



	Dr. Vinay Chopra MD (Pathology & Micr Chairman & Consultar	obiology)	M	am Chopra ID (Pathology) ant Pathologist	
NAME	: Mr. SAMEER				
AGE/ GENDER	: 49 YRS/MALE		PATIENT ID	: 178309	4
COLLECTED BY	:		REG. NO./LAB NO.	:01250	3080003
REFERRED BY	:		REGISTRATION DATE		/2025 07:07 AM
BARCODE NO. CLIENT CODE.	: 01526674 : KOS DIAGNOSTIC LAB		COLLECTION DATE REPORTING DATE		/2025 07:10AM /2025 08:47AM
CLIENT CODE. CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBA	ALA CANTI		. 00/ Wai	/ 2023 00.47 Alvi
Test Name		Value	Unit		Biological Reference interval
HAEMOGLOBIN (HE by CALORIMETRIC RED BLOOD CELL (F by HYDRO DYNAMIC FC PACKED CELL VOLU by CALCULATED BY AU MEAN CORPUSCULA by CALCULATED BY AU MEAN CORPUSCULA by CALCULATED BY AU RED CELL DISTRIBU by CALCULATED BY AU RED CELL DISTRIBU	COMP (RBCS) COUNT AND INDICES 3) RBC) COUNT DOUSING, ELECTRICAL IMPEDENCE ME (PCV) JTOMATED HEMATOLOGY ANALYZER		ELLNESS PANEL: (OOD COUNT (CBC) gm/dL Million % fL pg g/dL % fL RATIO	ns/cmm	12.0 - 17.0 3.50 - 5.00 40.0 - 54.0 80.0 - 100.0 27.0 - 34.0 32.0 - 36.0 11.00 - 16.00 35.0 - 56.0 BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA:
GREEN & KING IND by CALCULATED WHITE BLOOD CEL TOTAL LEUCOCYTE	LS (WBCS)	22.15 6810	RATIO /cmm		>13.0 BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0 4000 - 11000
by FLOW CYTOMETRY NUCLEATED RED BI	BY SF CUBE & MICROSCOPY LOOD CELLS (nRBCS) T HEMATOLOGY ANALYZER	NIL			0.00 - 20.00
	LOOD CELLS (nRBCS) % JTOMATED HEMATOLOGY ANALYZER	NIL	%		< 10 %





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

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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





Dr. Vinay Chopra

MD (Pathology & Microbiology) Chairman & Consultant Pathologist



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

NAME	: Mr. SAMEER		
AGE/ GENDER	: 49 YRS/MALE	PATIENT ID	: 1783094
COLLECTED BY	:	REG. NO./LAB NO.	: 012503080003
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Test Name		Value	Unit	Biological Reference interval
DIFFERENTIAL LEUCOCYTE	E COUNT (DLC)			
NEUTROPHILS by flow cytometry by sf cue	BE & MICROSCOPY	58	%	50 - 70
LYMPHOCYTES by flow cytometry by SF cue	BE & MICROSCOPY	34	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUE	BE & MICROSCOPY	2	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUE	BE & MICROSCOPY	6	%	2 - 12
BASOPHILS by flow cytometry by sf cue ABSOLUTE LEUKOCYTES (V		0	%	0 - 1
ABSOLUTE NEUTROPHIL CO by FLOW CYTOMETRY BY SF CUE		3950	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE CO by FLOW CYTOMETRY BY SF CUE	BE & MICROSCOPY	2315	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COU by FLOW CYTOMETRY BY SF CUE		136	/cmm	40 - 440
ABSOLUTE MONOCYTE COUN by FLOW CYTOMETRY BY SF CUE	BE & MICROSCOPY	409	/cmm	80 - 880
ABSOLUTE BASOPHIL COUN by FLOW CYTOMETRY BY SF CUE	BE & MICROSCOPY	0	/cmm	0 - 110
ABSOLUTE IMMATURE GRAM by FLOW CYTOMETRY BY SF CUE	BE & MICROSCOPY	0	/cmm	0.0 - 999.0
PLATELETS AND OTHER PL	ATELET PREDICTIVE	E MARKERS.		
PLATELET COUNT (PLT) by hydro dynamic focusing, e	ELECTRICAL IMPEDENCE	348000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, E	ELECTRICAL IMPEDENCE	0.36 ^H	%	0.10 - 0.36
MEAN PLATELET VOLUME (M by HYDRO DYNAMIC FOCUSING, E		10	fL	6.50 - 12.0
PLATELET LARGE CELL COU by HYDRO DYNAMIC FOCUSING, E		105000 ^H	/cmm	30000 - 90000
PLATELET LARGE CELL RAT by HYDRO DYNAMIC FOCUSING, E		30	%	11.0 - 45.0
PLATELET DISTRIBUTION W by HYDRO DYNAMIC FOCUSING, E		16.6	%	15.0 - 17.0





