*Review Article*

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# MALE INFERTILITY AND SEMEN PARAMETERS: A DEMYSTIFIED REVIEW

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## ABSTRACT

Infertility and problems of impaired fecundity represents a significant social and medical problem, affecting 8–12% of couples worldwide. Nearly 40–50% of infertility can be ascribed to the males, amongst which, nearly 2% men suffer from suboptimal sperm parameters. Abnormalities of the sperm parameters affect one or a combination of low sperm concentration, poor sperm motility, or abnormal morphology. Infertility has a strong social taboo in developing countries like ours often leading to psychological distress within the family and the society. According to the International Committee for Monitoring Assisted Reproductive Technology, World Health Organization (WHO), infertility is defined as a disease characterized by a failure to achieve a clinical pregnancy after regular unprotected sexual intercourse of 12 months or more. It can also be defined as failure of a couple to conceive after 12 months of regular intercourse without the use of contraception in women <35 years; and after 6 months of regular intercourse without the use of contraception in women ≥35 years. Male infertility refers to a male's inability to impregnate a fertile female. "Male factor" infertility is diagnosed as an alteration in the sperm concentration and/or motility and/or morphology in at least one of the two samples, collected 1 to 4 weeks apart. This review article is intended to highlight the semen abnormalities and associated factors in infertility.

**KEYWORDS:** Male infertility, Sperm parameters, Fertility issues.

## INTRODUCTION

Infertility is a disease characterized by a failure to achieve a clinical pregnancy after 12 months or more of regular unprotected sexual intercourse (1-5).

Reliable global prevalence related data on infertility is lacking, (6) but it is estimated that as high as 72million couples experience fertility problems globally (7-8). In terms of proportion, nearly 8-12% of couples worldwide is estimated to suffer from infertility (9-10). For obvious reasons, countries with high fertility rates tend to have a higher incidence of infertility and vice versa (11). If we look at the more developed countries like the United States, approximately 10% of the couples are affected with infertility (9-10). According to National Center for Health Statistics, the absolute numbers of impaired fecundity increased by about 2.7 million women, from 1982 to 2002 before falling marginally between 2006 and 2010. In the same vein, the incidence of infertility in younger male (< 30 years of age) has also decreased worldwide by 15% (12-15).

A breakup of the infertility cases reveals that the etiology is symmetrically distributed, with 40% cases related to men, 40% related to women and the remaining 20% being ascribable to both (16). According to a multicentric study conducted by WHO which accrued patients from 1982 to 1985, only 20% of cases could be attributed to male factors while 38% were female related, 27% cases affected both partners, and in about 15% of the cases, the cause could not be satisfactorily elucidated (17). To make matters worse, over one fourth of these cases do not reveal themselves on routine tests, often described as unexplained infertility (18). The study by Ahmad et al brought our attention to the fact that an abnormal semen quality pertaining to the sperm quantity, quality, and bacterial infection could be a an important cause of male infertility (19).

Semen analysis forms the cornerstone in the investigation of infertility. The key advantages of a semen analysis lies include an ease of testing and the outpatient nature of the procedure. The parameters

examined in a semen sample include volume, pH, sperm concentration, motility, morphology, and vitality of the spermatozoa (19).

Defects in the semen quality and quantity are one of the major findings in semen analysis in male infertility. Azoospermia, which means the absence of spermatozoa in the semen ejaculate, and oligospermia, where the sperm count is <15 million/ml are often detected in these cases (1). Moreover, male suffering from oligospermia and infertility often show an evidence of a microbial infection in the seminal fluid, the exact significance of which still remains unelucidated. Nevertheless, a bacterial infection related infertility often result in a decrease in the sperm quantity and motility. Sexually transmitted infections like gonorrhea (caused by *Neisseria gonorrhea*) and sometimes a retrograde infection of an urinary tract infection, frequently caused by staphylococcus aureus, can infect and adverse damage the male sexual apparatus like the testicles, epididymis, and often impairing the production of testicular hormones (19).

