



# 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)

**NAME** : Mr. PARAS DHIMAN

**AGE/ GENDER** : 41 YRS/MALE

**COLLECTED BY** :

**REFERRED BY** :

**BARCODE NO.** : 12504529

**CLIENT CODE.** : P.K.R JAIN HEALTHCARE INSTITUTE

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

**PATIENT ID** : 1603965

**REG. NO./LAB NO.** : 122409060012

**REGISTRATION DATE** : 06/Sep/2024 12:17 PM

**COLLECTION DATE** : 06/Sep/2024 12:22PM

**REPORTING DATE** : 06/Sep/2024 01:28PM

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	16.5	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	4.56	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	46	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	100.9 <sup>H</sup>	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	36.1 <sup>H</sup>	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	35.8	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	14.4	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	54.5	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	22.13	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	31.79	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	11350 <sup>H</sup>	/cmm	4000 - 11000
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#### DIFFERENTIAL LEUCOCYTE COUNT (DLC)

NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	85 <sup>H</sup>	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	10 <sup>L</sup>	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0 <sup>L</sup>	%	1 - 6



  
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CONSULTANT PATHOLOGIST  
MBBS, MD (PATHOLOGY & MICROBIOLOGY)

  
DR.YUGAM CHOPRA  
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MBBS, MD (PATHOLOGY)





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MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	5	%	2 - 12
BASOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	%	0 - 1
<b>ABSOLUTE LEUKOCYTES (WBC) COUNT</b>			
ABSOLUTE NEUTROPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	9648 <sup>H</sup>	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1135	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0 <sup>L</sup>	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	568	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110
<b>PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.</b>			
PLATELET COUNT (PLT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	142000 <sup>L</sup>	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.13	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	9	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	31000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	21.7	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	16.5	%	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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## PROTHROMBIN TIME STUDIES (PT/INR)

PT TEST (PATIENT) by PHOTO OPTICAL CLOT DETECTION	13.8	SECS	11.5 - 14.5
PT (CONTROL) by PHOTO OPTICAL CLOT DETECTION	12	SECS	
ISI by PHOTO OPTICAL CLOT DETECTION	1.1		
INTERNATIONAL NORMALISED RATIO (INR) by PHOTO OPTICAL CLOT DETECTION	1.17		0.80 - 1.20
PT INDEX by PHOTO OPTICAL CLOT DETECTION	86.96	%	

### 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	Low Intensity	2.0 - 3.0
Treatment of pulmonary embolism		
Prevention of systemic embolism in tissue heart valves		
Valvular heart disease		
Acute myocardial infarction		
Atrial fibrillation		
Bileaflet mechanical valve in aortic position	High Intensity	2.5 - 3.5
Recurrent embolism		
Mechanical heart valve		
Antiphospholipid antibodies <sup>+</sup>		

COMMENTS:



*Chopra*

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*Chopra*

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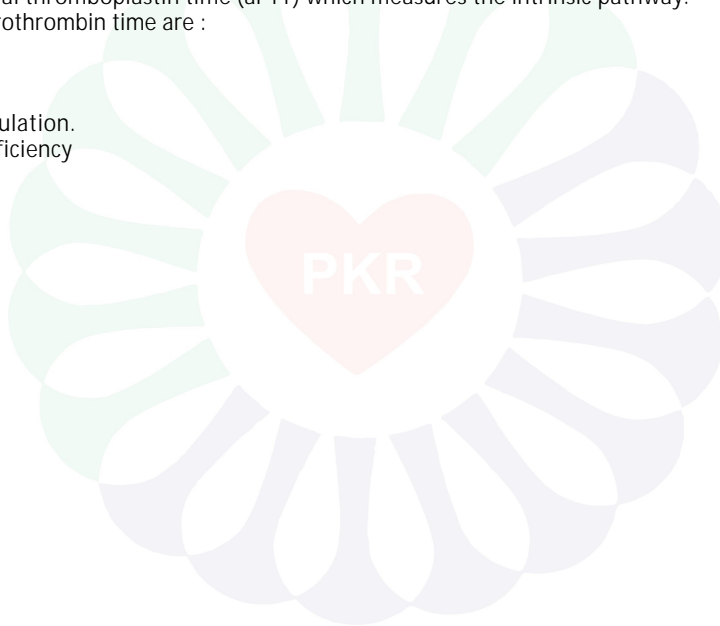
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The prothrombin time (PT) and its derived measures of prothrombin ratio (PR) and international normalized ratio (INR) are measures of the efficacy of the extrinsic pathway of coagulation. PT test reflects the adequacy of factors I (fibrinogen), II (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.
- 4.Disseminated intra vascular coagulation.
- 5.Factor 5, 7 , 10 or Prothrombin deficiency



