

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT



	<b>Dr. Vinay Chopra</b> MD (Pathology & Micr Chairman & Consultar	obiology)		(Pathology)
NAME	: Mrs. UPASNA JAIN			
AGE/ GENDER	: 54 YRS/FEMALE		PATIENT ID	: 1720682
COLLECTED BY	: SURJESH		REG. NO./LAB NO.	: 012501100005
<b>REFERRED BY</b>	:		<b>REGISTRATION DATE</b>	: 10/Jan/2025 09:29 AM
BARCODE NO.	: 01523702		COLLECTION DATE	: 10/Jan/2025 10:05AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 10/Jan/2025 10:26AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMB	ALA CANTI		
Test Name		Value	Unit	<b>Biological Reference interval</b>
	SWAST	THYA W	ELLNESS PANEL: G	
			OOD COUNT (CBC)	
RED BLOOD CELLS	G (RBCS) COUNT AND INDICES			
HAEMOGLOBIN (H		12.2	gm/dL	12.0 - 16.0
by CALORIMETRIC RED BLOOD CELL (	DDC) COUNT	4.37	Millions	/cmm 3.50 - 5.00
	COUNT COCUSING, ELECTRICAL IMPEDENCE	4.37	WIIIIOUS	3.50 - 5.00
PACKED CELL VOLU	JME (PCV) UTOMATED HEMATOLOGY ANALYZER	37.2	%	37.0 - 50.0
MEAN CORPUSCUL	AR VOLUME (MCV)	85.3	fL	80.0 - 100.0
MEAN CORPUSCUL	UTOMATED HEMATOLOGY ANALYZER AR HAEMOGLOBIN (MCH)	27.9	pg	27.0 - 34.0
MEAN CORPUSCUL	UTOMATED HEMATOLOGY ANALYZER AR HEMOGLOBIN CONC. (MCHC)	32.7	g/dL	32.0 - 36.0
	UTOMATED HEMATOLOGY ANALYZER UTION WIDTH (RDW-CV)	15.7	%	11.00 - 16.00
by CALCULATED BY A	UTOMATED HEMATOLOGY ANALYZER			
	UTION WIDTH (RDW-SD) UTOMATED HEMATOLOGY ANALYZER	50.4	fL	35.0 - 56.0
MENTZERS INDEX		19.52	RATIO	BETA THALASSEMIA TRAIT: <
by CALCULATED				13.0 IRON DEFICIENCY ANEMIA:
				>13.0
GREEN & KING IND	DEX	30.63	RATIO	BETA THALASSEMIA TRAIT:<= 65.0
<i>by 0/12002/1120</i>				IRON DEFICIENCY ANEMIA: >
WILLITE DI AAN CE				65.0
WHITE BLOOD CE		6780	/cmm	4000 - 11000
by FLOW CYTOMETRY	Y BY SF CUBE & MICROSCOPY		/ cinili	
	BLOOD CELLS (nRBCS) RT HEMATOLOGY ANALYZER	NIL		0.00 - 20.00
NUCLEATED RED B	LOOD CELLS (nRBCS) %	NIL	%	< 10 %
by CALCULATED BY A	UTOMATED HEMATOLOGY ANALYZER			





