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Dr. Yugam Chopra  
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CEO & Consultant Pathologist

NAME : Mr. HARWINDER SINGH  
AGE/ GENDER : 54 YRS/MALE  
COLLECTED BY :  
REFERRED BY :  
BARCODE NO. : 01518483  
CLIENT CODE. : KOS DIAGNOSTIC LAB  
CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT

PATIENT ID : 1636555  
REG. NO./LAB NO. : 012410070047  
REGISTRATION DATE : 07/Oct/2024 11:24 AM  
COLLECTION DATE : 07/Oct/2024 11:30AM  
REPORTING DATE : 07/Oct/2024 11:43AM

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) by CALORIMETRIC	12.8	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	4.52	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	39 <sup>L</sup>	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	86.3	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	27.6	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	32 <sup>L</sup>	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	17.8 <sup>H</sup>	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	56.1 <sup>H</sup>	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	19.09	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	33.12	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	5550	/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 %

#### DIFFERENTIAL LEUCOCYTE COUNT (DLC)

NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	29 <sup>L</sup>	%	50 - 70
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LYMPHOCYTES <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	57 <sup>H</sup>	%	20 - 40
EOSINOPHILS <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	7 <sup>H</sup>	%	1 - 6
MONOCYTES <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	7	%	2 - 12
BASOPHILS <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	0	%	0 - 1
<b><u>ABSOLUTE LEUKOCYTES (WBC) COUNT</u></b>			
ABSOLUTE NEUTROPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	1610 <sup>L</sup>	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	3164	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	388	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	388	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	0	/cmm	0 - 110
<b><u>PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.</u></b>			
PLATELET COUNT (PLT) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	285000	/cmm	150000 - 450000
PLATELET CRIT (PCT) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	0.23	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	9	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	46000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	17.2	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	16.1	%	15.0 - 17.0
NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD			

RECHECKED



  
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### CLINICAL CHEMISTRY/BIOCHEMISTRY

#### LIVER FUNCTION TEST (COMPLETE)

BILIRUBIN TOTAL: SERUM <i>by DIAZOTIZATION, SPECTROPHOTOMETRY</i>	0.65	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM <i>by DIAZO MODIFIED, SPECTROPHOTOMETRY</i>	0.28	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.37	mg/dL	0.10 - 1.00
SGOT/AST: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	37.19	U/L	7.00 - 45.00
SGPT/ALT: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	75.7 <sup>H</sup>	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.49	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM <i>by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL</i>	73	U/L	40.0 - 150.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM <i>by SZASZ, SPECTROPHOTOMETRY</i>	90 <sup>H</sup>	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM <i>by BIURET, SPECTROPHOTOMETRY</i>	7.89	gm/dL	6.20 - 8.00
ALBUMIN: SERUM <i>by BROMOCRESOL GREEN</i>	4.93	gm/dL	3.50 - 5.50
GLOBULIN: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	2.96	gm/dL	2.30 - 3.50
A : G RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.67	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



  
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INTRAHEPATIC CHOLESTATIS	> 1.5		
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)		

**DECREASED:**

1. Acute Hepatitis due to virus, drugs, toxins (with AST increased 3 to 10 times upper limit of normal)
2. Extra Hepatic cholestasis: 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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#### ELECTROLYTES COMPLETE PROFILE

SODIUM: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	144.1	mmol/L	135.0 - 150.0
POTASSIUM: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	4.15	mmol/L	3.50 - 5.00
CHLORIDE: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	108.07	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 inadequate salt replacement.
3. Diuretics abuses.
4. Salt loosing nephropathy.
5. Metabolic acidosis.
6. Adrenocortical insufficiency .
7. Hepatic failure.

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

1. Hyperapnea (Prolonged)
2. Diabetes insipidus
3. Diabetic acidosis
4. Cushing's 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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## VITAMINS

### VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)	50.609	ng/mL	DEFICIENCY: < 20.0 INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0
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#### 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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**VITAMIN B12/COBALAMIN**

**VITAMIN B12/COBALAMIN: SERUM**
> 2000<sup>H</sup>
pg/mL
190.0 - 890.0

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

**INTERPRETATION:-**

INCREASED VITAMIN B12	DECREASED VITAMIN B12
1.Ingestion of Vitamin C	1.Pregnancy
2.Ingestion of Estrogen	2.DRUGS:Aspirin, Anti-convulsants, Colchicine
3.Ingestion of Vitamin A	3.Ethanol lgestion
4.Hepatocellular injury	4. Contraceptive Harmones
5.Myeloproliferative disorder	5.Haemodialysis
6.Uremia	6. Multiple Myeloma

1.Vitamin B12 (cobalamin) is necessary for hematopoiesis and normal neuronal function.  
 2.In humans, it is obtained only from animal proteins and requires intrinsic factor (IF) for absorption.  
 3.The body uses its vitamin B12 stores very economically, reabsorbing vitamin B12 from the ileum and returning it to the liver; very little is excreted.  
 4.Vitamin B12 deficiency may be due to lack of IF secretion by gastric mucosa (eg, gastrectomy, gastric atrophy) or intestinal malabsorption (eg, ileal resection, small intestinal diseases).  
 5.Vitamin B12 deficiency frequently causes macrocytic anemia, glossitis, peripheral neuropathy, weakness, hyperreflexia, ataxia, loss of proprioception, poor coordination, and affective behavioral changes. These manifestations may occur in any combination; many patients have the neurologic defects without macrocytic anemia.  
 6.Serum methylmalonic acid and homocysteine levels are also elevated in vitamin B12 deficiency states.  
 7.Follow-up testing for antibodies to intrinsic factor (IF) is recommended to identify this potential cause of vitamin B12 malabsorption.  
**NOTE:**A normal serum concentration of vitamin B12 does not rule out tissue deficiency of vitamin B12. The most sensitive test for vitamin B12 deficiency at the cellular level is the assay for MMA. If clinical symptoms suggest deficiency, measurement of MMA and homocysteine should be considered, even if serum vitamin B12 concentrations are normal.

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



  
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