



	Dr. Vinay Chopra MD (Pathology & Micr Chairman & Consultar	obiology)		(Pathology)
NAME	: Mr. SUBHASH			
AGE/ GENDER	: 43 YRS/MALE		PATIENT ID	: 1665157
COLLECTED BY	:		REG. NO./LAB NO.	:012411080033
REFERRED BY	:		REGISTRATION DATE	: 08/Nov/2024 10:24 AM
BARCODE NO.	: 01520363		COLLECTION DATE	: 08/Nov/2024 10:27AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 08/Nov/2024 10:52AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMB/	ALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	SWAST	HYA WE	LLNESS PANEL: 1.	2
			OOD COUNT (CBC)	-
RED BLOOD CELLS	(RBCS) COUNT AND INDICES			
HAEMOGLOBIN (HI		9.8 ^L	gm/dL	12.0 - 17.0
by CALORIMETRIC RED BLOOD CELL (1	DDC) COUNT			250 500
	COUNT OCUSING, ELECTRICAL IMPEDENCE	4.37	Millions	/cmm 3.50 - 5.00
PACKED CELL VOLU	JME (PCV) utomated hematology analyzer	32.9 ^L	%	40.0 - 54.0
MEAN CORPUSCUL	AR VOLUME (MCV)	75.3 ^L	fL	80.0 - 100.0
	UTOMATED HEMATOLOGY ANALYZER AR HAEMOGLOBIN (MCH)	22.4 ^L	pg	27.0 - 34.0
by CALCULATED BY A	UTOMATED HEMATOLOGY ANALYZER			
	AR HEMOGLOBIN CONC. (MCHC) UTOMATED HEMATOLOGY ANALYZER	29.7 ^L	g/dL	32.0 - 36.0
RED CELL DISTRIB	UTION WIDTH (RDW-CV)	20.1 ^H	%	11.00 - 16.00
,	utomated hematology analyzer JTION WIDTH (RDW-SD)	54.4	fL	35.0 - 56.0
by CALCULATED BY A	UTOMATED HEMATOLOGY ANALYZER			
MENTZERS INDEX by CALCULATED		17.23	RATIO	BETA THALASSEMIA TRAIT: < 13.0
				IRON DEFICIENCY ANEMIA:
GREEN & KING IND	FX	34.59	RATIO	>13.0 BETA THALASSEMIA TRAIT:<
by CALCULATED		51.53	INATIO	65.0
				IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CEI	LLS (WBCS)			05.0
FOTAL LEUCOCYTE	COUNT (TLC)	7630	/cmm	4000 - 11000
	' BY SF CUBE & MICROSCOPY LOOD CELLS (nRBCS)	NIL		0.00 - 20.00
	T HEMATOLOGY ANALYZER	INTE		0.00 - 20.00
by AUTOMATED 6 PAR	LOOD CELLS (nRBCS) %	NIL	%	< 10 %





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



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Dr. Vinay Chopra

MD (Pathology & Microbiology) Chairman & Consultant Pathologist



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

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Test Name	Value	Unit	Biological Reference interval
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	63	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	24	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	6 ^H	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	7	%	2 - 12
BASOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	%	0 - 1
ABSOLUTE LEUKOCYTES (WBC) COUNT			
ABSOLUTE NEUTROPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	4807	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1831	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by flow cytometry by sf cube & microscopy	458 ^H	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	534	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110
PLATELETS AND OTHER PLATELET PREDICTIVE	MARKERS.		
PLATELET COUNT (PLT) by hydro dynamic focusing, electrical impedence	498000 ^H	/cmm	150000 - 450000
PLATELETCRIT (PCT) by hydro dynamic focusing, electrical impedence	0.49 ^H	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by hydro dynamic focusing, electrical impedence	10	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by hydro dynamic focusing, electrical impedence	135000 ^H	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by hydro dynamic focusing, electrical impedence	27.2	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by hydro dynamic focusing, electrical impedence NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD	15.7	%	15.0 - 17.0



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Test Name	Value	Unit	Biological Reference interval

Test Name

RECHECKED



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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



