



	<b>Dr. Vinay Chopra</b> MD (Pathology & Micr Chairman & Consultar	obiology)	Dr. Yugam ( MD (F CEO & Consultant P	Pathology)
NAME	: Mr. JOGINDER SINGH			
AGE/ GENDER	: 70 YRS/MALE	PAT	FIENT ID	: 1738578
COLLECTED BY	:	REG	G. NO./LAB NO.	: 012501290003
REFERRED BY	:	REC	GISTRATION DATE	: 29/Jan/2025 09:19 AM
BARCODE NO.	: 01524582		LLECTION DATE	: 29/Jan/2025 09:26AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		PORTING DATE	: 29/Jan/2025 09:59AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMB/	ALA CANTT		
Test Name		Value	Unit	<b>Biological Reference interval</b>
	SWAST	THYA WELL	NESS PANEL: G	
	COMP	PLETE BLOO	D COUNT (CBC)	
RED BLOOD CELLS	S (RBCS) COUNT AND INDICES			
HAEMOGLOBIN (H	B)	15.1	gm/dL	12.0 - 17.0
by CALORIMETRIC RED BLOOD CELL (	RBC) COUNT	5.01 <sup>H</sup>	Millions/c	mm 3.50 - 5.00
by HYDRO DYNAMIC F	OCUSING, ELECTRICAL IMPEDENCE			
PACKED CELL VOLI	UME (PCV) UTOMATED HEMATOLOGY ANALYZER	45.2	%	40.0 - 54.0
MEAN CORPUSCUL	AR VOLUME (MCV)	90.2	fL	80.0 - 100.0
	UTOMATED HEMATOLOGY ANALYZER AR HAEMOGLOBIN (MCH)	30.1	pg	27.0 - 34.0
	UTOMATED HEMATOLOGY ANALYZER	22.4		22.0.20.0
	AR HEMOGLOBIN CONC. (MCHC) UTOMATED HEMATOLOGY ANALYZER	33.4	g/dL	32.0 - 36.0
	UTION WIDTH (RDW-CV) UTOMATED HEMATOLOGY ANALYZER	14.2	%	11.00 - 16.00
	UTION WIDTH (RDW-SD)	48.3	fL	35.0 - 56.0
	UTOMATED HEMATOLOGY ANALYZER	10	DATIO	
MENTZERS INDEX by CALCULATED		18	RATIO	BETA THALASSEMIA TRAIT: < 13.0
				IRON DEFICIENCY ANEMIA:
GREEN & KING INI	)FX	25.53	RATIO	>13.0 BETA THALASSEMIA TRAIT:<=
by CALCULATED		20.00	in the	65.0
				IRON DEFICIENCY ANEMIA: > 65.0
<b>WHITE BLOOD CE</b>	LLS (WBCS)			00.0
FOTAL LEUCOCYTE	E COUNT (TLC) ( by sf cube & microscopy	5850	/cmm	4000 - 11000
NUCLEATED RED E	BLOOD CELLS (nRBCS)	NIL		0.00 - 20.00
	RT HEMATOLOGY ANALYZER BLOOD CELLS (nRBCS) %	NIL	%	< 10 %
	UTOMATED HEMATOLOGY ANALYZER			





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NAME



Dr. Vinay Chopra Dr. Yugam Chopra MD (Pathology & Microbiology) MD (Pathology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** : Mr. JOGINDER SINGH **PATIENT ID** AGE/ GENDER : 70 YRS/MALE **COLLECTED BY** REG. NO./LAB NO. : **REFERRED BY REGISTRATION DATE** : **BARCODE NO.** :01524582 **COLLECTION DATE** CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT

## A <u>P</u>] PI PI Μ PI PI ΡI

MBBS, MD (PATHOLOGY & MICROBIOLOGY)

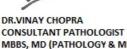
DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY)

:1738578 :012501290003 : 29/Jan/2025 09:19 AM : 29/Jan/2025 09:26AM

: 29/Jan/2025 09:59AM

Test Name	Value	Unit	<b>Biological Reference interval</b>
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS by flow cytometry by sf cube & microscopy	65	%	50 - 70
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	8	%	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	3803	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by flow cytometry by SF cube & microscopy	1462	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by flow cytometry by SF cube & microscopy	117	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	468	/cmm	80 - 880
PLATELETS AND OTHER PLATELET PREDICTIVE	MARKERS.		
PLATELET COUNT (PLT) by hydro dynamic focusing, electrical impedence	194000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.22	%	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	72000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	37.1	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by hydro dynamic focusing, electrical impedence	16.5	%	15.0 - 17.0
NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD			





