

(A Unit of KOS Healthcare)



Dr. Vinay Chopra
MD (Pathology & Microbiology)
Chairman & Consultant Pathologist

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

NAME : Mr. MUKESH KUMAR

**AGE/ GENDER** : 52 YRS/MALE **PATIENT ID** : 1811746

COLLECTED BY : REG. NO./LAB NO. : 012503300009

 REFERRED BY
 : 30/Mar/2025 08:29 AM

 BARCODE NO.
 : 01528010
 COLLECTION DATE
 : 30/Mar/2025 08:36AM

 CLIENT CODE.
 : KOS DIAGNOSTIC LAB
 REPORTING DATE
 : 30/Mar/2025 10:04AM

**CLIENT ADDRESS**: 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name Value Unit Biological Reference interval

## SWASTHYA WELLNESS PANEL: D COMPLETE BLOOD COUNT (CBC)

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

RED DECOD CELLS (RDCS) COUNT AND INDICES			
HAEMOGLOBIN (HB) by CALORIMETRIC	14.6	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	4.88	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	43.3	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	88.6	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	29.9	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	33.8	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	13.4	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	44.7	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	18.16	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	72.05	RATIO	BETA THALASSEMIA TRAIT: <= 74.1 IRON DEFICIENCY ANEMIA: >= 74.1
WHITE BLOOD CELLS (WBCS)			
TOTAL LEUCOCYTE COUNT (TLC)	$13100^{\mathrm{H}}$	/cmm	4000 - 11000

TOTAL LEUCOCYTE COUNT (TLC)
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY

NUCLEATED RED BLOOD CELLS (nRBCS)
by AUTOMATED 6 PART HEMATOLOGY ANALYZER

NUCLEATED RED BLOOD CELLS (nRBCS) %

NIL

4000 - 11000

0.00 - 20.00

0.00 - 20.00

× 10 %



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by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER			
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	77 <sup>H</sup>	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	17 <sup>L</sup>	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2	%	1 - 6
MONOCYTES  by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	4	%	2 - 12
BASOPHILS by flow cytometry by Sf cube & microscopy  ABSOLUTE LEUKOCYTES (WBC) COUNT	0	%	0 - 1
ABSOLUTE NEUTROPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	10087 <sup>H</sup>	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2227	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	262	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	524	/cmm	80 - 880
PLATELETS AND OTHER PLATELET PREDICTIV	E MARKERS.		
PLATELET COUNT (PLT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	199000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.26	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by hydro dynamic focusing, electrical impedence	13 <sup>H</sup>	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	93000 <sup>H</sup>	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	46.7 <sup>H</sup>	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD	16.1	%	15.0 - 17.0



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KOS Central Lab: 6349/1, Nicholson Road, Ambala Cantt -133 001, Haryana KOS Molecular Lab: IInd Floor, Parry Hotel, Staff Road, Opp. GPO, Ambala Cantt -133 001, Haryana



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# KOS Diagnostic Lab (A Unit of KOS Healthcare)



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DR.VINAY CHOPRA CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY & MICROBIOLOGY)



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#### GLYCOSYLATED HAEMOGLOBIN (HBA1C)

GLYCOSYLATED HAEMOGLOBIN (HbA1c): 8.6<sup>H</sup>

WHOLE BLOOD

by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)

ESTIMATED AVERAGE PLASMA GLUCOSE

by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)

mg/dL

60.00 - 140.00

4.0 - 6.4

: 30/Mar/2025 03:35PM

**INTERPRETATION:** 

CLIENT CODE.

REFERENCE GROUP	GLYCOSYLATED HEMOGL	OGIB (HBAIC) in %
Non diabetic Adults >= 18 years	<5.7	
At Risk (Prediabetes)	5.7 – 6.	4
Diagnosing Diabetes	>= 6.5	
	Age > 19 Y	ears
	Goals of Therapy:	< 7.0
Therapeutic goals for glycemic control	Actions Suggested:	>8.0
	Age < 19 Y	ears
	Goal of therapy:	<sub>-75</sub>

 $200.12^{H}$ 

#### COMMENTS:

- 1.Glycosylated hemoglobin (HbA1c) test is three monthly monitoring done to assess compliace with therapeutic regimen in diabetic patients. 2. 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 HbAlc. Converse is true for a diabetic previously under good control but now poorly controlled.
- 3. 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, targetting a goal of < 7.0% may not be
- 4.High HbA1c (>9.0 -9.5 %) is strongly associated with risk of development and rapid progression of microvascular and nerve complications 5.Any condition that shorten RBC life span like acute blood loss, hemolytic anemia falsely lower HbA1c results.
- 6.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 gycemic control.
- 7. Specimens from patients with polycythemia or post-splenctomy may exhibit increse in HbA1c values due to a somewhat longer life span of the red cells.



