

(A Unit of KOS Healthcare)



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

Dr. Yugam Chopra MD (Pathology) CEO & Consultant Pathologist

**NAME** : Mr. RAKESH

**AGE/ GENDER** : 27 YRS/MALE **PATIENT ID** : 1559146

**COLLECTED BY** REG. NO./LAB NO. : 012407240040

REFERRED BY **REGISTRATION DATE** : 24/Jul/2024 12:44 PM BARCODE NO. :01513741 **COLLECTION DATE** : 24/Jul/2024 12:45PM CLIENT CODE. : KOS DIAGNOSTIC LAB REPORTING DATE

**CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name Value Unit **Biological Reference interval** 

## **ENDOCRINOLOGY LUTEINISING HORMONE (LH)**

LUTEINISING HORMONE (LH): SERUM 5.22 mIU/mL MALES: 0.57 - 12.07

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

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

: 24/Jul/2024 03:26PM

**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 possible implantation.

4. LH supports thecal cells in the ovary that provide and organization 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 DECŘEÁSEĎ 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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### FOLLICLE STIMULATING HORMONE (FSH)

FOLLICLE STIMULATING HORMONE (FSH): SERUM by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

7.26 FEMALE FOLLICULAR PHASE: 3.03 -

FEMALE MID-CYCLE PEAK: 2.55 -

16.69

FEAMLE LUTEAL PHASE: 1.38 -

5.47

FEMALE POST-MENOPAUSAL:

26.72 - 133.41 MALE: 0.95 - 11.95

#### **INTERPRETATION:**

1. Gonadotropin-releasing hormone from the hypothalamus controls the secretion of the gonadotropins, follicle-stimulating hormone (FSH) and luteinizing hormone (LH) from the anterior pituitary.

2. The menstrual cycle is divided by a midcycle surge of both FSH and LH into a follicular phase and a luteal phase.

FSH appears to control gametogenesis in both males and females.The test is useful in the following settings:

- An adjunct in the evaluation of menstrual irregularities.
   Evaluating patients with suspected hypogonadism.
   Predicting ovulation

- 4. Evaluating infertility
- 5. Diagnosing pituitary disorders
- 6. In both males and females, primary hypogonadism results in an elevation of basal follicle-stimulating hormone (FSH) and luteinizing hormone (LH) levels

### **FSH and LH LEVELS ELEVATED IN:**

- Primary gonadal failure
   Complete testicular feminization syndrome.
- 3. Precocious puberty (either idiopathic or secondary to a central nervous system lesion)
- Menopause (postmenopausal FSH levels are generally >40 IU/L)
- 5. Primary ovarian hypofunction in females
- 6. Primary hypogonadism in males

#### NOTE:

- 1. Normal or decreased FSH is seen in polycystic ovarian disease in females
- 2. FSH and LH are both decreased in failure of the pituitary or hypothalamus.



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**TESTOSTERONE: TOTAL** 

TESTOSTERONE - TOTAL: SERUM 5.03 ng/mL 0.47 - 9.80

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

**INTERPRETATION:** 

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.

**CLINIC USE:** 

1.Assesment of testicular functions in males 2.Management of hirsutism and virilization in females

**INCREAŠED LEVELS:** 

1.Precocious puberty (Males)
2.Androgen resistance
3.Testoxicosis
4.Congenital Adrenal Hyperplasia

5. Polycystic ovarian disease

7. Ovárián tumors

#### **DECREASED LEVELS:**

1.Delayed puberty (Males)
2.Gonadotropin deficiency

3. Testicular defects

4. Systemic diseases



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## **CLINICAL PATHOLOGY** SEMEN ANALYSIS/SEMINOGRAM

#### PHYSICAL EXAMINATION

TIME OF SPECIMEN COLLECTION	24-07-2024	AM/PM	
DURATION OF ABSTINENCE	3 DAYS	DAYS	2 - 7
TYPE OF STONE	FRESH		
LIQUIFACTION TIME AT 37*C	< 30 MINS	MINS	30 - 60
VOLUME	1	ML	
COLOUR	WHITISH OPAQUE		WHITISH OPAQL