Altered consistency of the semen, where the semen sample is either too thick or too thin is another abnormal finding in seminal analysis in such patients. Often such findings correlate with lower sperm counts (20). As far as the relationship of duration between sexual activity and sperm parameters goes, prolonged abstinence often leads to an increased sperm concentration while a shorter period of abstinence leads to an improved sperm motility albeit with a lower sperm density. However, sexual abstinence does not affect the sperm morphology as such. It has been observed widely that other rare sperm defects like the asthenozoospermia and teratozoospermia are significantly more commonly encountered in oligospermic semen than in the normospermic semen (19).

**Normal semen parameters**

As mentioned earlier, semen analysis remains the cornerstone in the investigation of male infertility (21). However, one must ensure a consistently high standard of quality in such examinations to ensure that the results truly reflect the disease (22-23). One must also remember that semen analysis is only a screening tool and useful only in the initial evaluation of an infertile male, and for obvious reasons, cannot decipher the other causes of male infertility (24). Moreover, semen analysis is insufficient in understanding the functional potential of spermatozoon to undergo a subsequent maturation processes in order to achieve fertilization. Therefore, semen analysis has got some limitations (25-27).

The WHO has revised the lower reference limits of various parameters for semen analyses. From a study conducted on 1900 men who were fertile (as defined by impregnation of their female partners with a time-to-pregnancy of ≤12 months), the following inferences were made (the findings were within the 95% confidence interval, as depicted below): (28).

- Volume: 1.5 mL (95% CI: 1.4–1.7)
- Sperm concentration: 15 million spermatozoa/mL (95% CI: 12–16)
- Total sperm number: 39 million spermatozoa per ejaculate (95% CI: 33–46)
- Morphology: 4% normal forms (95% CI: 3–4), using a "strict" Tygerberg method (23)
- Vitality: 58% live (95% CI: 55–63)
- Progressive motility: 32% (95% CI: 31–34)
- Total (progressive + nonprogressive motility): 40% (95% CI: 38–42).

Sperm abnormalities are a critical factor in male infertility. These abnormalities are defined as follows (Table 1):

| Parameter | Lower reference limit |
|---|---|
| Semen volume (ml) | 1.5 (1.4-1.7) |
| Total sperm number ($10^6$ per ejaculate) | 39 (33-46) |
| Sperm concentration ($10^6$ per ml) | 15 (12-16) |
| Total motility (PR+ NP, %) | 40 (38-42) |
| Progressive motility (PR, %) | 32 (31-34) |
| Vitality (live spermatozoa, %) | 58 (55-63) |
| Sperm morphology (normal forms, %) | 4 (3.0-4.0) |
| Other consensus threshold values | |
| pH | ≥7.2 |

***Table 1: Lower Reference Limits for Semen (WHO 2010)***

| | |
|---|---|
| Peroxidase-positive leukocytes ($10^6$ per ml) | <1.0 |
| MAR test (motile spermatozoa with bound particles, %) | <50 |
| Immunobead test (motile spermatozoa with bound beads, %) | <50 |
| Seminal zinc (μ mol/ejaculate) | ≥2.4 |
| Seminal fructose (μ mol/ejaculate) | ≥13 |
| Seminal neutral glucosidase (mU/ejaculate) | ≥20 |

***Cont. Table 1: Lower Reference Limits for Semen (WHO 2010)***

| | **WHO 1999** | **WHO 2010** | **Nomenclature if below cut-off value** |
|---|---|---|---|
| Volume | 2 ml | 1.5 ml | Hypospermia* |
| Sperm concentration | 20 x $10^6$ spermatozoa/ ml | 15 x $10^6$ spermatozoa/ ml | Oligozoospermia** |
| Motility (A+ B)*** | 50% | 32% | Asthenozoospermia |
| Morphology | 30% normally formed | 4% normally formed**** | Teratozoospermia |