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	<b>Dr. Vinay Chop</b> MD (Pathology & M Chairman & Consul	licrobiology)	Dr. Yugam MD ( CEO & Consultant F	Pathology)
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Test Name		Value	Unit	Biological Reference interval
DIFFERENTIAL LE	UCOCYTE COUNT (DLC)			
NEUTROPHILS by FLOW CYTOMETRY	Y BY SF CUBE & MICROSCOPY	61	%	50 - 70
LYMPHOCYTES by flow cytometry	Y BY SF CUBE & MICROSCOPY	31	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY	Y BY SF CUBE & MICROSCOPY	2	%	1 - 6
MONOCYTES	' BY SF CUBE & MICROSCOPY	6	%	2 - 12
BASOPHILS	BY SF CUBE & MICROSCOPY	0	%	0 - 1
	CYTES (WBC) COUNT			
ABSOLUTE NEUTRO	DPHIL COUNT ' by sf cube & microscopy	4136	/cmm	2000 - 7500
ABSOLUTE LYMPHO	OCYTE COUNT ' BY SF CUBE & MICROSCOPY	2102	/cmm	800 - 4900
ABSOLUTE EOSINO by FLOW CYTOMETRY	PHIL COUNT ' by sf cube & microscopy	136	/cmm	40 - 440
ABSOLUTE MONOC by FLOW CYTOMETRY	YTE COUNT ( by sf cube & microscopy	407	/cmm	80 - 880
PLATELETS AND O	THER PLATELET PREDICTIVE	<u>E MARKERS.</u>		
PLATELET COUNT ( by HYDRO DYNAMIC F	(PLT) OCUSING, ELECTRICAL IMPEDENCE	384000	/cmm	150000 - 450000
PLATELETCRIT (PC by HYDRO DYNAMIC F	T) OCUSING, ELECTRICAL IMPEDENCE	0.36 <sup>H</sup>	%	0.10 - 0.36
MEAN PLATELET V by hydro dynamic f	OLUME (MPV) ocusing, electrical impedence	9	fL	6.50 - 12.0
	CELL COUNT (P-LCC) OCUSING, ELECTRICAL IMPEDENCE	86000	/cmm	30000 - 90000
	CELL RATIO (P-LCR) OCUSING, ELECTRICAL IMPEDENCE	22.4	%	11.0 - 45.0
by HYDRO DYNAMIC F	UTION WIDTH (PDW) OCUSING, ELECTRICAL IMPEDENCE CTED ON EDTA WHOLE BLOOD	15.9	%	15.0 - 17.0





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CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 10/Jan/2025 02:35PM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A				
Test Name		Value	Unit	Biological Referen	nce interval
WHOLE BLOOD	EMOGLOBIN (HbA1c):	5.6	EMOGLOBIN (HBA1 %	4.0 - 6.4	
ESTIMATED AVERA	RMANCE LIQUID CHROMATOGRAPHY) GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY)	114.02	mg/dL	60.00 - 140.00	
	AS PER AMERICAN	DIABETES ASSOCI/	ATION (ADA):		
	REFERENCE GROUP	GL	YCOSYLATED HEMOGLOGIE	3 (HBAIC) in %	
	abetic Adults >= 18 years	1	<5.7		
	t Risk (Prediabetes)		5.7 – 6.4		
D	iagnosing Diabetes		>= 6.5		
			Age > 19 Years		
Theory			of Therapy:	< 7.0	
Therapeut	ic goals for glycemic control	Action	s Suggested:	>8.0	
			Ago < 10 Voore		
		<u> </u>	Age < 19 Years of therapy:	<7.5	

## COMMENTS:

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 appropiate.

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 faisely 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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LIENT ADDRESS	: 6349/1, NICHOLSON ROAI	D, AMBALA CANTT		
'est Name		Value	Unit	Biological Reference interval
by RED CELL AGGRE NTERPRETATION: . ESR is a non-specif mmune disease, but 2. An ESR can be affe is C-reactive protein 3. This test may also	DIMENTATION RATE (ESR) GATION BY CAPILLARY PHOTOME ic test because an elevated res does not tell the health practi cted by other conditions besid be used to monitor disease act	sult often indicates the pr tioner exactly where the i es inflammation. For this	mm/1st esence of inflammati oflammation is in the reason, the ESR is typ	hr 0 - 20
by RED CELL AGGRE <b>NTERPRETATION:</b> . ESR is a non-specifi nmune disease, but . An ESR can be affe s C-reactive protein . This test may also ystemic lupus eryth <b>ONDITION WITH LO</b> low ESR can be see polycythaemia), sigi s sickle cells in sick <b>IOTE:</b> . ESR and C - reactiv . Generally, ESR dog	DIMENTATION RATE (ESR) GATION BY CAPILLARY PHOTOME ic test because an elevated res does not tell the health practi cted by other conditions besid be used to monitor disease act ematosus W ESR n with conditions that inhibit t	17 TRY sult often indicates the pr tioner exactly where the i es inflammation. For this tivity and response to the he normal sedimentation count (leucocytosis), and e ESR. ers of inflammation. s CRP, either at the start of	mm/1st esence of inflammation for the	hr 0 - 20 on associated with infection, cancer and auto- 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





