



	Dr. Vinay Chopra MD (Pathology & Micr Chairman & Consultar	obiology)		(Pathology)	
NAME	: Mr. PRAVEEN KUMAR				
AGE/ GENDER	: 55 YRS/MALE		PATIENT ID	: 1732418	
COLLECTED BY	:		REG. NO./LAB NO.	: 012501230044	
REFERRED BY	: Dr. N.C.WADHAWAN (AMBALA CA	ANTT)	REGISTRATION DATE	: 23/Jan/2025 12:41 PM	
BARCODE NO.	: 01524311		COLLECTION DATE	: 23/Jan/2025 12:42PM	
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 23/Jan/2025 12:58PM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMB/	ALA CANTT			
Test Name		Value	Unit	Biological Reference	interval
			LLNESS PANEL: 1.1 00D COUNT (CBC)		
RED BLOOD CELLS	G (RBCS) COUNT AND INDICES				
HAEMOGLOBIN (H	B)	14.7	gm/dL	12.0 - 17.0	
RED BLOOD CELL (RBC) COUNT	4.34	Millions/	ýcmm 3.50 - 5.00	
PACKED CELL VOLU	JME (PCV) utomated hematology analyzer	43.1	%	40.0 - 54.0	
MEAN CORPUSCUL		99.4	fL	80.0 - 100.0	
MEAN CORPUSCUL	AR HAEMOGLOBIN (MCH) UTOMATED HEMATOLOGY ANALYZER	34	pg	27.0 - 34.0	
MEAN CORPUSCUL	AR HEMOGLOBIN CONC. (MCHC) UTOMATED HEMATOLOGY ANALYZER	34.2	g/dL	32.0 - 36.0	
RED CELL DISTRIB	UTION WIDTH (RDW-CV) UTOMATED HEMATOLOGY ANALYZER	16	%	11.00 - 16.00	
RED CELL DISTRIB	UTION WIDTH (RDW-SD) UTOMATED HEMATOLOGY ANALYZER	59.9 ^H	fL	35.0 - 56.0	
MENTZERS INDEX by CALCULATED		22.9	RATIO	BETA THALASSEMIA 13.0 IRON DEFICIENCY A >13.0	
GREEN & KING INE by CALCULATED		36.78	RATIO	BETA THALASSEMIA 65.0 IRON DEFICIENCY A 65.0	
WHITE BLOOD CE					
TOTAL LEUCOCYTE	E COUNT (TLC) / by sf cube & microscopy	11550 ^H	/cmm	4000 - 11000	
NUCLEATED RED B	BLOOD CELLS (nRBCS) RT HEMATOLOGY ANALYZER	NIL		0.00 - 20.00	
NUCLEATED RED B	LOOD CELLS (nRBCS) % UTOMATED HEMATOLOGY ANALYZER	NIL	%	< 10 %	





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NAME



Dr. Yugam Chopra Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologist MD (Pathology) **CEO & Consultant Pathologist** : Mr. PRAVEEN KUMAR AGE/ GENDER **PATIENT ID** : 55 YRS/MALE **COLLECTED BY** REG. NO./LAB NO. : **REFERRED BY REGISTRATION DATE** : Dr. N.C.WADHAWAN (AMBALA CANTT) **BARCODE NO.** :01524311 **COLLECTION DATE CLIENT CODE.** : KOS DIAGNOSTIC LAB **REPORTING DATE CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT

:1732418 :012501230044 : 23/Jan/2025 12:41 PM : 23/Jan/2025 12:42PM : 23/Jan/2025 12:58PM

Value	Unit	Biological Reference interval
63	%	50 - 70
30	%	20 - 40
1	%	1 - 6
6	%	2 - 12
0	%	0 - 1
7277	/cmm	2000 - 7500
3465	/cmm	800 - 4900
116	/cmm	40 - 440
693	/cmm	80 - 880
0	/cmm	0 - 110
E MARKERS.		
181000	/cmm	150000 - 450000
0.22	%	0.10 - 0.36
12 ^H	fL	6.50 - 12.0
75000	/cmm	30000 - 90000
41.6	%	11.0 - 45.0
16.3	%	15.0 - 17.0
	63 30 1 6 0 7277 3465 116 693 0 EMARKERS. 181000 0.22 12H 75000 41.6	63 % 30 % 30 % 1 % 6 % 6 % 0 % 7277 /cmm 3465 /cmm 116 /cmm 693 /cmm 0 /cmm 0 /cmm 0 /cmm 0.22 % 12H fL 75000 /cmm 41.6 %