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#### **ERYTHROCYTE SEDIMENTATION RATE (ESR)**

ERYTHROCYTE SEDIMENTATION RATE (ESR)

26<sup>H</sup>

mm/1st hr

0 - 20

by RED CELL AGGREGATION BY CAPILLARY PHOTOMETRY

#### INTERPRETATION:

- 1. ESR is a non-specific test because an elevated result often indicates the presence of inflammation associated with infection, cancer and autoimmune 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:

- ESR and C reactive protein (C-RP) are both markers of inflammation.
   Generally, ESR does not change as rapidly as does CRP, either at the start of inflammation or as it resolves.
   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

156.7<sup>H</sup> mg/dL NORMAL: < 100.0 by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD)

PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 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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Test Name	Value	Unit	Biological Reference interval
	LIPID PROFILE	: BASIC	
CHOLESTEROL TOTAL: SERUM by CHOLESTEROL OXIDASE PAP	187.81	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)	156.82 <sup>H</sup>	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	36.7	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMETRY	119.75	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	151.11 <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	31.36	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM by CALCULATED, SPECTROPHOTOMETRY	532.44	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	5.12 <sup>H</sup>	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0



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Test Name	Value	Unit	Biological Reference interval
			MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0
LDL/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	3.26 <sup>H</sup>	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/HDL RATIO: SERUM by CALCULATED. SPECTROPHOTOMETRY	4.27	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

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 atherogeniclipoproteins such as LDL, VLDL, IDL, Lpa, 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 LDL.

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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#### LIVER FUNCTION TEST (COMPLETE)

BILIRUBIN TOTAL: SERUM by DIAZOTIZATION, SPECTROPHOTOMETRY	0.76	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.18	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.58	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	25.25	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	23.64	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM  by CALCULATED, SPECTROPHOTOMETRY	1.07	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL	98.23	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	19.89	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	7.36	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	4.24	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	3.12	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.36	RATIO	1.00 - 2.00

**INTERPRETATION** 

NOTE:- To be correlated in individuals having SGOT and SGPT values higher than Normal Referance 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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Test Name	Value	Unit	Biological Reference interval
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS		> 1.3 (Slightly Increased)	
DECDEASED.			

REPORTING DATE

#### DECREASED:

CLIENT CODE.

- 1. Acute Hepatitis due to virus, drugs, toxins (with AST increased 3 to 10 times upper limit of normal)
- 2. Extra Hepatic cholestatis: 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 by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)	19.47	mg/dL	10.00 - 50.00
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETERY	0.94	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY	9.1	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	9.68 <sup>L</sup>	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	20.71	RATIO	
URIC ACID: SERUM by URICASE - OXIDASE PEROXIDASE	3.6	mg/dL	3.60 - 7.70
CALCIUM: SERUM by ARSENAZO III, SPECTROPHOTOMETRY	9.3	mg/dL	8.50 - 10.60
PHOSPHOROUS: SERUM by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY	3.4	mg/dL	2.30 - 4.70
ELECTROLYTES			
SODIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)	143.5	mmol/L	135.0 - 150.0
POTASSIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)	4.54	mmol/L	3.50 - 5.00
CHLORIDE: SERUM by ISE (ION SELECTIVE ELECTRODE)	107.63	mmol/L	90.0 - 110.0

#### **ESTIMATED GLOMERULAR FILTERATION RATE**

ESTIMATED GLOMERULAR FILTERATION RATE 97.5

(eGFR): SERUM by CALCULATED 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.



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**NAME** : Mr. MUKESH KUMAR

AGE/ GENDER : 52 YRS/MALE **PATIENT ID** : 1811746

**COLLECTED BY** :012503300009 REG. NO./LAB NO.

REFERRED BY **REGISTRATION DATE** : 30/Mar/2025 08:29 AM BARCODE NO. :01528010 **COLLECTION DATE** : 30/Mar/2025 08:36AM CLIENT CODE. : KOS DIAGNOSTIC LAB REPORTING DATE :30/Mar/2025 11:53AM

**CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name Value Unit **Biological Reference interval** 

- 2. Catabolic states with increased tissue breakdown.
- 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:

- 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 inappropiate antidiuretic harmone) 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.

#### **INAPPROPIATE 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 FILTERATION RATE**:

LUTINIATED GEOWIERGEART	ILI LIKATION KATE.		
CKD STAGE	DESCRIPTION	GFR ( mL/min/1.73m2 )	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 m2 (G3) and without any marker of Kidney damage, It is recommended to measure

4. eGFR category G1 OR G2 does not fullfill 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).

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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#### **VITAMINS**

#### VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM ng/mL DEFICIENCY: < 20.0

by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0

TOXICITY: > 100.0

**INTERPRETATION:** 

DEFICIENT:	< 20	ng/mL
INSUFFICIENT:	21 - 29	ng/mL
PREFFERED RANGE:	30 - 100	ng/mL
INTOXICATION:	> 100	ng/ml

1. Vitamin D compounds are derived from dietary ergocalciferol (from plants, Vitamin D2), or cholecalciferol (from animals, Vitamin D3), or by conversion of 7- dihydrocholecalciferol to Vitamin D3 in the skin upon Ultraviolet exposure.

