

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



	Dr. Vinay Chop MD (Pathology & Mi Chairman & Consult	crobiology)		(Pathology)
NAME	: Mr. RAJINDER KUMAR			
AGE/ GENDER	: 63 YRS/MALE		PATIENT ID	: 1697125
COLLECTED BY	: SURJESH		REG. NO./LAB NO.	: 012412120013
REFERRED BY	:		<b>REGISTRATION DATE</b>	: 12/Dec/2024 09:55 AM
BARCODE NO.	: 01522341		COLLECTION DATE	: 12/Dec/2024 12:23PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 12/Dec/2024 10:22AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	BALA CANTT		
Test Name		Value	Unit	Biological Reference interval
			ELLNESS PANEL: G DOD COUNT (CBC)	
RED BLOOD CELLS	(RBCS) COUNT AND INDICES			
HAEMOGLOBIN (H)		12 <sup>L</sup>	gm/dL	12.0 - 16.0
RED BLOOD CELL (		4.56	Millions/	cmm 3.50 - 5.00
PACKED CELL VOLU	OCUSING, ELECTRICAL IMPEDENCE JME (PCV) UTOMATED HEMATOLOGY ANALYZER	38.3	%	37.0 - 50.0
MEAN CORPUSCUL		84	fL	80.0 - 100.0
	AR HAEMOGLOBIN (MCH) UTOMATED HEMATOLOGY ANALYZER	26.4 <sup>L</sup>	pg	27.0 - 34.0
MEAN CORPUSCUL	AR HEMOGLOBIN CONC. (MCHC UTOMATED HEMATOLOGY ANALYZER	) <b>31.5<sup>L</sup></b>	g/dL	32.0 - 36.0
	JTION WIDTH (RDW-CV) UTOMATED HEMATOLOGY ANALYZER	14.7	%	11.00 - 16.00
RED CELL DISTRIB	JTION WIDTH (RDW-SD) UTOMATED HEMATOLOGY ANALYZER	46.2	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED		18.42	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING IND by CALCULATED		27.17	RATIO	BETA THALASSEMIA TRAIT:< 65.0 IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CEI				
TOTAL LEUCOCYTE	BY SF CUBE & MICROSCOPY	8420	/cmm	4000 - 11000
•		NIL		0.00 - 20.00
NUCLEATED RED B	LOOD CELLS (nRBCS) RT HEMATOLOGY ANALYZER	NIL		

KOS Diagnostic Lab (A Unit of KOS Healthcare)





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

DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY)

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Dr. Vinay Chopra Dr. Yugam Chopra MD (Pathology & Microbiology) MD (Pathology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** NAME : Mr. RAJINDER KUMAR **AGE/ GENDER** : 63 YRS/MALE **PATIENT ID** :1697125 **COLLECTED BY** : SURJESH REG. NO./LAB NO. :012412120013 **REFERRED BY REGISTRATION DATE** : 12/Dec/2024 09:55 AM : **BARCODE NO.** :01522341 **COLLECTION DATE** :12/Dec/2024 12:23PM CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE** : 12/Dec/2024 10:22AM **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Unit **Biological Reference interval DIFFERENTIAL LEUCOCYTE COUNT (DLC)** NEUTROPHILS 57 % 50 - 70 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY LYMPHOCYTES 32 % 20 - 40 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY EOSINOPHILS 5 % 1 - 6 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY MONOCYTES 6 % 2 - 12by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY BASOPHILS 0 % 0 - 1 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY **ABSOLUTE LEUKOCYTES (WBC) COUNT** ABSOLUTE NEUTROPHIL COUNT 4799 2000 - 7500 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE LYMPHOCYTE COUNT 2694 800 - 4900 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE EOSINOPHIL COUNT 421/cmm 40 - 440 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE MONOCYTE COUNT 505 /cmm 80 - 880 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE BASOPHIL COUNT 0 /cmm 0 - 110 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS. PLATELET COUNT (PLT) 150000 - 450000 116000<sup>L</sup> /cmm by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELETCRIT (PCT) 0.16 % 0.10 - 0.36 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE MEAN PLATELET VOLUME (MPV) 14<sup>H</sup> fL 6.50 - 12.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE 30000 - 90000 PLATELET LARGE CELL COUNT (P-LCC) 64000 /cmm by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE % PLATELET LARGE CELL RATIO (P-LCR) 54.9<sup>H</sup> 11.0 - 45.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET DISTRIBUTION WIDTH (PDW) 15.0 - 17.0 16.6% by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE

ADVICE



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

**KINDLY CORRELATE CLINICALLY** 







	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbiology) Chairman & Consultant Pathologis		(Pathology)
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Test Name	Value	Unit	Biological Reference interval

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

RECHECKED.



