



	Dr. Vinay Chopra MD (Pathology & Micr Chairman & Consultar	obiology)	ME	m Chopra D (Pathology) nt Pathologist
IAME	: Mr. DILPREET SINGH			
GE/ GENDER	: 25 YRS/MALE		PATIENT ID	: 1722673
OLLECTED BY	:		REG. NO./LAB NO.	: 012503050016
REFERRED BY	:		<b>REGISTRATION DATE</b>	: 05/Mar/2025 09:56 AM
ARCODE NO.	: 01526490		COLLECTION DATE	:05/Mar/202509:56AM
LIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 05/Mar/2025 10:39AM
LIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMB.	ALA CANT'	r	
Fest Name		Value	Unit	Biological Reference interva
	SWAST	THYA W	ELLNESS PANEL: I	D
	COMP	PLETE BI	LOOD COUNT (CBC)	
ED BLOOD CELLS	S (RBCS) COUNT AND INDICES			
IAEMOGLOBIN (H by Calorimetric	B)	16.2	gm/dL	12.0 - 17.0
ED BLOOD CELL (	(RBC) COUNT	5.4 <sup>H</sup>	Millions	s/cmm 3.50 - 5.00
-	FOCUSING, ELECTRICAL IMPEDENCE	49	%	40.0 - 54.0
ACKED CELL VOL	AUTOMATED HEMATOLOGY ANALYZER	49	70	40.0 - 54.0
	AR VOLUME (MCV) AUTOMATED HEMATOLOGY ANALYZER	90.7	fL	80.0 - 100.0
IEAN CORPUSCUL	AR HAEMOGLOBIN (MCH)	30	pg	27.0 - 34.0
	AUTOMATED HEMATOLOGY ANALYZER AR HEMOGLOBIN CONC. (MCHC)	33.1	g/dL	32.0 - 36.0
by CALCULATED BY A	UTOMATED HEMATOLOGY ANALYZER		Ŭ	
	UTION WIDTH (RDW-CV)	13.8	%	11.00 - 16.00
	UTION WIDTH (RDW-SD)	47	fL	35.0 - 56.0
IENTZERS INDEX	AUTOMATED HEMATOLOGY ANALYZER	16.8	RATIO	BETA THALASSEMIA TRAIT
by CALCULATED				13.0
				IRON DEFICIENCY ANEMIA >13.0
		23.18	RATIO	BETA THALASSEMIA TRAIT
REEN & KING INI	DEX	20.10		05.0
REEN & KING INI	DEX	23.10		65.0 IDON DEFICIENCY ANEMIA
	DEX	23.10		65.0 IRON DEFICIENCY ANEMIA 65.0
by CALCULATED		23.16		IRON DEFICIENCY ANEMIA
by CALCULATED NHITE BLOOD CE COTAL LEUCOCYTI	<u>lls (wbcs)</u> E count (tlc)	4790	/cmm	IRON DEFICIENCY ANEMIA
by CALCULATED <b>WHITE BLOOD CE</b> COTAL LEUCOCYTH by FLOW CYTOMETR	LLS (WBCS)	4790	/cmm	IRON DEFICIENCY ANEMIA 65.0
<b>WHITE BLOOD CE</b> FOTAL LEUCOCYTI by flow cytometr NUCLEATED RED E by AUTOMATED 6 PA	<b>LLS (WBCS)</b> E COUNT (TLC) y by sf cube & microscopy		/cmm %	IRON DEFICIENCY ANEMIA 65.0 4000 - 11000





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

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







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

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

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Test Name	Value	Unit	<b>Biological Reference interval</b>
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	56	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	28	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	8 <sup>H</sup>	%	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	2682	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1341	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	383	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	383	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110
ABSOLUTE IMMATURE GRANULOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0.0 - 999.0
PLATELETS AND OTHER PLATELET PREDICTIVE	MARKERS.		
PLATELET COUNT (PLT) by hydro dynamic focusing, electrical impedence	238000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by hydro dynamic focusing, electrical impedence	0.26	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by hydro dynamic focusing, electrical impedence	11	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	77000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	32.3	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by hydro dynamic focusing, electrical impedence	16.4	%	15.0 - 17.0



