



	Dr. Vinay Chopra MD (Pathology & Micr Chairman & Consultar	obiology)		(Pathology)
NAME	: Mr. JYOTI SAROOP			
AGE/ GENDER	: 53 YRS/MALE		PATIENT ID	: 1793349
COLLECTED BY	:		REG. NO./LAB NO.	: 012503160011
REFERRED BY	: CENTRAL PHOENIX CLUB (AMBA)	LA CANTT)	REGISTRATION DATE	: 16/Mar/2025 08:18 AM
BARCODE NO.	: 01527162		COLLECTION DATE	: 16/Mar/2025 08:33AM
CLIENT CODE. CLIENT ADDRESS	: KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON ROAD, AMB.	ALA CANTI	REPORTING DATE	: 16/Mar/2025 08:58AM
Test Name		Value	Unit	Biological Reference interval
			LLNESS PANEL: 1.3 OOD COUNT (CBC)	2
RED BLOOD CELLS	<u>S (RBCS) COUNT AND INDICES</u>			
HAEMOGLOBIN (H by CALORIMETRIC	(B)	13.8	gm/dL	12.0 - 17.0
RED BLOOD CELL (4.86	Millions	/cmm 3.50 - 5.00
PACKED CELL VOL	OCUSING, ELECTRICAL IMPEDENCE UME (PCV) AUTOMATED HEMATOLOGY ANALYZER	41.7	%	40.0 - 54.0
MEAN CORPUSCUL	AR VOLUME (MCV) AUTOMATED HEMATOLOGY ANALYZER	85.7	fL	80.0 - 100.0
MEAN CORPUSCUL	AR HAEMOGLOBIN (MCH)	28.4	pg	27.0 - 34.0
	AR HEMOGLOBIN CONC. (MCHC)	33.2	g/dL	32.0 - 36.0
	UTION WIDTH (RDW-CV) automated hematology analyzer	14	%	11.00 - 16.00
	SUTION WIDTH (RDW-SD) AUTOMATED HEMATOLOGY ANALYZER	45	fL	35.0 - 56.0
MENTZERS INDEX by calculated		17.63	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INI		24.69	RATIO	BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CE FOTAL LEUCOCYTI	E COUNT (TLC)	5630	/cmm	4000 - 11000
NUCLEATED RED H	Y BY SF CUBE & MICROSCOPY BLOOD CELLS (nRBCS)	NIL		0.00 - 20.00
NUCLEATED RED H	rt hematology analyzer BLOOD CELLS (nRBCS) % automated hematology analyzer	NIL	%	< 10 %





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Dr. Vinay Chopra Dr. Yugam Chopra MD (Pathology & Microbiology) MD (Pathology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** NAME : Mr. JYOTI SAROOP AGE/ GENDER : 53 YRS/MALE **PATIENT ID** :1793349 **COLLECTED BY** :012503160011 REG. NO./LAB NO. **REFERRED BY** : CENTRAL PHOENIX CLUB (AMBALA CANTT) **REGISTRATION DATE** : 16/Mar/2025 08:18 AM **BARCODE NO.** :01527162 **COLLECTION DATE** : 16/Mar/2025 08:33AM CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE** : 16/Mar/2025 08:58AM **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Unit **Biological Reference interval DIFFERENTIAL LEUCOCYTE COUNT (DLC)** NEUTROPHILS 60 % 50 - 70 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY LYMPHOCYTES 32 % 20 - 40 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY EOSINOPHILS 2 % 1 - 6 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY MONOCYTES 6 % 2 - 12by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY BASOPHILS 0 % 0 - 1 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY **ABSOLUTE LEUKOCYTES (WBC) COUNT** ABSOLUTE NEUTROPHIL COUNT 3378 2000 - 7500 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE LYMPHOCYTE COUNT 1802 800 - 4900 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE EOSINOPHIL COUNT 113 /cmm 40 - 440 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE MONOCYTE COUNT 338 /cmm 80 - 880 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE BASOPHIL COUNT 0 /cmm 0 - 110 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE IMMATURE GRANULOCYTE COUNT 0.0 - 999.00 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS. PLATELET COUNT (PLT) 317000 /cmm 150000 - 450000 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELETCRIT (PCT) 0.27 % 0.10 - 0.36 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE MEAN PLATELET VOLUME (MPV) 8 fL 6.50 - 12.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET LARGE CELL COUNT (P-LCC) 52000 /cmm 30000 - 90000 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET LARGE CELL RATIO (P-LCR) 16.3% 11.0 - 45.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET DISTRIBUTION WIDTH (PDW) 16.1% 15.0 - 17.0