VISCOSITY **VISCOUS VISCOUS** ρН 5.0 - 7.5pН

### AUTOMMATED SEMEN ANALYSIS, GOLD STANDARD, WHO APPROVED (SQA GOLD)

TOTAL SPERM CONCENTRATION	3.2	Millions/mL	12 - 16
	3.2	IVIIIIIOHS/THL	12 - 10
by ELECTRO-OPTICS SIGNAL & COMPUTER ALOGRITHM		0/	40.0
TOTAL MOTILITY (GRADE A + GRABE B + GRADE C)	2	%	> = 42.0
by ELECTRO-OPTICS SIGNAL & COMPUTER ALOGRITHM			
RAPIDLY PROGRESSIVE MOTILITY (GRADE A)	0	%	> = 30.0
by ELECTRO-OPTICS SIGNAL & COMPUTER ALOGRITHM			
SLOWLY PROGRESSIVE MOTILITY (GRADE B)	1	%	>= 30
by ELECTRO-OPTICS SIGNAL & COMPUTER ALOGRITHM			
NON PROGRESSIVE MOTILITY (GRADE C)	1	%	<= 1
by ELECTRO-OPTICS SIGNAL & COMPUTER ALOGRITHM			
IMMOTILE	98	%	
by ELECTRO-OPTICS SIGNAL & COMPUTER ALOGRITHM			
MORPHOLOGY NORMAL	0	%	> = 4.0
by ELECTRO-OPTICS SIGNAL & COMPUTER ALOGRITHM			
VELOCITY (AVERAGE PATH VELOCITY)	0	Mic/sec	> = 5
by ELECTRO-OPTICS SIGNAL & COMPUTER ALOGRITHM			
SPERM MOTILE INDEX (SMI)	NIL		> = 80
by ELECTRO-OPTICS SIGNAL & COMPUTER ALOGRITHM	=		
.,			

**TOTAL PER EJACULATION** 



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TOTAL SPERM NUMBER by ELECTRO-OPTICS SIGNAL & COMPUTER ALOGRITHM	3.2	Millions/ejc.	> = 39.0
TOTAL MOTILE SPERM  by electro-optics signal & computer alogrithm	N.A	Millions/ejc.	> = 16.0
TOTAL PROGRESSIVE MOTILE SPERM by electro-optics signal & computer alogrithm	N.A	Millions/ejc.	> = 12.0
TOTAL FUNCTIONAL SPERM by ELECTRO-OPTICS SIGNAL & COMPUTER ALOGRITHM	N.A	Millions/ejc.	
TOTAL MORPHOLOGY NORMAL SPERM by ELECTRO-OPTICS SIGNAL & COMPUTER ALOGRITHM MANUAL MICROSCOPY AND MORPHOLOGY	N.A	Millions/ejc.	> = 2.0
VITALITY by MICROSCOPY	66	%	
RED BLOOD CELLS (RBCs) by MICROSCOPY	NOT DETECTED	/HPF	NOT DETECTED
PUS CELLS by MICROSCOPY	3-6	/HPF	0 - 5
AGGLUTINATES by MICROSCOPY	NOT DETECTED		NOT DETECTED
AMORPHOUS DEPOSITS/ROUND CELLS/DEBRIS by microscopy	NOT DETECTED		NOT DETECTED
BACTERIA by MICROSCOPY	NEGATIVE (-ve)		NEGATIVE (-ve)
CHEMICAL EXAMINATION			
SEMEN FRUCTOSE (QUALITATIVE) by QUALITATIVE METHOD USING RESORCINOL INTERPRETATION:	POSITIVE (+ve)		POSITIVE (+ve)

1.Fructose is the energy source for sperm motility. A positive fructose is considered normal.

2.Azoospermia and fructose negative results may indicate an absence of seminal vesicles / vas deferens in the area of seminal vesicles / obstruction of seminal vesicles.

\*\*\* End Of Report \*\*\*



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