



	Dr. Vinay Chopra MD (Pathology & Microbi Chairman & Consultant P			(Pathology)
NAME :	Mrs. SHUBHANGI			
AGE/ GENDER :	28 YRS/FEMALE		PATIENT ID	: 1814850
COLLECTED BY :			REG. NO./LAB NO.	: 012504020002
REFERRED BY			REGISTRATION DATE	: 02/Apr/2025 07:46 AM
BARCODE NO.	01528189		COLLECTION DATE	: 02/Apr/2025 07:50AM
CLIENT CODE.	KOS DIAGNOSTIC LAB		REPORTING DATE	: 02/Apr/2025 09:04AM
CLIENT ADDRESS :	6349/1, NICHOLSON ROAD, AMBALA	A CANTT		
Test Name	V	alue	Unit	Biological Reference interval
			LLNESS PANEL: 1 DOD COUNT (CBC)	.5
RED BLOOD CELLS	(RBCS) COUNT AND INDICES		(
HAEMOGLOBIN (HB) by CALORIMETRIC		10.9 ^L	gm/dL	12.0 - 16.0
RED BLOOD CELL (R	BC) COUNT CUSING, ELECTRICAL IMPEDENCE	3.98	Millions	2.50 - 5.00
PACKED CELL VOLU	ME (PCV) OMATED HEMATOLOGY ANALYZER	33.5 ^L	%	37.0 - 50.0
MEAN CORPUSCULA by CALCULATED BY AUT	R VOLUME (MCV) OMATED HEMATOLOGY ANALYZER	84.2	fL	80.0 - 100.0
	R HAEMOGLOBIN (MCH) OMATED HEMATOLOGY ANALYZER	27.3	pg	27.0 - 34.0
	AR HEMOGLOBIN CONC. (MCHC) TOMATED HEMATOLOGY ANALYZER	32.4	g/dL	32.0 - 36.0
	UTION WIDTH (RDW-CV) TOMATED HEMATOLOGY ANALYZER	16.9 ^H	%	11.00 - 16.00
	JTION WIDTH (RDW-SD) TOMATED HEMATOLOGY ANALYZER	53.1	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED		21.16	RATIO	BETA THALASSEMIA TRAIT: - 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDI by CALCULATED	EX	109.92	RATIO	BETA THALASSEMIA TRAIT: <= 65.0 IRON DEFICIENCY ANEMIA: 2
				65.0
WHITE BLOOD CEI		(0.00		1000 11000
FOTAL LEUCOCYTE by FLOW CYTOMETRY B	COUNT (TLC) Y SF CUBE & MICROSCOPY	6980	/cmm	4000 - 11000
	LOOD CELLS (nRBCS)	NIL		0.00 - 20.00
by AUTOMATED 6 PART	TILIVIATOLOGT ANALTZER			





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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





Dr. Vinay Chopra Dr. Yugam Chopra MD (Pathology & Microbiology) MD (Pathology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** NAME : Mrs. SHUBHANGI **AGE/ GENDER** : 28 YRS/FEMALE **PATIENT ID** :1814850 **COLLECTED BY** :012504020002 REG. NO./LAB NO. **REFERRED BY REGISTRATION DATE** :02/Apr/2025 07:46 AM **BARCODE NO.** :01528189 **COLLECTION DATE** :02/Apr/2025 07:50AM CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE** :02/Apr/2025 09:04AM **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Unit **Biological Reference interval** by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER DIFFERENTIAL LEUCOCYTE COUNT (DLC) **NEUTROPHILS** 62 50 - 70 % by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY LYMPHOCYTES 28 % 20 - 40 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY EOSINOPHILS 5 % 1 - 6by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY 5 MONOCYTES % 2 - 12 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY BASOPHILS 0 % 0 - 1by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE LEUKOCYTES (WBC) COUNT ABSOLUTE NEUTROPHIL COUNT 2000 - 7500 4328 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE LYMPHOCYTE COUNT 1954 /cmm 800 - 4900 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE EOSINOPHIL COUNT 349 40 - 440 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE MONOCYTE COUNT 349 /cmm 80 - 880 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE BASOPHIL COUNT 0 0 - 110/cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS. PLATELET COUNT (PLT) 224000 /cmm 150000 - 450000 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELETCRIT (PCT) 0.32 % 0.10 - 0.36 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE MEAN PLATELET VOLUME (MPV) fL. 6.50 - 12.0 14^H by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET LARGE CELL COUNT (P-LCC) /cmm 30000 - 90000 123000^H by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET LARGE CELL RATIO (P-LCR) % 11.0 - 45.0 54.8^H by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET DISTRIBUTION WIDTH (PDW) 16.4 % 15.0 - 17.0



