



	Dr. Vinay Chop MD (Pathology & Mi Chairman & Consult	crobiology)		Pathology)
NAME	: Mr. RITESH NARANG			
AGE/ GENDER	: 50 YRS/MALE		PATIENT ID	: 1616865
COLLECTED BY	:		REG. NO./LAB NO.	: 012409180009
<b>REFERRED BY</b>	:		REGISTRATION DATE	: 18/Sep/2024 07:27 AM
BARCODE NO.	:01517168		COLLECTION DATE	: 18/Sep/2024 07:30AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 18/Sep/2024 09:03AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	BALA CANT I		
Test Name		Value	Unit	Biological Reference interval
	SWA	STHYA WE	ELLNESS PANEL: G	
	CO	MPLETE BLC	DOD COUNT (CBC)	
RED BLOOD CELLS (R	BCS) COUNT AND INDICES			
HAEMOGLOBIN (HB) by calorimetric		13.4	gm/dL	12.0 - 17.0
RED BLOOD CELL (RB	C) COUNT OCUSING, ELECTRICAL IMPEDENCE	4.96	Millions/cm	am 3.50 - 5.00
PACKED CELL VOLUM		42.7	%	40.0 - 54.0
MEAN CORPUSCULA		85.9	fL	80.0 - 100.0
MEAN CORPUSCULA	R HAEMOGLOBIN (MCH)	27.1	pg	27.0 - 34.0
MEAN CORPUSCULA	R HEMOGLOBIN CONC. (MCHC) UTOMATED HEMATOLOGY ANALYZER	31.5 <sup>L</sup>	g/dL	32.0 - 36.0
RED CELL DISTRIBUT	ION WIDTH (RDW-CV) UTOMATED HEMATOLOGY ANALYZER	14.8	%	11.00 - 16.00
RED CELL DISTRIBUT	ION WIDTH (RDW-SD) UTOMATED HEMATOLOGY ANALYZER	47.7	fL	35.0 - 56.0
MENTZERS INDEX		17.32	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDE	X	25.71	RATIO	BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CELLS	<u>(WBCS)</u>			
	OUNT (TLC) ' by sf cube & microscopy	6680	/cmm	4000 - 11000
NUCLEATED RED BLC		NIL		0.00 - 20.00
NUCLEATED RED BLC	OD CELLS (nRBCS) % UTOMATED HEMATOLOGY ANALYZER	NIL	%	< 10 %
NEUTROPHILS by flow cytometry	Y BY SF CUBE & MICROSCOPY	49 <sup>L</sup>	%	50 - 70

