

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



	Dr. Vinay Chopra MD (Pathology & Micr Chairman & Consultan	licrobiology)		am Chopra MD (Pathology) tant Pathologist	
IAME	: Miss. SANYA				
AGE/ GENDER	: 24 YRS/FEMALE		PATIENT ID	: 1732307	
COLLECTED BY	:		REG. NO./LAB NO.	: 012501230028	
REFERRED BY	:		REGISTRATION DATE	: 23/Jan/2025 11:10 AM	
BARCODE NO.	: 01524295		COLLECTION DATE	: 23/Jan/2025 11:15AM	
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 23/Jan/2025 12:03PM	
LIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBA	ALA CANTT			
Fest Name		Value	Unit	Biological Reference interval	
	SWAST	HYA WE	LLNESS PANEL: G	т	
	СОМР	LETE BL	OOD COUNT (CBC)		
RED BLOOD CELLS	(RBCS) COUNT AND INDICES				
HAEMOGLOBIN (H)	B)	10.4 ^L	gm/dL	12.0 - 16.0	
RED BLOOD CELL (RBC) COUNT OCUSING, ELECTRICAL IMPEDENCE	4.34	Millions	s/cmm 3.50 - 5.00	
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER		32.5 ^L	%	37.0 - 50.0	
MEAN CORPUSCULAR VOLUME (MCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER		74.9 ^L	fL	80.0 - 100.0	
	AR HAEMOGLOBIN (MCH) utomated hematology analyzer	23.9 ^L	pg	27.0 - 34.0	
	AR HEMOGLOBIN CONC. (MCHC) UTOMATED HEMATOLOGY ANALYZER	31.9 ^L	g/dL	32.0 - 36.0	
	UTION WIDTH (RDW-CV) utomated hematology analyzer	15.9	%	11.00 - 16.00	
	UTION WIDTH (RDW-SD) utomated hematology analyzer	44.6	fL	35.0 - 56.0	
MENTZERS INDEX		17.26	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0	
GREEN & KING IND by CALCULATED	θEX	27.37	RATIO	BETA THALASSEMIA TRAIT:< 65.0 IRON DEFICIENCY ANEMIA: > 65.0	
WHITE BLOOD CEI	LLS (WBCS)			-	
FOTAL LEUCOCYTE	COUNT (TLC) ' by sf cube & microscopy	5490	/cmm	4000 - 11000	
	LOOD CELLS (nRBCS) RT HEMATOLOGY ANALYZER	NIL		0.00 - 20.00	
by AUTOMATED OF AN		NIL	%		





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

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Dr. Vinay Chopra

EXCELLENCE IN HEALTHCARE & DIAGNOSTICS

Dr. Yugam Chopra

MD (Pathology & Microbiology) MD (Pathology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** NAME : Miss. SANYA **AGE/ GENDER** : 24 YRS/FEMALE **PATIENT ID** :1732307 **COLLECTED BY** :012501230028 REG. NO./LAB NO. **REFERRED BY REGISTRATION DATE** : 23/Jan/2025 11:10 AM **BARCODE NO.** :01524295 **COLLECTION DATE** : 23/Jan/2025 11:15AM CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE** : 23/Jan/2025 12:03PM **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 3294 2000 - 7500 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE LYMPHOCYTE COUNT 1757 800 - 4900 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE EOSINOPHIL COUNT 110 /cmm 40 - 440 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE MONOCYTE COUNT 329 /cmm 80 - 880 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE BASOPHIL COUNT 0 /cmm 0 - 110 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS. PLATELET COUNT (PLT) 150000 - 450000 292000 /cmm by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELETCRIT (PCT) 0.35 % 0.10 - 0.36 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE MEAN PLATELET VOLUME (MPV) 12 fL 6.50 - 12.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE 30000 - 90000 PLATELET LARGE CELL COUNT (P-LCC) /cmm 120000^H by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE % PLATELET LARGE CELL RATIO (P-LCR) 41.211.0 - 45.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET DISTRIBUTION WIDTH (PDW) 15.0 - 17.0 16.2% by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD



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Test Name	Valu	e Unit	Biological Reference interval



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MBBS, MD (PATHOLOGY)

