



	Dr. Vinay Chopr MD (Pathology & Mic Chairman & Consulta	crobiology)		(Pathology)
NAME	: Mrs. SONALI			
AGE/ GENDER	: 34 YRS/FEMALE		PATIENT ID	: 1735597
COLLECTED BY	:		<b>REG. NO./LAB NO.</b>	: 012501260012
REFERRED BY	:		REGISTRATION DATE	: 26/Jan/2025 09:33 AM
BARCODE NO.	: 01524443		COLLECTION DATE	: 26/Jan/2025 09:34AM
CLIENT CODE. CLIENT ADDRESS	: KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON ROAD, AMH	BALA CANTT	REPORTING DATE	: 26/Jan/2025 10:08AM
Test Name		Value	Unit	Biological Reference interval
			LLNESS PANEL: 1.5 OOD COUNT (CBC)	5
RED BLOOD CELLS	S (RBCS) COUNT AND INDICES			
HAEMOGLOBIN (H	B)	11 <sup>L</sup>	gm/dL	12.0 - 16.0
by CALORIMETRIC RED BLOOD CELL ( by HYDRO DYNAMIC F	(RBC) COUNT FOCUSING, ELECTRICAL IMPEDENCE	3.58	Millions/	/cmm 3.50 - 5.00
PACKED CELL VOL		33.4 <sup>L</sup>	%	37.0 - 50.0
	AR VOLUME (MCV) Automated hematology analyzer	93.3	fL	80.0 - 100.0
	AR HAEMOGLOBIN (MCH)	30.8	pg	27.0 - 34.0
MEAN CORPUSCUL by CALCULATED BY A	AR HEMOGLOBIN CONC. (MCHC)	33	g/dL	32.0 - 36.0
	UTION WIDTH (RDW-CV) AUTOMATED HEMATOLOGY ANALYZER	15.7	%	11.00 - 16.00
	UTION WIDTH (RDW-SD) AUTOMATED HEMATOLOGY ANALYZER	54.4	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED		26.06	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INI		41.01	RATIO	BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0
<b>WHITE BLOOD CE</b> TOTAL LEUCOCYTI	E COUNT (TLC)	6410	/cmm	4000 - 11000
NUCLEATED RED H	Y BY SF CUBE & MICROSCOPY BLOOD CELLS (nRBCS)	NIL		0.00 - 20.00
NUCLEATED RED H	rt hematology analyzer BLOOD CELLS (nRBCS) % automated hematology analyzer	NIL	%	< 10 %





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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





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

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

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Test Name	Value	Unit	<b>Biological Reference interval</b>
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	68	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	27	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	4	%	2 - 12
BASOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	%	0 - 1
ABSOLUTE LEUKOCYTES (WBC) COUNT			
ABSOLUTE NEUTROPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	4359	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1731	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	64	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	256	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110
PLATELETS AND OTHER PLATELET PREDICTIVE	E MARKERS.		
PLATELET COUNT (PLT) by hydro dynamic focusing, electrical impedence	150000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by hydro dynamic focusing, electrical impedence	0.19	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by hydro dynamic focusing, electrical impedence	13 <sup>H</sup>	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by hydro dynamic focusing, electrical impedence	72000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	47.8 <sup>H</sup>	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by hydro dynamic focusing, electrical impedence	16.4	%	15.0 - 17.0
ADVICE	KINDLY CORRE	ELATE CLINICALLY	





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	Dr. Vinay Chopr MD (Pathology & Mic Chairman & Consulta	robiology) ME	m <b>Chopra</b> D (Pathology) ht Pathologist
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Test Name		Value Unit	Biological Reference interval

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

RECHECKED.



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TEST PERFORMED AT KOS DIAGNOSTIC LAB. AMBALA CANTT



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BARCODE NO.	: 01524443		CTION DATE	: 26/Jan/2025 09:34AM	
CLIENT CODE.	: KOS DIAGNOSTIC LAB		RTING DATE	: 26/Jan/2025 01:52PM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,				
Test Name		Value	Unit	Biological Reference inte	erval
WHOLE BLOOD	EMOGLOBIN (HbA1c): RMANCE LIQUID CHROMATOGRAPHY)	4.8	%	4.0 - 6.4	
	GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY)	91.06	mg/dL	60.00 - 140.00	
		DIABETES ASSOCIATION (			
	REFERENCE GROUP		ATED HEMOGLOGIB (H	BAIC) in %	
Non di	abetic Adults >= 18 years		<5.7		
A	t Risk (Prediabetes)		5.7 - 6.4		
D	iagnosing Diabetes		>= 6.5		
		Goals of Ther	Age > 19 Years	7.0	
	Therapeutic goals for diversic control		apy:	< 7.0	
Therapout			stad		
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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		x Microbiology) onsultant Pathologist		n Chopra (Pathology) : Pathologist
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LIENT ADDRESS	: 6349/1, NICHOLSON ROAI	D, AMBALA CANTT		
by RED CELL AGGREC NTERPRETATION: . ESR is a non-specifi mmune disease, but 2. An ESR can be affer as C-reactive protein	DIMENTATION RATE (ESR) GATION BY CAPILLARY PHOTOME ic test because an elevated res does not tell the health practil cted by other conditions besid	sult often indicates the p tioner exactly where the es inflammation. For this	mm/1st resence of inflammat inflammation is in th reason, the ESR is ty	hr 0 - 20 ion associated with infection, cancer and auto





