

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



	<b>Dr. Vinay Chopra</b> MD (Pathology & Micr Chairman & Consultar	obiology)		) (Pathology)
NAME	: Miss. ANJALI			
AGE/ GENDER	: 24 YRS/FEMALE		PATIENT ID	: 1729923
<b>COLLECTED BY</b>	:		<b>REG. NO./LAB NO.</b>	:012501210033
<b>REFERRED BY</b>	:		<b>REGISTRATION DATE</b>	: 21/Jan/2025 12:31 PM
BARCODE NO.	: 01524193		COLLECTION DATE	: 21/Jan/2025 12:33PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 21/Jan/2025 01:19PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMB	ALA CAN I	1	
Test Name		Value	Unit	<b>Biological Reference interval</b>
	SWAST	HYA WI	ELLNESS PANEL: 1.(	0
	COMP	PLETE BI	LOOD COUNT (CBC)	
RED BLOOD CELL	<u>S (RBCS) COUNT AND INDICES</u>			
HAEMOGLOBIN (H	IB)	9.6 <sup>L</sup>	gm/dL	12.0 - 16.0
RED BLOOD CELL	(RBC) COUNT FOCUSING, ELECTRICAL IMPEDENCE	4.36	Millions/	3.50 - 5.00
PACKED CELL VOL		31.5 <sup>L</sup>	%	37.0 - 50.0
	AR VOLUME (MCV) automated hematology analyzer	72.2 <sup>L</sup>	fL	80.0 - 100.0
	LAR HAEMOGLOBIN (MCH) AUTOMATED HEMATOLOGY ANALYZER	22.1 <sup>L</sup>	pg	27.0 - 34.0
	LAR HEMOGLOBIN CONC. (MCHC) AUTOMATED HEMATOLOGY ANALYZER	30.6 <sup>L</sup>	g/dL	32.0 - 36.0
	BUTION WIDTH (RDW-CV) AUTOMATED HEMATOLOGY ANALYZER	15.9	%	11.00 - 16.00
	BUTION WIDTH (RDW-SD) AUTOMATED HEMATOLOGY ANALYZER	43.2	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED		16.56	RATIO	BETA THALASSEMIA TRAIT: 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INI by CALCULATED WHITE BLOOD CE		26.43	RATIO	BETA THALASSEMIA TRAIT: 65.0 IRON DEFICIENCY ANEMIA: 65.0
TOTAL LEUCOCYT		4280	/cmm	4000 - 11000
NUCLEATED RED I	BLOOD CELLS (nRBCS) RT HEMATOLOGY ANALYZER	NIL		0.00 - 20.00
NUCLEATED RED I	BLOOD CELLS (nRBCS) % AUTOMATED HEMATOLOGY ANALYZER	NIL	%	< 10 %



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

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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>
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS	64	%	50 - 70
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	24	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	10	%	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	2739	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by flow cytometry by sf cube & microscopy	1027	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	86 <sup>L</sup>	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	428	/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	308000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.34	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	11	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	113000 <sup>H</sup>	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	36.8	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by hydro dynamic focusing, electrical impedence NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD	16.5	%	15.0 - 17.0



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Test Name	Value	Unit	Biological Reference interval