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CLIENT CODE.	: KOS DIAGNOSTIC LAB	R	EPORTING DATE	: 12/Dec/2024 01:51PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	/IBALA CANTT		
Test Name		Value	Unit	Biological Reference interva
	EMOGLOBIN (HbA1c):	5.8	EMOGLOBIN (HBA1)	4.0 - 6.4
		0.0	70	4.0 - 6.4
WHOLE BLOOD by HPLC (HIGH PERFOR	RMANCE LIQUID CHROMATOGRAPHY)	0.0	70	4.0 - 0.4
by HPLC (HIGH PERFOR	RMANCE LIQUID CHROMATOGRAPHY) GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY)	119.76	/o mg/dL	4.0 - 6.4 60.00 - 140.00
by HPLC (HIGH PERFOR ESTIMATED AVERA by HPLC (HIGH PERFOR	GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY)	119.76	mg/dL	
by HPLC (HIGH PERFOI ESTIMATED AVERA by HPLC (HIGH PERFOI INTERPRETATION:	GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN D	119.76	mg/dL	60.00 - 140.00
by HPLC (HIGH PERFOI ESTIMATED AVERA by HPLC (HIGH PERFOI INTERPRETATION:	GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN D REFERENCE GROUP	119.76	mg/dL TION (ADA): COSYLATED HEMOGLOGIB	60.00 - 140.00
by HPLC (HIGH PERFOI ESTIMATED AVERA by HPLC (HIGH PERFOI INTERPRETATION: Non dia	GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN D REFERENCE GROUP abetic Adults >= 18 years	119.76	mg/dL TION (ADA): COSYLATED HEMOGLOGIB <5.7	60.00 - 140.00
by HPLC (HIGH PERFOI ESTIMATED AVERA by HPLC (HIGH PERFOI INTERPRETATION: Non dia A	GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN D REFERENCE GROUP abetic Adults >= 18 years t Risk (Prediabetes)	119.76	mg/dL TION (ADA): COSYLATED HEMOGLOGIB <5.7 5.7 - 6.4	60.00 - 140.00
by HPLC (HIGH PERFOI ESTIMATED AVERA by HPLC (HIGH PERFOI INTERPRETATION: Non dia A	GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN D REFERENCE GROUP abetic Adults >= 18 years	119.76	mg/dL TION (ADA): COSYLATED HEMOGLOGIB <5.7 5.7 - 6.4 >= 6.5	60.00 - 140.00
by HPLC (HIGH PERFOI ESTIMATED AVERA by HPLC (HIGH PERFOI INTERPRETATION: NOT dia A	GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN D REFERENCE GROUP abetic Adults >= 18 years t Risk (Prediabetes)	119.76	mg/dL TION (ADA): COSYLATED HEMOGLOGIB <5.7 5.7 - 6.4	60.00 - 140.00
by HPLC (HIGH PERFOI ESTIMATED AVERA by HPLC (HIGH PERFOI INTERPRETATION: Non dia A D	GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN D REFERENCE GROUP abetic Adults >= 18 years t Risk (Prediabetes)	119.76	mg/dL TION (ADA): COSYLATED HEMOGLOGIB <5.7 5.7 - 6.4 >= 6.5 Age > 19 Years	60.00 - 140.00 (HBAIC) in %
by HPLC (HIGH PERFOI ESTIMATED AVERA by HPLC (HIGH PERFOI INTERPRETATION: Non dia A D	GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN D REFERENCE GROUP abetic Adults >= 18 years t Risk (Prediabetes) iagnosing Diabetes	119.76 ABETES ASSOCIAT GLY GOals o Actions 5	mg/dL TION (ADA): COSYLATED HEMOGLOGIB <5.7 5.7 – 6.4 >= 6.5 Age > 19 Years f Therapy:	60.00 - 140.00 (HBAIC) in %

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# 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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LIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT		
Cest Name		Value	Unit	Biological Reference interva
oolycythaemia), sig s sickle cells in sick IOTE: . ESR and C - reactiv . Generally, ESR doe . CRP is not affected	n with conditions that inhibit the	Int (leucocytosis) , a R. of inflammation. RP, either at the sta , <b>making it a better</b>	and some protein abno rt of inflammation or a: <b>marker of inflammatior</b>	rmalities. Šome changes in red cell shape (s s it resolves.
. Drugs such as dext	ive a higher ESR, and menstruation tran, methyldopa, oral contracepti nd quinine may decrease it	and pregnancy čan ves, penicillamine p	cause temporary eleva procainamide, theophy	ations. Iline, and vitamin A can increase ESR, while