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		chopra v & Microbiology) onsultant Pathologist	Dr. Yugam MD CEO & Consultant	(Pathology)
NAME	: Mrs. SONALI			
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAL	D, AMBALA CANTT		
Test Name		Value	Unit	<b>Biological Reference interval</b>
	CLIN	ICAL CHEMISTRY/	BIOCHEMIST	RY
		GLUCOSE FAST	ING (F)	

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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CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPO	DRTING DATE	: 26/Jan/2025 11:43AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROA	D, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		LIPID PROFIL	F · BASIC	
	TAL. CEDIM			
CHOLESTEROL TO by CHOLESTEROL OX		162.51	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: S by GLYCEROL PHOSE	ERUM PHATE OXIDASE (ENZYMATIC)	88.52	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	57.27	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROI by CALCULATED, SPE		87.54	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 CHOLES by CALCULATED, SPE		105.24	mg/dL	VERT HIGH: > OK = 190.0 OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTER(		17.7	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SER by CALCULATED, SPE	RUM	413.54	mg/dL	350.00 - 700.00
CHOLESTEROL/HE by CALCULATED, SPE	DL RATIO: SERUM	2.84	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0



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Test Name		Value	Unit	<b>Biological Reference interval</b>
LDL/HDL RATIO: S by CALCULATED, SPE		1.53	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	1.55 <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.

 Low HDL levels are associated with increased risk for Atherosclerotic Cardiovascular disease (ASCVD) due to insufficient HDL being available to participate in reverse cholesterol transport, the process by which cholesterol is eliminated from peripheral tissues.
 NLA-2014 identifies Non HDL Cholesterol (an indicator of all atherogeniclipoproteins such as LDL, VLDL, IDL, Lpa, Chylomicron remnants) along with LDL-cholesterol as co- primary target for cholesterol lowering therapy. Note that major risk factors can modify treatment goals for LDL & Non HDL

5. Additional testing for Apolipoprotein B, hsCRP,Lp(a) & LP-PLA2 should be considered among patients with moderate risk for ASCVD for risk refinement





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Test Name		Value	Unit	Biological Reference interval
restranc		Vinue	Unit	biological actor ence microar
	LIVER	FUNCTION	TEST (COMPLETE)	
BILIRUBIN TOTAL: by DIAZOTIZATION, SF		1.12	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
	(CONJUGATED): SERUM	0.26	mg/dL	0.00 - 0.40
	CT (UNCONJUGATED): SERUM	0.86	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PY	RIDOXAL PHOSPHATE	26	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PY		26	U/L	0.00 - 49.00
AST/ALT RATIO: SI	ERUM	1	RATIO	0.00 - 46.00
ALKALINE PHOSPH by Para Nitropheny PROPANOL	IATASE: SERUM YL PHOSPHATASE BY AMINO METHYL	92.08	U/L	40.0 - 130.0
GAMMA GLUTAMY	L TRANSFERASE (GGT): SERUM	11.08	U/L	0.00 - 55.0
TOTAL PROTEINS: by BIURET, SPECTRON		7.67	gm/dL	6.20 - 8.00
ALBUMIN: SERUM		3.93	gm/dL	3.50 - 5.50
GLOBULIN: SERUM		3.74 <sup>H</sup>	gm/dL	2.30 - 3.50
A : G RATIO: SERUN		1.05	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
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)





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INTERPRETATION





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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).