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CLIENT ADDRESS	: 6349/1, NICHO	LSON ROAD, AME	BALA CANTT		
Test Name			Value	Unit	Biological Reference interval
Immune disease, but 2. An ESR can be affe as C-reactive protein 3. This test may also systemic lupus erythin CONDITION WITH LO A low ESR can be see (polycythaemia), sigr as sickle cells in sickl NOTE: 1. ESR and C - reactiv	does not tell the he ected by other cond be used to monitor ematosus W ESR en with conditions t hificantly high white le cell anaemia) als re protein (C-RP) are	ealth practitioner itions besides infla disease activity a hat inhibit the nor e blood cell count o lower the ESR. e both markers of	exactly where ammation. For and response t rmal sediment (leucocytosis) inflammation.	the inflammation is in the this reason, the ESR is ty o therapy in both of the a ation of red blood cells s	ion associated with infection, cancer and auto e 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 (suc
 CRP is not affected If the ESR is elevat Women tend to ha 	I by as many other f ed, it is typically a r ave a higher ESR, an tran, methyldopa, c	actors as is ESR, m result of two types d menstruation ar oral contraceptives	aking it a bett s of proteins, g nd pregnancy of	er marker of inflammation plobulins or fibrinogen. an cause temporary eleva	1.





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		hopra & Microbiology) onsultant Pathologist	Dr. Yugam MD CEO & Consultant	(Pathology)
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Test Name		Value	Unit	Biological Reference interval
	CLINI	ICAL CHEMISTRY/	BIOCHEMIST	RY
		GLUCOSE FAST	ING (F)	

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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MBBS, MD (PATHOLOGY)

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				å DIAGNOSTICS	
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD), AMBALA CANTT			
Test Name		Value	Unit	Biological Reference interval	
		LIPID PR	OFILE : BASIC		
CHOLESTEROL TOT by CHOLESTEROL OX		151.72	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0	
TRIGLYCERIDES: SH by GLYCEROL PHOSPI	ERUM HATE OXIDASE (ENZYMATIC)	103.13	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0	
HDL CHOLESTEROL by SELECTIVE INHIBITI	. (DIRECT): SERUM on	43.36	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0	
DL CHOLESTEROL by CALCULATED, SPEC		87.73	mg/dL	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 CHOLEST by calculated, spec		108.36	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	
LDL CHOLESTERO		20.63	mg/dL	0.00 - 45.00	
OTAL LIPIDS: SER by CALCULATED, SPEC	UM	406.57	mg/dL	350.00 - 700.00	
by CALCULATED, SPEC	L RATIO: SERUM	3.5	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0	

CLIENT ADDRESS

by CALCULATED, SPECTROPHOTOMETRY	
NON HDL CHOLESTEROL: SERUM	
by CALCULATED, SPECTROPHOTOMETRY	

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Test Name		Value	Unit	Biological Reference interval
LDL/HDL RATIO: S by CALCULATED, SPE		2.02	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/H by CALCULATED, SPE		2.38 ^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

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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Biological Reference interval

Dr. Vinay Chopra Dr. Yugam Chopra MD (Pathology & Microbiology) MD (Pathology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** : Mr. SUBHASH AGE/ GENDER : 43 YRS/MALE **PATIENT ID COLLECTED BY** REG. NO./LAB NO. : **REFERRED BY REGISTRATION DATE** : **BARCODE NO.** :01520363 **COLLECTION DATE** CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Unit

LIVER	FUNCTION TEST (CO	MPLETE)	
BILIRUBIN TOTAL: SERUM by DIAZOTIZATION, SPECTROPHOTOMETRY	0.69	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.14	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by Calculated, spectrophotometry	0.55	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	24.7	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	25.8	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by calculated, spectrophotometry	0.96	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL	108.06	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by szasz, spectrophtometry	15.89	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by biuret, spectrophotometry	7.62	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	4.31	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	3.31	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by Calculated, spectrophotometry	1.3	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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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 T	EST (COMPLETE)	
UREA: SERUM		23.5	mg/dL	10.00 - 50.00
-	NATE DEHYDROGENASE (GLDH)	0.05		0.40 1.40
CREATININE: SERI		0.95	mg/dL	0.40 - 1.40
	ROGEN (BUN): SERUM	10.98	mg/dL	7.0 - 25.0
	ROGEN (BUN)/CREATININE	11.56	RATIO	10.0 - 20.0
RATIO: SERUM				
by CALCULATED, SPE UREA/CREATININ		24.74	RATIO	
by CALCULATED, SPE	ECTROPHOTOMETRY			
URIC ACID: SERUM		6.97	mg/dL	3.60 - 7.70
CALCIUM: SERUM	SET ERONIDAGE	10.16	mg/dL	8.50 - 10.60
by ARSENAZO III, SPE		2.20		2 20 4 70
PHOSPHOROUS: SE by PHOSPHOMOLYBE	ZKUM DATE, SPECTROPHOTOMETRY	3.29	mg/dL	2.30 - 4.70
ELECTROLYTES				
SODIUM: SERUM		142.6	mmol/L	135.0 - 150.0
by ISE (ION SELECTIV POTASSIUM: SERU		4.58	mmol/L	3.50 - 5.00
by ISE (ION SELECTIV	/E ELECTRODE)			
CHLORIDE: SERUM by ISE (ION SELECTIVE ELECTRODE)		106.95	mmol/L	90.0 - 110.0
	<u>MERULAR FILTERATION RATE</u>			
(eGFR): SERUM by CALCULATED	ERULAR FILTERATION RATE	101.9		
ESTIMATED GLOM (eGFR): SERUM		101.9		

