



# 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.

<b>NAME</b>	: Mr. KAMAL	<b>PATIENT ID</b>	: 1578263
<b>AGE/ GENDER</b>	: 24 YRS/MALE	<b>REG. NO./LAB NO.</b>	: 122408120014
<b>COLLECTED BY</b>	:	<b>REGISTRATION DATE</b>	: 12/Aug/2024 02:23 PM
<b>REFERRED BY</b>	:	<b>COLLECTION DATE</b>	: 12/Aug/2024 02:26PM
<b>BARCODE NO.</b>	: 12504121	<b>REPORTING DATE</b>	: 12/Aug/2024 05:04PM
<b>CLIENT CODE.</b>	: P.K.R JAIN HEALTHCARE INSTITUTE		
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Test Name	Value	Unit	Biological Reference interval
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## HAEMATOLOGY

### HAEMOGLOBIN (HB)

**HAEMOGLOBIN (HB)**

7.6<sup>L</sup>

gm/dL

12.0 - 17.0

by CALORIMETRIC

#### INTERPRETATION:-

Hemoglobin is the protein molecule in red blood cells that carries oxygen from the lungs to the body's tissues and returns carbon dioxide from the tissues back to the lungs.

A low hemoglobin level is referred to as ANEMIA or low red blood count.

#### ANEMIA ( DECREASED HAEMOGLOBIN):

- 1) Loss of blood (traumatic injury, surgery, bleeding, colon cancer or stomach ulcer)
- 2) Nutritional deficiency (iron, vitamin B12, folate)
- 3) Bone marrow problems (replacement of bone marrow by cancer)
- 4) Suppression by red blood cell synthesis by chemotherapy drugs
- 5) Kidney failure
- 6) Abnormal hemoglobin structure (sickle cell anemia or thalassemia).

#### POLYCYTHEMIA (INCREASED HAEMOGLOBIN):

- 1) People in higher altitudes (Physiological)
- 2) Smoking (Secondary Polycythemia)
- 3) Dehydration produces a falsely rise in hemoglobin due to increased haemoconcentration
- 4) Advanced lung disease (for example, emphysema)
- 5) Certain tumors
- 6) A disorder of the bone marrow known as polycythemia rubra vera,
- 7) Abuse of the drug erythropoetin (Epogen) by athletes for blood doping purposes (increasing the amount of oxygen available to the body by chemically raising the production of red blood cells).

**NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD**



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## CLINICAL CHEMISTRY/BIOCHEMISTRY

### POTASSIUM

**POTASSIUM: SERUM**

5.3<sup>H</sup>

mmol/L

3.50 - 5.00

by ISE (ION SELECTIVE ELECTRODE)

#### INTERPRETATION:-

##### **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
- 4.Hemolysis of blood



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### KIDNEY FUNCTION TEST (BASIC)

UREA: SERUM <i>by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)</i>	242.34 <sup>H</sup>	mg/dL	10.00 - 50.00
CREATININE: SERUM <i>by ENZYMATIC, SPECTROPHOTOMETRY</i>	14.55 <sup>H</sup>	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	113.24 <sup>H</sup>	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	7.78 <sup>L</sup>	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	16.66	RATIO	
URIC ACID: SERUM <i>by URICASE - OXIDASE PEROXIDASE</i>	8.85 <sup>H</sup>	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

#### HEPATITIS C VIRUS (HCV) ANTIBODIES SCREENING

HEPATITIS C ANTIBODY (HCV) TOTAL RESULT **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 \*\*\*



  
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