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Test Name	Value	Unit	Biological Reference interval

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD



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MBBS, MD (PATHOLOGY)





TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



	Dr. Vinay Chopra MD (Pathology & Micro Chairman & Consultant			Dr. Yugan MD CEO & Consultant	(Pathology)
AME	: Mr. DILPREET	SINGH			
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RCODE NO.	:01526490		CO	<b>LLECTION DATE</b>	:05/Mar/202509:56AM
IENT CODE.	: KOS DIAGNOST	IC LAB	RI	EPORTING DATE	:05/Mar/2025 11:18AM
IENT ADDRESS	: 6349/1, NICHO	LSON ROAD, AMBA	LA CANTT		
est Name			Value	Unit	Biological Reference interval
by RED CELL AGGRE ITERPRETATION: ESR is a non-speci- nmune disease, but An ESR can be affe s C-reactive protein	does not tell the he cted by other condi	TE (ESR) у рнотометку elevated result ofter ealth practitioner ex tions besides inflam	7 n indicates the actly where the nmation. For t	he inflammation is in the his reason, the ESR is ty	hr 0 - 20 ion associated with infection, cancer and auto body or what is causing it. pically used in conjunction with other test such
by RED CELL AGGRE ITERPRETATION: ESR is a non-speci- imune disease, but An ESR can be affe c -reactive protein This test may also stemic lupus eryth DNDITION WITH LO low ESR can be see olycythaemia), sig s sickle cells in sick OTE: ESR and C - reactiv Generally, ESR doo	GATION BY CAPILLAR ic test because an e does not tell the he icted by other condi be used to monitor ematosus W ESR in with conditions th ificantly high white e cell anaemia) also e protein (C-RP) are as not change as rap	TE (ESR) Y PHOTOMETRY elevated result ofter ealth practitioner ex- tions besides inflam disease activity and the blood cell count (He blood cell co	7 n indicates the actly where the mation. For t d response to hal sedimentate eucocytosis), flammation. ther at the sta	mm/1st presence of inflammat he inflammation is in the his reason, the ESR is ty therapy in both of the a ion of red blood cells, s	hr 0 - 20 ion associated with infection, cancer and auto body or what is causing it. pically used in conjunction with other test such bove diseases as well as some others, such as uch as a high red blood cell count rmalities. Some changes in red cell shape (such is it resolves.





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	MD (	<b>Vinay Chopra</b> Pathology & Microbiology) man & Consultant Pathologist	Dr. Yugam MD CEO & Consultant	(Pathology)
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CLIENT CODE.	: KOS DIAGNOSTIC	LAB RI	EPORTING DATE	:05/Mar/2025 12:22PM
CLIENT ADDRESS	: 6349/1, NICHOLS	ON ROAD, AMBALA CANTT		
Test Name		Value	Unit	<b>Biological Reference interval</b>
		CLINICAL CHEMIST	RY/BIOCHEMIST	'RY
		GLUCOSE FA	ASTING (F)	
GLUCOSE FASTING	G (F): PLASMA Se - peroxidase (god-f	90.24 POD)	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0

**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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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT		
Test Name		Value	Unit	<b>Biological Reference interval</b>
		LIPID PRO	FILE : BASIC	
CHOLESTEROL TO	TAL: SERUM	175.17	mg/dL	<b>OPTIMAL:</b> < 200.0
by CHOLESTEROL O			ing, al	BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR =
TRIGLYCERIDES: S	ERUM PHATE OXIDASE (ENZYMATIC)	116.7	mg/dL	240.0 OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 -
				199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTERO	L (DIRECT): SERUM 70N	78.34	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0
LDL CHOLESTERO		73.49	mg/dL	HIGH HDL: > OR = 60.0 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 CHOLES by Calculated, spe		96.83	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		23.34	mg/dL	0.00 - 45.00
by CALCULATED, SPE TOTAL LIPIDS: SEE by CALCULATED, SPE	RUM	467.04	mg/dL	350.00 - 700.00
CHOLESTEROL/HI by CALCULATED, SPE		2.24	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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		<b>hopra</b> & Microbiology) onsultant Patholog		(Pathology)
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Test Name		Value	Unit	<b>Biological Reference interval</b>
LDL/HDL RATIO: S		0.94	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	1.49 <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.

