CLIENT CODE.

HAEMOCLORINI (HR)



# PKR JAIN HEALTHCARE INSTITUTE NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

### A PIONEER DIAGNOSTIC CENTRE

**■** 0171-2532620, 8222896961 ■ pkrjainhealthcare@gmail.com

: 10/Aug/2024 01:34PM

120 170

**NAME** : Mr. JASBIR SINGH

**AGE/ GENDER** : 59 YRS/MALE **PATIENT ID** : 1576525

**COLLECTED BY** REG. NO./LAB NO. : 122408100021

REFERRED BY **REGISTRATION DATE** : 10/Aug/2024 11:34 AM BARCODE NO. : 12504097 **COLLECTION DATE** : 10/Aug/2024 11:42AM

**CLIENT ADDRESS** : NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA

: P.K.R JAIN HEALTHCARE INSTITUTE

**Test Name** Value Unit **Biological Reference interval** 

# **HAEMATOLOGY**

REPORTING DATE

### **COMPLETE BLOOD COUNT (CBC)**

RED BLOOD C	ELLS (RBCS) CO	OUNT AND I	NDICES

HAEMOGLOBIN (HB)	14	gm/dL	12.0 - 17.0
by CALORIMETRIC  RED BLOOD CELL (RBC) COUNT	4.65	Millions/cmm	3.50 - 5.00
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	40.8	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV)  by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	87.7	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH)  by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	30.2	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	34.4	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	16.6 <sup>H</sup>	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD)  by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	55	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	18.86	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	31.4	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  DIFFERENTIAL LEUCOCYTE COUNT (DLC)	7730	/cmm	4000 - 11000
NEUTROPHILS	58	%	50 - 70
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
LYMPHOCYTES  by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	32	%	20 - 40
EOSINOPHILS	3	%	1 - 6



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Test Name	Value	Unit	Biological Reference interval
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
MONOCYTES	7	%	2 - 12
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
BASOPHILS	0	%	0 - 1
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE LEUKOCYTES (WBC) COUNT			
ABSOLUTE NEUTROPHIL COUNT	4483	/cmm	2000 - 7500
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE LYMPHOCYTE COUNT	2474 <sup>L</sup>	/cmm	800 - 4900
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE EOSINOPHIL COUNT	232	/cmm	40 - 440
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	A. PAR /		
ABSOLUTE MONOCYTE COUNT	541	/cmm	80 - 880
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			0. 110
ABSOLUTE BASOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110
PLATELETS AND OTHER PLATELET PREDICTIVE MARKER	oc .		
	<u>13.</u>		
PLATELET COUNT (PLT)	124000 <sup>L</sup>	/cmm	150000 - 450000
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELETCRIT (PCT)	0.15	%	0.10 - 0.36
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.15	70	0.10 - 0.36
MEAN PLATELET VOLUME (MPV)	12 <sup>H</sup>	fL	6.50 - 12.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	12	12	0.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC)	54000	/cmm	30000 - 90000
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.1000	7 311111	7000
PLATELET LARGE CELL RATIO (P-LCR)	44	%	11.0 - 45.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELET DISTRIBUTION WIDTH (PDW)	16.3	%	15.0 - 17.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD			



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DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST





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**Test Name** Value Unit **Biological Reference interval** 

#### PROTHROMBIN TIME STUDIES (PT/INR)

	The fillend fill and a second of the second			
PT TEST (PATIENT)	11.9	SECS	11.5 - 14.5	
by PHOTO OPTICAL CLOT DETECTION				
PT (CONTROL)	12	SECS		
by PHOTO OPTICAL CLOT DETECTION				
ISI	1.1			
by PHOTO OPTICAL CLOT DETECTION				
INTERNATIONAL NORMALISED RATIO (INR)	0.99		0.80 - 1.20	
by PHOTO OPTICAL CLOT DETECTION				
PT INDEX	100.84	%		
by PHOTO OPTICAL CLOT DETECTION				

#### **INTERPRETATION:-**

- 1.INR is the parameter of choice in monitoring adequacy of oral anti-coagulant therapy. Appropriate therapeutic range varies with the disease and treatment intensity.
- 2. Prolonged INR suggests potential bleeding disorder /bleeding complications
- 3. Results should be clinically correlated.
- 4. Test conducted on Citrated Plasma

RECOMMENDED THERAPEUTIC RANGE FOR ORAL ANTI-COAGULANT THERAPY (INR)			
INDICATION		INTERNATIONAL NORMALIZED RATIO (INR)	
Treatment of venous thrombosis			
Treatment of pulmonary embolism			
Prevention of systemic embolism in tissue heart valves			
Valvular heart disease	Low Intensity	2.0 - 3.0	
Acute myocardial infarction			
Atrial fibrillation			
Bileaflet mechanical valve in aortic position			
Recurrent embolism			
Mechanical heart valve	High Intensity	2.5 - 3.5	
Antiphospholipid antibodies <sup>+</sup>			

**COMMENTS:** 



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The prothrombin time (PT) and its derived measures of prothrombin ratio (PR) and international normalized ratio (INR) are measures of the efficacy of the extrinsic pathway of coagulation. PT test reflects the adequacy of factors I (fibrinogen), II (prothrombin), V, VII, and X. It is used in conjunction with the activated partial thromboplastin time (aPTT) which measures the intrinsic pathway.