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



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Test Name		Value	Unit	<b>Biological Reference interv</b>
	KIDNE	Y FUNCTION	TEST (COMPLETE)	
UREA: SERUM		22.08	mg/dL	10.00 - 50.00
•	MATE DEHYDROGENASE (GLDH)	1.07	. / 11	0.40, 1.00
CREATININE: SERU by ENZYMATIC, SPEC		1.07	mg/dL	0.40 - 1.20
	ROGEN (BUN): SERUM	10.32	mg/dL	7.0 - 25.0
by CALCULATED, SPE BLOOD UREA NITE	ECTROPHOTOMETRY ROGEN (BUN)/CREATININE	9.64 <sup>L</sup>	RATIO	10.0 - 20.0
RATIO: SERUM	OULIN (DOIN)/ OILLATININL	9.64-	RATIO	10.0 - 20.0
by CALCULATED, SPE		20.04	DATIO	
UREA/CREATININ by CALCULATED, SPE		20.64	RATIO	
URIC ACID: SERUM		6.05	mg/dL	2.50 - 6.80
by URICASE - OXIDAS CALCIUM: SERUM	SE PEROXIDASE	9.28	mg/dL	8.50 - 10.60
by ARSENAZO III, SPE	ECTROPHOTOMETRY		Ű	0.00 10.00
PHOSPHOROUS: SE	ERUM DATE, SPECTROPHOTOMETRY	3.09	mg/dL	2.30 - 4.70
ELECTROLYTES	DATE, SI LETROI HOTOMETRI			
SODIUM: SERUM		142.5	mmol/L	135.0 - 150.0
by ISE (ION SELECTIV				
POTASSIUM: SERU by ISE (ION SELECTIV		4.96	mmol/L	3.50 - 5.00
CHLORIDE: SERUM	1	106.88	mmol/L	90.0 - 110.0
by ISE (ION SELECTIV	'E ELECTRODE) <b>IERULAR FILTERATION RATE</b>			
	ERULAR FILTERATION RATE	69.9		
(eGFR): SERUM	IERULAR FILTERATION RATE	09.9		
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.

3. GI haemorrhage.



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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT

