

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



	Dr. Vinay Chopr MD (Pathology & Micı Chairman & Consulta	robiology)		(Pathology)
NAME	: Mrs. SNEH PRABHA			
AGE/ GENDER	: 63 YRS/FEMALE		PATIENT ID	: 612561
<b>COLLECTED BY</b>	:		REG. NO./LAB NO.	: 042410050001
<b>REFERRED BY</b>	:		<b>REGISTRATION DATE</b>	: 05/Oct/2024 08:38 AM
BARCODE NO.	: A0465680		COLLECTION DATE	:05/Oct/202402:59PM
CLIENT CODE.	: KOS DIAGNOSTIC SHAHBAD		REPORTING DATE	: 05/Oct/2024 03:27PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMB	ALA CANT	Г	
Test Name		Value	Unit	Biological Reference interval
	SWAS	THYA W	ELLNESS PANEL: 1.4	
			OOD COUNT (CBC)	
RED BLOOD CELLS (R	BCS) COUNT AND INDICES			
HAEMOGLOBIN (HB)		10 <sup>L</sup>	gm/dL	12.0 - 16.0
RED BLOOD CELL (RB	C) COUNT DCUSING, ELECTRICAL IMPEDENCE	4.21	Millions/	cmm 3.50 - 5.00
PACKED CELL VOLUM		32.5 <sup>L</sup>	%	37.0 - 50.0
MEAN CORPUSCULAR		77.2 <sup>L</sup>	fL	80.0 - 100.0
MEAN CORPUSCULAR	R HAEMOGLOBIN (MCH)	23.8 <sup>L</sup>	pg	27.0 - 34.0
MEAN CORPUSCULAR	R HEMOGLOBIN CONC. (MCHC)	30.8 <sup>L</sup>	g/dL	32.0 - 36.0
<b>RED CELL DISTRIBUTI</b>	ON WIDTH (RDW-CV)	16.6 <sup>H</sup>	%	11.00 - 16.00
RED CELL DISTRIBUTI	ON WIDTH (RDW-SD) JTOMATED HEMATOLOGY ANALYZER	47.8	fL	35.0 - 56.0
MENTZERS INDEX		18.34	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX	(	30.5	RATIO	BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CELLS	(WBCS)			
TOTAL LEUCOCYTE CO	DUNT (TLC) by sf cube & microscopy	8960	/cmm	4000 - 11000
NUCLEATED RED BLO		NIL		0.00 - 20.00
NUCLEATED RED BLO	OD CELLS (nRBCS) % <i>itomated hematology analyzer</i>	NIL	%	< 10 %
DIFFERENTIAL LEUCO	<u>CYTE COUNT (DLC)</u>			
	BY SF CUBE & MICROSCOPY	57	%	50 - 70

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







	<b>Dr. Vinay Chop</b> MD (Pathology & Mi Chairman & Consult	crobiology)	Dr. Yugam MD ( CEO & Consultant	(Pathology)
NAME	: Mrs. SNEH PRABHA			
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Test Name		Value	Unit	Biological Reference interval
LYMPHOCYTES		31	%	20 - 40
EOSINOPHILS	Y BY SF CUBE & MICROSCOPY Y BY SF CUBE & MICROSCOPY	4	%	1 - 6
MONOCYTES	Y BY SF CUBE & MICROSCOPY	8	%	2 - 12
BASOPHILS	Y BY SF CUBE & MICROSCOPY	0	%	0 - 1
	PHIL COUNT Y BY SF CUBE & MICROSCOPY	5107	/cmm	2000 - 7500
ABSOLUTE LYMPHO		2778	/cmm	800 - 4900
ABSOLUTE EOSINOP		358	/cmm	40 - 440
ABSOLUTE MONOCY by FLOW CYTOMETRY	TE COUNT y by sf cube & microscopy	717	/cmm	80 - 880
PLATELETS AND OTH	HER PLATELET PREDICTIVE MARKE	<u>RS.</u>		
PLATELET COUNT (P by hydro dynamic f	LT) FOCUSING, ELECTRICAL IMPEDENCE	277000	/cmm	150000 - 450000
PLATELETCRIT (PCT)	FOCUSING, ELECTRICAL IMPEDENCE	0.37 <sup>H</sup>	%	0.10 - 0.36
MEAN PLATELET VO		13 <sup>H</sup>	fL	6.50 - 12.0
PLATELET LARGE CEI	LL COUNT (P-LCC) Focusing, electrical impedence	141000 <sup>H</sup>	/cmm	30000 - 90000
	FOCUSING, ELECTRICAL IMPEDENCE	50.8 <sup>H</sup>	%	11.0 - 45.0
PLATELET DISTRIBU by HYDRO DYNAMIC F	TION WIDTH (PDW) FOCUSING, ELECTRICAL IMPEDENCE	16.1	%	15.0 - 17.0