 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>
	LIVER	FUNCTION	TEST (COMPLETE)	
BILIRUBIN TOTAL	: SERUM PECTROPHOTOMETRY	2.33 <sup>H</sup>	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
	Г (CONJUGATED): SERUM SPECTROPHOTOMETRY	0.46 <sup>H</sup>	mg/dL	0.00 - 0.40
BILIRUBIN INDIRE	ECT (UNCONJUGATED): SERUM	1.87 <sup>H</sup>	mg/dL	0.10 - 1.00
SGOT/AST: SERUM	[ /RIDOXAL PHOSPHATE	19.6	U/L	7.00 - 45.00
SGPT/ALT: SERUM		32.6	U/L	0.00 - 49.00
AST/ALT RATIO: S		0.6	RATIO	0.00 - 46.00
ALKALINE PHOSPI by PARA NITROPHEN PROPANOL	HATASE: SERUM IYL PHOSPHATASE BY AMINO METHYL	96.23	U/L	40.0 - 130.0
GAMMA GLUTAMY by SZASZ, SPECTRO	L TRANSFERASE (GGT): SERUM	24.59	U/L	0.00 - 55.0
TOTAL PROTEINS: by BIURET, SPECTRO		6.67	gm/dL	6.20 - 8.00
ALBUMIN: SERUM		4.25	gm/dL	3.50 - 5.50
GLOBULIN: SERUN by CALCULATED, SPE	Л	2.42	gm/dL	2.30 - 3.50
A : G RATIO: SERU		1.76	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)





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Test Name		Value Unit	Biological Reference interval

## DECREASED:

1. Acute Hepatitis due to virus, drugs, toxins (with AST increased 3 to 10 times upper limit of normal)

2. Extra Hepatic cholestatis: 0.8 (normal or slightly decreased).

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



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







	Dr. Vinay Choj MD (Pathology & M		Dr. Yugam	Chopra Pathology)
	Chairman & Consul		CEO & Consultant	
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Test Name		Value	Unit	Biological Reference interval
1 est Maine		Value	Unit	biological Reference interval
	KIDNE	Y FUNCTION	TEST (COMPLETE)	
UREA: SERUM		28.9	mg/dL	10.00 - 50.00
•	MATE DEHYDROGENASE (GLDH)	1.00		0.40 1.40
CREATININE: SERU		1.09	mg/dL	0.40 - 1.40
BLOOD UREA NITR by CALCULATED, SPE	ROGEN (BUN): SERUM	13.5	mg/dL	7.0 - 25.0
	ROGEN (BUN)/CREATININE	12.39	RATIO	10.0 - 20.0
RATIO: SERUM by CALCULATED, SPE	CTROPHOTOMETRY			
UREA/CREATININ		26.51	RATIO	
by CALCULATED, SPE		450		3 60 7 70
URIC ACID: SERUM by URICASE - OXIDAS		4.53	mg/dL	3.60 - 7.70
CALCIUM: SERUM by ARSENAZO III, SPE		10.4	mg/dL	8.50 - 10.60
PHOSPHOROUS: SE		2.63	mg/dL	2.30 - 4.70
	DATE, SPECTROPHOTOMETRY		J. J	
<u>ELECTROLYTES</u> SODIUM: SERUM		138.6	mmol/L	135.0 - 150.0
by ISE (ION SELECTIV	E ELECTRODE)	138.0	IIIII01/ L	133.0 - 130.0
POTASSIUM: SERU		3.99	mmol/L	3.50 - 5.00
by ISE (ION SELECTIV CHLORIDE: SERUM		103.95	mmol/L	90.0 - 110.0
by ISE (ION SELECTIV	(E ELECTRODE)			
	<b>IERULAR FILTERATION RATE</b>	0.0.0		
ESTIMATED GLOM (eGFR): SERUM	ERULAR FILTERATION RATE	96.6		
by CALCULATED				
INTERPRETATION:				