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Page 4 of 12





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Test Name		Value	Unit	<b>Biological Reference interval</b>
	CLINI	CAL CHEMISTI GLUCOSE FA	RY/BIOCHEMIST ASTING (F)	'nY

**IN ACCRDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES:** 1. A fasting plasma glucose level below 100 mg/dl is considered normal. 2. 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. 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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LIENT ADDRESS	: 6349/1, NICHOLSON ROAD	, AMBALA CANTT		
Fest Name		Value	Unit	<b>Biological Reference interval</b>
		LIPID PRC	OFILE : BASIC	
HOLESTEROL TOT	'AL: SERUM	105.71	mg/dL	<b>OPTIMAL:</b> < 200.0
by CHOLESTEROL OX		100.71	ing, ui	BORDERLINE HIGH: 200.0 -
				239.0
				HIGH CHOLESTEROL: > OR = 240.0
RIGLYCERIDES: SH		83.25	mg/dL	OPTIMAL: < 150.0
by GLYCEROL PHOSPI	HATE OXIDASE (ENZYMATIC)			BORDERLINE HIGH: 150.0 - 199.0
				HIGH: 200.0 - 499.0
				VERY HIGH: $> OR = 500.0$
IDL CHOLESTEROL by SELECTIVE INHIBITI	L (DIRECT): SERUM	45.3	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0
				60.0
				HIGH HDL: $> OR = 60.0$
DL CHOLESTEROL by CALCULATED, SPEC		43.76	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
ION HDL CHOLEST	'EROL: SERUM	60.41	mg/dL	OPTIMAL: < 130.0
by CALCULATED, SPEC	CTROPHOTOMETRY			ABOVE OPTIMAL: 130.0 - 159
				BORDERLINE HIGH: 160.0 - 189.0
				HIGH: 190.0 - 219.0
LDL CHOLESTERO	N. CEDUM	10.05		VERY HIGH: > OR = 220.0 0.00 - 45.00
by CALCULATED, SPE		16.65	mg/dL	0.00 - 45.00
OTAL LIPIDS: SER		<b>294.67<sup>L</sup></b>	mg/dL	350.00 - 700.00
by CALCULATED, SPEC		2.33	RATIO	LOW RISK: 3.30 - 4.40
by CALCULATED, SPEC				AVERAGE RISK: 4.50 - 7.0
				MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0

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Page 6 of 12





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Test Name		Value	Unit	<b>Biological Reference interval</b>
LDL/HDL RATIO: S by CALCULATED, SPE		0.97	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 ECTROPHOTOMETRY	1.84 <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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Test Name		Value	Unit	Biological Reference interval
BILIRUBIN TOTAL		<b>FUNCTIO</b> 0.46	<b>N TEST (COMPLETE)</b> mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT	Γ (CONJUGATED): SERUM SPECTROPHOTOMETRY	0.17	mg/dL	ADULI: 0.00 - 1.20 0.00 - 0.40
BILIRUBIN INDIRE	ECT (UNCONJUGATED): SERUM	0.29	mg/dL	0.10 - 1.00
SGOT/AST: SERUM		19.5	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PY	[ /RIDOXAL PHOSPHATE	17.5	U/L	0.00 - 49.00
AST/ALT RATIO: S by CALCULATED, SPE	ERUM ECTROPHOTOMETRY	1.11	RATIO	0.00 - 46.00
ALKALINE PHOSPI by Para Nitrophen propanol	HATASE: SERUM IYL PHOSPHATASE BY AMINO METHYL	44.23	U/L	40.0 - 130.0
GAMMA GLUTAMY by SZASZ, SPECTRO	L TRANSFERASE (GGT): SERUM PHTOMETRY	10.02	U/L	0.00 - 55.0
TOTAL PROTEINS: by BIURET, SPECTRO		6.93	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL G		4.1	gm/dL	3.50 - 5.50
GLOBULIN: SERUN by CALCULATED, SPE	I ECTROPHOTOMETRY	2.83	gm/dL	2.30 - 3.50
A : G RATIO: SERU by CALCULATED, SPE	M ectrophotometry	1.45	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:**

