



	<b>Dr. Vinay Chopra</b> MD (Pathology & Micr Chairman & Consultar	obiology)		(Pathology)
NAME	: Mr. AMANDEEP SINGH			
AGE/ GENDER	: 46 YRS/MALE		PATIENT ID	: 1734528
COLLECTED BY	:		REG. NO./LAB NO.	: 012501250018
REFERRED BY	:		<b>REGISTRATION DATE</b>	: 25/Jan/2025 10:15 AM
BARCODE NO.	: 01524397		COLLECTION DATE	: 25/Jan/2025 10:17AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 25/Jan/2025 10:26AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMB/	ALA CANTI		
Test Name		Value	Unit	Biological Reference interval
	SWAST	HYA WE	LLNESS PANEL: 1.	0
	COMP	PLETE BL	OOD COUNT (CBC)	
RED BLOOD CELLS	(RBCS) COUNT AND INDICES			
HAEMOGLOBIN (H	B)	15.4	gm/dL	12.0 - 17.0
by CALORIMETRIC RED BLOOD CELL (	RBC) COUNT	4.92	Millions	/cmm 3.50 - 5.00
by HYDRO DYNAMIC F	OCUSING, ELECTRICAL IMPEDENCE	15.0		
PACKED CELL VOLU	JME (PCV) UTOMATED HEMATOLOGY ANALYZER	45.8	%	40.0 - 54.0
	AR VOLUME (MCV) utomated hematology analyzer	93	fL	80.0 - 100.0
MEAN CORPUSCUL	AR HAEMOGLOBIN (MCH)	31.4	pg	27.0 - 34.0
	UTOMATED HEMATOLOGY ANALYZER AR HEMOGLOBIN CONC. (MCHC)	33.7	g/dL	32.0 - 36.0
by CALCULATED BY A	UTOMATED HEMATOLOGY ANALYZER		, i i i i i i i i i i i i i i i i i i i	
	UTION WIDTH (RDW-CV) UTOMATED HEMATOLOGY ANALYZER	13.5	%	11.00 - 16.00
RED CELL DISTRIB	UTION WIDTH (RDW-SD)	47.3	fL	35.0 - 56.0
by CALCULATED BY A	UTOMATED HEMATOLOGY ANALYZER	18.9	RATIO	BETA THALASSEMIA TRAIT: <
by CALCULATED		10.0	101110	13.0
				IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INI	DEX	25.6	RATIO	BETA THALASSEMIA TRAIT:<=
by CALCULATED				65.0
				IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CE	LLS (WBCS)			4000 11000
WHITE BLOOD CE	E COUNT (TLC)	8200	/cmm	4000 - 11000
TOTAL LEUCOCYTE		8200 NIL	/cmm	4000 - 11000 0.00 - 20.00
TOTAL LEUCOCYTE by FLOW CYTOMETRY NUCLEATED RED E by AUTOMATED 6 PAF	COUNT (TLC) / by sf cube & microscopy		/cmm	





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

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NAME

AGE/ GENDER

**COLLECTED BY** 

**REFERRED BY** 

**BARCODE NO.** 

**CLIENT CODE.** 

**CLIENT ADDRESS** 



Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologist : Mr. AMANDEEP SINGH : 46 YRS/MALE :01524397 : KOS DIAGNOSTIC LAB

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

: Mr. AMANDEEP SINGH		
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: 6349/1, NICHOLSON ROAD, AMBALA CANTT	[	

Test Name	Value	Unit	Biological Reference interval
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	68	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	22	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	3	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	7	%	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	5576	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1804	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	246	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	574	/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	215000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.25	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	12	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	82000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	38.1	%	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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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANT	Т	
Test Name	Value	Unit	<b>Biological Reference interval</b>



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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



	MD (Pathology & Microbiology)		MD	Dr. Yugam Chopra MD (Pathology) CEO & Consultant Pathologist	
IAME	: Mr. AMANDEEP SINGH				
GE/ GENDER	: 46 YRS/MALE	PAT	ENT ID	: 1734528	
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ARCODE NO.	: 01524397	COL	ECTION DATE	: 25/Jan/2025 10:17AM	
LIENT CODE.	: KOS DIAGNOSTIC LAB	REP	DRTING DATE	: 25/Jan/2025 10:43AM	
IENT ADDRESS	: 6349/1, NICHOLSON ROAL	D, AMBALA CANTT			
est Name		Value	Unit	Biological Reference interval	
by RED CELL AGGRE NTERPRETATION: . ESR is a non-specif mmune disease, but 2. An ESR can be affe is C-reactive protein	does not tell the health practit ected by other conditions beside	sult often indicates the p tioner exactly where the es inflammation. For this	nflammation is in the reason, the ESR is ty	on associated with infection, cancer and auto-	





