



U 9001 : 2008 GERIIFIED LAB					
	Dr. Vinay Chopra MD (Pathology & Microl Chairman & Consultant			(Pathology)	
IAME : Mrs. USH	IA				
GE/ GENDER : 48 YRS/F	EMALE		PATIENT ID	: 1722681	
OLLECTED BY :			REG. NO./LAB NO.	: 012501130027	
EFERRED BY :			REGISTRATION DATE	: 13/Jan/2025 12:28 PM	
ARCODE NO. : 01523818			COLLECTION DATE	: 13/Jan/2025 12:29PM	
	GNOSTIC LAB		REPORTING DATE	: 13/Jan/2025 01:00PM	
CLIENT ADDRESS : 6349/1, 1	NICHOLSON ROAD, AMBAI	LA CANTT			
Test Name		Value	Unit	Biological Reference in	nterval
	SWASTH	YA WEI	LLNESS PANEL: 1.0	D	
			DOD COUNT (CBC)		
ED BLOOD CELLS (RBCS) CO					
IAEMOGLOBIN (HB)		11.4 ^L	gm/dL	12.0 - 16.0	
RED BLOOD CELL (RBC) COUN		4.06	Millions/	/cmm 3.50 - 5.00	
by HYDRO DYNAMIC FOCUSING, ELE PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED H		35.3 ^L	%	37.0 - 50.0	
AEAN CORPUSCULAR VOLUME by CALCULATED BY AUTOMATED H	E (MCV)	87	fL	80.0 - 100.0	
IEAN CORPUSCULAR HAEMO		28	pg	27.0 - 34.0	
MEAN CORPUSCULAR HEMOG	LOBIN CONC. (MCHC)	32.2	g/dL	32.0 - 36.0	
RED CELL DISTRIBUTION WID by CALCULATED BY AUTOMATED H		14.5	%	11.00 - 16.00	
RED CELL DISTRIBUTION WID	TH (RDW-SD)	47.4	fL	35.0 - 56.0	
MENTZERS INDEX by CALCULATED		21.43	RATIO	BETA THALASSEMIA 13.0 IRON DEFICIENCY AN >13.0	
REEN & KING INDEX by CALCULATED		30.98	RATIO	BETA THALASSEMIA 65.0 IRON DEFICIENCY AN	
WHITE BLOOD CELLS (WBCS)			65.0	
OTAL LEUCOCYTE COUNT (TI	LC)	3790 ^L	/cmm		
NHITE BLOOD CELLS (WBCS) FOTAL LEUCOCYTE COUNT (T by flow cytometry by sf cube NUCLEATED RED BLOOD CELI by automated 6 part hematolo	LC) & <i>microscopy</i> LS (nRBCS)	3790^L NIL	/cmm	65.0	





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

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

NAME	: Mrs. USHA		
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Test Name	Value	Unit	Biological Reference interval
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS by flow cytometry by SF cube & microscopy	62	%	50 - 70
LYMPHOCYTES by flow cytometry by SF cube & microscopy	30	%	20 - 40
EOSINOPHILS by flow cytometry by SF cube & microscopy	3	%	1 - 6
MONOCYTES by flow cytometry by SF cube & microscopy	5	%	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	2350	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by flow cytometry by sf cube & microscopy	1137	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by flow cytometry by sf cube & microscopy	114	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by flow cytometry by sf cube & microscopy	190	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT by flow cytometry by sf cube & microscopy	0	/cmm	0 - 110
PLATELETS AND OTHER PLATELET PREDICTIVE	MARKERS.		
PLATELET COUNT (PLT) by hydro dynamic focusing, electrical impedence	161000	/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	12	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	66000	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	41	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by hydro dynamic focusing, electrical impedence NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD	16.2	%	15.0 - 17.0



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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



