

**Dr. Vinay Chopra**  
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 Chairman & Consultant Pathologist

**Dr. Yugam Chopra**  
 MD (Pathology)  
 CEO & Consultant Pathologist

<b>NAME</b>	: Mr. SUKHRAJ	<b>PATIENT ID</b>	: 1761824
<b>AGE/ GENDER</b>	: 82 YRS/MALE	<b>REG. NO./LAB NO.</b>	: 012502180053
<b>COLLECTED BY</b>	: SURJESH	<b>REGISTRATION DATE</b>	: 18/Feb/2025 05:15 PM
<b>REFERRED BY</b>	: CENTRAL PHOENIX CLUB (AMBALA CANTT)	<b>COLLECTION DATE</b>	: 18/Feb/2025 05:19PM
<b>BARCODE NO.</b>	: 01525735	<b>REPORTING DATE</b>	: 18/Feb/2025 05:56PM
<b>CLIENT CODE.</b>	: KOS DIAGNOSTIC LAB		
<b>CLIENT ADDRESS</b>	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name	Value	Unit	Biological Reference interval
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**SWASTHYA WELLNESS PANEL: G**  
**COMPLETE BLOOD COUNT (CBC)**


**RED BLOOD CELLS (RBCS) COUNT AND INDICES**


HAEMOGLOBIN (HB) <i>by CALORIMETRIC</i>	13.3	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDEANCE</i>	4.38	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	<b>40.8<sup>L</sup></b>	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	93.1	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	30.4	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	32.6	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	13.7	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	47.9	fL	35.0 - 56.0
MENTZERS INDEX <i>by CALCULATED</i>	21.26	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX <i>by CALCULATED</i>	29.15	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 &amp; MICROSCOPY</i>	<b>14470<sup>H</sup></b>	/cmm	4000 - 11000
NUCLEATED RED BLOOD CELLS (nRBCS) <i>by AUTOMATED 6 PART HEMATOLOGY ANALYZER</i>	NIL		0.00 - 20.00
NUCLEATED RED BLOOD CELLS (nRBCS) % <i>by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER</i>	NIL	%	< 10 %



  
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<b><u>DIFFERENTIAL LEUCOCYTE COUNT (DLC)</u></b>			
NEUTROPHILS <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	91 <sup>H</sup>	%	50 - 70
LYMPHOCYTES <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	6 <sup>L</sup>	%	20 - 40
EOSINOPHILS <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	0 <sup>L</sup>	%	1 - 6
MONOCYTES <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	3	%	2 - 12
BASOPHILS <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	0	%	0 - 1
<b><u>ABSOLUTE LEUKOCYTES (WBC) COUNT</u></b>			
ABSOLUTE NEUTROPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	13168 <sup>H</sup>	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	868 <sup>L</sup>	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	0 <sup>L</sup>	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	434	/cmm	80 - 880
<b><u>PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.</u></b>			
PLATELET COUNT (PLT) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	381000	/cmm	150000 - 450000
PLATELETCRIT (PCT) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	0.34	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	9	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	71000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	18.8	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	16	%	15.0 - 17.0


**ADVICE**

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

**KINDLY CORRELATE CLINICALLY**



  
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RECHECKED



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**GLYCOSYLATED HAEMOGLOBIN (HBA1C)**

GLYCOSYLATED HAEMOGLOBIN (HbA1c): WHOLE BLOOD <i>by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)</i>	5.7	%	4.0 - 6.4
ESTIMATED AVERAGE PLASMA GLUCOSE <i>by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)</i>	116.89	mg/dL	60.00 - 140.00

**INTERPRETATION:**

AS PER AMERICAN DIABETES ASSOCIATION (ADA):		
REFERENCE GROUP	GLYCOSYLATED HEMOGLOBIN (HBA1C) in %	
Non diabetic Adults >= 18 years	<5.7	
At Risk (Prediabetes)	5.7 – 6.4	
Diagnosing Diabetes	>= 6.5	
Therapeutic goals for glycemic control	<b>Age &gt; 19 Years</b>	
	Goals of Therapy:	< 7.0
	Actions Suggested:	>8.0
	<b>Age &lt; 19 Years</b>	
	Goal of therapy:	<7.5