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Test Name	Va	lue Unit	Biological Reference interval

by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD



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CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 02/Apr/2025 12:23PM
CLIENT CODE.	: 6349/1, NICHOLSON ROAD, AN		MELOWING DATE	. <i>ownipi/ wowo iw.woi</i> im
CLIENI ADDRESS	. 0349/1, MCHOLSON KOAD, AN	ADALA CANT I		
Test Name		Value	Unit	Biological Reference interval
WHOLE BLOOD	IAEMOGLOBIN (HbA1c):	4.5	%	4.0 - 6.4
ESTIMATED AVER by HPLC (HIGH PERFO	RMANCE LIQUID CHROMATOGRAPHY) AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY)	82.45	mg/dL	60.00 - 140.00
ESTIMATED AVER by HPLC (HIGH PERFO	AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY)			60.00 - 140.00
ESTIMATED AVER by HPLC (HIGH PERFO INTERPRETATION:	AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN D	IABETES ASSOCIA	TION (ADA):	
ESTIMATED AVER by HPLC (HIGH PERFO INTERPRETATION:	AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN D REFERENCE GROUP	IABETES ASSOCIA		
ESTIMATED AVER by HPLC (HIGH PERFO INTERPRETATION: Non di	AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN D	IABETES ASSOCIA	TION (ADA): /COSYLATED HEMOGLOGIB	
ESTIMATED AVER by HPLC (HIGH PERFO INTERPRETATION: Non di A	AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN D REFERENCE GROUP abetic Adults >= 18 years	IABETES ASSOCIA	TION (ADA): /COSYLATED HEMOGLOGIB <5.7 5.7 - 6.4 >= 6.5	
ESTIMATED AVER by HPLC (HIGH PERFO INTERPRETATION: Non di A	AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN D REFERENCE GROUP abetic Adults >= 18 years tt Risk (Prediabetes)	IABETES ASSOCIA GLY	TION (ADA): /COSYLATED HEMOGLOGIB <5.7 5.7 - 6.4 >= 6.5 Age > 19 Years	(HBAIC) in %
ESTIMATED AVER by HPLC (HIGH PERFO INTERPRETATION: Non di A	AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN D REFERENCE GROUP abetic Adults >= 18 years tt Risk (Prediabetes) biagnosing Diabetes	IABETES ASSOCIA GLY Goals of	TION (ADA): /COSYLATED HEMOGLOGIB <5.7	(HBAIC) in % < 7.0
ESTIMATED AVER by HPLC (HIGH PERFO INTERPRETATION: Non di A	AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN D REFERENCE GROUP abetic Adults >= 18 years tt Risk (Prediabetes)	IABETES ASSOCIA GLY Goals of	TION (ADA): /COSYLATED HEMOGLOGIB <5.7 5.7 - 6.4 >= 6.5 Age > 19 Years	(HBAIC) in %

KOS Diagnostic Lab

(A Unit of KOS Healthcare)

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 appropriate.

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 falsely 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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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



