



	Dr. Vinay Chopr MD (Pathology & Mic Chairman & Consulta	robiology)		(Pathology)
NAME	: Mrs. KUSUM			
AGE/ GENDER	: 49 YRS/FEMALE		PATIENT ID	: 1808001
COLLECTED BY	:		REG. NO./LAB NO.	: 012503270004
REFERRED BY	:		<b>REGISTRATION DATE</b>	: 27/Mar/2025 07:35 AM
BARCODE NO.	: 01527836		COLLECTION DATE	: 27/Mar/2025 07:39AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 27/Mar/2025 09:41AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AME	BALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	SWASTI	HYA WE	LLNESS PANEL: 1	.5
	COMP	PLETE BLO	OOD COUNT (CBC)	
RED BLOOD CELL	S (RBCS) COUNT AND INDICE	<u>S</u>		
HAEMOGLOBIN (HE	3)	12.9	gm/dL	12.0 - 16.0
by CALORIMETRIC RED BLOOD CELL (	RBC) COUNT	4.71	Millions	/cmm 3.50 - 5.00
by HYDRO DYNAMIC FO	CUSING, ELECTRICAL IMPEDENCE			
PACKED CELL VOL	UME (PCV) ITOMATED HEMATOLOGY ANALYZER	39.9	%	37.0 - 50.0
MEAN CORPUSCUL	AR VOLUME (MCV)	84.6	fL	80.0 - 100.0
MEAN CORPUSCUL	ITOMATED HEMATOLOGY ANALYZER AR HAEMOGLOBIN (MCH)	27.4	pg	27.0 - 34.0
	ITOMATED HEMATOLOGY ANALYZER AR HEMOGLOBIN CONC. (MCH	HC) 32.4	g/dL	32.0 - 36.0
by CALCULATED BY AU	ITOMATED HEMATOLOGY ANALYZER	í.		
	UTION WIDTH (RDW-CV)	13.8	%	11.00 - 16.00
RED CELL DISTRIB	UTION WIDTH (RDW-SD)	43.7	fL	35.0 - 56.0
MENTZERS INDEX		17.96	RATIO	BETA THALASSEMIA TRAIT:
by CALCULATED				13.0
				IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INI	DEX	76.56	RATIO	BETA THALASSEMIA TRAIT:
by CALCULATED				<= 65.0
				IRON DEFICIENCY ANEMIA: 65.0
WHITE BLOOD CE	LLS (WBCS)			
TOTAL LEUCOCYT		5050	/cmm	4000 - 11000
•	BY SF CUBE & MICROSCOPY BLOOD CELLS (nRBCS)	NIL		0.00 - 20.00
by AUTOMATED 6 PAR	T HEMATOLOGY ANALYZER			0.00 20.00
NUCLEATED RED F	BLOOD CELLS (nRBCS) %	NIL	%	< 10 %
NOCLEATED RED I				





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

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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



