

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



	Chopra gy & Microbiology) Consultant Pathologist	Dr. Yugam MD (CEO & Consultant	(Pathology)
NAME: Mr. MOHIT PAL SINGHAGE/ GENDER: 30 YRS/MALECOLLECTED BY: .REFERRED BY: .BARCODE NO.: 01517175CLIENT CODE.: KOS DIAGNOSTIC LABCLIENT ADDRESS: 6349/1, NICHOLSON ROA	RE RE CO RE	FIENT ID G. NO./LAB NO. GISTRATION DATE LLECTION DATE PORTING DATE	: 1616887 : 012409180016 : 18/Sep/2024 09:03 AM : 18/Sep/2024 09:04AM : 18/Sep/2024 09:58AM
Test Name	Value	Unit	Biological Reference interval
	SWASTHYA WELLI COMPLETE BLOOD		
RED BLOOD CELLS (RBCS) COUNT AND INDICES			
HAEMOGLOBIN (HB) by calorimetric	14.3	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT	5.43 ^H	Millions/c	mm 3.50 - 5.00
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDI PACKED CELL VOLUME (PCV)	44.7	%	40.0 - 54.0
by CALCULATED BY AUTOMATED HEMATOLOGY ANAL MEAN CORPUSCULAR VOLUME (MCV)	LYZER 82.3	fL	80.0 - 100.0
by CALCULATED BY AUTOMATED HEMATOLOGY ANAL		IL.	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by CALCULATED BY AUTOMATED HEMATOLOGY ANA	26.3 ^L	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MC	HC) 32	g/dL	32.0 - 36.0
by CALCULATED BY AUTOMATED HEMATOLOGY ANAL RED CELL DISTRIBUTION WIDTH (RDW-CV)	15.1	%	11.00 - 16.00
by CALCULATED BY AUTOMATED HEMATOLOGY ANA	LYZER		
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANAL	46.5 LYZER	fL	35.0 - 56.0
MENTZERS INDEX	15.16	RATIO	BETA THALASSEMIA TRAIT: < 13.0
by CALCULATED GREEN & KING INDEX	22.86	RATIO	IRON DEFICIENCY ANEMIA: >13.0 BETA THALASSEMIA TRAIT:<= 65.0
by CALCULATED	22.00	i i i i i i i i i i i i i i i i i i i	IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CELLS (WBCS)			
TOTAL LEUCOCYTE COUNT (TLC) by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	5920	/cmm	4000 - 11000
NUCLEATED RED BLOOD CELLS (nRBCS)	NIL		0.00 - 20.00
by AUTOMATED 6 PART HEMATOLOGY ANALYZER NUCLEATED RED BLOOD CELLS (nRBCS) %	NIL	%	< 10 %
by CALCULATED BY AUTOMATED HEMATOLOGY ANA		/0	× 10 /0
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS	52	%	50 - 70

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Dr. Yugam Chopra Dr. Vinay Chopra MD (Pathology & Microbiology) MD (Pathology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** NAME : Mr. MOHIT PAL SINGH AGE/ GENDER : 30 YRS/MALE **PATIENT ID** :1616887 **COLLECTED BY** :012409180016 REG. NO./LAB NO. **REFERRED BY REGISTRATION DATE** : 18/Sep/2024 09:03 AM **BARCODE NO.** :01517175 **COLLECTION DATE** :18/Sep/2024 09:04AM CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE** :18/Sep/2024 09:58AM **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Unit **Biological Reference interval** 20 - 40 LYMPHOCYTES 40 % by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY **EOSINOPHILS** 2 % 1-6 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY MONOCYTES % 2 - 12 6 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY BASOPHILS 0 % 0 - 1 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE LEUKOCYTES (WBC) COUNT ABSOLUTE NEUTROPHIL COUNT 3078 /cmm 2000 - 7500 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY 800 - 4900 ABSOLUTE LYMPHOCYTE COUNT 2368 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE EOSINOPHIL COUNT 118 40 - 440 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE MONOCYTE COUNT 355 80 - 880 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE BASOPHIL COUNT 0 - 110 0 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS. 278000 150000 - 450000 PLATELET COUNT (PLT) /cmm by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE 0.10 - 0.36 PLATELETCRIT (PCT) 0.31 % by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE MEAN PLATELET VOLUME (MPV) 11 fL 6.50 - 12.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET LARGE CELL COUNT (P-LCC) 30000 - 90000 91000^H /cmm by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET LARGE CELL RATIO (P-LCR) 32.8 % 11.0 - 45.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET DISTRIBUTION WIDTH (PDW) % 15.0 - 17.0 16 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD



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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	Value	Unit	Biological Reference interval
	FDVTI	HROCYTE SEDIMEN		
immune disease, but 2. An ESR can be affe as C-reactive protein 3. This test may also systemic lupus erythe CONDITION WITH LOV A low ESR can be see (polycythaemia), sigr as sickle cells in sickl NOTE: 1. ESR and C - reactiv 2. Generally, ESR doe 3. CRP is not affected 4. If the ESR is elevat 5. Women tend to ha 6. Drugs such as dext	does not tell the health practitic cted by other conditions besides be used to monitor disease active matosus W ESR n with conditions that inhibit th ificantly high white blood cell c e cell anaemia) also lower the E e protein (C-RP) are both marker is not change as rapidly as does by as many other factors as is ES ed, it is typically a result of two ve a higher ESR, and menstruation	oner exactly where the s inflammation. For this vity and response to the e normal sedimentation count (leucocytosis) , an ESR. rs of inflammation. CRP, either at the start SR, making it a better m types of proteins, globu on and pregnancy can c	inflammation is in the reason, the ESR is typ rapy in both of the at n of red blood cells, su d some protein abnor of inflammation or as arker of inflammation lins or fibrinogen. ause temporary elevat	bicallý used in conjunction with other test such bove diseases as well as some others, such as uch as a high red blood cell count rmalities. Some changes in red cell shape (such tit resolves.





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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD), AMBALA CANTT		Biological Reference interval
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD), AMBALA CANTT	Unit RY/BIOCHEMISTR	Biological Reference interval

KOS Diagnostic Lab (A Unit of KOS Healthcare)

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



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Test Name		Value	Unit	Biological Reference interval
		LIPID PROFILE	: BASIC	
CHOLESTEROL TOTAL by CHOLESTEROL OXI		135.14	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239 HIGH CHOLESTEROL: > OR = 240
TRIGLYCERIDES: SERI	UM HATE OXIDASE (ENZYMATIC)	78.8	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROL (E		55.36	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SI by CALCULATED, SPEC		64.02	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.0 BORDERLINE HIGH: 130.0 - 159 HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0
NON HDL CHOLESTER by CALCULATED, SPEC		79.78	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTEROL: by CALCULATED, SPEC		15.76	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUN		349.08 ^L	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL R by CALCULATED, SPEC	RATIO: SERUM	2.44	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0
LDL/HDL RATIO: SERU		1.16	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0

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CLIENT ADDRESS	: 6349/1, NICHOLSON ROA	D, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
TRIGLYCERIDES/HD		1.42 ^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
	LIVE		N TEST (COMPLETE)	
BILIRUBIN TOTAL: S	ERUM	0.73	mg/dL	INFANT: 0.20 - 8.00
-	PECTROPHOTOMETRY			ADULT: 0.00 - 1.20
	CONJUGATED): SERUM	0.19	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT	(UNCONJUGATED): SERUM	0.54	mg/dL	0.10 - 1.00
SGOT/AST: SERUM	IRIDOXAL PHOSPHATE	128.55 ^H	U/L	7.00 - 45.00
SGPT/ALT: SERUM	IRIDOXAL PHOSPHATE	141.94 ^H	U/L	0.00 - 49.00
AST/ALT RATIO: SER	UM	0.91	RATIO	0.00 - 46.00
ALKALINE PHOSPHA		62.9	U/L	40.0 - 130.0
PROPANOL GAMMA GLUTAMYL by SZASZ, SPECTROF	. TRANSFERASE (GGT): SERUM PHTOMETRY	12.81	U/L	0.00 - 55.0
TOTAL PROTEINS: S		6.18 ^L	gm/dL	6.20 - 8.00
ALBUMIN: SERUM		3.57	gm/dL	3.50 - 5.50
GLOBULIN: SERUM		2.61	gm/dL	2.30 - 3.50