	Dr. Vinay Chop MD (Pathology & Mi Chairman & Consult	icrobiology)	Dr. Yugam MD (CEO & Consultant	(Pathology)
NAME	: Mrs. SHUBHANGI			
AGE/ GENDER	: 28 YRS/FEMALE	PAT	TIENT ID	: 1814850
COLLECTED BY	:	REC	. NO./LAB NO.	: 012504020002
REFERRED BY	:	REC	SISTRATION DATE	: 02/Apr/2025 07:46 AM
BARCODE NO.	: 01528189	COI	LECTION DATE	: 02/Apr/2025 07:50AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REF	ORTING DATE	: 02/Apr/2025 10:06AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	BALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	ERYTHROC	CYTE SEDIME	NTATION RATE ((ESR)
by RED CELL AGGREG INTERPRETATION: 1. ESR is a non-specifimmune disease, but 2. An ESR can be affe as C-reactive protein 3. This test may also systemic lupus erythy CONDITION WITH LO A low ESR can be see (polycythaemia), sigras sickle cells in sickl NOTE: 1. ESR and C - reactive 3. CRP is not affected 4. If the ESR is elevat 5. Women tend to ha 6. Drugs such as dext	does not tell the health practitioner cted by other conditions besides inf be used to monitor disease activity ematosus W ESR n with conditions that inhibit the non ificantly high white blood cell coun e cell anaemia) also lower the ESR. e protein (C-RP) are both markers of s not change as rapidly as does CRP by as many other factors as is ESR, r ed, it is typically a result of two type ve a higher ESR, and menstruation a	r exactly where the lammation. For thi and response to th prmal sedimentation t (leucocytosis), a f inflammation. , either at the star making it a better n es of proteins, glob and pregnancy can	e inflammation is in the s reason, the ESR is typ herapy in both of the ab on of red blood cells, su nd some protein abnor t of inflammation or as narker of inflammation . ulins or fibrinogen. cause temporary elevat	on associated with infection, cancer and auto- body or what is causing it. ically used in conjunction with other test such bove diseases as well as some others, such as och as a high red blood cell count malities. Some changes in red cell shape (such it resolves.





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	M	r. Vinay Cho D (Pathology & I airman & Const		Dr. Yugam MD CEO & Consultant	(Pathology)
NAME	: Mrs. SHUBHA	IGI			
AGE/ GENDER	: 28 YRS/FEMAL	E	P	ATIENT ID	: 1814850
COLLECTED BY	:		F	REG. NO./LAB NO.	: 012504020002
REFERRED BY	:		F	REGISTRATION DATE	: 02/Apr/2025 07:46 AM
BARCODE NO.	:01528189		C	COLLECTION DATE	: 02/Apr/2025 07:50AM
CLIENT CODE.	: KOS DIAGNOST	'IC LAB	F	REPORTING DATE	: 02/Apr/2025 11:26AM
CLIENT ADDRESS	: 6349/1, NICHO	LSON ROAD, A	MBALA CANTT		
			Value	Unit	Biological Reference interval
Test Name					
lest Name		CLINICA	L CHEMIST	FRY/BIOCHEMIS	TRY
l est Name		CLINICA		FRY/BIOCHEMIS FASTING (F)	TRY

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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	Dr. Vinay Cl MD (Pathology Chairman & Co			(Pathology)
NAME : N	Ars. SHUBHANGI			
AGE/ GENDER : 2	8 YRS/FEMALE		PATIENT ID	: 1814850
COLLECTED BY :			REG. NO./LAB NO.	: 012504020002
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	XOS DIAGNOSTIC LAB		REPORTING DATE	: 02/Apr/2025 11:26AM
CLIENT ADDRESS : 6	349/1, NICHOLSON ROAD	, AMBALA CANTI		
Test Name		Value	Unit	Biological Reference interval
		LIPID PRO	FILE : BASIC	
CHOLESTEROL TOTAL by CHOLESTEROL OXIDAS		121.27	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR =
TRIGLYCERIDES: SER by GLYCEROL PHOSPHAT		53.17	mg/dL	240.0 OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROL (I by SELECTIVE INHIBITION	DIRECT): SERUM	49.8	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: S by calculated, spectro		70.84	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.0 BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0
NON HDL CHOLESTER by CALCULATED, SPECTR		71.47	mg/dL	VERY HIGH: > OR = 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 CHOLESTEROL: by CALCULATED, SPECTR		10.63	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUN		305.71 ^L	mg/dL	350.00 - 700.00
by CALCULATED, SPECTR CHOLESTEROL/HDL R by CALCULATED, SPECTR	ATIO: SERUM	2.44	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0
	lt	6	hopra	

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		hopra & Microbiology) onsultant Pathologist	Dr. Yugam MD CEO & Consultant	(Pathology)
NAME	: Mrs. SHUBHANGI			
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAI	D, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
				MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0
LDL/HDL RATIO: S by CALCULATED, SPE		1.42	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/H by CALCULATED, SPE	HDL RATIO: SERUM	1.07 ^L	RATIO	3.00 - 5.00

INTERPRETATION:

1.Measurements in the same patient can show physiological& analytical variations. Three serial samples 1 week apart are recommended for Total Cholesterol, Triglycerides, HDL & LDL Cholesterol. 2. As per NLA-2014 guidelines, all adults above the age of 20 years should be screened for lipid status. Selective screening of children above the age of 2 years with a family history of premature cardiovascular disease or those with at least one parent with high total cholesterol is recommended.

