

(A Unit of KOS Healthcare)



Dr. Vinay Chopra
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Chairman & Consultant Pathologist

Dr. Yugam Chopra MD (Pathology) CEO & Consultant Pathologist

NAME : Mrs. ANITA RANI

**AGE/ GENDER** : 58 YRS/FEMALE **PATIENT ID** : 1565100

COLLECTED BY : REG. NO./LAB NO. : 012407300025

 REFERRED BY
 : 30/Jul/2024 10:25 AM

 BARCODE NO.
 : 01514120
 COLLECTION DATE
 : 30/Jul/2024 10:32AM

 CLIENT CODE.
 : KOS DIAGNOSTIC LAB
 REPORTING DATE
 : 30/Jul/2024 10:36AM

**CLIENT ADDRESS**: 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name Value Unit Biological Reference interval

## HAEMATOLOGY COMPLETE BLOOD COUNT (CBC)

#### RED BLOOD CELLS (RBCS) COUNT AND INDICES

HAEMOGLOBIN (HB) by CALORIMETRIC	8.9 <sup>L</sup>	gm/dL	12.0 - 16.0
RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	3.92	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	29.4 <sup>L</sup>	%	37.0 - 50.0
MEAN CORPUSCULAR VOLUME (MCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	74.9 <sup>L</sup>	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	22.7 <sup>L</sup>	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	30.3 <sup>L</sup>	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	20.1 <sup>H</sup>	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	54.9	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	19.11	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	38.4	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	9700	/cmm	4000 - 11000
NUCLEATED RED BLOOD CELLS (nRBCS) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER & MICROSCOPY	NIL		0.00 - 20.00
NUCLEATED RED BLOOD CELLS (nRBCS) %	NIL	%	< 10 %

# by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER & MICROSCOPY DIFFERENTIAL LEUCOCYTE COUNT (DLC)



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Test Name	Value	Unit	Biological Reference interval
NEUTROPHILS by Flow cytometry by SF cube & microscopy	79 <sup>H</sup>	%	50 - 70
LYMPHOCYTES by Flow cytometry by SF cube & Microscopy by Flow cytometry by SF cube & Microscopy	16 <sup>L</sup>	%	20 - 40
EOSINOPHILS  by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1 <sup>L</sup>	%	1 - 6
MONOCYTES  by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	4	%	2 - 12
BASOPHILS  by Flow cytometry by SF cube & microscopy  ABSOLUTE LEUKOCYTES (WBC) COUNT	0	%	0 - 1
ABSOLUTE NEUTROPHIL COUNT  by Flow cytometry by SF cube & Microscopy	7663 <sup>H</sup>	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT  by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1552	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT  by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	97	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT  by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	388	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT  by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110
PLATELETS AND OTHER PLATELET PREDICTIVE MARKE	<u>RS.</u>		
PLATELET COUNT (PLT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	390000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by hydro dynamic focusing, electrical impedence	0.4 <sup>H</sup>	%	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	107000 <sup>H</sup>	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	27.3	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by hydro dynamic focusing, electrical impedence NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD	15.8	%	15.0 - 17.0



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Value Unit **Biological Reference interval** Test Name

#### ERYTHROCYTE SEDIMENTATION RATE (ESR)

**ERYTHROCYTE SEDIMENTATION RATE (ESR)** 

mm/1st hr 82<sup>H</sup>

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 services and quiping may decrease it. aspirin, cortisone, and quinine may decrease it



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#### PERIPHERAL BLOOD SMEAR

#### **TEST NAME:**

#### PERIPHERAL BLOOD FILM/SMEAR (PBF)

#### RED BLOOD CELLS (RBC'S):

Anisocytosis with microcytosis & occ. macrocytes.RBCs reveal mild to moderate hypochromia.No polychromatic cells or normobkasts noted.

#### WHITE BLOOD CELLS (WBC'S):

No immature leucocytes seen.

#### PI ATFI FTS

Platelets are adequate.

#### **HEMOPARASITES:**

NOT SEEN.

#### IMPRESSION:

Microcytic hypochromic anemia.