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

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Test Name		Value	Unit	Biological Reference interval
	GL	YCOSYLATED HAE	MOGLOBIN (HBA1C)	
GLYCOSYLATED HAEM		YCOSYLATED HAE	MOGLOBIN (HBA1C) %	4.0 - 6.4
WHOLE BLOOD	DGLOBIN (HbA1c):			4.0 - 6.4
WHOLE BLOOD by HPLC (HIGH PERFORM	DGLOBIN (HbA1c):			4.0 - 6.4 60.00 - 140.00
ESTIMATED AVERAGE I by HPLC (HIGH PERFORM	DGLOBIN (HbA1c):	6.3	%	
WHOLE BLOOD by HPLC (HIGH PERFORM ESTIMATED AVERAGE I by HPLC (HIGH PERFORM	DGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY)	6.3 134.11	% mg/dL	
WHOLE BLOOD by HPLC (HIGH PERFORM ESTIMATED AVERAGE I by HPLC (HIGH PERFORM INTERPRETATION:	DGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN DIAB	6.3 134.11 SETES ASSOCIATION (A	% mg/dL DA):	60.00 - 140.00
WHOLE BLOOD by HPLC (HIGH PERFORM ESTIMATED AVERAGE I by HPLC (HIGH PERFORM INTERPRETATION: RE	DGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN DIAB FERENCE GROUP	6.3 134.11 SETES ASSOCIATION (A	% mg/dL DA): ITED HEMOGLOGIB (HBAIC) i	60.00 - 140.00
WHOLE BLOOD by HPLC (HIGH PERFORM ESTIMATED AVERAGE I by HPLC (HIGH PERFORM INTERPRETATION: RE Non diab	DGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN DIAB FERENCE GROUP Metic Adults >= 18 years	6.3 134.11 SETES ASSOCIATION (A	% mg/dL DA): ITED HEMOGLOGIB (HBAIC) i <5.7	60.00 - 140.00
WHOLE BLOOD by HPLC (HIGH PERFORM ESTIMATED AVERAGE I by HPLC (HIGH PERFORM INTERPRETATION: RE Non diab At I	DGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN DIAB FERENCE GROUP	6.3 134.11 SETES ASSOCIATION (A	% mg/dL DA): ITED HEMOGLOGIB (HBAIC) i	60.00 - 140.00

GLYCOSYLATED HEMOGL	OGIB (HBAIC) in %
<5.7	
5.7 - 6.4	1
>= 6.5	
Age > 19 Ye	ears
Goals of Therapy:	< 7.0
Actions Suggested:	>8.0
Age < 19 Ye	ears
Goal of therapy:	<7.5
	5.7 – 6.2 >= 6.5 Age > 19 Ye Goals of Therapy:

#### COMMENTS:

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

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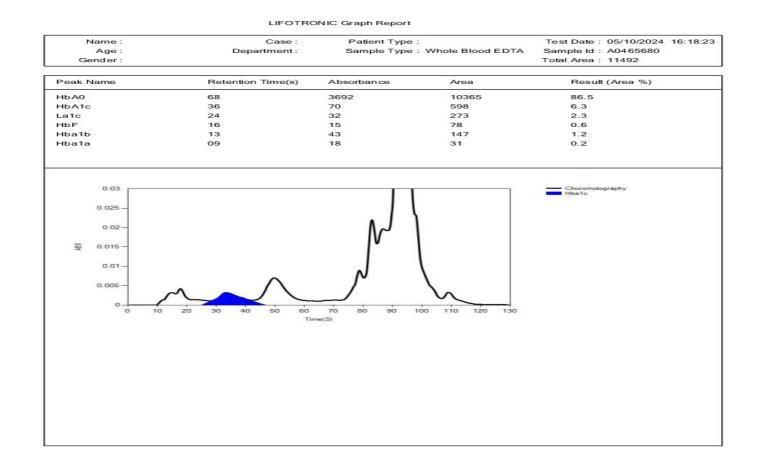
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4.High





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Test Name	Val	ue Unit	Biological Reference interval







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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBAI	LA CANTT	
Test Name		Value Unit	Biological Reference interval
	ERYTHROCY	TE SEDIMENTATION RATE (ES	SR)
	MENTATION RATE (ESR) GATION BY CAPILLARY PHOTOMETRY	78 <sup>H</sup> mm/1st	hr 0 - 20
1. ESR is a non-specif immune disease, but 2. An ESR can be affe as C-reactive protein	does not tell the health practitioner exa cted by other conditions besides inflam be used to monitor disease activity and	actly where the inflammation is in th mation. For this reason, the ESR is ty	tion associated with infection, cancer and auto- ne body or what is causing it. ypically used in conjunction with other test such above diseases as well as some others, such as
<b>CONDITION WITH LOV</b> A low ESR can be see (polycythaemia), sign	<b>W ESR</b> n with conditions that inhibit the norma	al sedimentation of red blood cells, s eucocytosis) , and some protein abno	such as a high red blood cell count ormalities. Some changes in red cell shape (such

 ESR and C - reactive protein (C-RP) are both markers of inflammation.
 Generally, ESR does not change as rapidly as does CRP, either at the start of inflammation or as it resolves.
 **CRP is not affected by as many other factors as is ESR, making it a better marker of inflammation.** If the ESR is elevated, it is typically a result of two types of proteins, globulins or fibrinogen.
 Women tend to have a higher ESR, and menstruation and pregnancy can cause temporary elevations.
 Drugs such as dextran, methyldopa, oral contraceptives, penicillamine procainamide, theophylline, and vitamin A can increase ESR, while exprise contrace and quiping may decrease it. aspirin, cortisone, and quinine may decrease it





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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
GLUCOSE FASTING ( by GLUCOSE OXIDAS	r): PLASIVIA SE - PEROXIDASE (GOD-POD)	101.72 <sup>H</sup>	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0
1. A fasting plasma g 2. A fasting plasma g test (after consumpt 3. A fasting plasma g	ion of 75 ams of alucose) is recom	considered normal ng/dl is considerec nmended for all su s highly suggestive	l as glucose intolerant or ch patients. e of diabetic state. A repe	prediabetic. A fasting and post-prandial blood at post-prandial is strongly recommended for al atory for diabetic state.

KOS Diagnostic Lab (A Unit of KOS Healthcare)





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Test Name		Value	Unit	Biological Reference interval
		LIPID PROFILE :	BASIC	
CHOLESTEROL TOTAL by CHOLESTEROL OXI		158.06	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.
TRIGLYCERIDES: SER by GLYCEROL PHOSP	UM HATE OXIDASE (ENZYMATIC)	193.87 <sup>H</sup>	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROL (I by SELECTIVE INHIBITI		43.51	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: S by CALCULATED, SPEC		75.78	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 CHOLESTEF by CALCULATED, SPEC		114.55	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 CHOLESTEROL: by CALCULATED, SPEC		38.77	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUN	Λ	509.99	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL R by CALCULATED, SPEC	ATIO: SERUM	3.63	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: SERI by CALCULATED, SPEC		1.74	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0