 Cow 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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	Chairman & Consultar	nt Pathologis	t CEO & Consultant	Pathologist
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Test Name		Value	Unit	Biological Reference interval
	LIVER F	UNCTIO	N TEST (COMPLETE	
BILIRUBIN TOTAL		0.67	mg/dL	INFANT: 0.20 - 8.00
•	PECTROPHOTOMETRY			ADULT: 0.00 - 1.20
	T (CONJUGATED): SERUM SPECTROPHOTOMETRY	0.18	mg/dL	0.00 - 0.40
BILIRUBIN INDIRI by CALCULATED, SPE	ECT (UNCONJUGATED): SERUM	0.49	mg/dL	0.10 - 1.00
SGOT/AST: SERUN by IFCC, WITHOUT PY	Л /RIDOXAL PHOSPHATE	11.85	U/L	7.00 - 45.00
SGPT/ALT: SERUN by IFCC, WITHOUT PY	Í /RIDOXAL PHOSPHATE	18.42	U/L	0.00 - 49.00
AST/ALT RATIO: S		0.64	RATIO	0.00 - 46.00
ALKALINE PHOSP		67.41	U/L	40.0 - 130.0
GAMMA GLUTAM by SZASZ, SPECTROF	YL TRANSFERASE (GGT): SERUN Phtometry	1 7.66	U/L	0.00 - 55.0
TOTAL PROTEINS by BIURET, SPECTRO		6.29	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL G		4.21	gm/dL	3.50 - 5.50
GLOBULIN: SERUN by CALCULATED, SPE	M	2.08 ^L	gm/dL	2.30 - 3.50
A : G RATIO: SERU by CALCULATED, SPE	JM	2.02 ^H	RATIO	1.00 - 2.00

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.

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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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	BALA CANTT	
Test Name		Value Unit	Biological Reference interval
HEPATOCELLULAR C	ARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly I	ncreased)

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).

DDOONIOCTIC	CICNIIFICANICE.
PRUGNUNTI.	SIGNIFICANCE:
110001000110	SIGINITION HOL.

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

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

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Dr. Vinay C MD (Pathology Chairman & Co		icrobiology)		(Pathology)
NAME	: Mrs. SHUBHANGI			
AGE/ GENDER	: 28 YRS/FEMALE		PATIENT ID	: 1814850
COLLECTED BY	:		REG. NO./LAB NO.	: 012504020002
REFERRED BY	:		REGISTRATION DATE	: 02/Apr/2025 07:46 AM
BARCODE NO.	: 01528189		COLLECTION DATE	: 02/Apr/2025 07:50AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 02/Apr/2025 11:26AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	IBALA CANT	Г	
Test Name		Value	Unit	Biological Reference interval
	KIDNEY	FUNCTI	ON TEST (COMPLETI	E)
UREA: SERUM		18.91	mg/dL	10.00 - 50.00
-	TE DEHYDROGENASE (GLDH)			
CREATININE: SERUN by ENZYMATIC, SPECTR		0.79	mg/dL	0.40 - 1.20
	OGEN (BUN): SERUM	8.84	mg/dL	7.0 - 25.0
by CALCULATED, SPECT	TROPHOTOMETRY			
BLOOD UREA NITRO RATIO: SERUM	OGEN (BUN)/CREATININE	11.19	RATIO	10.0 - 20.0
by CALCULATED, SPECT	TROPHOTOMETRY			
UREA/CREATININE		23.94	RATIO	
by CALCULATED, SPECT	TROPHOTOMETRY	2 21	I I.)	2.50 (.80
URIC ACID: SERUM by URICASE - OXIDASE	PEROXIDASE	3.21	mg/dL	2.50 - 6.80
CALCIUM: SERUM		9.41	mg/dL	8.50 - 10.60
by ARSENAZO III, SPECT		2.40	(17	2.20 4.70
PHOSPHOROUS: SER by PHOSPHOMOLYBDA	KUM TE, SPECTROPHOTOMETRY	3.42	mg/dL	2.30 - 4.70
ELECTROLYTES				
SODIUM: SERUM		139.5	mmol/L	135.0 - 150.0
by ISE (ION SELECTIVE		2.65		
POTASSIUM: SERUN by ISE (ION SELECTIVE I		3.88	mmol/L	3.50 - 5.00
CHLORIDE: SERUM by ISE (ION SELECTIVE I		104.63	mmol/L	90.0 - 110.0
ESTIMATED GLOM	ERULAR FILTERATION RAT	<u>E</u>		
(eGFR): SERUM by CALCULATED	ERULAR FILTERATION RATE	104.4		
INTERPRETATION:	en pre- and post renal azotemia			

