



	Dr. Vinay Chopra MD (Pathology & Micr Chairman & Consultar	obiology)		(Pathology)
NAME	: Mrs. MONA AHUJA			
AGE/ GENDER	: 43 YRS/FEMALE		PATIENT ID	: 1769389
COLLECTED BY	:		REG. NO./LAB NO.	: 012502250008
REFERRED BY	: CENTRAL PHOENIX CLUB (AMBAI	LA CANTT)	REGISTRATION DATE	: 25/Feb/2025 08:41 AM
BARCODE NO.	:01526102		COLLECTION DATE	: 25/Feb/2025 08:42AM
CLIENT CODE. CLIENT ADDRESS	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 25/Feb/2025 09:57AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMB/	ALA CANTI		
Test Name		Value	Unit	Biological Reference interval
	COMP		LLNESS PANEL: 1.0 OOD COUNT (CBC)	0
	(RBCS) COUNT AND INDICES	10	()	
HAEMOGLOBIN (H)	В)	13	gm/dL	12.0 - 16.0
RED BLOOD CELL (RBC) COUNT OCUSING, ELECTRICAL IMPEDENCE	4.6	Millions/	/cmm 3.50 - 5.00
PACKED CELL VOLU		39.8	%	37.0 - 50.0
by CALCULATED BY A MEAN CORPUSCUL	UTOMATED HEMATOLOGY ANALYZER	86.6	fL	80.0 - 100.0
by CALCULATED BY A	UTOMATED HEMATOLOGY ANALYZER		IL	
	AR HAEMOGLOBIN (MCH) UTOMATED HEMATOLOGY ANALYZER	28.3	pg	27.0 - 34.0
	AR HEMOGLOBIN CONC. (MCHC) UTOMATED HEMATOLOGY ANALYZER	32.7	g/dL	32.0 - 36.0
	UTION WIDTH (RDW-CV) utomated hematology analyzer	14.1	%	11.00 - 16.00
RED CELL DISTRIB	UTION WIDTH (RDW-SD) UTOMATED HEMATOLOGY ANALYZER	46	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED		18.83	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INE by CALCULATED		26.58	RATIO	BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CE				
TOTAL LEUCOCYTE	COUNT (TLC) y by sf cube & microscopy	6450	/cmm	4000 - 11000
NUCLEATED RED B	LOOD CELLS (nRBCS)	NIL		0.00 - 20.00
NUCLEATED RED B	LOOD CELLS (nRBCS) % UTOMATED HEMATOLOGY ANALYZER	NIL	%	< 10 %



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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





Dr. Yugam Chopra

CEO & Consultant Pathologist

MD (Pathology)

NAME : Mrs. MONA AHUJA AGE/ GENDER : 43 YRS/FEMALE **PATIENT ID** :1769389 **COLLECTED BY** :012502250008 REG. NO./LAB NO. **REFERRED BY** : CENTRAL PHOENIX CLUB (AMBALA CANTT) **REGISTRATION DATE** : 25/Feb/2025 08:41 AM **BARCODE NO.** :01526102 **COLLECTION DATE** : 25/Feb/2025 08:42AM CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE** : 25/Feb/2025 09:57AM **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Unit **Biological Reference interval DIFFERENTIAL LEUCOCYTE COUNT (DLC) NEUTROPHILS** 52% 50 - 70 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY LYMPHOCYTES 36 % 20 - 40 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ЯH EOSINOPHILS % 1 - 6 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY MONOCYTES 4 % 2 - 12by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY BASOPHILS 0 % 0 - 1 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY **ABSOLUTE LEUKOCYTES (WBC) COUNT** ABSOLUTE NEUTROPHIL COUNT 3354 2000 - 7500 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE LYMPHOCYTE COUNT 2322 800 - 4900 /cmm by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE EOSINOPHIL COUNT 516^H /cmm 40 - 440 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE MONOCYTE COUNT 258 /cmm 80 - 880 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE BASOPHIL COUNT 0 /cmm 0 - 110 by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY PLATELETS AND OTHER PLATELET PREDICTIVE MARKERS. PLATELET COUNT (PLT) 150000 - 450000 270000 /cmm by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELETCRIT (PCT) 0.34 % 0.10 - 0.36 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE MEAN PLATELET VOLUME (MPV) 13^H fL 6.50 - 12.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE 119000^H 30000 - 90000 PLATELET LARGE CELL COUNT (P-LCC) /cmm by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE % PLATELET LARGE CELL RATIO (P-LCR) 44 11.0 - 45.0 by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET DISTRIBUTION WIDTH (PDW) 15.0 - 17.0 15.9%