KOS Central Lab: 6349/1, Nicholson Road, Ambala Cantt -133 001, Haryana

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

Page 1 of 12





Dr. Vinay Chop MD (Pathology & M Chairman & Consul		crobiology)	Chopra (Pathology) Pathologist	
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Test Name		Value	Unit	Biological Reference interval
LYMPHOCYTES		43 <sup>H</sup>	%	20 - 40
EOSINOPHILS	RY BY SF CUBE & MICROSCOPY	4	%	1 - 6
by FLOW CYTOMETR	Y BY SF CUBE & MICROSCOPY			
MONOCYTES		4	%	2 - 12
BASOPHILS	Y BY SF CUBE & MICROSCOPY	0	%	0 - 1
	Y BY SF CUBE & MICROSCOPY	Ŭ	,,,	
ABSOLUTE LEUKOCY	<u>YTES (WBC) COUNT</u>			
ABSOLUTE NEUTRO		3273	/cmm	2000 - 7500
	Y BY SF CUBE & MICROSCOPY	0070		000 1000
ABSOLUTE LYMPHO	CYTE COUNT Y BY SF CUBE & MICROSCOPY	2872	/cmm	800 - 4900
ABSOLUTE EOSINOP		267	/cmm	40 - 440
	Y BY SF CUBE & MICROSCOPY			
ABSOLUTE MONOCY	TE COUNT Y BY SF CUBE & MICROSCOPY	267	/cmm	80 - 880
ABSOLUTE BASOPHI		0	/cmm	0 - 110
by FLOW CYTOMETR	Y BY SF CUBE & MICROSCOPY			
PLATELETS AND OT	HER PLATELET PREDICTIVE MARKEI	<u>RS.</u>		
PLATELET COUNT (P		190000	/cmm	150000 - 450000
by HYDRO DYNAMIC I PLATELETCRIT (PCT)	FOCUSING, ELECTRICAL IMPEDENCE	0.26	%	0.10 - 0.36
	FOCUSING, ELECTRICAL IMPEDENCE	0.20	/0	0.10 - 0.30
MEAN PLATELET VO	LUME (MPV)	14 <sup>H</sup>	fL	6.50 - 12.0
PLATELET LARGE CE	FOCUSING, ELECTRICAL IMPEDENCE	96000 <sup>H</sup>	/cmm	30000 - 90000
by HYDRO DYNAMIC	FOCUSING, ELECTRICAL IMPEDENCE			
PLATELET LARGE CE	LL RATIO (P-LCR) FOCUSING, ELECTRICAL IMPEDENCE	50.4 <sup>H</sup>	%	11.0 - 45.0
PLATELET DISTRIBU		16.7	%	15.0 - 17.0
by HYDRO DYNAMIC I	FOCUSING, ELECTRICAL IMPEDENCE			
NOTE: TEST CONDU	ICTED ON EDTA WHOLE BLOOD			

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD



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BARCODE NO.	: 01517168		LLECTION DATE	: 18/Sep/2024 07:30AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	RE	PORTING DATE	: 18/Sep/2024 01:43PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
GLYCOSYLATED HAEI WHOLE BLOOD	MOGLOBIN (HbA1c):	5.4	%	4.0 - 6.4
ESTIMATED AVERAG		108.28	mg/dL	60.00 - 140.00
	AS PER AMERICAN	DIABETES ASSOCIATIO		
	REFERENCE GROUP	GLYCC	GLYCOSYLATED HEMOGLOGIB (HBAIC) in %	
	abetic Adults >= 18 years	/	<5.7	
	t Risk (Prediabetes)		5.7 - 6.4	
D	liagnosing Diabetes		>= 6.5	
		Goals of T	Age > 19 Years	< 7.0
Therapeutic goals for glycemic control				>8.0
Therapeut	ic goals for glycemic control	Actions Su	Age < 19 Years	20.0

**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 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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TEST PERFORMED AT KOS DIAGNOSTIC LAB. AMBALA CANTT





	Dr. Vinay Cho MD (Pathology & N Chairman & Consu	Microbiology)	Dr. Yugam C MD (Pa CEO & Consultant Pa	athology)
NAME	: Mr. RITESH NARANG			
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CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPOR	TING DATE	: 18/Sep/2024 09:18AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, Al	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	FRYTHR	ROCYTE SEDIMENT	ATION RATE (ESR)	
by RED CELL AGGREG INTERPRETATION: 1. ESR is a non-specified immune disease, but 2. An ESR can be affe as C-reactive protein 3. This test may also systemic lupus erythe CONDITION WITH LOW A low ESR can be see (polycythaemia), sigras sickle cells in sickling NOTE: 1. ESR and C - reactive 2. Generally, ESR doe 3. CRP is not affected 4. If the ESR is elevate 5. Women tend to ha 6. Drugs such as dext	does not tell the health practition cted by other conditions besides in be used to monitor disease activity ematosus <b>N ESR</b> n with conditions that inhibit the r ificantly high white blood cell cou e cell anaemia) also lower the ESF e protein (C-RP) are both markers of s not change as rapidly as does CR <b>by as many other factors as is ESR</b> , ed, it is typically a result of two typ ve a higher ESR, and menstruation	often indicates the pres er exactly where the inf nflammation. For this re y and response to thera normal sedimentation c int (leucocytosis), and R. of inflammation. RP, either at the start of <b>, making it a better mar</b> pes of proteins, globulir and pregnancy can cau	flammation is in the be eason, the ESR is typica apy in both of the above of red blood cells, such some protein abnorm inflammation or as it ker of inflammation. is or fibrinogen. se temporary elevatio	ally used in conjunction with other test such ve diseases as well as some others, such as n as a high red blood cell count alities. Some changes in red cell shape (such resolves.