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.

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		& Microbiology)	Dr. Yugam MD & Consultant	(Pathology)		
NAME	: Mrs. SHUBHANGI					
AGE/ GENDER	: 28 YRS/FEMALE	PATIENT I	D	: 1814850		
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CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTIN		: 02/Apr/2025 11:26AM		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAI), AMBALA CANTT		1		
Test Name		Value	Unit	Biological Referen	nce interval	
1. Acute tubular necr						
 Low protein diet and 3. Severe liver diseas Other causes of decomposition of the second dialysis of the second	e. creased urea synthesis. (urea rather than creatinine dif monemias (urea is virtually ab of inappropiate antidiuretic har 10:1) WITH INCREASED CREATIN py (accelerates conversion of c eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false	mone) due to tubular secretion IINE: creatine to creatinine). increase in creatinine with cert	of urea.	gies,resulting in normal ratio wł	nen dehydratio	
 Low protein diet and Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido should produce an in Cephalosporin the 	e. creased urea synthesis. (urea rather than creatinine dif monemias (urea is virtually ab of inappropiate antidiuretic har 10:1) WITH INCREASED CREATIN py (accelerates conversion of c eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false creased BUN/creatinine ratio). rapy (interferes with creatinine	sent in blood). mone) due to tubular secretion I INE: creatine to creatinine). increase in creatinine with cert	of urea.	gies,resulting in normal ratio wł	nen dehydratio	
 Low protein diet and a severe liver diseas Severe liver diseas Other causes of destination of the severe dialysis Inherited hyperam SIADH (syndrome of the severe disease) Pregnancy. DECREASED RATIO (< Phenacimide theration of the severe disease) Muscular patients MAPPROPIATE RATIO Diabetic ketoacido should produce an in the set of the sector of th	e. creased urea synthesis. (urea rather than creatinine dif monemias (urea is virtually ab of inappropiate antidiuretic har 10:1) WITH INCREASED CREATIN py (accelerates conversion of c eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false creased BUN/creatinine ratio). rapy (interferes with creatinine JLAR FILTERATION RATE: DESCRIPTION	sent in blood). mone) due to tubular secretion IINE: creatine to creatinine). increase in creatinine with cert measurement). GFR (mL/min/1.73	of urea. ain methodolo	SOCIATED FINDINGS	nen dehydratio	
. Low protein diet an . Severe liver diseas . Other causes of de . Repeated dialysis . Inherited hyperam . SIADH (syndrome of . Pregnancy. PECREASED RATIO (< . Phenacimide thera . Rhabdomyolysis (r . Muscular patients NAPPROPIATE RATIO . Diabetic ketoacido hould produce an in . Cephalosporin the STIMATED GLOMER	e. creased urea synthesis. (urea rather than creatinine dif monemias (urea is virtually ab of inappropiate antidiuretic har 10:1) WITH INCREASED CREATIN py (accelerates conversion of c eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false creased BUN/creatinine ratio). apy (interferes with creatinine JLAR FILTERATION RATE:	sent in blood). mone) due to tubular secretion IINE: creatine to creatinine). increase in creatinine with cert measurement). GFR (mL/min/1.73 inction >90	of urea. ain methodolo m2) AS		nen dehydrati	

