



P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

☎ 0171-2532620, 8222896961 ✉ pkrjainhealthcare@gmail.com

NAME : Mr. KRISHAN LAL
AGE/ GENDER : 64 YRS/MALE
COLLECTED BY :
REFERRED BY :
BARCODE NO. : 12507838
CLIENT CODE. : P.K.R JAIN HEALTHCARE INSTITUTE
CLIENT ADDRESS : NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA

PATIENT ID : 1813693
REG. NO./LAB NO. : 122504010011
REGISTRATION DATE : 01/Apr/2025 10:54 AM
COLLECTION DATE : 01/Apr/2025 11:03AM
REPORTING DATE : 01/Apr/2025 01:22PM

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	9.2 ^L	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	3.3 ^L	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	27.5 ^L	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	83.5	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	27.9	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	33.5	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	19.3 ^H	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	60 ^H	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	25.3	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	146.27	RATIO	BETA THALASSEMIA TRAIT: <= 74.1 IRON DEFICIENCY ANEMIA: >= 74.1

WHITE BLOOD CELLS (WBCS)

TOTAL LEUCOCYTE COUNT (TLC) by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	5790	/cmm	4000 - 11000
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DIFFERENTIAL LEUCOCYTE COUNT (DLC)




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CONSULTANT PATHOLOGIST
MBBS, MD (PATHOLOGY & MICROBIOLOGY)


DR.YUGAM CHOPRA
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MBBS, MD (PATHOLOGY)





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NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	74 ^H	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	20	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0 ^L	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	6	%	2 - 12
BASOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	%	0 - 1
<u>ABSOLUTE LEUKOCYTES (WBC) COUNT</u>			
ABSOLUTE NEUTROPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	4285	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1158	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0 ^L	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	347	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110
<u>PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.</u>			
PLATELET COUNT (PLT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	140000 ^L	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.14	%	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	46000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	32.6	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	15.9	%	15.0 - 17.0

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD



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CLINICAL CHEMISTRY/BIOCHEMISTRY

GLUCOSE RANDOM (R)

GLUCOSE RANDOM (R): PLASMA by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)	97.33	mg/dL	NORMAL: < 140.00 PREDIABETIC: 140.0 - 200.0 DIABETIC: > OR = 200.0
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INTERPRETATION

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-prandial 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 TEST (COMPLETE)

BILIRUBIN TOTAL: SERUM <i>by DIAZOTIZATION, SPECTROPHOTOMETRY</i>	0.43	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM <i>by DIAZO MODIFIED, SPECTROPHOTOMETRY</i>	0.24	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.19	mg/dL	0.10 - 1.00
SGOT/AST: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	22.81	U/L	7.00 - 45.00
SGPT/ALT: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	16.3	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.4	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM <i>by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL</i>	167.87 ^H	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM <i>by SZASZ, SPECTROPHOTOMETRY</i>	46.24	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM <i>by BIURET, SPECTROPHOTOMETRY</i>	6.17 ^L	gm/dL	6.20 - 8.00
ALBUMIN: SERUM <i>by BROMOCRESOL GREEN</i>	3.72	gm/dL	3.50 - 5.50
GLOBULIN: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	2.45	gm/dL	2.30 - 3.50
A : G RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.52	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




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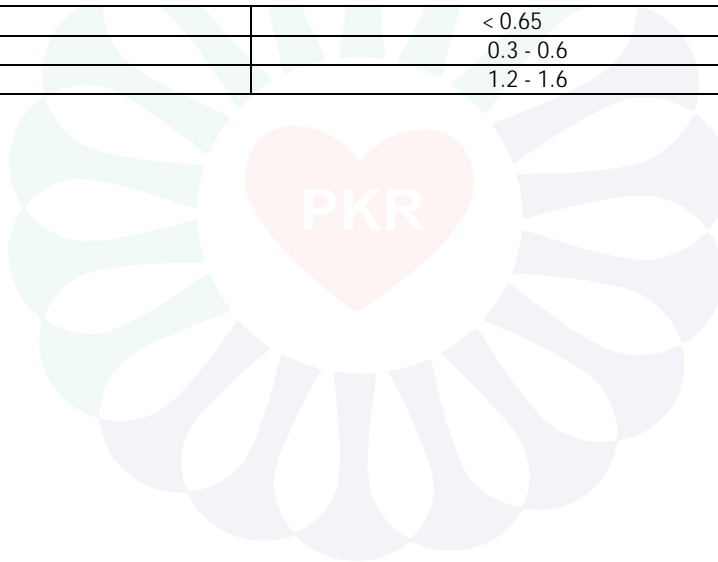
Test Name	Value	Unit	Biological Reference interval
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS		> 1.3 (Slightly Increased)	

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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KIDNEY FUNCTION TEST (BASIC)

UREA: SERUM by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)	46.11	mg/dL	10.00 - 50.00
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETRY	5.03 ^H	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY	21.55	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	4.28 ^L	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	9.17	RATIO	
URIC ACID: SERUM by URICASE - OXIDASE PEROXIDASE	4.13	mg/dL	3.60 - 7.70

NOTE 2

ADVICE

RESULT RECHECKED TWICE

KINDLY CORRELATE CLINICALLY



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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

HEPATITIS C VIRUS (HCV) ANTIBODIES SCREENING

HEPATITIS C ANTIBODY (HCV) TOTAL
RESULT NON - REACTIVE

by IMMUNOCHROMATOGRAPHY

INTERPRETATION:


1. Anti HCV total antibody assay identifies presence IgG antibodies in the serum . It is a useful screening test with a specificity of nearly 99%.
2. It becomes positive approximately 24 weeks after exposure. The test can not isolate an active ongoing HCV infection from an old infection that has been cleared. All positive results must be confirmed for active disease by an HCV PCR test .

FALSE NEGATIVE RESULTS SEEN IN:

- 1.Window period
- 2.Immunocompromised states.




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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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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)