Dr. Vinay Chopra

MD (Pathology & Microbiology)

Chairman & Consultant Pathologist

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



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





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BARCODE NO.	:01526102		COLLECTION DATE	: 25/Feb/2025 08:42AM
CLIENT CODE.	: KOS DIAGNOSTIC LAP	3	REPORTING DATE	: 25/Feb/2025 11:24AM
CLIENT ADDRESS	: 6349/1, NICHOLSON	ROAD, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
by RED CELL AGGRE	DIMENTATION RATE (E gation by capillary pho	CSR) 17 Tometry	MENTATION RATE (mm/1st	





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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	CLINICA	AL CHEMISTRY	/BIOCHEMIST	RY
		GLUCOSE FAS	TING (F)	

KOS Diagnostic Lab (A Unit of KOS Healthcare)

A fasting plasma glucose level below 100 mg/dl is considered normal.
 A fasting plasma glucose level between 100 - 125 mg/dl is considered as glucose intolerant or prediabetic. A fasting and post-prandial blood test (after consumption of 75 gms of glucose) is recommended for all such patients.
 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.



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		hopra & Microbiology) nsultant Pathologist			
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Test Name		Value	Unit	Biological Reference interval	
		LIPID PROFII	F · BASIC		
CHOLESTEROL TOT by CHOLESTEROL OX		182.83	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0	
TRIGLYCERIDES: SI by GLYCEROL PHOSP	ERUM HATE OXIDASE (ENZYMATIC)	72.13	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0	
HDL CHOLESTEROI by SELECTIVE INHIBITI		60.71	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0	
LDL CHOLESTEROL by CALCULATED, SPE		107.69	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129. BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0	
NON HDL CHOLEST by CALCULATED, SPE		122.12	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159. BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0	
VLDL CHOLESTERC by CALCULATED, SPE	CTROPHOTOMETRY	14.43	mg/dL	0.00 - 45.00	
TOTAL LIPIDS: SER by CALCULATED, SPE		437.79	mg/dL	350.00 - 700.00	
CHOLESTEROL/HD by CALCULATED, SPE		3.01	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0	



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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT				
Test Name		Value	Unit	Biological Reference interval	
LDL/HDL RATIO: S		1.77	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	1.19 ^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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U/L

40.0 - 130.0

0.00 - 55.0

6.20 - 8.00

3.50 - 5.50

2.30 - 3.50

1.00 - 2.00

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Test Name		Value	Unit	Biological Reference interval
	LIVER	E FUNCTIO	N TEST (COMPLETE)	
BILIRUBIN TOTAL: SERUM by DIAZOTIZATION, SPECTROPHOTOMETRY		0.61	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
	C (CONJUGATED): SERUM	0.11	mg/dL	0.00 - 0.40
BILIRUBIN INDIRE	CT (UNCONJUGATED): SERUM	0.5	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PY	RIDOXAL PHOSPHATE	20.5	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PY	RIDOXAL PHOSPHATE	18.8	U/L	0.00 - 49.00
AST/ALT RATIO: S		1.09	RATIO	0.00 - 46.00

GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	25.92	U/L
TOTAL PROTEINS: SERUM by biuret, spectrophotometry	6.38	gm/dL
ALBUMIN: SERUM by BROMOCRESOL GREEN	4.26	gm/dL
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.12 ^L	gm/dL
A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.01 ^H	RATIO
ΙΝΤΕΦΦΕΤΛΤΙΩΝ		

INTERPRETATION

ALKALINE PHOSPHATASE: SERUM

by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL

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:

PROPANOL

> 2
> 2 (Highly Suggestive)
1.4 - 2.0
> 1.5
> 1.3 (Slightly Increased)

68.21





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Test Name	Value	Unit	Biological Reference interval

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).

