



	<b>Dr. Vinay Chopra</b> MD (Pathology & Micr Chairman & Consultar	obiology)	ME	n Chopra D (Pathology) ht Pathologist
NAME	: Mrs. RAJNI			
AGE/ GENDER	: 30 YRS/FEMALE		PATIENT ID	: 1800175
COLLECTED BY	:		<b>REG. NO./LAB NO.</b>	: 012503210004
<b>REFERRED BY</b>	:		<b>REGISTRATION DATE</b>	: 21/Mar/2025 07:49 AM
BARCODE NO.	: 01527469		COLLECTION DATE	: 21/Mar/2025 07:59AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 21/Mar/2025 09:30AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMB	ALA CANT	ſ	
Test Name		Value	Unit	<b>Biological Reference interval</b>
	SW/A ST	HVA WI	ELLNESS PANEL: 1.	0
			OOD COUNT (CBC)	
DED BLOOD CELLS	(RBCS) COUNT AND INDICES	LEIE DI		
<u>RED BLOOD CELLS</u> HAEMOGLOBIN (HE		10.3 <sup>L</sup>	gm/dL	12.0 - 16.0
by CALORIMETRIC			Ű	
RED BLOOD CELL (I	RBC) COUNT	4.22	Millions	s/cmm 3.50 - 5.00
PACKED CELL VOLU		33.3 <sup>L</sup>	%	37.0 - 50.0
MEAN CORPUSCULA	AR VOLUME (MCV)	78.9 <sup>L</sup>	fL	80.0 - 100.0
MEAN CORPUSCUL	UTOMATED HEMATOLOGY ANALYZER AR HAEMOGLOBIN (MCH)	24.3 <sup>L</sup>	pg	27.0 - 34.0
MEAN CORPUSCUL	UTOMATED HEMATOLOGY ANALYZER AR HEMOGLOBIN CONC. (MCHC) UTOMATED HEMATOLOGY ANALYZER	30.8 <sup>L</sup>	g/dL	32.0 - 36.0
RED CELL DISTRIBU	JTION WIDTH (RDW-CV)	16	%	11.00 - 16.00
	JTOMATED HEMATOLOGY ANALYZER JTION WIDTH (RDW-SD)	47.3	fL	35.0 - 56.0
	JTOMATED HEMATOLOGY ANALYZER	47.5		33.0 - 30.0
MENTZERS INDEX by CALCULATED		18.7	RATIO	BETA THALASSEMIA TRAIT: < 13.0
.,				IRON DEFICIENCY ANEMIA:
	EV	90 70	DATIO	>13.0 DETA THALASSENIA TRAFT.
GREEN & KING IND by calculated	EA	29.78	RATIO	BETA THALASSEMIA TRAIT:< 65.0
				IRON DEFICIENCY ANEMIA: >
WHITE BLOOD CEI	IS (WR(S)			65.0
TOTAL LEUCOCYTE	COUNT (TLC)	5600	/cmm	4000 - 11000
,	by sf cube & microscopy LOOD CELLS (nRBCS)	NIL		0.00 - 20.00
	T HEMATOLOGY ANALYZER			
	LOOD CELLS (nRBCS) %	NIL	%	< 10 %





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



TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





EXCELLENCE IN HEALTHCARE & DIAGNOSTICS

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

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Test Name	Value	Unit	Biological Reference interval
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS by flow cytometry by sf cube & microscopy	59	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	31	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	4	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	6	%	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	3304	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1736	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by flow cytometry by SF cube & microscopy	224	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	336	/cmm	80 - 880
PLATELETS AND OTHER PLATELET PREDICTIVE	MARKERS.		
PLATELET COUNT (PLT) by hydro dynamic focusing, electrical impedence	225000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.31	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by hydro dynamic focusing, electrical impedence	14 <sup>H</sup>	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	120000 <sup>H</sup>	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	53.3 <sup>H</sup>	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by hydro dynamic focusing, electrical impedence NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD	16.3	%	15.0 - 17.0



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	MD	Vinay Chopra (Pathology & Microbiology) rman & Consultant Patholog		(Pathology)
AME	: Mrs. RAJNI			
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est Name		Value	Unit	Biological Reference interval
by RED CELL AGGREG <b>ITERPRETATION:</b> . ESR is a non-specif nmune disease, but . An ESR can be affe s C-reactive protein	does not tell the hea cted by other condit be used to monitor o	TE (ESR) 9 PHOTOMETRY evated result often indicate alth practitioner exactly which ions besides inflammation.	ere the inflammation is in th For this reason, the ESR is ty	hr 0 - 20 ion associated with infection, cancer and auto