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	Chairman & Cons	<b>O</b> , ,	CEO & Consultant	
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Test Name		Value	Unit	Biological Reference interval
TRIGLYCERIDES/HDI	RATIO: SERUM	4.46	RATIO	3.00 - 5.00

TRIGLYCERIDES/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY

## **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
			ON TEST (COMPLETE)	
BILIRUBIN TOTAL: SI by diazotization, sf	ERUM PECTROPHOTOMETRY	0.73	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (C	CONJUGATED): SERUM	0.16	mg/dL	0.00 - 0.40
	(UNCONJUGATED): SERUM	0.57	mg/dL	0.10 - 1.00
SGOT/AST: SERUM	RIDOXAL PHOSPHATE	35.49	U/L	7.00 - 45.00
SGPT/ALT: SERUM	RIDOXAL PHOSPHATE	25.96	U/L	0.00 - 49.00
AST/ALT RATIO: SER by CALCULATED, SPE	UM	1.37	RATIO	0.00 - 46.00
ALKALINE PHOSPHA		65	U/L	40.0 - 150.0
GAMMA GLUTAMYL by szasz, spectrof	. TRANSFERASE (GGT): SERUM	21	U/L	0.00 - 55.0
TOTAL PROTEINS: SE	RUM	7.36	gm/dL	6.20 - 8.00
ALBUMIN: SERUM		4.51	gm/dL	3.50 - 5.50
GLOBULIN: SERUM		2.85	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by CALCULATED, SPE		1.58	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





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HEPATOCELLULAR C.	ARCINOMA & CHRONIC HEPATITIS		> 1.3 (Slightly Inc	reased)

THEFATOCELEOLAR CARCINOWA & CHRONIC HEFATTIS	
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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NAME	: Mrs. SNEH PRABHA			
AGE/ GENDER	: 63 YRS/FEMALE	PAT	TIENT ID	: 612561
COLLECTED BY	:	REG	. NO./LAB NO.	:042410050001
<b>REFERRED BY</b>	:	REG	SISTRATION DATE	: 05/Oct/2024 08:38 AM
BARCODE NO.	: A0465679	COI	LECTION DATE	:05/Oct/2024 02:59PM
CLIENT CODE.	: KOS DIAGNOSTIC SHAHBAD	REP	ORTING DATE	: 05/Oct/2024 04:27PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	кі	DNEY FUNCTION T	EST (COMPLETE)	
UREA: SERUM		19.68	mg/dL	10.00 - 50.00
	ATE DEHYDROGENASE (GLDH)	0.50	in a fall	0.40 1.20
CREATININE: SERUN by ENZYMATIC, SPEC		0.58	mg/dL	0.40 - 1.20
BLOOD UREA NITRC		9.2	mg/dL	7.0 - 25.0
by CALCULATED, SPE	<i>есткорнотометку</i> )GEN (BUN)/CREATININE	15.86	RATIO	10.0 - 20.0
RATIO: SERUM		15.00	KATIO	10.0 - 20.0
by CALCULATED, SPE			DATIO	
UREA/CREATININE F		33.93	RATIO	
URIC ACID: SERUM		4.3	mg/dL	2.50 - 6.80
by URICASE - OXIDAS	SE PEROXIDASE	0.27	ma/dl	0.50, 10.40
CALCIUM: SERUM by ARSENAZO III, SPE	CTROPHOTOMETRY	9.36	mg/dL	8.50 - 10.60
PHOSPHOROUS: SEF		4.24	mg/dL	2.30 - 4.70
by PHOSPHOMOLYBE ELECTROLYTES	DATE, SPECTROPHOTOMETRY			
SODIUM: SERUM		135.6	mmol/L	135.0 - 150.0
by ISE (ION SELECTIV		4.70		
POTASSIUM: SERUN by ISE (ION SELECTIV		4.79	mmol/L	3.50 - 5.00
CHLORIDE: SERUM		101.7	mmol/L	90.0 - 110.0
by ISE (ION SELECTIV	'E ELECTRODE) RULAR FILTERATION RATE			
		101 /		
estimated glome (egfr): serum by calculated	RULAR FILTERATION RATE	101.6		