PROGNOSTIC SIGNIFICANCE:

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



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	KIDNI	EY FUNCTIO)N TEST (COMPLETE)	
UREA: SERUM		23.85	mg/dL	10.00 - 50.00
by UREASE - GLUTAN	NATE DEHYDROGENASE (GLDH)		Ũ	
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETERY		0.89	mg/dL	0.40 - 1.20
BLOOD UREA NITE	ROGEN (BUN): SERUM	11.14	mg/dL	7.0 - 25.0
by CALCULATED, SPECTROPHOTOMETRY BLOOD UREA NITROGEN (BUN)/CREATININE		12.52	RATIO	10.0 - 20.0
RATIO: SERUM		12.02		10.0 20.0
by CALCULATED, SPE		00.0	DATIO	
UREA/CREATININ		26.8	RATIO	
URIC ACID: SERUM		2.52	mg/dL	2.50 - 6.80
by URICASE - OXIDAS	SE PEROXIDASE	0.01		0.50, 10.00
CALCIUM: SERUM by ARSENAZO III, SPE	CTROPHOTOMETRY	9.01	mg/dL	8.50 - 10.60
PHOSPHOROUS: SE		3.62	mg/dL	2.30 - 4.70
-	DATE, SPECTROPHOTOMETRY		J. J	
ELECTROLYTES		100		
SODIUM: SERUM by ISE (ION SELECTIV	/E ELECTRODE)	138	mmol/L	135.0 - 150.0
POTASSIUM: SERU		4.07	mmol/L	3.50 - 5.00
by ISE (ION SELECTIV				
CHLORIDE: SERUM		103.5	mmol/L	90.0 - 110.0
, ,	IERULAR FILTERATION RATE			
ESTIMATED GLOM (eGFR): SERUM by CALCULATED INTERPRETATION:	ERULAR FILTERATION RATE	82.4		