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



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Test Name	v	alue Unit	Biological Reference interva

### **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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CLIENT CODE.	: KOS DIAGNOSTIC LAB	]	REPORTING DATE	: 10/Jan/2025 11:18AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AN	MBALA CANTT		
Test Name		Value	Unit	<b>Biological Reference interval</b>
	KIDNE	EY FUNCTION	N TEST (COMPLETE)	
UREA: SERUM		16.1	mg/dL	10.00 - 50.00
-	ATE DEHYDROGENASE (GLDH)		0	
CREATININE: SERU by ENZYMATIC, SPEC		0.92	mg/dL	0.40 - 1.20
-	ROGEN (BUN): SERUM	7.52	mg/dL	7.0 - 25.0
BLOOD UREA NITH	ROGEN (BUN)/CREATININE	8.17 <sup>L</sup>	RATIO	10.0 - 20.0
RATIO: SERUM				
by CALCULATED, SPE UREA/CREATININ		17.5	RATIO	
by CALCULATED, SPE		17.0	in 110	
URIC ACID: SERUM		2.47 <sup>L</sup>	mg/dL	2.50 - 6.80
by URICASE - OXIDAS CALCIUM: SERUM	SEPERUXIDASE	9.23	mg/dL	8.50 - 10.60
by ARSENAZO III, SPE	CTROPHOTOMETRY		ing, ui	0.00 10.00
PHOSPHOROUS: SE	ERUM DATE, SPECTROPHOTOMETRY	3.82	mg/dL	2.30 - 4.70
ELECTROLYTES	DATE, SPECIROPHOTOMETRY			
SODIUM: SERUM		138.1	mmol/L	135.0 - 150.0
by ISE (ION SELECTIV	ELECTRODE)	100.1		100.0 100.0
POTASSIUM: SERU		4	mmol/L	3.50 - 5.00
by ISE (ION SELECTIV CHLORIDE: SERUM		103.57	mmol/L	90.0 - 110.0
by ISE (ION SELECTIV	(E ELECTRODE)			
ESTIMATED GLON	IERULAR FILTERATION RATE			
	ERULAR FILTERATION RATE	74		
(eGFR): SERUM				
INTERPRETATION:				
To differentiate betw	een 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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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