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







		Chopra gy & Microbiology) Consultant Pathologist	Dr. Yugan MD CEO & Consultan	(Pathology)	
NAME	: Mrs. SNEH PRABHA				
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROA	AD, AMBALA CANTT			
Test Name		Value	Unit	Biological Re	eference interval
2. Low protein diet a 3. Severe liver diseas					
<ol> <li>Severe liver diseas</li> <li>Other causes of de</li> <li>Repeated dialysis</li> <li>Inherited hyperam</li> <li>SIADH (syndrome of</li> <li>Pregnancy.</li> <li>DECREASED RATIO (&lt;</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>INAPPROPIATE RATIO</li> <li>Diabetic ketoacido</li> <li>should produce an in</li> <li>Cephalosporin thei</li> </ol>	e. Acreased urea synthesis. (urea rather than creatinine of monemias (urea is virtually a of inappropiate antidiuretic h 10:1) WITH INCREASED CREAT (by (accelerates conversion of eleases muscle creatinine). who develop renal failure. bis (acetoacetate causes falso creased BUN/creatinine ratio rapy (interferes with creatinin JLAR FILTERATION RATE:	absent in blood). armone) due to tubular s ININE: f creatine to creatinine). e increase in creatinine v b). he measurement).	ecretion of urea. vith certain methodolo		atio when dehydratio
<ol> <li>Severe liver diseas</li> <li>Other causes of de</li> <li>Repeated dialysis</li> <li>Inherited hyperam</li> <li>SIADH (syndrome of</li> <li>Pregnancy.</li> <li>PCREASED RATIO (</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>NAPPROPIATE RATIO</li> <li>Diabetic ketoacido</li> <li>Should produce an in</li> <li>Cephalosporin the</li> <li>CETIMATED GLOMERI</li> <li>CKD STAGE</li> </ol>	e. Acreased urea synthesis. (urea rather than creatinine of amonemias (urea is virtually a of inappropiate antidiuretic h 10:1) WITH INCREASED CREAT apy (accelerates conversion of eleases muscle creatinine). who develop renal failure. sis (acetoacetate causes false creased BUN/creatinine ratio rapy (interferes with creatinine JLAR FILTERATION RATE: DESCRIPTIC	absent in blood). armone) due to tubular s ININE: f creatine to creatinine). e increase in creatinine v b). ne measurement). DNGFR ( mL/r	ecretion of urea. vith certain methodolo nin/1.73m2 ) AS	SOCIATED FINDINGS	atio when dehydratio
<ol> <li>Severe liver diseas</li> <li>Other causes of de</li> <li>Repeated dialysis</li> <li>Inherited hyperam</li> <li>SIADH (syndrome of</li> <li>Pregnancy.</li> <li>DECREASED RATIO (</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>NAPPROPIATE RATIO</li> <li>Diabetic ketoacido</li> <li>Should produce an in</li> <li>Cephalosporin their</li> <li>ESTIMATED GLOMERI</li> <li>CKD STAGE</li> <li>G1</li> </ol>	e. Acreased urea synthesis. (urea rather than creatinine of imonemias (urea is virtually a of inappropiate antidiuretic h 10:1) WITH INCREASED CREAT apy (accelerates conversion of eleases muscle creatinine). who develop renal failure. b: sis (acetoacetate causes false creased BUN/creatinine ratio rapy (interferes with creatinine JLAR FILTERATION RATE: DESCRIPTIO Normal kidney fu	absent in blood). armone) due to tubular s ININE: f creatine to creatinine). e increase in creatinine v b). ne measurement). DN GFR (mL/m unction >	ecretion of urea. vith certain methodolo nin/1.73m2) AS 90	SOCIATED FINDINGS	atio when dehydratio
