Dr. Vinay Chopra
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NAME	**: Mrs. SARLA DEVI**		
AGE/ GENDER	: 41 YRS/FEMALE	**PATIENT ID**	: 1535746
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 012407020030**
REFERRED BY	: CIVIL HOSPITAL (AMBALA CANTT)	**REGISTRATION DATE**	: 02/Jul/2024 10:38 AM
BARCODE NO.	: 01512374	**COLLECTION DATE**	: 02/Jul/2024 10:39AM
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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)*	10.01	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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FOLLICLE STIMULATING HORMONE (FSH)

FOLLICLE STIMULATING HORMONE (FSH): SERUM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	19.7	mIU/mL	FEMALE FOLLICULAR PHASE: 3.03 - 8.08 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.
3. FSH appears to control gametogenesis in both males and females.

The test is useful in the following settings:

1. An adjunct in the evaluation of menstrual irregularities.
2. Evaluating patients with suspected hypogonadism.
3. 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:

1. Primary gonadal failure
2. Complete testicular feminization syndrome.
3. Precocious puberty (either idiopathic or secondary to a central nervous system lesion)
4. 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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Test Name	Value	Unit	Biological Reference interval

ESTRADIOL (E2)

ESTRADIOL (E2): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	< 10	pg/mL	FEMALE FOLLICULAR PHASE: 19.5 - 144.2 FEMALE MID CYCLE PHASE: 63.9 - 356.7 FEMALE PRE OVULATORY PHASE: 136.0 - 251.0 FEMALE LUTEAL PHASE: 55.8 - 214.2 POST MENOPAUSAL:< 50.0

INTEPRETATION:

OTHER MATERNAL FACTORS AND PREGNANCY	UNITS	RANGE
Hormonal Contraceptives	pg/mL	15.0 – 95.0
1st Trimester (0 – 12 Weeks)	pg/mL	38.0 – 3175.0
2nd Trimester (13 – 28 Weeks)	pg/mL	678.0 – 16633.0
3rd Trimester (29 – 40 Weeks)	pg/mL	43.0 – 33781.0
Post Menopausal	Pg/mL	< 50.0
MALES:	pg/mL	< 40.0

1. Estrogens are involved in development and maintenance of the female phenotype,germ cell maturation,and pregnancy.They also are important for many other,nongender-specific processes, including growth,nervous system maturation,bone metabolism/remodeling,and endothelial responsiveness.
2. E2 is produced primarily in ovaries and testes by aromatization of testosterone.
3. Small amounts are produced in the adrenal glands and some peripheral tissues,most notably fat.E2 levels in premenopausal women fluctuate during the menstrual cycle.
4. They are lowest during the early follicular phase. E2 levels then rise gradually until 2 to 3 days before ovulation, at which stage they start to increase much more rapidly and peak just before the ovulation-inducing luteinizing hormone (LH)/follicle stimulating hormone (FSH) surge at 5 to 10 times the early follicular levels.This is followed by a modest decline during the ovulatory phase.E2 levels then increase again gradually until the midpoint of the luteal phase and thereafter decline to trough,early follicular levels.

INDICATIONS FOR ASSAY: -

1. Evaluation of hypogonadism and oligo-amenorrhea in females.
2. Assessing ovarian status, including follicle development,for assisted reproduction protocols (eg, in vitro fertilization)
3. In conjunction with lutenizing hormone measurements,monitoring of estrogen replacement therapy in hypogonadal premenopausal women
4. Evaluation of feminization,including gynecomastia,in males.
5. Diagnosis of estrogen-producing neoplasms in males,and,to a lesser degree,females
6. As part of the diagnosis and work-up of precocious and delayed puberty in females,and,to a lesser degree, males

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7. As part of the diagnosis and work-up of suspected disorders of sex steroid metabolism,eg:aromatase deficiency and 17 alpha-hydroxylase deficiency
8. As an adjunct to clinical assessment,imaging studies and bone mineral density measurement in the fracture risk assessment of postmenopausal women,and,to a lesser degree,older men
9. Monitoring low-dose female hormone replacement therapy in post-menopausal women
10. Monitoring antiestrogen therapy (eg, aromatase inhibitor therapy).

CAUSES FOR INCREASED E2 LEVELS:

1. High androgen levels caused by tumors or androgen therapy (medical or sport performance enhancing),with secondary elevations in E1 and E2 due to aromatization
2. Obesity with increased tissue production of E1
3. Decreased E1 and E2 clearance in liver disease
4. Estrogen producing tumors
5. Estrogen Ingestion

*** End Of Report ***

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