NAME       : Mrs. UPASNA JAIN         AGE/ CERDER       : 54 YRS/FEMALE       PATIENT ID       : 1720682         COLLECTED BY       : SURJESH       REG.NO./LAB NO.       : 012501100005         REFEREED BY       :       REGISTRATION DATE       : 10/Jan/2025 09:29 AM         BARCODE NO.       : 01523702       COLLECTION DATE       : 10/Jan/2025 10:05AM         CLIENT CODE       : KOS DIAGNOSTIC LAB       REPORTING DATE       : 10/Jan/2025 11:18AM         CLIENT ADDRESS       : 6349/1, NICHOLSON ROAD, AMBALA CANTT       : 10/Jan/2025 11:18AM         Test Name       Value       Unit       Biological Reference Interva         4. High protein intake.       : Impaired renal function plus       : Gexes protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, burns, surgery, cachexia, high fever).          1. Unitor reabsoribin (e.g. ureter colostomy)       : Reduced muscle mass (subnormal creatinine production)       : Reduced muscle mass (subnormal creatinine production)         2. Certain drugs (e.g. tetracyclin, e.glucocorticoids)       INCREASED RATIO (-20:1) WITH EEVATED CREATININE ELVELS:          1. Postrenal azotemia Superimposed on renal disease.           2. Low protein dict and starvation.           3. Severe liver disease.		٢	Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologist		Dr. Yugam Chopra MD (Pathology) CEO & Consultant Pathologist					
COLLECTED BY       SURJESH       REG. NO. /LAB NO.       : 012501100005         REFERRED BY       ::       REGISTRATION DATE       : 10/Jan/2025 09:29 AM         BARCODE NO.       ::1523702       COLLECTION DATE       ::10/Jan/2025 10:05AM         CLIENT CODE       ::KOS DIAGNOSTIC LAB       REPORTING DATE       ::10/Jan/2025 11:18AM         CLIENT ADDRES       ::8349/1, NICHOLSON ROAD, AMBALA CANTT	NAME	: Mrs. UPASNA	JAIN							
REFEREND BY       I:       REGISTRATION DATE       I:0/Jan/2025 09:29 AM         BARCODE NO.       I:0.523702       COLLECTION DATE       I:0/Jan/2025 10:05AM         CLIENT CODE       I:0.53702       REPORTING DATE       I:0/Jan/2025 11:18AM         CLIENT ADDRESS       I:0.43an/2025 11:18AM       IIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIII	AGE/ GENDER	: 54 YRS/FEMA	LE	РА	TIENT ID	: 17	720682			
REFERRED BY       ::       REGISTRATION DATE       :10/Jan/2025 09:29 AM         BARCODE NO.       :01523702       COLLECTION DATE       :10/Jan/2025 10:05AM         CLIENT CODE       ::       KOS DIACNOSTIC LAB       REPORTING DATE       :10/Jan/2025 11:18AM         CLIENT ADDRESS       ::       :	COLLECTED BY	· SURIESH		RE	G. NO. / LAB NO.	: 0	125011000	05		
BARCODE NO. : 101523702 COLLECTION DATE : 10/Jan/2025 10:05AM CLIENT CODE : : KOS DIAGNOSTIC LAB BEPORTING DATE : 10/Jan/2025 11:18AM CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Unit Biological Reference interva 4. High protein intake. 5. Impaired renal function plus 6. Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, burns, surgery, cacheska, high fever). 1. Urine reabsorption (e.g. ureter colostomy) 8. Reduced muscle mass (subnormal creatinine production) 9. Certain drugs (e.g. tetracycline, glucocorticoids) 1. Verice reabsorption (e.g. ureter colostomy) 8. Reduced muscle mass (subnormal creatinine production) 9. Certain drugs (e.g. tetracycline, glucocorticoids) 1. Veroterab zotemia superimposed on renal disease. DECREASED RATIO (<0.21) WITH DECREASED BUN : 1. Acute tubular necrosis 3. Severe liver disease. 3. Own protein diet and starvation. 3. Severe liver disease. 4. Other causes of decreased urea synthesis. 5. Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid). 6. Inherited hyperammonemias (urea is virtually absent in blood). 7. SIADH (syndrome of inappropiate antidiuretic harmone) due to tubular secretion of urea. 8. Pregnancy. <b>DECRESED RATIO (&lt;0.1) WITH INCREASED CREATININE:</b> 1. Phenacimide therapy (accelerates conversion of creatine to creatinine). 2. Rhadomyolysis (releases muscle creatinine). 2. Rhadomyolysis (celeases muscle creatinine). 3. Muscular poleratiens who develop renal failure. <b>INEPPOPIATE RATIO</b> 1. Diabetic ketoacidosis (acctoacetate causes false increase in creatinine with certain methodologies, resulting in normal ratio when dehydrat should produce an increased BUN/creatinine ratio). 2. Cephalosporin therapy (Interferes with creatinine measurement). <b>STIMATEO CIGNERULAR TILERATION OF (R1 (mL/min/1.73m2)</b> ASSOCIATED FINDINCS G1 Normal Kidney damage with >90 Presence of Protein , Albumin or cast in uri		·								
