P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

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NAME	**: Mrs. HARJINDER KAUR**		
AGE/ GENDER	: 50 YRS/FEMALE	**PATIENT ID**	: 1794481
COLLECTED BY	:	**REG. NO./LAB NO.**	**: 122503170026**
REFERRED BY	:	**REGISTRATION DATE**	: 17/Mar/2025 02:01 PM
BARCODE NO.	: 12507553	**COLLECTION DATE**	: 17/Mar/2025 02:01PM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE	**REPORTING DATE**	: 17/Mar/2025 04:22PM
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HAEMATOLOGY

COMPLETE BLOOD COUNT (CBC)

Test Name	Value	Unit	Biological Reference interval
RED BLOOD CELLS (RBCS) COUNT AND INDICES			
HAEMOGLOBIN (HB) *by CALORIMETRIC*	12.5	gm/dL	12.0 - 16.0
RED BLOOD CELL (RBC) COUNT *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	4.04	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	**35.4^{L}**	%	37.0 - 50.0
MEAN CORPUSCULAR VOLUME (MCV) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	87.6	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	30.8	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	35.2	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	12.7	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	43.1	fL	35.0 - 56.0
MENTZERS INDEX *by CALCULATED*	21.68	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX *by CALCULATED*	27.41	RATIO	BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CELLS (WBCS)			
TOTAL LEUCOCYTE COUNT (TLC) *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	10080	/cmm	4000 - 11000
NUCLEATED RED BLOOD CELLS (nRBCS) *by AUTOMATED 6 PART HEMATOLOGY ANALYZER*	NIL		0.00 - 20.00
NUCLEATED RED BLOOD CELLS (nRBCS) % *by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER*	NIL	%	< 10 %

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Test Name	Value	Unit	Biological Reference interval
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	65	%	50 - 70
LYMPHOCYTES *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	29	%	20 - 40
EOSINOPHILS *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	2	%	1 - 6
MONOCYTES *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	4	%	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*	6552	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	**2923^{L}**	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT *by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY*	202	/cmm	40 - 440
PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.			
PLATELET COUNT (PLT) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	245000	/cmm	150000 - 450000
PLATELETCRIT (PCT) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	0.25	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	10	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	74000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	30	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) *by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE*	16.2	%	15.0 - 17.0

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

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Test Name	Value	Unit	Biological Reference interval

GLYCOSYLATED HAEMOGLOBIN (HBA1C)

GLYCOSYLATED HAEMOGLOBIN (HbA1c): WHOLE BLOOD *by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)*	**7.9**^{H}	%	4.0 - 6.4
ESTIMATED AVERAGE PLASMA GLUCOSE *by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)*	**180.03**^{H}	mg/dL	60.00 - 140.00

INTERPRETATION:

AS PER AMERICAN DIABETES ASSOCIATION (ADA):		
REFERENCE GROUP	**GLYCOSYLATED HEMOGLOGIB (HBAIC) 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:

1.Glycosylated hemoglobin (HbA1c) test is three monthly monitoring done to assess compliace with therapeutic regimen in diabetic patients.

2.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 HbAlc. Converse is true for a diabetic previously under good control but now poorly controlled.

3.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, targetting a goal of < 7.0% may not be appropiate.

4.High HbA1c (>9.0 -9.5 %) is strongly associated with risk of development and rapid progression of microvascular and nerve complications

5.Any condition that shorten RBC life span like acute blood loss, hemolytic anemia falsely lower HbA1c results.

6.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 gycemic control.

7.Specimens from patients with polycythemia or post-splenctomy may exhibit increse in HbA1c values due to a somewhat longer life span of the red cells.

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Test Name	Value	Unit	Biological Reference interval

ERYTHROCYTE SEDIMENTATION RATE (ESR)

ERYTHROCYTE SEDIMENTATION RATE (ESR)	6	mm/1st hr	0 - 20

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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Test Name	Value	Unit	Biological Reference interval
CLINICAL CHEMISTRY/BIOCHEMISTRY			
KIDNEY FUNCTION TEST (BASIC)			
UREA: SERUM *by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)*	40.46	mg/dL	10.00 - 50.00
CREATININE: SERUM *by ENZYMATIC, SPECTROPHOTOMETERY*	1.15	mg/dL	0.40 - 1.20
BLOOD UREA NITROGEN (BUN): SERUM *by CALCULATED, SPECTROPHOTOMETERY*	18.91	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETERY*	16.44	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM *by CALCULATED, SPECTROPHOTOMETERY*	35.18	RATIO	
URIC ACID: SERUM *by URICASE - OXIDASE PEROXIDASE*	3.85	mg/dL	2.50 - 6.80

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Test Name	Value	Unit	Biological Reference interval

INTERPRETATION:

Normal range for a healthy person on normal diet: 12 - 20

To Differentiate between pre- and postrenal azotemia.

