

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT



	<b>Dr. Vinay Chopra</b> MD (Pathology & Micr Chairman & Consultan	obiology)		) (Pathology)	
NAME : MI	r. <b>RAJ KIRA</b> N				
AGE/ GENDER : 21	YRS/MALE		PATIENT ID	: 175080	)5
<b>COLLECTED BY</b> :			REG. NO./LAB NO.	:01250	02090022
<b>REFERRED BY</b> :			<b>REGISTRATION DATE</b>		o/2025 10:50 AM
	525201		COLLECTION DATE		o/2025 10:59AM
	OS DIAGNOSTIC LAB		REPORTING DATE	:09/Feb	o/2025 11:28AM
CLIENT ADDRESS . 03	49/1, NICHOLSON ROAD, AMBA	ALA CANTI			
Test Name		Value	Unit		Biological Reference interval
	SWASTI	HYA WE	ELLNESS PANEL: 1.	0	
	СОМР	LETE BL	OOD COUNT (CBC)		
RED BLOOD CELLS (RB	<u>CS) COUNT AND INDICES</u>				
HAEMOGLOBIN (HB)		14.9	gm/dL		12.0 - 17.0
by CALORIMETRIC RED BLOOD CELL (RBC)	COUNT	5.46 <sup>H</sup>	Millions	/cmm	3.50 - 5.00
by HYDRO DYNAMIC FOCUSI	NG, ELECTRICAL IMPEDENCE				
PACKED CELL VOLUME ( by CALCULATED BY AUTOM.	(PCV) ATED HEMATOLOGY ANALYZER	45.2	%		40.0 - 54.0
MEAN CORPUSCULAR VO	DLUME (MCV) ated hematology analyzer	82.7	fL		80.0 - 100.0
MEAN CORPUSCULAR H		27.3	pg		27.0 - 34.0
MEAN CORPUSCULAR H	EMOGLOBIN CONC. (MCHC) ATED HEMATOLOGY ANALYZER	33.1	g/dL		32.0 - 36.0
RED CELL DISTRIBUTIO		15.1	%		11.00 - 16.00
RED CELL DISTRIBUTIO		47.1	fL		35.0 - 56.0
MENTZERS INDEX		15.15	RATIO		BETA THALASSEMIA TRAIT: <
by CALCULATED					13.0 IRON DEFICIENCY ANEMIA:
					>13.0
GREEN & KING INDEX by CALCULATED		22.88	RATIO		BETA THALASSEMIA TRAIT:<= 65.0
					IRON DEFICIENCY ANEMIA: >
WHITE BLOOD CELLS (	WR(S)				65.0
TOTAL LEUCOCYTE COU		8010	/cmm		4000 - 11000
by FLOW CYTOMETRY BY SF	CUBE & MICROSCOPY				
NUCLEATED RED BLOOI by AUTOMATED 6 PART HEM		NIL			0.00 - 20.00
NUCLEATED RED BLOOI		NIL	%		< 10 %





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

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

KOS Central Lab: 6349/1, Nicholson Road, Ambala Cantt -133 001, Haryana KOS Molecular Lab: IInd Floor, Parry Hotel, Staff Road, Opp. GPO, Ambala Cantt - 133 001, Haryana 0171-2643898, +91 99910 43898 | care@koshealthcare.com | www.koshealthcare.com







EXCELLENCE IN HEALTHCARE & DIAGNOSTICS

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

-- .

NAME	: Mr. RAJ KIRAN		
AGE/ GENDER	: 21 YRS/MALE	PATIENT ID	: 1750805
COLLECTED BY	:	REG. NO./LAB NO.	: 012502090022
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 09/Feb/2025 10:50 AM
BARCODE NO.	: 01525201	<b>COLLECTION DATE</b>	: 09/Feb/2025 10:59AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	<b>REPORTING DATE</b>	: 09/Feb/2025 11:28AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

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Test Name	Value	Unit	<b>Biological Reference interval</b>
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	63	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	26	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	9	%	2 - 12
BASOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	%	0 - 1
ABSOLUTE LEUKOCYTES (WBC) COUNT			
ABSOLUTE NEUTROPHIL COUNT by flow cytometry by sf cube & microscopy	5046	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by flow cytometry by sf cube & microscopy	2083	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	160	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by flow cytometry by sf cube & microscopy	721	/cmm	80 - 880
PLATELETS AND OTHER PLATELET PREDICTIVE	MARKERS.		
PLATELET COUNT (PLT) by hydro dynamic focusing, electrical impedence	213000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.26	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by hydro dynamic focusing, electrical impedence	12 <sup>H</sup>	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	90000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	42.3	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by hydro dynamic focusing, electrical impedence NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD	16.2	%	15.0 - 17.0



