

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



	Dr. Vinay Chopra MD (Pathology & Micr Chairman & Consultar	robiology)		(Pathology)
NAME	: Mr. SHUBHAM			
AGE/ GENDER	: 30 YRS/MALE		PATIENT ID	: 1716408
COLLECTED BY	:		REG. NO./LAB NO.	: 012501050012
REFERRED BY	:		REGISTRATION DATE	: 05/Jan/2025 09:44 AM
BARCODE NO.	: 01523458		COLLECTION DATE	: 05/Jan/2025 09:45AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 05/Jan/2025 09:58AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMB	ALA CAN'I'T		
Fest Name		Value	Unit	Biological Reference interval
	SWAST	HYA WE	LLNESS PANEL: 1.	0
	COMP	PLETE BLO	DOD COUNT (CBC)	
RED BLOOD CELLS	(RBCS) COUNT AND INDICES			
IAEMOGLOBIN (H	B)	15.3	gm/dL	12.0 - 17.0
by CALORIMETRIC RED BLOOD CELL (RBC) COUNT	5.2 ^H	Millions	/cmm 3.50 - 5.00
by HYDRO DYNAMIC F	OCUSING, ELECTRICAL IMPEDENCE			
PACKED CELL VOLU	JME (PCV) UTOMATED HEMATOLOGY ANALYZER	47.7	%	40.0 - 54.0
	AR VOLUME (MCV) UTOMATED HEMATOLOGY ANALYZER	91.9	fL	80.0 - 100.0
AEAN CORPUSCUL	AR HAEMOGLOBIN (MCH)	29.4	pg	27.0 - 34.0
MEAN CORPUSCUL	UTOMATED HEMATOLOGY ANALYZER AR HEMOGLOBIN CONC. (MCHC)	32	g/dL	32.0 - 36.0
	UTOMATED HEMATOLOGY ANALYZER UTION WIDTH (RDW-CV)	13.1	%	11.00 - 16.00
by CALCULATED BY A	UTOMATED HEMATOLOGY ANALYZER			
	UTION WIDTH (RDW-SD) UTOMATED HEMATOLOGY ANALYZER	45.1	fL	35.0 - 56.0
MENTZERS INDEX		17.67	RATIO	BETA THALASSEMIA TRAIT: <
by CALCULATED				13.0 IRON DEFICIENCY ANEMIA:
				>13.0
GREEN & KING IND by calculated	DEX	23.13	RATIO	BETA THALASSEMIA TRAIT:<< 65.0
2, ONEOOLATED				IRON DEFICIENCY ANEMIA: >
MITTE BLOOD OT				65.0
WHITE BLOOD CE		0E 40		4000 11000
	BY SF CUBE & MICROSCOPY	8540	/cmm	4000 - 11000
	LOOD CELLS (nRBCS) RT HEMATOLOGY ANALYZER	NIL		0.00 - 20.00
	LOOD CELLS (nRBCS) %	NIL	%	< 10 %





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

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

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Dr. Vinay Chopra

MD (Pathology & Microbiology) Chairman & Consultant Pathologist

Test Name	Value	Unit	Biological Reference interval
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS	64	%	50 - 70
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	25	%	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	5466	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by flow cytometry by sf cube & microscopy	2135	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by flow cytometry by sf cube & microscopy	171	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by flow cytometry by sf cube & microscopy	769	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT by flow cytometry by sf cube & microscopy	0	/cmm	0 - 110
PLATELETS AND OTHER PLATELET PREDICTIVE	MARKERS.		
PLATELET COUNT (PLT) by hydro dynamic focusing, electrical impedence	250000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by hydro dynamic focusing, electrical impedence	0.33	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by hydro dynamic focusing, electrical impedence	13 ^H	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	119000 ^H	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	47.5 ^H	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by hydro dynamic focusing, electrical impedence NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD	16.6	%	15.0 - 17.0