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		& Microbiology) onsultant Pathologist	Dr. Yugam MD CEO & Consultant	(Pathology)
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CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPO	RTING DATE	: 25/Jan/2025 12:01PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD	), AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	CLINI	CAL CHEMISTRY	BIOCHEMIST	RY
		GLUCOSE FAST	TING (F)	
		156.19 <sup>H</sup>	mg/dL	NORMAL: < 100.0

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. 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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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



		<b>hopra</b> & Microbiology) onsultant Pathologist	Dr. Yugam Chopra MD (Pathology) CEO & Consultant Pathologist	
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD	), AMBALA CANTT		
Test Name		Value	Unit	<b>Biological Reference interval</b>
		I IPID PRO	FILE : BASIC	
CHOLESTEROL TO	TAL · SEPLIM	200.73 <sup>H</sup>	mg/dL	OPTIMAL: < 200.0
by CHOLESTEROL O		200.73**	ing, uL	BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: S by GLYCEROL PHOSE	ERUM PHATE OXIDASE (ENZYMATIC)	225.92 <sup>H</sup>	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0
HDL CHOLESTERO by SELECTIVE INHIBIT	L (DIRECT): SERUM	61.24	mg/dL	VERY HIGH: > OR = 500.0 LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTERO		94.31	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.0 BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0
NON HDL CHOLES' by Calculated, spe		139.49 <sup>H</sup>	mg/dL	VERY HIGH: > OR = 190.0 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		45.18 <sup>H</sup>	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SEE by CALCULATED, SPE	RUM	627.38	mg/dL	350.00 - 700.00
by CALCULATED, SPE CHOLESTEROL/HE by CALCULATED, SPE	DL RATIO: SERUM	3.28	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0

KOS Diagnostic Lab (A Unit of KOS Healthcare)



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Test Name		Value	Unit	<b>Biological Reference interval</b>
LDL/HDL RATIO: S by CALCULATED, SPE		1.54	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/H by CALCULATED, SPE	IDL RATIO: SERUM	3.69	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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Test Name		Value	Unit	<b>Biological Reference interval</b>
BILIRUBIN DIRECT by DIAZO MODIFIED, S BILIRUBIN INDIRE by CALCULATED, SPE SGOT/AST: SERUM by IFCC, WITHOUT PY	: SERUM pectrophotometry Γ (CONJUGATED): SERUM spectrophotometry CCT (UNCONJUGATED): SERUM ectrophotometry μ μ μ	0.83 0.22 0.61 32	TEST (COMPLETE) mg/dL mg/dL mg/dL U/L	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20 0.00 - 0.40 0.10 - 1.00 7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PY	l /RIDOXAL PHOSPHATE	52.6 <sup>H</sup>	U/L	0.00 - 49.00
AST/ALT RATIO: S	ERUM	0.61	RATIO	0.00 - 46.00
by CALCULATED, SPE ALKALINE PHOSPI by PARA NITROPHEN PROPANOL		132.9 <sup>H</sup>	U/L	40.0 - 130.0
GAMMA GLUTAMY by szasz, spectrol	L TRANSFERASE (GGT): SERUM PHTOMETRY	118.13 <sup>H</sup>	U/L	0.00 - 55.0
TOTAL PROTEINS: by BIURET, SPECTRO		7.38	gm/dL	6.20 - 8.00
ALBUMIN: SERUM		4.34	gm/dL	3.50 - 5.50
by BROMOCRESOL G GLOBULIN: SERUN by CALCULATED, SPE	1	3.04	gm/dL	2.30 - 3.50
A : G RATIO: SERUI	M ECTROPHOTOMETRY	1.43	RATIO	1.00 - 2.00

by CALCULATED, SPECTROPHOTOMETRY

NOTE:- To be correlated in individuals having SGOT and SGPT values higher than Normal Referance Range. USE:- Differential diagnosis of diseases of hepatobiliary system and pancreas.

## **INCREASED:**

DRUG HEPATOTOXICITY	> 2
ALCOHOLIC HEPATITIS	> 2 (Highly Suggestive)
CIRRHOSIS	1.4 - 2.0
INTRAHEPATIC CHOLESTATIS	> 1.5
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)



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INTERPRETATION





	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbi Chairman & Consultant P	ology) ME	n Chopra D (Pathology) ht Pathologist
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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).