The common causes of prolonged prothrombin time are: 1. Oral Anticoagulant therapy.

2.Liver disease.

3. Vit K. deficiency.

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4. Disseminated intra vascular coagulation.

5. Factor 5, 7, 10 or Prothrombin dificiency



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### **ACTIVATED PARTIAL THROMBOPLASTIN TIME (APTT)**

**APTT (PATIENT VALUE)** 30.1 **SECS** 28.6 - 38.2

by PHOTO OPTICAL CLOT DETECTION

#### **INTERPRETATION:-**

The activated partial thromboplastin time (aPTT or APTT) is a performance indicator measuring the efficacy of both the intrinsic (now referred to as the contact activation pathway) and the common coagulation pathways. Apart from detecting abnormalities in blood clotting, it is also used to monitor the treatment effects with heparin, a major anticoagulant. It is used in conjunction with the prothrombin time (PT) which measures the extrinsic pathway.

#### **COMMON CAUSES OF PROLONGED APTT:-**

- 1. Disseminated intravascular coagulation.
- 2. Liver disease.
- 3. Massive transfusion with stored blood.
- 4. Heparin administration or contamination.
- 5. A circulating Anticogulant.
- 6. Deficiency of a coagulation Factor other than factor 7.



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### **CLINICAL CHEMISTRY/BIOCHEMISTRY**

### **KIDNEY FUNCTION TEST (BASIC)**

UREA: SERUM	31.26	mg/dL	10.00 - 50.00
by UREASE - GLUTAMATE DEHYDROGENASE	(GLDH)		
CREATININE: SERUM	1.11	mg/dL	0.40 - 1.40
by ENZYMATIC, SPECTROPHOTOMETERY			
BLOOD UREA NITROGEN (BUN): SERUM	14.61	mg/dL	7.0 - 25.0
by CALCULATED, SPECTROPHOTOMETERY			
BLOOD UREA NITROGEN (BUN)/CREATIN	INE 13.16	RATIO	10.0 - 20.0
RATIO: SERUM			
by CALCULATED, SPECTROPHOTOMETERY			
UREA/CREATININE RATIO: SERUM	28.16	RATIO	
by CALCULATED, SPECTROPHOTOMETERY			
URIC ACID: SERUM	3.72	mg/dL	3.60 - 7.70
by URICASE - OXIDASE PEROXIDASE			



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**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.

Ž.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 (pia (PLIN rices diegrapartic particular partic

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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#### **ELECTROLYTES COMPLETE PROFILE**

SODIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)	138.1	mmol/L	135.0 - 150.0
POTASSIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)	4.8	mmol/L	3.50 - 5.00
CHLORIDE: SERUM by ISE (ION SELECTIVE ELECTRODE)	103.57	mmol/L	90.0 - 110.0

#### **INTERPRETATION:-**

#### SODIUM:-

Sodium is the major cation of extra-cellular fluid. Its primary function in the body is to chemically maintain osmotic pressure & acid base balance & to transmit nerve impulse.

#### HYPONATREMIA (LOW SODIUM LEVEL) CAUSES:-

- 1. Low sodium intake.
- 2. Sodium loss due to diarrhea & vomiting with adequate water and iadequate salt replacement.
- 3. Diuretics abuses.
- 4. Salt loosing nephropathy.
- 5. Metabolic acidosis.
- 6. Adrenocortical issuficiency.
- 7. Hepatic failure.

#### HYPERNATREMIA (INCREASED SODIUM LEVEL) CAUSES:-

- 1. Hyperapnea (Prolonged)
- 2. Diabetes insipidus
- 3. Diabetic acidosis
- 4. Cushings syndrome
- 5.Dehydration

#### POTASSIUM:-

Potassium is the major cation in the intracellular fluid. 90% of potassium is concentrated within the cells. When cells are damaged, potassium is released in the blood.

#### HYPOKALEMIA (LOW POTASSIUM LEVELS):-

- 1. Diarrhoea, vomiting & malabsorption.
- 2. Severe Burns.
- 3. Increased Secretions of Aldosterone

#### HYPERKALEMIA (INCREASED POTASSIUM LEVELS):-

- 1.Oliguria
- 2. Renal failure or Shock
- 3. Respiratory acidosis



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4. Hemolysis of blood

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End Of Report



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