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		Chopra & Microbiology) onsultant Pathologist	Dr. Yugam MD CEO & Consultant	(Pathology)
IAME	: Mrs. RAJNI			
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAI	D, AMBALA CANTT		
Test Name		Value	Unit	<b>Biological Reference interval</b>
	CLIN	ICAL CHEMISTRY	BIOCHEMIST	RY
		GLUCOSE FAST	'ING (F)	
		die code moi		

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. 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 CODE.	: KOS DIAGNOSTIC LAB	RE	PORTING DATE	: 21/Mar/2025 11:57AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	, AMBALA CANTT		
Test Name		Value	Unit	<b>Biological Reference interval</b>
		LIPID PROFI	I.F · BASIC	
CHOLESTEROL TO by CHOLESTEROL O.		174.75	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0
FRIGLYCERIDES: S	SERUM PHATE OXIDASE (ENZYMATIC)	252.08 <sup>H</sup>	mg/dL	HIGH CHOLESTEROL: > OR = 240.0 OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0
IDL CHOLESTERO	DL (DIRECT): SERUM	58.16	mg/dL	HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0 LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0
DL CHOLESTERO	L: SERUM ECTROPHOTOMETRY	66.17	mg/dL	60.0 HIGH HDL: > OR = 60.0 OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129. BORDERLINE HIGH: 130.0 -
NON HDL CHOLES by Calculated, spi	TEROL: SERUM ECTROPHOTOMETRY	116.59	mg/dL	159.0 HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0 OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159. BORDERLINE HIGH: 160.0 -
/LDL CHOLESTER		50.42 <sup>H</sup>	mg/dL	189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0 0.00 - 45.00
FOTAL LIPIDS: SEI		601.58	mg/dL	350.00 - 700.00
CHOLESTEROL/HI	ECTROPHOTOMETRY DL RATIO: SERUM ECTROPHOTOMETRY	3	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 PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





Dr. Vinay ChopraDr. Yugam ChopraMD (Pathology & Microbiology)MD (Pathology)Chairman & Consultant PathologistCEO & Consultant Pathologist				
NAME	: Mrs. RAJNI			
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Test Name		Value	Unit	<b>Biological Reference interval</b>
LDL/HDL RATIO: S by CALCULATED, SPE		1.14	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/H by CALCULATED, SPE		4.33	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.

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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Test Name	Value	Unit	Biological Reference interval
LIVER	FUNCTION TE	ST (COMPLETE)	
BILIRUBIN TOTAL: SERUM by DIAZOTIZATION, SPECTROPHOTOMETRY	0.94	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	0.73	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	19.7	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	17.1	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.15	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by Para NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL	111.19	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	15.14	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	7.63	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	4.36	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	3.27	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.33	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
	. 0043/1, Menolson Rond, Am		
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	MD (Pathology & M Chairman & Consult	icrobiology) M[	D (Pathology)
	Dr. Vinay Chop	ora I Dr. Yugai	n Chopra

### 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. Vinay Chopra Dr. Yugam Chopra MD (Pathology) MD (Pathology & Microbiology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** NAME : Mrs. RAJNI **AGE/ GENDER** : 30 YRS/FEMALE **PATIENT ID** :1800175 **COLLECTED BY** :012503210004 REG. NO./LAB NO. **REFERRED BY REGISTRATION DATE** : 21/Mar/2025 07:49 AM **BARCODE NO.** :01527469 **COLLECTION DATE** : 21/Mar/2025 07:59AM CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE** : 21/Mar/2025 11:57AM **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Value Unit Test Name **KIDNEY FUNCTION TEST (COMPLETE)** UREA: SERUM 39.73 mg/dL by UREASE - GLUTAMATE DEHYDROGENASE (GLDH) **CREATININE: SERUM** 0.94 mg/dL by ENZYMATIC, SPECTROPHOTOMETERY BLOOD UREA NITROGEN (BUN): SERUM 18.57 mg/dL by CALCULATED, SPECTROPHOTOMETRY RATIO BLOOD UREA NITROGEN (BUN)/CREATININE 19.76RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY **UREA/CREATININE RATIO: SERUM** 42.27 RATIO by CALCULATED, SPECTROPHOTOMETRY URIC ACID: SERUM 5.67 mg/dL by URICASE - OXIDASE PEROXIDASE CALCIUM: SERUM 9.32 mg/dL by ARSENAZO III, SPECTROPHOTOMETRY PHOSPHOROUS: SERUM mg/dL 3.6

by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY **ELECTROLYTES** SODIUM: SERUM 139.2 mmol/L by ISE (ION SELECTIVE ELECTRODE) POTASSIUM: SERUM 4.27mmol/L by ISE (ION SELECTIVE ELECTRODE) CHLORIDE: SERUM 104.4mmol/L by ISE (ION SELECTIVE ELECTRODE) **ESTIMATED GLOMERULAR FILTERATION RATE** 83.7

ESTIMATED GLOMERULAR FILTERATION RATE (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.