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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			PORTING DATE	: 05/ Mar/ 2025 01:4	ZZPIVI
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD	, AMDALA CANTT			
Test Name		Value	Unit	Biologica	al Reference interval
7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. <b>INCREASED RATIO (&gt;2</b> 1. Postrenal azotemia 2. Prerenal azotemia	xia, high fever). (e.g. ureter colostomy) ass (subnormal creatinine proc tetracycline, glucocorticoids) <b>0:1) WITH ELEVATED CREATININ</b> (BUN rises disproportionately superimposed on renal disease	<b>E LEVELS:</b> more than creatinine)	(e.g. obstructive uro	pathy).	
7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (<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. DECREASED RATIO (<1 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther	(e.g. ureter colostomy) ass (subnormal creatinine proc tetracycline, glucocorticoids) <b>0:1) WITH ELEVATED CREATININ</b> (BUN rises disproportionately superimposed on renal disease <b>0:1) WITH DECREASED BUN :</b> osis. id starvation. e. creased urea synthesis. urea rather than creatinine diff monemias (urea is virtually abs of inappropiate antidiuretic harn <b>0:1) WITH INCREASED CREATINI</b> py (accelerates conversion of c eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false i creased BUN/creatinine ratio). apy (interferes with creatinine <b>ULAR FILTERATION RATE:</b> <u>DESCRIPTION Normal kidney fun</u> Kidney damage w	Tuses out of extracelle ent in blood). none) due to tubular NE: reatine to creatinine) ncrease in creatinine measurement). GFR (mL/ ction	ular fluid). secretion of urea. with certain methodo <u>min/1.73m2 )</u>	blogies,resulting in norm ASSOCIATED FINDINGS No proteinuria Presence of Protein ,	nal ratio when dehydratio
7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. <b>INCREASED 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 of 8. Pregnancy. <b>DECREASED RATIO (</b> <1 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients <b>INAPPROPIATE RATIO</b> 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther <b>ESTIMATED GLOMERL</b> <u>G1</u> <u>G2</u>	(e.g. ureter colostomy) ass (subnormal creatinine proc tetracycline, glucocorticoids) <b>0:1) WITH ELEVATED CREATININ</b> (BUN rises disproportionately superimposed on renal disease <b>0:1) WITH DECREASED BUN :</b> osis. d starvation. e. creased urea synthesis. urea rather than creatinine diff monemias (urea is virtually abs of inappropiate antidiuretic harn <b>0:1) WITH INCREASED CREATINI</b> py (accelerates conversion of c eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false i creased BUN/creatinine ratio). apy (interferes with creatinine <b>ILAR FILTERATION RATE:</b> <u>DESCRIPTION</u> <u>Normal kidney fun</u> Kidney damage w normal or high G	IE LEVELS:         more than creatinine;         fuses out of extracelluent in blood).         none) due to tubular         NE:         reatine to creatinine)         ncrease in creatinine         measurement).         GFR (mL/         ith         FR	ular fluid). secretion of urea. with certain methodo <u>min/1.73m2 )</u>	ologies,resulting in norm ASSOCIATED FINDINGS No proteinuria	nal ratio when dehydratio
7. Urine reabsorption 3. Reduced muscle m 4. Certain drugs (e.g. <b>INCREASED RATIO (&gt;2</b> 1. Postrenal azotemia 2. Prerenal 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 of 8. Pregnancy. <b>DECREASED RATIO (</b> <1 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients <b>INAPPROPIATE RATIO</b> 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther <b>ESTIMATED GLOMERL</b> G1 G2 G3 G3a	(e.g. ureter colostomy) ass (subnormal creatinine proc tetracycline, glucocorticoids) <b>0:1) WITH ELEVATED CREATININ</b> (BUN rises disproportionately superimposed on renal disease <b>0:1) WITH DECREASED BUN :</b> osis. ad starvation. e. creased urea synthesis. urea rather than creatinine diff monemias (urea is virtually abs of inappropiate antidiuretic harm <b>0:1) WITH INCREASED CREATINI</b> py (accelerates conversion of c eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false i creased BUN/creatinine ratio). apy (interferes with creatinine <b>ILAR FILTERATION RATE:</b> DESCRIPTION Normal kidney fun Kidney damage w normal or high G Mild decrease in 0	IE LEVELS:         more than creatinine;         fuses out of extracelluent in blood).         none) due to tubular         NE:         reatine to creatinine;         measurement).         GFR (mL/         ith         FR         GFR	ular fluid). secretion of urea. with certain methodo <u>min/1.73m2 ) / / / / / / / / / / / / / / / / / / </u>	blogies,resulting in norm ASSOCIATED FINDINGS No proteinuria Presence of Protein ,	nal ratio when dehydratio
7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. <b>INCREASED 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 of 8. Pregnancy. <b>DECREASED RATIO (</b> <1 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients <b>INAPPROPIATE RATIO</b> 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther <b>ESTIMATED GLOMERL</b> <u>G1</u> <u>G2</u>	(e.g. ureter colostomy) ass (subnormal creatinine proc tetracycline, glucocorticoids) <b>0:1) WITH ELEVATED CREATININ</b> (BUN rises disproportionately superimposed on renal disease <b>0:1) WITH DECREASED BUN :</b> osis. d starvation. e. creased urea synthesis. urea rather than creatinine diff monemias (urea is virtually abs of inappropiate antidiuretic harn <b>0:1) WITH INCREASED CREATINI</b> py (accelerates conversion of c eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false i creased BUN/creatinine ratio). apy (interferes with creatinine <b>ILAR FILTERATION RATE:</b> <u>DESCRIPTION</u> <u>Normal kidney fun</u> Kidney damage w normal or high G	IE LEVELS:         more than creatinine;         fuses out of extracellue         ent in blood).         none) due to tubular         NE:         reatine to creatinine;         measurement).         Ction         ith         FR         GFR       6         n GFR       3	ular fluid). secretion of urea. with certain methodo <u>min/1.73m2 )</u>	blogies,resulting in norm ASSOCIATED FINDINGS No proteinuria Presence of Protein ,	nal ratio when dehydratic