by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE



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

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NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD



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: 6349/1, NICHOLSON ROAD, AMBALA CAN	TT	
Value	Unit	Biological Reference interval
does not tell the health practitioner exactly whether the backward of the conditions besides inflammation	here the inflammation is in th	tion associated with infection, cancer and auto-
fic test because an elevated result often indicated does not tell the health practitioner exactly whether conditions besides inflammation be used to monitor disease activity and resport ematosus W ESR w with conditions that inhibit the normal sedin	here the inflammation is in th . For this reason, the ESR is ty nse to therapy in both of the a nentation of red blood cells.	tion associated with infection, cancer and auto- e body or what is causing it. pically used in conjunction with other test such above diseases as well as some others, such as
fic test because an elevated result often indicated does not tell the health practitioner exactly whether conditions besides inflammation be used to monitor disease activity and responematosus W ESR en with conditions that inhibit the normal sedin inficantly high white blood cell count (leucocytile cell anaemia) also lower the ESR.	here the inflammation is in th . For this reason, the ESR is ty nse to therapy in both of the a nentation of red blood cells, s osis), and some protein abno	tion associated with infection, cancer and auto- e body or what is causing it. pically used in conjunction with other test such above diseases as well as some others, such as
fic test because an elevated result often indicat does not tell the health practitioner exactly whe ected by other conditions besides inflammation be used to monitor disease activity and respor ematosus W ESR In with conditions that inhibit the normal sedin hificantly high white blood cell count (leucocyt le cell anaemia) also lower the ESR. The protein (C-RP) are both markers of inflammat es not change as rapidly as does CRP, either at a l by as many other factors as is ESR, making it a led. it is typically a result of two types of protei	here the inflammation is in the . For this reason, the ESR is ty nee to therapy in both of the a nentation of red blood cells, so osis), and some protein abno tion. the start of inflammation or a better marker of inflammation ns. globulins or fibrinogen.	tion associated with infection, cancer and auto- e body or what is causing it. pically used in conjunction with other test such above diseases as well as some others, such as such as a high red blood cell count prmalities. Some changes in red cell shape (such s it resolves. n .
fic test because an elevated result does not tell the health practition ected by other conditions besides i be used to monitor disease activit ematosus W ESR en with conditions that inhibit the hificantly high white blood cell cou le cell anaemia) also lower the ES re protein (C-RP) are both markers es not change as rapidly as does CF I by as many other factors as is ESR ed. it is typically a result of two ty	often indica er exactly winflammation by and resport normal sedir unt (leucocyt R. of inflammat RP, either at , making it a pes of protei	often indicates the presence of inflammation er exactly where the inflammation is in the nflammation. For this reason, the ESR is ty y and response to therapy in both of the a normal sedimentation of red blood cells, s unt (leucocytosis), and some protein abno R. of inflammation. R, either at the start of inflammation or a , making it a better marker of inflammation.