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CLIENT CODE.	: KOS DIAGNOSTIC LAB		RTING DATE	: 23/Jan/2025 02:39PM	
CLIENT CODE. CLIENT ADDRESS			WING DATE	jan/	
ULIENI ADDKË33	: 6349/1, NICHOLSON ROAD, A	AIVIDALA UAINTT			
Test Name		Value	Unit	Biological Reference	interva
WHOLE BLOOD	EMOGLOBIN (HbA1c): RMANCE LIQUID CHROMATOGRAPHY)	9.9 ^H	%	4.0 - 6.4	
ESTIMATED AVERA	GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY)	237.43 ^H	mg/dL	60.00 - 140.00	
INTERPRETATION:					
		DIABETES ASSOCIATION (
	REFERENCE GROUP	GLYCOSYLATED HEMOGLOGIB (HBAIC) in %		BAIC) in %	
	abetic Adults >= 18 years	/	<5.7		
	t Risk (Prediabetes)		5.7 - 6.4 >= 6.5		
U	iagnosing Diabetes	_	>= 0.5 Age > 19 Years		
		Goals of The		< 7.0	
	Therapeutic goals for glycemic control			>8.0	
Therapeut	ic goals for glycemic control	Actions Suade	sted:	>8.0	
Therapeut	ic goals for glycemic control	Actions Sugge	sted: Age < 19 Years	>8.0	

1.Glycosylated hemoglobin (HbA1c) test is three monthly monitoring done to assess compliace with therapeutic regimen in diabetic patients. 2. Since Hb1c reflects long term fluctuations in blood glucose concentration, a diabetic patient who has recently under good control may still have high concentration of HbAlc. Converse is true for a diabetic previously under good control but now poorly controlled.

3. Target goals of < 7.0 % may be beneficial in patients with short duration of diabetes, long life expectancy and no significant cardiovascular disease. In patients with significant complications of diabetes, limited life expectancy or extensive co-morbid conditions, targetting a goal of < 7.0% may not be appropiate.

4. High HbA1c (>9.0 -9.5 %) is strongly associated with risk of development and rapid progression of microvascular and nerve complications 5. Any condition that shorten RBC life span like acute blood loss, hemolytic anemia faisely lower HbA1c results.

6.HbA1c results from patients with HbSS,HbSC and HbD must be interpreted with caution, given the pathological processes including anemia, increased red cell turnover, and transfusion requirement that adversely impact HbA1c as a marker of long-term gycemic control.

7.Specimens from patients with polycythemia or post-splenctomy may exhibit increse in HbA1c values due to a somewhat longer life span of the red cells.



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CLIENT CODE.	: KOS DIAGNO	STIC LAB	R	EPORTING DATE	: 23/Jan/2025 12:36P	М
LIENT ADDRESS	: 6349/1, NICH	HOLSON ROAD,	AMBALA CANTT			
Test Name			Value	Unit	Biological H	Reference interval
systemic lupus eryth CONDITION WITH LO' A low ESR can be see polycythaemia), sign as sickle cells in sickle NOTE: 1. ESR and C - reactive 2. Generally, ESR doe 3. CRP is not affected 4. If the ESR is elevat 5. Women tend to ha	be used to monit ematosus W ESR in with conditions ificantly high wh e cell anaemia) a e protein (C-RP) a es not change a by as many othe ed, it is typically we a higher ESR, tran, methyldopa	s that inhibit the nite blood cell co also lower the E are both marker rapidly as does (r factors as is ES a result of two t and menstruation, oral contracep	e normal sedimenta ount (leucocytosis) SR. so of inflammation. CRP, either at the st R, making it a bette types of proteins, gl on and pregnancy ca	tion of red blood cells, so , and some protein abno art of inflammation or as r marker of inflammatior obulins or fibrinogen. In cause temporary eleva	n.	ell count in red cell shape (sucl





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		hopra & Microbiology) onsultant Pathologist	Dr. Yugam MD CEO & Consultant	(Pathology)
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD), AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		CAL CHEMISTRY/	BIOCHEMIST	RY
	CLINI			
	CLINI	GLUCOSE FAST		

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
		LIPID PROFILE	: BASIC	
CHOLESTEROL TO by CHOLESTEROL OX		216.61 ^H	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: S by GLYCEROL PHOSP	ERUM HATE OXIDASE (ENZYMATIC)	74.36	mg/dL	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	79.27	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROI by CALCULATED, SPE		122.48	mg∕dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129. BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0
NON HDL CHOLEST by CALCULATED, SPE		137.34 ^H	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159. BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTER(14.87	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SER	UM	507.59	mg/dL	350.00 - 700.00
CHOLESTEROL/HD by CALCULATED, SPE	L RATIO: SERUM	2.73	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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Page 7 of 15





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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD), AMBALA CANTT				
Test Name		Value	Unit	Biological Reference interval		
LDL/HDL RATIO: S by CALCULATED, SPE		1.55	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	0.94 ^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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6.20 - 8.00