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LIENT CODE.	: KOS DIAGNOSTIC LAB		ORTING DATE	: 21/Jan/2025 01:44PM	
LIENT ADDRESS	: 6349/1, NICHOLSON ROAD	D, AMBALA CANTT			
Fest Name		Value	Unit	<b>Biological Reference interval</b>	
. ESR is a non-speci nmune disease, but . An ESR can be affe s C-reactive protein . This test may also ystemic lupus eryth	does not tell the health practit ected by other conditions beside be used to monitor disease act ematosus	ioner exactly where the es inflammation. For this	inflammation is in the reason, the ESR is typ	e body or what is causing it.	
mmune disease, but 2. An ESR can be affe is C-reactive protein 3. This test may also ystemic lupus eryth <b>CONDITION WITH LO</b> A low ESR can be see polycythaemia), sign is sickle cells in sick <b>JOTE:</b> . ESR and C - reactive 2. Generally, ESR dod 6. <b>CRP is not affected</b> 4. If the ESR is elevat	does not tell the health practit ected by other conditions beside be used to monitor disease act ematosus <b>W ESR</b> In with conditions that inhibit the hificantly high white blood cell le cell anaemia) also lower the eprotein (C-RP) are both marked es not change as rapidly as does led, it is typically a result of two	ioner exactly where the es inflammation. For this ivity and response to the he normal sedimentation count (leucocytosis), an ESR. ers of inflammation. s CRP, either at the start <b>ESR, making it a better m</b> b types of proteins, alobu	inflammation is in the reason, the ESR is typ erapy in both of the a n of red blood cells, su d some protein abno of inflammation or as <b>arker of inflammatior</b> lins or fibrinogen.	bicallý 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 (such s it resolves. <b>1.</b>	
. ESR is a non-specifi mmune disease, but 2. An ESR can be affet is C-reactive protein 3. This test may also (SONDITION WITH LO VIOW ESR can be see polycythaemia), sign is sickle cells in sick IOTE: . ESR and C - reactive 3. CRP is not affected b. If the ESR is elevat 5. Women tend to ha b. Drugs such as dex	does not tell the health practit acted by other conditions beside be used to monitor disease act ematosus <b>W ESR</b> In with conditions that inhibit the hificantly high white blood cell le cell anaemia) also lower the e protein (C-RP) are both marke es not change as rapidly as does I by as many other factors as is is ed, it is typically a result of two and higher ESR, and menstruat	ioner exactly where the es inflammation. For this ivity and response to the he normal sedimentation count (leucocytosis), an ESR. ers of inflammation. s CRP, either at the start <b>ESR, making it a better m</b> o types of proteins, globution and pregnancy can c	inflammation is in the reason, the ESR is typ erapy in both of the a n of red blood cells, su d some protein abno of inflammation or as <b>arker of inflammatior</b> llins or fibrinogen. ause temporary eleva	e body or what is causing it. bically 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 (such s it resolves.	





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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	, AMBALA CANTT		
Test Name		Value	Unit	<b>Biological Reference interval</b>
	CLINI	CAL CHEMISTRY	/BIOCHEMIST	'RY
		<b>GLUCOSE FAS</b>	TING (F)	

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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		<b>Chopra</b> gy & Microbiology) Consultant Pathologist	Dr. Yugam MD CEO & Consultant	(Pathology)
NAME AGE/ GENDER COLLECTED BY REFERRED BY BARCODE NO. CLIENT CODE. CLIENT ADDRESS	: Miss. ANJALI : 24 YRS/FEMALE : : : 01524193 : KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON ROA	R R C R	ATIENT ID EG. NO./LAB NO. EGISTRATION DATE OLLECTION DATE EPORTING DATE	: 1729923 <b>: 012501210033</b> : 21/Jan/2025 12:31 PM : 21/Jan/2025 12:33PM : 21/Jan/2025 03:06PM
Test Name		Value	Unit	Biological Reference interval
		LIPID PROI	FILE : BASIC	
CHOLESTEROL TOT by CHOLESTEROL OX		158.8	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SI by GLYCEROL PHOSPI	ERUM HATE OXIDASE (ENZYMATIC)	47.69	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	L (DIRECT): SERUM ON	51.21	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL by CALCULATED, SPE		98.05	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		107.59	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 CHOLESTERO	CTROPHOTOMETRY	9.54	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SER by CALCULATED, SPEC CHOLESTEROL/HD by CALCULATED, SPEC	<i>сткорнотометку</i> L RATIO: SERUM	365.29 3.1	mg/dL RATIO	350.00 - 700.00 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 by CALCULATED, SPE		1.91	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/H by CALCULATED, SPE		0.93 <sup>L</sup>	RATIO	3.00 - 5.00

## INTERPRETATION:

1. Measurements in the same patient can show physiological analytical variations. Three serial samples 1 week apart are recommended for Total Cholesterol, Triglycerides, HDL & LDL Cholesterol.