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Test NameValueUnitBiological Reference interval

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD



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BARCODE NO.	: 01526674	COLLI	CTION DATE	: 08/Mar/2025 07:10AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		RTING DATE	: 08/Mar/2025 11:46AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,			
CLIENT ADDRESS	. 0545/ 1, MCHOLSON ROAD, .	ANIDALA CANTI		
Test Name		Value	Unit	Biological Reference interva
WHOLE BLOOD	AEMOGLOBIN (HbA1c):	12.6 ^H	%	4.0 - 6.4
by HPLC (HIGH PERFO ESTIMATED AVERA	RMANCE LIQUID CHROMATOGRAPHY)	314.92 ^H	mg/dL	60.00 - 140.00
by HPLC (HIGH PERFO INTERPRETATION:	RMANCE LIQUID CHROMATOGRAPHY)	I DIABETES ASSOCIATION (ADA):	
			пену	
	REFERENCE GROUP	GLYCOSYI	ATED HEMOGLOGIB	(HBAIC) IN %
	REFERENCE GROUP abetic Adults >= 18 years	GLYCOSYI	ATED HEMOGLOGIB <5.7	
Non di		GLYCOSY	<5.7 5.7 – 6.4	
Non di A	abetic Adults >= 18 years	GLYCOSY	<5.7 5.7 - 6.4 >= 6.5	
Non di A	abetic Adults >= 18 years .t Risk (Prediabetes)		<5.7 5.7 – 6.4 >= 6.5 Age > 19 Years	
Non di A D	abetic Adults >= 18 years t Risk (Prediabetes) Diagnosing Diabetes	Goals of The	<5.7 5.7 - 6.4 >= 6.5 Age > 19 Years "apy:	< 7.0
Non di A D	abetic Adults >= 18 years .t Risk (Prediabetes)		<5.7 5.7 - 6.4 >= 6.5 Age > 19 Years "apy:	

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	RI	PORTING DATE	: 08/Mar/2025 09:01AM	
LIENT ADDRESS	: 6349/1, NICHOLSON ROAD	, AMBALA CANTT			
Test Name		Value	Unit	Biological Refer	ence interval
systemic lupus erythe CONDITION WITH LO A low ESR can be see polycythaemia), sigr	be used to monitor disease acti ematosus	ne normal sedimentat	ion of red blood cells.	such as a high red blood cell co	unt
C Generally, ESR doe CCRP is not affected If the ESR is elevat Women tend to ha Drugs such as dext	e protein (C-RP) are both marke s not change as rapidly as does by as many other factors as is E ed, it is typically a result of two ve a higher ESR, and menstruati ran, methyldopa, oral contrace d quinine may decrease it	CRP, either at the sta SR, making it a better types of proteins, glo ion and pregnancy car	marker of inflammatio bulins or fibrinogen. a cause temporary eleva	n. ations.	se ESR, while





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



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CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPO	RTING DATE	:08/Mar/2025 11:48AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROA	D, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	CLIN	ICAL CHEMISTRY/	BIOCHEMISTI	RY
		GLUCOSE FAST	'ING (F)	
	G (F): PLASMA	251.84 ^H	mg/dL	NORMAL: < 100.0

KOS Diagnostic Lab (A Unit of KOS Healthcare)

A fasting plasma glucose level below 100 mg/dl is considered normal.
 A fasting plasma glucose level between 100 - 125 mg/dl is considered as glucose intolerant or prediabetic. A fasting and post-prandial blood