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Test Name	Value	Unit	Biological Reference interval





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LIENT CODE.	: KOS DIAGNOSTIC LAB	RE	PORTING DATE	: 23/Jan/2025 01:34PM
LIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	BALA CANTT		
Fest Name		Value	Unit	Biological Reference interval
by RED CELL AGGREG NTERPRETATION: . ESR is a non-specif nmune disease, but . An ESR can be affe s C-reactive protein	does not tell the health practitioner cted by other conditions besides infl be used to monitor disease activity a	exactly where the ammation. For the	e inflammation is in the his reason, the ESR is ty	ion associated with infection, cancer and auto-
low ESR can be see bolycythaemia), sigr s sickle cells in sickl IOTE: . ESR and C - reactiv . Generally, ESR doe . CRP is not affected . If the ESR is elevat . Women tend to ha . Drugs such as dext	n with conditions that inhibit the no nificantly high white blood cell count e cell anaemia) also lower the ESR. e protein (C-RP) are both markers of s not change as rapidly as does CRP, by as many other factors as is ESR, n ed, it is typically a result of two type ye a higher ESR, and menstruation a	t (leucocytosis) , inflammation. either at the sta naking it a better s of proteins, glo nd pregnancy car	and some protein abno rt of inflammation or a: marker of inflammatior bulins or fibrinogen. i cause temporary eleva	ormalities. Šome changes in red cell shape (such s it resolves. n.





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BARCODE NO.	: 01524311	(COLLECTION DATE	: 23/Jan/2025 12:42PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	F	REPORTING DATE	: 23/Jan/2025 03:30PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	CLINI	CAL CHEMIST	RY/BIOCHEMIST	'RY
		GLUCOSE I	FASTING (F)	
GLUCOSE FASTING	G (F): PLASMA E - PEROXIDASE (GOD-POD)	99.42	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0

KOS Diagnostic Lab (A Unit of KOS Healthcare)

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 CANTI	2	
Test Name		Value	Unit	Biological Reference interval
		I IPIN PR	OFILE : BASIC	
CHOLESTEROL TO	TAL·SERIM			OPTIMAL: < 200.0
by CHOLESTEROL O		200.34 ^H	ling/ uL	BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR =
				240.0
TRIGLYCERIDES: S		398.7 ^H	mg/dL	OPTIMAL: < 150.0
by GLYCEROL PHOSP	PHATE OXIDASE (ENZYMATIC)			BORDERLINE HIGH: 150.0 -
				199.0 HIGH: 200.0 - 499.0
				VERY HIGH: $> OR = 500.0$
	L (DIRECT): SERUM	65.87	mg/dL	LOW HDL: < 30.0
by SELECTIVE INHIBIT	ION			BORDERLINE HIGH HDL: 30.0 60.0
				HIGH HDL: $> OR = 60.0$
LDL CHOLESTERO	L: SERUM	54.73	mg/dL	OPTIMAL: < 100.0
by CALCULATED, SPE	ECTROPHOTOMETRY			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		134.47 ^H	mg/dL	OPTIMAL: < 130.0
by CALCULATED, SPE	ECTROPHOTOMETRY			ABOVE OPTIMAL: 130.0 - 159.0
				BORDERLINE HIGH: 160.0 - 189.0
				HIGH: 190.0 - 219.0
				VERY HIGH: $> OR = 220.0$
VLDL CHOLESTER(by CALCULATED, SPE		79.74 ^H	mg/dL	0.00 - 45.00
бу CALCULATED, SPE ГОТАL LIPIDS: SEF		799.38 ^H	mg/dL	350.00 - 700.00
by CALCULATED, SPE	CTROPHOTOMETRY			
CHOLESTEROL/HE by CALCULATED, SPE		3.04	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0
				HIGH RISK: > 11.0
	ok .		Ghopra	