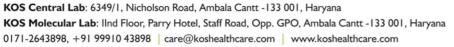




	٨	Dr. Vinay Chopr 1D (Pathology & Micr Chairman & Consultar	obiology)			thology)			
NAME	: Mrs. SONALI								
AGE/ GENDER	: 34 YRS/FEMA	LE		PATIENT ID		: 1735597			
COLLECTED BY	:			REG. NO./LAB NO.		: 0125012600	)12		
REFERRED BY				<b>REGISTRATION DA</b>		: 26/Jan/2025			
BARCODE NO.	:01524443			COLLECTION DAT		: 26/Jan/2025 (			
CLIENT CODE.	: KOS DIAGNOS			REPORTING DATE	L	: 26/Jan/2025	11:43AM		
CLIENT ADDRESS	: 6349/1, NICE	IOLSON ROAD, AMB	ALA CANTT						
Test Name			Value	Uni	it	Biolo	gical Ref	ference i	nterval
<ol> <li>Reduced muscle m</li> <li>Certain drugs (e.g.</li> <li>INCREASED RATIO (&gt;2</li> <li>Postrenal azotemia</li> </ol>	tetracycline, gluc 0:1) WITH ELEVA (BUN rises dispr	reatinine productior cocorticoids) <b>TED CREATININE LEV</b> oportionately more	ELS:	ne) (e.g. obstructive	e uropathy	).			
<ol> <li>Reduced muscle m</li> <li>Certain drugs (e.g.,</li> <li>INCREASED RATIO (&gt;2</li> <li>Postrenal azotemia</li> <li>Prerenal azotemia</li> <li>DECREASED RATIO (&lt;</li> <li>Acute tubular necr</li> <li>Low protein diet ar</li> <li>Severe liver diseas</li> <li>Other causes of de</li> <li>Repeated dialysis (</li> <li>SIADH (syndrome of</li> <li>Pregnancy.</li> <li>DECREASED RATIO (</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>Muscular patients</li> <li>Mappropiate RATIO</li> <li>Diabetic ketoacido</li> <li>Should produce an in</li> <li>Cephalosporin their</li> <li>ESTIMATED GLOMERI</li> <li>CKD STAGE</li> </ol>	ass (subnormal c tetracycline, gluc 0:1) WITH ELEVA (BUN rises dispr superimposed or 0:1) WITH DECRE osis. Id starvation. 2. creased urea syn urea rather than monemias (urea of inappropiate al 0:1) WITH INCRE. py (accelerates c eleases muscle c who develop ren : sis (acetoacetate creased BUN/cre apy (interferes w ULAR FILTERATION	reatinine production cocorticoids) <b>TED CREATININE LEV</b> oportionately more in renal disease. <b>CASED BUN :</b> thesis. creatinine diffuses of is virtually absent in ntidiuretic harmone) <b>ASED CREATININE:</b> onversion of creatine reatinine). al failure. e causes false increase atinine ratio). vith creatinine measu. <b>I RATE:</b> <b>DESCRIPTION</b>	ELS: Than creatinin but of extract blood). due to tubul e to creatinin e in creatinin rement).	ellular fluid). lar secretion of urea ne). ne with certain metl	i. hodologie ASSOC	s,resulting in n		io when d	ehydrati
A. Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Prerenal azotemia Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet an Severe liver diseas Other causes of de Repeated dialysis ( Niherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (< Nescular patients NappROPIATE RATIO Diabetic ketoacido should produce an in Cephalosporin their ESTIMATED GLOMERI CKD STAGE G1	ass (subnormal c tetracycline, gluc 0:1) WITH ELEVA (BUN rises dispr superimposed or 0:1) WITH DECRE osis. Id starvation. 2. creased urea syn urea rather than monemias (urea of inappropiate al 0:1) WITH INCRE. py (accelerates c eleases muscle c who develop ren : sis (acetoacetate creased BUN/cre apy (interferes w ULAR FILTERATION Norr	reatinine production cocorticoids) <b>TED CREATININE LEV</b> oportionately more in renal disease. <b>CASED BUN :</b> thesis. creatinine diffuses of is virtually absent in ntidiuretic harmone) <b>ASED CREATININE:</b> onversion of creatine reatinine). al failure. causes false increase atinine ratio). vith creatinine measu. <b>I RATE:</b> <b>DESCRIPTION</b> nal kidney function	ELS: Than creatinin but of extract blood). due to tubul e to creatinin e in creatinin rement).	ellular fluid). lar secretion of urea ne). ne with certain metl nL/min/1.73m2) >90	n. hodologie ASSOC	s,resulting in n <b>IATED FINDING</b> p proteinuria	S	io when d	ehydrati
A Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Prerenal azotemia Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet an Severe liver diseas Other causes of de Repeated dialysis ( Niherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (< Nescular patients Muscular patients Muscular patients MappROPIATE RATIO Diabetic ketoacido should produce an in Cephalosporin their ESTIMATED GLOMERI OKD STAGE	ass (subnormal c tetracycline, gluc 0:1) WITH ELEVA (BUN rises dispr superimposed or 0:1) WITH DECRE osis. Id starvation. 2. creased urea syn urea rather than monemias (urea of inappropiate al 0:1) WITH INCRE. py (accelerates c eleases muscle c who develop ren : sis (acetoacetate creased BUN/cre apy (interferes w ULAR FILTERATION Norr Kic	reatinine production cocorticoids) <b>TED CREATININE LEV</b> oportionately more in renal disease. <b>CASED BUN :</b> thesis. creatinine diffuses of is virtually absent in ntidiuretic harmone) <b>ASED CREATININE:</b> onversion of creating reatinine). al failure. causes false increase atinine ratio). vith creatinine measu. <b>J RATE:</b> <b>DESCRIPTION</b> nal kidney function iney damage with	ELS: Than creatinin but of extract blood). due to tubul e to creatinin e in creatinin rement).	ellular fluid). lar secretion of urea ne). ne with certain metl	hodologie ASSOC	s,resulting in n	S	io when d	ehydrati
A. Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Prerenal azotemia Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet an Severe liver diseas Other causes of de Repeated dialysis ( Repeated dialysis ( NIADH (syndrome of Pregnancy. DECREASED RATIO (< Neclar patients NAPPROPIATE RATIO Diabetic ketoacido should produce an in Cephalosporin there ESTIMATED GLOMERI CKD STAGE G1	ass (subnormal c tetracycline, gluc 0:1) WITH ELEVA (BUN rises dispr superimposed or 0:1) WITH DECRE osis. Id starvation. 2. creased urea syn urea rather than monemias (urea of inappropiate an 0:1) WITH INCRE. py (accelerates c eleases muscle c who develop ren : sis (acetoacetate creased BUN/cre apy (interferes w ILAR FILTERATION Norr Norr	reatinine production cocorticoids) <b>TED CREATININE LEV</b> oportionately more in renal disease. <b>CASED BUN :</b> thesis. creatinine diffuses of is virtually absent in ntidiuretic harmone) <b>ASED CREATININE:</b> onversion of creatine reatinine). al failure. causes false increase atinine ratio). vith creatinine measu. <b>I RATE:</b> <b>DESCRIPTION</b> nal kidney function	ELS: Than creatinin but of extract blood). due to tubul e to creatinin e in creatinin rement).	ellular fluid). lar secretion of urea ne). ne with certain metl nL/min/1.73m2) >90	hodologie ASSOC	s,resulting in n <b>HATED FINDING</b> p proteinuria ince of Protein	S	io when d	ehydrati
B. Reduced muscle m     Certain drugs (e.g.     INCREASED RATIO (>2     I. Postrenal azotemia     DECREASED RATIO (<         1. Acute tubular necr     Low protein diet ar     Severe liver diseas     Other causes of de     Severe liver diseas     Nother causes of de     Severe liver diseas     Severe liver diseas	ass (subnormal c tetracycline, gluc 0:1) WITH ELEVA (BUN rises dispr superimposed or 0:1) WITH DECRE osis. Id starvation. 2: creased urea syn urea rather than monemias (urea f inappropiate an 0:1) WITH INCRE. py (accelerates c eleases muscle c who develop ren : sis (acetoacetate creased BUN/cre apy (interferes w UAR FILTERATION Norr Kic no Mil	reatinine production cocorticoids) <b>TED CREATININE LEV</b> oportionately more in renal disease. <b>CASED BUN :</b> thesis. creatinine diffuses of is virtually absent in htidiuretic harmone) <b>ASED CREATININE:</b> onversion of creating reatinine). al failure. causes false increas atinine ratio). vith creatinine measu <b>I RATE:</b> <b>DESCRIPTION</b> mal kidney function Iney damage with rmal or high GFR d decrease in GFR rate decrease in GFR	ELS: than creatining but of extraction blood). due to tubul e to creatining e in creatining rement). GFR (m	ellular fluid). lar secretion of urea ne). ne with certain metl <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>second</u> <u>s</u>	hodologie ASSOC	s,resulting in n <b>HATED FINDING</b> p proteinuria ince of Protein	S	io when d	ehydrati
A. Reduced muscle m     Certain drugs (e.g.     NCREASED RATIO (>2     Prerenal azotemia     DECREASED RATIO (<         Acute tubular necr     Low protein diet ar     Severe liver diseas     Other causes of de     Severe liver diseas     Other causes of de     Severe liver diseas     A. Other causes of de     Severe liver diseas     Nuherited hyperam     SIADH (syndrome d     Severe liver diseas     Nuscular patients     NAPPROPIATE RATIO     Should produce an in     Cephalosporin the     STIMATED GLOMERI     G1     G2     G3a	ass (subnormal c tetracycline, gluc 0:1) WITH ELEVA (BUN rises dispr superimposed or 0:1) WITH DECRE osis. Id starvation. 2: creased urea syn urea rather than monemias (urea of inappropiate an 0:1) WITH INCRE. py (accelerates c eleases muscle c who develop ren : sis (acetoacetate creased BUN/cre apy (interferes w UAR FILTERATION Norr Norr Kic no Mode Seve	reatinine production cocorticoids) <b>TED CREATININE LEV</b> oportionately more in renal disease. <b>CASED BUN :</b> thesis. creatinine diffuses of is virtually absent in htidiuretic harmone) <b>ASED CREATININE:</b> onversion of creating reatinine). al failure. causes false increase atinine ratio). vith creatinine measu. <b>I RATE:</b> <b>DESCRIPTION</b> mal kidney function Iney damage with rmal or high GFR d decrease in GFR	ELS: than creatining but of extraction blood). due to tubul e to creatining e in creatining rement). GFR (m	ellular fluid). lar secretion of urea ne). ne with certain meth <u>&gt;90 &gt;90</u> >90 60 -89	hodologie ASSOC	s,resulting in n <b>HATED FINDING</b> p proteinuria ince of Protein	S	io when d	ehydrati