CKD STAGE	DESCRIPTION	GFR (mL/min/1.73m2)	ASSOCIATED FINDINGS
G1	Normal kidney function	>90	No proteinuria
G2	Kidney damage with	>90	Presence of Protein,
	normal or high GFR		Albumin or cast in urine
G3a	Mild decrease in GFR	60 -89	
G3b	Moderate decrease in GFR	30-59	
G4	Severe decrease in GFR	15-29	
G5	Kidney failure	<15	





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	Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Patholog		(Pathology)
NAME	: Mrs. SHUBHANGI		
AGE/ GENDER	: 28 YRS/FEMALE	PATIENT ID	: 1814850
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANT	Т	
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 Chopra MD (Pathology & Micr Chairman & Consultar		obiology)		(Pathology)	
NAME	: Mrs. SHUBH	ANGI			
AGE/ GENDER	: 28 YRS/FEMA	ALE		PATIENT ID	: 1814850
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CLIENT ADDRESS	: 6349/1, NICI	IOLSON ROAD, AMBA	ALA CANTT		
Test Name			Value	Unit	Biological Reference interval
			IRON	PROFILE	
IRON: SERUM by FERROZINE, SPEC	TROPHOTOMETRY		69.2	μg/dL	37.0 - 145.0
UNSATURATED II	RON BINDING (CAPACITY (UIBC)	243.4	μg/dL	150.0 - 336.0
SERUM by FERROZINE, SPEC	TROPHOTOMETER	N.			
TOTAL IRON BINI			312.6	μg/dL	230 - 430
:SERUM		(/		r.o. u.L	
by SPECTROPHOTON					
%TRANSFERRIN S by CALCULATED, SPE			22.14	%	15.0 - 50.0
TRANSFERRIN: SE			221.95	mg/dL	200.0 - 350.0
by SPECTROPHOTON	METERY (FERENE)			ç	
INTERPRETATION:-					
VARIAE	DLEJ	ANEMIA OF CHRONI	UDISEASE	IRON DEFICIENCY ANEMIA	A THALASSEMIA α/β TRAIT

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:

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 Pathologist	Dr. Yugam Ch MD (Path CEO & Consultant Path	nology)	
NAME	: Mrs. SHUBHANGI				
AGE/ GENDER	: 28 YRS/FEMALE	PATI	INT ID : 1	.814850	
COLLECTED BY	:	REG. I	NO./LAB NO. :	012504020002	
REFERRED BY	:	REGIS	TRATION DATE : (02/Apr/2025 07:46 AM	
BARCODE NO.	: 01528189	COLLI	CTION DATE : (02/Apr/2025 07:50AM	
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPO	RTING DATE : (02/Apr/2025 11:34AM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD), AMBALA CANTT			
Test Name		Value	Unit	Biological Reference inter	val
		ENDOCRINO	DLOGY		
	TH	IYROID FUNCTION	TEST: TOTAL		
TRIIODOTHYRON by CMIA (CHEMILUMIN	INE (T3): SERUM IESCENT MICROPARTICLE IMMUNO.	0.925 ASSAY)	ng/mL	0.35 - 1.93	
THYROXINE (T4):	SERUM IESCENT MICROPARTICLE IMMUNO	8.7 MASSAY)	μgm/dL	4.87 - 12.60	
			µIU/mL	0.35 - 5.50	
by CMIA (CHEMILUMIN THYROID STIMUL	ATING HORMONE (TSH): S		μιο/iiiL	0.33 - 5.50	
by CMIA (CHEMILUMIN THYROID STIMUL by CMIA (CHEMILUMIN 3rd GENERATION, ULT	IESCENT MICROPARTICLE IMMUNO		μισλιίε	0.55 - 5.50	
by CMIA (CHEMILUMIN THYROID STIMUL by CMIA (CHEMILUMIN 3rd GENERATION, ULT <u>INTERPRETATION</u> : TSH levels are subject to a day has influence on the l	IESCENT MICROPARTICLE IMMUNO. RASENSITIVE circadian variation, reaching peak leve measured serum TSH concentrations. ² lure at any level of regulation of the	ASSAY) els between 2-4 a.m and at a m TSH stimulates the production	inimum between 6-10 pm. Th and secretion of the metabo	e variation is of the order of 50%.Hence time lically active hormones, thyroxine (T4)and	