3.50 - 5.50

2.30 - 3.50

1.00 - 2.00

gm/dL

gm/dL

gm/dL

RATIO

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Test Name		Value	Unit	Biological Reference interval
	LIVER	FUNCTION T	TEST (COMPLETE)	
BILIRUBIN TOTAL: by DIAZOTIZATION, SF	: SERUM PECTROPHOTOMETRY	0.99	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
	C (CONJUGATED): SERUM	0.56 ^H	mg/dL	0.00 - 0.40
BILIRUBIN INDIRE by CALCULATED, SPE	CT (UNCONJUGATED): SERUM	0.43	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PY	RIDOXAL PHOSPHATE	24.33	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PY	RIDOXAL PHOSPHATE	21.76	U/L	0.00 - 49.00
AST/ALT RATIO: SI by CALCULATED, SPE		1.12	RATIO	0.00 - 46.00
ALKALINE PHOSPH		115	U/L	40.0 - 150.0
GAMMA GLUTAMY	L TRANSFERASE (GGT): SERUM	8	U/L	0.00 - 55.0

ALBUMIN: SERUM 5.22 by BROMOCRESOL GREEN **GLOBULIN: SERUM** 2.8 by CALCULATED, SPECTROPHOTOMETRY A : G RATIO: SERUM 1.86 by CALCULATED, SPECTROPHOTOMETRY INTERPRETATION

by SZASZ, SPECTROPHTOMETRY TOTAL PROTEINS: SERUM

by BIURET, 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:

> 2
> 2 (Highly Suggestive)
1.4 - 2.0
> 1.5
> 1.3 (Slightly Increased)

8.02^H



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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).

GOOD PROGNOSTIC SIGN 0.3 - 0.6	
POOR PROGNOSTIC SIGN 1.2 - 1.6	



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	KIDNE	Y FUNCTION	TEST (COMPLETE)	
UREA: SERUM		32.77	mg/dL	10.00 - 50.00
by UREASE - GLUTAM	ATE DEHYDROGENASE (GLDH)	02.11		
CREATININE: SERU		0.65	mg/dL	0.40 - 1.20
•	COGEN (BUN): SERUM	15.31	mg/dL	7.0 - 25.0
by CALCULATED, SPE			-	
BLOOD UREA NITR RATIO: SERUM	COGEN (BUN)/CREATININE	23.55 ^H	RATIO	10.0 - 20.0
by CALCULATED, SPE	CTROPHOTOMETRY			
UREA/CREATININ by CALCULATED, SPE		50.42	RATIO	
URIC ACID: SERUM		2.8	mg/dL	2.50 - 6.80
by URICASE - OXIDAS	E PEROXIDASE			
CALCIUM: SERUM by ARSENAZO III, SPE	CTROPHOTOMETRY	9.89	mg/dL	8.50 - 10.60
PHOSPHOROUS: SE	CRUM	3.14	mg/dL	2.30 - 4.70
	DATE, SPECTROPHOTOMETRY			
ELECTROLYTES		100 5	1/I	105.0 150.0
SODIUM: SERUM by ISE (ION SELECTIV	E ELECTRODE)	139.5	mmol/L	135.0 - 150.0
POTASSIUM: SERUI		3.94	mmol/L	3.50 - 5.00
by ISE (ION SELECTIV CHLORIDE: SERUM		104.63	mmol/L	90.0 - 110.0
by ISE (ION SELECTIV	E ELECTRODE)	104.00	IIIII01/ L	00.0 110.0
ESTIMATED GLOM	IERULAR FILTERATION RATE			
	ERULAR FILTERATION RATE	126		
(eGFR): SERUM by CALCULATED				
INTERPRETATION:				

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.



