

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



	Dr. Vinay Chopra MD (Pathology & Micr Chairman & Consultar	obiology)	Dr. Yugam Cl MD (Pat CEO & Consultant Patl	hology)
NAME	: Mr. AMIT SHARMA			
AGE/ GENDER	: 50 YRS/MALE	PATI	ENT ID :	1773270
COLLECTED BY	:	REG.	NO./LAB NO. :	012502280005
REFERRED BY	:	REGI	STRATION DATE :	28/Feb/2025 08:57 AM
BARCODE NO.	: 01526232			28/Feb/2025 09:04AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		RTING DATE :	28/Feb/2025 10:21AM
LIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMB/	ALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	SWAST	THYA WELLN	ESS PANEL: G	
	COMP	PLETE BLOOD	COUNT (CBC)	
RED BLOOD CELL	S (RBCS) COUNT AND INDICES			
HAEMOGLOBIN (H	B)	14.5	gm/dL	12.0 - 17.0
by CALORIMETRIC	(RBC) COUNT	5.19 ^H	Millions/cm	m 3.50 - 5.00
by HYDRO DYNAMIC I	FOCUSING, ELECTRICAL IMPEDENCE			
ACKED CELL VOL	UME (PCV) AUTOMATED HEMATOLOGY ANALYZER	44.9	%	40.0 - 54.0
	AR VOLUME (MCV) AUTOMATED HEMATOLOGY ANALYZER	86.5	fL	80.0 - 100.0
IEAN CORPUSCUI	AR HAEMOGLOBIN (MCH)	28	pg	27.0 - 34.0
MEAN CORPUSCUI	AUTOMATED HEMATOLOGY ANALYZER AR HEMOGLOBIN CONC. (MCHC)	32.3	g/dL	32.0 - 36.0
RED CELL DISTRIB	AUTOMATED HEMATOLOGY ANALYZER SUTION WIDTH (RDW-CV)	14.4	%	11.00 - 16.00
•	AUTOMATED HEMATOLOGY ANALYZER BUTION WIDTH (RDW-SD)	47	fL	35.0 - 56.0
	AUTOMATED HEMATOLOGY ANALYZER			
				BETA THALASSEMIA TRAIT: <
MENTZERS INDEX		16.67	RATIO	
MENTZERS INDEX		10.07	RATIO	13.0 IRON DEFICIENCY ANEMIA:
MENTZERS INDEX by CALCULATED				13.0 IRON DEFICIENCY ANEMIA: >13.0
MENTZERS INDEX by CALCULATED		24.05	RATIO	13.0 IRON DEFICIENCY ANEMIA: >13.0 BETA THALASSEMIA TRAIT:< 65.0
MENTZERS INDEX by calculated				13.0 IRON DEFICIENCY ANEMIA: >13.0 BETA THALASSEMIA TRAIT:< 65.0 IRON DEFICIENCY ANEMIA: >
MENTZERS INDEX by CALCULATED GREEN & KING INI by CALCULATED	DEX			13.0 IRON DEFICIENCY ANEMIA: >13.0 BETA THALASSEMIA TRAIT:< 65.0
MENTZERS INDEX by calculated GREEN & KING INI by calculated WHITE BLOOD CE	DEX LLS (WBCS) E COUNT (TLC)			13.0 IRON DEFICIENCY ANEMIA: >13.0 BETA THALASSEMIA TRAIT:< 65.0 IRON DEFICIENCY ANEMIA: >
MENTZERS INDEX by calculated GREEN & KING INI by calculated MHITE BLOOD CE TOTAL LEUCOCYTI by FLOW CYTOMETR	DEX LLS (WBCS)	24.05	RATIO	13.0 IRON DEFICIENCY ANEMIA: >13.0 BETA THALASSEMIA TRAIT:< 65.0 IRON DEFICIENCY ANEMIA: > 65.0
MENTZERS INDEX by CALCULATED GREEN & KING INI by CALCULATED MHITE BLOOD CE COTAL LEUCOCYTI by FLOW CYTOMETR NUCLEATED RED I by AUTOMATED 6 PA	DEX ELLS (WBCS) E COUNT (TLC) y by sf cube & microscopy	24.05	RATIO	13.0 IRON DEFICIENCY ANEMIA: >13.0 BETA THALASSEMIA TRAIT:< 65.0 IRON DEFICIENCY ANEMIA: > 65.0 4000 - 11000