**COMMENTS:**

- Glycosylated hemoglobin (HbA1c) test is three monthly monitoring done to assess compliance with therapeutic regimen in diabetic patients.
- Since Hb1c reflects long term fluctuations in blood glucose concentration, a diabetic patient who has recently under good control may still have high concentration of HbA1c. Converse is true for a diabetic previously under good control but now poorly controlled.
- Target goals of < 7.0 % may be beneficial in patients with short duration of diabetes, long life expectancy and no significant cardiovascular disease. In patients with significant complications of diabetes, limited life expectancy or extensive co-morbid conditions, targeting a goal of < 7.0% may not be appropriate. 4.High
- HbA1c (>9.0 -9.5 %) is strongly associated with risk of development and rapid progression of microvascular and nerve complications
- Any condition that shorten RBC life span like acute blood loss, hemolytic anemia falsely lower HbA1c results.
- HbA1c results from patients with HbSS, HbSC and HbD must be interpreted with caution, given the pathological processes including anemia, increased red cell turnover, and transfusion requirement that adversely impact HbA1c as a marker of long-term glycemic control.
- Specimens from patients with polycythemia or post-splenectomy may exhibit increase in HbA1c values due to a somewhat longer life span of the red cells.



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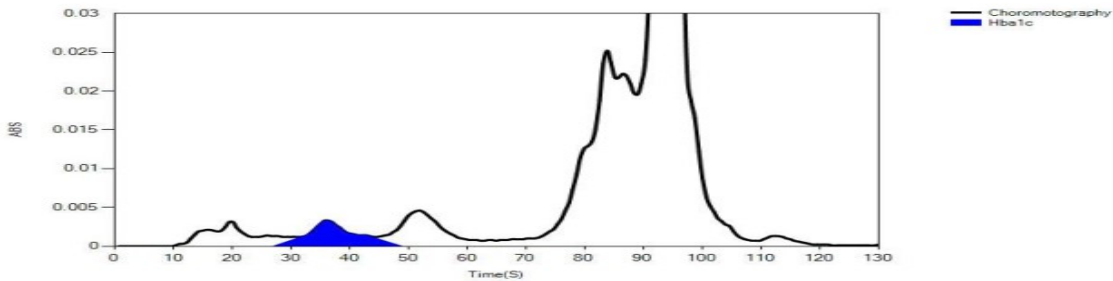
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LIFOTRONIC Graph Report

Name :	Case :	Patient Type :	Test Date : 18/02/2025 20:36:18
Age :	Department :	Sample Type : Whole Blood EDTA	Sample Id : 01525735
Gender :			Total Area : 7906

Peak Name	Retention Time(s)	Absorbance	Area	Result (Area %)
HbA0	69	1986	6957	81.7
HbA1c	38	46	485	5.7
La1c	26	34	240	2.8
HbF	19	14	22	0.2
Hba1b	14	32	120	1.4
Hba1a	11	21	82	1.0



  
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**ERYTHROCYTE SEDIMENTATION RATE (ESR)**

ERYTHROCYTE SEDIMENTATION RATE (ESR) <i>by RED CELL AGGREGATION BY CAPILLARY PHOTOMETRY</i>	<b>74<sup>H</sup></b>	mm/1st hr	0 - 20
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**INTERPRETATION:**

1. ESR is a non-specific test because an elevated result often indicates the presence of inflammation associated with infection, cancer and auto-immune disease, but does not tell the health practitioner exactly where the inflammation is in the body or what is causing it.
2. An ESR can be affected by other conditions besides inflammation. For this reason, the ESR is typically used in conjunction with other test such as C-reactive protein
3. This test may also be used to monitor disease activity and response to therapy in both of the above diseases as well as some others, such as systemic lupus erythematosus

**CONDITION WITH LOW ESR**

A low ESR can be seen with conditions that inhibit the normal sedimentation of red blood cells, such as a high red blood cell count (polycythaemia), significantly high white blood cell count (leucocytosis), and some protein abnormalities. Some changes in red cell shape (such as sickle cells in sickle cell anaemia) also lower the ESR.