  
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## CLINICAL CHEMISTRY/BIOCHEMISTRY

### LIVER FUNCTION TEST (COMPLETE)

<b>BILIRUBIN TOTAL: SERUM</b> <i>by DIAZOTIZATION, SPECTROPHOTOMETRY</i>	1.74 <sup>H</sup>	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
<b>BILIRUBIN DIRECT (CONJUGATED): SERUM</b> <i>by DIAZO MODIFIED, SPECTROPHOTOMETRY</i>	0.63 <sup>H</sup>	mg/dL	0.00 - 0.40
<b>BILIRUBIN INDIRECT (UNCONJUGATED): SERUM</b> <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.11 <sup>H</sup>	mg/dL	0.10 - 1.00
<b>SGOT/AST: SERUM</b> <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	65.5 <sup>H</sup>	U/L	7.00 - 45.00
<b>SGPT/ALT: SERUM</b> <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	102.27 <sup>H</sup>	U/L	0.00 - 49.00
<b>AST/ALT RATIO: SERUM</b> <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.64	RATIO	0.00 - 46.00
<b>ALKALINE PHOSPHATASE: SERUM</b> <i>by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL</i>	120.13	U/L	40.0 - 130.0
<b>GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM</b> <i>by SZASZ, SPECTROPHOTOMETRY</i>	292.62 <sup>H</sup>	U/L	0.00 - 55.0
<b>TOTAL PROTEINS: SERUM</b> <i>by BIURET, SPECTROPHOTOMETRY</i>	7.05	gm/dL	6.20 - 8.00
<b>ALBUMIN: SERUM</b> <i>by BROMOCRESOL GREEN</i>	4.31	gm/dL	3.50 - 5.50
<b>GLOBULIN: SERUM</b> <i>by CALCULATED, SPECTROPHOTOMETRY</i>	2.74	gm/dL	2.30 - 3.50
<b>A : G RATIO: SERUM</b> <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.57	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:**

- 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)	32.53	mg/dL	10.00 - 50.00
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETRY	0.66	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY	15.2	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	23.03 <sup>H</sup>	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	49.29	RATIO	
URIC ACID: SERUM by URICASE - OXIDASE PEROXIDASE	4.62	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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<b>NAME</b>	: Mr. PARAS DHIMAN	<b>PATIENT ID</b>	: 1603965
<b>AGE/ GENDER</b>	: 41 YRS/MALE	<b>REG. NO./LAB NO.</b>	: 122409060012
<b>COLLECTED BY</b>	:	<b>REGISTRATION DATE</b>	: 06/Sep/2024 12:17 PM
<b>REFERRED BY</b>	:	<b>COLLECTION DATE</b>	: 06/Sep/2024 12:22PM
<b>BARCODE NO.</b>	: 12504529	<b>REPORTING DATE</b>	: 06/Sep/2024 01:28PM
<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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## HEPATITIS B SURFACE ANTIGEN (HBsAg) SCREENING

HEPATITIS B SURFACE ANTIGEN (HBsAg) NON - REACTIVE  
RESULT

by IMMUNOCHROMATOGRAPHY

### INTERPRETATION:-

1.HBsAG is the first serological marker of HBV infection to appear in the blood (approximately 30-60 days after infection and prior to the onset of clinical disease). It is also the last viral protein to disappear from blood and usually disappears by three months after infection in self limiting acute Hepatitis B viral infection.

2.Persistence of HBsAg in blood for more than six months implies chronic infection. It is the most common marker used for diagnosis of an acute Hepatitis B infection but has very limited role in assessing patients suffering from chronic hepatitis.

### FALSE NEGATIVE RESULT SEEN IN:

- 1.Window period.
- 2.Infection with HBsAg mutant strains
- 3.Hepatitis B Surface antigen (HBsAg) is the earliest indicator of HBV infection. Usually it appears in 27 - 41 days (as early as 14 days).
- 4.Appears 7 - 26 days before biochemical abnormalities. Peaks as ALT rises. Persists during the acute illness. Usually disappears 12- 20 weeks after the onset of symptoms / laboratory abnormalities in 90% of cases.
- 5.Is the most reliable serologic marker of HBV infection. Persistence > 6 months defines carrier state. May also be found in chronic infection.Hepatitis B vaccination does not cause a positive HBsAg. Titers are not of clinical value.

### NOTE:-

- 1.All reactive HBsAG Should be reconfirmed with neutralization test(HBsAg confirmatory test).
- 2.Anti - HAV IgM appears at the same time as symptoms in > 99% of cases, peaks within the first month, becomes nondetectable in 12 months (usually 6 months). Presence confirms diagnosis of recent acute infection.

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



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

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