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Test Name		Value	Unit	Biological Reference interval	
LDL/HDL RATIO: S by CALCULATED, SPE		0.83	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0	
TRIGLYCERIDES/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY		6.05 ^H	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	Biological Reference interval				
1 est Maine		value	Unit	biological kelerence inter var				
	LIVER FUNCTION TEST (COMPLETE)							
BILIRUBIN TOTAL by DIAZOTIZATION, S	: SERUM PECTROPHOTOMETRY	0.33	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20				
	Г (CONJUGATED): SERUM spectrophotometry	0.11	mg/dL	0.00 - 0.40				
BILIRUBIN INDIRE	ECT (UNCONJUGATED): SERUM	0.22	mg/dL	0.10 - 1.00				
SGOT/AST: SERUN by IFCC, WITHOUT P	I YRIDOXAL PHOSPHATE	23.2	U/L	7.00 - 45.00				
SGPT/ALT: SERUM	I /RIDOXAL PHOSPHATE	25.2	U/L	0.00 - 49.00				
AST/ALT RATIO: S	ERUM ECTROPHOTOMETRY	0.92	RATIO	0.00 - 46.00				
ALKALINE PHOSP by PARA NITROPHEN PROPANOL	HATASE: SERUM IYL PHOSPHATASE BY AMINO METHYL	69.82	U/L	40.0 - 130.0				
GAMMA GLUTAMY by SZASZ, SPECTRO	L TRANSFERASE (GGT): SERUM	45.44	U/L	0.00 - 55.0				
TOTAL PROTEINS: by BIURET, SPECTRO		7.22	gm/dL	6.20 - 8.00				
ALBUMIN: SERUM		4.27	gm/dL	3.50 - 5.50				
GLOBULIN: SERUM		2.95	gm/dL	2.30 - 3.50				
A : G RATIO: SERU		1.45	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).

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
	KIDNI	EY FUNCTI	ON TEST (COMPLETE)	
UREA: SERUM	IATE DEHYDROGENASE (GLDH)	30.45	mg/dL	10.00 - 50.00
CREATININE: SER	UM	1.2	mg/dL	0.40 - 1.40
by ENZYMATIC, SPEC	CTROPHOTOMETERY ROGEN (BUN): SERUM	14.23	mg/dL	7.0 - 25.0
by CALCULATED, SPE	ECTROPHOTOMETRY	14.23	iiig/ uL	7.0 - 23.0
	ROGEN (BUN)/CREATININE	11.86	RATIO	10.0 - 20.0
RATIO: SERUM by CALCULATED, SPE	ECTROPHOTOMETRY			
UREA/CREATININ	E RATIO: SERUM	25.38	RATIO	
URIC ACID: SERUM	ECTROPHOTOMETRY [4.27	mg/dL	3.60 - 7.70
by URICASE - OXIDAS			-	
CALCIUM: SERUM by ARSENAZO III, SPE	ECTROPHOTOMETRY	8.69	mg/dL	8.50 - 10.60
PHOSPHOROUS: SH	ERUM	4.14	mg/dL	2.30 - 4.70
by PHOSPHOMOLYBE ELECTROLYTES	DATE, SPECTROPHOTOMETRY			
SODIUM: SERUM		140.7	mmol/L	135.0 - 150.0
by ISE (ION SELECTIV				
POTASSIUM: SERU		4.13	mmol/L	3.50 - 5.00
CHLORIDE: SERUN by ISE (ION SELECTIV	1	105.53	mmol/L	90.0 - 110.0
ESTIMATED GLON	MERULAR FILTERATION RATE			
ESTIMATED GLOM (eGFR): SERUM by CALCULATED INTERPRETATION:	IERULAR FILTERATION RATE	71.4		