***Table 2: The Cut-on Values of Sperm Parameters as per the WHO 1999 and 2010 Criteria and Nomenclature***

*No ejaculate is aspermia,
**If there are no spermatozoa in the ejaculate then it is called azoospermia,
***A-motility is fast forward progressive, 13-motility is slow progressive.
****According to the Tygerberg criteria (Kurger et al., 1988).

| **Semen characteristics** | **Lower reference limit** |
|---|---|
| Volume, mL | 1.5 |
| Sperm concentration, 106/mL | 39 |
| Total sperm number, 106 | 15 |
| Total motility (PR + NP), % | 40 |
| Progressive motility (PR), % | 32 |
| Vitality (live spermatozoa), % | 58 |
| Sperm morphology (normal forms), % | 4 |
| PH | >77.2 |
| Seminal fructose, gmol/ejaculate | >13 |

***Table 3: PR, Progressive Motility; NP, Non-progressive Motility***

| Semen characteristics | WHO 1980 | WHO 1987 | WHO 1992 | WHO 2010 | WHO 1999 |
|---|---|---|---|---|---|
| Volume (mL) | ND | ≥2 | ≥2 | 15 | ≥2 |
| Sperm count ($10^{-}$/mL) | 20-200 | ≥20 | ≥20 | 15 | ≥20 |
| Total sperm count ($10^{-}$) | ND | ≥40 | ≥40 | 39 | ≥40 |
| Total motility (% motile) | ≥6O | ≥50 | ≥50 | 40 | ≥50 |
| Progressive motility[2] | ≥2[3] | ≥25% | ≥25% (grade a) | 32% (a + b) | 205% (grade a) |
| Vitality (% alive) | ND | ≥50 | ≥75 | 58 | ≥75 |
| Morphology (%59<791 19<59) | 80.5 | ≥50 | <30 | 4[6] | (14)[5] |
| Leukocyte count($10^{6}$/mL) | < 4.7 | < 1.0 | < 1.0 | < 1.0 | < 1.0 |

***Table 4: Cut-off Reference Values for Semen Characteristics as Published in Consecutive WHO Manuals***

'Lower reference limits are generated from the lower fifth centile value; 2Grade a = rapid progressive motility (>25µm/s): grade b = slow/sluggish progressive motility (5-25 µm/s): Normal = 50% motility (grades a+b) or 25% progressive motility (grade a) within 60 min of ejaculation; 3Forward progession (scale 0-3); 4Arbitrary value; 5 Value is not defined but strict criterion is suggested: °Strict (Tygetherg) criterion: ND= not defined.

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| Parameter | WHO 1999 | WHO 2010 | % Decline |
|---|---|---|---|
| Semen volume (ml) | 2 | 1.5 | 25 |
| Concentration (106/ml) | 20 | 15 | 25 |
| Motility (%) | 50 | 40 | 20 |
| Normal Morphology (%) | 14 | 4 | 71 |

***Table 5: Cut-off and Reference Values and Percentage Decline in these Values with Shift from the WHO (1999) to WHO (2010) Criteria***

| | WHO1999 | | WHO2010 | |
|---|---|---|---|---|
| Category | N | % | N | % |
| Normal | 27 | 4 | 152 | 23 |
| Oligospermia | 5 | 1 | 38 | 6 |
| Asthenospermia | 91 | 14 | 121 | 18 |
| Teratospermia | 145 | 22 | 111 | 17 |
| Oligoasthenospermia | 17 | 3 | 32 | 5 |
| Oligoteratospermia | 34 | 5 | 43 | 6 |
| Asthenoteratospermia | 141 | 21 | 60 | 9 |
| Oligoasthenoteratospermia | 201 | 30 | 104 | 16 |

***Table 6: Number and Percentage of Men with Single and Multiple Abnormalities in the Standard Semen Analysis According to WHO1999 and 2010 Criteris***

As detailed in the beginning, the males are found to be solely responsible for 20-30% of infertility cases and contribute to 50% of all cases. However there exists a great deal of variation across the world and an accurate data is often lacking (29). It is estimated that 30 to 50% of men have poor semen quality, the cause of which remains poorly understood (30-31).