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.





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CLIENT ADDRESS	. 0349/ I, MCHOLSON KOAD,	, AMDALA CAN I I			
Test Name		Value	Unit	Biologic	cal Reference interva
INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia	ass (subnormal creatinine prod tetracycline, glucocorticoids) (0:1) WITH ELEVATED CREATININ (BUN rises disproportionately i superimposed on renal disease (0:1) WITH DECREASED BUN :	IE LEVELS: more than creatinine) (e.g.	obstructive uropa	ithy).	
INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (< 1. Acute tubular necr 2. Low protein diet ar 3. Severe liver diseas 4. Other causes of de 5. Repeated dialysis (6. Inherited hyperam 7. SIADH (syndrome of 8. Pregnancy. DECREASED RATIO (< 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERI CKD STAGE	tetracycline, glucocorticoids) i0:1) WITH ELEVATED CREATININ a (BUN rises disproportionately is superimposed on renal disease i0:1) WITH DECREASED BUN : osis. a starvation. b. creased urea synthesis. urea rather than creatinine diff monemias (urea is virtually absorb inappropiate antidiuretic harr i0:1) WITH INCREASED CREATINI py (accelerates conversion of cr eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false in creased BUN/creatinine ratio). Tapy (interferes with creatinine in JLAR FILTERATION RATE: DESCRIPTION	JE LEVELS: more than creatinine) (e.g. s. fuses out of extracellular flu sent in blood). mone) due to tubular secret INE: reatine to creatinine). ncrease in creatinine with c measurement). GFR (mL/min/1	iid). ion of urea. ertain methodolo	ogies,resulting in norn SOCIATED FINDINGS	nal ratio when dehydra
INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (< 1. Acute tubular necr 2. Low protein diet an 3. Severe liver diseas 4. Other causes of de 5. Repeated dialysis (6. Inherited hyperam 7. SIADH (syndrome of 8. Pregnancy. DECREASED RATIO (< 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERI CKD STAGE G1	tetracycline, glucocorticoids) i0:1) WITH ELEVATED CREATININ a (BUN rises disproportionately is superimposed on renal disease i0:1) WITH DECREASED BUN : osis. a starvation. b. creased urea synthesis. urea rather than creatinine diff monemias (urea is virtually absorb inappropiate antidiuretic harr i0:1) WITH INCREASED CREATINI py (accelerates conversion of cre eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false in creased BUN/creatinine ratio). apy (interferes with creatinine in JLAR FILTERATION RATE: <u>DESCRIPTION</u> Normal kidney fund	JE LEVELS: more than creatinine) (e.g. s. fuses out of extracellular flu sent in blood). mone) due to tubular secret INE: reatine to creatinine). ncrease in creatinine with c measurement). GFR (mL/min/1 ction >90	iid). ion of urea. ertain methodolo 73m2) AS	ogies,resulting in norn SOCIATED FINDINGS No proteinuria	nal ratio when dehydra
INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (< 1. Acute tubular necr 2. Low protein diet ar 3. Severe liver diseas 4. Other causes of de 5. Repeated dialysis (6. Inherited hyperam 7. SIADH (syndrome of 8. Pregnancy. DECREASED RATIO (< 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERI OKD STAGE	tetracycline, glucocorticoids) i0:1) WITH ELEVATED CREATININ a (BUN rises disproportionately is superimposed on renal disease i0:1) WITH DECREASED BUN : osis. and starvation. b. creased urea synthesis. urea rather than creatinine diff monemias (urea is virtually absorb inappropiate antidiuretic harrist i0:1) WITH INCREASED CREATINI py (accelerates conversion of cre eleases muscle creatinine). who develop renal failure. i: sis (acetoacetate causes false in creased BUN/creatinine ratio). apy (interferes with creatinine) JLAR FILTERATION RATE: DESCRIPTION Normal kidney fund Kidney damage w	JE LEVELS: more than creatinine) (e.g. s. fuses out of extracellular flu sent in blood). mone) due to tubular secret INE: reatine to creatinine). ncrease in creatinine with c measurement). GFR (mL/min/1 ction >90 vith >90	iid). ion of urea. ertain methodolo 73m2) AS	ogies,resulting in norm SOCIATED FINDINGS No proteinuria resence of Protein ,	
INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (< 1. Acute tubular necr 2. Low protein diet an 3. Severe liver diseas 4. Other causes of de 5. Repeated dialysis (6. Inherited hyperam 7. SIADH (syndrome of 8. Pregnancy. DECREASED RATIO (< 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERI CKD STAGE G1	tetracycline, glucocorticoids) i0:1) WITH ELEVATED CREATININ a (BUN rises disproportionately is superimposed on renal disease i0:1) WITH DECREASED BUN : osis. a starvation. b. creased urea synthesis. urea rather than creatinine diff monemias (urea is virtually absorb inappropiate antidiuretic harr i0:1) WITH INCREASED CREATINI py (accelerates conversion of cre eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false in creased BUN/creatinine ratio). apy (interferes with creatinine in JLAR FILTERATION RATE: <u>DESCRIPTION</u> Normal kidney fund	JE LEVELS: more than creatinine) (e.g. sent in blood). mone) due to tubular secret INE: reatine to creatinine). ncrease in creatinine with c measurement). GFR (mL/min/1 ction >90 vith >90 FR	iid). ion of urea. ertain methodolo 73m2) AS	ogies,resulting in norn SOCIATED FINDINGS No proteinuria	
INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (< 1. Acute tubular necr 2. Low protein diet ar 3. Severe liver diseas 4. Other causes of de 5. Repeated dialysis (6. Inherited hyperam 7. SIADH (syndrome of 8. Pregnancy. DECREASED RATIO (< 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERI G1 G2 G3a G3b	tetracycline, glucocorticoids) i0:1) WITH ELEVATED CREATININ a (BUN rises disproportionately is superimposed on renal disease i0:1) WITH DECREASED BUN : osis. and starvation. b. creased urea synthesis. urea rather than creatinine diff monemias (urea is virtually absorb inappropiate antidiuretic harrist i0:1) WITH INCREASED CREATINI py (accelerates conversion of cre eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false in creased BUN/creatinine ratio). apy (interferes with creatinine) LAR FILTERATION RATE: DESCRIPTION Normal kidney function Kidney damage w normal or high G Moderate decrease in C	JE LEVELS: more than creatinine) (e.g. fuses out of extracellular flu sent in blood). mone) due to tubular secret INE: reatine to creatinine). INC measurement). GFR (mL/min/1 ction >90 vith >90 GFR 60 -89 in GFR 30-59	iid). ion of urea. ertain methodolo 73m2) AS	ogies,resulting in norm SOCIATED FINDINGS No proteinuria resence of Protein ,	
INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (< 1. Acute tubular necr 2. Low protein diet ar 3. Severe liver diseas 4. Other causes of de 5. Repeated dialysis (6. Inherited hyperam 7. SIADH (syndrome of 8. Pregnancy. DECREASED RATIO (< 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERI CKD STAGE G1 G2 G3a	tetracycline, glucocorticoids) i0:1) WITH ELEVATED CREATININ a (BUN rises disproportionately is superimposed on renal disease i0:1) WITH DECREASED BUN : osis. and starvation. b. creased urea synthesis. urea rather than creatinine diff monemias (urea is virtually absorb inappropiate antidiuretic harrist i0:1) WITH INCREASED CREATINI py (accelerates conversion of cre eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false in creased BUN/creatinine ratio). apy (interferes with creatinine) ULAR FILTERATION RATE: DESCRIPTION Normal kidney function Kidney damage w normal or high G Mild decrease in (0)	JE LEVELS: more than creatinine) (e.g. s. fuses out of extracellular flu- sent in blood). mone) due to tubular secret INE: reatine to creatinine). INE: reatine to creatinine with con- measurement). GFR (mL/min/1 ction >90 /ith >90 /ith >90 /ith >90 /ith 30 /ith	iid). ion of urea. ertain methodolo 73m2) AS	ogies,resulting in norm SOCIATED FINDINGS No proteinuria resence of Protein ,	