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CLIENT CODE.	: KOS DIAGNOSTIC LAB	R	EPORTING DATE	: 12/Dec/2024 11:15AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD	, AMBALA CANTT		
Test Name		Value	Unit	<b>Biological Reference interval</b>
	CLINI	CAL CHEMIST	RY/BIOCHEMIST ASTING (F)	'nY
GLUCOSE FASTING by GLUCOSE OXIDAS	E (F): PLASMA E - PEROXIDASE (GOD-POD)	102.7 <sup>H</sup>	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0

**IN ACCRDANCE 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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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	G	LUCOSE POST	F PRANDIAL (PP)	
	ANDIAL (PP): PLASMA E - PEROXIDASE (GOD-POD)	126.99	mg/dL	NORMAL: < 140.00 PREDIABETIC: 140.0 - 200.0 DIABETIC: > 0R = 200.0

TEST PERFORMED AT KOS DIAGNOSTIC LAB. AMBALA CANTT

INTERPRETATION IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES: 1. A post-prandial plasma glucose level below 140 mg/dl is considered normal. 2. A post-prandial glucose level between 140 - 200 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 post-prandial plasma glucose level of above 200 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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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD	AMBALA CANTT		
Test Name		Value	Unit	<b>Biological Reference interval</b>
		LIPID PROFI	LE : BASIC	
CHOLESTEROL TOT	AL: SERUM	114.92	mg/dL	OPTIMAL: < 200.0
by CHOLESTEROL OX			8	BORDERLINE HIGH: 200.0 -
				239.0 HIGH CHOLESTEROL: > OR =
				240.0
TRIGLYCERIDES: SI		150.93 <sup>H</sup>	mg/dL	OPTIMAL: < 150.0
by GLYCEROL PHOSPI	HATE OXIDASE (ENZYMATIC)			BORDERLINE HIGH: 150.0 - 199.0
				HIGH: 200.0 - 499.0
		00 77		VERY HIGH: $> OR = 500.0$
by SELECTIVE INHIBITI	L (DIRECT): SERUM ON	33.77	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0
				60.0
LDL CHOLESTEROL	· CEDIIM	50.96	mg/dL	HIGH HDL: > OR = 60.0 OPTIMAL: < 100.0
by CALCULATED, SPE		50.90	liig/ uL	ABOVE OPTIMAL: < 100.0 - 129.
				BORDERLINE HIGH: 130.0 -
				159.0 HIGH: 160.0 - 189.0
				VERY HIGH: $> OR = 190.0$
NON HDL CHOLEST		81.15	mg/dL	OPTIMAL: < 130.0
by CALCULATED, SPE	CIROPHOIOMEIRY			ABOVE OPTIMAL: 130.0 - 159. BORDERLINE HIGH: 160.0 -
				189.0
				HIGH: 190.0 - 219.0
VLDL CHOLESTERO	DL: SERUM	30.19	mg/dL	VERY HIGH: > OR = 220.0 0.00 - 45.00
by CALCULATED, SPE	CTROPHOTOMETRY			
FOTAL LIPIDS: SER by CALCULATED, SPE		380.77	mg/dL	350.00 - 700.00
CHOLESTEROL/HD	L RATIO: SERUM	3.4	RATIO	LOW RISK: 3.30 - 4.40
by CALCULATED, SPE	CTROPHOTOMETRY			AVERAGE RISK: 4.50 - 7.0
				MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0



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Test Name		Value	Unit	<b>Biological Reference interval</b>
LDL/HDL RATIO: S by CALCULATED, SPE		1.51	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/H by CALCULATED, SPE	IDL RATIO: SERUM	4.47	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.

