



Comparative study of psychosexual aspects between male factor and female factor infertility

Thesis

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Dermatology & Andrology*

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Abstract

Objective: The present study was performed to compare sexual, marital and psychological aspects of infertile males and infertile females.

Method: After obtaining their consents, 50 infertile males and 50 infertile females answered 3 questionnaires of symptoms checklist 90, index of marital satisfaction (IMS) questionnaire & sexual satisfaction scale (SSS) questionnaire.

Results: The study showed that infertile females tend to have higher levels of depression, anxiety, somatization and interpersonal sensitivity as compared to infertile males. Age, infertility duration and most of psychiatric disorders of subjects have no effect on marital or sexual satisfaction. Marital & sexual satisfactions are affected by each other positively in both genders.

Conclusion: Infertility diagnosis is an important factor in assessing the differences in infertility distress, marital satisfaction and sexual satisfaction particularly for women. Adequate attention to these patients psychologically is of such importance as physical attention.

Keywords: male factor infertility, female factor infertility, Psychiatric disorders marital satisfaction, sexual satisfaction

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List of abbreviations

ACTH	Adrenal Corticotrophin Hormone
ART	Assisted Reproductive Technique
AZF	Azoospermia Factor Region
CAH	Congenital Adrenal Hyperplasia
CBAVD	Congenital Bilateral Absence of VasDeferens
CF	Cystic Fibrosis
CFTR	Cystic Fibrosis Trans membrane Conductance Regulator gene
CRH	Corticotropin releasing hormone
DES	Diethylstilboestrol
FSH	Follicle Stimulating Hormone
GABA	γ -aminobutyric acid
GAD	Generalized Anxiety Disorder
GALP	Galanin-like peptide
GNRH	Gonadotropin Releasing Hormone
HA	Hypothalamic Amenorrhea
HCG	Human Chronic Gonadotropin
HH	Hypogonadotropic Hypogonadism
HMG	Human Menopausal Gonadotropin
HPG	Hypothalamic Pituitary Gonadal axis
ICSI	IntraCytoplasmic Sperm Injection
IUI	IntraUterine Insemination
IVF	In Vitro Fertilization
LH	Luteinizing Hormone
NPY	Neuropeptide Y
OCD	Obsessive Compulsive Disorder
PCOS	PolyCystic Ovary Syndrome
PID	Pelvic Inflammatory Disease
POF	Premature Ovarian Failure
PTSD	Post Traumatic Stress Disorder
SHBG	Sex hormone binding globulin
TESE	Testicular Sperm Extraction
TSH	Thyroid Stimulating Hormone

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Introduction

Infertility, defined as the inability to conceive after one year of regular unprotected intercourse, is a prevalent condition and represents a significant social and public health problem (*Khayata et al., 2003*).

Infertility affects about 7% of all men. The etiology of impaired sperm production and function can be related to factors acting at pre-testicular, post-testicular or directly at the testicular level. Primary testicular failure accounts for about 75% of all male factor infertility. Genetic factors can be identified in about 15% of cases (e.g. congenital hypogonadotropic hypogonadism, congenital absence of vas deferens). Despite progresses, mainly in the field of genetics, the etiology is still unknown in about 50% cases and it is termed “idiopathic infertility” (*krausz, 2011*).

In a World Health Organization (WHO) study of 8500 infertile couples, female factor infertility was reported in 37 percent of infertile couples in developed countries. The most common identifiable female factors are: Ovulatory disorders, Endometriosis, Pelvic adhesions, Tubal blockage, other tubal abnormalities and Hyperprolactinemia (*WHO, 1992; Smith et al., 2003*).

Marital satisfaction is a global evaluation of the state of one's marriage or current long-term romantic relationship. This global evaluation can be a reflection of a how happy people are in their marriage in general or a composite of satisfaction with several specific facets of the marital relationship (*Roehling & Bultman, 2002; Schoen et al., 2002*).

Social, psychological and infertility related issues as well as gender may be of relevance in determining the impact of infertility on marital relationships (*Henning and strauss, 2002*).

Sexual satisfaction is defined as a multidimensional experience involving thoughts, feelings, personal and socio-cultural attitudes beliefs, combined with biological factors (*Gil, 2007*).

