

(A Unit of KOS Healthcare)



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

NAME : Mrs. KIRAN

**AGE/ GENDER** : 73 YRS/FEMALE **PATIENT ID** : 1621341

COLLECTED BY : REG. NO./LAB NO. : 012409220011

 REFERRED BY
 : 22/Sep/2024 07:45 AM

 BARCODE NO.
 : 01517446
 COLLECTION DATE
 : 22/Sep/2024 07:45 AM

 CLIENT CODE.
 : KOS DIAGNOSTIC LAB
 REPORTING DATE
 : 22/Sep/2024 08:42 AM

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

Test Name Value Unit Biological Reference interval

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

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

HAEMOGLOBIN (HB) by CALORIMETRIC	10.5 <sup>L</sup>	gm/dL	12.0 - 16.0
RED BLOOD CELL (RBC) COUNT  by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	3.8	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZE	33.6 <sup>L</sup>	%	37.0 - 50.0
MEAN CORPUSCULAR VOLUME (MCV) by calculated by automated hematology analyzei	88.5 R	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZEI	27.7 R	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by calculated by automated hematology analyze	31.3 <sup>L</sup>	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by Calculated by automated hematology analyzed	14.1 R	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by Calculated by automated hematology analyzed	46.5 R	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	23.29	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	32.92	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	8480	/cmm	4000 - 11000
NUCLEATED RED BLOOD CELLS (nRBCS) by automated 6 part hematology analyzer	NIL		0.00 - 20.00
NUCLEATED RED BLOOD CELLS (nRBCS) % by calculated by automated hematology analyzed DIFFERENTIAL LEUCOCYTE COUNT (DLC)	NIL R	%	< 10 %
	/7	0/	F0 70
NEUTROPHILS by Flow cytometry by SF cube & Microscopy	67	%	50 - 70



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Test Name	Value	Unit	Biological Reference interval
YMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	23	%	20 - 40
OSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	6	%	1 - 6
MONOCYTES by flow cytometry by sf cube & microscopy	4	%	2 - 12
ASOPHILS by flow cytometry by sf cube & microscopy BSOLUTE LEUKOCYTES (WBC) COUNT	0	%	0 - 1
BSOLUTE NEUTROPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	5682	/cmm	2000 - 7500
BSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1950	/cmm	800 - 4900
BSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	509 <sup>H</sup>	/cmm	40 - 440
BSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	339	/cmm	80 - 880
BSOLUTE BASOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY LATELETS AND OTHER PLATELET PREDICTIVE MARKER	0 <b>RS.</b>	/cmm	0 - 110
LATELET COUNT (PLT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	308000	/cmm	150000 - 450000
LATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.3	%	0.10 - 0.36
1EAN PLATELET VOLUME (MPV) by hydro dynamic focusing, electrical impedence	10	fL	6.50 - 12.0
LATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	74000	/cmm	30000 - 90000
LATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	23.8	%	11.0 - 45.0
LATELET DISTRIBUTION WIDTH (PDW) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE IOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD	16	%	15.0 - 17.0



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Test Name Value Unit Biological Reference interval

### **GLYCOSYLATED HAEMOGLOBIN (HBA1C)**

GLYCOSYLATED HAEMOGLOBIN (HbA1c): 6.2 % 4.0 - 6.4

WHOLE BLOOD

by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)

ESTIMATED AVERAGE PLASMA GLUCOSE 131.24 mg/dL 60.00 - 140.00

by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)

#### **INTERPRETATION:**

AS PER AMERICAN DIABETES ASSOCIATION (ADA):				
REFERENCE GROUP GLYCOSYLATED HEMOGLOGIB (HBAIC) in %				
Non diabetic Adults >= 18 years <5.7				
At Risk (Prediabetes) 5.7 – 6.4		4		
Diagnosing Diabetes	>= 6.5			
	Age > 19 Years			
Therapeutic goals for glycemic control	Goals of Therapy:	< 7.0		
	Actions Suggested:	>8.0		
	Age < 19 Years			
	Goal of therapy:	<7.5		