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Page 4 of 12





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Test Name		Value	Unit	Biological Reference interval
	CLIN	ICAL CHEMISTRY	/BIOCHEMISTR	Y
			STING (F)	
		GLUCOSE FAS		

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.
 A fasting plasma glucose level in excess of 125 mg/dl on both occasions is confirmatory for diabetic state.



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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT





SU 9001:2008 CERT	IFIED LAB		EXCELLENCE IN HEALTHCARE	
		c <b>hopra</b> / & Microbiology) onsultant Pathologist	Dr. Yugam MD CEO & Consultant	(Pathology)
NAME	: Mr. RITESH NARANG			
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROA	D, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		LIPID PROFI	LE : BASIC	
CHOLESTEROL TOTA by CHOLESTEROL OX		126.65	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.
TRIGLYCERIDES: SER by GLYCEROL PHOSE	RUM PHATE OXIDASE (ENZYMATIC)	105.15	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROL ( by SELECTIVE INHIBIT		49.97	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: 5 by CALCULATED, SPE		55.65	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 CHOLESTE by calculated, spe		76.68	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189. HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTEROL		21.03	mg/dL	0.00 - 45.00
by CALCULATED, SPE TOTAL LIPIDS: SERU by CALCULATED, SPE	M	358.45	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL by CALCULATED, SPE	RATIO: SERUM	2.53	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: SER by CALCULATED, SPE		1.11	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
สตอรมดอาส		0		

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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

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Page 6 of 12





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Test Name		Value	Unit	Biological Reference interval
TRIGLYCERIDES/HD		2.1 <sup>L</sup>	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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Unit

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Value

Dr. Vinay Chopra MD (Pathology & Microbiolog Chairman & Consultant Patho

LIV	ER FUNCTION TES	T (COMPLETE)	
BILIRUBIN TOTAL: SERUM by diazotization, spectrophotometry	0.78	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.21	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.57	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	16.54	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	23.62	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by calculated, spectrophotometry	0.7	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by Para Nitrophenyl phosphatase by amino methyl propanol	124.22	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by szasz, spectrophtometry	14.5	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	6.75	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	3.61	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	3.14	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.15	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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**Biological Reference interval** 

Test Name

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Test Name		Value	Unit	Biological Ref	ference interval
HEPATOCELLULAR C	ARCINOMA & CHRONIC HEPATITIS		> 1.3 (Slightly Inc	reased)	

