



P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

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

NAME	: Miss. RAVINDER KAUR	PATIENT ID	: 1769537
AGE/ GENDER	: 38 YRS/FEMALE	REG. NO./LAB NO.	: 122502250013
COLLECTED BY	:	REGISTRATION DATE	: 25/Feb/2025 11:45 AM
REFERRED BY	:	COLLECTION DATE	: 25/Feb/2025 12:58PM
BARCODE NO.	: 12507223	REPORTING DATE	: 25/Feb/2025 04:25PM
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>	12.9	gm/dL	12.0 - 16.0
RED BLOOD CELL (RBC) COUNT <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	5.15 ^H	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	40.3	%	37.0 - 50.0
MEAN CORPUSCULAR VOLUME (MCV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	78.3 ^L	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	25 ^L	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	31.9 ^L	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	16.1 ^H	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	47.7	fL	35.0 - 56.0
MENTZERS INDEX <i>by CALCULATED</i>	15.2	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX <i>by CALCULATED</i>	24.43	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>	5180	/cmm	4000 - 11000
NUCLEATED RED BLOOD CELLS (nRBCS) <i>by AUTOMATED 6 PART HEMATOLOGY ANALYZER</i>	0		0.00 - 20.00
NUCLEATED RED BLOOD CELLS (nRBCS) % <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	0	%	< 10 %




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CORRECTED TOTAL LEUCOCYTE COUNT (C-TLC) <i>by MICROSCOPY ON EDTA SMEAR</i>	5180	/cmm	4000 - 11000
<u>DIFFERENTIAL LEUCOCYTE COUNT (DLC)</u>			
NEUTROPHILS <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	62	%	50 - 70
LYMPHOCYTES <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	26	%	20 - 40
EOSINOPHILS <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	2	%	1 - 6
MONOCYTES <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	10	%	2 - 12
BASOPHILS <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	0	%	0 - 1
IMMATURE GRANULOCYTE (IG) % <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	0	%	0 - 5.0
<u>ABSOLUTE LEUKOCYTES (WBC) COUNT</u>			
ABSOLUTE NEUTROPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	3212	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	1347 ^L	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	104	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	518	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	0	/cmm	0 - 110
ABSOLUTE IMMATURE GRANULOCYTE COUNT <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	0	/cmm	0.0 - 999.0
<u>PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.</u>			
PLATELET COUNT (PLT) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	93000 ^L	/cmm	150000 - 450000
PLATELETCRIT (PCT) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	0.15	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	17 ^H	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	67000	/cmm	30000 - 90000



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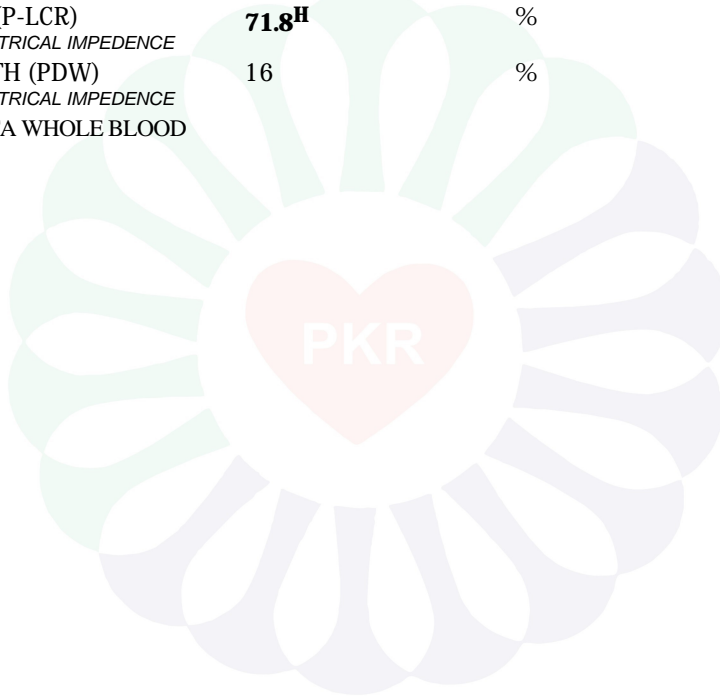
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PLATELET LARGE CELL RATIO (P-LCR) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	71.8 ^H	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	16	%	15.0 - 17.0

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD




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GLYCOSYLATED HAEMOGLOBIN (HbA1C)

GLYCOSYLATED HAEMOGLOBIN (HbA1c): WHOLE BLOOD by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)	5	%	4.0 - 6.4
ESTIMATED AVERAGE PLASMA GLUCOSE by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)	96.8	mg/dL	60.00 - 140.00

INTERPRETATION:

AS PER AMERICAN DIABETES ASSOCIATION (ADA):

REFERENCE GROUP	GLYCOSYLATED HEMOGLOBIN (HbA1C) in %	
Non diabetic Adults >= 18 years	<5.7	
At Risk (Prediabetes)	5.7 – 6.4	
Diagnosing Diabetes	>= 6.5	
Therapeutic goals for glycemic control	Age > 19 Years	
	Goals of Therapy:	< 7.0
	Actions Suggested:	>8.0
	Age < 19 Years	
	Goal of therapy:	<7.5

COMMENTS:

- Glycosylated hemoglobin (HbA1c) test is three monthly monitoring done to assess compliance with therapeutic regimen in diabetic patients.
- Since Hb1c reflects long term fluctuations in blood glucose concentration, a diabetic patient who has recently under good control may still have high concentration of HbA1c. Converse is true for a diabetic previously under good control but now poorly controlled.
- Target goals of < 7.0 % may be beneficial in patients with short duration of diabetes, long life expectancy and no significant cardiovascular disease. In patients with significant complications of diabetes, limited life expectancy or extensive co-morbid conditions, targeting a goal of < 7.0% may not be appropriate.
- High HbA1c (>9.0 -9.5 %) is strongly associated with risk of development and rapid progression of microvascular and nerve complications
- Any condition that shorten RBC life span like acute blood loss, hemolytic anemia falsely lower HbA1c results.
- HbA1c results from patients with HbSS, HbSC and HbD must be interpreted with caution, given the pathological processes including anemia, increased red cell turnover, and transfusion requirement that adversely impact HbA1c as a marker of long-term glycemic control.
- Specimens from patients with polycythemia or post-splenectomy may exhibit increase in HbA1c values due to a somewhat longer life span of the red cells.




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ENDOCRINOLOGY

THYROID STIMULATING HORMONE (TSH)

THYROID STIMULATING HORMONE (TSH): SERUM 2.27 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.
- 7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.



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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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VITAMINS

VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM
by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

10.4^L

ng/mL

DEFICIENCY: < 20.0
INSUFFICIENCY: 20.0 - 30.0
SUFFICIENCY: 30.0 - 100.0
TOXICITY: > 100.0

INTERPRETATION:

DEFICIENT:	< 20	ng/mL
INSUFFICIENT:	21 - 29	ng/mL
PREFERRED RANGE:	30 - 100	ng/mL
INTOXICATION:	> 100	ng/mL

- Vitamin D compounds are derived from dietary ergocalciferol (from plants, Vitamin D2), or cholecalciferol (from animals, Vitamin D3), or by conversion of 7- dihydrocholecalciferol to Vitamin D3 in the skin upon Ultraviolet exposure.
- 25-OH--Vitamin D represents the main body reservoir and transport form of Vitamin D and transport form of Vitamin D, being stored in adipose tissue and tightly bound by a transport protein while in circulation.
- Vitamin D plays a primary role in the maintenance of calcium homeostasis. It promotes calcium absorption, renal calcium absorption and phosphate reabsorption, skeletal calcium deposition, calcium mobilization, mainly regulated by parathyroid hormone (PTH).
- Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults.

DECREASED:

- Lack of sunshine exposure.
- Inadequate intake, malabsorption (celiac disease)
- Depressed Hepatic Vitamin D 25- hydroxylase activity
- Secondary to advanced Liver disease
- Osteoporosis and Secondary Hyperparathyroidism (Mild to Moderate deficiency)
- Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism.

INCREASED:

- Hypervitaminosis D is Rare, and is seen only after prolonged exposure to extremely high doses of Vitamin D. When it occurs, it can result in severe hypercalcemia and hyperphosphatemia.

CAUTION: Replacement therapy in deficient individuals must be monitored by periodic assessment of Vitamin D levels in order to prevent hypervitaminosis D

NOTE:- Dark coloured individuals as compare to whites, is at higher risk of developing Vitamin D deficiency due to excess of melanin pigment which interfere with Vitamin D absorption.



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TUMOUR MARKER

CANCER ANTIGEN 125 (CA 125): OVARIAN CANCER MARKER

CANCER ANTIGEN (CA) -125: SERUM	26.3	U/mL	0.0 - 35.0
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by CMIA (CHEMILUMINESCENCE MICROPARTICLE IMMUNOASSAY)

INTERPRETATION:

1. Cancer antigen 125 (CA 125) is a glycoprotein antigen normally expressed in tissues derived from coelomic epithelia (ovary, fallopian tube, peritoneum, pleura, pericardium, colon, kidney, stomach).
2. Serum CA 125 is elevated in approximately 80% of women with advanced epithelial ovarian cancer, but assay sensitivity is suboptimal in early disease stages. The average reported sensitivities are 50% for stage I and 90% for stage II or greater.
3. Elevated serum CA 125 levels have been reported in individuals with a variety of nonovarian malignancies including cervical, liver, pancreatic, lung, colon, stomach, biliary tract, uterine, fallopian tube, breast, and endometrial carcinomas.

SIGNIFICANCE:

1. Evaluating patients' response to cancer therapy, especially for ovarian carcinoma
2. Predicting recurrent ovarian cancer or intra-peritoneal tumor. In monitoring studies, elevations of cancer antigen 125 (CA 125) >35 U/mL after de-bulking surgery and chemotherapy indicate that residual disease is likely (>95% accuracy). However, normal levels do not rule-out recurrence.
3. A persistently rising CA 125 value suggests progressive malignant disease and poor therapeutic response.
4. Physiologic half-life of CA 125 is approximately 5 days.
5. In patients with advanced disease who have undergone cyto-reductive surgery and are on chemotherapy, a prolonged half-life (>20 days) may be associated with a shortened disease-free survival.

NOTE:

1. CA 125 levels. Hence this assay, regardless of level, should not be interpreted as absolute evidence for the presence or absence of malignant disease. The assay value should be used in conjunction with findings from clinical evaluation and other diagnostic procedures. It is not recommended to use this test for the initial diagnosis of ovarian cancer.
2. Falsely Elevated serum CA 125 levels have been reported in individuals with a variety of nonmalignant conditions including: cirrhosis, hepatitis, endometriosis, first trimester pregnancy, ovarian cysts, and pelvic inflammatory disease. Elevated levels during the menstrual cycle also have been reported.

*** End Of Report ***



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