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CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 09/Feb/2025 11:45AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	IBALA CANTT	
Test Name		Value Unit	Biological Reference interval
	EDVTUDA	OVTE CEDIMENTATION DATE	
DVTHDOOVTE CEI	DIMENTATION RATE (ESR)	25 <sup>H</sup> mm/1	
<ol> <li>An ESR can be affe as C-reactive protein</li> <li>This test may also systemic lupus erythic CONDITION WITH LO' A low ESR can be see (polycythaemia), sigr as sickle cells in sickl NOTE:</li> <li>ESR and C - reactive</li> <li>Generally, ESR doe</li> <li>CRP is not affected</li> <li>If the ESR is elevat</li> <li>Women tend to ha</li> <li>Drugs such as dext</li> </ol>	cted by other conditions besides in be used to monitor disease activity ematosus <b>W ESR</b> n with conditions that inhibit the n- hificantly high white blood cell cour e cell anaemia) also lower the ESR e protein (C-RP) are both markers o es not change as rapidly as does CRF <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 a	and response to therapy in both of the ormal sedimentation of red blood cells and (leucocytosis), and some protein ab f inflammation. P, either at the start of inflammation or <b>making it a better marker of inflammat</b> es of proteins, globulins or fibrinogen. and pregnancy can cause temporary ele	typicallý used in conjunction with other test such e above diseases as well as some others, such as s, such as a high red blood cell count normalities. Some changes in red cell shape (such r as it resolves. <b>ion.</b>





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MBBS, MD (PATHOLOGY)







		<b>/ Chopra</b> ogy & Microbiology) « Consultant Pathologist	Dr. Yugam MD CEO & Consultant	(Pathology)
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CLIENT ADDRESS	: 6349/1, NICHOLSON R0	DAD, AMBALA CANTT		
Test Name		Value	Unit	<b>Biological Reference interval</b>
	CLI	NICAL CHEMISTI	RY/BIOCHEMIST	RY
		GLUCOSE FA	ASTING (F)	
	G (F): PLASMA	95.04	mg/dL	NORMAL: < 100.0

KOS Diagnostic Lab (A Unit of KOS Healthcare)

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

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.

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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<b>JENT ADDRESS</b> : 6349/1, ]	NICHOLSON ROAD	), AMBALA CANTT		
est Name		Value	Unit	<b>Biological Reference interval</b>
		LIPID PROF	ILE : BASIC	
HOLESTEROL TOTAL: SERUN by CHOLESTEROL OXIDASE PAP	1	187.34	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR =
		70 70	( ) T	240.0
RIGLYCERIDES: SERUM by GLYCEROL PHOSPHATE OXIDAS	E (ENZYMATIC)	73.78	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0
DL CHOLESTEROL (DIRECT):	SEDIM	47.32	mg/dL	VERY HIGH: > OR = 500.0 LOW HDL: < 30.0
by SELECTIVE INHIBITION	SEROM	47.32	liig/ uL	BORDERLINE HIGH HDL: $30.0$ 60.0 HIGH HDL: > OR = $60.0$
DL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTO	METRY	125.26	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
ON HDL CHOLESTEROL: SER by CALCULATED, SPECTROPHOTO		140.02 <sup>H</sup>	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
LDL CHOLESTEROL: SERUM	METRY	14.76	mg/dL	0.00 - 45.00
DTAL LIPIDS: SERUM		448.46	mg/dL	350.00 - 700.00
HOLESTEROL/HDL RATIO: S. by CALCULATED, SPECTROPHOTOL	ERUM	3.96	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0



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Test Name		Value	Unit	Biological Reference interval
LDL/HDL RATIO: S	ERUM ECTROPHOTOMETRY	2.65	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/H	IDL RATIO: SERUM	1.56 <sup>L</sup>	RATIO	3.00 - 5.00

by 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 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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**REPORTING DATE** 

Dr. Yugam Chopra

MD (Pathology)

:1750805

:012502090022

:09/Feb/2025 10:50 AM

:09/Feb/2025 10:59AM

:09/Feb/202501:03PM

Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** : Mr. RAJ KIRAN : 21 YRS/MALE **PATIENT ID** REG. NO./LAB NO. : **REGISTRATION DATE** : **COLLECTION DATE** 