Apart from abnormalities of the sperm, the other aetiological factors responsible for male infertility include an absence of testicular tissues, bilateral castration, impaired sperm production and function, AZF gene deletion (y-deletion), hypogonadotropic hypogonadism (cryptorchidism), testicular cancer and varicocele, age > 55 years, genitourinary infection, environmental agents such as extremes of temperature, irradiation, occupational exposure, drugs, tobacco abuse, alcohol, and nutritional deficiency like trace elements e.g. selenium, zinc and vitamins. Impaired sperm transport as often seen in autoimmune infertility, epididymitis, blockage of vas deferens, ejaculatory failure, impotence, previous vasectomy and disturbance in sperm oocyte fusion e.g, abnormal egg binding proteins could be the other causes of male infertility. This makes it difficult to declare a person fertile with absolute certainty (32).

Although the clinical value of the analysis of human semen has previously been questioned (33) and semen analysis is an imperfect tool, Semen analysis remains the cornerstone in the evaluation male infertility, despite its imperfections and questions raised about their utility in the past (34). A thorough history, physical examination and a semen analysis forms the basic three steps in the evaluation of male infertility (34).

A report published in 2013 "Falling sperm count twenty years on, where we are now" alarmed the world about the problem and led others to investigate the phenomenon. One of the major criticism was whether there has truly been a global decline in sperm counts in recent decades. Variations in terms of location, donor selection criteria, analytical methods, age distribution, ejaculation frequency, socio-economic background and racial composition, independent of any differences in environmental or life-style exposures preclude a generalized, universally accepted statement. Literature has provided a conflicting account in this respect so far. (35).

Mereino et.al, proved that an increasing age beyond 40 years contributed to a decline in the sperm motility and morphology in men (36). In this study, a normal sperm count was found in 12 (16.4%) participants. While this figure was comparable (14.5%) to another study done in Islamabad (Pakistan) [6], another study in Bangladeshi population, 38.5% of the participants had normozoospermia (37). Globally, males are considered to be a responsible in nearly one-third couples affected by infertility (38).

The original meta-analysis that sperm density has decreased globally by about 50% over the past fifty to sixty years attracted considerable attention and generated much controversy (39-40). An important point to consider is that male infertility is not an entity but reflects a variety of different pathogenic mechanisms (41). A study on the South African population (42) showed 84% abnormal sperm parameters on the basis of concentration, motility, morphology and WBC in semen. This study indicated that male infertility to the tune of 70%, a figure comparable to our study. Another study done in Rome between 2004-2009 revealed that nearly 65% men were responsible for infertility and had alteration in at least one seminal parameter (43).

Nalka K.P in his study concluded that the sperm motility provided more accurate information than the morphology (WHO and Tygerberg's criteria) during the fertility evaluation. They proposed that redefining the reference concentration and morphology could significantly increase the importance of routine semen analysis (44). Several studies have demonstrated the correlation of motility with the fertilization rate in vivo and in vitro. Krause W also noted a predictable impact of sperm concentration and percentage of motile spermatozoa on fertility outcome in vivo (45)

There is a continuing debate over the role of normal morphology in male infertility and its value in the evaluation and management of the infertile men (46). Various parameters may be abnormal in such cases. A useful guide to the prognosis is that one factor abnormality tends to be associated with a better prognosis than two factors which, in turn was better than three factors (abnormality factors are count, motility and morphology) (47). In this study oligoasthenoteratozoospermia was reported in 2 (2.7%) patients, which was lower than a study in which the prevalence was 11% (28) but comparable to another study in which it was reported as 1.39% (48).