Dr. Vinay Chopra

A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY

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)

1.37





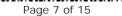
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DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY)



RATIO

1.00 - 2.00







	Dr. Vinay Chopra MD (Pathology & Micro Chairman & Consultan	obiology) MI	m Chopra D (Pathology) nt Pathologist
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Test Name		Value Unit	Biological Reference interval

DECREASED:

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

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

NORMAL	< 0.65
GOOD PROGNOSTIC SIGN	0.3 - 0.6
POOR PROGNOSTIC SIGN	1.2 - 1.6



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Test Name		Value	llmit	Dialogical Deference interval
Test Name		Value	Unit	Biological Reference interval
	KI	DNEY FUNCTION	TEST (COMPLETE)	
UREA: SERUM		25.58	mg/dL	10.00 - 50.00
by UREASE - GLUTAN	NATE DEHYDROGENASE (GLDH)	20.00		
CREATININE: SERUN by ENZYMATIC, SPEC		1.06	mg/dL	0.40 - 1.40
-	GEN (BUN): SERUM	11.95	mg/dL	7.0 - 25.0
•		11.07		
RATIO: SERUM	OGEN (BUN)/CREATININE	11.27	RATIO	10.0 - 20.0
by CALCULATED, SPE	ECTROPHOTOMETRY			
UREA/CREATININE I		24.13	RATIO	
by CALCULATED, SPE URIC ACID: SERUM	ECTROPHOTOMETRY	5.6	mg/dL	3.60 - 7.70
by URICASE - OXIDAS	SE PEROXIDASE			
CALCIUM: SERUM by ARSENAZO III, SPE		9.04	mg/dL	8.50 - 10.60
PHOSPHOROUS: SEF		2.9	mg/dL	2.30 - 4.70
-	DATE, SPECTROPHOTOMETRY			
ELECTROLYTES		1 10 5		105.0.150.0
SODIUM: SERUM by ISE (ION SELECTIN	/E ELECTRODE)	140.5	mmol/L	135.0 - 150.0
POTASSIUM: SERUM		4.06	mmol/L	3.50 - 5.00
by ISE (ION SELECTIN	/E ELECTRODE)	105.20	mmol/l	00.0 110.0
CHLORIDE: SERUM by ISE (ION SELECTIV	/E ELECTRODE)	105.38	mmol/L	90.0 - 110.0
	RULAR FILTERATION RATE			
	RULAR FILTERATION RATE	96.8		
(eGFR): SERUM by CALCULATED				

by CALCULATED 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.