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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'est Name		Value U	nit Biologi	cal Reference interval	
NCREASED RATIO (>2 . Postrenal azotemia . Prerenal azotemia	tetracycline, glucocorticoids) 20:1) WITH ELEVATED CREATININE LEVI a (BUN rises disproportionately more superimposed on renal disease. 10:1) WITH DECREASED BUN :		re uropathy).		
NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet al Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin the STIMATED GLOMERI CKD STAGE	20:1) WITH ELEVATED CREATININE LEVI a (BUN rises disproportionately more is superimposed on renal disease. 10:1) WITH DECREASED BUN : rosis. nd starvation. e. creased urea synthesis. (urea rather than creatinine diffuses of inappropiate antidiuretic harmone) 10:1) WITH INCREASED CREATININE: py (accelerates conversion of creating eleases muscle creatinine). who develop renal failure. e: sis (acetoacetate causes false increase creased BUN/creatinine ratio). rapy (interferes with creatinine measu JLAR FILTERATION RATE: DESCRIPTION	than creatinine) (e.g. obstructiv but of extracellular fluid). blood). due to tubular secretion of ure e to creatinine). te in creatinine with certain me urement). GFR (mL/min/1.73m2)	a. thodologies,resulting in nor ASSOCIATED FINDINGS	mal ratio when dehydratic	
VCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia ECREASED RATIO (< Acute tubular necr Low protein diet al Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. ECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients VAPPROPIATE RATIO Diabetic ketoacido nould produce an in Cephalosporin ther STIMATED GLOMERT CKD STAGE G1	20:1) WITH ELEVATED CREATININE LEVI a (BUN rises disproportionately more superimposed on renal disease. 10:1) WITH DECREASED BUN : rosis. nd starvation. e. creased urea synthesis. (urea rather than creatinine diffuses of inappropiate antidiuretic harmone) 10:1) WITH INCREASED CREATININE: up (accelerates conversion of creating eleases muscle creatinine). who develop renal failure. b: reased BUN/creatinine ratio). rapy (interferes with creatinine measu JLAR FILTERATION RATE: DESCRIPTION Normal kidney function	than creatinine) (e.g. obstructiv but of extracellular fluid). blood). due to tubular secretion of ure e to creatinine). the in creatinine with certain me urement). GFR (mL/min/1.73m2) >90	a. thodologies,resulting in nor ASSOCIATED FINDINGS No proteinuria	mal ratio when dehydratic	
ICREASED RATIO (>2 Postrenal azotemia Prerenal azotemia ECREASED RATIO (< Acute tubular necr Low protein diet al 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 the STIMATED GLOMERI CKD STAGE	20:1) WITH ELEVATED CREATININE LEVI a (BUN rises disproportionately more is superimposed on renal disease. 10:1) WITH DECREASED BUN : rosis. nd starvation. e. creased urea synthesis. (urea rather than creatinine diffuses of inappropiate antidiuretic harmone) 10:1) WITH INCREASED CREATININE: up (accelerates conversion of creating eleases muscle creatinine). who develop renal failure. b: reased BUN/creatinine ratio). rapy (interferes with creatinine measu JLAR FILTERATION RATE: DESCRIPTION Normal kidney function	than creatinine) (e.g. obstructiv but of extracellular fluid). blood). due to tubular secretion of ure e to creatinine). te in creatinine with certain me urement). GFR (mL/min/1.73m2)	a. thodologies,resulting in nor ASSOCIATED FINDINGS		