2. As per NLA-2014 guidelines, all adults above the age of 20 years should be screened for lipid status. Selective screening of children above the age of 2 years with a family history of premature cardiovascular disease or those with at least one parent with high total cholesterol is recommended.

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

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





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MD (Pathology & Microbiology)

Chairman & Consultant Pathologist

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

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Test Name	Value	Unit	<b>Biological Reference interval</b>
LIVER	FUNCTION TE	ST (COMPLETE)	
BILIRUBIN TOTAL: SERUM by DIAZOTIZATION, SPECTROPHOTOMETRY	0.29	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.1	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.19	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	18.6	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	12.8	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by calculated, spectrophotometry	1.45	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL	70.55	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	14.45	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	8.22 <sup>H</sup>	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	4.38	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	3.84 <sup>H</sup>	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by calculated, spectrophotometry	1.14	RATIO	1.00 - 2.00

## 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
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)





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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 FUNCTIO	)N TEST (COMPLETE)		
UREA: SERUM		18.16	mg/dL	10.00 - 50.00	
	ATE DEHYDROGENASE (GLDH)	10.10	ilig/ dL	10.00 30.00	
CREATININE: SERU		0.86	mg/dL	0.40 - 1.20	
by ENZYMATIC, SPEC		8.49	mg/dL	7.0 - 25.0	
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY		0.45	iiig/ uL	7.0 - 23.0	
BLOOD UREA NITROGEN (BUN)/CREATININE		9.87 <sup>L</sup>	RATIO	10.0 - 20.0	
RATIO: SERUM					
by CALCULATED, SPE UREA/CREATININ		21.12	RATIO		
by CALCULATED, SPE		61.16	RATIO		
URIC ACID: SERUM		4.05	mg/dL	2.50 - 6.80	
by URICASE - OXIDAS CALCIUM: SERUM	E PEROXIDASE	9.26	mg/dL	8.50 - 10.60	
by ARSENAZO III, SPE	CTROPHOTOMETRY	9.20	ilig/ uL	8.50 - 10.00	
PHOSPHOROUS: SE		3.19	mg/dL	2.30 - 4.70	
-	DATE, SPECTROPHOTOMETRY				
ELECTROLYTES					
SODIUM: SERUM by ISE (ION SELECTIV	(F ELECTRODE)	142.3	mmol/L	135.0 - 150.0	
POTASSIUM: SERU		4.75	mmol/L	3.50 - 5.00	
by ISE (ION SELECTIV	(E ELECTRODE)				
CHLORIDE: SERUM		106.73	mmol/L	90.0 - 110.0	
	<b>IERULAR FILTERATION RATE</b>				
	ERULAR FILTERATION RATE	96.7			
(eGFR): SERUM		0.011			
by CALCULATED					
INTERPRETATION:	icon pro, and post ronal azatamia				