**NOTE:**

1. ESR and C - reactive protein (C-RP) are both markers of inflammation.
2. Generally, ESR does not change as rapidly as does CRP, either at the start of inflammation or as it resolves.
3. **CRP is not affected by as many other factors as is ESR, making it a better marker of inflammation.**
4. If the ESR is elevated, it is typically a result of two types of proteins, globulins or fibrinogen.
5. Women tend to have a higher ESR, and menstruation and pregnancy can cause temporary elevations.
6. Drugs such as dextran, methyldopa, oral contraceptives, penicillamine procainamide, theophylline, and vitamin A can increase ESR, while aspirin, cortisone, and quinine may decrease it



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**CLINICAL CHEMISTRY/BIOCHEMISTRY**

**GLUCOSE FASTING (F)**


GLUCOSE FASTING (F): PLASMA <i>by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)</i>	<b>107.67<sup>H</sup></b>	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > OR = 126.0
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**INTERPRETATION**

**IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:**

1. A fasting plasma glucose level below 100 mg/dl is considered normal.
2. A fasting plasma glucose level between 100 - 125 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 fasting plasma glucose level of above 125 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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<b>LIPID PROFILE : BASIC</b>			
CHOLESTEROL TOTAL: SERUM <i>by CHOLESTEROL OXIDASE PAP</i>	160.8	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM <i>by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)</i>	93.32	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROL (DIRECT): SERUM <i>by SELECTIVE INHIBITION</i>	62.34	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	79.8	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.0 BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0
NON HDL CHOLESTEROL: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	98.46	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTEROL: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	18.66	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	414.92	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	2.58	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0



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LDL/HDL RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.28	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/HDL RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	<b>1.5<sup>L</sup></b>	RATIO	3.00 - 5.00

**INTERPRETATION:**

1. Measurements in the same patient can show physiological & analytical variations. Three serial samples 1 week apart are recommended for Total Cholesterol, Triglycerides, HDL & LDL Cholesterol.
2. As per NLA-2014 guidelines, all adults above the age of 20 years should be screened for lipid status. Selective screening of children above the age of 2 years with a family history of premature cardiovascular disease or those with at least one parent with high total cholesterol is recommended.
3. Low HDL levels are associated with increased risk for Atherosclerotic Cardiovascular disease (ASCVD) due to insufficient HDL being available to participate in reverse cholesterol transport, the process by which cholesterol is eliminated from peripheral tissues.
4. NLA-2014 identifies Non HDL Cholesterol (an indicator of all atherogenic lipoproteins such as LDL, VLDL, IDL, Lp(a), Chylomicron remnants) along with LDL-cholesterol as co-primary target for cholesterol lowering therapy. Note that major risk factors can modify treatment goals for LDL & Non HDL.
5. Additional testing for Apolipoprotein B, hsCRP, Lp(a) & LP-PLA2 should be considered among patients with moderate risk for ASCVD for risk refinement

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Test Name	Value	Unit	Biological Reference interval
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**LIVER FUNCTION TEST (COMPLETE)**

BILIRUBIN TOTAL: SERUM <i>by DIAZOTIZATION, SPECTROPHOTOMETRY</i>	0.65	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM <i>by DIAZO MODIFIED, SPECTROPHOTOMETRY</i>	0.17	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.48	mg/dL	0.10 - 1.00
SGOT/AST: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	32.4	U/L	7.00 - 45.00
SGPT/ALT: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	34.9	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.93	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM <i>by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL</i>	82.26	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM <i>by SZASZ, SPECTROPHOTOMETRY</i>	31.46	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM <i>by BIURET, SPECTROPHOTOMETRY</i>	7.05	gm/dL	6.20 - 8.00
ALBUMIN: SERUM <i>by BROMOCRESOL GREEN</i>	<b>3.43<sup>L</sup></b>	gm/dL	3.50 - 5.50
GLOBULIN: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	<b>3.62<sup>H</sup></b>	gm/dL	2.30 - 3.50
A : G RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	<b>0.95<sup>L</sup></b>	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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**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 (COMPLETE)**