## :01525201

: KOS DIAGNOSTIC LAB

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

Test Name	Value	Unit	<b>Biological Reference interval</b>		
LIVER	FUNCTION TES	ST (COMPLETE)			
BILIRUBIN TOTAL: SERUM by diazotization, spectrophotometry	0.74	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20		
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.17	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	91 <sup>H</sup>	U/L	7.00 - 45.00		
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	73.1 <sup>H</sup>	U/L	0.00 - 49.00		
AST/ALT RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.24	RATIO	0.00 - 46.00		
ALKALINE PHOSPHATASE: SERUM by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL	81.36	U/L	40.0 - 130.0		
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	21.63	U/L	0.00 - 55.0		
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	7.2	gm/dL	6.20 - 8.00		
ALBUMIN: SERUM by bromocresol green	4.33	gm/dL	3.50 - 5.50		
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.87	gm/dL	2.30 - 3.50		
A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.51	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:**

> 2
> 2 (Highly Suggestive)
1.4 - 2.0
> 1.5
> 1.3 (Slightly Increased)





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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



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Dr. Yugam Chopra

mmol/L

90.0 - 110.0

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Test Name		Value	Unit	Biological Reference interval		
	KIDNE		TEST (COMPLETE)			
UREA: SERUM by UREASE - GLUTAM	IATE DEHYDROGENASE (GLDH)	20.29	mg/dL	10.00 - 50.00		
CREATININE: SERU	JM	1.15	mg/dL	0.40 - 1.40		
BLOOD UREA NITR by CALCULATED, SPE	COGEN (BUN): SERUM	9.48	mg/dL	7.0 - 25.0		
BLOOD UREA NITR RATIO: SERUM by CALCULATED, SPE	COGEN (BUN)/CREATININE	8.24 <sup>L</sup>	RATIO	10.0 - 20.0		
UREA/CREATININ by CALCULATED, SPE		17.64	RATIO			
URIC ACID: SERUM by URICASE - OXIDAS		6.54	mg/dL	3.60 - 7.70		
CALCIUM: SERUM by ARSENAZO III, SPE	CTROPHOTOMETRY	9.67	mg/dL	8.50 - 10.60		
PHOSPHOROUS: SE by PHOSPHOMOLYBE	RUM DATE, SPECTROPHOTOMETRY	3.07	mg/dL	2.30 - 4.70		
<b>ELECTROLYTES</b>						
SODIUM: SERUM by ISE (ION SELECTIV	'E ELECTRODE)	142.56	mmol/L	135.0 - 150.0		
POTASSIUM: SERU		4.58	mmol/L	3.50 - 5.00		

Dr. Vinay Chopra

CHLORIDE: SERUM 106.92 by ISE (ION SELECTIVE ELECTRODE) **ESTIMATED GLOMERULAR FILTERATION RATE** 92.9

ESTIMATED GLOMERULAR FILTERATION RATE (eGFR): SERUM

by ISE (ION SELECTIVE ELECTRODE)