Dr Vinav

C



Dr. Vinay Chop MD (Pathology & Mic Chairman & Consult		icrobiology)	Dr. Yugam MD CEO & Consultant	(Pathology)
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	IBALA CANTI		
Test Name		Value	Unit	Biological Reference interval
	AUTOMATED HEMATOLOGY ANALYZER			
<u>DIFFERENTIAL L</u>	<u>LEUCOCYTE COUNT (DLC)</u>			
NEUTROPHILS by FLOW CYTOMETR	RY BY SF CUBE & MICROSCOPY	68	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETR	RY BY SF CUBE & MICROSCOPY	23	%	20 - 40
EOSINOPHILS		2	%	1 - 6
	RY BY SF CUBE & MICROSCOPY			
MONOCYTES	RY BY SF CUBE & MICROSCOPY	7	%	2 - 12
BASOPHILS		0	%	0 - 1
•	RY BY SF CUBE & MICROSCOPY			
	<u>KOCYTES (WBC) COUNT</u>			
ABSOLUTE NEUT	ROPHIL COUNT RY BY SF CUBE & MICROSCOPY	3434	/cmm	2000 - 7500
ABSOLUTE LYMP		1162	/cmm	800 - 4900
	RY BY SF CUBE & MICROSCOPY			
ABSOLUTE EOSIN		101	/cmm	40 - 440
by FLOW CYTOMETR ABSOLUTE MONO	RY BY SF CUBE & MICROSCOPY	354	/cmm	80 - 880
	RY BY SF CUBE & MICROSCOPY	334	/ciiiii	80 - 880
PLATELETS AND	OTHER PLATELET PREDICTIV	/E MARKERS.		
PLATELET COUN	T (PLT) FOCUSING. ELECTRICAL IMPEDENCE	202000	/cmm	150000 - 450000
PLATELETCRIT (1	PCT)	0.25	%	0.10 - 0.36
MEAN PLATELET	FOCUSING, ELECTRICAL IMPEDENCE	12 <sup>H</sup>	fL	6.50 - 12.0
	FOCUSING, ELECTRICAL IMPEDENCE	12		0.30 12.0
	E CELL COUNT (P-LCC)	82000	/cmm	30000 - 90000
	FOCUSING, ELECTRICAL IMPEDENCE	40.7	04	11.0 45.0
	E CELL RATIO (P-LCR) FOCUSING, ELECTRICAL IMPEDENCE	40.7	%	11.0 - 45.0
	BUTION WIDTH (PDW)	16.5	%	15.0 - 17.0
by HYDRO DYNAMIC	FOCUSING, ELECTRICAL IMPEDENCE			
NOTE: TEST CONDU	UCTED ON EDTA WHOLE BLOOD			



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







	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbiol Chairman & Consultant Pat		(Pathology)
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA (	CANTT	
Test Name	Val	luo Unit	Biological Deference interval





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CLIENT CODE.	: KOS DIAGNOSTIC LAB		<b>REPORTING DATE</b>	: 27/Mar/2025 02:31PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	ABALA CANTT		
	10010/1,1101101201110112,11			
Test Name		Value	Unit	<b>Biological Reference interva</b>
WHOLE BLOOD by HPLC (HIGH PERFO. ESTIMATED AVER.	IAEMOGLOBIN (HbA1c): RMANCE LIQUID CHROMATOGRAPHY) AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY)	5.6	% mg/dL	4.0 - 6.4 60.00 - 140.00
	AS PER AMERICAN D	IABETES ASSOCI	ATION (ADA):	
	REFERENCE GROUP	GLYCOSYLATED HEMOGLOGIB (H		(HBAIC) in %
Non di	abetic Adults >= 18 years	/	<5.7	
	t Risk (Prediabetes)		5.7 – 6.4	
D	iagnosing Diabetes		>= 6.5	
D			Age > 19 Years of Therapy:	< 7.0
	ic goals for glycemic control		s Suggested:	>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 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.



	<b>Dr. Vinay Chop</b> MD (Pathology & M Chairman & Consul	licrobiology)	Dr. Yugam MD CEO & Consultant	(Pathology)
NAME	: Mrs. KUSUM			
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CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPO	RTING DATE	: 27/Mar/2025 11:32AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	IBALA CANTT		
Test Name		Value	Unit	<b>Biological Reference interval</b>
	ERYTHROO	CYTE SEDIMEN'	<b>FATION RATE</b>	(ESR)
by RED CELL AGGRE	EDIMENTATION RATE (ESR) GATION BY CAPILLARY PHOTOMETRY ic test because an elevated result o	25 <sup>H</sup>	mm/1st h sence of inflammati	r 0 - 20
as C-reactive protein 3. This test may also systemic lupus erythe <b>CONDITION WITH LON</b> A low ESR can be see polycythaemia), sigr as sickle cells in sickl <b>NOTE:</b> 1. ESR and C - reactive 2. Generally, ESR doe 3. <b>CRP is not affected</b> 4. If the ESR is elevate 5. Women tend to ha 5. Drugs such as dext	be used to monitor disease activity ematosus <b>W ESR</b> in with conditions that inhibit the ne ificantly high white blood cell cour e cell anaemia) also lower the ESR e protein (C-RP) are both markers of s not change as rapidly as does CRF <b>by as many other factors as is ESR</b> , ed, it is typically a result of two typ ve a higher ESR, and menstruation a	and response to ther ormal sedimentation of (leucocytosis), and finflammation. P, either at the start o making it a better mar es of proteins, globuli and pregnancy can cat	apy in both of the a of red blood cells, si some protein abno f inflammation or as <b>rker of inflammatior</b> ns or fibrinogen. use temporary eleva	rmalities. Šome changes in red cell shape (such s it resolves. <b>1.</b>