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





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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAI	D, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		LIPID PROFILE	: BASIC	
CHOLESTEROL TO	TAL: SERUM	296.67 ^H	mg/dL	OPTIMAL: < 200.0
by CHOLESTEROL O		230.07		BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR =
DICI VCEDIDES, S	EDIM	191.94	mg/dI	240.0 OPTIMAL: 150.0
RIGLYCERIDES: S	ERUM PHATE OXIDASE (ENZYMATIC)	121.24	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0
				HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
	L (DIRECT): SERUM	81.98 ^H	mg/dL	LOW HDL: < 30.0
by SELECTIVE INHIBIT	10N			BORDERLINE HIGH HDL: 30 60.0 HIGH HDL: > OR = 60.0
DL CHOLESTERO		190.44 ^H	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129 BORDERLINE HIGH: 130.0 -
				159.0 HIGH: 160.0 - 189.0
				VERY HIGH: > OR = 190.0
NON HDL CHOLES ⁷ by Calculated, spe		214.69 ^H	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 15 BORDERLINE HIGH: 160.0 - 189.0
				HIGH: 190.0 - 219.0
				VERY HIGH: $> OR = 220.0$
LDL CHOLESTER(by CALCULATED, SPE		24.25	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SEF	RUM	714.58 ^H	mg/dL	350.00 - 700.00
HOLESTEROL/HI by CALCULATED, SPE		3.62	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, A	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
LDL/HDL RATIO: S by CALCULATED, SPE		2.32	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/H by CALCULATED, SPE		1.48 ^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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Test Name	Value	Unit	Biological Reference interval
LIVER	FUNCTION TI	EST (COMPLETE)	
BILIRUBIN TOTAL: SERUM by DIAZOTIZATION, SPECTROPHOTOMETRY	0.75	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.15	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.6	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	13.75	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	11.34	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.21	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL	90.11	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	18.48	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	7.33	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	4.38	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.95	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.48	RATIO	1.00 - 2.00

INTERPRETATION

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

INCREASED:

> 2 (Highly Suggestive)
1.4 - 2.0
> 1.5
> 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).

PROGNOSTIC SIGNIFICANCE:	

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	Biological Reference interval
	KIDNE	Y FUNCTION 1	FEST (COMPLETE)	
UREA: SERUM		42.69	mg/dL	10.00 - 50.00
by UREASE - GLUTAM	ATE DEHYDROGENASE (GLDH)		Ũ	
CREATININE: SERU		0.91	mg/dL	0.40 - 1.40
	OGEN (BUN): SERUM	19.95	mg/dL	7.0 - 25.0
	OGEN (BUN)/CREATININE	21.92 ^H	RATIO	10.0 - 20.0
RATIO: SERUM by CALCULATED, SPE	CTROPHOTOMETRY			
UREA/CREATININE	E RATIO: SERUM	46.91	RATIO	
by CALCULATED, SPE		2.0	m e /dI	2.00 7.70
URIC ACID: SERUM by URICASE - OXIDASE		3.6	mg/dL	3.60 - 7.70
CALCIUM: SERUM		10.37	mg/dL	8.50 - 10.60
by ARSENAZO III, SPEC PHOSPHOROUS: SE		3.39	mg/dL	2.30 - 4.70
	ATE, SPECTROPHOTOMETRY	5.59	IIIg/ UL	2.30 - 4.70
ELECTROLYTES				
SODIUM: SERUM		143.5	mmol/L	135.0 - 150.0
by ISE (ION SELECTIVE POTASSIUM: SERUN		4.26	mmol/L	3.50 - 5.00
by ISE (ION SELECTIVE	E ELECTRODE)			
CHLORIDE: SERUM		107.63	mmol/L	90.0 - 110.0
, ,	ERULAR FILTERATION RATE			
ESTIMATED GLOMI (eGFR): SERUM by CALCULATED INTERPRETATION:	ERULAR FILTERATION RATE	103.3		

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.





