



# P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

**A PIONEER DIAGNOSTIC CENTRE**

☎ 0171-2532620, 8222896961 ✉ pkrjainhealthcare@gmail.com

**NAME** : Mrs. JASVINDER KAUR  
**AGE/ GENDER** : 30 YRS/FEMALE  
**COLLECTED BY** :  
**REFERRED BY** :  
**BARCODE NO.** : 12504837  
**CLIENT CODE.** : P.K.R JAIN HEALTHCARE INSTITUTE  
**CLIENT ADDRESS** : NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA

**PATIENT ID** : 1620182  
**REG. NO./LAB NO.** : 122409210009  
**REGISTRATION DATE** : 21/Sep/2024 09:19 AM  
**COLLECTION DATE** : 21/Sep/2024 09:23 AM  
**REPORTING DATE** : 21/Sep/2024 01:52 PM

Test Name	Value	Unit	Biological Reference interval
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## HAEMATOLOGY

### ERYTHROCYTE SEDIMENTATION RATE (ESR)

ERYTHROCYTE SEDIMENTATION RATE (ESR)	10	mm/1st hr	0 - 20
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by RED CELL AGGREGATION BY CAPILLARY PHOTOMETRY

#### INTERPRETATION:

1. ESR is a non-specific test because an elevated result often indicates the presence of inflammation associated with infection, cancer and auto-immune disease, but does not tell the health practitioner exactly where the inflammation is in the body or what is causing it.
2. An ESR can be affected by other conditions besides inflammation. For this reason, the ESR is typically used in conjunction with other test such as C-reactive protein
3. This test may also be used to monitor disease activity and response to therapy in both of the above diseases as well as some others, such as systemic lupus erythematosus

#### CONDITION WITH LOW ESR

A low ESR can be seen with conditions that inhibit the normal sedimentation of red blood cells, such as a high red blood cell count (polycythaemia), significantly high white blood cell count (leucocytosis), and some protein abnormalities. Some changes in red cell shape (such as sickle cells in sickle cell anaemia) also lower the ESR.

#### NOTE:

1. ESR and C - reactive protein (C-RP) are both markers of inflammation.
2. Generally, ESR does not change as rapidly as does CRP, either at the start of inflammation or as it resolves.
3. **CRP is not affected by as many other factors as is ESR, making it a better marker of inflammation.**
4. If the ESR is elevated, it is typically a result of two types of proteins, globulins or fibrinogen.
5. Women tend to have a higher ESR, and menstruation and pregnancy can cause temporary elevations.
6. Drugs such as dextran, methyldopa, oral contraceptives, penicillamine procainamide, theophylline, and vitamin A can increase ESR, while aspirin, cortisone, and quinine may decrease it



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## ENDOCRINOLOGY

### THYROID FUNCTION TEST: TOTAL

TRIIODOTHYRONINE (T3): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	1.27	ng/mL	0.35 - 1.93
THYROXINE (T4): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	6.19	µgm/dL	4.87 - 12.60
THYROID STIMULATING HORMONE (TSH): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	1.18	µIU/mL	0.35 - 5.50

3rd GENERATION, ULTRASENSITIVE

#### INTERPRETATION:

TSH levels are subject to circadian 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 concentrations.TSH stimulates the production and secretion of the metabolically active hormones, thyroxine (T4)and triiodothyronine (T3).Failure at any level of regulation of the hypothalamic-pituitary-thyroid axis will result in either underproduction (hypothyroidism) or overproduction(hyperthyroidism) of T4 and/or T3.

CLINICAL CONDITION	T3	T4	TSH
Primary Hypothyroidism:	Reduced	Reduced	Increased (Significantly)
Subclinical Hypothyroidism:	Normal or Low Normal	Normal or Low Normal	High
Primary Hyperthyroidism:	Increased	Increased	Reduced (at times undetectable)
Subclinical Hyperthyroidism:	Normal or High Normal	Normal or High Normal	Reduced

#### LIMITATIONS:-

1. T3 and T4 circulates in reversibly bound form with Thyroid binding globulins (TBG),and to a lesser extent albumin and Thyroid binding Pre Albumin so conditions in which TBG and protein levels alter such as pregnancy, excess estrogens, androgens, anabolic steroids and glucocorticoids may falsely affect the T3 and T4 levels and may cause false thyroid values for thyroid function tests.
2. Normal levels of T4 can also be seen in Hyperthyroid patients with :T3 Thyrotoxicosis, Decreased binding capacity due to hypoproteinemia or ingestion of certain drugs (eg: phenytoin , salicylates).
3. Serum T4 levels in neonates and infants are higher than values in the normal adult , due to the increased concentration of TBG in neonate serum.
4. TSH may be normal in central hypothyroidism , recent rapid correction of hyperthyroidism or hypothyroidism , pregnancy , phenytoin therapy.