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

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







AGE/ GENDER         : 25 YRS/MALE         PATIENT ID         : 1722673           COLLECTED BY         :         REG. NO./LAB NO.         : 012503050016           REFERRED BY         :         REGISTRATION DATE         : 05/Mar/2025 09:56 AM
AGE/ GENDER: 25 YRS/MALEPATIENT ID: 1722673
NAME : Mr. DILPREET SINGH

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



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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



KOS Diagnostic Lab (A Unit of KOS Healthcare)

	Dr. Vinay Chop MD (Pathology & Mid Chairman & Consulta	crobiology)	Dr. Yugam MD CEO & Consultant	(Pathology)
NAME	: Mr. DILPREET SINGH	•		
AGE/ GENDER	: 25 YRS/MALE	PATIE	NT ID	: 1722673
COLLECTED BY			0./LAB NO.	: 012503050016
REFERRED BY	· ·		<b>FRATION DATE</b>	: 05/Mar/2025 09:56 AM
BARCODE NO.	: 01526490		CTION DATE	: 05/Mar/2025 09:56AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		TING DATE	: 05/Mar/2025 12:22PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMI		IIII DAIL	
Test Name		Value	Unit	<b>Biological Reference interval</b>
		ENDOCRINO	LOGY	
		TESTOSTERONE		
TESTOSTERONE - T		6.09	ng/mL	0.47 - 9.80
	ESCENT MICROPARTICLE IMMUNOASSA		iig/ iiiL	0.47 - 0.00
4.The total testostero CLINIC USE: 1.Assesment of testic	e Hyperplasia lisease ales)	SHBG levels are affec	ted by medication,	disease, sex steroids and insulin.
	the	Ghops	2	



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

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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



KOS Diagnostic Lab (A Unit of KOS Healthcare)