**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.

3. GI haemorrhage.



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by CALCULATED





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Test Name			Value	Unit	t	Biologie	cal Refere	ence interv	/al
9. Certain drugs (e.g. <b>INCREASED RATIO (&gt;2</b> 1. Postrenal azotemia	tetracycline, gluc 20:1) WITH ELEVA a (BUN rises dispr	TED CREATININE LEV oportionately more	ELS:	(e.g. obstructive	uropathy).				
9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (< 1. Acute tubular necr 2. Low protein diet al 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 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERI OKD STAGE	tetracycline, gluc 20:1) WITH ELEVA a (BUN rises dispr superimposed of 10:1) WITH DECRE osis. ad starvation. e. creased urea syn (urea rather than monemias (urea of inappropiate al 10:1) WITH INCRE py (accelerates c eleases muscle c who develop ren sis (acetoacetate creased BUN/cre rapy (interferes w JLAR FILTERATION	cocorticoids) <b>TED CREATININE LEVI</b> oportionately more in renal disease. <b>ASED BUN :</b> thesis. creatinine diffuses of is virtually absent in ntidiuretic harmone) <b>ASED CREATININE:</b> onversion of creating reatinine). al failure. causes false increases atinine ratio). rith creatinine measu. <b>IRATE:</b> <b>DESCRIPTION</b>	ELS: than creatinine) but of extracellu blood). due to tubular s e to creatinine). e in creatinine v rement).	lar fluid). secretion of urea.	odologies,rest	D FINDINGS	mal ratio v	vhen dehyd	ratio
<ol> <li>Certain drugs (e.g., NCREASED RATIO (&gt;2</li> <li>Postrenal azotemia</li> <li>Prerenal azotemia</li> <li>Prerenal azotemia</li> <li>DECREASED RATIO (&lt;</li> <li>Acute tubular necr</li> <li>Low protein diet and</li> <li>Severe liver diseas</li> <li>Other causes of definition</li> <li>Repeated dialysis</li> <li>Inherited hyperam</li> <li>SIADH (syndrome of Pregnancy.</li> <li>Pregnancy.</li> <li>PCREASED RATIO (</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>NAPPROPIATE RATIO</li> <li>Diabetic ketoacido</li> <li>Should produce an in</li> <li>Cephalosporin their</li> <li>CKD STAGE</li> <li>G1</li> </ol>	tetracycline, gluc 20:1) WITH ELEVA a (BUN rises dispr superimposed of 10:1) WITH DECRE osis. ad starvation. e. creased urea syn (urea rather than monemias (urea of inappropiate al 10:1) WITH INCRE py (accelerates c eleases muscle c who develop ren creased BUN/cre rapy (interferes w JLAR FILTERATION Norr	cocorticoids) <b>TED CREATININE LEVI</b> oportionately more in renal disease. <b>ASED BUN :</b> thesis. creatinine diffuses of is virtually absent in ntidiuretic harmone) <b>ASED CREATININE:</b> onversion of creating reatinine). al failure. causes false increase atinine ratio). rith creatinine measu. <b>IRATE:</b> <b>DESCRIPTION</b> nal kidney function	ELS: than creatinine) but of extracellu blood). due to tubular s e to creatinine). e in creatinine v rement). GFR (mL/r	lar fluid). secretion of urea. with certain meth nin/1.73m2 )	odologies,resu ASSOCIATEI No prot	<b>D FINDINGS</b> einuria	mal ratio v	vhen dehyd	ratio
<ol> <li>Certain drugs (e.g., NCREASED RATIO (&gt;2</li> <li>Postrenal azotemia</li> <li>Prerenal azotemia</li> <li>Prerenal azotemia</li> <li>DECREASED RATIO (&lt;</li> <li>Acute tubular necr</li> <li>Low protein diet and</li> <li>Severe liver diseas</li> <li>Other causes of definition</li> <li>Repeated dialysis</li> <li>Inherited hyperam</li> <li>SIADH (syndrome of Pregnancy.</li> <li>Pregnancy.</li> <li>Pregnancy.</li> <li>Phenacimide theration</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>NAPPROPIATE RATIO</li> <li>Diabetic ketoacido</li> <li>Should produce an in</li> <li>Cephalosporin theration</li> <li>CKD STAGE</li> </ol>	tetracycline, gluc 20:1) WITH ELEVA a (BUN rises dispr superimposed of 10:1) WITH DECRE osis. ad starvation. e. creased urea syn (urea rather than monemias (urea of inappropiate al 10:1) WITH INCRE py (accelerates c eleases muscle c who develop ren creased BUN/cre rapy (interferes w JLAR FILTERATION Norr Kic	cocorticoids) <b>TED CREATININE LEVI</b> oportionately more in renal disease. <b>ASED BUN :</b> thesis. creatinine diffuses of is virtually absent in ntidiuretic harmone) <b>ASED CREATININE:</b> onversion of creating reatinine). al failure. causes false increases atinine ratio). rith creatinine measu. <b>IRATE:</b> <b>DESCRIPTION</b>	ELS: than creatinine) but of extracellu blood). due to tubular s e to creatinine). e in creatinine v rement). GFR (mL/r	lar fluid). secretion of urea. with certain meth	odologies,rest	<b>D FINDINGS</b> einuria f Protein ,		vhen dehyd	ratio