<ol> <li>Severe liver diseas</li> <li>Other causes of de</li> <li>Repeated dialysis</li> <li>Inherited hyperam</li> <li>SIADH (syndrome of</li> <li>Pregnancy.</li> <li>DECREASED RATIO (&lt;</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>NAPPROPIATE RATIO</li> <li>Diabetic ketoacido</li> <li>Should produce an in</li> <li>Cephalosporin the</li> <li>ESTIMATED GLOMERI</li> <li>CKD STAGE</li> </ol>	e. creased urea synthesis. (urea rather than creatinine of imonemias (urea is virtually a of inappropiate antidiuretic h 10:1) WITH INCREASED CREAT py (accelerates conversion of eleases muscle creatinine). who develop renal failure. b: sis (acetoacetate causes faise creased BUN/creatinine ratio rapy (interferes with creatinine JLAR FILTERATION RATE: DESCRIPTIO Normal kidney fai Kidney damage	absent in blood).         armone) due to tubular s <b>ININE:</b> f creatine to creatinine).         e increase in creatinine voltation         b).         ne measurement). <b>DN GFR ( mL/r</b> unction         e with	ecretion of urea. vith certain methodolo nin/1.73m2) AS 90 P	SOCIATED FINDINGS No proteinuria resence of Protein ,	atio when dehydratio
<ol> <li>Severe liver diseas</li> <li>Other causes of de</li> <li>Repeated dialysis</li> <li>Inherited hyperam</li> <li>SIADH (syndrome of</li> <li>Pregnancy.</li> <li>DECREASED RATIO (</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>INAPPROPIATE RATIO</li> <li>Diabetic ketoacido</li> <li>Should produce an in</li> <li>Cephalosporin the</li> <li>ESTIMATED GLOMERI</li> <li>G1</li> <li>G2</li> </ol>	e. creased urea synthesis. (urea rather than creatinine of imonemias (urea is virtually a of inappropiate antidiuretic h <b>10:1) WITH INCREASED CREAT</b> upy (accelerates conversion of eleases muscle creatinine). who develop renal failure. b: sis (acetoacetate causes false creased BUN/creatinine ratio rapy (interferes with creatinine JLAR FILTERATION RATE: DESCRIPTIO Normal kidney fal Kidney damage normal or high	absent in blood).         armone) due to tubular s <b>ININE:</b> f creatine to creatinine).         e increase in creatinine voltation         b).         ne measurement). <b>DN GFR ( mL/r</b> aunction         a with         a GFR	ecretion of urea. vith certain methodolo nin/1.73m2) AS 90 P 90 P Alb	SOCIATED FINDINGS	atio when dehydratio
<ol> <li>Severe liver diseas</li> <li>Other causes of de</li> <li>Repeated dialysis</li> <li>Inherited hyperam</li> <li>SIADH (syndrome of</li> <li>Pregnancy.</li> <li>DECREASED RATIO (</li> <li>Rhabdomyolysis (r</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>NAPPROPIATE RATIO</li> <li>Diabetic ketoacido</li> <li>cephalosporin their</li> <li>CED STAGE</li> <li>CKD STAGE</li> <li>G1</li> </ol>	e. creased urea synthesis. (urea rather than creatinine of imonemias (urea is virtually a of inappropiate antidiuretic h 10:1) WITH INCREASED CREAT py (accelerates conversion of eleases muscle creatinine). who develop renal failure. b: sis (acetoacetate causes faise creased BUN/creatinine ratio rapy (interferes with creatinine JLAR FILTERATION RATE: DESCRIPTIO Normal kidney fai Kidney damage	absent in blood).         armone) due to tubular s <b>ININE:</b> f creatine to creatinine).         e increase in creatinine v         b).         ne measurement).         DN         GFR (mL/r         unction         a with         b GFR         in GFR         600	ecretion of urea. vith certain methodolo nin/1.73m2) AS 90 P	SOCIATED FINDINGS No proteinuria resence of Protein ,	atio when dehydratio