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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	1	Dr. Vinay Chopra MD (Pathology & Micr Chairman & Consultar	obiology)		<b>Yugam Ch</b> MD (Path nsultant Path	iology)			
IAME	: Miss. ANJAL	[							
AGE/ GENDER	: 24 YRS/FEM	LE		PATIENT ID	:1	729923			
COLLECTED BY	:			REG. NO./LAB NO.	. :(	)125012100	33		
REFERRED BY				REGISTRATION D	ATE · 🤅	21/Jan/2025 1	2:31 PM		
BARCODE NO.	:01524193			COLLECTION DAT		21/Jan/2025 1			
CLIENT CODE.	: KOS DIAGNO	STIC I AR		REPORTING DATI		21/Jan/2025 0			
				KEI OKIING DAII		17 Jail/ 2023 0	5.001 1		
CLIENT ADDRESS	: 0349/1, MICI	IOLSON ROAD, AMB	ALA CANT I						
Test Name			Value	Un	it	Biolog	gical Refe	erence int	erval
8. Reduced muscle m 9. Certain drugs (e.g. I <b>NCREASED RATIO (&gt;2</b>	tetracycline, glu <b>0:1) WITH ELEVA</b> (BUN rises displ superimposed o	creatinine production cocorticoids) <b>TED CREATININE LEVE</b> roportionately more t n renal disease.	LS:	ne) (e.g. obstructive	e uropathy).				
<ol> <li>Reduced muscle m Certain drugs (e.g. INCREASED RATIO (&gt;2 1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (&lt; 1. Acute tubular necr 2. Low protein diet ar 3. Severe liver disease 4. Other causes of de 5. Repeated dialysis ( 6. Inherited hyperam 7. SIADH (syndrome c 8. Pregnancy. DECREASED RATIO (</li> <li>Inherited hyperam 7. SIADH (syndrome c 8. Pregnancy. DECREASED RATIO (</li> <li>Inherited hyperam 7. SIADH (syndrome c 8. Pregnancy. DECREASED RATIO (</li> <li>Inbenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERL CKD STAGE G1     </li> </ol>	ass (subnormal of tetracycline, glu 0:1) WITH ELEVA (BUN rises dispi- superimposed of 0:1) WITH DECRI osis. Ind starvation. 2: creased urea syr urea rather thar monemias (urea of inappropiate a 0:1) WITH INCRE py (accelerates of eleases muscle of who develop rer sis (acetoacetate creased BUN/crea apy (interferes v ULAR FILTERATION	creatinine production cocorticoids) <b>TED CREATININE LEVE</b> roportionately more to n renal disease. <b>EASED BUN :</b> thesis. creatinine diffuses of is virtually absent in ntidiuretic harmone) <b>ASED CREATININE:</b> conversion of creatine reatinine). hal failure. e causes false increas eatinine ratio). vith creatinine measu <b>V RATE:</b> <b>DESCRIPTION</b> mal kidney function	LS: han creatinin ut of extrace blood). due to tubul to creatinin e in creatinin rement).	ellular fluid). ar secretion of urea ne). he with certain met uL/min/1.73m2 ) >90	hodologies, <b>ASSOCI</b> No	ATED FINDINGS	S	o when deł	nydrati
<ol> <li>Reduced muscle mu</li></ol>	ass (subnormal of tetracycline, glu 0:1) WITH ELEVA (BUN rises dispi- superimposed of 0:1) WITH DECRI osis. Ind starvation. 2. creased urea syr urea rather thar monemias (urea of inappropiate a 0:1) WITH INCRE py (accelerates of eleases muscle of who develop rer : sis (acetoacetate creased BUN/crea apy (interferes v ULAR FILTERATION Nor	creatinine production cocorticoids) <b>TED CREATININE LEVE</b> roportionately more to n renal disease. <b>EASED BUN :</b> thesis. creatinine diffuses of is virtually absent in ntidiuretic harmone) <b>ASED CREATININE:</b> conversion of creatine reatinine). hal failure. e causes false increas eatinine ratio). vith creatinine measu <b>V RATE:</b> DESCRIPTION mal kidney function dney damage with	LS: han creatinin ut of extrace blood). due to tubul to creatinin e in creatinin rement).	ellular fluid). ar secretion of urea ne). ne with certain met	hodologies, ASSOCI No Presen	ATED FINDING proteinuria ce of Protein ,	S	o when deł	nydrati
A. Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis ( Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (       B. Pregnancy. DECREASED RATIO (       B. Pregnancy. DECREASED RATIO (       B. Phenacimide thera Rhabdomyolysis (r Sidde tetoacido should produce an in Cephalosporin ther ESTIMATED GLOMERL G1 G2	ass (subnormal of tetracycline, glu 0:1) WITH ELEVA (BUN rises dispi- superimposed of 0:1) WITH DECRI osis. ad starvation. b: creased urea syr urea rather thar monemias (urea of inappropiate a 0:1) WITH INCRE py (accelerates of eleases muscle of who develop rer sis (acetoacetate creased BUN/crea apy (interferes v ILAR FILTERATION Nor King	creatinine production cocorticoids) <b>TED CREATININE LEVE</b> roportionately more to n renal disease. <b>EASED BUN :</b> thesis. creatinine diffuses of is virtually absent in ntidiuretic harmone) <b>ASED CREATININE:</b> conversion of creatine reatinine). hal failure. causes false increas extinine ratio). vith creatinine measu <b>NATE:</b> <b>DESCRIPTION</b> mal kidney function dney damage with prmal or high GFR	LS: han creatinin ut of extrace blood). due to tubul to creatinin e in creatinin rement).	ellular fluid). ar secretion of urea ne). ne with certain met L/min/1.73m2 ) >90 >90	hodologies, ASSOCI No Presen	ATED FINDINGS	S	o when deł	nydrati
B. Reduced muscle m Certain drugs (e.g. NCREASED RATIO (>2 Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis ( Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (< Nuscular patients NAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin ther STIMATED GLOMERL G1 G2 G3a	ass (subnormal of tetracycline, glu 0:1) WITH ELEVA (BUN rises dispi- superimposed of 0:1) WITH DECRI osis. Ind starvation. 2: creased urea syr urea rather thar monemias (urea of inappropiate a 0:1) WITH INCRE py (accelerates of eleases muscle of who develop rer sis (acetoacetate creased BUN/crea apy (interferes v UAR FILTERATION Nor Nor Kid not	creatinine production cocorticoids) <b>TED CREATININE LEVE</b> roportionately more to n renal disease. <b>EASED BUN :</b> thesis. creatinine diffuses of is virtually absent in ntidiuretic harmone) <b>ASED CREATININE:</b> conversion of creatine reatinine). hal failure. e causes false increas eatinine ratio). vith creatinine measu <b>X RATE:</b> DESCRIPTION mal kidney function dney damage with ormal or high GFR id decrease in GFR	LS: han creatinin ut of extrace blood). due to tubul to creatinin e in creatinin rement).	ellular fluid). ar secretion of urea ne). he with certain met <u>L/min/1.73m2 ) &gt;90 &gt;90 60 -89</u>	hodologies, ASSOCI No Presen	ATED FINDING proteinuria ce of Protein ,	S	o when def	nydrati
<ol> <li>Reduced muscle m</li> <li>Certain drugs (e.g.</li> <li>INCREASED RATIO (&gt;2</li> <li>Postrenal azotemia</li> <li>DECREASED RATIO (</li> <li>Acute tubular necr</li> <li>Low protein diet ar</li> <li>Severe liver disease</li> <li>Other causes of de</li> <li>Repeated dialysis (</li> <li>Inherited hyperam</li> <li>SIADH (syndrome c</li> <li>Pregnancy.</li> <li>DECREASED RATIO (</li> <li>Rhabdomyolysis (r</li> <li>Muscular patients</li> <li>INAPPROPIATE RATIO</li> <li>Diabetic ketoacido</li> <li>Should produce an in</li> <li>Cephalosporin ther</li> <li>ESTIMATED GLOMERL</li> <li>G1</li> <li>G2</li> </ol>	ass (subnormal of tetracycline, glu 0:1) WITH ELEVA (BUN rises dispi- superimposed of 0:1) WITH DECRI osis. Ind starvation. E. creased urea syr urea rather thar monemias (urea of inappropiate a 0:1) WITH INCRE py (accelerates of eleases muscle of who develop rer sis (acetoacetate creased BUN/crea aby (interferes v UAR FILTERATION Nor Nor Nor Nor Nor	creatinine production cocorticoids) <b>TED CREATININE LEVE</b> roportionately more to n renal disease. <b>EASED BUN :</b> thesis. creatinine diffuses of is virtually absent in ntidiuretic harmone) <b>ASED CREATININE:</b> conversion of creatine reatinine). hal failure. causes false increas extinine ratio). vith creatinine measu <b>NATE:</b> <b>DESCRIPTION</b> mal kidney function dney damage with prmal or high GFR	LS: han creatinin ut of extrace blood). due to tubul to creatinin e in creatinin rement).	ellular fluid). ar secretion of urea ne). ne with certain met L/min/1.73m2 ) >90 >90	hodologies, ASSOCI No Presen	ATED FINDING proteinuria ce of Protein ,	S	o when deł	nydrati



