



	Dr. Vinay Chopr MD (Pathology & Mic Chairman & Consulta	robiology)	MI	m <b>Chopra</b> D (Pathology) nt Pathologist
NAME	: Mrs. SEETU REHAN			
AGE/ GENDER	: 47 YRS/FEMALE		PATIENT ID	: 1576446
COLLECTED BY	: SURJESH		REG. NO./LAB NO.	: 012408100017
REFERRED BY	:		<b>REGISTRATION DATE</b>	: 10/Aug/2024 10:15 AM
BARCODE NO.	: 01514814		<b>COLLECTION DATE</b>	: 10/Aug/2024 10:19AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		<b>REPORTING DATE</b>	: 10/Aug/2024 10:44AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AME	BALA CANTI	2	
Test Name		Value	Unit	Biological Reference interval
	SWA	STHYA W	ELLNESS PANEL: G	
	CON	<b>VIPLETE BL</b>	OOD COUNT (CBC)	
RED BLOOD CELLS (F	RBCS) COUNT AND INDICES			
HAEMOGLOBIN (HB		11.5 <sup>L</sup>	gm/dL	12.0 - 16.0
				/omm 2.50 5.00
RED BLOOD CELL (RE	SC) COUNT FOCUSING, ELECTRICAL IMPEDENCE	4.16	Millions	/cmm 3.50 - 5.00
PACKED CELL VOLUN		36.8 <sup>L</sup>	%	37.0 - 50.0
MEAN CORPUSCULA	AUTOMATED HEMATOLOGY ANALYZER	88.6	fL	80.0 - 100.0
by CALCULATED BY A	AUTOMATED HEMATOLOGY ANALYZER			
	AR HAEMOGLOBIN (MCH) AUTOMATED HEMATOLOGY ANALYZER	27.7	pg	27.0 - 34.0
MEAN CORPUSCULA	R HEMOGLOBIN CONC. (MCHC)	31.2 <sup>L</sup>	g/dL	32.0 - 36.0
	AUTOMATED HEMATOLOGY ANALYZER FION WIDTH (RDW-CV)	14	%	11.00 - 16.00
by CALCULATED BY A	AUTOMATED HEMATOLOGY ANALYZER		/0	11.00 10.00
	TION WIDTH (RDW-SD) AUTOMATED HEMATOLOGY ANALYZER	46.4	fL	35.0 - 56.0
MENTZERS INDEX	TO TOWATED TEWATOLOGIT AWALIZER	21.3	RATIO	BETA THALASSEMIA TRAIT: < 13.
by CALCULATED				IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDE	ΞX	29.88	RATIO	BETA THALASSEMIA TRAIT: < =
Jy UALOULATED				65.0 IRON DEFICIENCY ANEMIA: > 65.
WHITE BLOOD CELL	<u>S (WBCS)</u>			
	COUNT (TLC) y by sf cube & microscopy	5560	/cmm	4000 - 11000
NUCLEATED RED BL		NIL		0.00 - 20.00
MICROSCOPY				
	OOD CELLS (nRBCS) % AUTOMATED HEMATOLOGY ANALYZER &	NIL	%	< 10 %
by CALCULATED BY A MICROSCOPY	AUTOMATED HEMATOLOGY ANALYZER &			
DIFFERENTIAL LEUC	OCYTE COUNT (DLC)			



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

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







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

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Test Name		Value	Unit	Biological Reference interval
NEUTROPHILS	Y BY SF CUBE & MICROSCOPY	59	%	50 - 70

Dr. Vinay Chopra

MD (Pathology & Microbiology) Chairman & Consultant Pathologist

NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	59	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	31	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	3	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	7	%	2 - 12
BASOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE LEUKOCYTES (WBC) COUNT	0	%	0 - 1
ABSOLUTE NEUTROPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	3280	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1724	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	167	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	389	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110
PLATELETS AND OTHER PLATELET PREDICTIVE MARKER	<u>RS.</u>		
PLATELET COUNT (PLT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	173000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.26	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by hydro dynamic focusing, electrical impedence	15 <sup>H</sup>	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	103000 <sup>H</sup>	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	59.6 <sup>H</sup>	<b>%</b>	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by hydro dynamic focusing, electrical impedence NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD	16.2	%	15.0 - 17.0