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









	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbiology Chairman & Consultant Patholo		(Pathology)
NAME	: Mrs. SONALI		
AGE/ GENDER	: 34 YRS/FEMALE	PATIENT ID	: 1735597
COLLECTED BY	:	<b>REG. NO./LAB NO.</b>	: 012501260012
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 26/Jan/2025 09:33 AM
BARCODE NO.	: 01524443	<b>COLLECTION DATE</b>	: 26/Jan/2025 09:34AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	<b>REPORTING DATE</b>	: 26/Jan/2025 11:43AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CAN	TT	
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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	Dr. Vinay Chopi MD (Pathology & Mic Chairman & Consulta	crobiology)		
NAME	: Mrs. SONALI			
AGE/ GENDER	: 34 YRS/FEMALE	PA	TIENT ID	: 1735597
<b>COLLECTED BY</b>	:	RE	G. NO./LAB NO.	: 012501260012
<b>REFERRED BY</b>	:	RE	GISTRATION DATE	: 26/Jan/2025 09:33 AM
<b>BARCODE NO.</b> : 01524443		CO	LLECTION DATE	: 26/Jan/2025 09:34AM
<b>CLIENT CODE.</b> : KOS DIAGNOSTIC LAB		RE	PORTING DATE	: 26/Jan/2025 11:43AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMI	BALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		IRON PR	OFILE	
IRON: SERUM by FERROZINE, SPECT	ROPHOTOMETRY	96.5	μg/dL	37.0 - 145.0
UNSATURATED IRC :SERUM by FERROZINE, SPECT	ON BINDING CAPACITY (UIBC)	229.3	μg/dL	150.0 - 336.0
•	NG CAPACITY (TIBC)	325.8	µg/dL	230 - 430
%TRANSFERRIN SA	TURATION: SERUM CTROPHOTOMETERY (FERENE)	29.62	%	15.0 - 50.0
TRANSFERRIN: SEF		231.32	mg/dL	200.0 - 350.0