	MD (Pathology	Dr. Vinay ChopraDr. Yugam ChopraMD (Pathology & Microbiology)MD (Pathology)Chairman & Consultant PathologistCEO & Consultant Pathologist			
AME	: Mr. SAMEER				
GE/ GENDER	: 49 YRS/MALE	PATIE	NT ID	: 1783094	
OLLECTED BY	•	REG. N	O./LAB NO.	: 012503080003	
EFERRED BY			FRATION DATE	: 08/Mar/2025 07:0	
ARCODE NO.	: 01526674		CTION DATE	: 08/Mar/2025 07:	
				: 08/Mar/2025 12:5	
LIENT CODE.	: KOS DIAGNOSTIC LAB		TING DATE	: 08/Mar/ 2025 12:3	34PM
LIENT ADDRESS	: 6349/1, NICHOLSON ROAD), AMBALA CANTT			
Test Name		Value	Unit	Biologica	al Reference interval
NCREASED RATIO (>2 . Postrenal azotemia	ass (subnormal creatinine proc tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATINI (BUN rises disproportionately	NE LEVELS: more than creatinine) (e.g	. obstructive uropa	ithy).	
NCREASED RATIO (>2 Postrenal azotemia Perenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet and Severe liver diseas Other causes of de Repeated dialysis Nherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin ther STIMATED GLOMERL CKD STAGE	tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATINI (BUN rises disproportionately superimposed on renal disease 10:1) WITH DECREASED BUN : osis. ad starvation. b. creased urea synthesis. urea rather than creatinine dif monemias (urea is virtually absorbed in appropiate antidiuretic har 10:1) WITH INCREASED CREATIN py (accelerates conversion of co eleases muscle creatinine). who develop renal failure. 1: sis (acetoacetate causes false in creased BUN/creatinine ratio). apy (interferes with creatinine) JLAR FILTERATION RATE: DESCRIPTION	NE LEVELS: more than creatinine) (e.g e. ffuses out of extracellular f sent in blood). mone) due to tubular secre IINE: creatine to creatinine). increase in creatinine with measurement).	luid). etion of urea. certain methodolo	ogies,resulting in norm SOCIATED FINDINGS	nal ratio when dehydrat
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 GLOMERI CKD STAGE G1	tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATINI (BUN rises disproportionately superimposed on renal disease 10:1) WITH DECREASED BUN : osis. ad starvation. b. creased urea synthesis. urea rather than creatinine dif monemias (urea is virtually ab- of inappropiate antidiuretic har 10:1) WITH INCREASED CREATIN py (accelerates conversion of co- eleases muscle creatinine). who develop renal failure. 1: sis (acetoacetate causes false in creased BUN/creatinine ratio). apy (interferes with creatinine) JLAR FILTERATION RATE: DESCRIPTION Normal kidney fun	NE LEVELS: more than creatinine) (e.g. ffuses out of extracellular f sent in blood). mone) due to tubular secret IINE: creatine to creatinine). increase in creatinine with measurement). Image: Image:	luid). etion of urea. certain methodolo 1.73m2) AS	ogies,resulting in norm SOCIATED FINDINGS No proteinuria	nal ratio when dehydrat
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, glucocorticoids) 0:1) WITH ELEVATED CREATINI (BUN rises disproportionately superimposed on renal disease 10:1) WITH DECREASED BUN : osis. ad starvation. b. creased urea synthesis. urea rather than creatinine dif monemias (urea is virtually ab- of inappropiate antidiuretic har 10:1) WITH INCREASED CREATIN py (accelerates conversion of co- eleases muscle creatinine). who develop renal failure. 10:1) WITH INCREASED CREATIN py (accelerates conversion of co- eleases muscle creatinine). who develop renal failure. 11: sis (acetoacetate causes false in creased BUN/creatinine ratio). apy (interferes with creatinine) JLAR FILTERATION RATE: DESCRIPTION Normal kidney fun-	NE LEVELS: more than creatinine) (e.g. ffuses out of extracellular f sent in blood). mone) due to tubular secret IINE: creatine to creatinine). increase in creatinine with measurement). I GFR (mL/min/ nction >90 with >90	luid). etion of urea. certain methodolo 1.73m2) AS	ogies,resulting in norm SOCIATED FINDINGS No proteinuria resence of Protein ,	al ratio when dehydrat