Infection of the male genital tract is an important factor. It may affect the seminal quality by directly affecting the spermatozoa or their environment, including local inflammatory reaction (49). Stutz G et al concluded in their study that alcohol, tobacco and aspirin use could have detrimental effects on the seminal parameters and that men who wish to procreate should be warned about such effects (50).

**Evaluation of male reproductive failures:**

Following tables will elaborate the parameters which need to be evaluated in cases of male infertility with their desired normal reference range.

| Hormone (units) | Normal reference range |
|---|---|
| Total testosterone (ng dl-1) >20 years | 240–950 |
| Testosterone, bioavailable (ng dl-1) | |
| 20–39 years | 72–257 |
| 40–69 years | 40–213 |
| Testosterone, free (ng dl-1) | |
| 20–39 years | 1.4–20.3 |
| 40–69 years | 0.6–16.8 |
| Estradiol adult (pg ml-1)* | 11.6–41.6 |
| Follicle stimulating hormone adult (mIU ml-1)* | 0.9–15 |
| Luteinizing hormone adult (mIU ml-1)* | 1.3–13 |
| Inhibin B (pg ml-1) | 47–308 |
| Prolactin (ng ml-1) | 2–15 |

***Table 7: Reference Values of Male Reproductive Hormones***

*Some variation can be observed between various reference laboratories

| Clinical condition | Follicle stimulating hormone | Luteinizing hormone | Testosterone | Prolactin |
|---|---|---|---|---|
| Normal spermatogenesis | Normal | Normal | Normal | Normal |
| Abnormal spermatogenesis | High | Normal | Normal | Normal |
| Hypogonadotropic hypogonadism | Low | Low | Low | Normal |
| Hypergonadotropic hypogonadism/ complete testicular failure | High | High | Low | Normal |
| Prolactin-secreting pituitary tumor | Normal/low | Normal/low | Low | High |

***Table 8: Male Reproductive Hormone Evaluation Profile as Related to Clinical Condition***

| Characteristics | Units | Normal | Borderline | Pathological | Notes |
|---|---|---|---|---|---|
| Volume | ml | 2.0–6.0 | 1.5–1.9 | <1.5 | a |
| Sperm concentration | 106 ml-1 | 20–250 | 10–20 | <10 | a, b |
| Total sperm count | 106/ejaculate | ≥80 | 20–79 | <20 | a, b |
| Motility | % motile (total) | ≥60 | 40-59 | <40 | c, d |
| | % Progressive | ≥50 | 35-49 | <35 | c, d |
| | % rapid progressive | ≥25 | - | - | c, d |
| | Progression rate | 3 or 4 | 2 | 1 or 2 | c, d, e |
| Morphology | % typical head forms | ≥14 | 4–13 | <4 | f |
| Viability | Percent Viable | >75 | 50–70 | <50 | g |

***Table 9: General (consensus - based) Reference Values for Evaluation of Key Semen Parameters 49***

a: evaluated after 2–4 days of abstinence; b: for specimen with 2.0–6.0 ml volume;

c: evaluated at 30 min post-ejaculation; d: evaluated at 37°C; e: based on a scale

of 0–4 - 0: no progression; 1: poor; 2: medium; 3: good; 4: very good/excellent;

f: evaluated using Tygerber "strict criteria;" g: evaluated by eosin dye exclusion at

30 min post ejaculation.

Kruger's criteria are considered a standard for measuring the sperm morphology, or its shape. The Kruger's criteria are considered more in-depth and critical than the standard "crude morphology" assessment performed during a basic sperm analysis. The process is typically employed in men who have sperm that appears to be visually competent, but, along with a partner, are struggling with unexplained infertility.