MENTATION RATE (ESR) TION BY CAPILLARY PHOTOM test because an elevated re bes not tell the health pract	REA REA COU REI AD, AMBALA CANTT Value HROCYTE SEDIME 29 ^H ETRY esult often indicates the itioner exactly where the	mm/1st	
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MENTATION RATE (ESR) TION BY CAPILLARY PHOTOM test because an elevated re bes not tell the health pract	29 ^H ETRY esult often indicates the itioner exactly where the	mm/1st	
e used to monitor disease ad natosus ESR with conditions that inhibit icantly high white blood cel cell anaemia) also lower th protein (C-RP) are both mar not change as rapidly as dow y as many other factors as is t, it is typically a result of tw a higher ESR, and menstrue	des inflammation. For th ctivity and response to th the normal sedimentation locunt (leucocytosis), a e ESR. kers of inflammation. es CRP, either at the star s ESR, making it a better in yo types of proteins, glob ation and pregnancy can	e inflammation is in the is reason, the ESR is typ herapy in both of the a on of red blood cells, si nd some protein abno t of inflammation or as marker of inflammatior pulins or fibrinogen. cause temporary eleva	e body or what is causing it. pically used in conjunction with other test such bove diseases as well as some others, such as uch as a high red blood cell count rmalities. Some changes in red cell shape (suc s it resolves. 1. tions.
not y as l, it e a h in, r	change as rapidly as do s many other factors as is is typically a result of tw higher ESR, and menstru- methyldopa, oral contra	s many other factors as is ESR, making it a better r is typically a result of two types of proteins, glob higher ESR, and menstruation and pregnancy can methyldopa, oral contraceptives, penicillamine p	change as rapidly as does CRP, either at the start of inflammation or as s many other factors as is ESR, making it a better marker of inflammatior is typically a result of two types of proteins, globulins or fibrinogen. higher ESR, and menstruation and pregnancy can cause temporary eleva methyldopa, oral contraceptives, penicillamine procainamide, theophy





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	MD (Pathol	y Chopra ogy & Microbiology) & Consultant Pathologist	Dr. Yugan MD CEO & Consultant	(Pathology)
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CLIENT ADDRESS	: 6349/1, NICHOLSON R	OAD, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		INICAL CHEMISTR	Y/BIOCHEMIST	'nY
	CL			
	CL	GLUCOSE FA		

KOS Diagnostic Lab (A Unit of KOS Healthcare)

IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:

 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.

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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		C hopra y & Microbiology) Consultant Pathologist		(Pathology)
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Test Name		Value	Unit	Biological Reference interval
		LIPID PRO	FILE : BASIC	
CHOLESTEROL TOT by CHOLESTEROL OXI		198.07	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SE by GLYCEROL PHOSP	ERUM HATE OXIDASE (ENZYMATIC)	108.93	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROL by SELECTIVE INHIBITI		68.48	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL by CALCULATED, SPEC		107.8	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 CHOLEST by CALCULATED, SPEC		129.59	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 CHOLESTERO		21.79	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SER	UM	505.07	mg/dL	350.00 - 700.00
CHOLESTEROL/HD by CALCULATED, SPEC	L RATIO: SERUM	2.89	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0

DR.YUGAM CHOPRA

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Test Name		Value	Unit	Biological Reference interval
LDL/HDL RATIO: S by CALCULATED, SPE		1.57	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	1.59 ^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.

 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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Dr. Yugam Chopra