CLIENT CODE       K. KOS DIAGNOSTIC LAB       REPORTING DATE       10/Jan/2025 11:18AM         CLIENT ADDRESS       6349/1, NICHOLSON ROAD, AMBALA CANTF         Test Name       Value       Unit       Biological Reference interva         4. High protein intake.       Simpaired renal function plus       Simpaired renal function plus         6. Excess protein intake or production or tissue breakdown (e.g. infection, Gl bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, burns, surgery, cachexia, high fever).         7. Urine reabsorption (e.g. ureter colostomy)       Reduced muscle mass (ubnormal creatinine production)         9. Certain drugs (e.g. tetracycline, glucocorticoids)       NORKASED ATIO (2017) WITH ELEVATED CREATININE LEVELS:         1. Postrenal azotemia Superimposed on renal disease.       DECCEMASED RATIO (2017) WITH DECCEASED BUN :         1. Acute tubular necrosis.       2. Ore arostin diet and starvation.         3. Severe liver disease.       4. Other causes of decreased urea synthesis.         5. Repeated diskyis (urea rather than creatinine diffuses out of extracellular fluid).         6. Inherited hyperammonemias (urea is virtually absent in blood).         7. SIADH (syndrome of inappropiate antidiuretic harmone) due to ubular secretion of urea.         8. Prepancy.         DeceeASED RATO (-010) WITH INCREASED CREATININE.         1. Dabetic ketoacidostis (acetoacetate causes false increase in creatinine with certain methodologies, resulting i										
CLENT ADRESS       : 6349/1, NICHOLSON ROAD, AMBALA CANTT         Test Name       Value       Unit       Biological Reference interva         4. High protein intake.       S.       Impaired renal function plus         6. Excess protein intake or production or tissue breakdown (e.g. infection, Gl bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, burns, surgery, cachexia, high fever).         7. Urine reabsorption (e.g. ureter colostomy)       8. Reduced muscle mass (subnormal creatinine production)         9. Certain drugs (e.g. tetracycline, gluccoorticoids)       IVENESS DRATIO (>20:1) WITH ELEVATED CREATININE LEVELSI:         1. Postrenal azotemia superimposed on renal disease.       DecreaseD RATIO (>10:1) WITH DECREASED BUN :         1. Acute tubular necrosis.       1. Acute tubular necrosis.         2. Or protein diel and starvation.       3. Severe liver disease.         9. Other causes of decreased urea synthesis.       5. Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid).         6. Inherited hyperamonemias (urea is virtually absent in blood).       7. SIADH (syndrome of inappropiate antidiuretic harmone) due to tubular secretion of urea.         7. Byte statemets where causes false increase in creatinine with certain methodologies, resulting in normal ratio when dehydra should produce an increased BUN/creatinine measurement).         10. The cause and burder at the tract causes false increase in creatinine with certain methodologies, resulting in normal ratio when dehydra should produce an increased BUN										
Test Name       Value       Unit       Biological Reference interva         4. High protein intake.       5. Impaired renal function plus       6. Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, burns, surgery, cachexia, high fever).         1. Urine reabsorption (e.g. ureter colostomy)       8. Reduced muscle mass (subnormal creatinine production)         9. Certain drugs (e.g. tetracycline, gluccorticoids)       INORCASED RATIO (-20:1) WITH ELEVATED CREATININE LEVELS:         1. Postnenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).       2. Prerenal azotemia superimposed on renal disease.         2. Percenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).       2. Percenal azotemia superimposed on renal disease.         3. Percenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).       2. Percenal asotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).         2. Prerenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).       3. Percenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).         3. Bergera (BUN creatinine).       1. Acute tubular necrosis.       3. Gauge (Gue erates conversion of creatine to creatinine).         3. Muscular patients who develop renal failure.       1. Phenacimide therapy (accelerate causes false increase in creatinine with certain methodologies, resulting in norm	CLIENT CODE.	: KOS DIAGNOS	TIC LAB	RE	PORTING DATE	: 10	)/Jan/2025 1	1:18AM		