As per Kruger's strict criteria assessment, the sample of sperm is examined under a microscopic magnification of 1000x. Any minor deformity in the sperm's shape or structure is enough to classify the sperm as abnormal according to the criteria. A standard sperm contains four distinct components:

- An oblong head, where the DNA is stored.
- A thick mid part or body called the midpiece that houses the mitochondria, the energy source of the sperm.
- A tail
- An acrosomal cap, lining the tip and a very crucial structure aiding in fertilization.

Any abnormalities of these features are enough to classify the sperm as damaged. Examples of the abnormalities include:

- Abnormally sized heads
- Double heads
- Missing or disfigured midpiece
- Double tails
- Short tails
- Kinked tails
- Missing acrosomal cap
- Disfigured acrosomal cap

The final percentage of entirely undamaged sperm determines the resulting Kruger's strict criteria score. Therefore, a sperm sample with high Kruger's scores is more likely to contribute to pregnancy, whether introduced to an egg during the sexual intercourse or through assisted reproductive treatments (ART), including in vitro fertilization (IVF).

Strict morphology scores and their indications:

- Over 14% normal – high fertility potential
- 4-14% normal – somewhat impacted fertility potential
- 0-3% normal – severe impairment, possibly unable to fertilize through sexual intercourse, and may require fertility treatment

In addition to Kruger's strict criteria, the World Health Organization (WHO) is the other widely used sperm analysis criteria. The WHO follows a similar scale, and as of 2010, also considers 4% and higher of normal sperm as the advantageous composition for fertility (51-54).

## CONCLUSION

Male component of infertility is often an under recognised entity. The conventional semen analysis is a cheap, reliable and non-invasive technique representing the cornerstone in the initial workup schedule. It is clear that the male factor infertility has not been researched or studied to truly understand its magnitude and prevalence, especially in our society due likely to cultural and social barriers. Therefore, we have a great challenge in front of us, in term of diagnosis, awareness, prevention and treatment of infertility emanating from a male source. A falling sperm count hypothesis appears very likely but needs a more elaborate multicentered research for any definitive conclusions. In our population, main contributors are smoking and tobacco chewing.

## REFERENCES

1. Gurunath S, Pandian Z, Anderson RA, et al. Defining infertility-a systematic review of prevalence studies. Hum Reprod Update 2011;17:575-588.
2. Naina Kumar, Amit Kant Singh, Trends of male factor infertility, an important cause of infertility: A review of literature J Hum Reprod Sci. 2015; 8(4): 191-196.
3. Zegers-Hochschild F, Adamson GD, de Mouzon J, et al. International Committee for Monitoring Assisted Reproductive Technology (ICMART) and the World Health Organization (WHO) revised glossary of ART terminology, 2009. Fertil Steril. 2009;92:1520-1524
4. Practice Committee of the American Society for Reproductive Medicine. Definitions of infertility and recurrent pregnancy loss. Fertil Steril. 2008;90(5 Suppl):S60.
5. World Health Organization. WHO Laboratory Manual for the Examination of Human Semen and Semen-Cervical Mucus Interaction. 4th ed. Cambridge: Cambridge University Press; 1999.

