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Commentary Open Access

Seeking the Insight of Fertility Specialist in Managing Obesity in Men

Atif A Katib*

Department of Urology/Andrology, King Abdul-Aziz Hospital, Makkah, Saudi Arabia

***Corresponding author:** Atif A. Katib, Clinical research, 88 seneca drive, Ancaster, L9G 3B8, Hamilton, Ontario, Canada, Tel: 9059665237; Fax: 9059665237; E-mail: atifkatib@gmail.com

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Commentary

The author has presented a basic science review surrounding the biological and metabolic derangements that occur in overweight males and its relation to fertility potentials. Infertility, defined as the absence of pregnancy after one year of unprotected intercourse. Male factor infertility constitutes 25 to 30% of all cases of infertility and contributes, in combination with female factors, to another 30%. Reduced semen quality has been found to be a universal trend in the last few decades because of the dramatic changes in the life-style of civilized communities around the world. Alarmingly, one of the health problems in modernized societies that lead to infertility is obesity.

Recent estimates show that the increasing prevalence of obesity is recognized worldwide, with few exceptions. The International Obesity TaskForce estimates that at least 1.1 billion adults are currently overweight, including 312 million who are obese; and that with the new Asian Body Mass Index (BMI) criteria, the number is even higher [1]. Most importantly, there is emerging evidence that overweight is increasing not only in adults, but in children too [1].

Subfertile men have significantly higher BMI than the general population. Overly, an abnormal BMI (less than 19 kg/m^2 or more than 30 kg/m^2) has been associated with reduced testicular volume (WHO 1987), and reduced semen quality suggesting impairment of spermatogenesis (Jensen et al. 2004). Czernichow and his colleagues gathered data from 14 previous studies, including nearly 10,000 men. They looked at each participant's sperm count and BMI, which is a measure of body fatness, and is calculated based on weight and height. Men with BMI of more than 25 kg/m^2 are considered overweight, and those with a BMI exceeding 30 kg/m^2 are considered obese. Interestingly, they found that among men, who were of normal weight, 24 percent had low sperm counts and 2.6 percent had no viable sperms. Moreover, among the overweight men, 25.6 percent had low sperm counts, and 4.7 percent had no viable sperms. Of the men who were obese, 32.4 percent had low sperm counts and 6.9 percent had no viable sperms [2].

The article in focus has tackled the condition "traumatic epididymo-orchitis" to be a novel possible cause of infertility in obese males, and has presented a fairly arguable reasoning. Obese men have redundant lumps of fat at the suprapubic and inner thigh regions. These chunks of fat could cause mechanical inflammation to the scrotal contents, including the testes and epididymi, by rubbing and sheering forces; especially while sitting, walking, jogging and performing various physical activities. Looking at the clinical data, epididymo-orchitis post mumps infection, causes sterility in 7-13% of affected patients; as orchitis affects the testicular interstitium more than the Leydig and Sertoli cells, but sperm counts, mobility, and morphology could be affected. Testicular varicocele, the commonest surgical condition that disturbs the thermoregulation within the testes, can cause infertility too. I support the view that, regardless of the cause of epididymo-orchitis being microbial, mechanical, or thermal; it could cause infertility. Chronic inflammation of the testes and epididymi will affect their functions, changes the environment within the delicate tubules, thus affects sperm maturation, and subsequently fertilization ability. Recurrent inflammation could also cause epididymal duct blockage by scarring and cysts formation. Epididymo-orchitis might occur in one or either sides. Bilateral cases are more likely to compromise male fertility potentials. Urological references have pointed up "conventional" trauma to cause epididymo-orchitis; whereas friction blunt trauma by the fat lumps surrounding the genitalia is yet to have a sound proof. I believe that gaps exist in the prevalence and pathogenesis of epididymitis in obese males.