CREASED RATIO (>2 Postrenal azotemia Prerenal azotemia ECREASED RATIO (< Acute tubular necr Low protein diet al Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. ECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients APPROPIATE RATIO Diabetic ketoacido ould produce an in Cephalosporin the ETIMATED GLOMERI CKD STAGE G1	20:1) WITH ELEVATED CREATININE LEVI a (BUN rises disproportionately more superimposed on renal disease. 10:1) WITH DECREASED BUN : rosis. ad starvation. e. creased urea synthesis. (urea rather than creatinine diffuses of inappropiate antidiuretic harmone) 10:1) WITH INCREASED CREATININE: up (accelerates conversion of creating eleases muscle creatinine). who develop renal failure. b: reased BUN/creatinine ratio). rapy (interferes with creatinine measu JLAR FILTERATION RATE: DESCRIPTION Normal kidney function Kidney damage with normal or high GFR Mild decrease in GFR	than creatinine) (e.g. obstructiv but of extracellular fluid). blood). due to tubular secretion of ure e to creatinine). the in creatinine with certain me irement). GFR (mL/min/1.73m2) >90 >90 60 -89	a. thodologies,resulting in nor ASSOCIATED FINDINGS No proteinuria Presence of Protein ,		
ICREASED RATIO (>2 Postrenal azotemia Prerenal azotemia ECREASED RATIO (< Acute tubular necr Low protein diet al 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 the STIMATED GLOMERI G1 G2 G3a G3a	20:1) WITH ELEVATED CREATININE LEVI a (BUN rises disproportionately more superimposed on renal disease. 10:1) WITH DECREASED BUN : rosis. ad starvation. e. creased urea synthesis. (urea rather than creatinine diffuses of inappropiate antidiuretic harmone) 10:1) WITH INCREASED CREATININE: up (accelerates conversion of creating eleases muscle creatinine). who develop renal failure. b: rass (acetoacetate causes false increased urea subhycreatinine ratio). rapy (interferes with creatinine measu JLAR FILTERATION RATE: DESCRIPTION Normal kidney function Kidney damage with normal or high GFR Moderate decrease in GFR	than creatinine) (e.g. obstructiv but of extracellular fluid). blood). due to tubular secretion of ure e to creatinine). the in creatinine with certain me irement). GFR (mL/min/1.73m2) >90 >90 60 -89 30-59	a. thodologies,resulting in nor ASSOCIATED FINDINGS No proteinuria Presence of Protein ,		
ICREASED RATIO (>2 Postrenal azotemia Prerenal azotemia ECREASED RATIO (< Acute tubular necr Low protein diet al 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 the STIMATED GLOMERI G1 G2 G3a	20:1) WITH ELEVATED CREATININE LEVI a (BUN rises disproportionately more superimposed on renal disease. 10:1) WITH DECREASED BUN : rosis. ad starvation. e. creased urea synthesis. (urea rather than creatinine diffuses of inappropiate antidiuretic harmone) 10:1) WITH INCREASED CREATININE: up (accelerates conversion of creating eleases muscle creatinine). who develop renal failure. b: reased BUN/creatinine ratio). rapy (interferes with creatinine measu JLAR FILTERATION RATE: DESCRIPTION Normal kidney function Kidney damage with normal or high GFR Mild decrease in GFR	than creatinine) (e.g. obstructiv but of extracellular fluid). blood). due to tubular secretion of ure e to creatinine). the in creatinine with certain me irement). GFR (mL/min/1.73m2) >90 >90 60 -89	a. thodologies,resulting in nor ASSOCIATED FINDINGS No proteinuria Presence of Protein ,		