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CLIENT CODE.	: KOS DIAGNOSTIC LAB		RTING DATE	: 29/Jan/2025 12:17PM
			KIING DATE	. 29/Jan/ 2023 12.17PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interva
WHOLE BLOOD	EMOGLOBIN (HbA1c):	7.8 <sup>H</sup>	%	4.0 - 6.4
ESTIMATED AVERA	GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY)	177.16 <sup>H</sup>	mg/dL	60.00 - 140.00
		DIABETES ASSOCIATION		
	REFERENCE GROUP		LATED HEMOGLOGIB (	HBAIC) in %
	abetic Adults >= 18 years		<5.7	
	t Risk (Prediabetes)		5.7 - 6.4	
A		>= 6.5		
	iagnosing Diabetes	Age > 19 Years		
	iagnosing Diabetes			
D		Goals of The	erapy:	< 7.0
D	iagnosing Diabetes ic goals for glycemic control	Goals of The Actions Sugg	ested:	< 7.0 >8.0
D			erapy: ested: Age < 19 Years	

**KOS Diagnostic Lab** 

(A Unit of KOS Healthcare)

1.Glycosylated hemoglobin (HbA1c) test is three monthly monitoring done to assess compliace with therapeutic regimen in diabetic patients. 2. Since Hb1c reflects long term fluctuations in blood glucose concentration, a diabetic patient who has recently under good control may still have high concentration of HbAlc. Converse is true for a diabetic previously under good control but now poorly controlled.

3. Target goals of < 7.0 % may be beneficial in patients with short duration of diabetes, long life expectancy and no significant cardiovascular disease. In patients with significant complications of diabetes, limited life expectancy or extensive co-morbid conditions, targetting a goal of < 7.0% may not be appropiate.

4. High HbA1c (>9.0 -9.5 %) is strongly associated with risk of development and rapid progression of microvascular and nerve complications 5. Any condition that shorten RBC life span like acute blood loss, hemolytic anemia faisely lower HbA1c results.

6.HbA1c results from patients with HbSS,HbSC and HbD must be interpreted with caution, given the pathological processes including anemia, increased red cell turnover, and transfusion requirement that adversely impact HbA1c as a marker of long-term gycemic control.

7.Specimens from patients with polycythemia or post-splenctomy may exhibit increse in HbA1c values due to a somewhat longer life span of the red cells.



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TEST PERFORMED AT KOS DIAGNOSTIC LAB. AMBALA CANTT



TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



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LIENT CODE.	: KOS DIAGNOSTIC LAB	R	EPORTING DATE	: 29/Jan/2025 11:02AM
LIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT		
fest Name		Value	Unit	<b>Biological Reference interval</b>
by RED CELL AGGRE <b>NTERPRETATION:</b> I. ESR is a non-specit mmune disease, but 2. An ESR can be affe as C-reactive protein	DIMENTATION RATE (ESR) GATION BY CAPILLARY PHOTOMETE fic test because an elevated resul does not tell the health practitic ected by other conditions besides be used to monitor disease activ	27 <sup>H</sup> It often indicates th oner exactly where the inflammation. For	the inflammation is in the this reason, the ESR is ty	hr 0 - 20



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CLIENT CODE.	: KOS DIAGNOSTIC LAB	RE	PORTING DATE	: 29/Jan/2025 11:00AM
CLIENT ADDRESS	: 6349/1, NICHOLSON R	COAD, AMBALA CANTT		
Test Name		Value	Unit	<b>Biological Reference interval</b>
	CL	INICAL CHEMISTR	Y/BIOCHEMIST	'RY
		GLUCOSE FA	STING (F)	