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		y <b>Chopra</b> ogy & Microbiology) Consultant Pathologist	Dr. Yugan MD CEO & Consultant	(Pathology)
NAME AGE/ GENDER COLLECTED BY REFERRED BY BARCODE NO. CLIENT CODE. CLIENT ADDRESS	: Mrs. KUSUM : 49 YRS/FEMALE : : : 01527836 : KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON RC	RI RI CO RI	ATIENT ID 2G. NO./LAB NO. 2GISTRATION DATE 2DLLECTION DATE 2PORTING DATE	: 1808001 <b>: 012503270004</b> : 27/Mar/2025 07:35 AM : 27/Mar/2025 07:39AM : 27/Mar/2025 11:18AM
Test Name		Value	Unit	Biological Reference interval
GLUCOSE FASTIN by GLUCOSE OXIDAS	G (F): PLASMA E - PEROXIDASE (GOD-POD)	GLUCOSE F 111.46 <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	on of 75 gms of glucose) is r	dl is considered normal. 125 mg/dl is considered a recommended for all such a/dl is highly suggestive o	n pătients. of diabetic state. A repe	prediabetic. A fasting and post-prandial blood at post-prandial is strongly recommended for al natory for diabetic state.

KOS Diagnostic Lab (A Unit of KOS Healthcare)





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	<b>Dr. Vinay Chopra</b> MD (Pathology & Micro Chairman & Consultant	obiology)	Dr. Yugam MD ( CEO & Consultant	(Pathology)
	EMALE	REGIST COLLEG REPOR	NT ID 0./LAB NO. TRATION DATE CTION DATE TING DATE	: 1808001 <b>: 012503270004</b> : 27/Mar/2025 07:35 AM : 27/Mar/2025 07:39AM : 27/Mar/2025 11:35AM
Test Name		Value	Unit	<b>Biological Reference interval</b>
		PID PROFILE	·BASIC	
CHOLESTEROL TOTAL: SERU by CHOLESTEROL OXIDASE PAP		204.12 <sup>H</sup>	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM by GLYCEROL PHOSPHATE OXIDAS	E (ENZYMATIC)	76.21	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROL (DIRECT by Selective inhibition	'): SERUM	72.13	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTON	METRY	116.75	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 CHOLESTEROL: SEI by CALCULATED, SPECTROPHOTON		131.99 <sup>H</sup>	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: SERUM by CALCULATED, SPECTROPHOTOM		15.24	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM by CALCULATED, SPECTROPHOTOM		484.45	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: S by CALCULATED, SPECTROPHOTON	SERUM	2.83	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0



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Test Name		Value	Unit	<b>Biological Reference interval</b>
				MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0
LDL/HDL RATIO: S by CALCULATED, SPE		1.62	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	1.06 <sup>L</sup>	RATIO	3.00 - 5.00

# INTERPRETATION:

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

3. Low HDL levels are associated with increased risk for Atherosclerotic Cardiovascular disease (ASCVD) due to insufficient HDL being available to participate in reverse cholesterol transport, the process by which cholesterol is eliminated from peripheral tissues. 4. NLA-2014 identifies Non HDL Cholesterol (an indicator of all atherogeniclipoproteins such as LDL, VLDL, IDL, Lpa, Chylomicron remnants) along with LDL-cholesterol as co- primary target for cholesterol lowering therapy. Note that major risk factors can modify treatment goals for LDL & Non HDL