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	Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Patholog		(Pathology)
NAME	: Mr. PRAVEEN KUMAR		
AGE/ GENDER	: 55 YRS/MALE	PATIENT ID	: 1732418
COLLECTED BY	:	REG. NO./LAB NO.	: 012501230044
REFERRED BY	: Dr. N.C.WADHAWAN (AMBALA CANTT)	REGISTRATION DATE	: 23/Jan/2025 12:41 PM
BARCODE NO.	: 01524311	COLLECTION DATE	: 23/Jan/2025 12:42PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 23/Jan/2025 04:54PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANT	ГТ	
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

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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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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT		
		Valaa	Unit	Biological Reference interva
	ATING HORMONE (TSH): SERU	DID STIMULAT	CINCLOGY CING HORMONE (TS μIU/mL	
THYROID STIMUL by CMIA (CHEMILUMI Brd GENERATION, UL1	ATING HORMONE (TSH): SERU	ENDOCE DID STIMULAT	RINOLOGY FING HORMONE (TS	5H)
THYROID STIMUL by CMIA (CHEMILUMII Brd GENERATION, UL1	ATING HORMONE (TSH): SERU NESCENT MICROPARTICLE IMMUNOA TRASENSITIVE	ENDOCE DID STIMULAT	RINOLOGY F ING HORMONE (TS μIU/mL	SH) 0.35 - 5.50
THYROID STIMUL by CMIA (CHEMILUMII Brd GENERATION, UL1	ATING HORMONE (TSH): SERU	ENDOCE DID STIMULAT	RINOLOGY FING HORMONE (TS	SH) 0.35 - 5.50 (µlU/mL)
THYROID STIMUL by CMIA (CHEMILUMII Brd GENERATION, UL1	ATING HORMONE (TSH): SERU NESCENT MICROPARTICLE IMMUNOAS TRASENSITIVE AGE	ENDOCE DID STIMULAT	REFFERENCE RANGE	бН) 0.35 - 5.50 (µlU/mL)
THYROID STIMUL by CMIA (CHEMILUMI Brd GENERATION, UL1	ATING HORMONE (TSH): SERU VESCENT MICROPARTICLE IMMUNOAS TRASENSITIVE AGE 0 – 5 DAYS 6 Days – 2 Months 3 – 11 Months	ENDOCE DID STIMULAT	REFFERENCE RANGE 0.70 – 15.20 0.70 – 8.40	бН) 0.35 - 5.50 (µlU/mL)
THYROID STIMUL by CMIA (CHEMILUMII Brd GENERATION, UL1	ATING HORMONE (TSH): SERU VESCENT MICROPARTICLE IMMUNOAS RASENSITIVE AGE 0 – 5 DAYS 6 Days – 2 Months 3 – 11 Months 1 – 5 Years	ENDOCE DID STIMULAT	REFFERENCE RANGE 0.70 – 15.20 0.70 – 11.00 0.70 – 8.40 0.70 – 7.00	бН) 0.35 - 5.50 (µlU/mL)
THYROID STIMUL by CMIA (CHEMILUMII Brd GENERATION, UL1	ATING HORMONE (TSH): SERU VESCENT MICROPARTICLE IMMUNOAS RASENSITIVE AGE 0 – 5 DAYS 6 Days – 2 Months 3 – 11 Months 1 – 5 Years 6 – 10 Years	ENDOCE DID STIMULAT	REFFERENCE RANGE 0.70 – 15.20 0.70 – 11.00 0.70 – 8.40 0.70 – 7.00 0.60 – 5.50	бН) 0.35 - 5.50 (µlU/mL)
THYROID STIMUL	ATING HORMONE (TSH): SERU VESCENT MICROPARTICLE IMMUNOAS TRASENSITIVE AGE 0 – 5 DAYS 6 Days – 2 Months 3 – 11 Months 1 – 5 Years 6 – 10 Years 11 - 15	ENDOCE DID STIMULAT	REFFERENCE RANGE 0.70 – 15.20 0.70 – 15.20 0.70 – 11.00 0.70 – 8.40 0.70 – 7.00 0.60 – 5.50 0.50 – 5.50	бН) 0.35 - 5.50 (µlU/mL)
THYROID STIMUL by CMIA (CHEMILUMII Brd GENERATION, UL1	ATING HORMONE (TSH): SERU VESCENT MICROPARTICLE IMMUNOAS RASENSITIVE AGE 0 – 5 DAYS 6 Days – 2 Months 3 – 11 Months 1 – 5 Years 6 – 10 Years	ENDOCR DID STIMULAT JM 1.519 SSAY)	REFFERENCE RANGE 0.70 – 15.20 0.70 – 11.00 0.70 – 8.40 0.70 – 7.00 0.60 – 5.50	бН) 0.35 - 5.50 (µlU/mL)
THYROID STIMUL by CMIA (CHEMILUMII Brd GENERATION, UL1	ATING HORMONE (TSH): SERU VESCENT MICROPARTICLE IMMUNOAS TRASENSITIVE AGE 0 – 5 DAYS 6 Days – 2 Months 3 – 11 Months 1 – 5 Years 6 – 10 Years 11 - 15 > 20 Years (Adults)	ENDOCE DID STIMULAT	REFFERENCE RANGE 0.70 – 15.20 0.70 – 15.20 0.70 – 11.00 0.70 – 8.40 0.70 – 7.00 0.60 – 5.50 0.50 – 5.50 0.27 – 5.50	бН) 0.35 - 5.50 (µlU/mL)
THYROID STIMUL by CMIA (CHEMILUMII 3rd GENERATION, UL1	ATING HORMONE (TSH): SERU VESCENT MICROPARTICLE IMMUNOAS TRASENSITIVE AGE 0 – 5 DAYS 6 Days – 2 Months 3 – 11 Months 1 – 5 Years 6 – 10 Years 11 - 15	ENDOCR DID STIMULAT JM 1.519 SSAY)	REFFERENCE RANGE 0.70 – 15.20 0.70 – 15.20 0.70 – 11.00 0.70 – 8.40 0.70 – 7.00 0.60 – 5.50 0.50 – 5.50	бН) 0.35 - 5.50 (µlU/mL)