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< 7.0

>8.0

<7.5

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NAME	: Mrs. SEETU REHAN			
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REFERRED BY	:	REG	<b>GISTRATION DATE</b>	: 10/Aug/2024 10:15 AM
BARCODE NO.	:01514814	CO	LECTION DATE	: 10/Aug/2024 10:19AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	RE	PORTING DATE	: 10/Aug/2024 02:39PM
CLIENT ADDRESS				
Test Name		Value	Unit	Biological Reference interval
	GL	YCOSYLATED HAEM	OGLOBIN (HBA1C)	
GLYCOSYLATED HAEM	DGLOBIN (HbA1c):	5.6	%	4.0 - 6.4
	ANCE LIQUID CHROMATOGRAPHY)			
ESTIMATED AVERAGE F by HPLC (HIGH PERFORM INTERPRETATION:	PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY)	114.02	mg/dL	60.00 - 140.00
	AS PER AMERICAN DIAB	ETES ASSOCIATION (ADA	):	
			, D HEMOGLOGIB (HBAIC) in	1%
RE	FERENCE GROUP	GLYCOSYLATE	D HEIVIOGLOGIB (HEAIC) III	1 /0
Non diab	etic Adults >= 18 years	GLYCOSYLATE	<5.7	
Non diab At F		GLYCOSYLATE	, ,	

turnover, and transfu	sion requirement	that adversely impact HDA IC	as a marker of long-term g	gycemic control.
7 Specimons from pat	ionte with polyey	thomia or post splonstomy m	av avhibit incress in UbA1c	c values due to a semewhat longer life span of the red cells

Goals of Therapy

Actions Suggested

Goal of therapy

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

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

6.HbA1c results from patients with HbSS,HbSC and HbD must be interpreted with caution, given the pathological processes including anemia, increased red cell

1.Glycosylated hemoglobin (HbA1c) test is three monthly monitoring done to assess compliace with therapeutic regimen in diabetic patients.

significant complications of diabetes, limited life expectancy or extensive co-morbid conditions, targetting a goal of < 7.0% may not be

5.Any condition that shorten RBC life span like acute blood loss, hemolytic anemia falsely lower HbA1c results.

HbA1c (>9.0 -9.5 %) is strongly associated with risk of development and rapid progression of microvascular and nerve complications

Age < 19 Years

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.



COMMENTS:

appropiate

Therapeutic goals for glycemic control

HbAlc. Converse is true for a diabetic previously under good control but now poorly controlled.

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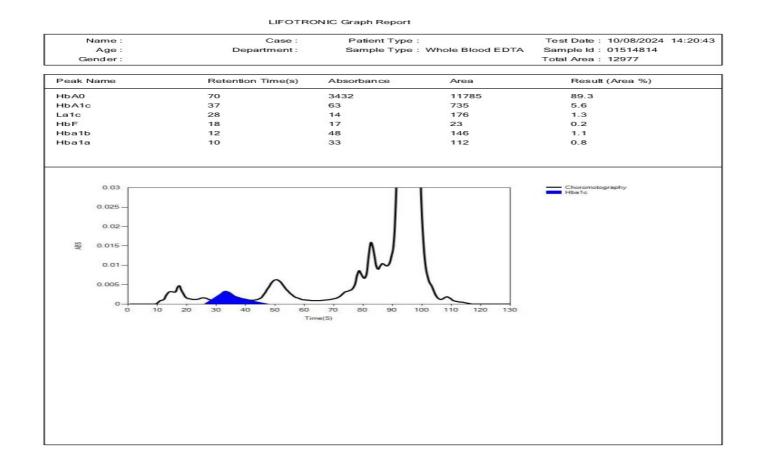
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4.High





	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbiolo Chairman & Consultant Path	0, ,	(Pathology)
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA C	ANTT	
Test Name	Valu	e Unit	<b>Biological Reference interval</b>







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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



