

Dr. Vinay Chopra  
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Chairman & Consultant Pathologist

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

NAME : Mr. VIKAS AGGARWAL  
AGE/ GENDER : 52 YRS/MALE  
COLLECTED BY : SURJESH  
REFERRED BY : CENTRAL PHOENIX CLUB (AMBALA CANTT)  
BARCODE NO. : 01513774  
CLIENT CODE : KOS DIAGNOSTIC LAB  
CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT

PATIENT ID : 1560059  
REG. NO./LAB NO. : 012407250019  
REGISTRATION DATE : 25/Jul/2024 10:11 AM  
COLLECTION DATE : 25/Jul/2024 10:22 AM  
REPORTING DATE : 25/Jul/2024 11:40 AM

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) by CALORIMETRIC	14.1	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	5.17 <sup>H</sup>	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	43.8	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	84.7	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	27.3	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	32.2	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	14.8	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	47	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	16.38	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	24.27	RATIO	BETA THALASSEMIA TRAIT: < = 65.0 IRON DEFICIENCY ANEMIA: > 65.0

WHITE BLOOD CELLS (WBCS)

TOTAL LEUCOCYTE COUNT (TLC) by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	8770	/cmm	4000 - 11000
NUCLEATED RED BLOOD CELLS (nRBCS) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER & MICROSCOPY	NIL		0.00 - 20.00
NUCLEATED RED BLOOD CELLS (nRBCS) % by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER & MICROSCOPY	NIL	%	< 10 %



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<b>DIFFERENTIAL LEUCOCYTE COUNT (DLC)</b>			
NEUTROPHILS <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	38 <sup>L</sup>	%	50 - 70
LYMPHOCYTES <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	27	%	20 - 40
EOSINOPHILS <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	30 <sup>H</sup>	%	1 - 6
MONOCYTES <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	5	%	2 - 12
BASOPHILS <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	0	%	0 - 1
IMMATURE GRANULOCYTE (IG) % <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	0	%	0 - 5.0
<b>ABSOLUTE LEUKOCYTES (WBC) COUNT</b>			
ABSOLUTE NEUTROPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	3333	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	2368	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	2631 <sup>H</sup>	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	438	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	0	/cmm	0 - 110
ABSOLUTE IMMATURE GRANULOCYTE COUNT <i>by FLOW CYTOMETRY BY SF CUBE &amp; MICROSCOPY</i>	0	/cmm	0.0 - 999.0
<b>PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS.</b>			
PLATELET COUNT (PLT) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	214000	/cmm	150000 - 450000
PLATELET CRIT (PCT) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	0.24	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) <i>by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE</i>	11	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)	33.3	%	11.0 - 45.0



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by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET DISTRIBUTION WIDTH (PDW)	16.6	%	15.0 - 17.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD			

RECHECKED.



  
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**GLYCOSYLATED HAEMOGLOBIN (HbA1c)**

GLYCOSYLATED HAEMOGLOBIN (HbA1c): WHOLE BLOOD by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)	5.8	%	4.0 - 6.4
ESTIMATED AVERAGE PLASMA GLUCOSE by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)	119.76	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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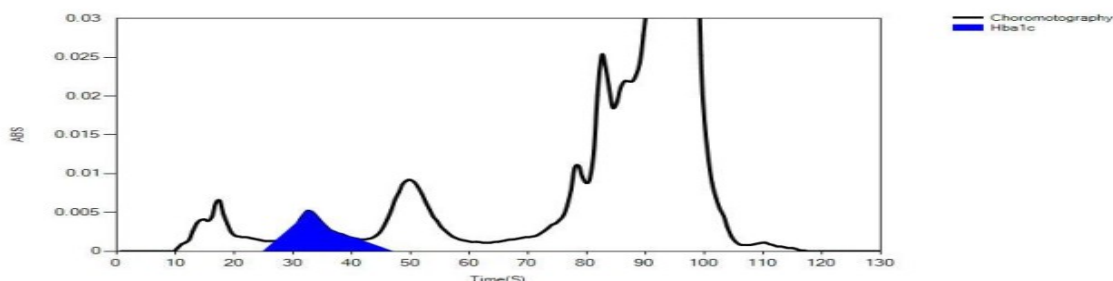
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LIFOTRONIC Graph Report

