The Role of Testosterone in the Elderly: What Do We Know? International Journal of Molecular Sciences, 23(7), p.3535. DOI: 10.3390/ijms23073535.

Blüher, M. (2020). Metabolically healthy obesity. In Endocrine Reviews (Vol. 41, Issue 3, pp. 405–420). Endocrine Society. https://doi.org/10.1210/endrev/bnaa004

Chooi, Y.C., Ding, C., & Magkos, F., 2018. The epidemiology of obesity. Metabolism, [online] Available at: https://doi.org/10.1016/j.metabol.2018.09.005 [Accessed 6 December 2024].

Colafella, K.M., & Denton, K.M., 2018. Sex-specific differences in hypertension and associated cardiovascular disease. Nature Reviews Nephrology, 14, pp.185-202. DOI: 10.1038/nrneph.2017.189.

Dandona, P., Dhindsa, S., Ghanim, H., Saad, F., & Batra, M., 2020. Mechanisms underlying the metabolic actions of testosterone in the human: A narrative review. Diabetes, Obesity and Metabolism. DOI: 10.1111/dom.14206.

Diaz-Arjonilla, M., Schwarcz, M., Swerdloff, R.S., & Wang, C., 2008. Obesity, low testosterone levels and erectile dysfunction. International Journal of Impotence Research, 21, pp.89-98. DOI: 10.1038/ijir.2008.42.

Elagizi, A., Köhler, T.S., & Lavie, C.J., 2018. Testosterone and

Cardiovascular Health. Mayo Clinic Proceedings, 93(1), pp.83-100. DOI: 10.1016/j.mayocp.2017.11.006.

Erenpreiss, J., Fodina, V., Pozarska, R., Zubkova, K., Dudorova, A., & Pozarskis, A., 2019. Prevalence of testosterone deficiency among aging men with and without morbidities. The Aging Male. DOI: 10.1080/13685538.2019.1621832.

Fernández-Miró, M., Chillarón, J. J., & Pedro-Botet, J. (2016). Testosterone deficiency, metabolic syndrome, and diabetes mellitus. In Medicina Clinica (Vol. 146, Issue 2, pp. 69–73). Edicione, Doyma, S.L. https://doi.org/10.1016/j.medcli.2015.06.020

Fui, M. N. T., Dupuis, P., & Grossmann, M. (2014). Lowered testosterone in male obesity: Mechanisms, morbidity and management. In Asian Journal of Andrology (Vol. 16, Issue 2, pp. 223–231). https://doi.org/10.4103/1008-682X.122365

Guyton, A. C. and Hall, J. E. (2011) Reproductive and hormonal functions of the male. In textbook of medical th physiology. Philadelphia, Saunders: Elsevier.

Hisasue, S. ichi. (2015). Contemporary perspective and management of testosterone deficiency: Modifiable factors and variable management. In International Journal of Urology (Vol. 22, Issue 12, pp. 1084–1095). Blackwell Publishing. https://doi.org/10.1111/iju.12880

https://doi.org/10.1001/jamanetworkopen.2023.2145

Kruljac, M., Finnbogadóttir, H., Bobjer, J., Giraldi, A., Fugl-Meyer, K., & Giwercman, A., 2020. Symptoms of sexual dysfunction among men from infertile couples: prevalence and association with testosterone deficiency. Andrology, 8, pp.160-165. DOI: 10.1111/andr.12678.

Kurniawan, L. B. (2021) "Hypotestosterone in Male with Obesity," Indonesian Journal of Clinical Pathology and Medical Laboratory, 27(2), pp. 217–223. doi: 10.24293/ijcpml.v27i2.1525.

Leisegang, K., Roychoudhury, S., Slama, P., & Finelli, R., 2021. The Mechanisms and Management of Age-Related Oxidative Stress in Male Hypogonadism Associated with Non-communicable Chronic Disease. Antioxidants, 10(11), p.1834. DOI: 10.3390/antiox10111834

Masliukov, P. and Nozdrachev, A.D., 2021. Hypothalamic Regulatory Mechanisms of Aging. Journal of Evolutionary Biochemistry and Physiology, 57(3), pp.473-491. DOI: 10.1134/S0022093021030030.

Mehrzad, R. 2020. Global Impact and Epidemiology Obesity First Edition. Cambridge : Elsevier.

Mulhall, J.P., Trost, L.W., Brannigan, R.E., Kurtz, E.G., Redmon, J.B., Chiles, K.A., Lightner, D.J., Miner, M.M., Murad, M.H., Nelson, C.J., Platz, E.A., Ramanathan, L.V., & Lewis, R.W., 2018. Evaluation and management of testosterone deficiency: AUA guideline. Journal of Urology, 200(423-432). DOI: 10.1016/j.juro.2018.03.115.

Ottarsdottir, K., Nilsson, A.G., Hellgren, M., Lindblad, U. & Daka, B., 2018. The association between serum testosterone and insulin resistance: a longitudinal study. Endocrine Connections, 7(12), pp.1491–1500. DOI: 10.1530/EC-18-0480.

Pangkahila, W. 2006. Seks yang Membahagiakan. Penerbit Buku, Jakarta.

Pelusi, C., & Pasquali, R. (2012). The Significance of Low Testosterone Levels in Obese Men. In Current Obesity Reports (Vol. 1, Issue 4, pp. 181–190). Current Medicine Group LLC 1. https://doi.org/10.1007/s13679-012-0029-4

Rosner, W., Auchus, R. J., Azziz, R., Sluss, P. M., & Raff, H. (2007). Position statement: Utility, limitations, and pitfalls in measuring testosterone: An endocrine society position statement. Journal of Clinical Endocrinology and Metabolism, 92(2), 405–413. https://doi.org/10.1210/jc.2006-1864

Shores, M. M., Smith, N. L., Forsberg, C. W., Anawalt, B. D., & Matsumoto, A. M. (2012). Testosterone treatment and mortality in men with low testosterone levels. Journal of Clinical Endocrinology and Metabolism, 97(6), 2050–2058. https://doi.org/10.1210/jc.2011-2591

Wang, J. S., Xia, P. F., Ma, M. N., Li, Y., Geng, T. T., Zhang, Y. B., Tu, Z. Z., Jiang, L., Zhou, L. R., Zhang, B. F., Tong, W. W., Shan, Z., Liu, G., Yang, K., & Pan, A. (2023). Trends in the Prevalence of Metabolically Healthy Obesity among US Adults, 1999-2018. JAMA Network Open, 6(3), E232145.

Weinbauer, G. F., Luetjens, C. M., Simoni, M., & Nieschlag, E. (2010). Physiology of testicular function. In Andrology: Male Reproductive Health and Dysfunction (pp. 11–59). Springer Berlin Heidelberg. https://doi.org/10.1007/978-3-540- 78355-8_2