6. Mascarenhas MN, Cheung H, Mathers CD, et al. Measuring infertility in populations: Constructing a standard definition for use with demographic and reproductive health surveys. Popul Health Metr. 2012;10:17.
7. Boivin J, Bunting L, Collins JA, et al. International estimates of infertility prevalence and treatment-seeking: Potential need and demand for infertility medical care. Hum Reprod. 2007; 22:1506-1512.
8. Calverton, Maryland. World Health Organization. Infecundity, Infertility, and Childlessness in Developing Countries. USA: ORC Macro and the World Health Organization; 2004.
9. Sciarra J. Infertility: An international health problem. Int J Gynaecol Obstet. 1994;46:155-163.
10. Population Council. Infertility. Looking Back, Looking Forward: A Profile of Sexual and Reproductive Health in India. New Delhi: Population Council; 2004.
11. Van Balen F, Gerrits T. Quality of infertility care in poor-resource areas and the introduction of new reproductive technologies. Hum Reprod. 2001;16:215-219.
12. Stephen EH, Chandra A. Declining estimates of infertility in the United States: 1982-2002. Fertil Steril. 2006;86:516-523.
13. Stephen EH, Chandra A. Updated projections of infertility in the United States: 1995-2025. Fertil Steril. 1998;70:30-34.
14. Chandra A, Copen CE, Stephen EH. Infertility and impaired fecundity in the United States, 1982-2010: Data from the National Survey of Family Growth. National Health Statistics Reports; No 67. Available from: http://www.cdc.gov/nchs/data/nhsr/nhsr067.pdf.
15. Martin JA, Hamilton BE, Sutton PD, et al. Births: Final data for 2004. Natl Vital Stat Rep. 2006; 55:1-101.
16. Sadock BJ, Sadock VA. Kaplans and Sadocks Symptoms of Psychiatry Behavioral Sciences Clinical Psychiatry. 9th ed. Philadelphia: Lippincott Williams and Wilkins; 2003.
17. World Health Organization. Towards more objectivity in diagnosis and management of male infertility. Int J Androl. 1987; 7:1-53.
18. Kumar TCA. Fertility and in-vitro fertilization in India. Curr Sci. 2004;86:254-256.
19. Ahmad S, Wasim S, Tiwari N, et al. Evaluation of Bacteriospermia as Etiology for Oligospermia: An Analysis. Int J Sci Stud. 2016;4(2):194-197.
20. Nabi A, Khalili MA, Halvaei I, et al. Seminal bacterial contaminations: Probable factor in unexplained recurrent pregnancy loss. Iran J Reprod Med. 2013;11:925-932.
21. Barratt CL. Semen analysis is the cornerstone of investigation for male infertility. Practitioner. 2007; 251: 8.
22. World Health Organization. WHO Laboratory Manual for the Examination of Human Semen and Semen-Cervical Mucus Interaction. 4th ed. Cambridge: Cambridge University Press; 1999.
23. World Health Organization Department of Reproductive Health and Research. World Health Organization Laboratory Manual for the Examination and Processing of Human Semen. 5th ed. Geneva, Switzerland: World Health Organization; 2010.
24. Jequier AM. Semen analysis: A new manual and its application to the understanding of semen and its pathology. Asian J Androl. 2010;12:11-13.
25. Guzick DS, Overstreet JW, Factor-Litvak P, et al. Sperm morphology, motility, and concentration in fertile and infertile men. N Engl J Med. 2001;345:1388-1393.
26. Smith KD, Rodriguez-Rigau LJ, Steinberger E. Relation between indices of semen analysis and pregnancy rate in infertile couples. Fertil Steril. 1977;28:1314-1319.
27. Brazil C. Practical semen analysis: From A to Z. Asian J Androl. 2010;12:14-20.
28. Cooper TG, Noonan E, von Eckardstein S, et al. World Health Organization reference values for human semen characteristics. Hum Reprod Update. 2010;16:231-245.
28. Agarwal A, Mulgund A, Hamada A, et al. A unique view on male infertility around the globe. Reproductive Biol Endocrinol. 2015;13:37.
29. Nieschlag E, Behre HM. Andrology: Male reproductive health and dysfunction. Heidelberg: Springer;1997.
30. Farley TM, Belsey FH. The prevalence and aetiology of infertility. In: Proceedings of the African population Conference. Dakar, Senegal. Liege: International Union for the scientific study of population; 1988.
31. Khan MS, Deepa F, Ahmed Z, et al. Assessment of male reproductive health by conventional method of semen analysis. J Ayub Med Coll Abbottabad. 2011; 23:84-88.
32. Franken DR, Oehninger S. Semen analysis and

sperm function testing. Asian J Androl 2012;14:6-13.