	MD (Pathology & Mi Chairman & Consult	crobiology)	Dr. Tugam MD (CEO & Consultant	(Pathology)
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Test Name		Value	Unit	Biological Reference interval
	LIVER	FUNCTION	TEST (COMPLETE)	
BILIRUBIN TOTAL by DIAZOTIZATION, SI	: SERUM PECTROPHOTOMETRY	0.57	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT	Г (CONJUGATED): SERUM spectrophotometry	0.14	mg/dL	0.00 - 0.40
	CCT (UNCONJUGATED): SERUM	0.43	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PY	[/RIDOXAL PHOSPHATE	20.9	U/L	7.00 - 45.00
	RIDOXAL PHOSPHATE	15.2	U/L	0.00 - 49.00
AST/ALT RATIO: S by CALCULATED, SPE	ERUM ectrophotometry	1.38	RATIO	0.00 - 46.00
ALKALINE PHOSPI by para nitrophen propanol	HATASE: SERUM IYL PHOSPHATASE BY AMINO METHYL	80.94	U/L	40.0 - 130.0
GAMMA GLUTAMY by SZASZ, SPECTRO	L TRANSFERASE (GGT): SERUM PHTOMETRY	11.99	U/L	0.00 - 55.0
TOTAL PROTEINS: by BIURET, SPECTRO		6.47	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by bromocresol G		4.44	gm/dL	3.50 - 5.50
GLOBULIN: SERUN		2.03 ^L	gm/dL	2.30 - 3.50

Dr. Vinay Chopra

by CALCULATED, SPECTROPHOTOMETRY INTERPRETATION

A : G RATIO: SERUM

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:

by CALCULATED, SPECTROPHOTOMETRY

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)

2.19^H





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RATIO

1.00 - 2.00

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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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	KIDNE	Y FUNCTION T	EST (COMPLETE)		
UREA: SERUM		31.04	mg/dL	10.00 - 50.00	
	ATE DEHYDROGENASE (GLDH)	51.04	ilig/ uL	10.00 - 50.00	
CREATININE: SERU		1.09	mg/dL	0.40 - 1.20	
by ENZYMATIC, SPEC	COGEN (BUN): SERUM	14.5	mg/dL	7.0 - 25.0	
by CALCULATED, SPE		14.5	iiig/ uL	7.0 - 23.0	
	OGEN (BUN)/CREATININE	13.3	RATIO	10.0 - 20.0	
RATIO: SERUM by CALCULATED, SPE					
UREA/CREATININ		28.48	RATIO		
by CALCULATED, SPE					
URIC ACID: SERUM		5.36	mg/dL	2.50 - 6.80	
by URICASE - OXIDAS CALCIUM: SERUM	DE FERUXIDASE	10.05	mg/dL	8.50 - 10.60	
by ARSENAZO III, SPE	CTROPHOTOMETRY	10.00	ing, ui	0.00 10.00	
PHOSPHOROUS: SE		2.73	mg/dL	2.30 - 4.70	
ELECTROLYTES	DATE, SPECTROPHOTOMETRY				
SODIUM: SERUM		140.2	mmol/L	135.0 - 150.0	
by ISE (ION SELECTIV	'E ELECTRODE)	110.6	IIIII01/ L	100.0 - 100.0	
POTASSIUM: SERU		3.86	mmol/L	3.50 - 5.00	
by ISE (ION SELECTIVE ELECTRODE) CHLORIDE: SERUM		105.15	mmol/L	90.0 - 110.0	
by ISE (ION SELECTIV FSTIMATED CLOM	'E ELECTRODE) IERULAR FILTERATION RATE				
		69.7			
eGFR): SERUM by CALCULATED	ERULAR FILTERATION RATE	62.7			
INTERPRETATION.					