The ability to reproduce is intimately tied to sexuality, self image and self-esteem. Sexuality and sexual activity are also important means of expressing feelings of closeness and intimacy in relationship. During infertility treatment, the pleasurable experience of sexual intimacy may be negatively affected and this may contribute to marital distress (*Oddens et al., 1999; Wischmann et al., 2001*).

These feelings of distress, sometimes combined with lack of social support, may result in several physiological and psychological symptoms of distress, such as health complaints, depression, anxiety and even complicated bereavement (*Berghuis and Stanton, 2002; van den Akker, 2005; Verhaak et al., 2005*).

Aim of the work

The aim of the work is to compare sexual, marital and psychological aspects of infertile male and infertile female.

Male Factor Infertility

Introduction

Infertility affects about 7% of all men. The etiology of impaired sperm production and function can be related to factors acting at pre-testicular, post-testicular or directly at the testicular level. Primary testicular failure accounts for about 75% of all male factor infertility. Genetic factors can be identified in about 15% of cases (e.g. congenital hypogonadotropic hypogonadism, congenital absence of vas deferens). Despite progresses, mainly in the field of genetics (*Krausz, 2011*), the etiology is still unknown in about 50% cases and it is termed “idiopathic infertility” (*Hamada et al., 2011*).

The Causes of Male Infertility

Pre-testicular, testicular and post-testicular causes.

I Pre-testicular causes

Pretesticular causes of infertility include congenital or acquired diseases of the hypothalamus, pituitary, or peripheral organs that alter the hypothalamic-pituitary axis (*Huhtaniemi & Alevizaki, 2007*).

1-Hypothalamic disorders:

Disorders of the hypothalamus lead to hypogonadotropic hypogonadism. If GnRH is not secreted, the pituitary does not release LH and FSH. Ideally, patients respond to replacement with exogenous GnRH or HCG, an LH analogue, although this does not always occur (*Huhtaniemi & Alevizaki, 2007*).