CLINICAL CONDITION	Т3	T4	TSH
Primary Hypothyroidism:	Reduced	Reduced	Increased (Significantly)
Subclinical Hypothyroidism:	Normal or Low Normal	Normal or Low Normal	High
Primary Hyperthyroidism:	Increased	Increased	Reduced (at times undetectable)
Subclinical Hyperthyroidism:	Normal or High Normal	Normal or High Normal	Reduced

LIMITATIONS:-

1. T3 and T4 circulates in reversibly bound form with Thyroid binding globulins (TBG), and to a lesser extent albumin and Thyroid binding Pre Albumin so conditions in which TBG and protein levels alter such as pregnancy, excess estrogens, androgens, anabolic steroids and glucocorticoids may falsely affect the T3 and T4 levels and may cause false thyroid values for thyroid function tests.

2. Normal levels of T4 can also be seen in Hyperthyroid patients with :T3 Thyrotoxicosis, Decreased binding capacity due to hypoproteinemia or ingestion of certain drugs (e.g.: phenytoin , salicylates).

3. Serum T4 levels in neonates and infants are higher than values in the normal adult , due to the increased concentration of TBG in neonate serum.

4. TSH may be normal in central hypothyroidism, recent rapid correction of hyperthyroidism or hypothyroidism, pregnancy, phenytoin therapy.

TRIIODOTH	RIIODOTHYRONINE (T3)		THYROXINE (T4)		ATING 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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Test Name			Value	Unit	t	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	
	RECOM	MENDATIONS OF TSH L	EVELS DURING PRE	GNANCY (µIU/mL)	•	
	1st Trimester			0.10 - 2.50		
	2nd Trimester			0.20 - 3.00		
	3rd Trimester			0.30 - 4.10		

INCREASED TSH LEVELS:

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

2.Hypothyroid patients receiving insufficient thyroid replacement therapy.

3. Hashimotos thyroiditis

4.DRUGS: Amphetamines, iodine containing agents & dopamine antagonist.

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

DECREASED TSH LEVELS:

1.Toxic multi-nodular goiter & Thyroiditis.

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

3. Autonomously functioning Thyroid adenoma

4.Secondary pituitary or hypothalamic hypothyroidism

5.Acute psychiatric illness

6.Severe dehydration.

7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.

8. Pregnancy: 1st and 2nd Trimester



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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		VITA	MINS	
	VITA	MIN D/25 HYD	ROXY VITAMIN D	03
	(DROXY VITAMIN D3): SERU escence immunoassay)	⁷ M 20 ^L	ng/mL	DEFICIENCY: < 20.0 INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0
INTERPRETATION:		20		
DEFI	CIENT:	< 20	ng	g/mL

DEFICIENT:	< 20	ng/mL		
INSUFFICIENT:	21 - 29	ng/mL		
PREFFERED RANGE:	30 - 100	ng/mL		
INTOXICATION:	> 100	ng/mL	I	

1. Vitamin D compounds are derived from dietary ergocalciferol (from plants, Vitamin D2), or cholecalciferol (from animals, Vitamin D3), or by conversion of 7- dihydrocholecalciferol to Vitamin D3 in the skin upon Ultraviolet exposure.

2.25-OH--Vitamin D represents the main body resevoir and transport form of Vitamin D and transport form of Vitamin D, being stored in adipose tissue and tightly bound by a transport protein while in circulation.

3. Vitamin D plays a primary role in the maintenance of calcium homeostatis. It promotes calcium absorption, renal calcium absorption and phosphate reabsorption, skeletal calcium deposition, calcium mobilization, mainly regulated by parathyroid harmone (PTH). 4. Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults. DECREASED:

1.Lack of sunshine exposure.

2.Inadequate intake, malabsorption (celiac disease) 3.Depressed Hepatic Vitamin D 25- hydroxylase activity

4.Secondary to advanced Liver disease

5. Osteoporosis and Secondary Hyperparathroidism (Mild to Moderate deficiency)

6.Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism.

INCREASED: 1. Hypervitaminosis D is Rare, and is seen only after prolonged exposure to extremely high doses of Vitamin D. When it occurs, it can result in

severe hypercalcemia and hyperphophatemia. CAUTION: Replacement therapy in deficient individuals must be monitored by periodic assessment of Vitamin D levels in order to prevent hypervitaminosis D

NOTE:-Dark coloured individuals as compare to whites, is at higher risk of developing Vitamin D deficiency due to excess of melanin pigment which interefere with Vitamin D absorption.