<ol> <li>Certain drugs (e.g., NCREASED RATIO (&gt;2</li> <li>Postrenal azotemia</li> <li>Prerenal azotemia</li> <li>Prerenal azotemia</li> <li>CECREASED RATIO (&lt;</li> <li>Acute tubular necr</li> <li>Low protein diet and</li> <li>Severe liver diseas</li> <li>Other causes of definition</li> <li>Severe liver diseas</li> <li>Other causes of definition</li> <li>Severe liver diseas</li> <li>Other causes of definition</li> <li>Severe liver diseas</li> <li>Acute tubular necr</li> <li>Severe liver diseas</li> <li>Other causes of definition</li> <li>Severe liver diseas</li> <li>Other causes of definition</li> <li>Severe liver diseas</li> <li>Pregnancy.</li> <li>DECREASED RATIO (</li> <li>Phenacimide thera</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>NAPPROPIATE RATIO</li> <li>Diabetic ketoacido</li> <li>cephalosporin their</li> <li>STIMATED GLOMERI</li> <li>CKD STAGE</li> <li>G1</li> </ol>	tetracycline, gluc 20:1) WITH ELEVA a (BUN rises dispr superimposed of 10:1) WITH DECRE osis. ad starvation. e. creased urea syn (urea rather than monemias (urea of inappropiate al 10:1) WITH INCRE py (accelerates c eleases muscle c who develop ren creased BUN/cre rapy (interferes w JLAR FILTERATION Norr Kic no	cocorticoids) <b>TED CREATININE LEVI</b> oportionately more in renal disease. <b>ASED BUN :</b> thesis. creatinine diffuses of is virtually absent in ntidiuretic harmone) <b>ASED CREATININE:</b> onversion of creating reatinine). al failure. causes false increase atinine ratio). ith creatinine measu. <b>IRATE:</b> <b>DESCRIPTION</b> nal kidney function Iney damage with	ELS: than creatinine) but of extracellu blood). due to tubular s e to creatinine). e in creatinine v rement). GFR (mL/r	lar fluid). secretion of urea. with certain meth nin/1.73m2 )	nodologies,rest ASSOCIATEI No prot Presence c	<b>D FINDINGS</b> einuria f Protein ,		vhen dehyd	ratio
<ul> <li>P. Certain drugs (e.g.</li> <li>INCREASED RATIO (&gt;2</li> <li>Prerenal azotemia</li> <li>DECREASED RATIO (&lt;</li> <li>1. Acute tubular necr</li> <li>Low protein diet and</li> <li>Severe liver diseas</li> <li>Other causes of definition of the second dialysis</li> <li>Severe liver diseas</li> <li>Other causes of definition of the second dialysis</li> <li>Repeated dialysis</li> <li>SIADH (syndrome of the second dialysis)</li> <li>Pregnancy.</li> <li>DECREASED RATIO (&lt;</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>NAPPROPIATE RATIO</li> <li>Cephalosporin the second dialysis</li> <li>CEDED GLOMERI</li> <li>CAL STAGE</li> <li>G1</li> <li>G2</li> </ul>	tetracycline, gluc 20:1) WITH ELEVA a (BUN rises dispr superimposed of 10:1) WITH DECRE osis. ad starvation. e. creased urea syn (urea rather than monemias (urea of inappropiate an 10:1) WITH INCRE py (accelerates c eleases muscle c who develop ren : sis (acetoacetate creased BUN/cre rapy (interferes w JLAR FILTERATION Norr Norr Kic no Mill Mode	cocorticoids) <b>TED CREATININE LEVI</b> oportionately more in renal disease. <b>ASED BUN :</b> thesis. creatinine diffuses of is virtually absent in intidiuretic harmone) <b>ASED CREATININE:</b> onversion of creating reatinine ratio). al failure. causes false increase atinine ratio). ith creatinine measu. <b>IRATE:</b> <b>DESCRIPTION</b> mal kidney function Iney damage with rmal or high GFR d decrease in GFR rate decrease in GFR	ELS: than creatinine) but of extracellu blood). due to tubular s e to creatinine v rement). GFR (mL/r 30 30 30 30 30 30 30 30 30 30	lar fluid). secretion of urea. with certain meth nin/1.73m2) .90 .90 .90 .90	nodologies,rest ASSOCIATEI No prot Presence c	<b>D FINDINGS</b> einuria f Protein ,		vhen dehyd	ratio
<ul> <li>P. Certain drugs (e.g.,</li> <li>INCREASED RATIO (&gt;2</li> <li>Prerenal azotemia</li> <li>Prerenal azotemia</li> <li>DECREASED RATIO (&lt;</li> <li>Acute tubular necr</li> <li>Low protein diet and</li> <li>Severe liver diseas</li> <li>Other causes of decision</li> <li>Repeated dialysis</li> <li>SIADH (syndrome of the second secon</li></ul>	tetracycline, gluc 20:1) WITH ELEVA a (BUN rises dispr superimposed of 10:1) WITH DECRE osis. ad starvation. e. creased urea syn (urea rather than monemias (urea of inappropiate an 10:1) WITH INCRE py (accelerates c eleases muscle c who develop ren : sis (acetoacetate creased BUN/cre rapy (interferes w JLAR FILTERATION Norr Norr Kic no Mill Mode	cocorticoids) <b>TED CREATININE LEVI</b> oportionately more in renal disease. <b>ASED BUN :</b> thesis. creatinine diffuses of is virtually absent in intidiuretic harmone) <b>ASED CREATININE:</b> onversion of creating reatinine). al failure. causes false increase atinine ratio). ith creatinine measu. <b>IRATE:</b> <b>DESCRIPTION</b> nal kidney function Iney damage with rmal or high GFR d decrease in GFR	ELS: than creatinine) but of extracellu blood). due to tubular s e to creatinine v rement). GFR (mL/r 60 30 15	lar fluid). secretion of urea. with certain meth nin/1.73m2 ) .90 .90	nodologies,rest ASSOCIATEI No prot Presence c	<b>D FINDINGS</b> einuria f Protein ,		vhen dehyd	ratio