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Test Name		Value	Unit	Biological Ref	ference interval
DECREASED RATIO (<	superimposed on renal diseas 10:1) WITH DECREASED BUN :		ictive uropathy)		
DECREASED RATIO (< 1. Acute tubular necr 2. Low protein diet al 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	10:1) WITH DECREASED BUN : rosis. nd starvation. e. ccreased urea synthesis. (urea rather than creatinine di monemias (urea is virtually ab of inappropiate antidiuretic ha 10:1) WITH INCREASED CREATIN py (accelerates conversion of	iffuses out of extracellular fluid). osent in blood). rmone) due to tubular secretion of NINE:			
DECREASED RATIO (< 1. Acute tubular necr 2. Low protein diet a 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 ir 2. Cephalosporin the	10:1) WITH DECREASED BUN : rosis. and starvation. e. creased urea synthesis. (urea rather than creatinine di monemias (urea is virtually ab of inappropiate antidiuretic ha 10:1) WITH INCREASED CREATIN apy (accelerates conversion of eleases muscle creatinine). who develop renal failure. D: osis (acetoacetate causes false increased BUN/creatinine ratio) rapy (interferes with creatinine	iffuses out of extracellular fluid). osent in blood). rmone) due to tubular secretion of NINE: creatine to creatinine).	urea.		atio when dehydratio
DECREASED RATIO (< 1. Acute tubular necr 2. Low protein diet a 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 ir 2. Cephalosporin the	10:1) WITH DECREASED BUN : rosis. and starvation. e. ccreased urea synthesis. (urea rather than creatinine di monemias (urea is virtually ak of inappropiate antidiuretic ha 10:1) WITH INCREASED CREATIN apy (accelerates conversion of releases muscle creatinine). who develop renal failure. b: visis (acetoacetate causes false icreased BUN/creatinine ratio) rapy (interferes with creatinine JLAR FILTERATION RATE:	iffuses out of extracellular fluid). osent in blood). rmone) due to tubular secretion of NINE: creatine to creatinine). increase in creatinine with certain).	urea. methodologies		atio when dehydratio
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DECREASED RATIO (< 1. Acute tubular necr 2. Low protein diet al 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 ir 2. Cephalosporin the ESTIMATED GLOMERI CKD STAGE	10:1) WITH DECREASED BUN : Tosis. Ind starvation. e. ecreased urea synthesis. (urea rather than creatinine di imonemias (urea is virtually ab of inappropiate antidiuretic ha 10:1) WITH INCREASED CREATIN apy (accelerates conversion of releases muscle creatinine). who develop renal failure. bis (acetoacetate causes false icreased BUN/creatinine ratio) rapy (interferes with creatinine) JLAR FILTERATION RATE: DESCRIPTION	iffuses out of extracellular fluid). osent in blood). rmone) due to tubular secretion of NINE: creatine to creatinine). increase in creatinine with certain). e measurement). N GFR (mL/min/1.73m2 nction >90 with >90	urea. methodologies 2) ASSOCI	resulting in normal ra	atio when dehydratio
CEREASED 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 ir Cephalosporin the STIMATED GLOMERI CKD STAGE G1	10:1) WITH DECREASED BUN : rosis. nd starvation. e. cereased urea synthesis. (urea rather than creatinine diationemias (urea is virtually abof inappropiate antidiuretic hate) 10:1) WITH INCREASED CREATINAL app (accelerates conversion of releases muscle creatinine). who develop renal failure. bis (acetoacetate causes false increased BUN/creatinine ratio) rapy (interferes with creatinine cation) app (interferes with creatinine cation) App (interferes with creation) App (interferes) App (inter	iffuses out of extracellular fluid). osent in blood). rmone) due to tubular secretion of NINE: creatine to creatinine). increase in creatinine with certain). e measurement). N GFR (mL/min/1.73m2 nction >90 with >90 GFR	urea. methodologies 2) ASSOCI	resulting in normal ra ATED FINDINGS proteinuria ce of Protein ,	atio when dehydrat