The process of spermatogenesis is highly sensitive to heat, with optimal temperature ranging between 34-35°C in humans [3]. It could be that elevated temperatures within the scrotum, due to fatter tissue, harm sperm cells. The deleterious effect of heat is associated with reduced sperm motility, increased sperm DNA fragmentation and increased sperm oxidative stress [4-6]. Changes to testicular temperature can occur via a number of mechanisms such as varicoceles, using a laptop computer in a laptop position [7], and immersion in a sauna bath [8]. Increased scrotal adiposity, and the scrotum being surrounded all the way around by suprapubic and thighs' lumps of fat; all are associated with reduced sperm function and subfertility [9-15]. It is therefore, not surprising that increased testicular heat caused by increased adiposity in obese men has been proposed as a possible mechanism. It is noteworthy that increased sperm DNA damage and oxidative stress is commonly noted in obese patients, and that a single study which investigated the surgical removal of scrotal fat reported an improvement in sperm parameters [12]. The prevalence and severity of varicoceles inversely correlated with obesity. The prevalence of varicocele decreases with increasing BMI. One explanation is increased adipose tissue preventing compression of the left renal vein. Another explanation is decreased detection due to adipose tissue in the spermatic cord. The decrease in varicocele prevalence as a function of BMI regardless of varicocele grade suggests this explanation is less likely [16]. Celiktas M, and associates have shown a decreases in varicocele incidence in obese men due to the increased retroperitoneal fat thickness and the adipose tissue deposition within the inguinal cord structures and scrotal content [17]. The present data support the explanation that obesity may result in a decreased nutcracker effect, which accounts for prevention of the renal vein compression by the adipose tissue [18]. Albert O, and associates in 2013, has published an interesting article to point up [19]. It shows the disruptive effect of the three mild anti-inflammatory agents, aspirin, paracetamol, and indomethacin on the testicular function. Obese persons often use them for joint pain relief. In therapeutically effective doses, they appear in the interstitial component of the testes in the same range as those measured in blood

plasma. Therefore, the production of testosterone, inhibin B and insulin-like factor 3 by leydig cells were altered by exposure to these drugs.

A positive part of the article in focus is the reversibility section. As knowledge advances, turning the tide back has become possible when treating obesity pathology. While it is becoming clearer that male obesity has negative impacts on fertility, sperm function and long-term impacts on the health burden of the offspring; It is equally clear that simple interventions such as changes to diet, and exercise can reverse both the disease state and the offspring outcomes. There is emerging evidence that intake of selenium enriched probiotics by obese rodents improves both their metabolic health and fertility measures (sperm count and motility) [20]. Amending BMI status, correcting endocrinopathy, improving metabolic health such as a return of plasma concentrations of glucose, insulin and cholesterol to normal levels result in improvements in sperm motility and morphology; concomitant with improvements to molecular composition such as reductions in oxidative stress and reduced DNA damage [21]. To date, there is little information about the impact of diet and exercise interventions in obese men with regard to semen parameters in the human. The mechanisms accounting for reduced total testosterone levels in obese men are various and are defined within a reversible hypogonadotropic hypogonadism pathway [22].

Research run against each other's surrounding the effect of bariatric procedures on semen quality. Reis LO, and associates show that weight loss surgeries don't show to improve sperm quality, but they do ameliorate the quality of life of sexual function. Moreover, they increase the total and free testosterone, and FSH; while they decrease prolactin levels [23]. On the contrary, Sermondade N, and associates, in 2012, revised three case series and found a severe worsening of semen parameters during the months following bariatric surgery, including extreme oligoasthenoteratozoospermia. Interestingly, they noticed a recovery in spermatogenesis in one of the series [24]. The author has produced a developed flowchart showing how obesity factors are linked to infertility [25].

In conclusion, there is substantial evidence elaborated in the original treatise that male obesity has negative impacts on fertility through changes at hormonal levels; as well as direct changes to sperm function and sperm molecular composition. Reversibility is a thrilling topic; as enthusiastic overweight individuals could turn the tide back, and have their condition improved. Imposing a fertility specialist in the obesity-management team will expand our knowledge in this promising field, and will extend the team`s care to cover a crucial aspect of the patient`s life, that is fertility and sexuality. The patient would be aware of the odds of being infertile after undergoing a given bariatric procedure. Furthermore, in case the procedure, in the future, turned to carry a significant risk on spermatogenesis, the fertility specialist might be having the enough evidence to offer the patient sperm-banking beforehand.

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