Name :	Case :	Patient Type :	Test Date : 25/07/2024 12:42:07
Age :	Department :	Sample Type : Whole Blood EDTA	Sample Id : 01513774
Gender :			Total Area : 16789

Peak Name	Retention Time(s)	Absorbance	Area	Result (Area %)
HbA0	70	4354	15159	88.0
HbA1c	36	93	1007	5.8
La1c	28	19	234	1.4
HbF	21	16	17	0.1
Hba1b	12	67	244	1.4
Hba1a	10	41	128	0.7



  
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### ERYTHROCYTE SEDIMENTATION RATE (ESR)

ERYTHROCYTE SEDIMENTATION RATE (ESR)	12	mm/1st hr	0 - 20
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by MODIFIED WESTERGREN AUTOMATED METHOD

#### 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	93.48	mg/dL	NORMAL: < 100.0
by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)			PREDIABETIC: 100.0 - 125.0
			DIABETIC: > OR = 126.0

#### 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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LIPID PROFILE : BASIC			
CHOLESTEROL TOTAL: SERUM by CHOLESTEROL OXIDASE PAP	190.81	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)	105.52	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 by SELECTIVE INHIBITION	41.76	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMETRY	127.95	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 by CALCULATED, SPECTROPHOTOMETRY	149.05 <sup>H</sup>	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 by CALCULATED, SPECTROPHOTOMETRY	21.1	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM by CALCULATED, SPECTROPHOTOMETRY	487.14	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	4.57 <sup>H</sup>	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0
LDL/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	3.06 <sup>H</sup>	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0



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TRIGLYCERIDES/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.53 <sup>L</sup>	RATIO	3.00 - 5.00

**INTERPRETATION:**

- 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.
- 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.
- 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.
- 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.
- 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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<b>BARCODE NO.</b>	: 01513774	<b>REPORTING DATE</b>	: 25/Jul/2024 12:09PM
<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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#### LIVER FUNCTION TEST (COMPLETE)

BILIRUBIN TOTAL: SERUM <i>by DIAZOTIZATION, SPECTROPHOTOMETRY</i>	0.64	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM <i>by DIAZO MODIFIED, SPECTROPHOTOMETRY</i>	0.21	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.43	mg/dL	0.10 - 1.00
SGOT/AST: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	19.26	U/L	7.00 - 45.00
SGPT/ALT: SERUM <i>by IFCC, WITHOUT PYRIDOXAL PHOSPHATE</i>	26.46	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	0.73	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM <i>by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL</i>	94.5	U/L	40.0 - 150.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM <i>by SZASZ, SPECTROPHOTOMETRY</i>	18.4	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM <i>by BIURET, SPECTROPHOTOMETRY</i>	6.81	gm/dL	6.20 - 8.00
ALBUMIN: SERUM <i>by BROMOCRESOL GREEN</i>	4.34	gm/dL	3.50 - 5.50
GLOBULIN: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	2.47	gm/dL	2.30 - 3.50
A : G RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	1.76	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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<b>NAME</b>	: Mr. VIKAS AGGARWAL		
<b>AGE/ GENDER</b>	: 52 YRS/MALE	<b>PATIENT ID</b>	: 1560059
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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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Test Name	Value	Unit	Biological Reference interval
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#### KIDNEY FUNCTION TEST (COMPLETE)

UREA: SERUM <i>by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)</i>	17.59	mg/dL	10.00 - 50.00
CREATININE: SERUM <i>by ENZYMATIC, SPECTROPHOTOMETRY</i>	0.84	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	8.22	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	9.79 <sup>L</sup>	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM <i>by CALCULATED, SPECTROPHOTOMETRY</i>	20.94	RATIO	
URIC ACID: SERUM <i>by URICASE - OXIDASE PEROXIDASE</i>	4.7	mg/dL	3.60 - 7.70
CALCIUM: SERUM <i>by ARSENazo III, SPECTROPHOTOMETRY</i>	8.45 <sup>L</sup>	mg/dL	8.50 - 10.60
PHOSPHOROUS: SERUM <i>by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY</i>	4.28	mg/dL	2.30 - 4.70

#### ELECTROLYTES

SODIUM: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	140.3	mmol/L	135.0 - 150.0
POTASSIUM: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	4.08	mmol/L	3.50 - 5.00
CHLORIDE: SERUM <i>by ISE (ION SELECTIVE ELECTRODE)</i>	105.23	mmol/L	90.0 - 110.0

#### ESTIMATED GLOMERULAR FILTRATION RATE

ESTIMATED GLOMERULAR FILTRATION RATE (eGFR): SERUM <i>by CALCULATED</i>	104.9		
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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.