ICREASED RATIO (>2 Postrenal azotemia Prerenal azotemia ECREASED RATIO (< Acute tubular necr Low protein diet an 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 CKD STAGE G1	tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATINI (BUN rises disproportionately superimposed on renal disease 10:1) WITH DECREASED BUN : osis. ad starvation. b. creased urea synthesis. urea rather than creatinine dif monemias (urea is virtually ab- of inappropiate antidiuretic har 10:1) WITH INCREASED CREATIN py (accelerates conversion of co- eleases muscle creatinine). who develop renal failure. 1: sis (acetoacetate causes false in creased BUN/creatinine ratio). apy (interferes with creatinine) JLAR FILTERATION RATE: DESCRIPTION Normal kidney fun	NE LEVELS: more than creatinine) (e.g. ffuses out of extracellular f sent in blood). mone) due to tubular secret IINE: creatine to creatinine). increase in creatinine with measurement). I GFR (mL/min/ ortholder >90 with >90	luid). etion of urea. certain methodolo 1.73m2) AS	ogies,resulting in norm SOCIATED FINDINGS No proteinuria	al ratio when dehydrat
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 (Repeated dialysis (Inherited hyperam SIADH (syndrome of Pregnancy. ECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients JAPPROPIATE RATIO Diabetic ketoacido nould produce an in Cephalosporin ther STIMATED GLOMERI G1 G2 G3a G3a	tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATINI (BUN rises disproportionately superimposed on renal disease 10:1) WITH DECREASED BUN : osis. ad starvation. b. creased urea synthesis. urea rather than creatinine dif monemias (urea is virtually ab- of inappropiate antidiuretic har 10:1) WITH INCREASED CREATIN py (accelerates conversion of co- eleases muscle creatinine). who develop renal failure. 10:1) WITH INCREASED CREATIN py (accelerates conversion of co- eleases muscle creatinine). who develop renal failure. 10:1) WITH INCREASED CREATIN py (interferes with creatinine JLAR FILTERATION RATE: DESCRIPTION Normal kidney fun Kidney damage v normal or high C	NE LEVELS: more than creatinine) (e.g. ffuses out of extracellular f sent in blood). mone) due to tubular secret IINE: creatine to creatinine). increase in creatinine with measurement). I GFR (mL/min/ nction >90 with >90 GFR 60 - 89 in GFR 30-59	luid). etion of urea. certain methodolo 1.73m2) AS	ogies,resulting in norm SOCIATED FINDINGS No proteinuria resence of Protein ,	al ratio when dehydrat
VCREASED 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 JAPPROPIATE RATIO Diabetic ketoacido nould produce an in Cephalosporin ther STIMATED GLOMERI G1 G2 G3a	tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATINI (BUN rises disproportionately superimposed on renal disease 10:1) WITH DECREASED BUN : osis. ad starvation. b. creased urea synthesis. urea rather than creatinine dif monemias (urea is virtually ab- of inappropiate antidiuretic har 10:1) WITH INCREASED CREATIN py (accelerates conversion of c eleases muscle creatinine). who develop renal failure. 1: sis (acetoacetate causes false in creased BUN/creatinine ratio). apy (interferes with creatinine) 1/AR FILTERATION RATE: DESCRIPTION Normal kidney fun Kidney damage v normal or high (Mild decrease in	NE LEVELS: more than creatinine) (e.g. ffuses out of extracellular f sent in blood). mone) due to tubular secret IINE: creatine to creatinine). increase in creatinine with measurement). Image: offR GFR GFR GFR GFR GFR GFR GFR Monophic Soft GFR Soft Monophic Soft	luid). etion of urea. certain methodolo 1.73m2) AS	ogies,resulting in norm SOCIATED FINDINGS No proteinuria resence of Protein ,	al ratio when dehydrat