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

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









	Dr. Vinay Chopr MD (Pathology & Mic Chairman & Consulta	robiology) MI	m Chopra D (Pathology) nt Pathologist
NAME	: Mr. RAJ KIRAN		
AGE/ GENDER	: 21 YRS/MALE	PATIENT ID	: 1750805
COLLECTED BY	:	REG. NO./LAB NO.	: 012502090022
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 09/Feb/2025 10:50 AM
BARCODE NO.	: 01525201	COLLECTION DATE	: 09/Feb/2025 10:59AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	<b>REPORTING DATE</b>	: 09/Feb/2025 12:07PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AME	SALA CANTT	
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





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

KOS Central Lab: 6349/1, Nicholson Road, Ambala Cantt -133 001, Haryana KOS Molecular Lab: IInd Floor, Parry Hotel, Staff Road, Opp. GPO, Ambala Cantt -133 001, Haryana 0171-2643898, +91 99910 43898 care@koshealthcare.com www.koshealthcare.com







Dr. Vinay C MD (Pathology Chairman & Co				(Pathology)	
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BARCODE NO.	:01525201		LECTION DATE	:09/Feb/202510:59AM	
CLIENT CODE.	: KOS DIAGNOSTIC LAB		PORTING DATE	: 09/Feb/2025 11:28AM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	AMBALA CANTT			
Test Name		Value	Unit	Biological Reference interval	
		CLINICAL PA	THOLOGY		
	URINE RO		SCOPIC EXAMINA	ATION	
PHYSICAL EXAMI					
QUANTITY RECIEV		10	ml		
by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY	AMBER YELL	OW	PALE YELLOW	
	CTANCE SPECTROPHOTOMETRY	AMDER IELL	000	PALE FELLOW	
TRANSPARANCY	CTANCE SPECTROPHOTOMETRY	CLEAR		CLEAR	
SPECIFIC GRAVITY	<i>l</i>	1.01		1.002 - 1.030	
by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY				
REACTION	INATION	ACIDIC			
by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY				
PROTEIN by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)	
SUGAR		Negative		NEGATIVE (-ve)	
by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY	5.5		5.0 - 7.5	
by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY				
BILIRUBIN by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)	
NITRITE		Negative		NEGATIVE (-ve)	
UROBILINOGEN	CTANCE SPECTROPHOTOMETRY.	Normal	EU/dL	0.2 - 1.0	
	CTANCE SPECTROPHOTOMETRY				
KETONE BODIES by DIP STICK/REFLEC	CTANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)	
BLOOD	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)	
ASCORBIC ACID	STANCE SPECTROPHOTOMETRY	NEGATIVE (-v	ve)	NEGATIVE (-ve)	
RED BLOOD CELLS		NEGATIVE (-v	ve) /HPF	0 - 3	
RED DECOD CEEED	(11003)			0 0	





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 0171-2643898, +91 99910 43898
 care@koshealthcare.com

 www.koshealthcare.com
 www.koshealthcare.com



TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.







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

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Test Name	Val	lue Unit	Biological Reference interval

by MICROSCOLT ON CENTRI OGED ON MART SEDIMENT				
PUS CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	2-3	/HPF	0 - 5	
EPITHELIAL CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	0-1	/HPF	ABSENT	
CRYSTALS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)	
CASTS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)	
BACTERIA by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)	
OTHERS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)	
TRICHOMONAS VAGINALIS (PROTOZOA) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	ABSENT		ABSENT	

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





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

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