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ARCODE NO.	: 01514814		COLLECTION DATE	: 10/Aug/2024 10:19AM
LIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 10/Aug/2024 10:57AM
LIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	, AMBALA CANT	Г	
est Name		Value	Unit	Biological Reference interval
	ERYT	HROCYTE SED	IMENTATION RATE (ES	R)
	MENTATION RATE (ESR)	13	mm/1st h	ır 0 - 20
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 5. Drugs such as dext	nificantly high white blood cell c le cell anaemia) also lower the l e protein (C-RP) are both marke es not change as rapidly as does l by as many other factors as is Es ed, it is typically a result of two we a higher ESR, and menstruati	ount (leucocytos ESR. CRP, either at th <b>SR, making it a be</b> types of proteins on and pregnanc	is), and some protein abno n. estart of inflammation or as e <b>tter marker of inflammatior</b> s, globulins or fibrinogen. y can cause temporary eleva	n.
	there a		Guopra	





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BARCODE NO.	: 01514814		COLLECTION DATE	: 10/Aug/2024 10:19AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 10/Aug/2024 11:41AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	CLIN	IICAL CHEMIS	TRY/BIOCHEMISTR	Y
		GLUCOSE	FASTING (F)	
GLUCOSE FASTING (F by glucose oxidasi	): PLASMA E - PEROXIDASE (GOD-POD)	77.48	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0

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.
 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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NAME AGE/ GENDER COLLECTED BY REFERRED BY BARCODE NO. CLIENT CODE. CLIENT ADDRESS	: Mrs. SEETU REHAN : 47 YRS/FEMALE : SURJESH : : 01514814 : KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON ROAD,	REG. REGI COLI REPO	ENT ID NO./LAB NO. STRATION DATE ECTION DATE DRTING DATE	: 1576446 <b>: 012408100017</b> : 10/Aug/2024 10:15 AM : 10/Aug/2024 10:19AM : 10/Aug/2024 12:15PM
Test Name		Value	Unit	Biological Reference interval
		LIPID PROFILE	BASIC	
CHOLESTEROL TOTAL by CHOLESTEROL OX		125.96	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239. HIGH CHOLESTEROL: > OR = 240
TRIGLYCERIDES: SER by GLYCEROL PHOSP	UM HATE OXIDASE (ENZYMATIC)	61.7	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROL (I by SELECTIVE INHIBITI		66.1	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: S by CALCULATED, SPEC		47.52	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		59.86	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		12.34	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUI	M	313.62 <sup>L</sup>	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL F by CALCULATED, SPEC	ratio: serum	1.91	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: SER by CALCULATED, SPEC		0.72	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0

KOS Diagnostic Lab (A Unit of KOS Healthcare)

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CLIENT ADDRESS	: 6349/1, NICHOLSON ROA	D, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
TRIGLYCERIDES/HD		0.93 <sup>L</sup>	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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	. 00 10/ 1, 10011015010 100112, 11			
Test Name		Value	Unit	Biological Reference interval
	LIV	ER FUNCTION	I TEST (COMPLETE)	
BILIRUBIN TOTAL: SI by DIAZOTIZATION, SF		0.63	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
	CONJUGATED): SERUM	0.22	mg/dL	0.00 - 0.40
-	(UNCONJUGATED): SERUM	0.41	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PY	RIDOXAL PHOSPHATE	16.5	U/L	7.00 - 45.00
SGPT/ALT: SERUM	RIDOXAL PHOSPHATE	21	U/L	0.00 - 49.00
AST/ALT RATIO: SER	UM	0.79	RATIO	0.00 - 46.00
ALKALINE PHOSPHA		55.3	U/L	40.0 - 130.0
GAMMA GLUTAMYL by SZASZ, SPECTROF	TRANSFERASE (GGT): SERUM	13.06	U/L	0.00 - 55.0
TOTAL PROTEINS: SE	RUM	6.65	gm/dL	6.20 - 8.00
ALBUMIN: SERUM		3.82	gm/dL	3.50 - 5.50
GLOBULIN: SERUM		2.83	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by CALCULATED, SPE		1.35	RATIO	1.00 - 2.00

by CALCULATED, SPECTROPHOTOMETRY

NOTE:- To be correlated in individuals having SGOT and SGPT values higher than Normal Referance Range.

USE:- Differential diagnosis of diseases of hepatobiliary system and pancreas.