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





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EXCELLENCE IN HEALTHCARE & DIAGNOSTICS
Dr. Yugam Chopra MD (Pathology) CEO & Consultant Pathologist

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

Test Name	Value	Unit	<b>Biological Reference interval</b>
LIVER F	UNCTION TE	ST (COMPLETE)	
BILIRUBIN TOTAL: SERUM by DIAZOTIZATION, SPECTROPHOTOMETRY	0.5	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.13	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.37	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	20.2	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	19.4	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.04	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by para nitrophenyl phosphatase by amino methyl propanol	126.55	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUN by SZASZ, SPECTROPHTOMETRY	1 22.9	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	7.44	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	4.25	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	3.19	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.33	RATIO	1.00 - 2.00

<u>INTERPRETATION</u> 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:**

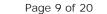
DRUG HEPATOTOXICITY	> 2		
ALCOHOLIC HEPATITIS	> 2 (Highly Suggestive)		
CIRRHOSIS	1.4 - 2.0		
INTRAHEPATIC CHOLESTATIS	> 1.5		





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

PROGNOSTIC	SIGNIFICANCE:

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



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	AKUS
EXC	ELLENCE IN HEALTHCARE & DIAGNOSTICS

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Test Name		Value	Unit	<b>Biological Reference interval</b>
	KIDNE	Y FUNCTION	TEST (COMPLETI	E)
UREA: SERUM		30.34	mg/dL	10.00 - 50.00
	IATE DEHYDROGENASE (GLDH)			
CREATININE: SER		0.82	mg/dL	0.40 - 1.20
-	ROGEN (BUN): SERUM	14.18	mg/dL	7.0 - 25.0
	ROGEN (BUN)/CREATININE	17.29	RATIO	10.0 - 20.0
by CALCULATED, SPE	ECTROPHOTOMETRY			
UREA/CREATININ		37	RATIO	
URIC ACID: SERUN by URICASE - OXIDAS		2.68	mg/dL	2.50 - 6.80
CALCIUM: SERUM by ARSENAZO III, SPE		10.39	mg/dL	8.50 - 10.60
PHOSPHOROUS: SI	ERUM DATE, SPECTROPHOTOMETRY	3.76	mg/dL	2.30 - 4.70
ELECTROLYTES				
SODIUM: SERUM by ISE (ION SELECTIV	'E ELECTRODE)	143.62	mmol/L	135.0 - 150.0
POTASSIUM: SERU		3.95	mmol/L	3.50 - 5.00
CHLORIDE: SERUN by ISE (ION SELECTIV ESTIMATED GLOI		107.72 <b>E</b>	mmol/L	90.0 - 110.0
	MERULAR FILTERATION RATE			

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.