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

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









Test Name		Value Unit	Biological Reference interval
	···· , · · · · , ·		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMI	BALA CANTT	
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 08/Nov/2024 11:51AM
BARCODE NO.	: 01520363	COLLECTION DATE	:08/Nov/2024 10:27AM
REFERRED BY	:	REGISTRATION DATE	: 08/Nov/2024 10:24 AM
COLLECTED BY	:	REG. NO./LAB NO.	: 012411080033
AGE/ GENDER	: 43 YRS/MALE	PATIENT ID	: 1665157
NAME	: Mr. SUBHASH		
	MD (Pathology & Mic Chairman & Consulta	crobiology) MI	D (Pathology)
	Dr. Vinay Chop	ra I Dr. Yuga	m Chopra

COMMENTS:

Estimated Glomerular filtration rate (eGFR) is the sum of filtration rates in all functioning nephrons and so an estimation of the GFR provides a measure of functioning nephrons of the kidney.
 eGFR calculated using the 2009 CKD-EPI creatinine equation and GFR category reported as per KDIGO guideline 2012
 In patients, with eGFR creatinine between 45-59 ml/min/1.73 m2 (G3) and without any marker of Kidney damage, It is recommended to measure of CFD with the commended to measure

3. In patients, with eGFR cleaning between 45-59 minimit 1.73 m2 (G3) and without any marker of Kidney damage, it is recommended to measure eGFR with Cystatin C for confirmation of CKD
4. eGFR category G1 OR G2 does not fulfill the criteria for CKD, in the absence of evidence of Kidney Damage
5. In a suspected case of Acute Kidney Injury (AKI), measurement of eGFR should be done after 48-96 hours of any Intervention or procedure
6. eGFR calculated by Serum Creatinine may be less accurate due to certain factors like Race, Muscle Mass, Diet, Certain Drugs. In such cases, eGFR should be calculated using Serum Cystatin C
7. A decrease in eGFR implies either progressive renal disease, or a reversible process causing decreased nephron function (eg, severe dehydration).

ADVICE:

KDIGO guideline, 2012 recommends Chronic Kidney Disease (CKD) should be classified based on cause, eGFR category and Albuminuria (ACR) category. GFR & ACR category combined together reflect risk of progression and helps Clinician to identify the individual who are progressing at more rapid rate than anticipated



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

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

KOS Central Lab: 6349/1, Nicholson Road, Ambala Cantt -133 001, Haryana KOS Molecular Lab: IInd Floor, Parry Hotel, Staff Road, Opp. GPO, Ambala Cantt -133 001, Haryana 0171-2643898, +91 99910 43898 care@koshealthcare.com www.koshealthcare.com







	Dr. Vinay Ch MD (Pathology & Chairman & Con		M	m Chopra D (Pathology) nt Pathologist
NAME	: Mr. SUBHASH			
AGE/ GENDER	: 43 YRS/MALE		PATIENT ID	: 1665157
COLLECTED BY	:		REG. NO./LAB NO.	: 012411080033
REFERRED BY	:		REGISTRATION DATE	: 08/Nov/2024 10:24 AM
BARCODE NO.	: 01520363		COLLECTION DATE	: 08/Nov/2024 10:27AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	:08/Nov/2024 11:48AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interv
		ENDOCI	RINOLOGY	
	TH	YROID FUNC	FION TEST: TOTAL	
TRIIODOTHYRONII		0.638	ng/mL	0.35 - 1.93
THYROXINE (T4): S	ESCENT MICROPARTICLE IMMUNOA ERUM ESCENT MICROPARTICLE IMMUNOA	5.34	µgm/d	L 4.87 - 12.60
	TING HORMONE (TSH): SERU		µIU/m	L 0.35 - 5.50
by CMIA (CHEMILUMIN 3rd GENERATION, ULT	ESCENT MICROPARTICLE IMMUNOA RASENSITIVE	SSAY)		
INTERPRETATION:				
day has influence on the i triiodothyronine (T3).Fai	measured serum TSH concentrations. TS	SH stimulates the pro	duction and secretion of the	pm. The variation is of the order of 50%.Hence time of metabolically active hormones, thyroxine (T4)and ther underproduction (hypothyroidism) or
CLINICAL CONDITION	T3		T4	TSH
Primary Hypothyroidis	n: Reduced		Reduced	Increased (Significantly)
Subclinical Hypothyroi	dism: Normal or Low	Normal	Normal or Low Normal	High