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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		Dr. Vinay Chopra MD (Pathology & Micr Chairman & Consultar	obiology)		u gam Cho MD (Pathol ultant Patholo	ogy)			
NAME	: Mrs. USHA								
AGE/ GENDER	: 48 YRS/FEM	AIF		PATIENT ID	· 175	22681			
	. 10 1100/11/201						~		
COLLECTED BY	:			REG. NO./LAB NO.		250113002			
REFERRED BY	:			REGISTRATION DA	TE : 13/	/Jan/2025 12:	:28 PM		
BARCODE NO.	:01523818			COLLECTION DATE	: 13/	/Jan/2025 12:	:29PM		
CLIENT CODE.	: KOS DIAGNO	STIC LAB		REPORTING DATE	: 13/	/Jan/2025 01:	:30PM		
CLIENT ADDRESS	: 6349/1, NIC	HOLSON ROAD, AMB	ALA CANTT						
Fest Name			Value	Unit	t	Biologie	cal Refer	ence inte	rval
 Reduced muscle m Certain drugs (e.g. INCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia PCREASED RATIO (<1 Acute tubular necr Low protein diet ar 	tetracycline, glu 0:1) WITH ELEV/ (BUN rises disp superimposed of 0:1) WITH DECR osis. Ind starvation.	creatinine productior acocorticoids) ATED CREATININE LEVE proportionately more to prin renal disease.	ELS:	ne) (e.g. obstructive (uropathy).				
 Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia Prerenal azotemia DECREASED RATIO (<1 Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome c Pregnancy. DECREASED RATIO (<1 Phenacimide thera Rabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido should produce an in Cephalosporin thera 	ass (subnormal tetracycline, glu 0:1) WITH ELEV/ (BUN rises disp superimposed of 0:1) WITH DECR osis. ad starvation. 2. creased urea sy urea rather tha monemias (urea of inappropiate a 0:1) WITH INCR py (accelerates eleases muscle who develop re sis (acetoacetat creased BUN/cr apy (interferes)	creatinine production accorticoids) ATED CREATININE LEVE proportionately more to on renal disease. EASED BUN : In thesis. In creatinine diffuses of a is virtually absent in antidiuretic harmone) EASED CREATININE: conversion of creatine creatinine). nal failure. e causes false increass eatinine ratio). with creatinine measu	ELS: Than creatini blood). due to tubu e to creatinin e in creatini	ellular fluid). lar secretion of urea. ne).		sulting in nori	mal ratio v	when dehy	ydratio
 Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia Perenal azotemia DECREASED RATIO (<1 Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome c Pregnancy. DECREASED RATIO (<1 Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin ther 	ass (subnormal tetracycline, glu 0:1) WITH ELEV/ (BUN rises disp superimposed of 0:1) WITH DECR osis. ad starvation. 2. creased urea sy urea rather tha monemias (urea of inappropiate a 0:1) WITH INCR py (accelerates eleases muscle who develop re sis (acetoacetat creased BUN/cr apy (interferes)	creatinine production accorticoids) ATED CREATININE LEVE proportionately more to on renal disease. EASED BUN : In thesis. In creatinine diffuses of a is virtually absent in antidiuretic harmone) EASED CREATININE: conversion of creatine creatinine). nal failure. e causes false increass eatinine ratio). with creatinine measu	ELS: Than creatini blood). due to tubu e to creatinin e in creatini rement).	ellular fluid). lar secretion of urea. ne). ne with certain methe	odologies,re:	sulting in nor	mal ratio v	when dehy	ydratio
Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (<1 Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome c Pregnancy. DECREASED RATIO (<1 Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in CEphalosporin ther STIMATED GLOMERL CKD STAGE	ass (subnormal tetracycline, glu 0:1) WITH ELEV/ (BUN rises disp superimposed of 0:1) WITH DECR osis. ad starvation. b. creased urea sy urea rather tha monemias (urea of inappropiate a 0:1) WITH INCR py (accelerates eleases muscle who develop re sis (acetoacetat creased BUN/cr apy (interferes ULAR FILTERATIO	creatinine production ucocorticoids) ATED CREATININE LEVE proportionately more to on renal disease. EASED BUN : In thesis. In creatinine diffuses of a is virtually absent in antidiuretic harmone) EASED CREATININE: conversion of creatine creatinine). nal failure. e causes false increass eatinine ratio). with creatinine measu N RATE: DESCRIPTION mal kidney function	ELS: Than creatini blood). due to tubu e to creatinin e in creatini rement).	ellular fluid). lar secretion of urea. ne).	odologies,re: ASSOCIATI	ED FINDINGS	mal ratio v	when dehy	ydratio
Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (<1 Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome c Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in CEphalosporin ther STIMATED GLOMERL CKD STAGE	ass (subnormal tetracycline, glu 0:1) WITH ELEV/ (BUN rises disp superimposed of 0:1) WITH DECR osis. ad starvation. creased urea sy urea rather tha monemias (urea of inappropiate a of inappropiate a of inappropiate a of inappropiate a finappropiate a sis (acetoacetat creased BUN/cr apy (interferes of DIAR FILTERATIO	creatinine production accorticoids) ATED CREATININE LEVE proportionately more to on renal disease. EASED BUN : In thesis. In creatinine diffuses of a is virtually absent in antidiuretic harmone) EASED CREATININE: conversion of creatine creatinine). nal failure. e causes false increas eatinine ratio). with creatinine measu N RATE: DESCRIPTION mal kidney function dney damage with	ELS: Than creatini blood). due to tubu e to creatinin e in creatini rement).	ellular fluid). lar secretion of urea. ne). ne with certain methe	odologies,re: ASSOCIATI	ED FINDINGS oteinuria of Protein ,	-	when dehy	ydratio
B. Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (<1 Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Diabetic disease Rabdomyolysis (r- CERASED RATIO (<1 Nenacimide thera Rabdomyolysis (r- CENTATED GLOMERL CENTATED GLOMERL G1 G2	ass (subnormal tetracycline, glu 0:1) WITH ELEV/ (BUN rises disp superimposed of 0:1) WITH DECR osis. ad starvation. 2. creased urea sy urea rather tha monemias (urea of inappropiate a of inappropiate a of inappropiate a finappropiate a of inappropiate a sis (acetoacetat creased BUN/cr apy (interferes of DLAR FILTERATIO	creatinine production accorticoids) ATED CREATININE LEVE proportionately more to on renal disease. EASED BUN : In thesis. In creatinine diffuses of a is virtually absent in antidiuretic harmone) EASED CREATININE: conversion of creatine creatinine). nal failure. e causes false increase eatinine ratio). with creatinine measu N RATE: DESCRIPTION mal kidney function dney damage with ormal or high GFR	ELS: Than creatini blood). due to tubu e to creatinin e in creatini rement).	ellular fluid). lar secretion of urea. ne). ne with certain metho <u>hL/min/1.73m2) >90 >90</u>	odologies,re: ASSOCIATI	ED FINDINGS	-	when dehy	ydratio
B. Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (<1 Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Diherited hyperam SIADH (syndrome c Rhabdomyolysis (r Rhabdomyolysis (r Diabetic ketoacido hould produce an in CEPhalosporin ther STIMATED GLOMERL G1 G2	ass (subnormal tetracycline, glu 0:1) WITH ELEV/ (BUN rises disp superimposed of 0:1) WITH DECR osis. ad starvation. creased urea sy urea rather tha monemias (urea of inappropiate a finappropiate a of inappropiate a finappropiate a finapp	creatinine production accorticoids) ATED CREATININE LEVE proportionately more to on renal disease. EASED BUN : In thesis. In creatinine diffuses of a is virtually absent in antidiuretic harmone) EASED CREATININE: conversion of creatine creatinine). nal failure. e causes false increase eatinine ratio). with creatinine measu N RATE: DESCRIPTION mal kidney function dney damage with ormal or high GFR ild decrease in GFR	ELS: than creatining but of extract blood). due to tubu e to creatining e in creatining rement). GFR (n	ellular fluid). lar secretion of urea. ne). ne with certain metho <u>hL/min/1.73m2) >90 >90 60 -89</u>	odologies,re: ASSOCIATI	ED FINDINGS oteinuria of Protein ,	-	when dehy	ydratio
A. Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Prerenal azotemia DECREASED RATIO (<1 Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome c Pregnancy. DECREASED RATIO (<1 Phenacimide thera Rhabdomyolysis (ro Muscular patients NAPPROPIATE RATIO Diabetic ketoacido should produce an in Cephalosporin ther ESTIMATED GLOMERL CKD STAGE G1 G2	ass (subnormal tetracycline, glu 0:1) WITH ELEV/ (BUN rises disp superimposed of 0:1) WITH DECR osis. Ind starvation. 2: creased urea sy urea rather tha monemias (urea f inappropiate a f inappropiate a of inappropiate a f inapp	creatinine production accorticoids) ATED CREATININE LEVE proportionately more to on renal disease. EASED BUN : In thesis. In creatinine diffuses of a is virtually absent in antidiuretic harmone) EASED CREATININE: conversion of creatine creatinine). nal failure. e causes false increase eatinine ratio). with creatinine measu N RATE: DESCRIPTION mal kidney function dney damage with ormal or high GFR	ELS: than creatining but of extract blood). due to tubu e to creatining e in creatining rement). GFR (n	ellular fluid). lar secretion of urea. ne). ne with certain metho <u>hL/min/1.73m2) >90 >90</u>	odologies,re: ASSOCIATI	ED FINDINGS oteinuria of Protein ,	-	when dehy	ydratio