TRIIODOTHYRONINE (T3)		THYROXINE (T4)		THYROID STIMULATING HORMONE (TSH)	
Age	Refferance Range (ng/mL)	Age	Refferance Range ( µg/dL)	Age	Reference Range ( µIU/mL)
0 - 7 Days	0.20 - 2.65	0 - 7 Days	5.90 – 18.58	0 - 7 Days	2.43 - 24.3
7 Days - 3 Months	0.36 - 2.59	7 Days - 3 Months	6.39 - 17.66	7 Days - 3 Months	0.58 - 11.00
3 - 6 Months	0.51 - 2.52	3 - 6 Months	6.75 – 17.04	3 Days – 6 Months	0.70 - 8.40



  
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6 - 12 Months	0.74 - 2.40	6 - 12 Months	7.10 - 16.16	6 - 12 Months	0.70 - 7.00
1 - 10 Years	0.92 - 2.28	1 - 10 Years	6.00 - 13.80	1 - 10 Years	0.60 - 5.50
11- 19 Years	0.35 - 1.93	11 - 19 Years	4.87- 13.20	11 - 19 Years	0.50 - 5.50
> 20 years (Adults)	0.35 - 1.93	> 20 Years (Adults)	4.87 - 12.60	> 20 Years (Adults)	0.35- 5.50

#### RECOMMENDATIONS OF TSH LEVELS DURING PREGNANCY ( $\mu$ U/mL)

1st Trimester	0.10 - 2.50
2nd Trimester	0.20 - 3.00
3rd Trimester	0.30 - 4.10

#### INCREASED TSH LEVELS:

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

#### DECREASED TSH LEVELS:

- 1.Toxic multi-nodular goitre & Thyroiditis.
- 2.Over replacement of thyroid hormone in treatment of hypothyroidism.
- 3.Autonomously functioning Thyroid adenoma
- 4.Secondary pituitary or hypothalamic hypothyroidism
- 5.Acute psychiatric illness
- 6.Severe dehydration.
- 7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.
- 8.Pregnancy: 1st and 2nd Trimester



  
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## LUTEINISING HORMONE (LH)

LUTEINISING HORMONE (LH): SERUM by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)	5.02	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
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### INTERPRETATION:

- 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.
- 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.
- 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.
- 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:

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

### FSH AND LH ELEVATED IN:

- Primary gonadal failure
- Complete testicular feminization syndrome
- Precocious puberty (either idiopathic or secondary to a central nervous system lesion)
- Menopause
- Primary ovarian hypo dysfunction in females
- Polycystic ovary disease in females
- Primary hypogonadism in males


### LH IS DECREASED IN:


- Primary ovarian hyper function in females
- Primary hypergonadism in males

### NOTE

- 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)	5.19	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
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### 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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## PROLACTIN

PROLACTIN: SERUM 17.29 ng/mL 3 - 25

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

### INTERPRETATION:

1. Prolactin is secreted by the anterior pituitary gland and controlled by the hypothalamus.  
2. The major chemical controlling prolactin secretion is dopamine, which inhibits prolactin secretion from the pituitary.  
3. Physiological function of prolactin is the stimulation of milk production. In normal individuals, the prolactin level rises in response to physiologic stimuli such as sleep, exercise, nipple stimulation, sexual intercourse, hypoglycemia, postpartum period, and also is elevated in the newborn infant.

### INCREASED (HYPERPROLACTEMIA):

1. Prolactin-secreting pituitary adenoma (prolactinoma, which is 5 times more frequent in females than males).  
2. Functional and organic disease of the hypothalamus.  
3. Primary hypothyroidism.  
4. Section compression of the pituitary stalk.  
5. Chest wall lesions and renal failure.  
6. Ectopic tumors.

7. DRUGS:- Anti-Dopaminergic drugs like antipsychotic drugs, anti-nausea/antiemetic drugs, Drugs that affect CNS serotonin metabolism, serotonin receptors, or serotonin reuptake (anti-depressants of all classes, ergot derivatives, some illegal drugs such as cannabis), Antihypertensive drugs, Opiates, High doses of estrogen or progesterone, anticonvulsants (valproic acid), anti-tuberculous medications (Isoniazid).

### SIGNIFICANCE:

1. In loss of libido, galactorrhea, oligomenorrhea, hyperprolactinemia often results in amenorrhea or menorrhagia, and infertility in premenopausal females.  
2. Loss of libido, impotence, infertility, and hypogonadism in males. Postmenopausal and premenopausal women, as well as men, can also suffer from decreased muscle mass and osteoporosis.  
3. In males, prolactin levels >13 ng/mL are indicative of hyperprolactinemia.  
4. In women, prolactin levels >27 ng/mL in the absence of pregnancy and postpartum lactation are indicative of hyperprolactinemia.  
5. Clear symptoms and signs of hyperprolactinemia are often absent in patients with serum prolactin levels <100 ng/mL.  
4. Mild to moderately increased levels of serum prolactin are not a reliable guide for determining whether a prolactin-producing pituitary adenoma is present, 5. Whereas levels >250 ng/mL are usually associated with a prolactin-secreting tumor.

### CAUTION:

Prolactin values that exceed the reference values may be due to macroprolactin (prolactin bound to immunoglobulin). Macroprolactin should be evaluated if signs and symptoms of hyperprolactinemia are absent, or pituitary imaging studies are not informative.



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## ESTRADIOL (E2)

ESTRADIOL (E2): SERUM  
by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

67

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

### INTERPRETATION:

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
<b>MALES:</b>	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 luteinizing 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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