 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 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 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
	LIVER	FUNCTIO	ON TEST (COMPLETE)	
BILIRUBIN TOTAL by DIAZOTIZATION, S		0.41	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
	Г (CONJUGATED): SERUM spectrophotometry	0.11	mg/dL	0.00 - 0.40
BILIRUBIN INDIRE	ECT (UNCONJUGATED): SERUM	0.3	mg/dL	0.10 - 1.00
SGOT/AST: SERUM	[ /RIDOXAL PHOSPHATE	20.7	U/L	7.00 - 45.00
SGPT/ALT: SERUM	[ /RIDOXAL PHOSPHATE	22.4	U/L	0.00 - 49.00
AST/ALT RATIO: S	ERUM ECTROPHOTOMETRY	0.92	RATIO	0.00 - 46.00
ALKALINE PHOSP by PARA NITROPHEN PROPANOL	HATASE: SERUM IYL PHOSPHATASE BY AMINO METHYL	2.09 <sup>L</sup>	U/L	40.0 - 130.0
GAMMA GLUTAMY by SZASZ, SPECTRO	L TRANSFERASE (GGT): SERUM	16.8	U/L	0.00 - 55.0
TOTAL PROTEINS: by BIURET, SPECTRO		6.56	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL G		4.1	gm/dL	3.50 - 5.50
GLOBULIN: SERUN by CALCULATED, SPI	I ECTROPHOTOMETRY	2.46	gm/dL	2.30 - 3.50
A : G RATIO: SERU	M	1.67	RATIO	1.00 - 2.00

by CALCULATED, SPECTROPHOTOMETRY

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
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)





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

DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY)



TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

INTERPRETATION





	<b>Dr. Vinay Chop</b> MD (Pathology & Mi Chairman & Consult	crobiology) ME	m Chopra D (Pathology) ht Pathologist
NAME	: Mr. RAJINDER KUMAR		
AGE/ GENDER	: 63 YRS/MALE	PATIENT ID	: 1697125
<b>COLLECTED BY</b>	: SURJESH	<b>REG. NO./LAB NO.</b>	: 012412120013
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 12/Dec/2024 09:55 AM
BARCODE NO.	: 01522341	COLLECTION DATE	: 12/Dec/2024 12:23PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	<b>REPORTING DATE</b>	: 12/Dec/2024 06:11PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	BALA CANTT	
Test Name		Value Unit	Biological Reference interval

### 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).

NORMAL	< 0.65
GOOD PROGNOSTIC SIGN	0.3 - 0.6
POOR PROGNOSTIC SIGN	1.2 - 1.6



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

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 www.koshealthcare.com







0 3001.2000 02.01					
	Dr. Vinay Cho MD (Pathology & N Chairman & Consu	1icrobiology)	Dr. Yugam MD CEO & Consultant	(Pathology)	
NAME	: Mr. RAJINDER KUMAR				
AGE/ GENDER	: 63 YRS/MALE	PAT	IENT ID	: 1697125	
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, Al				
Test Name		Value	Unit	Biological Reference interva	
	KIDNI	V FUNCTION T	EST (COMPLETE		
UREA: SERUM	<b>MD</b>	26.13	mg/dL	10.00 - 50.00	
	NATE DEHYDROGENASE (GLDH)	20.10	0	10.00 00.00	
CREATININE: SER		1.17	mg/dL	0.40 - 1.40	
by ENZYMATIC, SPEC	ROGEN (BUN): SERUM	12.21	mg/dL	7.0 - 25.0	
by CALCULATED, SPE	ECTROPHOTOMETRY				
	ROGEN (BUN)/CREATININE	10.44	RATIO	10.0 - 20.0	
RATIO: SERUM by CALCULATED, SPE	ECTROPHOTOMETRY				
UREA/CREATININ	E RATIO: SERUM	22.33	RATIO		
by CALCULATED, SPE URIC ACID: SERUM	ECTROPHOTOMETRY I	6.47	mg/dL	3.60 - 7.70	
by URICASE - OXIDAS		0.47	iiig/ uL	3.00 - 1.10	
CALCIUM: SERUM		9.17	mg/dL	8.50 - 10.60	
by ARSENAZO III, SPE PHOSPHOROUS: SI		3.03	mg/dL	2.30 - 4.70	
by PHOSPHOMOLYBL	DATE, SPECTROPHOTOMETRY	0.00	ing/ uL	2.00 1.10	
<u>ELECTROLYTES</u>					
SODIUM: SERUM by ISE (ION SELECTIV		140.2	mmol/L	135.0 - 150.0	
POTASSIUM: SERU		4.3	mmol/L	3.50 - 5.00	
by ISE (ION SELECTIV	/E ELECTRODE)				
CHLORIDE: SERUN by ISE (ION SELECTIV		105.15	mmol/L	90.0 - 110.0	
	IERULAR FILTERATION RATE				
	ERULAR FILTERATION RATE	70			
(eGFR): SERUM	mill				
by CALCULATED INTERPRETATION:					
	een pre- and post renal azotemia.				