## INCREASED:

DRUG HEPATOTOXICITY	> 2
ALCOHOLIC HEPATITIS	> 2 (Highly Suggestive)
CIRRHOSIS	1.4 - 2.0
INTRAHEPATIC CHOLESTATIS	> 1.5





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





**INTERPRETATION** 





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Test Name		Value	Unit	Biological Reference interval
HEPATOCELLULAR C	ARCINOMA & CHRONIC HEPATITIS		> 1.3 (Slightly Increa	used)

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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AGE/ GENDER	: 47 YRS/FEMALE	PA	ATIENT ID	: 1576446
COLLECTED BY	: SURJESH	RI	EG. NO./LAB NO.	: 012408100017
<b>REFERRED BY</b>	:	RI	EGISTRATION DATE	: 10/Aug/2024 10:15 AM
BARCODE NO.	:01514814	CC	DLLECTION DATE	: 10/Aug/2024 10:19AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	RI	EPORTING DATE	: 10/Aug/2024 12:15PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD	), AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	к	IDNEY FUNCTION	TEST (COMPLETE)	
UREA: SERUM		18.33	mg/dL	10.00 - 50.00
	ATE DEHYDROGENASE (GLDH)	0.04		0.40, 1.00
CREATININE: SERUN by ENZYMATIC, SPEC		0.84	mg/dL	0.40 - 1.20
	GEN (BUN): SERUM	8.57	mg/dL	7.0 - 25.0
	ectrophotometry DGEN (BUN)/CREATININE	10.2	RATIO	10.0 - 20.0
RATIO: SERUM	JOEN (DON)/ CREATININE	10.2	KATIO	10.0 - 20.0
	ECTROPHOTOMETRY			
UREA/CREATININE I	RATIO: SERUM ECTROPHOTOMETRY	21.82	RATIO	
URIC ACID: SERUM		3.37	mg/dL	2.50 - 6.80
by URICASE - OXIDAS CALCIUM: SERUM	SE PEROXIDASE	9.33	ma/dl	8.50 - 10.60
by ARSENAZO III, SPE	CTROPHOTOMETRY	9.33	mg/dL	8.30 - 10.60
PHOSPHOROUS: SEF		3.4	mg/dL	2.30 - 4.70
by PHOSPHOMOLYBL ELECTROLYTES	DATE, SPECTROPHOTOMETRY			
SODIUM: SERUM		137.9	mmol/L	135.0 - 150.0
by ISE (ION SELECTIV		4.2	mmol/L	3.50 - 5.00
POTASSIUM: SERUN by ISE (ION SELECTIV		4.3	mmoi/L	3.30 - 5.00
CHLORIDE: SERUM		103.43	mmol/L	90.0 - 110.0
by ISE (ION SELECTIN	/E ELECTRODE) RULAR FILTERATION RATE			
		04.0		
(eGFR): SERUM	RULAR FILTERATION RATE	86.2		
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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	MD (Pathology 8	Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologist				
NAME	: Mrs. SEETU REHAN					
AGE/ GENDER	: 47 YRS/FEMALE	PATIENT ID	:	1576446		
COLLECTED BY	: SURJESH	REG. NO./LAB NO		01240810001	17	
	. SUMESH					
REFERRED BY	:	REGISTRATION		10/Aug/2024 1		
BARCODE NO.	: 01514814	COLLECTION DA		10/Aug/2024 1		
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DAT	re :	10/Aug/2024 1	2:15PM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT				
Test Name		Value U	nit	Biologi	cal Reference	e interval
7. Urine reabsorption 3. Reduced muscle m 9. Certain drugs (e.g. <b>NCREASED RATIO (&gt;2</b> 1. Postrenal azotemia	xia, high fever). (e.g. ureter colostomy) ass (subnormal creatinine produ tetracycline, glucocorticoids) <b>0:1) WITH ELEVATED CREATININE</b> (BUN rises disproportionately n	E <b>LEVELS:</b> nore than creatinine) (e.g. obstructiv			,	otem diet,
7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. <b>INCREASED RATIO (&gt;2</b> 1. Postrenal azotemia 2. Prerenal azotemia <b>DECREASED RATIO (</b> <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 of 8. Pregnancy. <b>DECREASED RATIO (</b> <1 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients <b>INAPPROPIATE RATIO</b> 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther <b>ESTIMATED GLOMERL</b> <b>CKD STAGE</b>	xia, high fever). (e.g. ureter colostomy) ass (subnormal creatinine produ- tetracycline, glucocorticoids) <b>0:1) WITH ELEVATED CREATININE</b> (BUN rises disproportionately n superimposed on renal disease. <b>0:1) WITH DECREASED BUN :</b> osis. d starvation. 