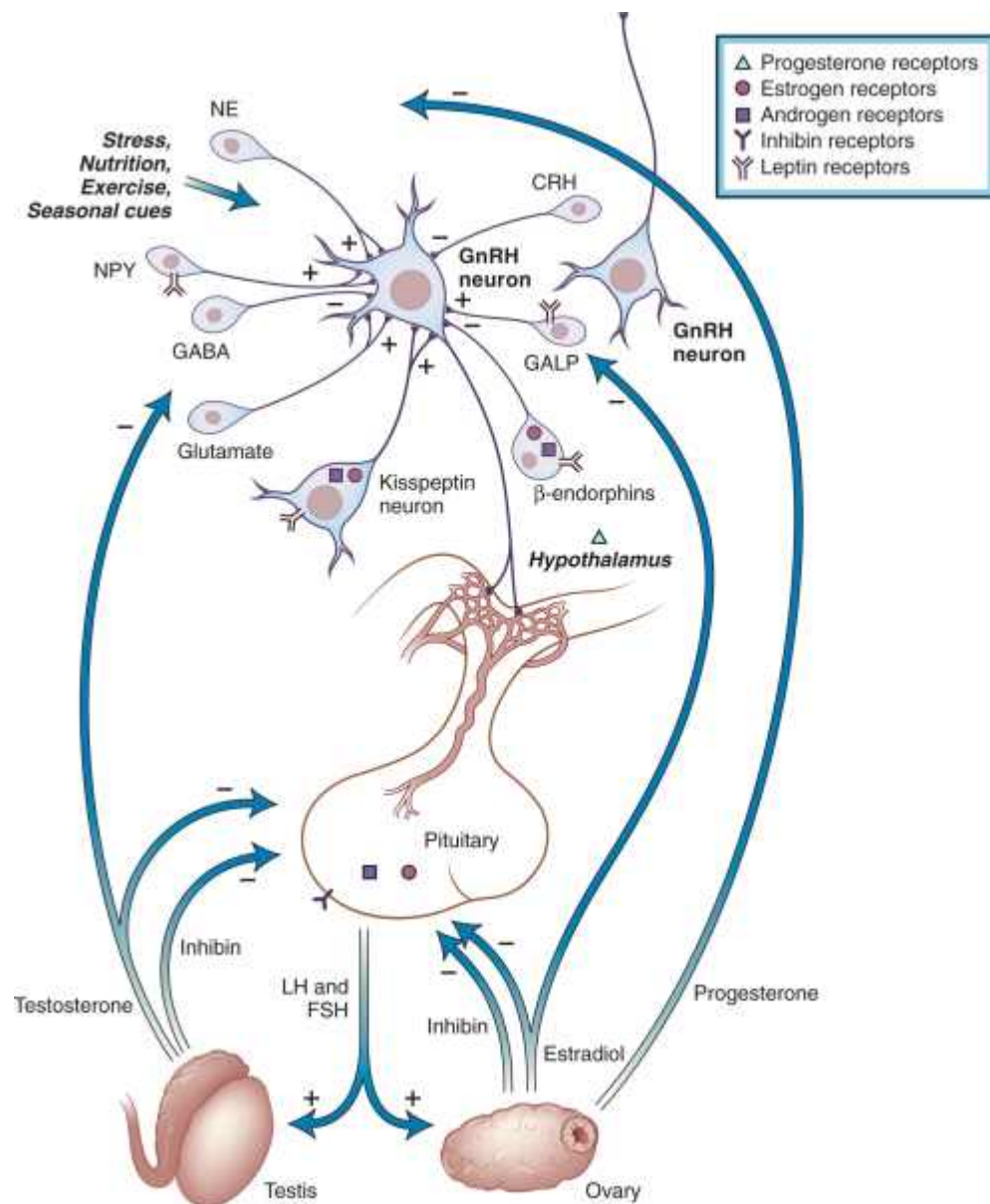


Fig.(1): Regulation of the hypothalamic-pituitary-gonadal axis. Schematic diagram of the hypothalamic-pituitary-gonadal axis showing neural systems that regulate GnRH secretion and feedback of gonadal steroid hormones at the level of the hypothalamus and pituitary. (Henry *et al.*, 2007)

Idiopathic hypogonadotropic hypogonadism: A failure of GnRH secretion without any discernible underlying cause may be observed alone (isolated) or as part of Kallmann syndrome, which is associated with midline defects such as anosmia, cleft lip and cleft palate, deafness,

cryptorchidism, and color blindness. Kallmann syndrome has been described in both familial (X-linked and autosomal) and sporadic forms, and its incidence is estimated as 1 case per 10,000-60,000 births (**Brugh & Lipshultz, 2004**).

A failure of GnRH neurons to migrate to the proper location in the hypothalamus has been implicated. Patients generally have long arms and legs due to a delayed closure of the epiphyseal plates, delayed puberty, and atrophic testis (**Whitten et al., 2006**).

Prader-Willi syndrome: Patients have characteristic obesity, mental retardation, small hands and feet, and hypogonadotropic hypogonadism due to a GnRH deficiency. Prader-Willi syndrome is caused by a disorder of genomic imprinting with deletions of paternally derived chromosome arm 15q11-13 (**Seminara et al., 2000**).

Laurence-Moon-Biedl syndrome: Patients with this syndrome have retinitis pigmentosa and polydactyly. Infertility is due to hypogonadotropic hypogonadism (**Seminara et al., 2000**).

Defects in Gn-RH secretion or the Gn-RH receptor lead to idiopathic hypogonadotropic hypogonadism. Various other lesions and diseases, such as CNS tumors, temporal lobe seizures, and many drugs (eg, dopamine antagonists) may interrupt the hypothalamic-pituitary axis at the hypothalamus (**Brugh & Lipshultz, 2004**).