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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AGE / GENDER       :63 YRS/MALE       PATIENT ID       :1697125         SOLLECTED BY       :SURJESH       REG. NO./LAB NO.       :012412120013         REFERED BY       :REGISTRATION DATE       :12/Dec/2024 09:55 AM         SARCODE NO.       :01522341       COLLECTION DATE       :12/Dec/2024 12:23PM         LIENT CODE       :KOS DIAGNOSTIC LAB       REPORTING DATE       :12/Dec/2024 12:23PM         SILENT CODE       :KOS DIAGNOSTIC LAB       REPORTING DATE       :12/Dec/2024 09:55 AM         SILENT ADDRESS       :6349/1, NICHOLSON ROAD, AMBALA CANTT       :12/Dec/2024 09:55 AM         SILENT CODE       Collectrion plus       :       :Scess protein intake.       :         1. Unife crabstorption (6.2, uretracultion production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, ururs, surgery, cachexia, high fever), .       :         2. Unife crabstorption (6.2, uretraculting, glucocorticoids)       :       :       :         NeckaSD RATIO (-20:1) WITH LEVATED CREATINNE LEVELS:       :       :       :         1. Postrenal azotemia Superimposed on renal disease.       :       :       :       :         2. Own protein diet and starvation.       :       :       :       :       :         3. Prognanz.       :       :       :       <		1	Dr. Vinay ChopraDr. Yugam ChopraMD (Pathology & Microbiology)MD (Pathology)Chairman & Consultant PathologistCEO & Consultant Pathologist				
COLLECTED BY       SURJESH       REG. NO./LAB NO.       SURJEST         REFERED BY       I. SURJESH       REGISTRATION DATE       I.2/Dec/2024 09:55 AM         SARCODE NO.       SUSJESSA       COLLECTION DATE       I.2/Dec/2024 12:23PM         SILENT CODE       KOS DIAGNOSTIC LAB       REPORTING DATE       I.2/Dec/2024 06:11PM         SILENT ADDRES       KGS DIAGNOSTIC LAB       REPORTING DATE       I.2/Dec/2024 06:11PM         SILENT ADDRES       KGS DIAGNOSTIC LAB       REPORTING DATE       I.2/Dec/2024 06:11PM         SILENT ADDRES       KGS DIAGNOSTIC LAB       REPORTING DATE       I.2/Dec/2024 06:11PM         SILENT ADDRES       KGS DIAGNOSTIC LAB       REPORTING DATE       I.2/Dec/2024 06:11PM         SILENT ADDRES       KGS DIAGNOSTIC LAB       REPORTING DATE       I.2/Dec/2024 06:11PM         SILENT ADDRES       KGS DIAGNOSTIC LAB       REPORTING DATE       I.2/Dec/2024 06:11PM         B. Medicad muscle mass (subnormal creatinine production)       P. Grand azotemia (SUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).       P. Freenal azotemia (SUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).       P. Freenal azotemia (SUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).       P. Freenal azotemia (gue a with reatinine offluxes out of extracellular fluid).       S. Otheretous of decrease durea synthesis.       P. Pere	NAME	: Mr. RAJINDI	ER KUMAR				
REFERED BY       I:       REGISTRATION DATE       I:2/Dec/2024 09:55 AM         SARCODE NO.       :01522341       COLLECTION DATE       I:2/Dec/2024 12:23PM         SLIENT CODE       : KOS DIAGNOSTIC LAB       REPORTING DATE       I:2/Dec/2024 06:11PM         SLIENT ADDRESS       : 6349/1, NICHOLSON ROAD, AMBALA CANTT       Biological Reference interva         1. High protein intake.       .       Impaired renal function plus       .         5. Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, Jurns, surgery, cachexia, high fever).       .       .         0. Urline reabsorption (e.g. urler colostomy)       .       .       .       .         1. Perstenal azotemia superimposed on renal disease.       .       .       .       .         2. Norte rauses of decreased urea synthesis.       .       .       .       .       .         3. Reported fide and starvation.       .       .       .       .       .       .       .       .       .         2. Severe liver disease.       .	AGE/ GENDER	: 63 YRS/MALI	3	PATIENT ID		: 1697125	