2. creased urea synthesis. urea rather than creatinine diffu- monemias (urea is virtually absection f inappropiate antidiuretic harm <b>0:1) WITH INCREASED CREATININ</b> py (accelerates conversion of creatinine). who develop renal failure. sis (acetoacetate causes false in creased BUN/creatinine ratio). apy (interferes with creatinine n <b>ILAR FILTERATION RATE:</b> <b>DESCRIPTION</b>	uction) E LEVELS: nore than creatinine) (e.g. obstructive uses out of extracellular fluid). ent in blood). none) due to tubular secretion of urea NE: eatine to creatinine). crease in creatinine with certain mean neasurement). GFR (mL/min/1.73m2)	ve uropathy) ea. ethodologies	). s,resulting in no <b>IATED FINDINGS</b>	rmal ratio wh	
<ol> <li>7. Urine reabsorption</li> <li>8. Reduced muscle m</li> <li>9. Certain drugs (e.g.</li> <li>NCREASED RATIO (&gt;2</li> <li>9. Postrenal azotemia</li> <li>2. Prerenal azotemia</li> <li>2. Prerenal azotemia</li> <li>2. Prerenal azotemia</li> <li>2. Prerenal azotemia</li> <li>2. Decreased RATIO (&lt;1</li> <li>1. Acute tubular necr</li> <li>2. Low protein diet ar</li> <li>3. Severe liver disease</li> <li>4. Other causes of de</li> <li>5. Repeated dialysis (</li> <li>6. Inherited hyperam</li> <li>7. SIADH (syndrome c</li> <li>8. Pregnancy.</li> <li>DECREASED RATIO (&lt;1</li> <li>1. Phenacimide thera</li> <li>2. Rhabdomyolysis (r</li> <li>3. Muscular patients</li> <li>NAPPROPIATE RATIO</li> <li>4. Diabetic ketoacido</li> <li>5. Hould produce an in</li> <li>2. Cephalosporin there</li> </ol>	xia, high fever). (e.g. ureter colostomy) ass (subnormal creatinine produ- tetracycline, glucocorticoids) <b>0:1) WITH ELEVATED CREATININE</b> (BUN rises disproportionately n superimposed on renal disease. <b>0:1) WITH DECREASED BUN :</b> osis. d starvation. 2. creased urea synthesis. urea rather than creatinine diffu- monemias (urea is virtually absect f inappropiate antidiuretic harm <b>0:1) WITH INCREASED CREATININ</b> py (accelerates conversion of creatine). who develop renal failure. : sis (acetoacetate causes false in creased BUN/creatinine ratio). apy (interferes with creatinine n <b>ILAR FILTERATION RATE:</b>	uction) E LEVELS: nore than creatinine) (e.g. obstructiv uses out of extracellular fluid). ent in blood). none) due to tubular secretion of ure NE: eatine to creatinine). crease in creatinine with certain me neasurement). GFR (mL/min/1.73m2) tion >90	ve uropathy) ea. ethodologies	). s,resulting in no	rmal ratio wh	
2. Urine reabsorption 3. Reduced muscle m 4. Certain drugs (e.g. NCREASED RATIO (>2 4. Postrenal azotemia 5. Prerenal azotemia 6. Certain drubular necr 7. Low protein diet ar 7. Severe liver disease 6. Other causes of de 6. Repeated dialysis ( 6. Inherited hyperam 7. SIADH (syndrome c 6. Pregnancy. 5. DECREASED RATIO (<1 7. Phenacimide thera 7. Rhabdomyolysis (r 7. Muscular patients 5. NAPPROPIATE RATIO 6. Diabetic ketoacido 6. Nould produce an in 6. Cephalosporin ther 6. STIMATED GLOMERL 6. G1 6. G1 6. G2	xia, high fever). (e.g. ureter colostomy) ass (subnormal creatinine produ- tetracycline, glucocorticoids) <b>0:1) WITH ELEVATED CREATININE</b> (BUN rises disproportionately n superimposed on renal disease. <b>0:1) WITH DECREASED BUN :</b> osis. d starvation. 