Severe decrease in GFR Kidney failure

Moderate decrease in GFR

30-59

15-29

<15

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G3b

G4

G5











	Dr. Vinay Chopra MD (Pathology & Microb Chairman & Consultant F	iology) ME	m Chopra D (Pathology) ht Pathologist
NAME	: Mr. MOHIT PAL SINGH		
AGE/ GENDER	: 30 YRS/MALE	PATIENT ID	: 1616887
COLLECTED BY	:	REG. NO./LAB NO.	:012409180016
REFERRED BY	:	REGISTRATION DATE	: 18/Sep/2024 09:03 AM
BARCODE NO.	: 01517175	COLLECTION DATE	: 18/Sep/2024 09:04AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 18/Sep/2024 11:18AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBAL	A CANTT	
Test Name	V	alue 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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	MD (Pathology & N	Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologist		r Chopra (Pathology) : Pathologist
NAME	: Mr. MOHIT PAL SINGH			
AGE/ GENDER	: 30 YRS/MALE		PATIENT ID	: 1616887
COLLECTED BY	:		REG. NO./LAB NO.	: 012409180016
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CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT				
Test Name		Value	Unit	Biological Reference interval
		ENDO	CRINOLOGY	
	TH	IYROID FU	NCTION TEST: TOTAL	
TRIIODOTHYRONINI	E (T3): SERUM VESCENT MICROPARTICLE IMMUNOASS	0.768 SAY)	ng/mL	0.35 - 1.93
THYROXINE (T4): SE		7.34	µgm/dL	4.87 - 12.60
	ING HORMONE (TSH): SERUM	2.051 SAY)	µIU/mL	0.35 - 5.50
3rd GENERATION, ULT INTERPRETATION:	RASENSITIVE			
day has influence on the trilodothyronine (T3).Fa		stimulates the p	production and secretion of the m	<i>m. The variation is of the order of 50%.Hence time of</i> etabolically active hormones, thyroxine (T4)and er underproduction (hypothyroidism) or

overproduction(hyperthyroidism) of T4 and/or T3.

CLINICAL CONDITION	T3	T4	TSH
Primary Hypothyroidism:	Reduced	Reduced	Increased (Significantly)
Subclinical Hypothyroidism:	Normal or Low Normal	Normal or Low Normal	High
Primary Hyperthyroidism:	Increased	Increased	Reduced (at times undetectable)
Subclinical Hyperthyroidism:	Normal or High Normal	Normal or High Normal	Reduced

LIMITATIONS:-

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.

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 (eg: phenytoin , salicylates).

3. Serum T4 levies 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 hypothroidism, pregnancy, phenytoin therapy.

TRIIODOTHYRONINE (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





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TEST PERFORMED AT KOS DIAGNOSTIC LAB. AMBALA CANTT





	Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologist CEO & Consultant Pathologist					gy)
NAME	: Mr. MOH	IT PAL SINGH				
AGE/ GENDER	: 30 YRS/N	IALE		PATIENT ID	: 161	6887
COLLECTED BY	:			REG. NO./LAB NO.	:012	409180016
REFERRED BY	:			REGISTRATION DA		Sep/2024 09:03 AM
BARCODE NO.	:01517175	õ		COLLECTION DATE	: 18/5	Sep/2024 09:04AM
CLIENT CODE.	: KOS DIAC	GNOSTIC LAB		REPORTING DATE	: 18/5	Sep/2024 11:18AM
CLIENT ADDRESS	S : 6349/1, 1	NICHOLSON ROAD, AN	MBALA CANTT			
Test Name		_	Value	Unit	:	Biological Reference interval
6 - 12 Months	0.74 - 2.40	6 - 12 Months	7.10 - 16.16	6 – 12 Months	0.70 - 7.00	
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	
	0.05 1.00	00 V/ (A-llt-)	4.07 10.00			

RECOMMENDATIONS OF TSH LEVELS DURING PREGNANCY (μIU/mL) 1st Trimester 0.10 - 2.50 2nd Trimester 0.20 - 3.00 3rd Trimester 0.20 - 4.10	> 20 years (Adults)	0.35 - 1.93	> 20 Years (Adults)	ts) 4.87 - 12.60 > 20 Years (Adults) 0.35– 5.50				
2nd Trimester 0.20 – 3.00	RECOMMENDATIONS OF TSH LEVELS DURING PREGNANCY (µIU/mL)							
		1st Trimester		0.10 – 2.50				
3rd Trimostor	2nd Trimester			0.20 - 3.00				
510 milliester 0.30 – 4.10		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, idonie 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 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.