EFFERED BY       I:       REGISTRATION DATE       I: 12/Dec/2024 09:55 AM         SARCODE NO.       : 01522341       COLLECTION DATE       I: 12/Dec/2024 12:23PM         JILENT CODE       : KOS DIAGNOSTIC LAB       REPORTING DATE       I: 2/Dec/2024 06:11PM         JILENT ADDRESS       : 6349/1, NICHOLSON ROAD, AMBALA CANTE       IIII: 12/Dec/2024 06:11PM         Feed       Value       Unit       Biological Reference interval         1. High protein intake.       : mgaired renal function plus       : Scess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, Jurns, surgery, cachexia, high fever).       : Urine reabsorption (e.g. uret colostomy)         1. Heigh protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, Jurns, surgery, cachexia, high fever).       : Orine reabsorption (e.g. uret colostomy)         2. Certain drugs (e.g. tetracycline, glucecontricoids)       : Networption (e.g. uret colostomy)         3. Reposed dialysis (ure a ration ad disease.       : Persenal azotemia superimposed on renal disease.         Severe liver disease.       : Other causes of decreased urea synthesis.         3. Owptein diet and starvation.       : Severe liver disease.         4. Other tabsorphytis (refeases muscle creatinne).       : Shodh (syndrome of inappropiate antidiureic harmone) due to tubular secretion of urea.	COLLECTED BY	: SURJESH		REG. NO./LAB	NO.	: 012412120013	
ARCODE NO. :: 01522341 COLLECTION DATE :: 12/Dec/2024 12:23PM ZIENT CODE :: KOS DIAGNOSTIC LAB REPORTING DATE :: 12/Dec/2024 06:11PM ZIENT ADDRESS :: 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Via Biological Reference interva 1. High protein intake. 3. Impaired renal function plus 5. Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, 1. Urine reabsorption (e.g. ureter colostomy) 3. Reduced muscle mass (subnormal creatinine production) 4. Certain drugs (e.g. tertacycline, glucocorticoids) MCREASDE RATIO (2021) WITH LEVATED CREATINNE LEVELS: Prostneal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy). 2. Percenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy). 3. Percenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy). 4. Percenal azotemia superimposed on renal disease. 5. Severe liver disease 5. Other causes of decreased urea synthesis. 7. Percenal azotemia superimposed on renal disease. 5. Other causes of decreased urea synthesis. 7. Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid). 5. MADE (syndrome of inappropriate antidiuretic harmone) due to tubular secretion of urea. 5. Pergenarizonemia superimosing or creatine to creatinine). 7. Stabdomyolysis (releases muscle creatinine). 7. Rehadomyolysis (releases muscle creatinine). 7. Rehadomyolysis (releases muscle creatinine). 7. Obtober text and therapy (accelerates conversion of creatine to creatinine with certain methodologies, resulting in normal ratio when dehydric 7. Cophosporin therapy (Interferes with creatinine measurement). 7. Stabdomyolysis (releases muscle creatinine ratio). 7. Obtober text and interversion of creatinine measurement). 7. Stabdomyolysis (releases muscle creatinine ratio). 7. Cophosporin therapy (Interferes with creatinine measurement). 7. Stabdomyolysis							55 AM
LIENT CODE       : KOS DIAGNOSTICLAB       REPORTING DATE       : 12/Dec/2024 06:11PM         LIENT ADDRESS       : 6349/1, NICHOLSON ROAD, AMBALA CANTF         Fest Name       Value       Unit       Biological Reference interva         I. High protein intake.       impaired remai function plus       impaired remai function plus         F. scess protein intake or production or tissue breakdown (e.g. infection, Gl bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, urns, surgery, cachexla, high fove).         View reabsorption (e.g. ureter colostomy)       impaired remai function plus         Reduced muscle mass (Subnormal creating production)       impaired remains (e.g. tetracycline, glucocorticoids)         Vertain drugs (e.g. tetracycline, glucocorticoids)       impaired indices and superimposed on renal disease.         VECREASED RATIO (C-201) WITH ELEVATED CREATININE LEVELS:       impoirted indie and starvation.         . Severe liver disease.       0 ther causes of decreased urea synthesis.         . Repeated displays (urea rather than creatinine diffuses out of extracellular fluid).         . Inherited hyperammonemias (urea is virtually absent in blood).         . Stored BATH Or (-10) WITH INCREASED CREATINNE).         . Repeated displays (urea restation of creatine to creatinine).         . Repeated displays (urea restation of displays of the antidure the harmone) due to tubular secretion of urea.         . Prognamo.         ECR		.01522341					
Item ADDRES       : 6349/1, NICHOLSON ROAD, AMBALA CANTS         Image:       Value       Unit       Biological Reference interval         Image:			STIC I AD				