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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Test Name		Value	Unit	Biological Reference interval	
	ки	ONEY FUNCTIO	N TEST (COMPLETE)		
UREA: SERUM		16.44	mg/dL	10.00 - 50.00	
	ATE DEHYDROGENASE (GLDH)		J		
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETERY BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY		0.97	mg/dL	0.40 - 1.40	
		7.68 n	ma/dl	7.0 - 25.0	
			mg/dL	7:0 - 23:0	
BLOOD UREA NITROGEN (BUN)/CREATININE		7.92 <sup>L</sup>	RATIO	10.0 - 20.0	
RATIO: SERUM					
by CALCULATED, SPE UREA/CREATININE R		16.95	RATIO		
by CALCULATED, SPE		10.95	KATIO		
URIC ACID: SERUM		5.45	mg/dL	3.60 - 7.70	
by URICASE - OXIDAS	E PEROXIDASE				
CALCIUM: SERUM		8.98	mg/dL	8.50 - 10.60	
by ARSENAZO III, SPE PHOSPHOROUS: SER		3.25	mg/dL	2.30 - 4.70	
	ATE, SPECTROPHOTOMETRY	5.25	Thy/de	2.30 - 4.70	
ELECTROLYTES					
Sodium: Serum		140.5	mmol/L	135.0 - 150.0	
by ISE (ION SELECTIV					
POTASSIUM: SERUM		4.11	mmol/L	3.50 - 5.00	
by ISE (ION SELECTIV CHLORIDE: SERUM	E ELECTRODE)	105.38	mmol/L	90.0 - 110.0	
by ISE (ION SELECTIV	E ELECTRODE)	100.30	TTITIOI/L	90.0 - 110.0	
	RULAR FILTERATION RATE				
ESTIMATED GLOMFI	RULAR FILTERATION RATE	95.1			
(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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	MD (Path	ay Chopra nology & Microbiology) n & Consultant Patholog	Ň	am Chopra 1D (Pathology) ant Pathologist	
NAME	: Mr. RITESH NARAN	G			
AGE/ GENDER	: 50 YRS/MALE		PATIENT ID	: 1616865	
COLLECTED BY			REG. NO./LAB NO.	: 012409180009	
	•				
REFERRED BY	:		REGISTRATION DATE	1	
BARCODE NO.	:01517168		COLLECTION DATE	: 18/Sep/2024 07:30	DAM
CLIENT CODE.	: KOS DIAGNOSTIC LAI	В	REPORTING DATE	: 18/Sep/2024 11:12	2AM
CLIENT ADDRESS	: 6349/1, NICHOLSON	ROAD, AMBALA CANT	Т		
Test Name		Value	Unit	Biological	Reference interval
<ol> <li>Low protein diet an</li> <li>Severe liver diseas</li> </ol>	е.				
<ol> <li>Severe liver diseas</li> <li>Other causes of de</li> <li>Repeated dialysis</li> <li>Inherited hyperam</li> <li>SIADH (syndrome of</li> <li>Pregnancy.</li> <li>DECREASED RATIO (</li> <li>1. Phenacimide thera</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>INAPPROPIATE RATIO</li> <li>Diabetic ketoacido</li> </ol>	e. creased urea synthesis. (urea rather than creatini monemias (urea is virtua of inappropiate antidiuret <b>10:1) WITH INCREASED CR</b> (py (accelerates conversional eleases muscle creatininal who develop renal failuret sis (acetoacetate causes)	Illy absent in blood). tic harmone) due to tul EATININE: on of creatine to creati e). e. false increase in creat	oular secretion of urea. nine).	ologies,resulting in norma	I ratio when dehydratio
<ol> <li>Severe liver diseas</li> <li>Other causes of de</li> <li>Repeated dialysis</li> <li>Inherited hyperam</li> <li>SIADH (syndrome of</li> <li>Pregnancy.</li> <li>DECREASED RATIO (&lt;</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>INAPPROPIATE RATIO</li> <li>Diabetic ketoacido</li> <li>should produce an in</li> <li>Cephalosporin the</li> </ol>	e. creased urea synthesis. (urea rather than creatini monemias (urea is virtua of inappropiate antidiuret <b>10:1) WITH INCREASED CR</b> upy (accelerates conversional eleases muscle creatining who develop renal failuret sis (acetoacetate causes creased BUN/creatining to rapy (interferes with creating)	Illy absent in blood). tic harmone) due to tul (EATININE: on of creatine to creati e). e. false increase in creat ratio).	oular secretion of urea. nine).	ologies,resulting in norma	l ratio when dehydratio