by SPECTROPHOTOMETERY (FERENE)

VARIABLES	<u>INTERPRETATION:-</u>	
	VARIABLES	1

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
IDON.			

### IRON:

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.
 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):** 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 PERFORMED AT KOS DIAGNOSTIC LAB. AMBALA CANTT





		hopra & Microbiology) onsultant Patholog	M	m Chopra D (Pathology) nt Pathologist
NAME	: Mrs. SONALI			
AGE/ GENDER	: 34 YRS/FEMALE		PATIENT ID	: 1735597
COLLECTED BY	:		REG. NO./LAB NO.	: 012501260012
<b>REFERRED BY</b>	:		<b>REGISTRATION DATE</b>	: 26/Jan/2025 09:33 AM
BARCODE NO.	:01524443		COLLECTION DATE	: 26/Jan/2025 09:34AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 26/Jan/2025 11:30AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD	), AMBALA CANT	Т	
Test Name		Value	Unit	<b>Biological Reference interval</b>
TRIIODOTHYRONII	NE (T3): SERUM	1.1	<b>CTION TEST: TOTAI</b> ng/mL	
by CMIA (CHEMILUMIN THYROXINE (T4): S	IESCENT MICROPARTICLE IMMUNO SERUM	ASSAY) 10.32	μgm/d	L 4.87 - 12.60
by CMIA (CHEMILUMIN	IESCENT MICROPARTICLE IMMUNO	ASSAY)	MB) ~	
	TING HORMONE (TSH): SEF		µIU/m	L 0.35 - 5.50
3rd GENERATION, ULT	IESCENT MICROPARTICLE IMMUNO RASENSITIVE	400AY)		
INTERPRETATION:				
day has influence on the triiodothyronine (T3).Fai	measured serum TSH concentrations.	TSH stimulates the p	production and secretion of the	<i>ppm. The variation is of the order of 50%.Hence time of t</i> 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 Lo	w Normal	Normal or Low Normal	High
Primary Hyperthyroidis	sm: Increase	d	Increased	Reduced (at times undetectable)
		1 1 1		

#### LIMITATIONS:-

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.

Normal or High Normal

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.

TRIIODOTH	YRONINE (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	

Normal or High Normal





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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT





	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbio Chairman & Consultant Pat	G, /	(Pathology)
NAME	: Mrs. SONALI		
AGE/ GENDER	: 34 YRS/FEMALE	PATIENT ID	: 1735597
COLLECTED BY	:	<b>REG. NO./LAB NO.</b>	: 012501260012
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 26/Jan/2025 09:33 AM
BARCODE NO.	: 01524443	<b>COLLECTION DATE</b>	: 26/Jan/2025 09:34AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	<b>REPORTING DATE</b>	: 26/Jan/2025 11:30AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA	CANTT	

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 LI	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





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





TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



KOS Diagnostic Lab (A Unit of KOS Healthcare)

