



P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

A PIONEER DIAGNOSTIC CENTRE

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

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

NAME	: Mr. NASIB SINGH	PATIENT ID	: 1738584
AGE/ GENDER	: 65 YRS/MALE	REG. NO./LAB NO.	: 122501290005
COLLECTED BY	:	REGISTRATION DATE	: 29/Jan/2025 09:36 AM
REFERRED BY	:	COLLECTION DATE	: 29/Jan/2025 10:49AM
BARCODE NO.	: 12506732	REPORTING DATE	: 29/Jan/2025 03:23PM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE		
CLIENT ADDRESS	: 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) <i>by CALORIMETRIC</i>	14.8	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDEANCE</i>	4.33	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	42.3	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	97.6	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	34.2^H	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	35	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	13.8	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	50.3	fL	35.0 - 56.0
MENTZERS INDEX <i>by CALCULATED</i>	22.54	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX <i>by CALCULATED</i>	31.12	RATIO	BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0


WHITE BLOOD CELLS (WBCS)


TOTAL LEUCOCYTE COUNT (TLC) <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	6710	/cmm	4000 - 11000
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DIFFERENTIAL LEUCOCYTE COUNT (DLC)

NEUTROPHILS <i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>	58	%	50 - 70
LYMPHOCYTES	34	%	20 - 40




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<i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>			
EOSINOPHILS	2	%	1 - 6
<i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>			
MONOCYTES	6	%	2 - 12
<i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>			
BASOPHILS	0	%	0 - 1
<i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>			
<u>ABSOLUTE LEUKOCYTES (WBC) COUNT</u>			
ABSOLUTE NEUTROPHIL COUNT	3892	/cmm	2000 - 7500
<i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>			
ABSOLUTE LYMPHOCYTE COUNT	2281	/cmm	800 - 4900
<i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>			
ABSOLUTE EOSINOPHIL COUNT	134	/cmm	40 - 440
<i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>			
ABSOLUTE MONOCYTE COUNT	403	/cmm	80 - 880
<i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>			
ABSOLUTE BASOPHIL COUNT	0	/cmm	0 - 110
<i>by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY</i>			
<u>PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.</u>			
PLATELET COUNT (PLT)	139000^L	/cmm	150000 - 450000
<i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>			
PLATELETCRIT (PCT)	0.18	%	0.10 - 0.36
<i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>			
MEAN PLATELET VOLUME (MPV)	13^H	fL	6.50 - 12.0
<i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>			
PLATELET LARGE CELL COUNT (P-LCC)	64000	/cmm	30000 - 90000
<i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>			
PLATELET LARGE CELL RATIO (P-LCR)	45.9^H	%	11.0 - 45.0
<i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>			
PLATELET DISTRIBUTION WIDTH (PDW)	16.8	%	15.0 - 17.0
<i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>			
NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD			




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CLINICAL CHEMISTRY/BIOCHEMISTRY

GLUCOSE RANDOM (R)

GLUCOSE RANDOM (R): PLASMA <i>by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)</i>	70.06	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>	0.98	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM <i>by DIAZO MODIFIED, SPECTROPHOTOMETRY</i>	0.35	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.63	mg/dL	0.10 - 1.00
SGOT/AST: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	27.36	U/L	7.00 - 45.00
SGPT/ALT: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	18.49	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.48	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM <i>by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL</i>	106.78	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM <i>by SZASZ, SPECTROPHOTOMETRY</i>	26.98	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM <i>by BIURET, SPECTROPHOTOMETRY</i>	6.18^L	gm/dL	6.20 - 8.00
ALBUMIN: SERUM <i>by BROMOCRESOL GREEN</i>	4.14	gm/dL	3.50 - 5.50
GLOBULIN: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	2.04^L	gm/dL	2.30 - 3.50
A : G RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	2.03^H	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 CHOLESTASIS	> 1.5
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)




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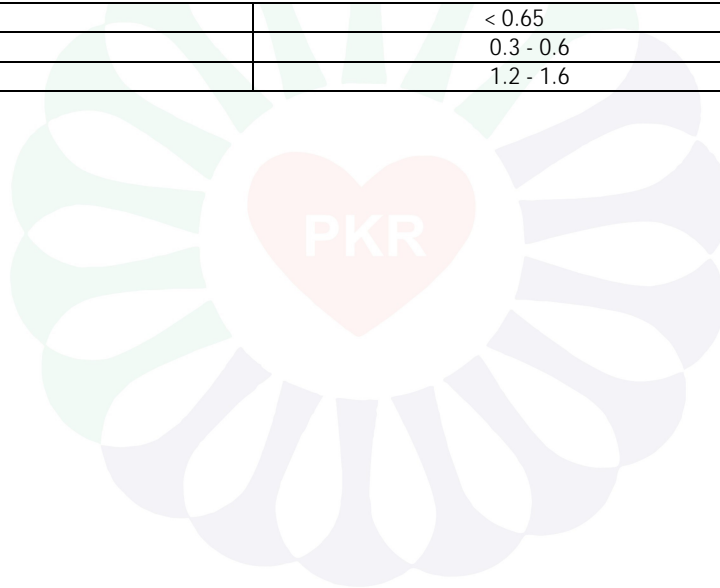
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DECREASED:

1. Acute Hepatitis due to virus, drugs, toxins (with AST increased 3 to 10 times upper limit of normal)
2. Extra Hepatic 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 <i>by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)</i>	28.67	mg/dL	10.00 - 50.00
CREATININE: SERUM <i>by ENZYMATIC, SPECTROPHOTOMETRY</i>	1.09	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	13.4	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	12.29	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	26.3	RATIO	
URIC ACID: SERUM <i>by URICASE - OXIDASE PEROXIDASE</i>	4.69	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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AMYLASE

AMYLASE - SERUM

184.7^H

IU/L

0 - 90

by CNPG 3, SPECTROPHOTOMETRY

INTERPRETATION COMMENTS

1. Amylase is produced in the Pancreas and most of the elevation in serum is due to increased rate of Amylase entry into the blood stream / decreased rate of clearance or both.
2. Serum Amylase rises within 6 to 48 hours of onset of Acute pancreatitis in 80% of patients, but is not proportional to the severity of the disease.
3. Activity usually returns to normal in 3-5 days in patients with milder edematous form of the disease.
4. Values persisting longer than this period suggest continuing necrosis of pancreas or Pseudocyst formation.
5. Approximately 20% of patients with Pancreatitis have normal or near normal activity.
6. Hyperlipemic patients with Pancreatitis also show spuriously normal Amylase levels due to suppression of Amylase activity by triglyceride.
7. Low Amylase levels are seen in Chronic Pancreatitis, Congestive Heart failure, 2nd & 3rd trimesters of pregnancy, Gastrointestinal cancer & bone fractures.



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LIPASE

LIPASE - SERUM <i>by METHYL RESORUFIN, SPECTROPHOTOMETRY</i>	52.53	U/L	0 - 60
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INTERPRETATION

1. Pancreas is the major and primary source of serum lipase though lipases are also present in liver, stomach, intestine, WBC, fat cells and milk.
2. In acute pancreatitis, serum lipase becomes elevated at the same time as amylase and remains high for 7-10 days.
3. Increased lipase activity rarely lasts longer than 14 days.
4. Prolonged increase suggests poor prognosis or presence of a cyst.
5. The combined use of serum lipase and serum amylase is effective in ruling out acute pancreatitis.

INCREASED LEVEL:

1. Acute & Chronic pancreatitis
2. Obstruction of pancreatic duct
3. Non pancreatic conditions like renal diseases, acute cholecystitis, intestinal obstruction, duodenal ulcer, alcoholism, diabetic ketoacidosis and following endoscopic retrograde cholangiopancreatography

NOTE:

1. Elevations 2 to 50 times the upper reference have been reported. The increase in serum lipase is not necessarily proportional to the severity of the attack. Normalization is not necessarily a sign of resolution.

ADVICE:

Concomitant testing of serum amylase and lipase is highly recommended to establish a diagnosis of pancreatic injury



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IMMUNOPATHOLOGY/SEROLOGY

HEPATITIS C VIRUS (HCV) ANTIBODIES SCREENING

HEPATITIS C ANTIBODY (HCV) TOTAL NON - REACTIVE

RESULT
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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A PIONEER DIAGNOSTIC CENTRE

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

TEST PERFORMED AT: KOS DIAGNOSTIC LAB, AMBALA CANTT.

NAME	: Mr. NASIB SINGH		
AGE/ GENDER	: 65 YRS/MALE	PATIENT ID	: 1738584
COLLECTED BY	:	REG. NO./LAB NO.	: 122501290005
REFERRED BY	:	REGISTRATION DATE	: 29/Jan/2025 09:36 AM
BARCODE NO.	: 12506732	COLLECTION DATE	: 29/Jan/2025 10:49AM
CLIENT CODE.	: P.K.R JAIN HEALTHCARE INSTITUTE	REPORTING DATE	: 29/Jan/2025 03:24PM
CLIENT ADDRESS	: NASIRPUR, HISSAR ROAD, AMBALA CITY - HARYANA		

Test Name	Value	Unit	Biological Reference interval
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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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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 ***




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