Test Name       Value       Unit       Biological Reference interval         1. High protein intake.       Impaired renal function plus       Impaired renal function plus         5. Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, jurns, surgery, cachexia, high fever).         1. Urine reabsorption (e.g. ureter colostomy)       Reduced muscle mass (subnormal creatinine production)         2. Certain drugs (e.g. tetracycline, glucocorticoids)       NorRASED RATIO (20:1) WITH ELEVATED CREATININE LEVELS:         1. Postnenal azotemia ligUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).       Percensia zotemia superimposed on renal disease.         2. Prerenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).       Percensia zotemia superimposed on renal disease.         2. Own protein field and starvation.       Severe liver disease.       Image: Superimposed on renal disease superimposed on renatinine offfuses out of extracellular fluid).         3. Inherited hyperammonemias (urea is virtually absent to blood).       Image: Superimposed area synthesis.         3. Repeared dialysis (urea rather than creatinine).       Percensize and superimposed on renatine to blood.         4. StADH (syndrome of inappropiate antidiuretic harmone) due to tubular secretion of urea.       Pregmancy.         EXCREASED RATIO (<10:1) WITH INCREASED CREATININE:					AIE	. 12/ Dec/ 2024 00.1	
A High protein intake.         9. High protein intake.         9. Impaired renal function plus         10. Excess protein intake or production or tissue breakdown (e.g. infection, Gl bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, urns, surgery, cachexia, high fever).         10. Urine reabsorption (e.g. ureter colostomy)         10. Reduced muscle mass (subnormal creatinine production)         2. Certain drugs (e.g. tetracycline, glucocorticoids)         NCREASED RATIO (<20:1) WITH ELEVATED CREATININE LEVELS:	LIEN I ADDKESS	: 6349/1, NICI	IULSUN RUAD, AMBA	ILA CANTT			
i. Impaired renal function plus i. Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, urns, surgery, cachexia, high fever). Urine reabsorption (e.g. ureter colostomy) i. Reduced muscle mass (subnormal creatinine production) Certain drugs (e.g. tetracycline, glucocorticoids) NCREASED RATIO (>20:1) WITH ELEVATED CREATININE LEVELS: Postrenal azotemia (BLW) rises disproportionately more than creatinine) (e.g. obstructive uropathy). Prerenal azotemia (BLW) rises disproportionately more than creatinine) (e.g. obstructive uropathy). Perenal azotemia (BLW) rises disproportionately more than creatinine) (e.g. obstructive uropathy). Perenal azotemia (BLW) rises disproportionately more than creatinine) (e.g. obstructive uropathy). Perenal azotemia (BLW) rises disproportionately more than creatinine) (e.g. obstructive uropathy). Perenal azotemia (BLW) rises disproportionately more than creatinine) (e.g. obstructive uropathy). Perenal azotemia (SLW) rises disproportionately more than creatinine) (e.g. obstructive uropathy). Other causes of decreased urea synthesis. I. Ouper causes of decreased urea synthesis. I. Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid). I. Inherited hyperanmonemias (urea is virtually absent in blood). SIADH (syndrome of inappropiate antidiuretic harmone) due to tubular secretion of urea. Pregnancy. Perenancy. Perenancy. Presence of Put INCREASED CREATININE: Phenacimide therapy (accelerates conversion of creatine to creatinine). NapPROPIATE RATIO: Diabetic ketoacidosis (acetoacetate causes false increase in creatinine with certain methodologies, resulting in normal ratio when dehydric hould produce an increased BUN/creatinine measurement). STIMATED GLOMERULAR FILTERATION RATE: CKD STAGE DESCRIPTION <u>GFR Albumin or cast in urine</u> G30 Mild decrease in GFR 60-89 G30 Moderate decrease in GFR 60-89 G30 Moderate decrease in GFR 15-29	ſest Name			Value	Unit	Biological	l Reference interval
G3aMild decrease in GFR60 - 89G3bModerate decrease in GFR30-59G4Severe decrease in GFR15-29			n renal disease.	nan creatinine) (e.g. obstruc	ctive uropa	ithy).	
G3bModerate decrease in GFR30-59G4Severe decrease in GFR15-29	DECREASED RATIO (< 1. Acute tubular necr 2. Low protein diet ar 3. Severe liver diseas 4. Other causes of de 5. Repeated dialysis ( 6. Inherited hyperam 7. SIADH (syndrome of 8. Pregnancy. DECREASED RATIO (< 1. Phenacimide thera 2. Rhabdomyolysis (r 8. Muscular patients NAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin thera 5. CKD STAGE G1	10:1) WITH DECRI rosis. nd starvation. se. ecreased urea syr (urea rather than nmonemias (urea of inappropiate a 10:1) WITH INCRE apy (accelerates of releases muscle of who develop rer D: osis (acetoacetate rapy (interferes v ULAR FILTERATION Norther Starter (Starter) Norther Starter)	n renal disease. <b>EASED BUN :</b> a creatinine diffuses o is virtually absent in ntidiuretic harmone) of <b>ASED CREATININE:</b> conversion of creatine reatinine). hal failure. e causes false increase eatinine ratio). vith creatinine measur <b>NATE:</b> <b>DESCRIPTION</b> mal kidney function	ut of extracellular fluid). blood). due to tubular secretion of t to creatinine). e in creatinine with certain rement). GFR (mL/min/1.73m2 >90	urea. methodolo	ogies,resulting in norma SOCIATED FINDINGS No proteinuria	al ratio when dehydratio