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Test Name		Value	Unit	Biological Reference interval
	CLIN		FRY/BIOCHEMIST FASTING (F)	TRY
	G (F): PLASMA	110.96 ^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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Test Name		Value	Unit	Biological Reference interval
			OFILE : BASIC	
CHOLESTEROL TO by CHOLESTEROL OX		146.98	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR =
FRIGLYCERIDES: S. by GLYCEROL PHOSP	ERUM HATE OXIDASE (ENZYMATIC)	92.26	mg/dL	240.0 OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTERO	L (DIRECT): SERUM ion	42.69	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROI by CALCULATED, SPE		85.84	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, SPE		104.29	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTER(18.45	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SER	RUM	386.22	mg/dL	350.00 - 700.00
CHOLESTEROL/HD by CALCULATED, SPE	DL RATIO: SERUM	3.44	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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Test Name	Value	Unit	Biological Reference interval
LDL/HDL RATIO: S		RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/H by CALCULATED, SPE	IDL RATIO: SERUM 2.16 ^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.

 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
	LIVER		N TEST (COMPLETE)	
BILIRUBIN TOTAL by DIAZOTIZATION, SI	: SERUM PECTROPHOTOMETRY	0.59	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
	Г (CONJUGATED): SERUM spectrophotometry	0.16	mg/dL	0.00 - 0.40
BILIRUBIN INDIRE	ECT (UNCONJUGATED): SERUM	0.43	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PY	[/RIDOXAL PHOSPHATE	24.8	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PY	[/RIDOXAL PHOSPHATE	35.6	U/L	0.00 - 49.00
AST/ALT RATIO: S		0.7	RATIO	0.00 - 46.00
ALKALINE PHOSPI by Para Nitrophen propanol	HATASE: SERUM IYL PHOSPHATASE BY AMINO METHYL	96.13	U/L	40.0 - 130.0
GAMMA GLUTAMY by SZASZ, SPECTRO	L TRANSFERASE (GGT): SERUM PHTOMETRY	63.01 ^H	U/L	0.00 - 55.0
TOTAL PROTEINS: by BIURET, SPECTRO	SERUM	6.73	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL G		4.45	gm/dL	3.50 - 5.50
GLOBULIN: SERUN by CALCULATED, SPE	Λ	2.28 ^L	gm/dL	2.30 - 3.50
A : G RATIO: SERUI		1.95	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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	KIDNI	TV FUNCTIO)N TEST (COMPLETE)	
UREA: SERUM	KIDI	22.2	mg/dL	10.00 - 50.00
	MATE DEHYDROGENASE (GLDH)	66.6	J	10.00 - 30.00
CREATININE: SERI		1.22	mg/dL	0.40 - 1.40
BLOOD UREA NITE	ROGEN (BUN): SERUM	10.37	mg/dL	7.0 - 25.0
	ROGEN (BUN)/CREATININE	8.5 ^L	RATIO	10.0 - 20.0
RATIO: SERUM		0.0		
by CALCULATED, SPE UREA/CREATININ		18.2	RATIO	
by CALCULATED, SPE	ECTROPHOTOMETRY			
URIC ACID: SERUM		5.17	mg/dL	3.60 - 7.70
CALCIUM: SERUM by ARSENAZO III, SPE		9.87	mg/dL	8.50 - 10.60
PHOSPHOROUS: SH		2.49	mg/dL	2.30 - 4.70
ELECTROLYTES	sine, or contornor omenti			
SODIUM: SERUM by ISE (ION SELECTIV		139.5	mmol/L	135.0 - 150.0
POTASSIUM: SERU	M	4.22	mmol/L	3.50 - 5.00
CHLORIDE: SERUN by ISE (ION SELECTIV	1	104.63	mmol/L	90.0 - 110.0
	IERULAR FILTERATION RATE			
ESTIMATED GLOM (eGFR): SERUM by calculated INTERPRETATION:	IERULAR FILTERATION RATE	70.9		