<ol> <li>Severe liver diseas</li> <li>Other causes of de</li> <li>Repeated dialysis</li> <li>Inherited hyperam</li> <li>SIADH (syndrome of</li> <li>Pregnancy.</li> <li>DECREASED RATIO (</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>INAPPROPIATE RATIO</li> <li>Diabetic ketoacido</li> <li>should produce an in</li> <li>Cephalosporin the</li> <li>ESTIMATED GLOMERI</li> <li>CKD STAGE</li> </ol>	e. creased urea synthesis. (urea rather than creatini monemias (urea is virtua of inappropiate antidiuret <b>10:1) WITH INCREASED CR</b> upy (accelerates conversional eleases muscle creatinine who develop renal failuret who develop renal failuret isis (acetoacetate causes creased BUN/creatininet rapy (interferes with creat JLAR FILTERATION RATE: DESCRII	Illy absent in blood). tic harmone) due to tul EATININE: on of creatine to creati e). e. false increase in creat ratio). tinine measurement). PTION GFR	oular secretion of urea. nine). inine with certain method (mL/min/1.73m2)	ASSOCIATED FINDINGS	I ratio when dehydratio
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<ol> <li>Severe liver diseas</li> <li>Other causes of de</li> <li>Repeated dialysis</li> <li>Inherited hyperam</li> <li>SIADH (syndrome of</li> <li>Pregnancy.</li> <li>DECREASED RATIO (</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>INAPPROPIATE RATIO</li> <li>Diabetic ketoacido</li> <li>should produce an in</li> <li>Cephalosporin the</li> <li>ESTIMATED GLOMERI</li> <li>G1</li> <li>G2</li> </ol>	e. creased urea synthesis. (urea rather than creatini monemias (urea is virtua of inappropiate antidiuret <b>10:1) WITH INCREASED CR</b> upy (accelerates conversic eleases muscle creatinine who develop renal failuret who develop renal failuret isis (acetoacetate causes creased BUN/creatinine to creased BUN/creased BUN/creatinine to creased BUN/creased BUN/creased BUN/creatinine to creased BUN/creased BUN/crease	Illy absent in blood). It harmone) due to tul EATININE: on of creatine to creati e). e. false increase in creati ratio). tinine measurement). PTION GFR nage with high GFR	oular secretion of urea. nine). inine with certain method (mL/min/1.73m2) >90 >90	ASSOCIATED FINDINGS No proteinuria	l ratio when dehydratio
<ol> <li>Severe liver diseas</li> <li>Other causes of de</li> <li>Repeated dialysis</li> <li>Inherited hyperam</li> <li>SIADH (syndrome of</li> <li>Pregnancy.</li> <li>DECREASED RATIO (</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>MAPPROPIATE RATIO</li> <li>Diabetic ketoacido</li> <li>Should produce an in</li> <li>Cephalosporin their</li> <li>CESTIMATED GLOMERI</li> <li>CKD STAGE</li> <li>G1</li> </ol>	e. creased urea synthesis. (urea rather than creatini monemias (urea is virtua of inappropiate antidiuret <b>10:1) WITH INCREASED CR</b> upy (accelerates conversic eleases muscle creatinine who develop renal failur who develop renal failur who develop renal failur sis (acetoacetate causes creased BUN/creatinine is creased BUN/creased BUN/creatinine is creased BUN/creased BUN/cre	Illy absent in blood). It harmone) due to tul EATININE: on of creatine to creati e). e. false increase in creat ratio). tinine measurement). PTION GFR nage with high GFR ase in GFR	oular secretion of urea. nine). inine with certain method (mL/min/1.73m2) >90 >90	ASSOCIATED FINDINGS No proteinuria Presence of Protein ,	l ratio when dehydrati



G4 G5

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Severe decrease in GFR

Kidney failure

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

15-29

<15









	Dr. Vinay Chopra MD (Pathology & Microt Chairman & Consultant	piology) MI	m <b>Chopra</b> D (Pathology) ht Pathologist
NAME	: Mr. RITESH NARANG		
AGE/ GENDER	: 50 YRS/MALE	PATIENT ID	: 1616865
COLLECTED BY	:	<b>REG. NO./LAB NO.</b>	: 012409180009
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 18/Sep/2024 07:27 AM
BARCODE NO.	:01517168	COLLECTION DATE	: 18/Sep/2024 07:30AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	<b>REPORTING DATE</b>	: 18/Sep/2024 11:12AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBAL	A CANTT	
Test Name	V	/alue 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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DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY)

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