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

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Dr. Yugam Chopra

MD (Pathology & Microbiology) MD (Pathology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** NAME : Mr. AMIT SHARMA AGE/ GENDER : 50 YRS/MALE **PATIENT ID** :1773270 **COLLECTED BY** :012502280005 REG. NO./LAB NO. **REFERRED BY REGISTRATION DATE** : 28/Feb/2025 08:57 AM **BARCODE NO.** :01526232 **COLLECTION DATE** : 28/Feb/2025 09:04AM CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE** : 28/Feb/2025 10:21AM **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Unit **Biological Reference interval DIFFERENTIAL LEUCOCYTE COUNT (DLC)** NEUTROPHILS 58 % 50 - 70 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY LYMPHOCYTES 29 % 20 - 40 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY 7H EOSINOPHILS % 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 4159 2000 - 7500 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE LYMPHOCYTE COUNT 2079 800 - 4900 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE EOSINOPHIL COUNT 502^H /cmm 40 - 440 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE MONOCYTE COUNT 430 /cmm 80 - 880 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS. PLATELET COUNT (PLT) 150000 - 450000 183000 /cmm by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELETCRIT (PCT) % 0.10 - 0.36 0.25by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE MEAN PLATELET VOLUME (MPV) 14^H fL. 6.50 - 12.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET LARGE CELL COUNT (P-LCC) 30000 - 90000 95000^H /cmm by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE 51.7^H PLATELET LARGE CELL RATIO (P-LCR) % 11.0 - 45.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET DISTRIBUTION WIDTH (PDW) 16.4% 15.0 - 17.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

Dr. Vinay Chopra





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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interva
WHOLE BLOOD	EMOGLOBIN (HbA1c):	7.3 ^H	%	4.0 - 6.4
ESTIMATED AVERA	RMANCE LIQUID CHROMATOGRAPHY) GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY)	162.81 ^H	mg/dL	60.00 - 140.00
INTERPRETATION:				
INTERI RETATION.				
<u>INTERI RETATION.</u>	AS PER AMERICAN	DIABETES ASSOCIATION (
	REFERENCE GROUP		ATED HEMOGLOGIB	(HBAIC) in %
 Non dia	REFERENCE GROUP abetic Adults >= 18 years		ATED HEMOGLOGIB <5.7	(HBAIC) in %
Non dia A	REFERENCE GROUP abetic Adults >= 18 years t Risk (Prediabetes)		ATED HEMOGLOGIB <5.7 5.7 - 6.4	(HBAIC) in %
Non dia A	REFERENCE GROUP abetic Adults >= 18 years		ATED HEMOGLOGIB <5.7 5.7 - 6.4 >= 6.5	(HBAIC) in %
Non dia	REFERENCE GROUP abetic Adults >= 18 years t Risk (Prediabetes)	GLYCOSYI	ATED HEMOGLOGIB <5.7 5.7 - 6.4 >= 6.5 Age > 19 Years	
Non dia A D	REFERENCE GROUP abetic Adults >= 18 years t Risk (Prediabetes) iagnosing Diabetes	GLYCOSYI Goals of The	ATED HEMOGLOGIB <5.7 5.7 – 6.4 >= 6.5 Age > 19 Years "apy:	< 7.0
Non dia A D	REFERENCE GROUP abetic Adults >= 18 years t Risk (Prediabetes)	GLYCOSYI	ATED HEMOGLOGIB <5.7 5.7 – 6.4 >= 6.5 Age > 19 Years "apy:	

KOS Diagnostic Lab

(A Unit of KOS Healthcare)

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 appropiate.

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 faisely 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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est Name				
		Value	Unit	Biological Reference interval
C-reactive protein This test may also be us stemic lupus erythema DNDITION WITH LOW E low ESR can be seen w olycythaemia), signific sickle cells in sickle ce DTE: ESR and C - reactive pr Generally, ESR does not CRP is not affected by . If the ESR is elevated,	used to monitor disease activity atosus SR ith conditions that inhibit the ne antly high white blood cell cour ell anaemia) also lower the ESR rotein (C-RP) are both markers o ot change as rapidly as does CRF as many other factors as is ESR, it is typically a result of two typ a higher ESR, and menstruation a	and response to the ormal sedimentation it (leucocytosis) , an f inflammation. P, either at the start making it a better m es of proteins, globu and pregnancy can c	erapy in both of the a n of red blood cells, si id some protein abno of inflammation or as arker of inflammatior Jlins or fibrinogen. ause temporary eleva	n.





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Test Name		Value	Unit	Biological Reference interval
	CLIN	ICAL CHEMISTRY	BIOCHEMISTR	Y
		GLUCOSE FAST	TING (F)	
GLUCOSE FASTING	G (F): PLASMA E - PEROXIDASE (GOD-POD)	127.95 ^H	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0

KOS Diagnostic Lab (A Unit of KOS Healthcare)

A fasting plasma glucose level below 100 mg/dl is considered normal.
 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.
 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.