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		Chopra y & Microbiology) consultant Pathologist	Dr. Yugam Chopra MD (Pathology) CEO & Consultant Pathologist	
NAME	: Mrs. KUSUM			
AGE/ GENDER	: 49 YRS/FEMALE	P	ATIENT ID	: 1808001
COLLECTED BY		D	REG. NO./LAB NO.	: 012503270004
	•			
REFERRED BY	:		EGISTRATION DATE	: 27/Mar/2025 07:35 AM
BARCODE NO.	:01527836	C	COLLECTION DATE	: 27/Mar/2025 07:39AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	F	REPORTING DATE	: 27/Mar/2025 11:35AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROA	D, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		value	Umt	biological Reference interval
burns, surgery, cache 7. Urine reabsorption	iction plus ke or production or tissue bre xia, high fever). (e.g. ureter colostomy)		n, GI bleeding, thyrotoxic	osis, Cushing's syndrome, high protein diet,
4. High protein intake 5. Impaired renal fur 6. Excess protein inta burns, surgery, cache 7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia DECREASED RATIO (< 1. Acute tubular necr 2. Low protein diet an 3. Severe liver diseas 4. Other causes of de 5. Repeated dialysis ( 6. Inherited hyperam 7. SIADH (syndrome of 8. Pregnancy. DECREASED RATIO (<	inction plus ke or production or tissue bre xia, high fever). (e.g. ureter colostomy) tass (subnormal creatinine pro- tetracycline, glucocorticoids) <b>co:1) WITH ELEVATED CREATIN</b> a (BUN rises disproportionately superimposed on renal disease <b>to:1) WITH DECREASED BUN :</b> osis. a d starvation. e. creased urea synthesis. (urea rather than creatinine d monemias (urea is virtually all of inappropiate antidiuretic ha <b>to:1) WITH INCREASED CREATIN</b>	oduction) INE LEVELS: y more than creatining se. iffuses out of extracel osent in blood). rmone) due to tubula NINE:	e) (e.g. obstructive uropa llular fluid). r secretion of urea.	
4. High protein intake 5. Impaired renal fur 6. Excess protein inta burns, surgery, cache 7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia DECREASED RATIO (< 1. Acute tubular necr 2. Low protein diet an 3. Severe liver diseas 4. Other causes of de 5. Repeated dialysis ( 6. Inherited hyperam 7. SIADH (syndrome of 8. Pregnancy. DECREASED RATIO (< 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients	inction plus ke or production or tissue bre xia, high fever). (e.g. ureter colostomy) lass (subnormal creatinine pro- tetracycline, glucocorticoids) <b>co:1) WITH ELEVATED CREATIN</b> a (BUN rises disproportionately superimposed on renal disease <b>to:1) WITH DECREASED BUN :</b> osis. a d starvation. e. creased urea synthesis. (urea rather than creatinine d monemias (urea is virtually all of inappropiate antidiuretic ha <b>to:1) WITH INCREASED CREATIN</b> py (accelerates conversion of eleases muscle creatinine). who develop renal failure.	oduction) INE LEVELS: y more than creatining se. iffuses out of extracel osent in blood). rmone) due to tubula NINE:	e) (e.g. obstructive uropa llular fluid). r secretion of urea.	
4. High protein intake 5. Impaired renal fur 6. Excess protein inta burns, surgery, cache 7. Urine reabsorption 8. Reduced muscle m 9. Certain drugs (e.g. INCREASED RATIO (>2 1. Postrenal azotemia DECREASED RATIO (< 1. Acute tubular necr 2. Low protein diet an 3. Severe liver diseas 4. Other causes of de 5. Repeated dialysis ( 6. Inherited hyperam 7. SIADH (syndrome of 8. Pregnancy. DECREASED RATIO (< 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients INAPPROPIATE RATIO	inction plus ke or production or tissue bre xia, high fever). (e.g. ureter colostomy) tass (subnormal creatinine pro- tetracycline, glucocorticoids) <b>co:1) WITH ELEVATED CREATIN</b> a (BUN rises disproportionately superimposed on renal disease <b>to:1) WITH DECREASED BUN :</b> osis. a d starvation. e. creased urea synthesis. (urea rather than creatinine d monemias (urea is virtually at of inappropiate antidiuretic ha <b>to:1) WITH INCREASED CREATIN</b> py (accelerates conversion of eleases muscle creatinine). who develop renal failure. :	oduction) INE LEVELS: y more than creatining se. iffuses out of extracel osent in blood). rmone) due to tubula NINE: creatine to creatinine	e) (e.g. obstructive uropa llular fluid). r secretion of urea.	

CKD STAGE	DESCRIPTION	GFR ( mL/min/1.73m2 )	ASSOCIATED FINDINGS
G1	Normal kidney function	>90	No proteinuria
G2	Kidney damage with normal or high GFR	>90	Presence of Protein , 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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	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbiology) Chairman & Consultant Patholo		(Pathology)
NAME	: Mrs. KUSUM		
AGE/ GENDER	: 49 YRS/FEMALE	PATIENT ID	: 1808001
<b>COLLECTED BY</b>	:	<b>REG. NO./LAB NO.</b>	: 012503270004
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 27/Mar/2025 07:35 AM
BARCODE NO.	: 01527836	<b>COLLECTION DATE</b>	: 27/Mar/2025 07:39AM
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CAN	TT	
Test Name	Value	Unit	<b>Biological Reference interval</b>