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



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	Dr. Vinay Chopra MD (Pathology & Microbiolog Chairman & Consultant Patho		(Pathology)
NAME	: Mr. SAMEER		
AGE/ GENDER	: 49 YRS/MALE	PATIENT ID	: 1783094
COLLECTED BY	:	REG. NO./LAB NO.	: 012503080003
REFERRED BY	:	REGISTRATION DATE	: 08/Mar/2025 07:07 AM
BARCODE NO.	: 01526674	COLLECTION DATE	:08/Mar/202507:10AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 08/Mar/2025 12:54PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CA	NTT	
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





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	Dr. Vinay ChopraDr. YugarMD (Pathology & Microbiology)MDChairman & Consultant PathologistCEO & Consultant			MD (Pathology)
NAME	: Mr. SAMEER				
AGE/ GENDER	: 49 YRS/MALE		PATIENT ID		: 1783094
COLLECTED BY	:		REG. NO./LAB NO).	: 012503080003
REFERRED BY	:		REGISTRATION D	DATE	: 08/Mar/2025 07:07 AM
BARCODE NO.	:01526674		COLLECTION DAT	ГЕ	:08/Mar/202507:10AM
CLIENT CODE.	: KOS DIAGNOSTIC L	AB	REPORTING DAT	Έ	:08/Mar/2025 11:48AM
CLIENT ADDRESS	: 6349/1, NICHOLSO	N ROAD, AMBALA CAN	TT		
Test Name		Value	Ur	nit	Biological Reference interv
		ENDO	OCRINOLOGY		
		THYROID FU	NCTION TEST: TO	DTAL	
TRIIODOTHYRONI	NE (T3): SERUM	0.75 IMMUNOASSAY)	ng	g/mL	0.35 - 1.93
THYROXINE (T4): S		6.81	μ	gm/dL	4.87 - 12.60
	TING HORMONE (TS		μΙ	U/mL	0.35 - 5.50
3rd GENERATION, ULT					
TSH levels are subject to day has influence on the triiodothyronine (T3).Fai	measured serum TSH concer	<i>trations</i> . TSH stimulates the	e production and secretion	of the me	. The variation is of the order of 50%.Hence time of tabolically active hormones, thyroxine (T4)and underproduction (hypothyroidism) or
CLINICAL CONDITION		T3	T4		TSH
Primary Hypothyroidis		Reduced	Reduced	Inc	reased (Significantly)
Subclinical Hypothyroi	dism: Nor	mal or Low Normal	Normal or Low Normal		High

LIMI	TAT	IONS	÷

Primary Hyperthyroidism:

Subclinical Hyperthyroidism:

1. T3 and T4 circulates in reversibly bound form with Thyroid binding globulins (TBG), and to a lesser extent albumin and Thyroid binding Pre Albumin so conditions in which TBG and protein levels alter such as pregnancy, excess estrogens, androgens, anabolic steroids and glucocorticoids may falsely affect the T3 and T4 levels and may cause false thyroid values for thyroid function tests.

Increased

Normal or High Normal

Reduced (at times undetectable)

Reduced

2. Normal levels of T4 can also be seen in Hyperthyroid patients with :T3 Thyrotoxicosis, Decreased binding capacity due to hypoproteinemia or ingestion of certain drugs (e.g.: phenytoin , salicylates).

3. Serum T4 levels in neonates and infants are higher than values in the normal adult , due to the increased concentration of TBG in neonate serum.

4. TSH may be normal in central hypothyroidism , recent rapid correction of hyperthyroidism or hypothyroidism , pregnancy , phenytoin therapy.

TRIIODOTH	RONINE (T3) THYROXINE (T4) THYROID STIMULATIN		THYROXINE (T4)		ATING HORMONE (TSH)
Age	Refferance Range (ng/mL)	Age	Refferance Range (µg/dL)	Age	Reference Range (μIU/mL)
0-7 Days	0.20 - 2.65	0 - 7 Days	5.90 - 18.58	0 - 7 Days	2.43 - 24.3
7 Days - 3 Months	0.36 - 2.59	7 Days - 3 Months	6.39 - 17.66	7 Days - 3 Months	0.58 - 11.00
3 - 6 Months	0.51 - 2.52	3 - 6 Months	6.75 - 17.04	3 Days – 6 Months	0.70 - 8.40
6 - 12 Months	0.74 - 2.40	6 - 12 Months	7.10 - 16.16	6 – 12 Months	0.70 - 7.00

Increased

Normal or High Normal





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 Pathologi		(Pathology)
NAME	: Mr. SAMEER		
AGE/ GENDER	: 49 YRS/MALE	PATIENT ID	: 1783094
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANT	Г	