UREA: SERUM <i>by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)</i>	31.6	mg/dL	10.00 - 50.00
CREATININE: SERUM <i>by ENZYMATIC, SPECTROPHOTOMETRY</i>	0.83	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	14.77	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	17.8	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	38.07	RATIO	
URIC ACID: SERUM <i>by URICASE - OXIDASE PEROXIDASE</i>	3.79	mg/dL	3.60 - 7.70
CALCIUM: SERUM <i>by ARSENAZO III, SPECTROPHOTOMETRY</i>	10	mg/dL	8.50 - 10.60
PHOSPHOROUS: SERUM <i>by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY</i>	2.82	mg/dL	2.30 - 4.70

**ELECTROLYTES**

SODIUM: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	139.3	mmol/L	135.0 - 150.0
POTASSIUM: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	4.11	mmol/L	3.50 - 5.00
CHLORIDE: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	104.48	mmol/L	90.0 - 110.0

**ESTIMATED GLOMERULAR FILTRATION RATE**

ESTIMATED GLOMERULAR FILTRATION RATE (eGFR): SERUM <i>by CALCULATED</i>	87.4		
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**INTERPRETATION:**

To differentiate between pre- and post renal 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 haemorrhage.



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- High protein intake.
  - Impaired renal function plus
  - Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, burns, surgery, cachexia, high fever).
  - Urine reabsorption (e.g. ureter colostomy)
  - Reduced muscle mass (subnormal creatinine production)
  - Certain drugs (e.g. tetracycline, glucocorticoids)
- INCREASED RATIO (>20:1) WITH ELEVATED CREATININE LEVELS:**
- Postrenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).
  - Prerenal azotemia superimposed on renal disease.
- DECREASED RATIO (<10:1) WITH DECREASED BUN :**
- Acute tubular necrosis.
  - Low protein diet and starvation.
  - Severe liver disease.
  - Other causes of decreased urea synthesis.
  - Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid).
  - Inherited hyperammonemias (urea is virtually absent in blood).
  - SIADH (syndrome of inappropriate antidiuretic hormone) due to tubular secretion of urea.
  - Pregnancy.
- DECREASED RATIO (<10:1) WITH INCREASED CREATININE:**
- Phenacimide therapy (accelerates conversion of creatine to creatinine).
  - Rhabdomyolysis (releases muscle creatinine).
  - Muscular patients who develop renal failure.
- INAPPROPRIATE RATIO:**
- Diabetic ketoacidosis (acetoacetate causes false increase in creatinine with certain methodologies, resulting in normal ratio when dehydration should produce an increased BUN/creatinine ratio).
  - Cephalosporin therapy (interferes with creatinine measurement).
- ESTIMATED GLOMERULAR FILTRATION RATE:**

CKD STAGE	DESCRIPTION	GFR ( mL/min/1.73m <sup>2</sup> )	ASSOCIATED FINDINGS
G1	Normal kidney function	>90	No proteinuria
G2	Kidney damage with normal or high GFR	>90	Presence of Protein , Albumin or cast in urine
G3a	Mild decrease in GFR	60 -89	
G3b	Moderate decrease in GFR	30-59	
G4	Severe decrease in GFR	15-29	
G5	Kidney failure	<15	