KOS Central Lab: 6349/1, Nicholson Road, Ambala Cantt -133 001, Haryana

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

Page 11 of 15







	1	Dr. Vinay ChopraDr. Yugam ChopraMD (Pathology & Microbiology)MD (Pathology)Chairman & Consultant PathologistCEO & Consultant Pathologist							
NAME	: Miss. SANYA								
AGE/ GENDER	: 24 YRS/FEMA	LE		PATIENT ID		: 1732307			
COLLECTED BY	:			REG. NO./LAB NO		: 012501230	0028		
	•								
REFERRED BY	:			REGISTRATION D		:23/Jan/2025			
BARCODE NO.	:01524295			COLLECTION DAT	E	:23/Jan/2025	5 11:15Al	M	
CLIENT CODE.	: KOS DIAGNOS	STIC LAB		REPORTING DAT	Е	:23/Jan/2025	5 12:42PM	M	
CLIENT ADDRESS	: 6349/1, NICH	IOLSON ROAD, AN	/IBALA CANTT	Г					
Test Name			Value	Un	nit	Bio	ogical R	eference	e interva
8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia	tetracycline, gluo 0:1) WITH ELEVA (BUN rises dispr	reatinine product cocorticoids) FED CREATININE L oportionately mo	EVELS:	nine) (e.g. obstructive	e uropathy	<i>)</i>).			
 Reduced muscle m Certain drugs (e.g. INCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (<1 Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome c Pregnancy. DECREASED RATIO (<1 Phenacimide thera Rhabdomyolysis (r Muscular patients INAPPROPIATE RATIO Diabetic ketoacido should produce an in Cephalosporin ther 	(e.g. ureter colos ass (subnormal c tetracycline, gluc 0:1) WITH ELEVA (BUN rises dispr superimposed or 0:1) WITH DECRE osis. Id starvation. by creased urea syn urea rather than monemias (urea f inappropiate an 0:1) WITH INCRE by (accelerates c eleases muscle c who develop ren sis (acetoacetate creased BUN/cre apy (interferes w	reatinine product cocorticoids) FED CREATININE L oportionately moin renal disease. ASED BUN : thesis. creatinine diffuse is virtually absent tidiuretic harmor ASED CREATININE: onversion of creat reatinine). al failure. causes false increat atinine ratio). tith creatinine meat IRATE:	EVELS: re than creatin es out of extra in blood). ne) due to tubu tine to creatin ease in creatin asurement).	icellular fluid). ular secretion of urea ine). hine with certain met	a. thodologie	es,resulting in		atio wher	n dehydra
 Reduced muscle m 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 CKD STAGE 	(e.g. ureter colos ass (subnormal c tetracycline, gluc 0:1) WITH ELEVA (BUN rises dispr superimposed of 0:1) WITH DECRE osis. Id starvation. 2: creased urea syn urea rather than monemias (urea f inappropiate al 0:1) WITH INCRE oy (accelerates c eleases muscle c who develop ren sis (acetoacetate creased BUN/cre apy (interferes w LAR FILTERATION	reatinine product cocorticoids) FED CREATININE L oportionately moin renal disease. ASED BUN : thesis. creatinine diffuse is virtually absent tidiuretic harmor ASED CREATININE: onversion of creat reatinine). al failure. causes false increatinine ratio). th creatinine meat IRATE: DESCRIPTION	EVELS: re than creatin es out of extra in blood). ne) due to tubu tine to creatin ease in creatin asurement).	icellular fluid). ular secretion of urea ine). nine with certain met mL/min/1.73m2)	a. thodologi€ ▲SSO	es,resulting in CIATED FINDIN		atio wher	n dehydra
 Reduced muscle m Certain drugs (e.g. INCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (<1 Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome c Pregnancy. DECREASED RATIO (<1 Phenacimide thera Rhabdomyolysis (r Muscular patients INAPPROPIATE RATIO Diabetic ketoacido should produce an in Cephalosporin ther 	(e.g. ureter colos ass (subnormal c tetracycline, gluc 0:1) WITH ELEVA (BUN rises dispr superimposed of 0:1) WITH DECRE osis. Id starvation. 2: creased urea syn urea rather than monemias (urea f inappropiate al 0:1) WITH INCRE oy (accelerates c eleases muscle c who develop ren sis (acetoacetate creased BUN/crea apy (interferes w LAR FILTERATION Norr Norr	reatinine product cocorticoids) FED CREATININE L oportionately mon nenal disease. ASED BUN : thesis. creatinine diffuse is virtually absent ntidiuretic harmor ASED CREATININE: onversion of creat reatinine). al failure. causes false increatinine ratio). ith creatinine maticity. ith creatinine maticity. IRATE: DESCRIPTION nal kidney function ney damage with	EVELS: re than creatin es out of extra in blood). ne) due to tubu tine to creatin ease in creatin asurement).	icellular fluid). ular secretion of urea ine). hine with certain met	a. thodologie ASSO N Preso	es,resulting in CIATED FINDIN o proteinuria ence of Protein	IGS	atio wher	ı dehydra
 Reduced muscle m Certain drugs (e.g. INCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (<1 Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome c Pregnancy. DECREASED RATIO (<1 Phenacimide thera Rhabdomyolysis (r Muscular patients INAPPROPIATE RATIO Diabetic ketoacido Should produce an in Cephalosporin ther ESTIMATED GLOMERL G1 G2 	(e.g. ureter colos ass (subnormal c tetracycline, gluc 0:1) WITH ELEVA (BUN rises dispr superimposed of 0:1) WITH DECRE osis. Id starvation. 2: creased urea syn urea rather than monemias (urea f inappropiate al 0:1) WITH INCRE oy (accelerates c eleases muscle c who develop ren sis (acetoacetate creased BUN/cre apy (interferes w LAR FILTERATION Norr Norr	reatinine product cocorticoids) FED CREATININE L oportionately mon in renal disease. ASED BUN : thesis. creatinine diffuse is virtually absent ntidiuretic harmor ASED CREATININE: onversion of creat reatinine). al failure. causes false increat reatinine ratio). ith creatinine meat IRATE: DESCRIPTION nal kidney function ney damage with rmal or high GFR.	EVELS: re than creatin es out of extra in blood). ne) due to tubu tine to creatin ease in creatin asurement). GFR (1 n	icellular fluid). ular secretion of urea ine). hine with certain met <u>mL/min/1.73m2) >90 >90</u>	a. thodologie ASSO N Preso	es,resulting in CIATED FINDIN o proteinuria	IGS	atio wher	n dehydra
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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 GLOMERU G1 G2	(e.g. ureter colos ass (subnormal c tetracycline, gluc 0:1) WITH ELEVA (BUN rises dispr superimposed of 0:1) WITH DECRE osis. Id starvation. creased urea syn urea rather than monemias (urea f inappropiate an 0:1) WITH INCRE oy (accelerates c eleases muscle c who develop ren sis (acetoacetate creased BUN/crea py (interferes w LAR FILTERATION Norr Kic nc Mill Mode	reatinine product cocorticoids) FED CREATININE L oportionately mon nenal disease. ASED BUN : thesis. creatinine diffuse is virtually absent ntidiuretic harmor ASED CREATININE: onversion of creat reatinine). al failure. causes false increat reatinine ratio). ith creatinine meat IRATE: DESCRIPTION nal kidney function ney damage with rmal or high GFR.	EVELS: re than creatin es out of extra in blood). ne) due to tubu tine to creatin ease in creatin easurement). GFR (n FR	icellular fluid). ular secretion of urea ine). hine with certain met <u>mL/min/1.73m2) >90 >90</u>	a. thodologie ASSO N Preso	es,resulting in CIATED FINDIN o proteinuria ence of Protein	IGS	atio wher	n dehydra