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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CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DAT	E : 16/Mar/20)25 12:28PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBAI	LA CANTT		
Test Name		Value Ur	it Bio	ological Reference interval
 7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 	xia, high fever). (e.g. ureter colostomy) ass (subnormal creatinine production) tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE LEVEL (BUN rises disproportionately more th	S:	rotoxicosis, Cushing's s	synerome, mgn protein aret,
7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia DECREASED RATIO (>1 1. Acute tubular necr 2. Low protein diet ar 3. Severe liver disease 4. Other causes of de 5. Repeated dialysis (6. Inherited hyperam 7. SIADH (syndrome c 8. Pregnancy. DECREASED RATIO (<1 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther	(e.g. ureter colostomy) ass (subnormal creatinine production) tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE LEVELS (BUN rises disproportionately more the superimposed on renal disease. 0:1) WITH DECREASED BUN : osis. d starvation. creased urea synthesis. urea rather than creatinine diffuses ou monemias (urea is virtually absent in b f inappropiate antidiuretic harmone) du 0:1) WITH INCREASED CREATININE: oy (accelerates conversion of creatine t eleases muscle creatinine). who develop renal failure. sis (acetoacetate causes false increase creased BUN/creatinine ratio). apy (interferes with creatinine measure LAR FILTERATION RATE: DESCRIPTION Normal kidney function Kidney damage with	S: an creatinine) (e.g. obstructive t of extracellular fluid). lood). ue to tubular secretion of urea to creatinine). in creatinine with certain met	e uropathy). a. thodologies,resulting in <u>ASSOCIATED FINDI</u> <u>No proteinuria</u> Presence of Prote	n normal ratio when dehydrat
7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (<1 1. Acute tubular necr 2. Low protein diet ar 3. Severe liver disease 4. Other causes of de 5. Repeated dialysis (6. Inherited hyperam 7. SIADH (syndrome c 8. Pregnancy. DECREASED RATIO (<1 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERL G1 G2	(e.g. ureter colostomy) ass (subnormal creatinine production) tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE LEVELS (BUN rises disproportionately more the superimposed on renal disease. 0:1) WITH DECREASED BUN : osis. d starvation. creased urea synthesis. urea rather than creatinine diffuses ou monemias (urea is virtually absent in b f inappropiate antidiuretic harmone) du 0:1) WITH INCREASED CREATININE: oy (accelerates conversion of creatine t eleases muscle creatinine). who develop renal failure. sis (acetoacetate causes false increase creased BUN/creatinine ratio). apy (interferes with creatinine measure LAR FILTERATION RATE: DESCRIPTION Normal kidney function Kidney damage with normal or high GFR	S: an creatinine) (e.g. obstructive t of extracellular fluid). lood). ue to tubular secretion of urea to creatinine). in creatinine with certain mere ement). GFR (mL/min/1.73m2) >90 >90	e uropathy). a. thodologies,resulting in ASSOCIATED FINDI No proteinuria	n normal ratio when dehydrat
7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia DECREASED RATIO (<1 1. Acute tubular necr 2. Low protein diet ar 3. Severe liver disease 4. Other causes of de 5. Repeated dialysis (6. Inherited hyperam 7. SIADH (syndrome c 8. Pregnancy. DECREASED RATIO (<1 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERU G1 G2 G3a	(e.g. ureter colostomy) ass (subnormal creatinine production) tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE LEVELS (BUN rises disproportionately more the superimposed on renal disease. 0:1) WITH DECREASED BUN : osis. d starvation. e. creased urea synthesis. urea rather than creatinine diffuses ou monemias (urea is virtually absent in b f inappropiate antidiuretic harmone) du 0:1) WITH INCREASED CREATININE: py (accelerates conversion of creatine t eleases muscle creatinine). who develop renal failure. sis (acetoacetate causes false increase creased BUN/creatinine ratio). apy (interferes with creatinine measure LAR FILTERATION RATE: DESCRIPTION Normal kidney function Kidney damage with normal or high GFR Mild decrease in GFR	S: an creatinine) (e.g. obstructive t of extracellular fluid). lood). ue to tubular secretion of urea to creatinine). in creatinine with certain me ement). GFR (mL/min/1.73m2) >90 >90 00-89	e uropathy). a. thodologies,resulting in <u>ASSOCIATED FINDI</u> <u>No proteinuria</u> Presence of Prote	n normal ratio when dehydrat
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	Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologis		(Pathology)
NAME	: Mr. JYOTI SAROOP		
AGE/ GENDER	: 53 YRS/MALE	PATIENT ID	: 1793349
COLLECTED BY	:	REG. NO./LAB NO.	: 012503160011
REFERRED BY	: CENTRAL PHOENIX CLUB (AMBALA CANTT)	REGISTRATION DATE	: 16/Mar/2025 08:18 AM
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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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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT	2	
Test Name	Value	Unit	Biological Reference interval
	ENDOC	RINOLOGY	
	THYROID FUNC	CTION TEST: TOTAL	
TRIIODOTHYRONI	NE (T3): SERUM 1.021 IESCENT MICROPARTICLE IMMUNOASSAY)	ng/mL	0.35 - 1.93
THYROXINE (T4): S	SERUM 8.73 IESCENT MICROPARTICLE IMMUNOASSAY)	µgm/dI	4.87 - 12.60
	ATING HORMONE (TSH): SERUM 0.968	µIU/mL	0.35 - 5.50
3rd GENERATION, ULT INTERPRETATION:			
TSH levels are subject to day has influence on the triiodothyronine (T3).Fai	circadian variation, reaching peak levels between 2-4 a.m and measured serum TSH concentrations. TSH stimulates the pr lure at any level of regulation of the hypothalamic-pituita proidism) of T4 and/or T3.	oduction and secretion of the r	metabolically active hormones, thyroxine (T4)and
CLINICAL CONDITION	T3	T4	TSH
Primary Hypothyroidis	m: Doducod	Doducod	Incroased (Significantly)