4. High protein intake.         5. Impaired renal function plus         6. Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, burns, surgery, cachexia, high fever).         7. Urine reabsorption (e.g. ureter colostomy)         8. Reduced muscle mass (Subnormal creatinine production)         9. Certain drugs (e.g. tetracycline, glucocorticoids)         INCREASED RATIO (>20:1) WITH ELEVATED CREATININE LEVELS:         1. Postrenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).         2. Prerenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).         2. Prerenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).         3. Postrenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).         2. Prerenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).         3. Postrenal azotemia (SUR rises)         3. Severe liver disease.         3. Severe liver disease.         4. Other causes of decreased urea synthesis.         5. Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid).         6. Inherited hyperaminomemias (urea is virtually absent in blood).         7. SIADH (syndrome of inappropiate antidiuretic harmone) due to tubular secretion of urea.         8. Pregnancy.	CLIENT ADDRESS	: 6349/1, NICH	OLSON ROAD, AMB	ALA CANTT						
5. Impaired renal function plus 6. Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, burns, surgery, cachexia, high fever). 7. Urine reabsorption (e.g. ureter colostomy) 8. Reduced muscle mass (subnormal creatinine production) 9. Certain drugs (e.g. tetracycline, glucocorticoids) <b>INCREASED RATIO (&gt;20:1) WITH ELEVATED CREATININE LEVELS:</b> 1. Postrenal azotemia (BUR rises disproportionately more than creatinine) (e.g. obstructive uropathy). 2. Prerenal azotemia (BUR rises disproportionately more than creatinine) (e.g. obstructive uropathy). 2. Prerenal azotemia (BUR rises disproportionately more than creatinine) (e.g. obstructive uropathy). 3. Pertere azotemia (BUR rises disproportionately more than creatinine) (e.g. obstructive uropathy). 4. Cute tubular necrosis. 5. Low protein diet and starvation. 3. Severe liver disease. 4. Other causes of decreased urea synthesis. 5. Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid). 6. Inherited hyperammonemias (urea is virtually absent in blood). 7. SIADH (syndrome of inappropiate antidiuretic harmone) due to tubular secretion of urea. 8. Pregnancy. <b>DECREASED RATIO (&lt;10:1) WITH INCREASED CREATININE:</b> 1. Phenacimide therapy (accelerates conversion of creatine to creatinine). 2. Rhabdomyolysis (releases muscle creatinine). 3. Muscular patients who develop renal failure. <b>INAPPROPIATE RATIO:</b> 1. Diabetic ketoacidosis (acetoacetate causes false increase in creatinine with certain methodologies, resulting in normal ratio when dehydra should produce an increased BUN/creatinine reasurement). <b>STIMATED GLOMERULAR FILTERATION RATE:</b> <b>CKD STAGE DESCRIPTION GFR (mL/min/1.73m2) ASSOCIATED FINDINGS</b> <b>G</b> Kidney damage with >90 Presence of Protein , Albumin or cast in urine <b>G</b> Kidney damage with >90 Presence of Protein , Albumin or cast in urine <b>G</b> Kidney damage with >90 Presence of Protein , Albumin or cast in urine <b>G</b> Kidne	Test Name			Value	Uni	t	Biolog	gical Refer	rence inter	val
G3aMild decrease in GFR60 -89G3bModerate decrease in GFR30-59G4Severe decrease in GFR15-29	1. Postrenal azotemia	20:1) WITH ELEVA a (BUN rises dispre	ED CREATININE LEVE		(e.g. obstructive	uropathy).				