111	ЛІТД	TIC)NS:	-

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	YRONINE (T3)	THYROXINE (T4)		THYROID STIMULATING 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)

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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT





	Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologis		(Pathology)
NAME	: Mr. SUBHASH		
AGE/ GENDER	: 43 YRS/MALE	PATIENT ID	: 1665157
COLLECTED BY	:	REG. NO./LAB NO.	: 012411080033
REFERRED BY	:	REGISTRATION DATE	: 08/Nov/2024 10:24 AM
BARCODE NO.	: 01520363	COLLECTION DATE	: 08/Nov/2024 10:27AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	:08/Nov/2024 11:48AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Cest 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





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

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

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		hopra & Microbiology) Insultant Pathologist	Dr. Yugan MD CEO & Consultant	(Pathology)
NAME	: Mr. SUBHASH			
AGE/ GENDER	: 43 YRS/MALE	PA	TIENT ID	: 1665157
COLLECTED BY	:	RE	G. NO./LAB NO.	: 012411080033
REFERRED BY	:	RE	GISTRATION DATE	: 08/Nov/2024 10:24 AM
BARCODE NO.	: 01520363	CO	LLECTION DATE	:08/Nov/2024 10:27AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	RE	PORTING DATE	: 08/Nov/2024 11:29AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD), AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		CLINICAL PA	THOLOGY	
	URINE R		SCOPIC EXAMINA	ATION
PHYSICAL EXAMIN				
QUANTITY RECIEV		10	ml	
	TANCE SPECTROPHOTOMETRY		OW	
COLOUR by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	AMBER YELI	2010	PALE YELLOW
TRANSPARANCY		CLEAR		CLEAR
SPECIFIC GRAVITY	TANCE SPECTROPHOTOMETRY	1.01		1.002 - 1.030
	TANCE SPECTROPHOTOMETRY			
CHEMICAL EXAMI	NATION	ACIDIC		
REACTION by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	ACIDIC		
PROTEIN	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
SUGAR	TANCE SPECIFICITIONETRY	Negative		NEGATIVE (-ve)
•	TANCE SPECTROPHOTOMETRY			50 75
pH by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	5.5		5.0 - 7.5
BILIRUBIN	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
NITRITE	TANCE SPECTROPHOTOMETRT	Negative		NEGATIVE (-ve)
	TANCE SPECTROPHOTOMETRY.	Normal		
UROBILINOGEN by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	Normai	EU/dL	0.2 - 1.0
KETONE BODIES	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
BLOOD	IANGE SPECIKOPHUIOMEIRY	Negative		NEGATIVE (-ve)
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	Ũ		
	TANCE SPECTROPHOTOMETRY	NEGATIVE (-	vej	NEGATIVE (-ve)
MICROSCOPIC EXA	MINATION			
RED BLOOD CELLS	(RBCs)	NEGATIVE (-	ve) /HPF	0 - 3





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

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 KOS Molecular Lab: IInd Floor, Parry Hotel, Staff Road, Opp. GPO, Ambala Cantt -133 001, Haryana

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

: Mr. SUBHASH		
: 43 YRS/MALE	PATIENT ID	: 1665157
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: KOS DIAGNOSTIC LAB	REPORTING DATE	: 08/Nov/2024 11:29AM
: 6349/1, NICHOLSON ROAD, AMBALA CANTT	,	
Value	Unit	Biological Reference interval
	: 43 YRS/MALE : : : 01520363 : KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON ROAD, AMBALA CANTT	: 43 YRS/MALEPATIENT ID:REG. NO./LAB NO.:REGISTRATION DATE: 01520363COLLECTION DATE: KOS DIAGNOSTIC LABREPORTING DATE: 6349/1, NICHOLSON ROAD, AMBALA CANTT

3-4	/HPF	0 - 5
1-3	/HPF	ABSENT
NEGATIVE (-ve)		NEGATIVE (-ve)
ABSENT		ABSENT
	1-3 NEGATIVE (-ve) NEGATIVE (-ve) NEGATIVE (-ve) NEGATIVE (-ve)	1-3 /HPF NEGATIVE (-ve) NEGATIVE (-ve) NEGATIVE (-ve) NEGATIVE (-ve)

** 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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