NAME       : Mr. DILPREET SING         AGE/ GENDER       : 25 YRS/MALE         COLLECTED BY       :         REFERRED BY       :         BARCODE NO.       : 01526490         CLIENT CODE.       : KOS DIAGNOSTIC L/         CLIENT CODE.       : 6349/1, NICHOLSOI         Test Name	AB N ROAD, AMBALA ( Vah VITAMIN D/2 : SERUM 25.; < 20	REGIST COLLEG REPOR CANTT ue VITAMIN 25 HYDROX	0./LAB NO. TRATION DATE CTION DATE TING DATE Unit	DEFICIENC INSUFFICIE	AM PM Reference interval Y: < 20.0
COLLECTED BY : REFERRED BY : BARCODE NO. : 01526490 CLIENT CODE. : KOS DIAGNOSTIC LA CLIENT ADDRESS : 6349/1, NICHOLSO Test Name VITAMIN D (25-HYDROXY VITAMIN D3): by CLIA (CHEMILUMINESCENCE IMMUNOASSAY) INTERPRETATION: DEFICIENT: PREFFERED RANGE: INTOXICATION: 1.Vitamin D compounds are derived from die conversion of 7- dihydrocholecalciferol to Vi 2.25-OHVitamin D represents the main bod tissue and tightly bound by a transport prote 3.Vitamin D plays a primary role in the main phosphate reabsorption, skeletal calcium de 4.Severe deficiency may lead to failure to min DECREASED: 1.Lack of sunshine exposure. 2.Inadequate intake, malabsorption (celiac of 3.Depressed Hepatic Vitamin D 25- hydroxyls 4.Secondary to advanced Liver disease	N ROAD, AMBALA C Vah VITAMIN D/2 SERUM 25. < 20	REG. N REGIST COLLE REPOR CANTT UE VITAMIN 25 HYDROX	0./LAB NO. CRATION DATE CTION DATE TING DATE Unit	: 012503050016 : 05/Mar/2025 09:56 : 05/Mar/2025 09:56/ : 05/Mar/2025 12:221 Biological F DEFICIENC INSUFFICIE	AM PM Reference interval Y: < 20.0
REFERRED BY       :         BARCODE NO.       : 01526490         CLIENT CODE.       : KOS DIAGNOSTIC LA         CLIENT ADDRESS       : 6349/1, NICHOLSON         Test Name	N ROAD, AMBALA C Vah VITAMIN D/2 SERUM 25. < 20	REGIST COLLEG REPOR CANTT ue VITAMIN 25 HYDROX	TRATION DATE CTION DATE TING DATE Unit	: 05/Mar/2025 09:56 : 05/Mar/2025 09:56 : 05/Mar/2025 12:22 Biological F DEFICIENC INSUFFICIE	AM PM Reference interval Y: < 20.0
REFERRED BY       :         BARCODE NO.       : 01526490         CLIENT CODE.       : KOS DIAGNOSTIC LA         CLIENT ADDRESS       : 6349/1, NICHOLSON         Test Name	N ROAD, AMBALA C Vah VITAMIN D/2 SERUM 25. < 20	REGIST COLLEG REPOR CANTT ue VITAMIN 25 HYDROX	TRATION DATE CTION DATE TING DATE Unit	: 05/Mar/2025 09:56 : 05/Mar/2025 09:56 : 05/Mar/2025 12:22 Biological F DEFICIENC INSUFFICIE	AM PM Reference interval Y: < 20.0
BARCODE NO. : 01526490 CLIENT CODE. : KOS DIAGNOSTIC LA CLIENT ADDRESS : 6349/1, NICHOLSO Test Name VITAMIN D (25-HYDROXY VITAMIN D3): by CLIA (CHEMILUMINESCENCE IMMUNOASSAY) MIERPRETATION: DEFICIENT: PREFFERED RANGE: INTOXICATION: 1. Vitamin D compounds are derived from die conversion of 7- dihydrocholecalciferol to Vi 2.25-OHVitamin D represents the main bod conversion of 7- dihydrocholecalciferol to vi issue and tightly bound by a transport prote 3. Vitamin D plays a primary role in the main boosphate reabsorption, skeletal calcium de 4. Severe deficiency may lead to failure to min DECREASED: 1. Lack of sunshine exposure. 2. Inadequate intake, malabsorption (celiac of 3. Depressed Hepatic Vitamin D 25- hydroxyls 4. Secondary to advanced Liver disease	N ROAD, AMBALA C Vah VITAMIN D/2 SERUM 25. < 20	COLLEG REPOR CANTT ue VITAMIN 25 HYDROX	CTION DATE TING DATE Unit S CY VITAMIN D3	: 05/Mar/2025 09:56/ : 05/Mar/2025 12:22I Biological F DEFICIENC INSUFFICIE	AM PM Reference interval Y: < 20.0