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









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ARCODE NO.	: 01523458	COI	LECTION DATE	: 05/Jan/2025 09:45AM
LIENT CODE.	: KOS DIAGNOSTIC LAB	REF	ORTING DATE	: 05/Jan/2025 10:34AM
IENT ADDRESS	: 6349/1, NICHOLSON ROAD	, AMBALA CANTT		
by RED CELL AGGREC NTERPRETATION: . ESR is a non-specif nmune disease, but . An ESR can be affer s C-reactive protein	DIMENTATION RATE (ESR) GATION BY CAPILLARY PHOTOMET ic test because an elevated resi does not tell the health practit cted by other conditions beside	ult often indicates the p ioner exactly where the is inflammation. For thi	mm/1st presence of inflammat inflammation is in the s reason, the ESR is ty	hr 0 - 20





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CLIENT ADDRESS	: 6349/1, NICHOLSON	I ROAD, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		CLINICAL CHEMISTRY	//BIOCHEMISTR	RY
		GLUCOSE FAS	TING (F)	
GLUCOSE FASTING	G (F): PLASMA E - PEROXIDASE (GOD-POL	86.58	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0

IN ACCRDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES: 1. A fasting plasma glucose level below 100 mg/dl is considered normal. 2. A fasting plasma glucose level between 100 - 125 mg/dl is considered as glucose intolerant or prediabetic. A fasting and post-prandial blood

test (after consumption of 75 gms of glucose) is recommended for all such patients. 3. A fasting plasma glucose level of above 125 mg/dl is highly suggestive of diabetic state. A repeat post-prandial is strongly recommended for all such patients. A fasting plasma glucose level in excess of 125 mg/dl on both occasions is confirmatory for diabetic state.



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Fest Name		Value	Unit	Biological Reference interval
		LIPID PR	OFILE : BASIC	
HOLESTEROL TO	TAL · SFRUM	164.58	mg/dL	OPTIMAL: < 200.0
by CHOLESTEROL OX		104.00	ing, dE	BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: S by GLYCEROL PHOSP	ERUM HATE OXIDASE (ENZYMATIC)	64.5	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTERO	L (DIRECT): SERUM	44.93	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROI by CALCULATED, SPE		106.85	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 CHOLEST by CALCULATED, SPE		119.65	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
VLDL CHOLESTER	DL: SERUM	12.9	mg/dL	VERY HIGH: > 0R = 220.0 0.00 - 45.00
by CALCULATED, SPE FOTAL LIPIDS: SER by CALCULATED, SPE	сткорнотометку UM	393.76	mg/dL	350.00 - 700.00
CHOLESTEROL/HD	L RATIO: SERUM	3.66	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0





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Test Name		Value	Unit	Biological Reference interval
LDL/HDL RATIO: S by CALCULATED, SPE		2.38	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/H by CALCULATED, SPE		1.44 ^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.

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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Dr. Vinay Chopra

MD (Pathology & Microbiology)

Chairman & Consultant Pathologist

HEALTHCARE & DIAGNOSTICS EXCELLENCE IN Dr. Yugam Chopra MD (Pathology) **CEO & Consultant Pathologist**

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Test Name	Value	Unit	Biological Reference interval
LIVER	FUNCTION TE	ST (COMPLETE)	
BILIRUBIN TOTAL: SERUM by diazotization, spectrophotometry	0.69	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	0.52	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	31.7	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	60.3 ^H	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	0.53	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL	86.97	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	37.65	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	6.85	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	4.16	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.69	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by calculated, spectrophotometry	1.55	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
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)
HEPATOCELLULAR CARCINOMIA & CHRONIC HEPATTIS	> 1.3 (Slightly Increased)





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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).