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**COMMENTS:**

1. Estimated Glomerular filtration rate (eGFR) is the sum of filtration rates in all functioning nephrons and so an estimation of the GFR provides a measure of functioning nephrons of the kidney.
2. eGFR calculated using the 2009 CKD-EPI creatinine equation and GFR category reported as per KDIGO guideline 2012
3. In patients, with eGFR creatinine between 45-59 ml/min/1.73 m<sup>2</sup> (G3) and without any marker of Kidney damage, It is recommended to measure eGFR with Cystatin C for confirmation of CKD
4. eGFR category G1 OR G2 does not fulfill the criteria for CKD, in the absence of evidence of Kidney Damage
5. In a suspected case of Acute Kidney Injury (AKI), measurement of eGFR should be done after 48-96 hours of any Intervention or procedure
6. eGFR calculated by Serum Creatinine may be less accurate due to certain factors like Race, Muscle Mass, Diet, Certain Drugs. In such cases, eGFR should be calculated using Serum Cystatin C
7. **A decrease in eGFR implies either progressive renal disease, or a reversible process causing decreased nephron function (eg, severe dehydration).**

**ADVICE:**  
KDIGO guideline, 2012 recommends Chronic Kidney Disease (CKD) should be classified based on cause, eGFR category and Albuminuria (ACR) category. GFR & ACR category combined together reflect risk of progression and helps Clinician to identify the individual who are progressing at more rapid rate than anticipated

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### ENDOCRINOLOGY

#### ANGIOTENSIN CONVERTING ENZYME (ACE): SERUM

ANGIOTENSIN CONVERTING ENZYME (ACE): SERUM	15.1	U/L	8.0 - 52.0
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by FURYACRYLOYLPHENYLALANYLGLYCYLGLYCINE (FAPPG)

#### INTERPRETATION

1. Angiotensin converting Enzyme (ACE) also known as kinase II, is present in many cells types such as neuronal cells, renal proximal tubular cells, and mostly in endothelial cells.
2. Angiotensin converting enzyme ( ACE) modulates peripheral vascular resistance as well as renal and cardiovascular function. It is responsible for conversion of Angiotensin I to Angiotensin II as well as inactivation of bradykinin
3. It is attached to endothelial surface membrane by an anchor peptide and can be cleaved to be released into the blood circulation as soluble enzyme. Serum ACE activity is significantly elevated in patients with untreated active disease.
4. Majority of ACE is tissue bound (> 90%) found predominantly in lungs & testes
5. It has been established as an important diagnostic parameter in Sarcoidosis. Spontaneous or induced remission of sarcoidosis has been seen, by decreasing serum ACE values.

#### FACTORS AFFECTING ACE LEVELS:

1. Smoking – ACE activity is 30% lower in smokers
2. Thyroid hormone- Stimulates ACE synthesis
3. Postmenopausal estrogen replacement – ACE activity is 20% lower

#### INCREASED LEVELS:

1. Sarcoidosis – ACE levels are used in the diagnosis and monitoring of this disease and are directly related to the number of organs affected and activity of granulomas. Mature granulomas produce less ACE than developing ones. ACE is more likely to be elevated with pulmonary involvement than with purely hilar adenopathy.
2. Pulmonary causes like Emphysema, Asthma, Small cell carcinoma & Squamous cell carcinoma, Idiopathic pulmonary fibrosis
3. Renal diseases – patients on hemodialysis show high ACE levels as compared to patients who are not on dialysis, chronic renal failure
4. Other causes – Multiple sclerosis, Addison’s disease, Hyperthyroidism, Diabetes Alcoholic hepatitis & cirrhosis & Peptic ulcer, histoplasmosis, hodgekins disease, gauchers disease, leprosy, amyloidosis, tuberculosis
5. Elevated ACE is thought to be a risk factor for myocardial infarction & cardiomyopathy.
7. ACE inhibitors have found wide spread application in treatment of systemic hypertension and Congestive Heart Failure (CHF). Monitoring of ACE may be beneficial to determine the optimum low dose of ACE inhibitor.

#### DECREASED LEVELS

1. Chronic liver disease.
2. Anorexia nervosa
3. Hypothyroidism

To be correlated clinically



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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.

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



  
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