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CLIENT ADDRESS	: 6349/1, NICHOLSON ROA	D, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		LIPID PRO	FILE : BASIC	
CHOLESTEROL TO	TAL: SERUM	130.46	mg/dL	OPTIMAL: < 200.0
by CHOLESTEROL OX		100110	ing, uz	BORDERLINE HIGH: 200.0 -
				239.0 HIGH CHOLESTEROL: > OR =
				HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: S		134.9	mg/dL	OPTIMAL: < 150.0
by GLYCEROL PHOSP	HATE OXIDASE (ENZYMATIC)			BORDERLINE HIGH: 150.0 -
				199.0 HIGH: 200.0 - 499.0
				VERY HIGH: $> OR = 500.0$
HDL CHOLESTEROI by SELECTIVE INHIBIT	L (DIRECT): SERUM	67.16	mg/dL	LOW HDL: < 30.0
by SELECTIVE INHIBITI	ON			BORDERLINE HIGH HDL: 30.0 60.0
				HIGH HDL: $> OR = 60.0$
LDL CHOLESTEROI		36.32	mg/dL	OPTIMAL: < 100.0
by CALCULATED, SPE	CIROPHOTOMETRY			ABOVE OPTIMAL: 100.0 - 129.0 BORDERLINE HIGH: 130.0 -
				159.0
				HIGH: 160.0 - 189.0
NON HDL CHOLEST		63.3	mg/dL	VERY HIGH: > OR = 190.0 OPTIMAL: < 130.0
by CALCULATED, SPE		03.3	liig/ uL	ABOVE OPTIMAL: 130.0 - 159.0
				BORDERLINE HIGH: 160.0 -
				189.0 HIGH: 190.0 - 219.0
				VERY HIGH: $> OR = 220.0$
VLDL CHOLESTERC		26.98	mg/dL	0.00 - 45.00
by CALCULATED, SPE TOTAL LIPIDS: SER		205 00	ma /dl	350.00 700.00
by CALCULATED, SPE		395.82	mg/dL	350.00 - 700.00
CHOLESTEROL/HD		1.94	RATIO	LOW RISK: 3.30 - 4.40
by CALCULATED, SPE	CIROPHUIOMEIRY			AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0
				$\mathbf{WODEWATEWOK, 1.10 - 11.0}$

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Test Name		Value	Unit	Biological Reference interval
LDL/HDL RATIO: S by CALCULATED, SPE		0.54	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	2.01 ^L	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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BILIRUBIN DIRECT	PECTROPHOTOMETRY Γ (CONJUGATED): SERUM SPECTROPHOTOMETRY	0.56 0.15	mg/dL mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20 0.00 - 0.40
BILIRUBIN INDIRE by CALCULATED, SPE	CCT (UNCONJUGATED): SERUM	0.41	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PY	[/RIDOXAL PHOSPHATE	25.65	U/L	7.00 - 45.00
SGPT/ALT: SERUM	[/RIDOXAL PHOSPHATE	36.64	U/L	0.00 - 49.00
AST/ALT RATIO: S		0.7	RATIO	0.00 - 46.00
ALKALINE PHOSPI by Para Nitrophen propanol	HATASE: SERUM IYL PHOSPHATASE BY AMINO METHYL	67.71	U/L	40.0 - 130.0
GAMMA GLUTAMY by SZASZ, SPECTRON	L TRANSFERASE (GGT): SERUM PHTOMETRY	44.7	U/L	0.00 - 55.0
TOTAL PROTEINS: by BIURET, SPECTRO	SERUM	6.82	gm/dL	6.20 - 8.00
ALBUMIN: SERUM		4.28	gm/dL	3.50 - 5.50
GLOBULIN: SERUN by CALCULATED, SPE	1	2.54	gm/dL	2.30 - 3.50
A : G RATIO: SERUI	M	1.69	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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INTERPRETATION