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	<b>Biological Reference interval</b>		
	KIDNE	EY FUNCTION	TEST (COMPLETE)			
UREA: SERUM by UREASE - GLUTAN	NATE DEHYDROGENASE (GLDH)	13.96	mg/dL	10.00 - 50.00		
CREATININE: SER	UM	1.09	mg/dL	0.40 - 1.40		
by ENZYMATIC, SPECTROPHOTOMETERY BLOOD UREA NITROGEN (BUN): SERUM		6.52 <sup>L</sup>	mg/dL	7.0 - 25.0		
by CALCULATED, SPE	ECTROPHOTOMETRY	0.32-				
BLOOD UREA NITH RATIO: SERUM	ROGEN (BUN)/CREATININE	5.98 <sup>L</sup>	RATIO	10.0 - 20.0		
	ECTROPHOTOMETRY					
UREA/CREATININ	E RATIO: SERUM	12.81	RATIO			
URIC ACID: SERUM		6.4	mg/dL	3.60 - 7.70		
by URICASE - OXIDAS	SE PEROXIDASE	0.70		0.50 10.00		
CALCIUM: SERUM by ARSENAZO III, SPE	ECTROPHOTOMETRY	8.76	mg/dL	8.50 - 10.60		
PHOSPHOROUS: SI		2.57	mg/dL	2.30 - 4.70		
ELECTROLYTES	DATE, SPECTROPHOTOMETRY					
SODIUM: SERUM by ISE (ION SELECTIV	/E ELECTRODE)	143.54	mmol/L	135.0 - 150.0		
POTASSIUM: SERU by ISE (ION SELECTIV	M	4.27	mmol/L	3.50 - 5.00		
CHLORIDE: SERUN by ISE (ION SELECTIV	A /E ELECTRODE)	107.66	mmol/L	90.0 - 110.0		
ESTIMATED GLON	MERULAR FILTERATION RATE					
ESTIMATED GLOM (eGFR): SERUM by CALCULATED	IERULAR FILTERATION RATE	84.8				
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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OLLECTED BY	:		RI	EG. NO./LAB NO.	:	0125012500	018		
EFERRED BY			RI	EGISTRATION DA	ATE ·	25/Jan/2025 1	10·15 AM		
ARCODE NO.	: 01524397			DLLECTION DATE					
LIENT CODE.	: KOS DIAGN			EPORTING DATE					
				EPURTING DATE	s :.	25/Jan/2025 1	12:13PM		
LIENT ADDRESS	: 6349/1, NIC	HOLSON ROAD, AMB	ALA CANTT						
Fest Name			Value	Uni	it	Biolog	gical Refe	erence in	nterval
NCREASED RATIO (>2 . Postrenal azotemia	tetracycline, gl 0:1) WITH ELEV (BUN rises disp	ATED CREATININE LEVE proportionately more t	LS:	) (e.g. obstructive	uropathy).				
Postrenal azotemia     Prerenal azotemia     CREASED RATIO (<         Acute tubular necr         Acute tubular necr         Low protein diet ar         Severe liver diseas         Other causes of de         Repeated dialysis         Inherited hyperam         SIADH (syndrome of         Pregnancy.         Phenacimide thera         Rhabdomyolysis (r         Muscular patients         NAPPROPIATE RATIO         Diabetic ketoacido         hould produce an in         Cephalosporin thei         STIMATED GLOMERL         CKD STAGE	tetracycline, gl <b>0:1) WITH ELEV</b> (BUN rises disp superimposed <b>0:1) WITH DECF</b> osis. Ind starvation. creased urea sy urea rather tha monemias (urea of inappropiate <b>0:1) WITH INCR</b> py (accelerates eleases muscle who develop re- sis (acetoaceta creased BUN/cr apy (interferes <b>ULAR FILTERATIC</b>	ucocorticoids) ATED CREATININE LEVE proportionately more to or renal disease. REASED BUN : In creatinine diffuses of a is virtually absent in antidiuretic harmone) EASED CREATININE: conversion of creatine creatinine). Inal failure. Ite causes false increas reatinine ratio). with creatinine measu IN RATE: DESCRIPTION	LS: han creatinine but of extracell blood). due to tubular to creatinine) e in creatinine rement).	ular fluid). secretion of urea. with certain meth min/1.73m2 )	hodologies	resulting in no		o when de	ehydrat
VCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet an Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients VAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin ther STIMATED GLOMERL CKD STAGE G1	tetracycline, gl 0:1) WITH ELEV (BUN rises disp superimposed 0:1) WITH DECF osis. Ind starvation. 2. creased urea sy urea rather tha monemias (urea of inappropiate 0:1) WITH INCR py (accelerates eleases muscle who develop re- sis (acetoaceta creased BUN/cr apy (interferes ULAR FILTERATIC No	Ared CREATININE LEVE proportionately more to proportionately more to prenal disease. AREASED BUN : In creatinine diffuses of a is virtually absent in antidiuretic harmone) EASED CREATININE: conversion of creatine creatinine). Inal failure. It causes false increas reatinine ratio). With creatinine measu IN RATE: DESCRIPTION rmal kidney function	LS: han creatinine but of extracell blood). due to tubular e to creatinine) e in creatinine rement). GFR ( mL/	ular fluid). secretion of urea. with certain meth <u>min/1.73m2 )</u> >90	hodologies ASSOCI	resulting in no <b>ATED FINDING</b>	S	o when de	ehydrat
VCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia VECREASED RATIO (< Acute tubular necr Low protein diet ar Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. VECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients VAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin ther STIMATED GLOMERU CKD STAGE	tetracycline, gl 0:1) WITH ELEV (BUN rises disp superimposed 0:1) WITH DECF osis. Ind starvation. 2. creased urea sy urea rather tha monemias (urea of inappropiate 0:1) WITH INCR py (accelerates eleases muscle who develop re- sis (acetoaceta creased BUN/cr apy (interferes ULAR FILTERATIO No	ucocorticoids) ATED CREATININE LEVE proportionately more to or renal disease. REASED BUN : In creatinine diffuses of a is virtually absent in antidiuretic harmone) EASED CREATININE: conversion of creatine creatinine). Inal failure. Ite causes false increas reatinine ratio). with creatinine measu IN RATE: DESCRIPTION	LS: han creatinine but of extracell blood). due to tubular e to creatinine) e in creatinine rement). GFR ( mL/	ular fluid). secretion of urea. with certain meth min/1.73m2 )	hodologies ASSOCI	resulting in no	<u>s</u>	o when de	ehydrat
ICREASED RATIO (>2 Postrenal azotemia Prerenal azotemia ECREASED RATIO (< Acute tubular necr Low protein diet ar Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. ECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients IAPPROPIATE RATIO Diabetic ketoacido nould produce an in Cephalosporin ther STIMATED GLOMERI G1 G2 G3a	tetracycline, gl 0:1) WITH ELEV (BUN rises disp superimposed 0:1) WITH DECF osis. Ind starvation. 2: creased urea sy urea rather tha monemias (urea of inappropiate 0:1) WITH INCR py (accelerates eleases muscle who develop re- sis (acetoaceta creased BUN/cr apy (interferes ULAR FILTERATIO No K No K No	Ared CREATININE LEVE proportionately more to proportionately more to prenal disease. REASED BUN : In creatinine diffuses of a is virtually absent in antidiuretic harmone) EASED CREATININE: conversion of creatine creatinine). Inal failure. Re causes false increas reatinine ratio). With creatinine measu IN RATE: DESCRIPTION IN RATE: DESCRIPTION Indley damage with iormal or high GFR ild decrease in GFR	LS: han creatinine but of extracell blood). due to tubular e to creatinine) e in creatinine rement). GFR ( mL/	ular fluid). secretion of urea. with certain meth min/1.73m2 ) >90 >90 0 -89	hodologies ASSOCI	resulting in no <b>ATED FINDING</b> proteinuria ice of Protein ,	<u>s</u>	o when de	ehydrat
VCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia VECREASED RATIO (< Acute tubular necr Low protein diet ar Severe liver diseas Other causes of de Repeated dialysis ( Inherited hyperam SIADH (syndrome of Pregnancy. VECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients VAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin ther STIMATED GLOMERI G1 G2 G3a G3b	tetracycline, gl 0:1) WITH ELEV (BUN rises disp superimposed 0:1) WITH DECF osis. Ind starvation. 2. creased urea sy urea rather tha monemias (urea f inappropiate 0:1) WITH INCR py (accelerates eleases muscle who develop re- sis (acetoaceta creased BUN/cr apy (interferes UAR FILTERATION NO K C NO K C NO MO MO MO	accorticoids) ATED CREATININE LEVE proportionately more t proportionately more t attack of the second attack of the	LS: han creatinine but of extracell blood). due to tubular e to creatinine rement). GFR ( mL/ 6 6 3	ular fluid). secretion of urea. with certain meth <u>min/1.73m2 ) &gt;90 &gt;90 0 -89 0-59</u>	hodologies ASSOCI	resulting in no <b>ATED FINDING</b> proteinuria ice of Protein ,	<u>s</u>	o when de	ehydrat
VCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia VECREASED RATIO (< Acute tubular necr Low protein diet ar Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. VECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients VAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin ther STIMATED GLOMERI G1 G2 G3a	tetracycline, gl 0:1) WITH ELEV (BUN rises disp superimposed 0:1) WITH DECF osis. Ind starvation. 2. creased urea sy urea rather tha monemias (urea f inappropiate 0:1) WITH INCR py (accelerates eleases muscle who develop re- sis (acetoaceta creased BUN/cr apy (interferes UAR FILTERATION NO K C NO K C NO MO MO MO	Ared CREATININE LEVE proportionately more to proportionately more to prenal disease. REASED BUN : In creatinine diffuses of a is virtually absent in antidiuretic harmone) EASED CREATININE: conversion of creatine creatinine). Inal failure. Re causes false increas reatinine ratio). With creatinine measu IN RATE: DESCRIPTION IN RATE: DESCRIPTION Indley damage with iormal or high GFR ild decrease in GFR	LS: han creatinine blood). due to tubular e to creatinine rement). GFR (mL/ 6 3 1	ular fluid). secretion of urea. with certain meth min/1.73m2 ) >90 >90 0 -89	hodologies ASSOCI	resulting in no <b>ATED FINDING</b> proteinuria ice of Protein ,	<u>s</u>	o when de	ehydrat





