

Main outcome measures Collected reproductive parameters included testosterone (T), sex hormone-binding globulin (SHBG), follicle-stimulating hormone (FSH), and luteinizing hormone (LH) serum hormonal values. A paired t test was done to evaluate differences across the before and after groups. Chi-square/Fisher exact test was used to find the significance of study parameters on a categorical scale between two or more groups. Results The mean BMI was significantly higher before weight loss (33.2) than after weight loss (30.4) in obese men. The weight loss increased the T to 35.40 ± 20.51 ng/mL compared with 27.16 ± 20.71 ng/mL, and SHBG to 23.72 ± 9.01 μ g/dL compared with 19.18 ± 10.44 μ g/dL, whereas FSH and LH were nonsignificant.

Conclusion The study showed that a high BMI at the baseline was associated with low values of serum T and SHBG. FSH and LH were considerably low in morbidly obese men before weight loss. Weight loss was associated with an increase in serum T and SHBG. FSH and LH were not statistically significant after weight loss except in morbidly obese men who showed clear aromatization influence. The hormonal profile among obese men evaluated in this study was characterized by abnormalities in the sex hormones, and weight loss improved some of the hormone levels; however, they were not normalized.

Keywords Body mass index, Male infertility, Male reproductive hormones, Obesity, Weight loss.

International Journal of Infertility and Fetal Medicine (2018): 10.5005/jp-journals-10016-1172

INTRODUCTION

Infertility is defined as the inability to conceive after 1 year of unprotected intercourse. Most of the developed countries are bearing the brunt of high frequency of overweight or obesity.¹ In the developing world, the prevalence of overweight and obesity is increasing at alarming rates,²⁻⁶ and, in some countries, has reached levels observed in the developed world.^{7,8} Although overweight and obesity have been demonstrated to affect female fertility,⁹ there is no consensus on the effects on male fertility and seminal parameters.¹⁰⁻¹² Epidemiologic studies performed at the population level and in infertile couples during recent years suggest that obesity is associated with reduced male fertility, although this effect seems to be modest.¹³ Male obesity has been linked to subfecundity and an effect-response relationship between increasing BMI and subfecundity has been proposed.¹⁴⁻¹⁶ A higher prevalence of obesity in developing countries is associated with the change from rural to urban lifestyle causing decreased levels of physical activity and an increased intake of energy-dense diet. In addition to lifestyle changes, genetics also play a role in increasing obesity in India. India and most of the Asia-Pacific population have a mutation of MC4R genes which put them into the high-risk population.¹⁷ WHO has reclassified the obesity limits for Asians/Indians as indicated in Table 1.¹⁸

Male obesity has been associated with abnormal semen characteristics,¹⁹⁻²⁶ although results are conflicting.²⁷⁻³³ Obesity has shown to affect the gonadotropin releasing hormone-luteinizing hormone/follicle stimulating hormone (GnRH-LH/FSH) pulse that may impair Leydig and Sertoli cell functions and with the release