G4

G5

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Severe decrease in GFR

Kidney failure

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

15-29

<15

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NAME	: Mrs. SNEH PRABHA		
AGE/ GENDER	: 63 YRS/FEMALE	PATIENT ID	: 612561
COLLECTED BY	:	<b>REG. NO./LAB NO.</b>	: 042410050001
REFERRED BY	:	<b>REGISTRATION DATE</b>	: 05/Oct/2024 08:38 AM
BARCODE NO.	: A0465679	COLLECTION DATE	:05/Oct/202402:59PM
CLIENT CODE.	: KOS DIAGNOSTIC SHAHBAD	<b>REPORTING DATE</b>	:05/Oct/202404:27PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA (	CANTT	
Test Name	Valu	ue 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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NAME	: Mrs. SNEH PRABHA			
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CLIENT CODE.	: KOS DIAGNOSTIC SHAHBAD	REPO	RTING DATE	:05/Oct/202404:27PM
Test Name		Value	Unit	Biological Reference interva
		IRON PROF	ILE	
IRON: SERUM by Ferrozine, spect		IRON PROF 28 <sup>L</sup>	FILE μg/dL	50.0 - 170.0
IRON: SERUM by Ferrozine, spect	BINDING CAPACITY (UIBC)	IRON PROF	ILE	
IRON: SERUM by FERROZINE, SPECT UNSATURATED IRON :SERUM by FERROZINE, SPECT TOTAL IRON BINDING :SERUM	BINDING CAPACITY (UIBC) <i>ROPHOTOMETERY</i> 5 CAPACITY (TIBC)	IRON PROF 28 <sup>L</sup>	FILE μg/dL	50.0 - 170.0
IRON: SERUM by FERROZINE, SPECT UNSATURATED IRON :SERUM by FERROZINE, SPECT TOTAL IRON BINDING :SERUM by SPECTROPHOTOME %TRANSFERRIN SATU	BINDING CAPACITY (UIBC) ROPHOTOMETERY S CAPACITY (TIBC) ETERY	<b>IRON PROF</b> 28 <sup>L</sup> 296.7	FILE μg/dL μg/dL	<b>50.0 - 170.0</b> 150.0 - 336.0

VARIABLES	ANEMIA OF CHRONIC DISEASE	IRON DEFICIENCY ANEMIA	THALASSEMIA α/β TRAIT
SERUM IRON:	Normal to Reduced	Reduced	Normal
TOTAL IRON BINDING CAPACITY:	Decreased	Increased	Normal
% TRANSFERRIN SATURATION:	Decreased	Decreased < 12-15 %	Normal
SERUM FERRITIN:	Normal to Increased	Decreased	Normal or Increased

IRON:

TEST PERFORMED AT KOS DIAGNOSTIC LAB. AMBALA CANTT

1.Serum iron studies is recommended for differential diagnosis of microcytic hypochromic anemia.i.e iron deficiency anemia, zinc deficiency anemia, anemia of chronic disease and thalassemia syndromes.

2. It is essential to isolate iron deficiency anemia from Beta thalassemia syndromes because during iron replacement which is therapeutic for iron deficiency anemia, is severely contra-indicated in Thalassemia.

**TOTAL IRON BINDING CAPACITY (TIBC):** 1.It is a direct measure of protein transferrin which transports iron from the gut to storage sites in the bone marrow.

## % TRANSFERRIN SATURATION:

1. Occurs in idiopathic hemochromatosis and transfusional hemosiderosis where no unsaturated iron binding capacity is available for iron mobilization. Similar condition is seen in congenital deficiency of transferrin.



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Test Name	: 6349/1, NICHOLSON ROAD, AM	Value	Unit	Biological Reference interval
		ENDOCRING	DLOGY	
	тн	YROID FUNCTION	TEST: TOTAL	
		1.243 AY)	ng/mL	0.35 - 1.93
THYROXINE (T4): SEE	E (T3): SERUM IESCENT MICROPARTICLE IMMUNOASS/	ау) 11.47	ng/mL µgm/dL	0.35 - 1.93 4.87 - 12.60

trilodothyronine (T3).Failure at any level of regulation of the hypothalamic-pituitary-thyroid axis will result in either underproduction (hypothyroidism) or overproduction(hyperthyroidism) of T4 and/or T3.