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 text (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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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD	, AMBALA CANTT		
Fest Name		Value	Unit	<b>Biological Reference interval</b>
		I IPID PRO	FILE : BASIC	
HOLESTEROL TO	TAL · SFRUM	147.07	mg/dL	<b>OPTIMAL:</b> < 200.0
by CHOLESTEROL OX		147.07	ling/ uL	BORDERLINE HIGH: 200.0 - 239.0
				HIGH CHOLESTEROL: > OR = 240.0
RIGLYCERIDES: S	ERUM	135.79	mg/dL	OPTIMAL: < 150.0
by GLYCEROL PHOSP	PHATE OXIDASE (ENZYMATIC)		0	BORDERLINE HIGH: 150.0 -
				199.0 HIGH: 200.0 - 499.0
				VERY HIGH: $> OR = 500.0$
	L (DIRECT): SERUM	47.04	mg/dL	LOW HDL: < 30.0
by SELECTIVE INHIBIT	ION			BORDERLINE HIGH HDL: 30.0 60.0
				HIGH HDL: $> OR = 60.0$
DL CHOLESTERO		72.87	mg/dL	OPTIMAL: < 100.0
by CALCULATED, SPE	CIROPHOIOMEIRY			ABOVE OPTIMAL: 100.0 - 129. BORDERLINE HIGH: 130.0 -
				159.0
				HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0
ION HDL CHOLEST	TEROL: SERUM	100.03	mg/dL	OPTIMAL: < 130.0
by CALCULATED, SPE		10000	ing, uz	ABOVE OPTIMAL: 130.0 - 159
				BORDERLINE HIGH: 160.0 -
				189.0 HIGH: 190.0 - 219.0
				VERY HIGH: $>$ OR = 220.0
LDL CHOLESTER(		27.16	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SER		429.93	mg/dL	350.00 - 700.00
by CALCULATED, SPE	CTROPHOTOMETRY			
CHOLESTEROL/HD by CALCULATED, SPE		3.13	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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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD	, AMBALA CANT	Т	
Test Name		Value	Unit	<b>Biological Reference interval</b>
LDL/HDL RATIO: S by CALCULATED, SPE		1.55	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	2.89 <sup>L</sup>	RATIO	3.00 - 5.00

## INTERPRETATION:

1. Measurements in the same patient can show physiological analytical variations. Three serial samples 1 week apart are recommended for Total Cholesterol, Triglycerides, HDL & LDL Cholesterol.

2. As per NLA-2014 guidelines, all adults above the age of 20 years should be screened for lipid status. Selective screening of children above the age of 2 years with a family history of premature cardiovascular disease or those with at least one parent with high total cholesterol is recommended.

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
			N TEST (COMPLETE)	
BILIRUBIN TOTAL by DIAZOTIZATION, S	: SERUM pectrophotometry	1.7 <sup>H</sup>	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
	Г (CONJUGATED): SERUM spectrophotometry	0.37	mg/dL	0.00 - 0.40
	ECT (UNCONJUGATED): SERUM	1.33 <sup>H</sup>	mg/dL	0.10 - 1.00
SGOT/AST: SERUM	I (RIDOXAL PHOSPHATE	28.5	U/L	7.00 - 45.00
SGPT/ALT: SERUM		23.4	U/L	0.00 - 49.00
AST/ALT RATIO: S		1.22	RATIO	0.00 - 46.00
ALKALINE PHOSP		107.86	U/L	40.0 - 130.0
GAMMA GLUTAMY by SZASZ, SPECTRO	L TRANSFERASE (GGT): SERUM	13.39	U/L	0.00 - 55.0
TOTAL PROTEINS: by BIURET, SPECTRO	SERUM	7.67	gm/dL	6.20 - 8.00
ALBUMIN: SERUM		4.37	gm/dL	3.50 - 5.50
GLOBULIN: SERUN		3.3	gm/dL	2.30 - 3.50
A : G RATIO: SERU		1.32	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** 