Catabolic states with increased tissue breakdown.

3. GI haemorrhage.



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**Biological Reference interval** 

10.00 - 50.00

0.40 - 1.20

7.0 - 25.0

10.0 - 20.0

2.50 - 6.80

8.50 - 10.60

2.30 - 4.70

135.0 - 150.0

3.50 - 5.00

90.0 - 110.0

AMBALA CANTT **FEST PERFORMED AT KOS DIAGNOSTIC LAB.** 





)9001:2008 CERTIFIED LAB			EXCELLENCE IN HEALTHCARE & DIAGNOSTICS						
	1	<b>Dr. Vinay Chopra</b> 1D (Pathology & Micro Chairman & Consultan	Dr. Yugam Chopra MD (Pathology) t CEO & Consultant Pathologist						
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Fest Name			Value	Uni	it	Biol	ogical R	eference	interva
7. Urine reabsorption 3. Reduced muscle m 9. Certain drugs (e.g. <b>NCREASED RATIO (&gt;2</b> 1. Postrenal azotemia 2. Prerenal azotemia <b>DECREASED RATIO (</b> <1 1. Acute tubular necr	ass (subnormal d tetracycline, glu <b>D:1) WITH ELEVA</b> (BUN rises dispi superimposed o <b>D:1) WITH DECRI</b> Dsis.	reatinine production) cocorticoids) <b>TED CREATININE LEVE</b> oportionately more tl n renal disease.	_S:	ne) (e.g. obstructive			ndrome,		
<ol> <li>Virine reabsorption</li> <li>Reduced muscle m</li> <li>Certain drugs (e.g.</li> <li>NCREASED RATIO (&gt;2</li> <li>Postrenal azotemia</li> <li>Perenal azotemia</li> <li>Perenal azotemia</li> <li>Acute tubular necr</li> <li>Low protein diet ar</li> <li>Severe liver disease</li> <li>Other causes of de</li> <li>Repeated dialysis (</li> <li>SIADH (syndrome c</li> <li>Pregnancy.</li> <li>PECREASED RATIO (&lt;1</li> <li>Phenacimide thera</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>NAPPROPIATE RATIO</li> <li>Diabetic ketoacido</li> <li>cephalosporin ther</li> </ol>	(e.g. ureter colo ass (subnormal of tetracycline, glu <b>D:1) WITH ELEVA</b> (BUN rises dispisuperimposed of <b>0:1) WITH DECRI</b> Disis. d starvation. creased urea syrurea rather than monemias (urea f inappropiate a <b>0:1) WITH INCRE</b> Dy (accelerates of eleases muscle of who develop rener sis (acetoacetate creased BUN/crea apy (interferes v	thesis. creatinine production) <b>TED CREATININE LEVE</b> oportionately more the n renal disease. <b>EASED BUN :</b> thesis. creatinine diffuses of is virtually absent in level ntidiuretic harmone) of <b>ASED CREATININE:</b> onversion of creatine reatinine). al failure. e causes false increase statinine ratio). vith creatinine measur <b>J RATE:</b>	S: han creatinin ut of extrace blood). due to tubula to creatinin e in creatinin ement).	ellular fluid). ar secretion of urea. e). e with certain meth	uropathy hodologie	). s,resulting in	normal ra		dehydra
2. Urine reabsorption 3. Reduced muscle m 4. Certain drugs (e.g. <b>NCREASED RATIO (&gt;2</b> 4. Postrenal azotemia 5. Prerenal azotemia 6. Certain drubular necr 7. Low protein diet ar 7. Severe liver disease 6. Other causes of de 6. Repeated dialysis ( 6. Inherited hyperam 7. SIADH (syndrome c 7. SIADH (syndrome c 7. Rhabdomyolysis (r 7. Rhabdomyolysis (r 7. Muscular patients 7. Muscular patients 7. Mapproplate RATIO 7. Diabetic ketoacido 7. Cephalosporin ther 7. STIMATED GLOMERL 7. CKD STAGE	(e.g. ureter colo ass (subnormal of tetracycline, glu <b>D:1) WITH ELEVA</b> (BUN rises dispi- superimposed of <b>0:1) WITH DECRI</b> osis. d starvation. creased urea syr- urea rather thar monemias (urea f inappropiate a <b>0:1) WITH INCRE</b> oy (accelerates of eleases muscle of who develop rer sis (acetoacetate creased BUN/crea apy (interferes v LAR FILTERATION	thesis. creatinine production) <b>TED CREATININE LEVE</b> oportionately more the n renal disease. <b>EASED BUN :</b> thesis. creatinine diffuses of is virtually absent in level ntidiuretic harmone) of <b>ASED CREATININE:</b> onversion of creatine reatinine). hal failure. e causes false increase tatinine ratio). vith creatinine measure <b>IRATE:</b> <b>DESCRIPTION</b>	S: han creatinin ut of extrace blood). due to tubula to creatinin e in creatinin ement).	Ilular fluid). ar secretion of urea. e). e with certain meth L/min/1.73m2 )	uropathy nodologie ASSOC	). s,resulting in	normal ra		dehydra