#### **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 appropriate.
- 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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Value Unit **Biological Reference interval** Test Name

### ERYTHROCYTE SEDIMENTATION RATE (ESR)

**ERYTHROCYTE SEDIMENTATION RATE (ESR)** 

mm/1st hr

0 - 20

: 22/Sep/2024 08:53AM

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:

- 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 services and quiping may decrease it. aspirin, cortisone, and quinine may decrease it



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**Test Name** Value Unit **Biological Reference interval** 

### CLINICAL CHEMISTRY/BIOCHEMISTRY **GLUCOSE FASTING (F)**

99.79 GLUCOSE FASTING (F): PLASMA 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	132.69	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)	87.22	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	48.77	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMETRY	66.48	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	83.92	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	17.44	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM by CALCULATED, SPECTROPHOTOMETRY	352.6	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.72	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	1.36	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 1.79<sup>L</sup> **RATIO** 3.00 - 5.00by CALCULATED, SPECTROPHOTOMETRY

**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 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

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 (C	OMPLETE)
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BILIRUBIN TOTAL: SERUM by DIAZOTIZATION, SPECTROPHOTOMETRY	0.43	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.13	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.3	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	14.55	U/L	7.00 - 45.00
SGPT/ALT: SERUM  by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	19.94	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM  by CALCULATED, SPECTROPHOTOMETRY	0.73	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by Para Nitrophenyl Phosphatase by Amino Methy PROPANOL	81.69 L	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	12.49	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	6.17 <sup>L</sup>	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	3.33 <sup>L</sup>	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.84	gm/dL	2.30 - 3.50
A : G RATIO: SERUM	1.17	RATIO	1.00 - 2.00

#### **INTERPRETATION**

by CALCULATED, SPECTROPHOTOMETRY

*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
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 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)	69.17 <sup>H</sup>	mg/dL	10.00 - 50.00
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETERY	2.02 <sup>H</sup>	mg/dL	0.40 - 1.20
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY	32.32 <sup>H</sup>	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM	16	RATIO	10.0 - 20.0
by CALCULATED, SPECTROPHOTOMETRY UREA/CREATININE RATIO: SERUM	34.24	RATIO	
by CALCULATED, SPECTROPHOTOMETRY  URIC ACID: SERUM  by URICASE - OXIDASE PEROXIDASE	6.9 <sup>H</sup>	mg/dL	2.50 - 6.80
CALCIUM: SERUM by ARSENAZO III, SPECTROPHOTOMETRY	8.98	mg/dL	8.50 - 10.60
PHOSPHOROUS: SERUM by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY	3.61	mg/dL	2.30 - 4.70
ELECTROLYTES			
SODIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)	138.6	mmol/L	135.0 - 150.0
POTASSIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)	4.42	mmol/L	3.50 - 5.00
CHLORIDE: SERUM  by ISE (ION SELECTIVE ELECTRODE)	103.95	mmol/L	90.0 - 110.0
ESTIMATED GLOMERULAR FILTERATION RATE			
ESTIMATED GLOMERULAR FILTERATION RATE	25.6		

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



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

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

: 22/Sep/2024 11:48AM

**NAME** : Mrs. KIRAN

AGE/ GENDER : 73 YRS/FEMALE **PATIENT ID** : 1621341

**COLLECTED BY** :012409220011 REG. NO./LAB NO.

REFERRED BY **REGISTRATION DATE** : 22/Sep/2024 07:45 AM BARCODE NO. :01517446 **COLLECTION DATE** : 22/Sep/2024 07:45AM

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

Test Name Value Unit **Biological Reference interval** 

REPORTING DATE

3. GI haemorrhage.

CLIENT CODE.

- 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 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:

ESTIMINATED SEGMENOLINATIES ENGLISH NATE:					
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

End Of Report \*\*\*



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