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









	Dr. Vinay Chopra MD (Pathology & Microbiolog Chairman & Consultant Patho		(Pathology)
NAME	: Miss. SANYA		
AGE/ GENDER	: 24 YRS/FEMALE	PATIENT ID	: 1732307
COLLECTED BY	:	REG. NO./LAB NO.	: 012501230028
REFERRED BY	:	REGISTRATION DATE	: 23/Jan/2025 11:10 AM
BARCODE NO.	: 01524295	COLLECTION DATE	: 23/Jan/2025 11:15AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 23/Jan/2025 12:42PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CA	NTT	
Test Name	Value	Unit	Biological Reference interval

COMMENTS:

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

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

ADVICE:

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





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MBBS, MD (PATHOLOGY)

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	MD (Pathology &	Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologist		m Chopra D (Pathology) nt Pathologist
NAME	: Miss. SANYA			
AGE/ GENDER	: 24 YRS/FEMALE]	PATIENT ID	: 1732307
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
			RINOLOGY	
	TH	IYROID FUNCT	TION TEST: TOTAI	
TRIIODOTHYRONI	NE (T3): SERUM iescent microparticle immunoa	1.32	ng/mL	0.35 - 1.93
THYROXINE (T4): S		10.24	µgm/d	L 4.87 - 12.60
	ATING HORMONE (TSH): SER		µIU/m	L 0.35 - 5.50
3rd GENERATION, ULT <u>INTERPRETATION</u> :	RASENSITIVE			
day has influence on the triiodothyronine (T3).Fai	measured serum TSH concentrations. T	SH stimulates the proc	luction and secretion of the	pm. The variation is of the order of 50%.Hence time of the metabolically active hormones, thyroxine (T4)and ther underproduction (hypothyroidism) or
CLINICAL CONDITION	Т3		T4	TSH
Primary Hypothyroidis			Reduced	Increased (Significantly)
Subclinical Hypothyroi	dism: Normal or Low	v ivormal N	ormal or Low Normal	High

LIMITATIONS:-

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.

TRIIODOTHYRONINE (T3)		THYROX	(INE (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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Increased

Normal or High Normal







	Dr. Vinay Chopra MD (Pathology & Microt Chairman & Consultant I	piology) MD	n Chopra 9 (Pathology) t Pathologist
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The set BI server		7-1 TL-*4	Biolo de l Biologue de Annuel

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

*** End Of Report ***





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