



P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

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

NAME	: Mrs. RITU	PATIENT ID	: 1588839
AGE/ GENDER	: 31 YRS/FEMALE	REG. NO./LAB NO.	: 122408230013
COLLECTED BY	:	REGISTRATION DATE	: 23/Aug/2024 12:38 PM
REFERRED BY	:	COLLECTION DATE	: 23/Aug/2024 12:51PM
BARCODE NO.	: 12504278	REPORTING DATE	: 23/Aug/2024 03:04PM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE		
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA		

Test Name	Value	Unit	Biological Reference interval
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HAEMATOLOGY COMPLETE BLOOD COUNT (CBC)

RED BLOOD CELLS (RBCS) COUNT AND INDICES

HAEMOGLOBIN (HB) <i>by CALORIMETRIC</i>	13.1	gm/dL	12.0 - 16.0
RED BLOOD CELL (RBC) COUNT <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	4.39	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	38.1	%	37.0 - 50.0
MEAN CORPUSCULAR VOLUME (MCV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	86.8	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	29.7	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	34.3	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	13.8	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	46.6	fL	35.0 - 56.0
MENTZERS INDEX <i>by CALCULATED</i>	19.77	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX <i>by CALCULATED</i>	27.16	RATIO	BETA THALASSEMIA TRAIT: <= 65.0 IRON DEFICIENCY ANEMIA: > 65.0

WHITE BLOOD CELLS (WBCS)

TOTAL LEUCOCYTE COUNT (TLC) <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	5910	/cmm	4000 - 11000
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DIFFERENTIAL LEUCOCYTE COUNT (DLC)

NEUTROPHILS <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	57	%	50 - 70
LYMPHOCYTES <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	38	%	20 - 40
EOSINOPHILS <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	0 ^L	%	1 - 6




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MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	5	%	2 - 12
BASOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	%	0 - 1
ABSOLUTE LEUKOCYTES (WBC) COUNT			
ABSOLUTE NEUTROPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	3369	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2246 ^L	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0 ^L	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	296	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110
PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.			
PLATELET COUNT (PLT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	101000 ^L	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.16	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	15 ^H	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	62000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	61.9 ^H	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	16.3	%	15.0 - 17.0

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD




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ERYTHROCYTE SEDIMENTATION RATE (ESR)

ERYTHROCYTE SEDIMENTATION RATE (ESR) 21^H mm/1st hr 0 - 20
by MODIFIED WESTERGREN AUTOMATED METHOD

INTERPRETATION:

1. ESR is a non-specific test because an elevated result often indicates the presence of inflammation associated with infection, cancer and autoimmune 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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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

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CLINICAL CHEMISTRY/BIOCHEMISTRY

GLUCOSE RANDOM (R)

GLUCOSE RANDOM (R): PLASMA
by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)

180.88^H

mg/dL

NORMAL: < 140.00
PREDIABETIC: 140.0 - 200.0
DIABETIC: > OR = 200.0

INTERPRETATION

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

1. A random plasma glucose level below 140 mg/dl is considered normal.
2. A random glucose level between 140 - 200 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 random glucose level of above 200 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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ENDOCRINOLOGY

THYROID STIMULATING HORMONE (TSH)

THYROID STIMULATING HORMONE (TSH): SERUM 4.286 μ IU/mL 0.35 - 5.50
by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

3rd GENERATION, ULTRASENSITIVE

INTERPRETATION:

AGE	REFERENCE RANGE (μ IU/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 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 concentration.

USE:- TSH controls biosynthesis and release of thyroid hormones 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 hormone in treatment of hypothyroidism.
- 3.Autonomously functioning Thyroid adenoma
- 4.Secondary pituitary or hypothalamic hypothyroidism
- 5.Acute psychiatric illness
- 6.Severe dehydration.



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7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.

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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LUTEINISING HORMONE (LH)

LUTEINISING HORMONE (LH): SERUM 4.05 mIU/mL
by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

MALES: 1.0 -12.5
FOLLICULAR PHASE: 1.2 - 12.7
MID-CYCLE PEAK: 15.5 - 90.0
LUTEAL PHASE: 0.50 - 14.6
POST MENOPAUSAL: 15.6 - 72.0

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 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 adjunct in 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 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
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)	7.4	mIU/mL	FEMALE FOLLICULAR PHASE: 2.5 - 11.4 FEMALE MID-CYCLE PEAK: 3.3 - 21.7 FEAMLE LUTEAL PHASE: 1.2 - 7.0 FEMALE POST-MENOPAUSAL: 18.8 - 132 MALE: 1.0 - 12.1
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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 11.149 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 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.

*** End Of Report ***



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