G4 Severe decrease in GFR 15-29	CKD STAGE CKD STAGE CKD STAGE CKD STAGE CKD STAGE CKD STAGE CKD STAGE CKD STAGE CKD STAGE CKD STAGE CACULANNESS	10:1) WITH DECRI rosis. nd starvation. se. ecreased urea syr (urea rather than nmonemias (urea of inappropiate a 10:1) WITH INCRE apy (accelerates of releases muscle of who develop rer Disis (acetoacetate rapy (interferes v ULAR FILTERATION Northing Kin Northing Kin Northin Northing Kin Northing Kin Nor	n renal disease. EASED BUN : The thesis. a creatinine diffuses of is virtually absent in a ntidiuretic harmone) of ASED CREATININE: conversion of creatine reatinine). hal failure. the causes false increase exatinine ratio). vith creatinine measure NATE: DESCRIPTION mal kidney function dney damage with prmal or high GFR	ut of extracellular fluid). blood). due to tubular secretion of t to creatinine). e in creatinine with certain rement). GFR (mL/min/1.73m2 >90	urea. methodolo	ogies,resulting in norma SOCIATED FINDINGS No proteinuria resence of Protein ,	al ratio when dehydratio
	Acute tubular necr Acute tubular necr Low protein diet ar Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin ther STIMATED GLOMERI G1 G2 G3a	10:1) WITH DECRI rosis. nd starvation. se. ecreased urea syr (urea rather than nmonemias (urea of inappropiate a 10:1) WITH INCRE apy (accelerates of releases muscle of who develop rer D: osis (acetoacetate rapy (interferes v ULAR FILTERATION ULAR FILTERATION Nor Nor	n renal disease. EASED BUN : The thesis. a creatinine diffuses of is virtually absent in a ntidiuretic harmone) of ASED CREATININE: conversion of creatine reatinine). hal failure. a causes false increase exatinine ratio). vith creatinine measure NATE: DESCRIPTION mal kidney function dney damage with ormal or high GFR Id decrease in GFR	ut of extracellular fluid). blood). due to tubular secretion of t to creatinine). e in creatinine with certain rement). GFR (mL/min/1.73m2 >90 >90 60 -89	urea. methodolo	ogies,resulting in norma SOCIATED FINDINGS No proteinuria resence of Protein ,	al ratio when dehydratio
G5 Kidney failure <15	Acute tubular necr Acute tubular necr Low protein diet ar Severe liver diseas Other causes of de Repeated dialysis ( Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin ther STIMATED GLOMERI G1 G2 G3a G3a G3b	10:1) WITH DECRI rosis. nd starvation. se. ecreased urea syr (urea rather than nonemias (urea of inappropiate a 10:1) WITH INCRE apy (accelerates of releases muscle of who develop rer D: osis (acetoacetate noreased BUN/cre rapy (interferes w ULAR FILTERATION ULAR FILTERATION Norm Kid norm Mode	n renal disease. <b>EASED BUN :</b> The thesis. The creatinine diffuses of is virtually absent in a ntidiuretic harmone) of <b>ASED CREATININE:</b> conversion of creatine reatinine). The causes false increase exatinine ratio). with creatinine measure <b>ARTE:</b> <b>DESCRIPTION</b> mal kidney function diney damage with ormal or high GFR id decrease in GFR erate decrease in GFR	ut of extracellular fluid). blood). due to tubular secretion of t to creatinine). e in creatinine with certain rement). GFR (mL/min/1.73m2 >90 >90 60 -89 30-59	urea. methodolo	ogies,resulting in norma SOCIATED FINDINGS No proteinuria resence of Protein ,	al ratio when dehydratio





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	Dr. Vinay Chopra MD (Pathology & Micro Chairman & Consultant	biology) MI	m Chopra D (Pathology) nt Pathologist
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Test Name		Value Unit	Biological Reference interval

COMMENTS:

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.
 eGFR calculated using the 2009 CKD-EPI creatinine equation and GFR category reported as per KDIGO guideline 2012
 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 of CFD with the commended to measure

3. In patients, with eGFR cleaning between 45-59 minimit 1.73 m2 (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

End Of Report \*\*\*





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