33. Vasan SS. Semen analysis and sperm function tests: How much to test? Indian J Urol. 2011;27:41-48.
34. World Health Organization. WHO Laboratory Manual for the Examination and Processing of Human Semen. 5th ed. Geneva: World Health Organization;2010.
35. Aitken RJ. Falling sperm counts twenty years on: where are we now? Asian J Androl. 2013;15:204-207.
36. Menrino G, Carranza LS. Semen characteristics, endocrine profile and testicular biopsies of infertile men of different ages. Arch Androl. 1995;32:219-224.
37. Muazzam MG. Investigation of Infertility-Semen Analysis. Bangladesh Journal of Medical sciences. 1996; 3: 32-35.
38. Seibel MM. Workup of the infertile couple. In: Infertility: A Comprehensive Text Book. Connecticut USA Norwalk: Appleton and Lange; 1990.
38. Carlsen E, Giwercman A, KeidingN, et al. Evidence for decreasing quality of semen during past 50 years. BMJ. 1992; 305: 609-613.
39. Fisch H. Declining Worldwide Sperm Counts: Disproving a Myth. Urol Clin North Am 2008; 35:137-146.
40. Shakkeback VE, Giwercman A, de-krester D. Pathogenesis and management of male infertility. Lancet. 1994;343:1473-1479.
41. Bornman MS, Schulenburg GW, Boomker D, et al. Observations in infertile African males at an andrology clinic in South Africa. Arch Androl. 1994;33:101-104.
42. Milardi D, Grande G, Sacchini D, et al. Male Fertility and Reduction in Semen Parameters: A single Tertiary-care Center Experience. Int J Endocrinol. 2012; 2012: 649149.
43. Shaikh AH, Khalique K, Tariq G, et al. Pattern of semen abnormalities in couples with male factor infertility. Pak J Surg. 2011; 27:204-208.
44. Krause W. The significance of computer assisted semen analysis (CASA) for diagnosis in andrology and fertility prognosis. Int J Androl 1995;18:32-35.
45. JajooS, Kalyani KR. Prevalence of abnormal semen analysis in patients of infertility at a rural setup in Central India. Int J Reprod Contracept Obstet Gynecol. 2013;2:161-164.
46. Menkveld R, Wong WY, Lombard CJ, et al. Semen parameters, including WHO and strict criteria morphology, in a fertile and sub-fertile population: an effort towards standardization of in-vivo thresholds. Hum Reprod. 2001;16:1165-1171.
47. Mathews T, Mati JK and Formulu JN. A study of infertility in Kenya: results of investigation of the infertile couple in Nairobi. East Afr Med J. 1981;58:258-267.
48. Mbizvo MT, Chinibira TH, Gwavava, et al. Azoospermic men. Br J Urol. 1989;63:423-427.
49. Khan AA, Khan FA, Sattar A, et al. Azoospermia in clinical practice at Rawalpindi. Pak Armed Force Med J. 1992;42:93-95.
50. Aziz N. The importance of semen analysis in the context of azoospermia. Clinics. 2013; 68(31): 35-38.
51. Dickey RP, Pyrak R, Lu PY, et al. Comparison of the sperm quality necessary for successful intrauterine insemination with World Health Organization threshold values for normal sperm. Fertil Steril. 1997;71:684-689.
52. Subhan F, Tahir F, Ahmad R, et al. The study of azoospermic patients in relation to their hormonal profile (LH, FSH and Testosterone). Rawal Med J. 1995;22:25-27.
53. Stutz G, ZaimudioJ, Santillan ME, et al. The effect of alcohol, tobacco and aspirin consumption on Semen quality among healthy young men. Arch Environ health. 2004;59:548-558.
54. Purohhit TM, Purohit MB, Dabhi BJ. Study of Semen Analysis and testicular biopsy in infertile men. Indian J Pathol Microbl. 2004;47:486-490.

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