<ol> <li>Virine reabsorption</li> <li>Reduced muscle m</li> <li>Certain drugs (e.g.</li> <li>NCREASED RATIO (&gt;2</li> <li>Postrenal azotemia</li> <li>Perenal azotemia</li> <li>Perenal azotemia</li> <li>CECREASED RATIO (&lt;1</li> <li>Acute tubular necr</li> <li>Low protein diet ar</li> <li>Severe liver disease</li> <li>Other causes of de</li> <li>Repeated dialysis (</li> <li>Inherited hyperam</li> <li>SIADH (syndrome c</li> <li>Pregnancy.</li> <li>PECREASED RATIO (&lt;1</li> <li>Phenacimide thera</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>NAPPROPIATE RATIO</li> <li>Diabetic ketoacido</li> <li>hould produce an in-</li> <li>Cephalosporin ther</li> </ol>	(e.g. ureter colo ass (subnormal of tetracycline, glu <b>D:1) WITH ELEVA</b> (BUN rises dispi- superimposed of <b>0:1) WITH DECRI</b> osis. d starvation. creased urea syr- urea rather thar monemias (urea f inappropiate a <b>0:1) WITH INCRE</b> oy (accelerates of eleases muscle of who develop rer sis (acetoacetate creased BUN/crea apy (interferes v LAR FILTERATION	thesis. creatinine production) <b>TED CREATININE LEVE</b> oportionately more the n renal disease. <b>EASED BUN :</b> thesis. creatinine diffuses of is virtually absent in level ntidiuretic harmone) of <b>ASED CREATININE:</b> onversion of creatine reatinine). al failure. e causes false increase statinine ratio). vith creatinine measur <b>J RATE:</b>	S: han creatinin ut of extrace blood). due to tubula to creatinin e in creatinin ement).	ellular fluid). ar secretion of urea. e). e with certain meth	uropathy nodologie <u>ASSOC</u>	). s,resulting in	normal ra		dehydra
Urine reabsorption     Reduced muscle m     Certain drugs (e.g.     NCREASED RATIO (>2     Postrenal azotemia     Prerenal azotemia     DECREASED RATIO (<1     Acute tubular necr     Low protein diet ar     Severe liver disease     Other causes of de     Repeated dialysis (     Inherited hyperam     SIADH (syndrome c     Rhabdomyolysis (r     Muscular patients     NAPPROPIATE RATIO     Diabetic ketoacido     hould produce an in     CEMBALED GLOMERL     CKD STAGE     G1     G2	(e.g. ureter colo ass (subnormal of tetracycline, glu <b>D:1) WITH ELEVA</b> (BUN rises dispi- superimposed of <b>0:1) WITH DECRI</b> osis. d starvation. creased urea syr- urea rather than monemias (urea f inappropiate a <b>0:1) WITH INCRE</b> oy (accelerates of eleases muscle of who develop rer sis (acetoacetate creased BUN/crea apy (interferes v LAR FILTERATION Normality (interferes v LAR FILTERATION Normality (interferes v Normality (interferes v LAR FILTERATION Normality (interferes v Normality (interferes v Normali	thesis. creatinine diffuses o is virtually absent in l <b>ASED CREATININE</b> thesis. creatinine diffuses o is virtually absent in l ntidiuretic harmone) o <b>ASED CREATININE:</b> onversion of creatine reatinine). al failure. causes false increase tatinine ratio). vith creatinine measur <b>IRATE:</b> <b>DESCRIPTION</b> mal kidney function Iney damage with ormal or high GFR	S: han creatinin ut of extrace blood). due to tubula to creatinin e in creatinin ement).	Ilular fluid). ar secretion of urea. e). e with certain meth L/min/1.73m2) >90 >90	uropathy nodologie <u>ASSOC</u> Prese	). s,resulting in <b>:IATED FINDIN</b> o proteinuria	normal ra		dehydra
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7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. <b>NCREASED RATIO (&gt;2</b> 1. Postrenal azotemia <b>DECREASED RATIO (</b> >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 8. Pregnancy. <b>DECREASED RATIO (</b> <1 1. Phenacimide thera 2. Rhabdomyolysis (ro 8. Muscular patients <b>NAPPROPIATE RATIO</b> 1. Diabetic ketoacido 5. Hould produce an in 2. Cephalosporin ther <b>ESTIMATED GLOMERL</b> <b>G1</b> <b>G2</b>	(e.g. ureter colo ass (subnormal of tetracycline, glu <b>D:1) WITH ELEVA</b> (BUN rises dispision superimposed of <b>0:1) WITH DECRI</b> osis. d starvation. creased urea syrurea rather than monemias (urea f inappropiate a <b>0:1) WITH INCRE</b> oy (accelerates of eleases muscle of who develop remissis (acetoacetate creased BUN/creased BUN/creased apy (interferes w LAR FILTERATION Normission (Normission) (No	thesis. creatinine diffuses o is virtually absent in l <b>ASED CREATININE</b> thesis. creatinine diffuses o is virtually absent in l ntidiuretic harmone) o <b>ASED CREATININE:</b> onversion of creatine reatinine). al failure. causes false increase tatinine ratio). vith creatinine measur <b>IRATE:</b> <b>DESCRIPTION</b> mal kidney function Iney damage with ormal or high GFR	S: han creatinin ut of extrace blood). due to tubula to creatinin e in creatinin ement).	Ilular fluid). ar secretion of urea. e). e with certain meth L/min/1.73m2) >90 >90	uropathy nodologie <u>ASSOC</u> Prese	). s,resulting in <u><b>CIATED FINDIN</b></u> o proteinuria ence of Proteir	normal ra		dehydra