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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Fest Name		Value	Unit	Biologic	al Reference inter	val
. Certain drugs (e.g. NCREASED RATIO (>2 . Postrenal azotemia	ass (subnormal creatinine produc tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE (BUN rises disproportionately mosed on ronal disease	LEVELS:	ctive uropath	y).		
Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet ar Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin ther STADE STADE STAGE	tetracycline, glucocorticoids) i0:1) WITH ELEVATED CREATININE a (BUN rises disproportionately me superimposed on renal disease. i0:1) WITH DECREASED BUN : osis. a starvation. b. creased urea synthesis. urea rather than creatinine diffu: monemias (urea is virtually abser- of inappropiate antidiuretic harmon i0:1) WITH INCREASED CREATININ py (accelerates conversion of crea- eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false inco- creased BUN/creatinine ratio). apy (interferes with creatinine me JLAR FILTERATION RATE: DESCRIPTION	LEVELS: ore than creatinine) (e.g. obstrue ses out of extracellular fluid). nt in blood). one) due to tubular secretion of E: atine to creatinine). crease in creatinine with certain easurement).	urea. methodologi) ASSO	es,resulting in norm	nal ratio when dehy	dratio
. Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia ECREASED RATIO (< Acute tubular necr Low protein diet ar Severe liver diseas Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome of Pregnancy. ECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin thei STIMATED GLOMERU CKD STAGE	tetracycline, glucocorticoids) i0:1) WITH ELEVATED CREATININE a (BUN rises disproportionately me superimposed on renal disease. i0:1) WITH DECREASED BUN : osis. a starvation. b. creased urea synthesis. urea rather than creatinine diffu: monemias (urea is virtually abser- of inappropiate antidiuretic harmon i0:1) WITH INCREASED CREATININ py (accelerates conversion of crea- eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false inco- creased BUN/creatinine ratio). apy (interferes with creatinine me JLAR FILTERATION RATE: DESCRIPTION Normal kidney functione Normal kidney functione	LEVELS: ore than creatinine) (e.g. obstruines) ses out of extracellular fluid). nt in blood). one) due to tubular secretion of E: atine to creatinine). crease in creatinine with certain easurement).	urea. methodologi) ASSO	es,resulting in norm CIATED FINDINGS o proteinuria	nal ratio when dehy	dratio
. Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia ECREASED RATIO (< Acute tubular necr Low protein diet ar Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. ECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin ther STIMATED GLOMERI CKD STAGE	tetracycline, glucocorticoids) i0:1) WITH ELEVATED CREATININE a (BUN rises disproportionately me superimposed on renal disease. i0:1) WITH DECREASED BUN : osis. a starvation. b. creased urea synthesis. urea rather than creatinine diffu: monemias (urea is virtually abser- of inappropiate antidiuretic harmon i0:1) WITH INCREASED CREATININ py (accelerates conversion of crea- eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false inco- creased BUN/creatinine ratio). apy (interferes with creatinine me <u>JLAR FILTERATION RATE:</u> <u>DESCRIPTION</u> Normal kidney function Kidney damage with	LEVELS: ore than creatinine) (e.g. obstruines) ses out of extracellular fluid). nt in blood). one) due to tubular secretion of E: atine to creatinine). crease in creatinine with certain easurement).	urea. methodologi) ASSO N Pres	es,resulting in norm CIATED FINDINGS o proteinuria ence of Protein ,	nal ratio when dehy	dratio
Certain drugs (e.g. JCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia ECREASED RATIO (< Acute tubular necr Low protein diet an Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. ECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients JAPPROPIATE RATIO Diabetic ketoacido nould produce an in Cephalosporin ther STIMATED GLOMERI G1 G2	tetracycline, glucocorticoids) i0:1) WITH ELEVATED CREATININE a (BUN rises disproportionately me superimposed on renal disease. i0:1) WITH DECREASED BUN : osis. a starvation. b. creased urea synthesis. urea rather than creatinine diffu: monemias (urea is virtually abser- of inappropiate antidiuretic harmon i0:1) WITH INCREASED CREATININ py (accelerates conversion of crea- eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false inco- creased BUN/creatinine ratio). apy (interferes with creatinine me <u>JLAR FILTERATION RATE:</u> <u>DESCRIPTION</u> Normal kidney function Kidney damage with normal or high GFF	LEVELS: ore than creatinine) (e.g. obstruents) ses out of extracellular fluid). nt in blood). one) due to tubular secretion of E: atine to creatinine). crease in creatinine with certain easurement).	urea. methodologi) ASSO N Pres	es,resulting in norm CIATED FINDINGS o proteinuria	nal ratio when dehy	dratio
Certain drugs (e.g. JCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia ECREASED RATIO (< Acute tubular necr Low protein diet an Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. ECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients JAPPROPIATE RATIO Diabetic ketoacido nould produce an in Cephalosporin ther STIMATED GLOMERI G1 G2 G3a	tetracycline, glucocorticoids) i0:1) WITH ELEVATED CREATININE a (BUN rises disproportionately me superimposed on renal disease. i0:1) WITH DECREASED BUN : osis. a starvation. b. creased urea synthesis. urea rather than creatinine diffu: monemias (urea is virtually abser- of inappropiate antidiuretic harmon i0:1) WITH INCREASED CREATININ py (accelerates conversion of crea- eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false inco- creased BUN/creatinine ratio). apy (interferes with creatinine me <u>JLAR FILTERATION RATE:</u> <u>DESCRIPTION</u> Normal kidney function Kidney damage with	LEVELS: ore than creatinine) (e.g. obstruents) ses out of extracellular fluid). nt in blood). one) due to tubular secretion of E: atine to creatinine). crease in creatinine with certain easurement). ion >90 h >90 R 60 - 89	urea. methodologi) ASSO N Pres	es,resulting in norm CIATED FINDINGS o proteinuria ence of Protein ,	nal ratio when dehy	dratio
. Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia ECREASED RATIO (< Acute tubular necr Low protein diet an Severe liver diseas Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome of Pregnancy. ECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin ther STIMATED GLOMERI G1 G2	tetracycline, glucocorticoids) i0:1) WITH ELEVATED CREATININE a (BUN rises disproportionately me superimposed on renal disease. i0:1) WITH DECREASED BUN : osis. a starvation. e. creased urea synthesis. urea rather than creatinine diffu: monemias (urea is virtually abser- of inappropiate antidiuretic harmon i0:1) WITH INCREASED CREATININ py (accelerates conversion of crea- eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false inco- creased BUN/creatinine ratio). apy (interferes with creatinine me <u>JLAR FILTERATION RATE:</u> <u>DESCRIPTION</u> Normal kidney function Kidney damage with normal or high GFF Mild decrease in GF	LEVELS: ore than creatinine) (e.g. obstruents) ses out of extracellular fluid). nt in blood). one) due to tubular secretion of E: atine to creatinine). crease in creatinine with certain easurement). ion >90 h >90 R 60 - 89 GFR 30-59	urea. methodologi) ASSO N Pres	es,resulting in norm CIATED FINDINGS o proteinuria ence of Protein ,	nal ratio when dehy	dratio