CLINICAL CONDITION	Т3	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 (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.

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	
6 - 12 Months	0.74 - 2.40	6 - 12 Months	7.10 - 16.16	6 – 12 Months	0.70 - 7.00	





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Test Name		Value		Unit	t	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 L	EVELS 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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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		CLINICAL PATHO	DIOCY	
	URINE RO	UTINE & MICROSCO		ATION
PHYSICAL EXAMINA				liion
QUANTITY RECIEVE	D	10	ml	
by DIP STICK/REFLECT/ COLOUR	ANCE SPECTROPHOTOMETRY	PALE YELLOW		PALE YELLOW
	ANCE SPECTROPHOTOMETRY			
TRANSPARANCY	ANCE SPECTROPHOTOMETRY	CLEAR		CLEAR
SPECIFIC GRAVITY		1.02		1.002 - 1.030
by DIP STICK/REFLECT/	ANCE SPECTROPHOTOMETRY			
REACTION		ACIDIC		
by DIP STICK/REFLECT/ PROTEIN	ANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
	ANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
SUGAR	ANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
pH		<=5.0		5.0 - 7.5
by DIP STICK/REFLECT/ BILIRUBIN	ANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
	ANCE SPECTROPHOTOMETRY			
NITRITE	ANCE SPECTROPHOTOMETRY.	Negative		NEGATIVE (-ve)
UROBILINOGEN		Normal	EU/dL	0.2 - 1.0
by DIP STICK/REFLECTA	ANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
by DIP STICK/REFLECT	ANCE SPECTROPHOTOMETRY	-		
BLOOD by DIP STICK/REFLECT/	ANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
ASCORBIC ACID		NEGATIVE (-ve)		NEGATIVE (-ve)
by DIP STICK/REFLECT/ MICROSCOPIC EXAI	ANCE SPECTROPHOTOMETRY MINATION			
RED BLOOD CELLS (NEGATIVE (-ve)	/HPF	0 - 3
· · · · · · · · · · · · · · · · · · ·		. /		





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

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Test Name	Value	Unit	Biological Reference interval

	by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
]	PUS CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	3-4	/HPF	0 - 5
]	EPITHELIAL CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	1-3	/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)
(THERS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)
	RICHOMONAS VAGINALIS (PROTOZOA) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	ABSENT		ABSENT

** End Of Report ***



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