GOOD PROGNOSTIC SIGN 0.3 - 0.6	
POOR PROGNOSTIC SIGN 1.2 - 1.6	



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kus diagnustic lab, ambala canti	Test Name		Value	Unit	Biological Reference interval			
I AI V	KIDNEY FUNCTION TEST (COMPLETE)							
JKIME	UREA: SERUM		19.79	mg/dL	10.00 - 50.00			
EKFC	by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)			0				
- - -	CREATININE: SERUM by enzymatic, spectrophotometery		1.03	mg/dL	0.40 - 1.40			
_	BLOOD UREA NITROGEN (BUN): SERUM		9.25	mg/dL	7.0 - 25.0			
	by CALCULATED, SPECTROPHOTOMETRY							
	BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM		8.98 ^L	RATIO	10.0 - 20.0			
	by CALCULATED, SPECTROPHOTOMETRY							
	UREA/CREATININE RATIO: SERUM		19.21	RATIO				
	by CALCULATED, SPECTROPHOTOMETRY URIC ACID: SERUM		6.14	mg/dL	3.60 - 7.70			
	by URICASE - OXIDASE PEROXIDASE			ing/ uL				
	CALCIUM: SERUM		10.26	mg/dL	8.50 - 10.60			
	-	by ARSENAZO III, SPECTROPHOTOMETRY PHOSPHOROUS: SERUM		mg/dL	2.30 - 4.70			
	by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY		3.06	ing, uz				
	ELECTROLYTES							
	SODIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)		138.6	mmol/L	135.0 - 150.0			
	POTASSIUM: SERUM by ISE (ION SELECTIVE ELECTRODE) CHLORIDE: SERUM by ISE (ION SELECTIVE ELECTRODE)		4.01	mmol/L	3.50 - 5.00			
			103.95	mmol/L	90.0 - 110.0			
		IERULAR FILTERATION RATE						
	(eGFR): SERUM by CALCULATED	ERULAR FILTERATION RATE	100.2					
	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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Test Name			Value	Uni	it	Biolog	jical Refer	ence interv	al
	tetracycline, glu 0:1) WITH ELEVA	TED CREATININE LEV	ELS:	no) (o a obstructivo	uronathy				
NCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (< 1. Acute tubular necr 2. Low protein diet an 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 NAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERL CKD STAGE G1	tetracycline, glu 0:1) WITH ELEVA (BUN rises disp superimposed o 10:1) WITH DECR osis. ad starvation. e. creased urea syn urea rather thar monemias (urea of inappropiate a 10:1) WITH INCRE py (accelerates of eleases muscle of who develop renti- sis (acetoacetate creased BUN/cro- apy (interferes of JLAR FILTERATIO Nor	cocorticoids) TED CREATININE LEV roportionately more n renal disease. EASED BUN : The thesis. a creatinine diffuses of is virtually absent in ntidiuretic harmone) EASED CREATININE: conversion of creating creatinine). hal failure. the causes false increase extinine ratio). vith creatinine measure V RATE: DESCRIPTION mal kidney function	ELS: than creatini blood). due to tubu e to creatinin e in creatini urement).	ellular fluid). lar secretion of urea ne). ne with certain metl nL/min/1.73m2) >90	n. hodologie: ASSOC	s,resulting in no I ATED FINDINGS proteinuria		when dehyd	atio
NCREASED RATIO (>2 Postrenal azotemia Perenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet an Severe liver diseas Other causes of de Repeated dialysis Nherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Anuscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in CED STAGE	tetracycline, glu 0:1) WITH ELEVA (BUN rises disp superimposed o 10:1) WITH DECR osis. ad starvation. e. creased urea syn urea rather thar monemias (urea of inappropiate a 10:1) WITH INCRE py (accelerates of eleases muscle of who develop renti- sis (acetoacetate creased BUN/cro- apy (interferes v JLAR FILTERATIO Nor	cocorticoids) TED CREATININE LEV roportionately more n renal disease. EASED BUN : The thesis. a creatinine diffuses of is virtually absent in ntidiuretic harmone) EASED CREATININE: conversion of creating creatinine). hal failure. the causes false increase extinine ratio). vith creatinine measure NATE: DESCRIPTION mal kidney function dney damage with	ELS: than creatini blood). due to tubu e to creatinin e in creatini urement).	ellular fluid). lar secretion of urea ne). ne with certain metl	hodologie: ASSOC	s,resulting in no IATED FINDINGS proteinuria nce of Protein ,	<u>.</u>	when dehyd	atio