Test Name			Value	Unit		Biological Reference interval
1 - 10 Years	0.92 - 2.28	1 - 10 Years	6.00 - 13.80	1 – 10 Years	0.60 - 5.50	
11- 19 Years	0.35 - 1.93	11 - 19 Years	4.87-13.20	11 – 19 Years	0.50 - 5.50	
> 20 years (Adults)	0.35 - 1.93	> 20 Years (Adults)	4.87 - 12.60	> 20 Years (Adults)	0.35-5.50	
	RECON	IMENDATIONS OF TSH LI	VELS DURING PRE	GNANCY (µIU/mL)		
	1st Trimester			0.10 - 2.50		
	2nd Trimester			0.20 - 3.00		
	3rd Trimester			0.30 - 4.10		

INCREASED TSH LEVELS:

1. Primary or untreated hypothyroidism may vary from 3 times to more than 100 times normal depending upon degree of hypofunction.

2. Hypothyroid patients receiving insufficient thyroid replacement therapy.

3.Hashimotos thyroiditis

4.DRUGS: Amphetamines, iodine containing agents & dopamine antagonist.

5.Neonatal period, increase in 1st 2-3 days of life due to post-natal surge

DECREASED TSH LEVELS:

1. Toxic multi-nodular goiter & Thyroiditis.

2. Over replacement of thyroid hormone in treatment of hypothyroidism.

3. Autonomously functioning Thyroid adenoma

4. Secondary pituitary or hypothalamic hypothyroidism

5. Acute psychiatric illness

6.Severe dehydration.

7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.

8.Pregnancy: 1st and 2nd Trimester





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	Dr. Vinay Cho MD (Pathology & M Chairman & Const	Microbiology)				
NAME	: Mr. SAMEER					
AGE/ GENDER	: 49 YRS/MALE	PAT	IENT ID	: 1783094		
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BARCODE NO.	:01526674	COL	LECTION DATE	:08/Mar/202507:10AM		
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REP	ORTING DATE	: 08/Mar/2025 12:27PM		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A), AMBALA CANTT				
Test Name		Value	Unit	Biological Reference interval		
DIIVCICAT EVAMI		CLINICAL PAT	FHOLOGY SCOPIC EXAMINA	ATION		
PHYSICAL EXAMIN		10				
0	QUANTITY RECIEVED by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY		ml			
COLOUR		AMBER YELLO	OW	PALE YELLOW		
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY TRANSPARANCY		HAZY		CLEAR		
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY						
SPECIFIC GRAVITY by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY		1.01		1.002 - 1.030		
CHEMICAL EXAMI						
REACTION		ACIDIC				
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY		Negative		NEGATIVE (-ve)		
PROTEIN by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY		Negative				
SUGAR	CTANCE SPECTROPHOTOMETRY	3+		NEGATIVE (-ve)		
pH		6.5		5.0 - 7.5		
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY		Negative		NEGATIVE (-ve)		
BILIRUBIN by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY		negative		NEGATIVE (-VE)		
		Negative		NEGATIVE (-ve)		
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY. UROBILINOGEN		Normal	EU/dL	0.2 - 1.0		
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY KETONE BODIES		Negative		NEGATIVE (-ve)		
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY BLOOD by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY						
		Negative		NEGATIVE (-ve)		
ASCORBIC ACID		NEGATIVE (-v	e)	NEGATIVE (-ve)		
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY					
RED BLOOD CELLS (RBCs) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT		NEGATIVE (-v	e) /HPF	0 - 3		





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











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

NAME	: Mr. SAMEER				
AGE/ GENDER	Y : Y : . :01526674		PATIENT ID	: 1783094 : 012503080003 : 08/Mar/2025 07:07 AM : 08/Mar/2025 07:10AM : 08/Mar/2025 12:27PM	
COLLECTED BY			REG. NO./LAB NO.		
REFERRED BY			REGISTRATION DATE		
BARCODE NO.			COLLECTION DATE		
CLIENT CODE.			REPORTING DATE		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AN	MBALA CANT	Т		
Test Name		Value	Unit	Biological Reference interval	
PUS CELLS	CENTRIFUGED URINARY SEDIMENT	2-3	/HPF	0 - 5	
			(IIDE		

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
EPITHELIAL CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	0-2	/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)	ABSENT		ABSENT

*** End Of Report ***



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

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