CLIENT CODE. : KOS DIAGNOSTIC LA CLIENT ADDRESS : 6349/1, NICHOLSO Test Name  VITAMIN D (25-HYDROXY VITAMIN D3): by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)  NITERPRETATION:  DEFICIENT: INSUFFICIENT: PREFFERED RANGE: INTOXICATION:  1.Vitamin D compounds are derived from dia conversion of 7- dihydrocholecalciferol to Vi 2.25-OHVitamin D represents the main bod issue and tightly bound by a transport proto issue and tightly bo	N ROAD, AMBALA C Vah VITAMIN D/2 SERUM 25. < 20	REPOR CANTT ue VITAMIN 25 HYDROX	Unit Unit S CY VITAMIN D3	: 05/Mar/2025 12:221 Biological F DEFICIENC INSUFFICIE	PM Reference interval Y: < 20.0
CLIENT ADDRESS       : 6349/1, NICHOLSOI         Fest Name	N ROAD, AMBALA C Vah VITAMIN D/2 SERUM 25. < 20	LANTT ue VITAMIN 25 HYDROX	Unit S XY VITAMIN D3	<b>Biological F</b> DEFICIENC INSUFFICIE	<b>Reference interval</b> Y: < 20.0
Test Name         VITAMIN D (25-HYDROXY VITAMIN D3):         by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)         INTERPRETATION:         DEFICIENT:         INSUFFICIENT:         PREFFERED RANGE:         INTOXICATION:         1. Vitamin D compounds are derived from die         conversion of 7- dihvdrocholecalciferol to Vi         1. Vitamin D compounds are derived from die         conversion of 7- dihvdrocholecalciferol to Vi         1. Vitamin D plays a primary role in the main bod         cissue and tightly bound by a transport prote         3. Vitamin D plays a primary role in the main         boosphate reabsorption, skeletal calcium de         4. Severe deficiency may lead to failure to mi         DECREASED:         1. Lack of sunshine exposure.         2. Inadequate intake, malabsorption (celiac of         3. Depressed Hepatic Vitamin D 25- hvdroxyli         4. Secondary to advanced Liver disease	Vah VITAMIN D/2 : SERUM 25.: < 20	<sup>ue</sup> VITAMIN 25 HYDROX	S KY VITAMIN D3	DEFICIENC INSUFFICIE	Y: < 20.0
VITAMIN D (25-HYDROXY VITAMIN D3): by CLIA (CHEMILUMINESCENCE IMMUNOASSAY) <u>NTERPRETATION:</u> <u>DEFICIENT:</u> <u>INSUFFICIENT:</u> <u>PREFFERED RANGE:</u> <u>INTOXICATION:</u> 1. Vitamin D compounds are derived from die conversion of 7- dihydrocholecalciferol to Vi 2.25-OHVitamin D represents the main bod conversion of 7- dihydrocholecalciferol to Vi 2.25-OHVitamin D represents the main bod 3. Vitamin D plays a primary role in the main boosphate reabsorption, skeletal calcium de 4. Severe deficiency may lead to failure to mi DECREASED: 1. Lack of sunshine exposure. 2. Inadequate intake, malabsorption (celiac of 3. Depressed Hepatic Vitamin D 25- hydroxyls 4. Secondary to advanced Liver disease	<b>VITAMIN D/</b> SERUM <b>25.</b> < 20	VITAMIN 25 HYDROX	S KY VITAMIN D3	DEFICIENC INSUFFICIE	Y: < 20.0
by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)  INTERPRETATION: DEFICIENT: INSUFFICIENT: PREFFERED RANGE: INTOXICATION: I.Vitamin D compounds are derived from die conversion of 7- dihvdrocholecalciferol to Vi conversion of 7- dihvdrocholecalciferol conversion of 2- dihvdrocholecalciferol conversion of 2- hvdrocholecalciferol conversion of 2- hvdrocholecalciferol conversion of 2- hvdrocholecalciferol conversion of 2- hvdrocholecalciferol conversion of 2- hvdrocholecalc	: SERUM <b>25.</b> : < 20	25 HYDROX	XY VITAMIN D3	INSUFFICIE	
by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)         NTERPRETATION:         DEFICIENT:         INSUFFICIENT:         PREFFERED RANGE:         INTOXICATION:         .Vitamin D compounds are derived from die onversion of 7- dihvdrocholecalciferol to Vi         .25-OHVitamin D represents the main bod issue and tightly bound by a transport protein the main hosphate reabsorption, skeletal calcium de .Severe deficiency may lead to failure to mistack of sunshine exposure.         .Lack of sunshine exposure.         .Inadequate intake, malabsorption (celiac of .Depressed Hepatic Vitamin D 25- hvdroxyli: .Secondary to advanced Liver disease	: SERUM <b>25.</b> : < 20	25 HYDROX	XY VITAMIN D3	INSUFFICIE	