Table 1. Genetic disorders leading to male infertility and treatment

Disorder	Cause	Treatment
<i>Disorders of GnRH secretion</i>		
Kallman's syndrome	Mutation in Kal gene (Xp22.3) resulting in ↓GnRH secretion	Replacement of FSH and HCG
GnRH receptor defects	Defects in G-protein coupled for GnRH	Replacement of FSH and HCG
↓GnRH secretion	Mutation in Convertase-1 gene (PC1)	Replacement of FSH and HCG
Prader-Willi syndrome	Mutation in 15q11q13	Replacement of FSH and HCG
<i>Disorders of LH and FSH function</i>	Defects in LH or FSH structure or receptor defects	Replacement of FSH and HCG if LH or FSH structural defect
<i>Disorders of androgen function</i>		
Congenital adrenal hyperplasia	Mutations in steroidogenic enzymes	Replacement of corticosteroids, mineralocorticosteroids, or androgens
Androgen insensitivity (Reifenstein's syndrome, testicular feminization, Lub syndrome, Rosewater's syndrome)	Mutations in the androgen receptor gene	These men may be candidates for TESE-IVF-ICSI although success rates for sperm extraction have not been reported nor have outcomes of IVF-ICSI
Kennedy's syndrome	Expansion of the polyglutamine tract in the AR transactivation domain	
5α-Reductase deficiency	Mutations in the 5α-reductase gene	

Data from Refs. (*de Roux et al.,1999, Wu et al.,2000, Casella et al.,2001*).

2-Pituitary disorders:

Both pituitary insufficiency and pituitary excess cause infertility. Pituitary failure may be congenital or acquired. Acquired causes include tumor, infarction, radiation, infection, or granulomatous disease. Nonfunctional pituitary tumors may compress the pituitary stalk or the gonadotropic cells, interrupting the proper chain of signals leading to pituitary failure. In contrast, functional pituitary tumors may lead to unregulated gonadotropin release or prolactin excess, interrupting the proper signaling (*Pavlovich et al., 2001*).

Prolactinoma: A prolactin-secreting adenoma is the most common functional pituitary tumor. Prolactin stimulates breast development and lactation; therefore, patients with infertility due to a prolactinoma may have gynecomastia and galactorrhea. In addition, loss of peripheral visual fields bilaterally may be due to compression of the optic chiasm by the growing pituitary tumor. A prolactin level of more than 150 mcg/L suggests a pituitary adenoma, while levels greater than 300 mcg/L are nearly diagnostic (*Pavlovich et al., 2001; Abdel-Razic et al., 2012*).

Isolated LH deficiency (fertile eunuch): In these patients, LH levels are decreased while FSH levels are within the reference range. Patients have eunuchoidal body habitus, large testis, and a low ejaculatory volume. The treatment of choice is exogenous HCG (*Wu et al., 2000*).

Isolated FSH deficiency: This is a very rare cause of infertility. Patients present with oligospermia but have LH levels within the reference range. Treatment is with human menopausal gonadotropin (HMG) or exogenous FSH (*Simoni et al., 1999*).

3-Peripheral organ disorders:

The hypothalamus-pituitary axis may be interrupted by hormonally active peripheral tumors or other exogenous factors, due to cortical excess, cortical deficiency, or estrogen excess (*Huhtaniemi & Alevizaki, 2007*).

Excess cortisol may be produced by adrenal hyperplasia, adenomas, carcinoma, or lung tumors. High cortisol levels may also be seen with exogenous steroid use, such as that administered to patients with ulcerative colitis, asthma, arthritis, or organ transplant. For example, high cortisol levels are seen in patients with Cushing syndrome, which causes negative feedback on the pituitary to decrease LH release (*Nieman & IIias, 2005*).

Cortical deficiency may be seen in patients with adrenal failure due to infection, infarction, or congenital adrenal hyperplasia (CAH). CAH may be due to the congenital deficiency of one of several adrenal enzymes, the most common of which is 21-hydroxylase deficiency. Because cortisol is not secreted, a lack of feedback inhibition on the pituitary gland occurs, leading to adrenocorticotrophic hormone (ACTH) hypersecretion. This leads to increased androgen secretion from the adrenal gland, causing feedback inhibition of GnRH release from the hypothalamus. Patients present with short stature, precocious puberty, small testis, and occasional bilateral testicular rests. Screening tests include increased plasma 17-hydroxylase and urine 17-ketosteroids (*Nieman & IIias, 2005*).

Estrogen excess may be seen in patients with Sertoli cell tumors, Leydig tumors, liver failure, or severe obesity. Estrogen causes negative