		/ & Microbiology) onsultant Pathologist	M	am Chopra 1D (Pathology) ant Pathologist		
JAME	: Mrs. SONALI					
AGE/ GENDER	: 34 YRS/FEMALE		PATIENT ID	: 1735597		
COLLECTED BY	:		REG. NO./LAB NO.	: 012501260012		
REFERRED BY		REGISTRATION COLLECTION DA				
	: 01524443					
				: 26/Jan/2025 09:34AM		
	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 26/Jan/2025 12:51PM		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROA	D, AMBALA CANTT				
Test Name		Value	Unit	Biological Reference interval		
WITAMIN D (25 HYDI	<b>VII</b> ROXY VITAMIN D3): SERU	TAMIN D/25 HY	AMINS YDROXY VITAMIN ng/mL			
INTERPRETATION:		M <b>16.207<sup>L</sup></b>	ng/ InL	INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0		
DEFICIE	INT.	< 20		ng/mL		
INSUFFIC		21 - 29		ng/mL		
PREFFERED		30 - 100		ng/mL		
conversion of 7- dihvdr 2.25-OHVitamin D rep tissue and tightly boun 3. Vitamin D plays a prir bhosphate reabsorption 4. Severe deficiency ma <b>DECREASED:</b> 1. Lack of sunshine expo 2. Inadequate intake, m 3. Depressed Hepatic Vi 4. Secondary to advance 5. Osteoporosis and Sec 5. Enzyme Inducing drug <b>INCREASED:</b> 1. Hypervitaminosis D is severe hypercalcemia a <b>CAUTION</b> : Replacement hypervitaminosis D	s are derived from dietary e ocholecalciferol to Vitamin resents the main body reser d by a transport protein wh mary role in the maintenand n, skeletal calcium depositic y lead to failure to mineraliz osure. alabsorption (celiac disease tamin D 25- hydroxylase act d Liver disease ondary Hyperparathroidism gs: anti-epileptic drugs like p s Rare, and is seen only afte nd hyperphophatemia. therapy in deficient individ dividuals as compare to white	D3 in the skin upon voir and transport fo- ile in circulation. se of calcium homeo- on, calcium mobiliza ze newly formed ost e) ivity (Mild to Moderate ohenytoin, phenobal r prolonged exposur uals must be monito	plants, Vitamin D2), or ch Ultraviolet exposure. form of Vitamin D and tran ostatis. It promotes calci- tion, mainly regulated by eoid in bone, resulting in deficiency) rbital and carbamazepine re to extremely high dose ored by periodic assessm	ng/mL cholecalciferol (from animals, Vitamin D3), or by ansport form of Vitamin D, being stored in adipo jum absorption, renal calcium absorption and by parathyroid harmone (PTH). n rickets in children and osteomalacia in adults the, that increases Vitamin D metabolism. ses of Vitamin D. When it occurs, it can result in hent of Vitamin D levels in order to prevent efficiency due to excess of melanin pigment which		





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		inay Chopra athology & Microbiology) an & Consultant Pathologist	Dr. Yugam MD CEO & Consultant	(Pathology)		
AME	: Mrs. SONALI					
GE/ GENDER	: 34 YRS/FEMALE	PAT	IENT ID	: 1735597		
OLLECTED BY	:	REG	NO./LAB NO.	: 012501260012		
EFERRED BY		REG	ISTRATION DATE	: 26/Jan/2025 09:33 AM		
ARCODE NO.	: 01524443		LECTION DATE	: 26/Jan/2025 09:33 AM		
LIENT CODE.	: KOS DIAGNOSTIC L		ORTING DATE			
			UKTING DATE	: 26/Jan/2025 11:43AM		
LIENT ADDRESS	: 6349/1, NICHOLSO	N ROAD, AMBALA CANTT				
Fest Name		Value	Unit	<b>Biological Reference interval</b>		
by CMIA (CHEMILUMIN NTERPRETATION:-	BALAMIN: SERUM	VITAMIN B12/C 114 <sup>L</sup>	pg/mL	190.0 - 890.0		
by CMIA (CHEMILUMIN NTERPRETATION:-	IESCENT MICROPARTICLE	114 <sup>L</sup>		190.0 - 890.0		
by CMIA (CHEMILUMIN NTERPRETATION:- INCREAS	IESCENT MICROPARTICLE	IMMUNOASSAY)				
by CMIA (CHEMILUMIN NTERPRETATION:- INCREA 1.Ingestion of Vitar	NESCENT MICROPARTICLE SED VITAMIN B12 nin C	IMMUNOASSAY)	pg/mL	I B12		
by CMIA (CHEMILUMIN <u>VTERPRETATION:-</u> INCREA: 1.Ingestion of Vitar 2.Ingestion of Estro	IESCENT MICROPARTICLE SED VITAMIN B12 nin C gen	III4 <sup>L</sup> IMMUNOASSAY)  114 <sup>L</sup> 1.Pregnancy 2.DRUGS:Asp	pg/mL DECREASED VITAMIN irin, Anti-convulsants,	I B12		
by CMIA (CHEMILUMIN <u>VTERPRETATION:-</u> INCREA: 1.Ingestion of Vitar 2.Ingestion of Estro 3.Ingestion of Vitan	IESCENT MICROPARTICLE SED VITAMIN B12 nin C gen nin A	IMMUNOASSAY)	pg/mL DECREASED VITAMIN irin, Anti-convulsants, stion	I B12		
by CMIA (CHEMILUMIN <u>NTERPRETATION:-</u> INCREA: 1.Ingestion of Vitar 2.Ingestion of Estro 3.Ingestion of Vitan 4.Hepatocellular in	IESCENT MICROPARTICLE SED VITAMIN B12 nin C gen nin A jury	III4 <sup>L</sup> IMMUNOASSAY)  114 <sup>L</sup> 1.Pregnancy 2.DRUGS:Asp 3.Ethanol Iges	pg/mL DECREASED VITAMIN irin, Anti-convulsants, stion ive Harmones	I B12		
by CMIA (CHEMILUMIN <u>NTERPRETATION:-</u> INCREA: 1.Ingestion of Vitar 2.Ingestion of Vitar 3.Ingestion of Vitar 4.Hepatocellular in 5.Myeloproliferativ 6.Uremia .Vitamin B12 (coba	SED VITAMIN B12 SED VITAMIN B12 gen nin C gen nin A jury re disorder lamin) is necessary for	III4 <sup>L</sup> IMMUNOASSAY)  114 <sup>L</sup> 1.Pregnancy 2.DRUGS:Asp 3.Ethanol Iges 4. Contracept	pg/mL DECREASED VITAMIN irin, Anti-convulsants, stion ive Harmones ysis yeloma onal function.	IB12 Colchicine		