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









	Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologi		(Pathology)
NAME	: Mrs. MONA AHUJA		
AGE/ GENDER	: 43 YRS/FEMALE	PATIENT ID	: 1769389
COLLECTED BY	:	REG. NO./LAB NO.	: 012502250008
REFERRED BY	: CENTRAL PHOENIX CLUB (AMBALA CANTT)	REGISTRATION DATE	: 25/Feb/2025 08:41 AM
BARCODE NO.	: 01526102	COLLECTION DATE	: 25/Feb/2025 08:42AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 25/Feb/2025 10:50AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT	ſ	
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.YUGAM CHOPRA CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY)







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Test Name		Value	Unit	Biological Reference interval		
		CLINICAL PATH	OLOCY			
	UDINE DO	UTINE & MICROSCO		TION		
PHYSICAL EXAMIN		UTINE & MICKUSCU	PIC EAAMINA	ATION		
QUANTITY RECIEVE		10	ml			
by DIP STICK/REFLECT	ANCE SPECTROPHOTOMETRY					
COLOUR by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY		PALE YELLOW		PALE YELLOW		
TRANSPARANCY		HAZY		CLEAR		
by DIP STICK/REFLECT. SPECIFIC GRAVITY	ANCE SPECTROPHOTOMETRY	1.01		1.002 - 1.030		
by DIP STICK/REFLECT	ANCE SPECTROPHOTOMETRY			1002 1000		
CHEMICAL EXAMIN	ATION	ACIDIC				
REACTION by DIP STICK/REFLECT	ANCE SPECTROPHOTOMETRY	ACIDIC				
PROTEIN	ANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)		
SUGAR	ANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)		
	ANCE SPECTROPHOTOMETRY	<=5.0		50 75		
pH by DIP STICK/REFLECT	ANCE SPECTROPHOTOMETRY	<=5.0		5.0 - 7.5		
BILIRUBIN	ANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)		
NITRITE		Negative		NEGATIVE (-ve)		
by DIP STICK/REFLECT. UROBILINOGEN	ANCE SPECTROPHOTOMETRY.	Normal	EU/dL	0.2 - 1.0		
• - • • • • • •	ANCE SPECTROPHOTOMETRY		E0/ uL			
KETONE BODIES by DIP STICK/REFLECT	ANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)		
BLOOD		Negative		NEGATIVE (-ve)		
by DIP STICK/REFLECT. ASCORBIC ACID	ANCE SPECTROPHOTOMETRY	NEGATIVE (-ve)		NEGATIVE (-ve)		
by DIP STICK/REFLECT	ANCE SPECTROPHOTOMETRY					
MICROSCOPIC EXA						
RED BLOOD CELLS ((RBCs)	NEGATIVE (-ve)	/HPF	0 - 3		





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

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by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
PUS CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	4-6	/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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V DR.YUGAM CHOPRA CONSULTANT PATHOLOGIST MBBS , MD (PATHOLOGY)

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