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	Dr. Vinay Chopra MD (Pathology & Microb Chairman & Consultant F	iology) M[	m <b>Chopra</b> D (Pathology) nt Pathologist
NAME	: Miss. ANJALI		
AGE/ GENDER	: 24 YRS/FEMALE	PATIENT ID	: 1729923
COLLECTED BY	:	<b>REG. NO./LAB NO.</b>	: 012501210033
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 21/Jan/2025 12:31 PM
BARCODE NO.	:01524193	<b>COLLECTION DATE</b>	: 21/Jan/2025 12:33PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	<b>REPORTING DATE</b>	: 21/Jan/2025 03:06PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBAL	A CANTT	
Test Name	v	alue 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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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



	<b>Dr. Vinay Ch</b> MD (Pathology & Chairman & Con		Dr. Yugam MD CEO & Consultant	(Pathology)
NAME :	Miss. ANJALI			
AGE/ GENDER :	24 YRS/FEMALE	PAT	FIENT ID	: 1729923
<b>COLLECTED BY</b> :		REG	G. NO./LAB NO.	: 012501210033
<b>REFERRED BY</b> :		REG	GISTRATION DATE	: 21/Jan/2025 12:31 PM
BARCODE NO. :	01524193	COL	LECTION DATE	: 21/Jan/2025 12:33PM
CLIENT CODE. :	KOS DIAGNOSTIC LAB	REI	PORTING DATE	: 21/Jan/2025 02:02PM
<b>CLIENT ADDRESS</b> :	6349/1, NICHOLSON ROAD,	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		CLINICAL PA	THOLOGY	
	URINE RO	UTINE & MICRO	SCOPIC EXAMINA	ATION
PHYSICAL EXAMINA	<u>FION</u>			
QUANTITY RECIEVED	ICE SPECTROPHOTOMETRY	10	ml	
COLOUR		PALE YELLO	N	PALE YELLOW
TRANSPARANCY		HAZY		CLEAR
SPECIFIC GRAVITY	ICE SPECTROPHOTOMETRY	1.02		1.002 - 1.030
CHEMICAL EXAMINA				
REACTION	ICE SPECTROPHOTOMETRY	ACIDIC		
PROTEIN	ICE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
SUGAR	ICE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
рН	ICE SPECTROPHOTOMETRY	<=5.0		5.0 - 7.5
BILIRUBIN by DIP STICK/REFLECTAN	ICE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
NITRITE	ICE SPECTROPHOTOMETRY.	Negative		NEGATIVE (-ve)
UROBILINOGEN	ICE SPECTROPHOTOMETRY	Normal	EU/dL	0.2 - 1.0
KETONE BODIES	ICE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
BLOOD		Negative		NEGATIVE (-ve)
ASCORBIC ACID	ICE SPECTROPHOTOMETRY ICE SPECTROPHOTOMETRY INATION	NEGATIVE (-י	ve)	NEGATIVE (-ve)
RED BLOOD CELLS (R		NEGATIVE (	ve) /HPF	0 - 3



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EXCELLENCE IN MEALTMCARE & DIAGNOSTICS

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

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

NAME	: Miss. ANJALI					
AGE/ GENDER			PATIENT ID	: 1729923 <b>: 012501210033</b>		
COLLECTED BY			REG. NO./LAB NO.			
<b>REFERRED BY</b>	:		<b>REGISTRATION DATE</b>	: 21/Jan/2025 12:31 PM		
BARCODE NO.	<b>NO.</b> : 01524193 <b>COLLECTION DATE</b>	3 COLL		: 21/Jan/2025 12:33PM : 21/Jan/2025 02:02PM		
CLIENT CODE.	: KOS DIAGNOSTIC LAB		<b>REPORTING DATE</b>			
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANT	Т			
Test Name		Value	Unit	Biological Reference interval		
by MICROSCOPY ON O	CENTRIFUGED URINARY SEDIMENT					
PUS CELLS		1-3	/HPF	0 - 5		

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT				
EPITHELIAL CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	5-6	/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)	ABSENT		ABSENT	

\*\* End Of Report \*\*\*



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