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







	<b>Dr. Vinay Cho</b> MD (Pathology & Chairman & Cons				
NAME	: Mrs. SONALI				
AGE/ GENDER	: 34 YRS/FEMALE	PATIEN	NT ID	: 1735597	
COLLECTED BY	:	REG. NO	D./LAB NO.	: 012501260012	
<b>REFERRED BY</b>	:	REGIST	<b>TRATION DATE</b>	: 26/Jan/2025 09:33 AM	
BARCODE NO.	: 01524443	COLLEG	CTION DATE	: 26/Jan/2025 09:34AM	
CLIENT CODE.	: KOS DIAGNOSTIC LAB	<b>REPORTING DATE</b>		: 26/Jan/2025 11:02AM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	AMBALA CANTT			
Test Name		Value	Unit	Biological Reference interval	
		CLINICAL PATH	IOLOGY		
	URINE RO	UTINE & MICROSC	OPIC EXAMINA	ATION	
PHYSICAL EXAMINA	TION				
QUANTITY RECIEVE		10	ml		
COLOUR		AMBER YELLOW	,	PALE YELLOW	
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY TRANSPARANCY by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY		HAZY		CLEAR	
SPECIFIC GRAVITY	NCE SPECTROPHOTOMETRY	1.01		1.002 - 1.030	
CHEMICAL EXAMIN					
REACTION		ACIDIC			
PROTEIN	NCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)	
	NCE SPECTROPHOTOMETRY				
SUGAR by DIP STICK/REFLECTA	NCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)	
pH		<=5.0		5.0 - 7.5	
BILIRUBIN	NCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)	
	NCE SPECTROPHOTOMETRY	C .			
NITRITE by DIP STICK/REFLECTA	NCE SPECTROPHOTOMETRY.	Negative		NEGATIVE (-ve)	
UROBILINOGEN	NCE SPECTROPHOTOMETRY	Normal	EU/dL	0.2 - 1.0	
KETONE BODIES		Negative		NEGATIVE (-ve)	
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY BLOOD		Negative		NEGATIVE (-ve)	
ASCORBIC ACID by DIP STICK/REFLECTA	NCE SPECTROPHOTOMETRY	NEGATIVE (-ve)		NEGATIVE (-ve)	
MICROSCOPIC EXAMINED BLOOD CELLS (		NEGATIVE (-ve)	/HPF	0 - 3	



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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. SONALI			
AGE/ GENDER	: 34 YRS/FEMALE	PA	<b>FIENT ID</b>	: 1735597
COLLECTED BY	:	REG	G. NO./LAB NO.	: 012501260012
<b>REFERRED BY</b>	:	REG	GISTRATION DATE	: 26/Jan/2025 09:33 AM
BARCODE NO.	: 01524443	COL	LECTION DATE	: 26/Jan/2025 09:34AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REI	PORTING DATE	: 26/Jan/2025 11:02AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
by MICROSCOPY ON (	CENTRIFUGED URINARY SEDIMENT			
PUS CELLS by MICROSCOPY ON (	CENTRIFUGED URINARY SEDIMENT	2-4	/HPF	0 - 5
EPITHELIAL CELLS	S	2-3	/HPF	ABSENT

EPITHELIAL CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	2-3	/HPF	ABSENT
CRYSTALS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)
CASTS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)
BACTERIA by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)
OTHERS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)		NEGATIVE (-ve)
TRICHOMONAS VAGINALIS (PROTOZOA) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	ABSENT		ABSENT

\*\* End Of Report



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

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