COMMENTS:

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

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

ADVICE:

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



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%

Decreased

mg/dL

15.0 - 50.0

200.0 - 350.0

Normal or Increased

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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AME	BALA CANTT			
Test Name		Value	Unit	Biological Reference interval	
		IRON P	ROFILE		
IRON: SERUM	TROPHOTOMETRY	76.4	μg/dL	37.0 - 145.0	
UNSATURATED IR SERUM	ON BINDING CAPACITY (UIBC)	286.18	μg/dL	150.0 - 336.0	
	DING CAPACITY (TIBC)	362.58	µg/dL	230 - 430	

by SPECTROPHOTOMETERY (FERENE)		Ū	
INTERPRETATION:-			
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

21.07

257.43

**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.

**SERUM FERRITIN:** 

Normal to Increased

## % TRANSFERRIN SATURATION:

by SPECTROPHOTOMETERY

TRANSFERRIN: SERUM

%TRANSFERRIN SATURATION: SERUM

by CALCULATED, SPECTROPHOTOMETERY (FERENE)

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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		hopra & Microbiology) onsultant Pathologist		m Chopra D (Pathology) nt Pathologist
NAME	: Mrs. KUSUM			
AGE/ GENDER	: 49 YRS/FEMALE	]	PATIENT ID	: 1808001
COLLECTED BY	:	1	REG. NO./LAB NO.	: 012503270004
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BARCODE NO.	: 01527836	(	COLLECTION DATE	: 27/Mar/2025 07:39AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	1	REPORTING DATE	: 27/Mar/2025 01:58PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD	), AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	TH		INOLOGY ION TEST: TOTA	L
TRIIODOTHYRON	INE (T3): SERUM	1.26 ASSAY)	ng/mL	0.35 - 1.93
THYROXINE (T4): by CMIA (CHEMILUMIN	SERUM IESCENT MICROPARTICLE IMMUNO	6.79 ASSAY)	µgm/dl	4.87 - 12.60
	ATING HORMONE (TSH): S iescent microparticle immuno rasensitive	0.014	µIU/ml	0.35 - 5.50
day has influence on the	measured serum TSH concentrations. lure at any level of regulation of the	TSH stimulates the proc	luction and secretion of the	pm. The variation is of the order of 50%.Hence time of the metabolically active hormones, thyroxine (T4) and her underproduction (hypothyroidism) or
CLINICAL CONDITION	Т3		T4	TSH
Primary Hypothyroidis Subclinical Hypothyroi		W Normal N	Reduced ormal or Low Normal	Increased (Significantly) High

#### LIMITATIONS:-

Primary Hyperthyroidism:

Subclinical Hyperthyroidism:

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

Increased

Normal or High Normal

Reduced (at times undetectable)

Reduced

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

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

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

TRIIODOTHYRONINE (T3)		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

Increased

Normal or High Normal





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	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbiology) Chairman & Consultant Pathologi		(Pathology)
NAME	: Mrs. KUSUM		
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Test Name		Value	Unit		<b>Biological Reference interval</b>	
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	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





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NAME	: Mrs. KUSUM			
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CLIENT CODE.	: KOS DIAGNOSTIC LAB		<b>REPORTING DATE</b>	: 27/Mar/2025 11:32AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMB	SALA CANTI	2	
Test Name		Value	Unit	<b>Biological Reference interval</b>
		VII	AMINS	
	VITAMI	N D/25 HY	YDROXY VITAMIN D	03
,	DROXY VITAMIN D3): SERUM escence immunoassay)	17.4 <sup>L</sup>	ng/mL	DEFICIENCY: < 20.0 INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0
INTERPRETATION:				