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USE:- TSH controls biosynthesis and release of thyroid harmones T4 & T3. It is a sensitive measure of thyroid function, especially useful in early or subclinical hypothyroidism, before the patient develops any clinical findings or goitre or any other thyroid function abnormality. **INCREASED LEVELS**:

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

2. Hypothyroid patients receiving insufficient thyroid replacement therapy.

3. Hashimotos thyroiditis.

4.DRUGS: Amphetamines, lodine containing agents and dopamine antagonist.

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

DECREASED LEVELS:

1. Toxic multi-nodular goitre & Thyroiditis.

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

3. Autonomously functioning Thyroid adenoma

4. Secondary pituatary or hypothalmic hypothyroidism

5. Acute psychiatric illness

6.Severe dehydration.

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



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NAME	: Mr. PRAVEEN KUMAR		
AGE/ GENDER	: 55 YRS/MALE	PATIENT ID	: 1732418
COLLECTED BY	:	REG. NO./LAB NO.	: 012501230044
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CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 23/Jan/2025 04:55PM
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Test Name	Value	Unit	Biological Reference interval

8.Pregnancy: 1st and 2nd Trimester LIMITATIONS:

1.TSH may be normal in central hypothyroidism, recent rapid correction of hyperthyroidism or hypothyroidism, pregnancy, phenytoin therapy. 2. Autoimmune disorders may produce spurious results.



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	Chairman & Cons	opra Microbiology) ultant Pathologist		n Chopra (Pathology) Pathologist
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CLIENT ADDRESS :	6349/1, NICHOLSON ROAD, A	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		CLINICAL PA	THOLOGY	
	URINE ROI		SCOPIC EXAMINA	ATION
PHYSICAL EXAMINA				
QUANTITY RECIEVED		10	ml	
by DIP STICK/REFLECTAI	NCE SPECTROPHOTOMETRY	PALE YELLO	A.7	PALE YELLOW
	NCE SPECTROPHOTOMETRY	FALE IELLO	vv	FALE TELEOW
TRANSPARANCY	NCE SPECTROPHOTOMETRY	CLEAR		CLEAR
SPECIFIC GRAVITY		>=1.030		1.002 - 1.030
by DIP STICK/REFLECTAI	NCE SPECTROPHOTOMETRY			
REACTION	<u>IIION</u>	ACIDIC		
by DIP STICK/REFLECTAI	NCE SPECTROPHOTOMETRY			
PROTEIN by DIP STICK/REFLECTAI	NCE SPECTROPHOTOMETRY	Trace		NEGATIVE (-ve)
SUGAR		Negative		NEGATIVE (-ve)
pH	NCE SPECTROPHOTOMETRY	5.5		5.0 - 7.5
	NCE SPECTROPHOTOMETRY	Nation		
BILIRUBIN by DIP STICK/REFLECTAI	NCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
NITRITE	NCE SPECTROPHOTOMETRY.	Negative		NEGATIVE (-ve)
UROBILINOGEN		Normal	EU/dL	0.2 - 1.0
by DIP STICK/REFLECTAI KETONE BODIES	NCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
by DIP STICK/REFLECTAI	NCE SPECTROPHOTOMETRY			
BLOOD by DIP STICK/REFLECTAI	NCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
ASCORBIC ACID	NCE SPECTROPHOTOMETRY	NEGATIVE (-	ve)	NEGATIVE (-ve)
RED BLOOD CELLS (R		NEGATIVE (-	ve) /HPF	0 - 3



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EXCELLENCE IN HEALTHCARE & DIAGNOSTICS

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

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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
by MICROSCOPY ON O	CENTRIFUGED URINARY SEDIMENT			
PUS CELLS by MICROSCOPY ON (CENTRIFUGED URINARY SEDIMENT	2-4	/HPF	0 - 5
EPITHELIAL CELLS	S CENTRIFUGED URINARY SEDIMENT	0-3	/HPF	ABSENT
CDVCTAIC				

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

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT

** End Of Report ***



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