8. Pregnancy: 1st and 2nd Trimester





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







	Dr. Vinay Ch MD (Pathology & Chairman & Cons			
NAME AGE/ GENDER COLLECTED BY REFERRED BY BARCODE NO. CLIENT CODE. CLIENT ADDRESS	: Mr. MOHIT PAL SINGH : 30 YRS/MALE : : : 01517175 : KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON ROAD, A	REGISTI COLLEC REPORT	T ID ./LAB NO. RATION DATE FION DATE FING DATE	: 1616887 : 012409180016 : 18/Sep/2024 09:03 AM : 18/Sep/2024 09:04AM : 18/Sep/2024 11:44AM
Test Name		Value	Unit	Biological Reference interval
PHYSICAL EXAMINA		CLINICAL PATHO		ΓΙΟΝ
QUANTITY RECIEVEL by DIP STICK/REFLEC COLOUR by DIP STICK/REFLEC TRANSPARANCY by DIP STICK/REFLEC SPECIFIC GRAVITY) TANCE SPECTROPHOTOMETRY TANCE SPECTROPHOTOMETRY TANCE SPECTROPHOTOMETRY	10 AMBER YELLOW CLEAR <=1.005	ml	PALE YELLOW CLEAR 1.002 - 1.030
PROTEIN	TANCE SPECTROPHOTOMETRY	ACIDIC Negative Negative		NEGATIVE (-ve) NEGATIVE (-ve)
pH by DIP STICK/REFLEC BILIRUBIN	TANCE SPECTROPHOTOMETRY	<=5.0 Negative		5.0 - 7.5 NEGATIVE (-ve)
NITRITE by DIP STICK/REFLEC UROBILINOGEN	TANCE SPECTROPHOTOMETRY	Negative Normal	EU/dL	NEGATIVE (-ve) 0.2 - 1.0
KETONE BODIES by DIP STICK/REFLEC BLOOD	TANCE SPECTROPHOTOMETRY TANCE SPECTROPHOTOMETRY	Negative Negative		NEGATIVE (-ve) NEGATIVE (-ve)
ASCORBIC ACID	TANCE SPECTROPHOTOMETRY TANCE SPECTROPHOTOMETRY	NEGATIVE (-ve)		NEGATIVE (-ve)

MICROSCOPIC EXAMINATION



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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





Dr. Vinay Chopra

EXCELLENCE IN HEALTHCARE & DIAGNOSTICS

Dr. Yugam Chopra

MD (Pathology) MD (Pathology & Microbiology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** NAME : Mr. MOHIT PAL SINGH **AGE/ GENDER** : 30 YRS/MALE **PATIENT ID** :1616887 **COLLECTED BY** :012409180016 REG. NO./LAB NO. : **REFERRED BY REGISTRATION DATE** : 18/Sep/2024 09:03 AM **BARCODE NO.** :01517175 **COLLECTION DATE** :18/Sep/2024 09:04AM CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE** :18/Sep/2024 11:44AM **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Unit **Biological Reference interval** NEGATIVE (-ve) **RED BLOOD CELLS (RBCs)** /HPF 0 - 3 by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT PUS CELLS 2-3 /HPF 0 - 5 by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT EPITHELIAL CELLS 1-2 /HPF ABSENT by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT CRYSTALS NEGATIVE (-ve) NEGATIVE (-ve) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT NEGATIVE (-ve) NEGATIVE (-ve) CASTS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT BACTERIA NEGATIVE (-ve) **NEGATIVE** (-ve)

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT OTHERS

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT TRICHOMONAS VAGINALIS (PROTOZOA)

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT

*** End Of Report ***

NEGATIVE (-ve)

ABSENT





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NEGATIVE (-ve)

ABSENT