NAME	**: Mrs. MADHU SHARMA**		
AGE/ GENDER	: 48 YRS/FEMALE	**PATIENT ID**	: 1781777
COLLECTED BY	: SURJESH	**REG. NO./LAB NO.**	**: 012503070039**
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval

CLINICAL CHEMISTRY/BIOCHEMISTRY

GLUCOSE FASTING (F)

GLUCOSE FASTING (F): PLASMA *by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)*	**117.89^{H}**	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0

INTERPRETATION

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

1. A fasting plasma glucose level below 100 mg/dl is considered normal.
2. A fasting plasma glucose level between 100 - 125 mg/dl is considered as glucose intolerant or prediabetic. A fasting and post-prandial blood test (after consumption of 75 gms of glucose) is recommended for all such patients.
3. A fasting plasma glucose level of above 125 mg/dl is highly suggestive of diabetic state. A repeat post-prandial is strongly recommended for all such patients. A fasting plasma glucose level in excess of 125 mg/dl on both occasions is confirmatory for diabetic state.

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Test Name	Value	Unit	Biological Reference interval

ENDOCRINOLOGY

THYROID STIMULATING HORMONE (TSH)

THYROID STIMULATING HORMONE (TSH): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)* **3rd GENERATION, ULTRASENSITIVE**	3.968	µIU/mL	0.35 - 5.50

INTERPRETATION:

AGE	REFFERENCE RANGE (µIU/mL)
0 – 5 DAYS	0.70 – 15.20
6 Days – 2 Months	0.70 – 11.00
3 – 11 Months	0.70 – 8.40
1 – 5 Years	0.70 – 7.00
6 – 10 Years	0.60 – 5.50
11 - 15	0.50 – 5.50
> 20 Years (Adults)	0.27 – 5.50
PREGNANCY	
1st Trimester	0.10 - 3.00
2nd Trimester	0.20 - 3.00
3rd Trimester	0.30 - 4.10

NOTE:-*TSH levels are subjected to circardian variation, reaching peak levels between 2-4 a.m and at a minimum between 6-10 pm. The variation is of the order of 50 %. Hence time of the day has influence on the measured serum TSH concentration.*

USE:- TSH controls biosynthesis and release of thyroid harmones T4 & T3. It is a sensitive measure of thyroid function, especially useful in early or subclinical hypothyroidism, before the patient develops any clinical findings or goitre or any other thyroid function abnormality.

INCREASED LEVELS:

1.Primary or untreated hypothyroidism, may vary from 3 times to more than 100 times normal depending on degree of hypofunction.
2.Hypothyroid patients receiving insufficient thyroid replacement therapy.
3.Hashimotos thyroiditis.
4.DRUGS: Amphetamines, Iodine containing agents and dopamine antagonist.
5.Neonatal period, increase in 1st 2-3 days of life due to post-natal surge.

DECREASED LEVELS:

1.Toxic multi-nodular goitre & Thyroiditis.
2.Over replacement of thyroid harmone in treatment of hypothyroidism.
3.Autonomously functioning Thyroid adenoma
4.Secondary pituatary or hypothalmic hypothyroidism
5.Acute psychiatric illness
6.Severe dehydration.
7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.

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Test Name	Value	Unit	Biological Reference interval

8.Pregnancy: 1st and 2nd Trimester

LIMITATIONS:

1.TSH may be normal in central hypothyroidism, recent rapid correction of hyperthyroidism or hypothyroidism, pregnancy, phenytoin therapy.

2.Autoimmune disorders may produce spurious results.

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Test Name	Value	Unit	Biological Reference interval

LUTEINISING HORMONE (LH)

LUTEINISING HORMONE (LH): SERUM *by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)*	16.1	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

FOLLICLE STIMULATING HORMONE (FSH)

FOLLICLE STIMULATING HORMONE (FSH): SERUM *by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)*	20.79	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
7. As part of the diagnosis and work-up of suspected disorders of sex steroid metabolism,eg:aromatase deficiency and 17 alpha-hydroxylase

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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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