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









	Dr. Vinay Chopra MD (Pathology & Microbiol Chairman & Consultant Pat	3, ,	(Pathology)
NAME	: Mrs. USHA		
AGE/ GENDER	: 48 YRS/FEMALE	PATIENT ID	: 1722681
COLLECTED BY	:	REG. NO./LAB NO.	: 012501130027
REFERRED BY	:	REGISTRATION DATE	: 13/Jan/2025 12:28 PM
BARCODE NO.	: 01523818	COLLECTION DATE	: 13/Jan/2025 12:29PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 13/Jan/2025 01:30PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA (CANTT	
Test Name	Val	ıe 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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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	AMBALA CANT	Т		
Test Name		Value	Unit	Biological Reference interval	
	IMM	UNOPATH	IOLOGY/SEROLOGY	Y	
		C-REACTIV	E PROTEIN (CRP)		
	EIN (CRP) QUANTITATIVE:	2.17	mg/L	0.0 - 6.0	

KOS Diagnostic Lab (A Unit of KOS Healthcare)

and the recovery being earlier than ESR. Unlike ESR, CRP levels are not influenced by hematologic conditions like Anemia, Polycythemia etc., 5. Elevated values are consistent with an acute inflammatory process. NOTE:

Elevated C-reactive protein (CRP) values are nonspecific and should not be interpreted without a complete clinical history.
 Oral contraceptives may increase CRP levels.





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	Dr. Vinay Cho MD (Pathology & Chairman & Cons					
NAME	: Mrs. USHA					
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CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE		: 13/Jan/2025 01:51PM		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT				
Test Name		Value	Unit	Biological Reference interval		
		CLINICAL PATH	HOLOGY			
	URINE ROI	UTINE & MICROSC	OPIC EXAMINA	ATION		
PHYSICAL EXAMIN	ATION					
QUANTITY RECIEV	ED TANCE SPECTROPHOTOMETRY	10	ml			
COLOUR		PALE YELLOW		PALE YELLOW		
TRANSPARANCY		HAZY		CLEAR		
SPECIFIC GRAVITY	TANCE SPECTROPHOTOMETRY	1.02		1.002 - 1.030		
CHEMICAL EXAMI						
REACTION		ACIDIC				
PROTEIN	TANCE SPECTROPHOTOMETRY	Trace		NEGATIVE (-ve)		
SUGAR	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)		
pH	TANCE SPECTROPHOTOMETRY	6.5		5.0 - 7.5		
BILIRUBIN	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)		
NITRITE	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)		
UROBILINOGEN	TANCE SPECTROPHOTOMETRY.	Normal	EU/dL	0.2 - 1.0		
KETONE BODIES	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)		
BLOOD	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)		
ASCORBIC ACID by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY TANCE SPECTROPHOTOMETRY	NEGATIVE (-ve)		NEGATIVE (-ve)		
MICROSCOPIC EXA	(RBCs)	NEGATIVE (-ve)	/HPF	0 - 3		

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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	Biological Reference interval

PUS CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	4-6	/HPF	0 - 5
EPITHELIAL CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	6-8	/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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