 CLINICAL CONDITION
 T3
 T4
 TSH

 Primary Hypothyroidism:
 Reduced
 Reduced
 Increased (Significantly)

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 levles 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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DR.YUGAM CHOPRA

CONSULTANT PATHOLOGIST CROBIOLOGY) MBBS , MD (PATHOLOGY)





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Dr. Vinay ChopraDr. Yugam ChopraMD (Pathology & Microbiology)MD (Pathology)Chairman & Consultant PathologistCEO & Consultant Pathologist				
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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	
> 20 years (Adults)	0.35 - 1.93	> 20 Years (Adults)	4.87 - 12.60	> 20 Years (Adults)	0.35- 5.50	
	RECON	MENDATIONS OF TSH LE	VELS DURING PRE	GNANCY ( µIU/mL)		
1st Trimester			0.10 - 2.50			
2nd Trimester			0.20 - 3.00			
	3rd Trimester			0.30 - 4.10		

### **INCREASED TSH LEVELS:**

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

2. Hypothyroid patients receiving insufficient thyroid replacement therapy.

3. Hashimotos thyroiditis

4.DRUGS: Amphetamines, 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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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, Al	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		value	onit	biological kererence interval
		CLINICAL PATHO	LOGY	
	URINE RO	UTINE & MICROSCO	PIC EXAMINAT	ION
<b>PHYSICAL EXAMINA</b>	TION			
OUANTITY RECIEVE		10	ml	
	TANCE SPECTROPHOTOMETRY			
COLOUR	TANCE SPECTROPHOTOMETRY	AMBER YELLOW		PALE YELLOW
TRANSPARANCY		HAZY		CLEAR
	TANCE SPECTROPHOTOMETRY	1 01		1 000 1 000
SPECIFIC GRAVITY by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	1.01		1.002 - 1.030
CHEMICAL EXAMINA				
REACTION		ACIDIC		
by DIP STICK/REFLEC PROTEIN	TANCE SPECTROPHOTOMETRY	Nogativo		NEGATIVE (-ve)
	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
SUGAR		Negative		NEGATIVE (-ve)
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	6.5		5.0 - 7.5
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	0.0		
BILIRUBIN		Negative		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY NITRITE		Negative		NEGATIVE (-ve)
	TANCE SPECTROPHOTOMETRY.			
UROBILINOGEN by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	Normal	EU/dL	0.2 - 1.0
KETONE BODIES		Negative		NEGATIVE (-ve)
by DIP STICK/REFLEC BLOOD	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
	TANCE SPECTROPHOTOMETRY			
ASCORBIC ACID		NEGATIVE (-ve)		NEGATIVE (-ve)
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY				

MICROSCOPIC EXAMINATION



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HEALTHCARE & DIAGNOSTIC EXCELLENCE IN

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

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

NAME	: Mrs. SNEH PRABHA				
AGE/ GENDER	: 63 YRS/FEMALE	PATIENT	ID	: 612561	
COLLECTED BY	:	REG. NO./	'LAB NO.	: 042410050001	
<b>REFERRED BY</b>	:	REGISTRA	ATION DATE	: 05/Oct/2024 08:38 AM	
BARCODE NO.	: A0465681	COLLECT	ION DATE	:05/Oct/202403:15PM	
CLIENT CODE.	: KOS DIAGNOSTIC SHAHBAD	REPORTI	NG DATE	: 05/Oct/2024 03:59PM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT				
Test Name		Value	Unit	Biological Reference interval	
			/LIDE	0.3	

			-
	NEGATIVE (-ve)	/HPF	0 - 3
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT PUS CELLS	10-12	/HPF	0 - 5
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
EPITHELIAL CELLS	4-6	/HPF	ABSENT
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT CRYSTALS	NEGATIVE (-ve)		NEGATIVE (-ve)
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
CASTS	NEGATIVE (-ve)		NEGATIVE (-ve)
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
BACTERIA by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)
OTHERS	NEGATIVE (-ve)		NEGATIVE (-ve)
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
	ABSENT		ABSENT
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			

End Of Report





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