Dr. Vinay Chopra
MD (Pathology & Microbiology)
Chairman & Consultant Pathologist

Dr. Yugam Chopra
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CEO & Consultant Pathologist

NAME	**: Mr. MAYANK**		
AGE/ GENDER	: 32 YRS/MALE	**PATIENT ID**	: 1436713
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 012409220015**
REFERRED BY	:	**REGISTRATION DATE**	: 22/Sep/2024 08:14 AM
BARCODE NO.	: 01517450	**COLLECTION DATE**	: 22/Sep/2024 08:16AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	**REPORTING DATE**	: 22/Sep/2024 11:45AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval

ENDOCRINOLOGY

LUTEINISING HORMONE (LH)

LUTEINISING HORMONE (LH): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	7.87	mIU/mL	MALES: 0.57 - 12.07 FOLLICULAR PHASE: 1.80 - 11.78 MID-CYCLE PEAK: 7.59 - 89.08 LUTEAL PHASE: 0.56 - 14.0 POST MENOPAUSAL WITHOUT HRT: 5.16 - 61.99

INTERPRETATION:

1. Luteinizing hormone (LH) is a glycoprotein hormone consisting of 2 non covalently bound subunits (alpha and beta). Gonadotropin-releasing hormone from the hypothalamus controls the secretion of the gonadotropins, FSH and LH, from the anterior pituitary.
2. In both males and females, LH is essential for reproduction. In females, the menstrual cycle is divided by a mid cycle surge of both LH and FSH into a follicular phase and a luteal phase.
3. This "LH surge" triggers ovulation thereby not only releasing the egg, but also initiating the conversion of the residual follicle into a corpus luteum that, in turn, produces progesterone to prepare the endometrium for a possiblei mplantation.
4. LH supports thecal cells in the ovary that provide androgens and hormonal precursors for estradiol production. LH in males acts on testicular interstitial cells of Leydig to cause increased synthesis of testosterone.

The test is useful in the following situations:

1. An adjunctin the evaluation of menstrual irregularities.
2. Evaluating patients with suspected hypogonadism
3. Predicting ovulation & Evaluating infertility
4. Diagnosing pituitary disorders
5. In both males and females, primary hypogonadism results in an elevation of basal follicle-stimulating hormone and luteinizing hormone levels.

FSH AND LH ELEVTED IN:

1. Primary gonadal failure
2. Complete testicular feminization syndrome
3. Precocious puberty (either idiopathic or secondary to a central nervous system lesion)
4. Menopause
5. Primary ovarian hypo dysfunction in females
6. Polycystic ovary disease in females
7. Primary hypogonadism in males

LH IS DECREASED IN:

1 .Primary ovarian hyper function in females
2. Primary hypergonadism in males

NOTE

1 .FSH and LH are both decreased in failure of the pituitary or hypothalamus.

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Test Name	Value	Unit	Biological Reference interval

TESTOSTERONE: FREE

TESTOSTERONE - FREE: SERUM	39.7	pg/mL	12.3 - 46.6

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

INERPRETATION:

1.Testosterone is secreted in females by the ovary and formed indirectly from androstenedione in adrenal glands.
2.In males it is secreted by the testes. It circulates in blood bound largely to sex hormone binding globulin (SHBG). Less than 1% of the total testosterone is in the free form.
3.The bioavailable fraction includes the free form and that "weakly bound" to albumin (40% of the total in men and 20% of the total in women) and bound to cortisol binding globulin (CBG). It is the most potent circulating androgenic hormone.
4.The total testosterone bound to SHBG fluctuates since SHBG levels are affected by medication, disease, sex steroids and insulin.

CLINICAL SIGNIFICANCE:

1.Usually, bioavailable and free testosterone levels parallel the total testosterone levels. However, a number of conditions and medications are known to increase or decrease the SHBG (SHBG / Sex Hormone Binding Globulin [SHBG], Serum) concentration, which may cause total testosterone concentration to change without necessarily influencing the bioavailable or free testosterone concentration, or vice versa.

CLINIC USE OF FREE TESTOSTERONE:

1.Assesment of testicular functions in males
2.Management of hirsutism and virilization in females.
3.Treatment with corticosteroids and sex steroids (particularly oral conjugated estrogen) can result in changes in SHBG levels and availability of sex-steroid binding sites on SHBG. This may make diagnosis of subtle testosterone abnormalities difficult.
4.Inherited abnormalities in SHBG binding.
5.Liver disease and severe systemic illness.
6.In pubertal boys and adult men, mild decreases of total testosterone without LH abnormalities can be associated with delayed puberty or mild hypogonadism. In this case, either bioavailable or free testosterone measurements are better indicators of mild hypogonadism than determination of total testosterone levels.
7.In polycystic ovarian syndrome and related conditions, there is often significant insulin resistance, which is associated with low SHBG levels. Consequently, bioavailable or free testosterone levels may be more significantly elevated.

INCREASED LEVELS:

1.Precocious puberty (Males)
2.Androgen resistance
3.Testoxicosis
4.Congenital Adrenal Hyperplasia
5.Polycystic ovarian disease
7.Ovarian tumors

DECREASED LEVELS:

1.Delayed puberty (Males)

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Test Name	Value	Unit	Biological Reference interval

2.Gonadotropin deficiency
3.Testicular defects
4.Systemic diseases

*** End Of Report ***

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