2.25-OH--Vitamin D represents the main body resevoir and transport form of Vitamin D and transport form of Vitamin D, being stored in adipose tissue and tightly bound by a transport protein while in circulation.

3. Vitamin D plays a primary role in the maintenance of calcium homeostatis. It promotes calcium absorption, renal calcium absorption and phosphate reabsorption, skeletal calcium deposition, calcium mobilization, mainly regulated by parathyroid harmone (PTH).

4. Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults.

#### **DECREASED:**

- 1.Lack of sunshine exposure.
- 2.Inadequate intake, malabsorption (celiac disease)
  3.Depressed Hepatic Vitamin D 25- hydroxylase activity
- 4. Secondary to advanced Liver disease
- 5. Osteoporosis and Secondary Hyperparathroidism (Mild to Moderate deficiency)
- 6.Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism.
- 1. Hypervitaminosis D is Rare, and is seen only after prolonged exposure to extremely high doses of Vitamin D. When it occurs, it can result in severe hypercalcemia and hyperphophatemia.

CAUTION: Replacement therapy in deficient individuals must be monitored by periodic assessment of Vitamin D levels in order to prevent hypervitaminosis D

NOTE:-Dark coloured individuals as compare to whites, is at higher risk of developing Vitamin D deficiency due to excess of melanin pigment which interefere with Vitamin D absorption.



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Test Name Value Unit Biological Reference interval

## CLINICAL PATHOLOGY URINE ROUTINE & MICROSCOPIC EXAMINATION

#### PHYSICAL EXAMINATION

QUANTITY RECIEVED 10 ml

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

COLOUR PALE YELLOW PALE YELLOW

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

TRANSPARANCY CLEAR CLEAR

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

SPECIFIC GRAVITY 1.02 1.002 - 1.030

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY.

#### CHEMICAL EXAMINATION

REACTION ACIDIC by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

PROTEIN Negative NEGATIVE (-ve)

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

SUGAR Negative NEGATIVE (-ve)

pH 5.5 5.0 - 7.5

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

BILIRUBIN Negative NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

NITRITE Negative NEGATIVE (-ve)

UROBILINOGEN Normal EU/dL 0.2 - 1.0

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

KETONE BODIES

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

NEGATIVE (-ve)

NEGATIVE (-ve)

BLOOD NEGATIVE (-ve) NEGATIVE (-ve)

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

ASCORBIC ACID NEGATIVE (-ve) NEGATIVE (-ve) by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

MICROSCOPIC EXAMINATION



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Test Name	Value	Unit	Biological Reference interval
RED BLOOD CELLS (RBCs) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)	/HPF	0 - 3
PUS CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	3-4	/HPF	0 - 5
EPITHELIAL CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	2-3	/HPF	ABSENT
CRYSTALS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)
CASTS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)
BACTERIA by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)
OTHERS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)
TRICHOMONAS VAGINALIS (PROTOZOA) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	ABSENT		ABSENT



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Value Unit Test Name **Biological Reference interval** 

#### MICROALBUMIN/CREATININE RATIO - RANDOM URINE

MICROALBUMIN: RANDOM URINE 20.19 mg/L by SPECTROPHOTOMETRY

108.97 CREATININE: RANDOM URINE mg/dL 20 - 320

by SPECTROPHOTOMETRY MICROALBUMIN/CREATININE RATIO -18.53 0 - 30mg/g

RANDOM URINE by SPECTROPHOTOMETRY

**INTERPRETATION:-**

PHYSIOLOGICALLY NORMAL:	mg/L	0 - 30
MICROALBUMINURIA:	mg/L	30 - 300
GROSS PROTEINURIA:	mg/L	> 300

Long standing un-treated Diabetes and Hypertension can lead to renal dysfunction.

2. Diabetic nephropathy or kidney disease is the most common cause of end stage renal disease(ERSD) or kidney failure. 3. Presence of Microalbuminuria is an early indicator of onset of compromised renal function in these patients.

4. Microalbuminuria is the condition when urinary albumin excretion is between 30-300 mg & above this it is called as macroalbuminuria, the

4.Microalbuminuma is the condition when urnary albumin excretion is between 30-300 mg & above this it is called as macroalbuminuma, the presence of which indicates serious kidney disease.

5.Microalbuminuma is not only associated with kidney disease but of cardiovascular disease in patients with dibetes & hypertension.

6.Microalbuminuma reflects vascular damage & appear to be a marker of of early arterial disease & endothelial dysfunction.

NOTE:- IF A PATIENT HAS = 1+ PROTEINURIA (30 mg/dl OR 300 mg/L) BY URINE DIPSTICK (URINEANALYSIS), OVERT PROTEINURIA IS PRESENT AND TESTING FOR MICROALBUMIN IS INAPPROPIATE. IN SUCH A CASE, URINE PROTEIN:CREATININE RATIO OR 24 HOURS TOTAL URINE MICROPROTEIN IS APPROPIATE.

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



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