2. creased urea synthesis. urea rather than creatinine diffu- monemias (urea is virtually absection f inappropiate antidiuretic harm <b>0:1) WITH INCREASED CREATININ</b> py (accelerates conversion of creatine). who develop renal failure. : sis (acetoacetate causes false in creased BUN/creatinine ratio). apy (interferes with creatinine n <b>ILAR FILTERATION RATE:</b> <u>DESCRIPTION</u> <u>Normal kidney func</u> Kidney damage wi normal or high GF	uction) E LEVELS: nore than creatinine) (e.g. obstructive uses out of extracellular fluid). ent in blood). none) due to tubular secretion of ure NE: eatine to creatinine). Crease in creatinine with certain measurement). Crease in crease in creatinine with certain measurement). Crease in crease in creatinine with certain measurement). Crease in crease in crease in crease in certain measurement). Crease in crease in crease in crease in certain measurement). Crease in crease in crease in crease in certain measurement). Crease in crease in crease in certain measurement). Crease in crease in crease in certain measurement). Crease in crease in certain certain measurement). Crease in certain cert	ve uropathy) ea. ethodologies ASSOC	). s,resulting in no <b>IATED FINDINGS</b> proteinuria	rmal ratio who	
Urine reabsorption     Reduced muscle m     Certain drugs (e.g.     NCREASED 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     Rhabdomyolysis (r     Muscular patients     NAPPROPIATE RATIO     Diabetic ketoacido     hould produce an in     CERD STAGE     G1     G2	xia, high fever). (e.g. ureter colostomy) ass (subnormal creatinine produ- tetracycline, glucocorticoids) <b>0:1) WITH ELEVATED CREATININE</b> (BUN rises disproportionately n superimposed on renal disease. <b>0:1) WITH DECREASED BUN :</b> osis. Id starvation. a. creased urea synthesis. urea rather than creatinine diffu- monemias (urea is virtually abse- f inappropiate antidiuretic harm <b>0:1) WITH INCREASED CREATININ</b> 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 n <b>ILAR FILTERATION RATE:</b> <u>DESCRIPTION</u> <u>Normal kidney func</u> <u>Kidney damage wi</u> <u>normal or high GF</u> <u>Mild decrease in G</u>	uction) <b>E LEVELS:</b> nore than creatinine) (e.g. obstructive uses out of extracellular fluid). ent in blood). none) due to tubular secretion of urea <b>VE:</b> eatine to creatinine). ucrease in creatinine with certain mean neasurement). <u>GFR (mL/min/1.73m2)</u> tion >90 th >90 FR 60 -89	ve uropathy) ea. ethodologies ASSOC	). s,resulting in no IATED FINDINGS proteinuria nce of Protein ,	rmal ratio who	
Y. Urine reabsorption     Reduced muscle m     Certain drugs (e.g.     NCREASED RATIO (>2     Postrenal azotemia     DECREASED RATIO (<1     Acute tubular necr     Low protein diet ar     Severe liver disease     Other causes of de     Repeated dialysis (     SIADH (syndrome c     Rhabdomyolysis (r     Rhabdomyolysis (r     Rhabdomyolysis (r     SMUSCULAR PATIO     Diabetic ketoacido     hould produce an in     CENTATED GLOMERL     CKD STAGE     G1     G2	xia, high fever). (e.g. ureter colostomy) ass (subnormal creatinine produ- tetracycline, glucocorticoids) <b>0:1) WITH ELEVATED CREATININE</b> (BUN rises disproportionately n superimposed on renal disease. <b>0:1) WITH DECREASED BUN :</b> osis. d starvation. b. creased urea synthesis. urea rather than creatinine diffu- monemias (urea is virtually absection f inappropiate antidiuretic harm <b>0:1) WITH INCREASED CREATININ</b> py (accelerates conversion of creatine). who develop renal failure. sis (acetoacetate causes false in creased BUN/creatinine ratio). apy (interferes with creatinine n <b>ILAR FILTERATION RATE:</b> <u>DESCRIPTION</u> <u>Normal kidney func</u> Kidney damage wi normal or high GF	uction) <b>E LEVELS:</b> nore than creatinine) (e.g. obstructive uses out of extracellular fluid). ent in blood). none) due to tubular secretion of urea <b>NE:</b> eatine to creatinine). acrease in creatinine with certain mean neasurement). <u>GFR (mL/min/1.73m2)</u> tion >90 th	ve uropathy) ea. ethodologies ASSOC	). s,resulting in no IATED FINDINGS proteinuria nce of Protein ,	rmal ratio who	