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

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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		<b>Chopra</b> ogy & Microbiology) Consultant Pathologist	Dr. Yugam MD CEO & Consultant	(Pathology)			
NAME	: Mrs. KUSUM						
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BARCODE NO.	: 01527836		COLLECTION DATE	: 27/Mar/2025 07:39AM			
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 27/Mar/2025 11:46AM			
CLIENT ADDRESS	: 6349/1, NICHOLSON RC		LEI OKTING DATE				
Test Name		Value	Unit	Biological Reference interval			
		VITAMIN B12	2/COBALAMIN				
by CMIA (CHEMILUMII	BALAMIN: SERUM	531 INOASSAY)	pg/mL	190.0 - 890.0			
<u>NTERPRETATION:-</u>	SED VITAMIN B12		DECREASED VITAMIN	N B12			
1.Ingestion of Vitamin C		1.Pregnar	1.Pregnancy				
2.Ingestion of Estro	gen	2.DRUGS:	Aspirin, Anti-convulsants	, Colchicine			
3.Ingestion of Vitamin A		3.Ethanol					
4.Hepatocellular injury 5.Myeloproliferative disorder			4. Contraceptive Harmones 5.Haemodialysis				
6.Uremia			6. Multiple Myeloma				
2.In humans, it is ob 3.The body uses its v excreted. 4.Vitamin B12 deficio leal resection, smal 5.Vitamin B12 defici	ency may be due to lack of IF I intestinal diseases). ency frequently causes macr	teins and requires intri omically, reabsorbing vi secretion by gastric mu ocytic anemia, glossitis behavioral changes. Th a. evels 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			





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NAME	: Mrs. KUSUM					
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BARCODE NO.	:01527836	COLLEC	TION DATE	: 27/Mar/2025 07:39AM		
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPOR'	TING DATE	: 27/Mar/2025 09:28AM		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	AMBALA CANTT				
Test Name		Value	Unit	<b>Biological Reference interva</b>		
		CLINICAL PATH	IOLOGY			
	URINE ROU	TINE & MICROSC	OPIC EXAMI	NATION		
PHYSICAL EXAM	INATION					
QUANTITY RECIE by DIP STICK/REFLEC	VED STANCE SPECTROPHOTOMETRY	10	ml			
COLOUR by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY		PALE YELLOW		PALE YELLOW		
TRANSPARANCY	TANCE SPECTROPHOTOMETRY	HAZY		CLEAR		
SPECIFIC GRAVIT		1.01		1.002 - 1.030		
CHEMICAL EXAN	<b>MINATION</b>					
REACTION by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	ACIDIC				
PROTEIN by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)		
SUGAR by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)		
pH by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	6		5.0 - 7.5		
BILIRUBIN by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)		
NITRITE by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY.	Negative		NEGATIVE (-ve)		
UROBILINOGEN	TANCE SPECTROPHOTOMETRY	Normal	EU/dL	0.2 - 1.0		
KETONE BODIES by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)		
BLOOD by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	NEGATIVE (-ve)		NEGATIVE (-ve)		
	TANCE SPECTROPHOTOMETRY	NEGATIVE (-ve)		NEGATIVE (-ve)		
MICDOSCODICE	V A MINI A THON					

MICROSCOPIC EXAMINATION



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

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

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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

NAME	: Mrs. KUSUM				
AGE/ GENDER	: 49 YRS/FEMALE	PATIENT	' ID	: 1808001 <b>: 012503270004</b> : 27/Mar/2025 07:35 AM : 27/Mar/2025 07:39AM : 27/Mar/2025 09:28AM	
COLLECTED BY	:	REG. NO.	/LAB NO.		
<b>REFERRED BY</b>	:	REGISTR	ATION DATE		
BARCODE NO.	: 01527836	COLLECT	ION DATE		
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORT	ING DATE		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AN	MBALA CANTT			
Test Name		Value	Unit	Biological Reference interval	
RED BLOOD CELL	S (RBCs) CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)	/HPF	0 - 3	
DUCCELLC		2.4	/LIDE	0 5	

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)	/HPF	0 - 3	
PUS CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	3-4	/HPF	0 - 5	
EPITHELIAL CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	5-7	/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 \*\*\*





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