NCREASED RATIO (>2 . Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (< . Acute tubular necr 2. Low protein diet and 3. Severe liver diseas 4. Other causes of de 5. Repeated dialysis (5. Inherited hyperam 7. SIADH (syndrome of 8. Pregnancy. DECREASED RATIO (< 7. Phenacimide thera 9. Muscular patients NAPPROPIATE RATIO 1. Diabetic ketoacido hould produce an in 1. Cephalosporin ther <u>STIMATED GLOMERU</u> <u>G1</u> <u>G2</u>	tetracycline, glu 0:1) WITH ELEVA (BUN rises disp superimposed o 10:1) WITH DECR osis. ad starvation. e. creased urea syn urea rather thar monemias (urea of inappropiate a 10:1) WITH INCRE py (accelerates of eleases muscle of who develop ref sis (acetoacetate creased BUN/cro apy (interferes v JLAR FILTERATIO Nor Kin	cocorticoids) TED CREATININE LEV roportionately more n renal disease. EASED BUN : The thesis. a creatinine diffuses of is virtually absent in ntidiuretic harmone) EASED CREATININE: conversion of creating creatinine). hal failure. the causes false increase extinine ratio). vith creatinine measure V RATE: DESCRIPTION mal kidney function	ELS: than creatini blood). due to tubu e to creatinin e in creatini urement).	ellular fluid). lar secretion of urea ne). ne with certain metl nL/min/1.73m2) >90	hodologie: ASSOC	s,resulting in no I ATED FINDINGS proteinuria	<u>.</u>	when dehyd	atio
NCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (< 1. Acute tubular necr 2. Low protein diet an 3. Severe liver diseas 4. Other causes of de 5. Repeated dialysis (6. Inherited hyperam 7. SIADH (syndrome of 8. Pregnancy. DECREASED RATIO (< 8. Phenacimide thera 9. Rhabdomyolysis (r 8. Muscular patients NAPPROPIATE RATIO 1. Diabetic ketoacido 5. Nould produce an in 2. Cephalosporin thera 5. CKD STAGE G1	tetracycline, glu 0:1) WITH ELEVA (BUN rises disp superimposed o 10:1) WITH DECR osis. ad starvation. 2. creased urea syr urea rather thar monemias (urea of inappropiate a 10:1) WITH INCRE py (accelerates of eleases muscle of who develop rei : sis (acetoacetate creased BUN/cro apy (interferes v JLAR FILTERATIO Nor Kin no	cocorticoids) TED CREATININE LEV roportionately more n renal disease. EASED BUN : The thesis. a creatinine diffuses of is virtually absent in ntidiuretic harmone) EASED CREATININE: conversion of creating treatinine). hal failure. the causes false increase extinine ratio). vith creatinine measure NATE: DESCRIPTION mal kidney function dney damage with prmal or high GFR	ELS: than creatini blood). due to tubu e to creatinin e in creatini urement). GFR (n	ellular fluid). lar secretion of urea ne). ne with certain meth nL/min/1.73m2) >90 >90	hodologie: ASSOC	s,resulting in no IATED FINDINGS proteinuria nce of Protein ,	<u>.</u>	when dehyd	atio
NCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (< 1. Acute tubular necr 2. Low protein diet an 3. Severe liver diseas 4. Other causes of de 5. Repeated dialysis (5. Inherited hyperam 7. SIADH (syndrome of 8. Pregnancy. DECREASED RATIO (< 6. Phenacimide thera 2. Rhabdomyolysis (r 8. Muscular patients NAPPROPIATE RATIO 1. Diabetic ketoacido 5. Nould produce an in 2. Cephalosporin ther 5. STIMATED GLOMERI CKD STAGE G1 G2 G3a	tetracycline, glu 0:1) WITH ELEVA (BUN rises disp superimposed o 10:1) WITH DECR osis. ad starvation. ad starvation. acceased urea syr urea rather thar monemias (urea of inappropiate a 10:1) WITH INCRE py (accelerates of eleases muscle of who develop rei sis (acetoacetate creased BUN/crea apy (interferes v JLAR FILTERATIO Nor Kin Model Model Model	cocorticoids) TED CREATININE LEV roportionately more n renal disease. EASED BUN : The thesis. n creatinine diffuses of is virtually absent in ntidiuretic harmone) EASED CREATININE: conversion of creatine creatinine). hal failure. the causes false increase extinine ratio). with creatinine measu N RATE: DESCRIPTION mal kidney function dney damage with prmal or high GFR_ Id decrease in GFR	ELS: than creatini blood). due to tubu e to creatinin e in creatini urement). GFR (n	ellular fluid). lar secretion of urea ne). ne with certain meth <u>>90 >90</u> >90 60 -89	hodologie: ASSOC	s,resulting in no IATED FINDINGS proteinuria nce of Protein ,	<u>.</u>	when dehyd	atio