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







	Dr. Vinay Chopr MD (Pathology & Mic Chairman & Consulta	robiology) ME	m <b>Chopra</b> D (Pathology) ht Pathologist
NAME	: Mrs. SEETU REHAN		
AGE/ GENDER	: 47 YRS/FEMALE	PATIENT ID	: 1576446
COLLECTED BY	: SURJESH	<b>REG. NO./LAB NO.</b>	: 012408100017
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 10/Aug/2024 10:15 AM
BARCODE NO.	:01514814	COLLECTION DATE	: 10/Aug/2024 10:19AM
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Test Name		Value Unit	<b>Biological Reference interval</b>

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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	Chairman & Co		CEO & Consultant Pat	
IAME	: Mrs. SEETU REHAN			
AGE/ GENDER	: 47 YRS/FEMALE	PA	TIENT ID :	: 1576446
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LIENT ADDRESS	: 6349/1, NICHOLSON ROAI	D, AMBALA CANTT		
Cost Namo				
HYROID STIMULAT	ING HORMONE (TSH): SERUM			Biological Reference interval
THYROID STIMULAT by CMIA (CHEMILUMIN Frd GENERATION, ULT	ING HORMONE (TSH): SERUM	ENDOCRIN YROID STIMULATIN 1 1.183	IOLOGY G HORMONE (TSH)	
HYROID STIMULAT by CMIA (CHEMILUMIN ind Generation, ult	ING HORMONE (TSH): SERUM	ENDOCRIN YROID STIMULATIN 1 1.183	IOLOGY G HORMONE (TSH)	0.35 - 5.50
HYROID STIMULAT by CMIA (CHEMILUMIN ind Generation, ult	ING HORMONE (TSH): SERUM vescent microparticle immuno rasensitive	ENDOCRIN YROID STIMULATIN 1 1.183	OLOGY G HORMONE (TSH) μIU/mL REFFERENCE RANGE (μι 0.70 – 15.20	0.35 - 5.50
HYROID STIMULAT by CMIA (CHEMILUMIN rd GENERATION, ULT	ING HORMONE (TSH): SERUM VESCENT MICROPARTICLE IMMUNO TRASENSITIVE AGE 0 – 5 DAYS 6 Days – 2 Months	ENDOCRIN YROID STIMULATIN 1 1.183	OLOGY G HORMONE (TSH) μIU/mL <u>REFFERENCE RANGE (μIL</u> 0.70 – 15.20 0.70 – 11.00	0.35 - 5.50
HYROID STIMULAT by CMIA (CHEMILUMIN ind Generation, ult	ING HORMONE (TSH): SERUM NESCENT MICROPARTICLE IMMUNO TRASENSITIVE AGE 0 – 5 DAYS 6 Days – 2 Months 3 – 11 Months	ENDOCRIN YROID STIMULATIN 1 1.183	OLOGY G HORMONE (TSH) μIU/mL <u>REFFERENCE RANGE (μIL</u> 0.70 – 15.20 0.70 – 11.00 0.70 – 8.40	0.35 - 5.50
THYROID STIMULAT by CMIA (CHEMILUMIN Brd GENERATION, ULT	ING HORMONE (TSH): SERUM NESCENT MICROPARTICLE IMMUNO TRASENSITIVE AGE 0 – 5 DAYS 6 Days – 2 Months 3 – 11 Months 1 – 5 Years	ENDOCRIN YROID STIMULATIN 1 1.183	IOLOGY G HORMONE (TSH) μIU/mL REFFERENCE RANGE (μIL 0.70 – 15.20 0.70 – 11.00 0.70 – 8.40 0.70 – 7.00	0.35 - 5.50
	ING HORMONE (TSH): SERUM VESCENT MICROPARTICLE IMMUNO TRASENSITIVE AGE 0 – 5 DAYS 6 Days – 2 Months 3 – 11 Months 1 – 5 Years 6 – 10 Years	ENDOCRIN YROID STIMULATIN 1 1.183	IOLOGY G HORMONE (TSH) μIU/mL REFFERENCE RANGE (μIL 0.70 – 15.20 0.70 – 11.00 0.70 – 8.40 0.70 – 7.00 0.60 – 5.50	0.35 - 5.50