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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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	Dr. Vinay Cho MD (Pathology & N Chairman & Consu	1icrobiology)		(Pathology)
NAME	: Mr. AMIT SHARMA			
AGE/ GENDER	: 50 YRS/MALE		PATIENT ID	: 1773270
COLLECTED BY	:		REG. NO./LAB NO.	: 012502280005
REFERRED BY	:		REGISTRATION DATE	: 28/Feb/2025 08:57 AM
BARCODE NO.	:01526232		COLLECTION DATE	: 28/Feb/2025 09:04AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 28/Feb/2025 11:11AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, Al	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interva
	KIDNI	EY FUNCTIO	N TEST (COMPLETE)	
UREA: SERUM	IATE DEHYDROGENASE (GLDH)	24.53	mg/dL	10.00 - 50.00
CREATININE: SERI	UM	0.96	mg/dL	0.40 - 1.40
	ROGEN (BUN): SERUM	11.46	mg/dL	7.0 - 25.0
RATIO: SERUM	ROGEN (BUN)/CREATININE	11.94	RATIO	10.0 - 20.0
UREA/CREATININ		25.55	RATIO	
URIC ACID: SERUM by URICASE - OXIDAS		4.89	mg/dL	3.60 - 7.70
CALCIUM: SERUM by ARSENAZO III, SPE	ECTROPHOTOMETRY	8.62	mg/dL	8.50 - 10.60
-	ERUM DATE, SPECTROPHOTOMETRY	3.1	mg/dL	2.30 - 4.70
ELECTROLYTES		149 5		125.0 150.0
SODIUM: SERUM by ISE (ION SELECTIV	/E ELECTRODE)	142.5	mmol/L	135.0 - 150.0
POTASSIUM: SERU by ISE (ION SELECTIV	Μ	4.33	mmol/L	3.50 - 5.00
CHLORIDE: SERUN by ISE (ION SELECTIV	1	106.88	mmol/L	90.0 - 110.0
ESTIMATED GLON	MERULAR FILTERATION RATE			
ESTIMATED GLOM (eGFR): SERUM by CALCULATED INTERPRETATION:	IERULAR FILTERATION RATE	96.3		
	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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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT





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CLIENT ADDRESS	: 6349/1, NI	CHOLSON ROAD, AMB	ALA CANTT					
Test Name			Value	Un	nit	Biolog	gical Referenc	e interval
burns, surgery, cache 7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (<1 1. Acute tubular necr	ke or producti kia, high fever (e.g. ureter co ass (subnorma tetracycline, g D:1) WITH ELE (BUN rises di superimposed 0:1) WITH DEC osis.	lostomy) Il creatinine productior lucocorticoids) /ATED CREATININE LEVE sproportionately more t on renal disease.) LS:				drome, high pro	tein diet,
5. Excess protein inta burns, surgery, cache 7. Urine reabsorption 3. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia DECREASED RATIO (<1 1. Acute tubular necr 2. Low protein diet ar 3. Severe liver disease 4. Other causes of de 5. Repeated dialysis (6. Inherited hyperam 7. SIADH (syndrome c 3. Pregnancy. DECREASED RATIO (<1 1. Phenacimide thera 2. Rhabdomyolysis (ro 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERU CKD STAGE G1 G2 G3a	ke or producti kia, high fever (e.g. ureter co ass (subnorma tetracycline, g D:1) WITH ELE (BUN rises di- superimposed 0:1) WITH DEC osis. d starvation. creased ureas urea rather th nonemias (ur f inappropiate 0:1) WITH INC oy (accelerate eleases muscl- who develop i sis (acetoacet creased BUN/ apy (interfere LAR FILTERATI). Il creatinine production Il creatinine production Il creatinine production Il creatinine production /ATED CREATININE LEVE sproportionately more to on renal disease. REASED BUN : ynthesis. an creatinine diffuses of the areatinine diffuse) LS: han creatinin blood). due to tubul to creatinin e in creatinin rement).	ne) (e.g. obstructive ellular fluid). ar secretion of urea ne). he with certain met <u>hL/min/1.73m2) >90 >90 60 -89</u>	e uropath a. thodologi	y).	ormal ratio whe	
5. Excess protein inta burns, surgery, cache 7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia DECREASED RATIO (<1 1. Acute tubular necr 2. Low protein diet ar 3. Severe liver disease 4. Other causes of de 5. Repeated dialysis (6. Inherited hyperam 7. SIADH (syndrome c 3. Pregnancy. DECREASED RATIO (<1 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERL CKD STAGE G1 G2	ke or producti kia, high fever (e.g. ureter co ass (subnorma tetracycline, g D:1) WITH ELE (BUN rises di- superimposed 0:1) WITH DEC osis. d starvation. creased ureas urea rather th nonemias (ur f inappropiate 0:1) WITH INC oy (accelerate eleases muscl- who develop n sis (acetoacet creased BUN/ apy (interfere LAR FILTERATI). Ilostomy) Il creatinine production Iucocorticoids) /ATED CREATININE LEVE sproportionately more to on renal disease. REASED BUN : ynthesis. an creatinine diffuses of the asses of the second and creatinine diffuses of the asses of the second e creatinine diffuses of the antidiuretic harmone) REASED CREATININE: s conversion of creatine the causes false increases creatinine ratio). s with creatinine measu ON RATE: DESCRIPTION tormal kidney function Kidney damage with normal or high GFR) LS: han creatinin blood). due to tubul to creatinin e in creatinin rement).	ne) (e.g. obstructive ellular fluid). ar secretion of urea ne). he with certain met <u>hL/min/1.73m2) >90 >90</u>	e uropath a. thodologi	y). es,resulting in no CIATED FINDING lo proteinuria sence of Protein ,	ormal ratio whe	





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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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