



# P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

**A PIONEER DIAGNOSTIC CENTRE**

☎ 0171-2532620, 8222896961 ✉ [pkrajainhealthcare@gmail.com](mailto:pkrajainhealthcare@gmail.com)

<b>NAME</b>	: Mr. LAKHVINDER SINGH	<b>PATIENT ID</b>	: 1559043
<b>AGE/ GENDER</b>	: 27 YRS/MALE	<b>REG. NO./LAB NO.</b>	: 122407240024
<b>COLLECTED BY</b>	:	<b>REGISTRATION DATE</b>	: 24/Jul/2024 11:55 AM
<b>REFERRED BY</b>	:	<b>COLLECTION DATE</b>	: 24/Jul/2024 12:13PM
<b>BARCODE NO.</b>	: 12503789	<b>REPORTING DATE</b>	: 24/Jul/2024 02:02PM
<b>CLIENT CODE.</b>	: P.K.R JAIN HEALTHCARE INSTITUTE		
<b>CLIENT ADDRESS</b>	: 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) by CALORIMETRIC	13.8	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	4.48	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	40.4	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	90.1	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	34.2	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	12.6	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	42.7	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	20.11	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	25.34	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	6880	/cmm	4000 - 11000
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### DIFFERENTIAL LEUCOCYTE COUNT (DLC)

NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	66	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	28	%	20 - 40



DR.VINAY CHOPRA  
CONSULTANT PATHOLOGIST  
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

DR.YUGAM CHOPRA  
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<b>EOSINOPHILS</b> by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0 <sup>L</sup>	%	1 - 6
<b>MONOCYTES</b> by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	6	%	2 - 12
<b>BASOPHILS</b> by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	%	0 - 1
<b><u>ABSOLUTE LEUKOCYTES (WBC) COUNT</u></b>			
<b>ABSOLUTE NEUTROPHIL COUNT</b> by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	4541	/cmm	2000 - 7500
<b>ABSOLUTE LYMPHOCYTE COUNT</b> by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1926	/cmm	800 - 4900
<b>ABSOLUTE EOSINOPHIL COUNT</b> by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0 <sup>L</sup>	/cmm	40 - 440
<b>ABSOLUTE MONOCYTE COUNT</b> by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	413	/cmm	80 - 880
<b>ABSOLUTE BASOPHIL COUNT</b> by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110
<b><u>PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.</u></b>			
<b>PLATELET COUNT (PLT)</b> by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	294000	/cmm	150000 - 450000
<b>PLATELET CRIT (PCT)</b> by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.26	%	0.10 - 0.36
<b>MEAN PLATELET VOLUME (MPV)</b> by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	9	fL	6.50 - 12.0
<b>PLATELET LARGE CELL COUNT (P-LCC)</b> by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	60000	/cmm	30000 - 90000
<b>PLATELET LARGE CELL RATIO (P-LCR)</b> by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	20.4	%	11.0 - 45.0
<b>PLATELET DISTRIBUTION WIDTH (PDW)</b> by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	16	%	15.0 - 17.0
NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD			



  
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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

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## ERYTHROCYTE SEDIMENTATION RATE (ESR)

ERYTHROCYTE SEDIMENTATION RATE (ESR)	6	mm/1st hr	0 - 20
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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 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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## CLINICAL CHEMISTRY/BIOCHEMISTRY

### LIVER FUNCTION TEST (COMPLETE)

BILIRUBIN TOTAL: SERUM <i>by DIAZOTIZATION, SPECTROPHOTOMETRY</i>	0.47	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM <i>by DIAZO MODIFIED, SPECTROPHOTOMETRY</i>	0.18	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.29	mg/dL	0.10 - 1.00
SGOT/AST: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	18.47	U/L	7.00 - 45.00
SGPT/ALT: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	14.93	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.24	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM <i>by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL</i>	90.36	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM <i>by SZASZ, SPECTROPHOTOMETRY</i>	17.53	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM <i>by BIURET, SPECTROPHOTOMETRY</i>	7.22	gm/dL	6.20 - 8.00
ALBUMIN: SERUM <i>by BROMOCRESOL GREEN</i>	4.58	gm/dL	3.50 - 5.50
GLOBULIN: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	2.64	gm/dL	2.30 - 3.50
A : G RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.73	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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Test Name	Value	Unit	Biological Reference interval
INTRAHEPATIC CHOLESTATIS	> 1.5		
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)		

**DECREASED:**


- Acute Hepatitis due to virus, drugs, toxins (with AST increased 3 to 10 times upper limit of normal)
- 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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### KIDNEY FUNCTION TEST (BASIC)

UREA: SERUM by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)	14.51	mg/dL	10.00 - 50.00
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETRY	0.71	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY	6.78 <sup>L</sup>	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	9.55 <sup>L</sup>	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	20.44	RATIO	
URIC ACID: SERUM by URICASE - OXIDASE PEROXIDASE	4.14	mg/dL	3.60 - 7.70



  
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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.
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 inappropriate antidiuretic hormone) 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.

#### **INAPPROPRIATE 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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## IMMUNOPATHOLOGY/SEROLOGY

### ANTI HUMAN IMMUNODEFICIENCY VIRUS (HIV) ANTIBODIES HIV (1 & 2) SCREENING

HIV 1/2 AND P24 ANTIGEN RESULT NON - REACTIVE

by IMMUNOCHROMATOGRAPHY

#### INTERPRETATION:-

- 1.AIDS is caused by at least 2 known types of HIV viruses, HIV-1 and HIV HIV-2.
- 2.This NACO approved immuno-chromatographic solid phase ELISA assay detects antibodies against both HIV-1 and HIV-2 viruses.
- 3.The test is used for routine serologic screening of patients at risk for HIV-1 or HIV-2 infection.
- 4.All screening ELISA assays for HIV antibody detection have high sensitivity but have low specificity.
- 5.At this laboratory, all positive samples are cross checked for positivity with two alternate assays prior to reporting.

#### NOTE:-

- 1.Confirmatory testing by Western blot is recommended for patients who are reactive for HIV by this assay.
- 2.Antibodies against HIV-1 and HIV-2 are usually not detectable until 6 to 12 weeks following exposure (window period) and are almost always detectable by 12 months.
- 3.The test is not recommended for children born to HIV infected mothers till the child turns two years old (as HIV antibodies may be transmitted passively to the child trans-placentally).

#### FALSE NEGATIVE RESULT SEEN IN:

- 1.Window period
- 2.Severe immuno-suppression including advanced AIDS.



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## VITAMINS

### VITAMIN B12/COBALAMIN

**VITAMIN B12/COBALAMIN: SERUM** 147.3<sup>L</sup> pg/mL 200.0 - 1100.0

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 Igestion
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 \*\*\*



  
DR.VINAY CHOPRA  
CONSULTANT PATHOLOGIST  
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

  
DR.YUGAM CHOPRA  
CONSULTANT PATHOLOGIST  
MBBS, MD (PATHOLOGY)