THYROID STIMULAT by CMIA (CHEMILUMIN Frd GENERATION, ULT	ING HORMONE (TSH): SERUM VESCENT MICROPARTICLE IMMUNO RASENSITIVE AGE 0 – 5 DAYS 6 Days – 2 Months 3 – 11 Months 1 – 5 Years 6 – 10 Years 11 - 15	ENDOCRIN YROID STIMULATIN 1 1.183	IOLOGY G HORMONE (TSH) μIU/mL REFFERENCE RANGE (μIL 0.70 – 15.20 0.70 – 11.00 0.70 – 8.40 0.70 – 7.00 0.60 – 5.50 0.50 – 5.50	0.35 - 5.50
HYROID STIMULAT by CMIA (CHEMILUMIN rd GENERATION, ULT	ING HORMONE (TSH): SERUM VESCENT MICROPARTICLE IMMUNO TRASENSITIVE AGE 0 – 5 DAYS 6 Days – 2 Months 3 – 11 Months 1 – 5 Years 6 – 10 Years	ENDOCRIN YROID STIMULATIN 1 1.183 DASSAY)	IOLOGY G HORMONE (TSH) μIU/mL REFFERENCE RANGE (μIL 0.70 – 15.20 0.70 – 11.00 0.70 – 8.40 0.70 – 7.00 0.60 – 5.50	0.35 - 5.50
HYROID STIMULAT by CMIA (CHEMILUMIN rd GENERATION, ULT	ING HORMONE (TSH): SERUM VESCENT MICROPARTICLE IMMUNO RASENSITIVE AGE 0 – 5 DAYS 6 Days – 2 Months 3 – 11 Months 1 – 5 Years 6 – 10 Years 11 - 15 > 20 Years (Adults)	ENDOCRIN YROID STIMULATIN 1 1.183	IOLOGY G HORMONE (TSH) μIU/mL REFFERENCE RANGE (μIL 0.70 – 15.20 0.70 – 11.00 0.70 – 8.40 0.70 – 7.00 0.60 – 5.50 0.50 – 5.50 0.27 – 5.50	0.35 - 5.50
THYROID STIMULAT by CMIA (CHEMILUMIN Frd GENERATION, ULT	ING HORMONE (TSH): SERUM VESCENT MICROPARTICLE IMMUNO RASENSITIVE AGE 0 – 5 DAYS 6 Days – 2 Months 3 – 11 Months 1 – 5 Years 6 – 10 Years 11 - 15	ENDOCRIN YROID STIMULATIN 1 1.183 DASSAY)	IOLOGY G HORMONE (TSH) μIU/mL REFFERENCE RANGE (μIL 0.70 – 15.20 0.70 – 11.00 0.70 – 8.40 0.70 – 7.00 0.60 – 5.50 0.50 – 5.50	0.35 - 5.50

**KOS Diagnostic Lab** 

(A Unit of KOS Healthcare)

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

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

2. Hypothyroid patients receiving insufficient thyroid replacement therapy.

3.Hashimotos thyroiditis.

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

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

DECREASED LEVELS:

1. Toxic multi-nodular goitre & Thyroiditis.

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

3. Autonomously functioning Thyroid adenoma

4. Secondary pituatary or hypothalmic hypothyroidism

5. Acute psychiatric illness

6.Severe dehydration.



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	Dr. Vinay Chc MD (Pathology & Chairman & Const	Microbiology)		n <b>Chopra</b> (Pathology) : Pathologist
NAME	: Mrs. SEETU REHAN			
AGE/ GENDER	: 47 YRS/FEMALE	PATIENT ID		: 1576446
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Test Name		Value	Unit	Biological Reference interval

7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis. 8.Pregnancy: 1st and 2nd Trimester

## LIMITATIONS:

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

\*\*\* End Of Report \*\*?



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