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









	Dr. Vinay Chopra MD (Pathology & Microb Chairman & Consultant I	viology) MI	m Chopra D (Pathology) nt Pathologist
NAME	: Mr. AMANDEEP SINGH		
AGE/ GENDER	: 46 YRS/MALE	PATIENT ID	: 1734528
COLLECTED BY	:	<b>REG. NO./LAB NO.</b>	: 012501250018
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 25/Jan/2025 10:15 AM
BARCODE NO.	: 01524397	<b>COLLECTION DATE</b>	: 25/Jan/2025 10:17AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	<b>REPORTING DATE</b>	: 25/Jan/2025 12:15PM
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





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

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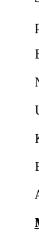


	<b>Dr. Vinay Ch</b> MD (Pathology & Chairman & Cons	Microbiology)	Dr. Yugam MD O & Consultant	(Pathology)		
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BARCODE NO.	: 01524397	COLLECTI		: 25/Jan/2025 10:17AM		
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTI	: 25/Jan/2025 11:04AM			
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	AMBALA CANTT				
Test Name		Value	Unit	<b>Biological Reference interval</b>		
		CLINICAL PATHO	LOGY			
	URINF RO	UTINE & MICROSCOP		ATION		
PHYSICAL EXAMI						
QUANTITY RECIEV	ΈD	10	ml			
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	AMBER YELLOW		PALE YELLOW		
	TANCE SPECTROPHOTOMETRY	CLEAR				
	TRANSPARANCY by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			CLEAR		
SPECIFIC GRAVITY	7	1.01		1.002 - 1.030		
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY					
REACTION		ACIDIC				
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY					
PROTEIN by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)		
SUGAR	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)		
pH	TANCE SPECTROPHOTOMETRY	6		5.0 - 7.5		
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	Negotive				
	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)		
NITRITE	TANCE SPECTROPHOTOMETRY.	Negative		NEGATIVE (-ve)		
UROBILINOGEN		Normal	EU/dL	0.2 - 1.0		
by DIP STICK/REFLEC	by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY KETONE BODIES by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY BLOOD by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY			NEGATIVE (-ve)		
				NEGATIVE (-ve)		
ASCORBIC ACID by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY		NEGATIVE (-ve)		NEGATIVE (-ve)		
MICROSCOPIC EX						
RED BLOOD CELLS	(RBCs)	NEGATIVE (-ve)	/HPF	0 - 3		
RED BLOOD CELLS	(RBCs)	NEGATIVE (-ve)	/HPF	0 - 3		





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



TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

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

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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANT	Т	
Test Name		Value	Unit	Biological Reference interval
by MICROSCOPY ON	CENTRIFUGED URINARY SEDIMENT			
PUS CELLS		2-4	/HPF	0 - 5

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	~ 1	,	0 0
EPITHELIAL CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	1-3	/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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