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Test Name Value Unit **Biological Reference interval** 

#### **CLINICAL CHEMISTRY/BIOCHEMISTRY GLUCOSE RANDOM (R)**

125.58 GLUCOSE RANDOM (R): PLASMA mg/dL NORMAL: < 140.00

by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD) PREDIABETIC: 140.0 - 200.0 DIABETIC: > OR = 200.0

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-prinadial 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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LIVER FUNCTION	IESI (	(COMPLETI	E)
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BILIRUBIN TOTAL: SERUM by DIAZOTIZATION, SPECTROPHOTOMETRY	0.24	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.11	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.13	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	35.11	U/L	7.00 - 45.00
SGPT/ALT: SERUM  by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	43.32	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM  by CALCULATED, SPECTROPHOTOMETRY	0.81	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL	98.29	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	24.99	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM  by BIURET, SPECTROPHOTOMETRY	7.99	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by Bromocresol green	3.45 <sup>L</sup>	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	4.54 <sup>H</sup>	gm/dL	2.30 - 3.50
A: GRATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	0.76 <sup>L</sup>	RATIO	1.00 - 2.00

**INTERPRETATION** 

**NOTE**:- To be correlated in individuals having SGOT and SGPT values higher than Normal Reference Range. **USE**:- Differential diagnosis of diseases of hepatobiliary system and pancreas.

#### **INCREASED:**

DRUG HEPATOTOXICITY	> 2
ALCOHOLIC HEPATITIS	> 2 (Highly Suggestive)
CIRRHOSIS	1.4 - 2.0
INTRAHEPATIC CHOLESTATIS	> 1.5
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)



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#### **DECREASED:**

1. Acute Hepatitis due to virus, drugs, toxins (with AST increased 3 to 10 times upper limit of normal)

2. Extra Hepatic cholestatis: 0.8 (normal or slightly decreased).

#### PROGNOSTIC SIGNIFICANCE:

NORMAL	< 0.65
GOOD PROGNOSTIC SIGN	0.3 - 0.6
POOR PROGNOSTIC SIGN	1.2 - 1.6



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**UREA** 

UREA: SERUM 28.99 mg/dL 10.00 - 50.00

by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)



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by ENZYMATIC, SPECTROPHOTOMETRY

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**CREATININE** 

CREATININE: SERUM 1.18 mg/dL 0.40 - 1.20



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#### **ELECTROLYTES COMPLETE PROFILE**

SODIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)	140.5	mmol/L	135.0 - 150.0
POTASSIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)	4.59	mmol/L	3.50 - 5.00
CHLORIDE: SERUM  by ISE (ION SELECTIVE ELECTRODE)	105.38	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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**Test Name** Value Unit **Biological Reference interval** 

4.Hemolysis of blood



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Test Name	Value	Unit	Biological Reference interval
	IRON PRO	FILE	
IRON: SERUM by FERROZINE, SPECTROPHOTOMETRY	36.6 <sup>L</sup>	μg/dL	37.0 - 145.0
UNSATURATED IRON BINDING CAPACITY (UIBC)	467.1 <sup>H</sup>	μg/dL	150.0 - 336.0

UNSATURATED IRON BINDING CAPACITY (UIBC)	467.1⊓	μg/aL	150.0 - 336.0
:SERUM			
by FERROZINE, SPECTROPHOTOMETERY			
TOTAL IRON BINDING CAPACITY (TIBC)	503.7 <sup>H</sup>	μg/dL	230 - 430
:SERUM	503.7	F-87	
10=110111			
by SPECTROPHOTOMETERY			
%TRANSFERRIN SATURATION: SERUM	7.27 <sup>L</sup>	%	15.0 - 50.0

by CALCULATED, SPECTROPHOTOMETERY (FERENE) TRANSFERRIN: SERUM 357.63<sup>H</sup> mg/dL 200.0 - 350.0

by SPECTROPHOTOMETERY (FERENE)

**INTERPRETATION:-**

VARIABLES	ANEMIA OF CHRONIC DISEASE	IRON DEFICIENCY ANEMIA	THALASSEMIA α/β TRAIT
SERUM IRON:	Normal to Reduced	Reduced	Normal
TOTAL IRON BINDING CAPACITY:	Decreased	Increased	Normal
% TRANSFERRIN SATURATION:	Decreased	Decreased < 12-15 %	Normal
SERUM FERRITIN:	Normal to Increased	Decreased	Normal or Increased

1. Serum iron studies is recommended for differential diagnosis of microcytic hypochromic anemia.i.e iron deficiency anemia, zinc deficiency anemia, anemia of chronic disease and thalassemia syndromes.

2. It is essential to isolate in deficiency anemia from Beta thalassemia syndromes because during iron replacement which is therapeutic for

iron deficiency anemia, is severely contra-indicated in Thalassemia.

TOTAL IRON BÍNDING CAPACITY (TÍBC):

1.It is a direct measure of protein transferrin which transports iron from the gut to storage sites in the bone marrow. **% TRANSFERRIN SATURATION:** 

1.Occurs in idiopathic hemochromatosis and transfusional hemosiderosis where no unsaturated iron binding capacity is available for iron mobilization. Similar condition is seen in congenital deficiency of transferrin.

\*\*\* End Of Report \*\*\*



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