INCREASED RATIO (>20:1) WITH NORMAL CREATININE:

1.Prerenal azotemia (BUN rises without increase in creatinine) e.g. heart failure, salt depletion,dehydration, blood loss) due to decreased glomerular filtration rate.
2.Catabolic states with increased tissue breakdown.
3.GI hemorrhage.
4.High protein intake.
5.Impaired renal function plus .
6.Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushings syndrome, high protein diet, burns,surgery, cachexia, high fever).
7.Urine reabsorption (e.g. ureterocolostomy)
8.Reduced muscle mass (subnormal creatinine production)
9.Certain drugs (e.g. tetracycline, glucocorticoids)

INCREASED RATIO (>20:1) WITH ELEVATED CREATININE LEVELS:

1.Postrenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).
2.Prerenal azotemia superimposed on renal disease.

DECREASED RATIO (<10:1) WITH DECREASED BUN :

1.Acute tubular necrosis.
2.Low protein diet and starvation.
3.Severe liver disease.
4.Other causes of decreased urea synthesis.
5.Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid).
6.Inherited hyperammonemias (urea is virtually absent in blood).
7.SIADH (syndrome of inappropiate antidiuretic harmone) due to tubular secretion of urea.
8.Pregnancy.

DECREASED RATIO (<10:1) WITH INCREASED CREATININE:

1.Phenacimide therapy (accelerates conversion of creatine to creatinine).
2.Rhabdomyolysis (releases muscle creatinine).
3.Muscular patients who develop renal failure.

INAPPROPIATE RATIO:

1.Diabetic ketoacidosis (acetoacetate causes false increase in creatinine with certain methodologies,resulting in normal ratio when dehydration should produce an increased BUN/creatinine ratio).
2.Cephalosporin therapy (interferes with creatinine measurement).

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Test Name	Value	Unit	Biological Reference interval
HOMOCYSTEINE			
HOMOCYSTEINE: SERUM *by SPECTROPHOTOMETRY*	**20.1^{H}**	µmol/L	3.0 - 18.0

INTERPRETATION:

1.Homocysteine is a sulphur containing amino acid. There is an association between elevated levels of circulating homocysteine and various vascular and cardiovascular disorders
2.Serum Homocystein level aid in screening patients suspected of having an inherited disorder of methionine metabolism including genetic defects in vitamin cofactors (vitamin B6, B12, and folate).
3.Nutritional deficiency of B12 and folate also lead to abnormal homocysteine accumulation.
4.Homocysteine concentration is an indicator of acquired folate or cobalamin deficiency, and is a contributing factor in the pathogenesis of neural tube defects.
5.Homocystenemia was previously thought to be an independent risk factor for coronary artery disease but current understanding suggests that the use of homocysteine for assessment of cardiovascular risk is uncertain and controversial. Based on several meta-analyses, at present, homocysteine may be regarded as a weak risk factor for coronary heart disease, and there is a lack of direct causal relationship between hyperhomocysteinemia and cardiovascular disease. It is most likely an indicator of poor lifestyle and diet.
6.Specially useful in young CVD patients (< 40 yrs) In known cases of CVD, high homocysteine levels should be used as a prognostic marker for CVD events and mortality CVD patients with homocysteine levels > 15 umol/L belong to a high risk group Increased homocysteine levels with low vitamin concentrations should be handled as a potential vitamin deficiency case.
7.This test should be used in conjunction with plasma amino acids and urine organic acids to aid in the biochemical screening for primary and secondary disorders of methionine metabolism.
8.Note:-Homocysteine concentrations >13 mcmol/L are considered abnormal in patients evaluated for suspected nutritional deficiencies (B12, folate) and inborn errors of metabolism. Measurement of methylmalonic acid (MMA) distinguishes between B12 (cobalamin) and folate deficiencies, as MMA is only elevated in B12 deficiency. Response to dietary treatment can be evaluated by monitoring serum homocysteine concentrations over time.
9.Homocysteine concentrations < or =10 mcmol/L are desirable when utilized for cardiovascular risk.
10.Other factors that may influence and increase serum homocysteine include: Age, Smoking, Poor diet, Chronic renal,disease,Hypothyroidism

NOTE:

1.Medications that may increase homocysteine concentrations include: Methotrexate, Azuridine, Nitrous Oxide, Phenytoin, Carbamazepine, Oral Contraceptives
2.A fasting specimen is recommended; however, nonfasting homocysteine concentrations produce slightly higher, but likely clinically insignificant changes.

*** End Of Report ***

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