  
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Test Name	Value	Unit	Biological Reference interval
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3. GI haemorrhage.
4. High protein intake.
5. Impaired renal function plus
6. 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).
7. Urine reabsorption (e.g. ureter colostomy)
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).

**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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Test Name	Value	Unit	Biological Reference interval
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### IMMUNOPATHOLOGY/SEROLOGY

#### C-REACTIVE PROTEIN (CRP)

C-REACTIVE PROTEIN (CRP) QUANTITATIVE: SERUM	1.58	mg/L	0.0 - 6.0
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by NEPHLOMETRY

#### INTERPRETATION:

1. C-reactive protein (CRP) is one of the most sensitive acute-phase reactants for inflammation.
2. CRP levels can increase dramatically (100-fold or more) after severe trauma, bacterial infection, inflammation, surgery, or neoplastic proliferation.
3. CRP levels (Quantitative) has been used to assess activity of inflammatory disease, to detect infections after surgery, to detect transplant rejection, and to monitor these inflammatory processes.
4. As compared to ESR, CRP shows an earlier rise in inflammatory disorders which begins in 4-6 hrs, the intensity of the rise being higher than ESR and the recovery being earlier than ESR. Unlike ESR, CRP levels are not influenced by hematologic conditions like Anemia, Polycythemia etc.,
5. Elevated values are consistent with an acute inflammatory process.

#### NOTE:

1. Elevated C-reactive protein (CRP) values are nonspecific and should not be interpreted without a complete clinical history.
2. Oral contraceptives may increase CRP levels.



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**BLOOD ELEMENTS/METALS AND MINERALS AND NUTRIENTS**  
**LEAD**

LEAD: BLOOD	56.05	µg/L	< 150.0
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by ICP-MS (INDUCTIVELY COUPLED PLASMA-MASS SPECTROMETRY)

**INTERPRETATION:**

Lead is the most ubiquitous toxic metal detectable in practically all phases of the inert environment and in all biological systems. Industrial exposure to lead is seen in industries manufacturing lead containing paints & ceramic glazes, batteries, water pipes & ammunition. Major exposure of the general population is through food & water. Lead containing toys & paints are a primary source of lead exposure in children. Centre for Disease Control (CDC) recommends universal screening of children from 6 months of age. Acute toxicity is uncommon as compared to chronic toxicity leading to intellectual deficit and lead induced anemias in children

LEAD INTERPRETATION	ASSOCIATED CONDITIONS
Lead exposure can occur as – 1. Occupational exposure in gasoline industries, Lead planting, auto repair plants and construction sites. 2. Other sources are like leaded house paints, drinking water from lead plumbing, vegetables grown in lead contaminated soil, certain Chinese and Ayurveda herbal preparations etc. In children exposure through soil as in Pica and toys containing lead can be sources of exposure.	Prolonged exposure to lead is often associated with Microcytic hypochromic anaemia, renal dysfunction, hypertension, anorexia, muscle discomfort, constipation, and metallic taste.

LEAD LEVELS IN (µg/L)	ACTION REQUIRED
0 - 9	No additional action
10 – 19	Identify and minimise exposure:
20 – 39	Exposure removal if symptomatic
40 – 79	Immediate evaluation
>=100	Toxicity, Chelate immediately

- Whole Blood metal testing is used for the detection of recent exposure or poisoning with the toxic element. However, blood metal levels in healthy subjects can vary considerably with exposure to the particular metal present in the diet and in the environment.
- It should be noted that low or within acceptable levels in blood do not always exclude that the element is uninvolved in contributing to the patient's symptoms because certain elements may be sequestered in tissues.
- Lower metal levels in patients on follow-up imply that the toxic element exposure is reduced in the patient's immediate environment or that the body has efficiently eliminated the toxic element.

**NOTE:**

- Inductively Coupled Plasma-Mass Spectrometry is used to determine the level of heavy / trace metals in biological tissues
- To assess occupational exposure sample should be collected at the end of the shift on the last day of the work week

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



  
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