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

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









: 01523458 : KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON ROAD, AMBALA CANTT	COLLECTION DATE REPORTING DATE	: 05/Jan/2025 09:45AM : 05/Jan/2025 11:08AM
: KOS DIAGNOSTIC LAB	REPORTING DATE	
: 01523458	COLLECTION DATE	: 05/Jan/2025 09:45AM
:	REGISTRATION DATE	: 05/Jan/2025 09:44 AM
:	REG. NO./LAB NO.	: 012501050012
: 30 YRS/MALE	PATIENT ID	: 1716408
: Mr. SHUBHAM		
		(Pathology) Pathologist
Dr. Vinay Chopra	Dr. Yugam	
	MD (Pathology & Microbiology) Chairman & Consultant Pathologis : Mr. SHUBHAM : 30 YRS/MALE	MD (Pathology & Microbiology) Chairman & Consultant Pathologist CEO & Consultant Mr. SHUBHAM : 30 YRS/MALE PATIENT ID REG. NO./LAB NO. REGISTRATION DATE

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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	Dr. Vinay Cł MD (Pathology & Chairman & Cor		Dr. Yugam MD CEO & Consultant	(Pathology)
NAME : Mr	r. SHUBHAM			
AGE/ GENDER : 30	YRS/MALE	PA	TIENT ID	: 1716408
COLLECTED BY :		RE	G. NO./LAB NO.	: 012501050012
REFERRED BY :		RE	GISTRATION DATE	: 05/Jan/2025 09:44 AM
	523458		LLECTION DATE	: 05/Jan/2025 09:45AM
	OS DIAGNOSTIC LAB			: 05/Jan/2025 10:28AM
CLIENT ADDRESS : 63	49/1, NICHOLSON ROAD,	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		CLINICAL PA	THOLOGY	
	URINE RO	OUTINE & MICRO	DSCOPIC EXAMINA	ATION
PHYSICAL EXAMINATIO	<u>DN</u>			
QUANTITY RECIEVED		10	ml	
COLOUR	SPECIROPHOTOMETRY	PALE YELLC	W	PALE YELLOW
by DIP STICK/REFLECTANCE TRANSPARANCY	by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			CLEAR
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY SPECIFIC GRAVITY by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY		CLEAR		
		>=1.030		1.002 - 1.030
CHEMICAL EXAMINATI				
REACTION		ACIDIC		
by DIP STICK/REFLECTANCE PROTEIN	SPECIROPHOIOMEIRY	Negative		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE	SPECTROPHOTOMETRY	-		
SUGAR by DIP STICK/REFLECTANCE	SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
pH	SPECTROPHOTOMETRY	<=5.0		5.0 - 7.5
by DIP STICK/REFLECTANCE BILIRUBIN	SPECIROPHOTOMETRY	Negative		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE NITRITE	SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE	SPECTROPHOTOMETRY.			
UROBILINOGEN		Normal	EU/dL	0.2 - 1.0
by DIP STICK/REFLECTANCE SPECTROPHOTOMET		Negative		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE BLOOD	SPECIKUPHUIOMEIRY	Negative		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE	SPECTROPHOTOMETRY	-)	
ASCORBIC ACID by DIP STICK/REFLECTANCE		NEGATIVE (-vej	NEGATIVE (-ve)
MICROSCOPIC EXAMIN			(LIDE	0.2
RED BLOOD CELLS (RBC	S)	NEGATIVE (-ve) /HPF	0 - 3



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EXCELLENCE IN HEALTHCARE & DIAGNOSTICS

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

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

NAME	: Mr. SHUBHAM		
AGE/ GENDER	: 30 YRS/MALE	PATIENT ID	: 1716408
COLLECTED BY	:	REG. NO./LAB NO.	: 012501050012
REFERRED BY	:	REGISTRATION DATE	: 05/Jan/2025 09:44 AM
BARCODE NO.	: 01523458	COLLECTION DATE	: 05/Jan/2025 09:45AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 05/Jan/2025 10:28AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		
Test Name	Value	Unit	Biological Reference interval

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
PUS CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	2-3	/HPF	0 - 5
EPITHELIAL CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	1-2	/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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