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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD), AMBALA CANTT			
Test Name		Value	Unit	Biological Reference interval	
		VITAMIN B12	2/COBALAMIN		
VITAMIN B12/COB. by CMIA (CHEMILUMIN	ALAMIN: SERUM	210	pg/mL	190.0 - 890.0	
INTERPRETATION:-					
	SED VITAMIN B12		DECREASED VITAMIN	I B12	
1.Ingestion of Vitamin C		1.Pregnar		2 1 1 1 1	
2.Ingestion of Estrogen 3.Ingestion of Vitamin A		2.DRUGS: 3.Ethanol	Aspirin, Anti-convulsants	, Colchicine	
			ceptive Harmones		
		5.Haemo			
6.Uremia			e Myeloma		
2.In humans, it is obt 3.The body uses its vi excreted. 4.Vitamin B12 deficie ileal resection, small 5.Vitamin B12 deficie proprioception, poor the neurologic defect	ency may be due to lack of IF se intestinal diseases). ency frequently causes macrocy coordination, and affective be ts without macrocytic anemia. nic acid and homocysteine leve or antibodies to intrinsic factor	ns and requires intri ically, reabsorbing v cretion by gastric m ytic anemia, glossitis havioral changes. Th els are also elevated	nsic factor (IF) for absorp tamin B12 from the ileun ucosa (eg, gastrectomy, g , peripheral neuropathy, ese manifestations may d in vitamin B12 deficiency d to identify this potentia	n and returning it to the liver; very little is astric atrophy) or intestinal malabsorption (eg, weakness, hyperreflexia, ataxia, loss of occur in any combination; many patients have states. I cause of vitamin B12 malabsorption.	





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

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







	Dr. Vinay Cho MD (Pathology & Chairman & Cons	Microbiology)	Dr. Yugam MD CEO & Consultant	(Pathology)	
NAME	: Mrs. SHUBHANGI				
AGE/ GENDER	: 28 YRS/FEMALE	PATIEN	T ID	: 1814850	
COLLECTED BY :		REG. NO./LAB NO.		: 012504020002	
REFERRED BY	:	REGISTRATION DATE		: 02/Apr/2025 07:46 AM	
BARCODE NO. : 01528189		COLLECTION DATE		: 02/Apr/2025 07:50AM	
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPOR	TING DATE	: 02/Apr/2025 08:42AM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT			
Test Name		Value	Unit	Biological Reference interv	
		CLINICAL PATH	IOLOGY		
	URINE ROU	TINE & MICROSC	OPIC EXAMI	NATION	
PHYSICAL EXAM	INATION				
QUANTITY RECIEVED by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY COLOUR by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY TRANSPARANCY by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY		10	ml		
		AMBER YELLOW	7	PALE YELLOW	
		CLEAR		CLEAR	
SPECIFIC GRAVIT	Y TANCE SPECTROPHOTOMETRY	1.02		1.002 - 1.030	
CHEMICAL EXAM	MINATION				
REACTION		ACIDIC			
PROTEIN	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)	
SUGAR	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)	
pH by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	6		5.0 - 7.5	
BILIRUBIN	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)	
NITRITE	TANCE SPECTROPHOTOMETRY.	Negative		NEGATIVE (-ve)	
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY.		Normal	EU/dL	0.2 - 1.0	
KETONE BODIES	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)	
BLOOD		Negative		NEGATIVE (-ve)	
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY ASCORBIC ACID by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY		NEGATIVE (-ve)		NEGATIVE (-ve)	
MICDOSCODIC F	V A MINIA THON				

MICROSCOPIC EXAMINATION



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

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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, Al	MBALA CANTT				
Test Name		Value	Unit	Biological Reference interval		
RED BLOOD CELL by MICROSCOPY ON (S (RBCs) CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)	/HPF	0 - 3		
PUS CELLS by MICROSCOPY ON (CENTRIFUGED URINARY SEDIMENT	1-2	/HPF	0 - 5		
EPITHELIAL CELL	S	2-4	/HPF	ABSENT		

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT		,	
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 ***





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