G4 Severe decrease in GFR 15-29	1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (< 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 of 8. Pregnancy. DECREASED RATIO (< 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERI G1 G2	20:1) WITH ELEVAT a (BUN rises dispr- superimposed or 10:1) WITH DECRE tosis. and starvation. e. creased urea syn (urea rather than monemias (urea of inappropiate ar 10:1) WITH INCRE/ upy (accelerates co eleases muscle co who develop ren creased BUN/cre- rapy (interferes w JLAR FILTERATION Norm Kid no	TED CREATININE LEVE oportionately more to renal disease. ASED BUN : ASED BUN : thesis. creatinine diffuses of is virtually absent in tidiuretic harmone) ASED CREATININE: onversion of creatine reatinine). al failure. causes false increas atinine ratio). ith creatinine measu RATE: DESCRIPTION hal kidney function ney damage with rmal or high GFR	han creatinine) but of extracellu blood). due to tubular s e to creatinine). e in creatinine v rement). GFR (mL/r	ular fluid). secretion of urea. with certain meth <u>min/1.73m2 )</u> >90	nodologies,ru ASSOCIA No pu Presence	<b>TED FINDINGS</b> roteinuria e of Protein ,	6	when dehyc	ratio
	1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (< 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 of 8. Pregnancy. DECREASED RATIO (< 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERU G1 G2 G3a	20:1) WITH ELEVAT a (BUN rises dispri- superimposed or 10:1) WITH DECRE tosis. and starvation. e. creased urea syn (urea rather than monemias (urea of inappropiate ar 10:1) WITH INCRE/ upy (accelerates co eleases muscle cr who develop ren creased BUN/cre- rapy (interferes w JLAR FILTERATION Norm Kid no	TED CREATININE LEVE oportionately more to a renal disease. ASED BUN : ASED BUN : ASED BUN : Creatinine diffuses of is virtually absent in tidiuretic harmone) ASED CREATININE: onversion of creatine reatinine). al failure. Causes false increase atinine ratio). ith creatinine measu RATE: DESCRIPTION nal kidney function ney damage with rmal or high GFR	than creatinine) but of extracellublood). due to tubular set to creatinine). e in creatinine v rement).	llar fluid). secretion of urea. with certain meth <u>min/1.73m2 )</u> >90 >90 >90	nodologies,ru ASSOCIA No pu Presence	<b>TED FINDINGS</b> roteinuria e of Protein ,	6	when dehyd	ratio
G5 Kidney failure <15	1. Postrenal azotemia 2. Prerenal azotemia DECREASED RATIO (< 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 of 8. Pregnancy. DECREASED RATIO (< 1. Phenacimide thera 2. Rhabdomyolysis (r 3. Muscular patients INAPPROPIATE RATIO 1. Diabetic ketoacido should produce an in 2. Cephalosporin ther ESTIMATED GLOMERU G1 G2 G3a G3a G3b	20:1) WITH ELEVAT a (BUN rises dispr- superimposed or 10:1) WITH DECRE tosis. and starvation. e. creased urea syn (urea rather than monemias (urea of inappropiate ar 10:1) WITH INCRE/ upy (accelerates ca eleases muscle cr who develop ren creased BUN/cre- rapy (interferes w <u>JAR FILTERATION</u> Norm Kid no Mile Mode	TED CREATININE LEVE oportionately more to renal disease. ASED BUN : ASED BUN : ASED BUN : Creatinine diffuses of is virtually absent in tidiuretic harmone) ASED CREATININE: onversion of creatine reatinine). al failure. Causes false increas atinine ratio). ith creatinine measu RATE: DESCRIPTION nal kidney function ney damage with rmal or high GFR d decrease in GFR rate decrease in GFR	than creatinine) but of extracellublood). due to tubular set to creatinine). e in creatinine verement). GFR (mL/r	ular fluid). secretion of urea. with certain meth <u>min/1.73m2 )</u> >90 >90 >90 >90 >59	nodologies,ru ASSOCIA No pu Presence	<b>TED FINDINGS</b> roteinuria e of Protein ,	6	when dehyd	ratio





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	Dr. Vinay Chopra MD (Pathology & Microbio Chairman & Consultant Pat		(Pathology)
NAME	: Mrs. UPASNA JAIN		
AGE/ GENDER	: 54 YRS/FEMALE	PATIENT ID	: 1720682
COLLECTED BY	: SURJESH	<b>REG. NO./LAB NO.</b>	: 012501100005
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 10/Jan/2025 09:29 AM
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA	CANTT	
Test Name	Val	ue 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

End Of Report \*\*\*





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