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









	<b>Dr. Vinay Chopra</b> MD (Pathology & Micro Chairman & Consultan	obiology) ME	m <b>Chopra</b> D (Pathology) ht Pathologist
NAME	: Mrs. RAJNI		
AGE/ GENDER	: 30 YRS/FEMALE	PATIENT ID	: 1800175
COLLECTED BY	:	<b>REG. NO./LAB NO.</b>	: 012503210004
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 21/Mar/2025 07:49 AM
BARCODE NO.	: 01527469	COLLECTION DATE	: 21/Mar/2025 07:59AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	<b>REPORTING DATE</b>	: 21/Mar/2025 11:57AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBA	ALA 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)

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CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT					
Test Name	Value	Unit	<b>Biological Reference interval</b>		
	CLINICAL PATHO JTINE & MICROSCOP		ATION		
<u>PHYSICAL EXAMINATION</u> QUANTITY RECIEVED	10	ml			
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY	10	1111			
COLOUR by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY	PALE YELLOW		PALE YELLOW		
TRANSPARANCY	HAZY		CLEAR		
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY SPECIFIC GRAVITY by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY	>=1.030		1.002 - 1.030		
CHEMICAL EXAMINATION					
REACTION by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY	ACIDIC				
PROTEIN by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)		
SUGAR by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)		
pH by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY	5.5		5.0 - 7.5		
BILIRUBIN by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)		
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY NITRITE by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY.	Negative		NEGATIVE (-ve)		
UROBILINOGEN by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY	Normal	EU/dL	0.2 - 1.0		
KETONE BODIES by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)		
BLOOD by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)		
ASCORBIC ACID by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY MICROSCOPIC EXAMINATION	NEGATIVE (-ve)		NEGATIVE (-ve)		
RED BLOOD CELLS (RBCs)	NEGATIVE (-ve)	/HPF	0 - 3		

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

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

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Test Name	Value	Unit	<b>Biological Reference interval</b>

I est Maine	value	ome	Diviogical Meler ence inter var
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
PUS CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	4-6	/HPF	0 - 5
EPITHELIAL CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	10-12	/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 \*\*\*



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