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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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SU 9001 : 2008 CERTIFIED LAB			EXCELLENCE IN HEALTHCAKE & DIAGNOSTICS		
				(Pathology)	
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Test Name		Value	Unit	Biological Reference interval	
	KIDNE	Y FUNCTION	TEST (COMPLETE)		
UREA: SERUM by UREASE - GLUTAMA	TE DEHYDROGENASE (GLDH)	34	mg/dL	10.00 - 50.00	
CREATININE: SERUM		1.44 <sup>H</sup>	mg/dL	0.40 - 1.40	
by ENZYMATIC, SPECTROPHOTOMETERY BLOOD UREA NITROGEN (BUN): SERUM		15.89	mg/dL	7.0 - 25.0	
by CALCULATED, SPECTROPHOTOMETRY					
BLOOD UREA NITROGEN (BUN)/CREATININE		11.03	RATIO	10.0 - 20.0	
RATIO: SERUM by CALCULATED, SPEC	TROPHOTOMETRY				
UREA/CREATININE		23.61	RATIO		
by CALCULATED, SPECTROPHOTOMETRY URIC ACID: SERUM		2.97 <sup>L</sup>	mg/dL	3.60 - 7.70	
by URICASE - OXIDASE PEROXIDASE					
CALCIUM: SERUM by ARSENAZO III, SPECTROPHOTOMETRY		9.13	mg/dL	8.50 - 10.60	
PHOSPHOROUS: SERUM		3.2	mg/dL	2.30 - 4.70	
by PHOSPHOMOLYBDA <u>ELECTROLYTES</u>	TE, SPECTROPHOTOMETRY				
SODIUM: SERUM		140.4	mmol/L	135.0 - 150.0	
by ISE (ION SELECTIVE ELECTRODE)			mm01/ L		
POTASSIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)		4.71	mmol/L	3.50 - 5.00	
CHLORIDE: SERUM		105.3	mmol/L	90.0 - 110.0	
by ISE (ION SELECTIVE					
	ERULAR FILTERATION RATE				
ESTIMATED GLOME (eGFR): SERUM by CALCULATED	RULAR FILTERATION RATE	52.3			
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 PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbiology) Chairman & Consultant Pathologist		Dr. Yugam Chopra MD (Pathology) CEO & Consultant Pathologist		
NAME	: Mr. JOGINDER SINGH				
AGE/ GENDER	: 70 YRS/MALE	PAT	IENT ID	: 1738578	
COLLECTED BY	:	REG	NO./LAB NO.	: 012501290003	
REFERRED BY	:	REG	ISTRATION DATE	: 29/Jan/2025 09:19 AM	
BARCODE NO.	: 01524582		LECTION DATE	: 29/Jan/2025 09:26AM	
CLIENT CODE.	: KOS DIAGNOSTIC LAB		ORTING DATE	: 29/Jan/2025 11:36AM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,			. 20/341/ 2020 11:00/14	
CLIENT ADDRESS	. 0349/1, MCHOLSON ROAD,	AMDALA CANT I			
Test Name		Value	Unit	Biological Refe	erence interval
8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia	xia, high fever). (e.g. ureter colostomy) lass (subnormal creatinine prod tetracycline, glucocorticoids) 20:1) WITH ELEVATED CREATININ a (BUN rises disproportionately r superimposed on renal disease 19:1) WITH DECEMENTS	E <b>LEVELS:</b> nore than creatinine) (e	e.g. obstructive urop	athy).	
8. Reduced muscle m 9. Certain drugs (e.g. INCREASED 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 INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin thei ESTIMATED GLOMERI OKD STAGE	(e.g. ureter colostomy) hass (subnormal creatinine prod tetracycline, glucocorticoids) (0:1) WITH ELEVATED CREATININ (BUN rises disproportionately r superimposed on renal disease (0:1) WITH DECREASED BUN : osis. nd starvation. e. creased urea synthesis. (urea rather than creatinine diff monemias (urea is virtually abso of inappropiate antidiuretic harm (urea rather than creatinine diff monemias (urea is virtually abso of inappropiate antidiuretic harm (0:1) WITH INCREASED CREATINII py (accelerates conversion of cr eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false in creased BUN/creatinine ratio). Tapy (interferes with creatinine r JLAR FILTERATION RATE: DESCRIPTION	E LEVELS: nore than creatinine) (e uses out of extracellula ent in blood). none) due to tubular se VE: eatine to creatinine). crease in creatinine wi neasurement). GFR ( mL/mi	r fluid). cretion of urea. th certain methodol n/1.73m2 ) A	ogies,resulting in normal ratio	o when dehydrat
A. Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Prerenal azotemia Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet an Severe liver diseas Other causes of de Repeated dialysis ( Repeated dialysis ( NIADH (syndrome of Pregnancy. DECREASED RATIO (< Neclar patients NAPPROPIATE RATIO Diabetic ketoacido should produce an in Cephalosporin there ESTIMATED GLOMERI CKD STAGE G1	(e.g. ureter colostomy) hass (subnormal creatinine prod tetracycline, glucocorticoids) (0:1) WITH ELEVATED CREATININ (BUN rises disproportionately r superimposed on renal disease (0:1) WITH DECREASED BUN : osis. nd starvation. e. creased urea synthesis. (urea rather than creatinine diff monemias (urea is virtually abso of inappropiate antidiuretic harm (urea rather than creatinine diff monemias (urea is virtually abso of inappropiate antidiuretic harm (0:1) WITH INCREASED CREATINII py (accelerates conversion of cr eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false in creased BUN/creatinine ratio). Tapy (interferes with creatinine r JLAR FILTERATION RATE: DESCRIPTION Normal kidney fund	E LEVELS: nore than creatinine) (e uses out of extracellula ent in blood). none) due to tubular se VE: eatine to creatinine). crease in creatinine wi neasurement). GFR (mL/mi tion >9	r fluid). cretion of urea. th certain methodol <u>n/1.73m2 ) A</u>	ogies,resulting in normal ratio SOCIATED FINDINGS No proteinuria	o when dehydrat
A Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Prerenal 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 (< Nuscular patients Muscular patients Muscular patients MappROPIATE RATIO Diabetic ketoacido should produce an in Cephalosporin their ESTIMATED GLOMERI OKD STAGE	(e.g. ureter colostomy) hass (subnormal creatinine prod tetracycline, glucocorticoids) (0:1) WITH ELEVATED CREATININ (BUN rises disproportionately r superimposed on renal disease (0:1) WITH DECREASED BUN : osis. nd starvation. e. creased urea synthesis. (urea rather than creatinine diff monemias (urea is virtually abso of inappropiate antidiuretic harm (urea rather than creatinine diff monemias (urea is virtually abso of inappropiate antidiuretic harm (urea rather than creatinine diff monemias (urea is virtually abso of inappropiate antidiuretic harm (urea rather than creatinine diff monemias (urea is virtually abso of inappropiate antidiuretic harm (urea rather than creatinine diff monemias (urea is virtually abso of inappropiate antidiuretic harm (urea rather than creatinine diff monemias (urea is virtually abso of inappropiate antidiuretic harm (urea rather than creatinine), who develop renal failure. : sis (acetoacetate causes false in creased BUN/creatinine ratio). Tapy (interferes with creatinine r JLAR FILTERATION RATE: DESCRIPTION Normal kidney function Kidney damage w	E LEVELS: nore than creatinine) (e uses out of extracellula ent in blood). none) due to tubular se VE: eatine to creatinine). crease in creatinine wi neasurement). GFR (mL/mi tion >9 th >9	r fluid). cretion of urea. th certain methodol n/1.73m2 ) As 0 F	ogies,resulting in normal ratio SOCIATED FINDINGS No proteinuria resence of Protein ,	o when dehydrat
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8. Reduced muscle m 9. Certain drugs (e.g. INCREASED 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 INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERI CKD STAGE G1 G2 G3a	(e.g. ureter colostomy)         ass (subnormal creatinine prod         tetracycline, glucocorticoids)         co:1) WITH ELEVATED CREATININ         a (BUN rises disproportionately r         superimposed on renal disease         to:1) WITH DECREASED BUN :         osis.         nd starvation.         e.         creased urea synthesis.         (urea rather than creatinine diff         monemias (urea is virtually absoft inappropiate antidiuretic harm         to:1) WITH INCREASED CREATINII         py (accelerates conversion of createleases muscle creatinine).         who develop renal failure.         :         sis (acetoacetate causes false in creased BUN/creatinine ratio).         rapy (interferes with creatinine ratio).         rapy (interferes with creatinine ratio).         app (interferes with creatinine ratio).         may (interferes with creatinine ratio).         may (interferes with creatinine ratio).         may (interferes with creatinine ratio).         app (and kidney function)         Normal kidney function)         Normal or high G         Mild decrease in C	E LEVELS: nore than creatinine) (e uses out of extracellula ent in blood). none) due to tubular se VE: eatine to creatinine). Crease in creatinine wi neasurement). GFR (mL/mi tion >9 th >9 FR 60 - n GFR 30-	r fluid). cretion of urea. th certain methodol n/1.73m2 ) A: 0 6 0 7 Alt 89 59 6 29	ogies,resulting in normal ratio SOCIATED FINDINGS No proteinuria resence of Protein ,	o when dehydrat



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	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbiology) Chairman & Consultant Pathologi		(Pathology)
NAME	: Mr. JOGINDER SINGH		
AGE/ GENDER	: 70 YRS/MALE	PATIENT ID	: 1738578
COLLECTED BY	:	REG. NO./LAB NO.	: 012501290003
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 29/Jan/2025 09:19 AM
BARCODE NO.	: 01524582	COLLECTION DATE	: 29/Jan/2025 09:26AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	<b>REPORTING DATE</b>	: 29/Jan/2025 11:36AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANT	Г	
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

End Of Report \*\*\*





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