by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)         NTERPRETATION:         DEFICIENT:         INSUFFICIENT:         PREFFERED RANGE:         INTOXICATION:         .Vitamin D compounds are derived from die         onversion of 7- dihvdrocholecalciferol to Vi         .25-OHVitamin D represents the main bod         issue and tightly bound by a transport protein the main         .Vitamin D plays a primary role in the main         hosphate reabsorption, skeletal calcium de         .Severe deficiency may lead to failure to mi         ECREASED:         .ladequate intake, malabsorption (celiac of Depressed Hepatic Vitamin D 25- hvdroxylis)         .Secondary to advanced Liver disease	: SERUM <b>25.</b> : < 20			INSUFFICIE	
DEFICIENT: INSUFFICIENT: PREFFERED RANGE: INTOXICATION: .Vitamin D compounds are derived from die onversion of 7- dihvdrocholecalciferol to Vi .25-OHVitamin D represents the main bod issue and tightly bound by a transport prote .Vitamin D plays a primary role in the main hosphate reabsorption, skeletal calcium de .Severe deficiency may lead to failure to mi ECREASED: .Lack of sunshine exposure. .Inadequate intake, malabsorption (celiac of .Depressed Hepatic Vitamin D 25- hvdroxyl: .Secondary to advanced Liver disease				TOXICITY: 2	ENCY: 20.0 - 30.0 CY: 30.0 - 100.0 > 100.0
INSUFFICIENT: PREFFERED RANGE: INTOXICATION: .Vitamin D compounds are derived from die onversion of 7- dihvdrocholecalciferol to Vi .25-OHVitamin D represents the main bod ssue and tightly bound by a transport prote .Vitamin D plays a primary role in the main hosphate reabsorption, skeletal calcium de .Severe deficiency may lead to failure to mi ECREASED: .Lack of sunshine exposure. .Inadequate intake, malabsorption (celiac of .Depressed Hepatic Vitamin D 25- hydroxyl; .Secondary to advanced Liver disease			-		
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onversion of 7- dihvdrocholecalciferol to Vi .25-OHVitamin D represents the main bod ssue and tightly bound by a transport prote .Vitamin D plays a primary role in the main hosphate reabsorption, skeletal calcium de .Severe deficiency may lead to failure to mi <b>ECREASED:</b> .Lack of sunshine exposure. .Inadeguate intake, malabsorption (celiac of Depressed Hepatic Vitamin D 25- hydroxyl: .Secondary to advanced Liver disease	> 100		ng/n		
5. Osteoporosis and Secondary Hyperparathr b.Enzyme Inducing drugs: anti-epileptic drug NCREASED: I. Hypervitaminosis D is Rare, and is seen on severe hypercalcemia and hyperphophatemi CAUTION: Replacement therapy in deficient i hypervitaminosis D NOTE:-Dark coloured individuals as compare to nterefere with Vitamin D absorption.	ein while in circulat tenance of calcium position, calcium m ineralize newly form disease) ase activity oidism (Mild to Mo is like phenytoin, ph Iv after prolonged e a. individuals must be	ion. homeostatis. I hobilization, ma ned osteoid in I derate deficier henobarbital an exposure to ext monitored by p	t promotes calcium a sinly regulated by par pone, resulting in rick acv) d carbamazepine, tha remely high doses of periodic assessment c	absorption, renal calciu athyroid harmone (PT tets in children and ost at increases Vitamin D Vitamin D. When it oc of Vitamin D levels in o	Im absorption and H). reomalacia in adults. metabolism. curs, it can result in rder to prevent
	*** End	Of Report	* * *		

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

