



# P K R JAIN HEALTHCARE INSTITUTE

NASIRPUR, Hissar Road, AMBALA CITY- (Haryana)

**A PIONEER DIAGNOSTIC CENTRE**

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

<b>NAME</b>	: Mr. KULDEEP SINGH	<b>PATIENT ID</b>	: 1257633
<b>AGE/ GENDER</b>	: 30 YRS/MALE	<b>REG. NO./LAB NO.</b>	: 122407010002
<b>COLLECTED BY</b>	:	<b>REGISTRATION DATE</b>	: 01/Jul/2024 09:10 AM
<b>REFERRED BY</b>	:	<b>COLLECTION DATE</b>	: 01/Jul/2024 09:46AM
<b>BARCODE NO.</b>	: 12503384	<b>REPORTING DATE</b>	: 01/Jul/2024 04:32PM
<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

### HAEMOGLOBIN (HB)

HAEMOGLOBIN (HB)	16.5	gm/dL	12.0 - 17.0
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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 erythropoietin (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

### KIDNEY FUNCTION TEST (BASIC)

UREA: SERUM <i>by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)</i>	27.74	mg/dL	10.00 - 50.00
CREATININE: SERUM <i>by ENZYMATIC, SPECTROPHOTOMETRY</i>	1.16	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	12.96	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	11.17	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	23.91	RATIO	
URIC ACID: SERUM <i>by URICASE - OXIDASE PEROXIDASE</i>	2.95 <sup>L</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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## ELECTROLYTES COMPLETE PROFILE

SODIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)	141.7	mmol/L	135.0 - 150.0
POTASSIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)	3.91	mmol/L	3.50 - 5.00
CHLORIDE: SERUM by ISE (ION SELECTIVE ELECTRODE)	106.28	mmol/L	90.0 - 110.0

### INTERPRETATION:-

#### SODIUM:-

Sodium is the major cation of extra-cellular fluid. Its primary function in the body is to chemically maintain osmotic pressure & acid base balance & to transmit nerve impulse.

#### HYPONATREMIA (LOW SODIUM LEVEL) CAUSES:-

1. Low sodium intake.
2. Sodium loss due to diarrhea & vomiting with adequate water and iadequate salt replacement.
3. Diuretics abuses.
4. Salt loosing nephropathy.
5. Metabolic acidosis.
6. Adrenocortical issuficiency .
- 7.Hepatic failure.

#### HYPERNATREMIA (INCREASED SODIUM LEVEL) CAUSES:-

- 1.Hyperapnea (Prolonged)
- 2.Diabetes insipidus
- 3.Diabetic acidosis
- 4.Cushings syndrome
- 5.Dehydration

#### 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



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4.Hemolysis of blood

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



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