



	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbiol Chairman & Consultant Pat		Dr. Yugam C MD (Pat EO & Consultant Pat	hology)
NAME : Mrs. MI	ENAKSHI GUPTA			
AGE/ GENDER : 36 YRS/	FEMALE	PATIENT	<b>ID</b> :	1709121
COLLECTED BY :		REG. NO.	LAB NO. :	012412260015
REFERRED BY :				26/Dec/2024 10:37 AM
BARCODE NO. : 0152302				26/Dec/2024 11:23AM
	GNOSTIC LAB NICHOLSON ROAD, AMBALA (	<b>REPORTI</b> CANTT	NG DATE :	26/Dec/2024 11:16AM
Test Name	Val	ue	Unit	<b>Biological Reference interval</b>
		A WELLNES		
<u>RED BLOOD CELLS (RBCS) C</u>	OUNT AND INDICES			
HAEMOGLOBIN (HB)	11,	4 <sup>L</sup>	gm/dL	12.0 - 16.0
RED BLOOD CELL (RBC) COU		1 <sup>H</sup>	Millions/cm	m 3.50 - 5.00
PACKED CELL VOLUME (PCV)	37.	.4	%	37.0 - 50.0
by CALCULATED BY AUTOMATED MEAN CORPUSCULAR VOLUM by CALCULATED BY AUTOMATED	TE (MCV) 73	2 <sup>L</sup>	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEM	OGLOBIN (MCH) 22	.2 <sup>L</sup>	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMO	GLOBIN CONC. (MCHC) 30	3 <sup>L</sup>	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WI		8 <sup>H</sup>	%	11.00 - 16.00
RED CELL DISTRIBUTION WI	DTH (RDW-SD) 54.	2	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	14.	32	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	28.	22	RATIO	BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CELLS (WBC	<u>5)</u>			
TOTAL LEUCOCYTE COUNT (T by FLOW CYTOMETRY BY SF CUB		50	/cmm	4000 - 11000
		L		0.00 - 20.00
NUCLEATED RED BLOOD CEL by AUTOMATED 6 PART HEMATOL				





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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





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

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

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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANT	Г	

Test Name	Value	Unit	<b>Biological Reference interval</b>
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	49 <sup>L</sup>	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	41 <sup>H</sup>	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	8	%	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	2818	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2358	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	115	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	460	/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	515000 <sup>H</sup>	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.52 <sup>H</sup>	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	10	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	141000 <sup>H</sup>	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by Hydro Dynamic Focusing, electrical impedence	27.4	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD	16.1	%	15.0 - 17.0



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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA	A CANTT	
Test Name	Va	alue Unit	Biological Reference interval



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BARCODE NO.	: 01523022		ECTION DATE	: 26/Dec/2024 11:23AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		RTING DATE	: 26/Dec/2024 02:51PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interva
		DSYLATED HAEMO		
WHOLE BLOOD by HPLC (HIGH PERFO	EMOGLOBIN (HbA1c):	8.4 <sup>H</sup>	%	4.0 - 6.4
WHOLE BLOOD by hplc (high perfo ESTIMATED AVERA	EMOGLOBIN (HbA1c):			
WHOLE BLOOD by HPLC (HIGH PERFO ESTIMATED AVERA by HPLC (HIGH PERFO	AEMOGLOBIN (HbA1c): RMANCE LIQUID CHROMATOGRAPHY) AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY)	8.4 <sup>H</sup>	% mg/dL	4.0 - 6.4
WHOLE BLOOD by HPLC (HIGH PERFO. ESTIMATED AVERA by HPLC (HIGH PERFO. INTERPRETATION:	AEMOGLOBIN (HbA1c): RMANCE LIQUID CHROMATOGRAPHY) AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN REFERENCE GROUP	8.4 <sup>H</sup> 194.38 <sup>H</sup> DIABETES ASSOCIATION	% mg/dL	4.0 - 6.4 60.00 - 140.00
WHOLE BLOOD by HPLC (HIGH PERFO. ESTIMATED AVERA by HPLC (HIGH PERFO. INTERPRETATION:	AEMOGLOBIN (HbA1c): RMANCE LIQUID CHROMATOGRAPHY) AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN REFERENCE GROUP abetic Adults >= 18 years	8.4 <sup>H</sup> 194.38 <sup>H</sup> DIABETES ASSOCIATION	% mg/dL (ADA): LATED HEMOGLOGIB <5.7	4.0 - 6.4 60.00 - 140.00
WHOLE BLOOD by HPLC (HIGH PERFO. ESTIMATED AVERA by HPLC (HIGH PERFO. INTERPRETATION: NON dia	AEMOGLOBIN (HbA1c): RMANCE LIQUID CHROMATOGRAPHY) AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN REFERENCE GROUP abetic Adults >= 18 years t Risk (Prediabetes)	8.4 <sup>H</sup> 194.38 <sup>H</sup> DIABETES ASSOCIATION	% mg/dL (ADA): LATED HEMOGLOGIB <5.7 5.7 - 6.4	4.0 - 6.4 60.00 - 140.00
WHOLE BLOOD by HPLC (HIGH PERFO. ESTIMATED AVERA by HPLC (HIGH PERFO. INTERPRETATION: Non dia A	AEMOGLOBIN (HbA1c): RMANCE LIQUID CHROMATOGRAPHY) AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN REFERENCE GROUP abetic Adults >= 18 years	8.4 <sup>H</sup> 194.38 <sup>H</sup> DIABETES ASSOCIATION	% mg/dL (ADA): LATED HEMOGLOGIB <5.7 5.7 - 6.4 >= 6.5	4.0 - 6.4 60.00 - 140.00
WHOLE BLOOD by HPLC (HIGH PERFO. ESTIMATED AVERA by HPLC (HIGH PERFO. INTERPRETATION: Non dia A	AEMOGLOBIN (HbA1c): RMANCE LIQUID CHROMATOGRAPHY) AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN REFERENCE GROUP abetic Adults >= 18 years t Risk (Prediabetes)	8.4 <sup>H</sup> 194.38 <sup>H</sup> DIABETES ASSOCIATION GLYCOSY	% mg/dL (ADA): LATED HEMOGLOGIB <5.7 5.7 - 6.4 >= 6.5 Age > 19 Years	4.0 - 6.4 60.00 - 140.00 (HBAIC) in %
WHOLE BLOOD by HPLC (HIGH PERFO. ESTIMATED AVERA by HPLC (HIGH PERFO. INTERPRETATION:	AEMOGLOBIN (HbA1c): RMANCE LIQUID CHROMATOGRAPHY) AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN REFERENCE GROUP abetic Adults >= 18 years t Risk (Prediabetes) iagnosing Diabetes	8.4 <sup>H</sup> 194.38 <sup>H</sup> DIABETES ASSOCIATION GLYCOSY GOals of The	% mg/dL (ADA): LATED HEMOGLOGIB <5.7 5.7 - 6.4 >= 6.5 Age > 19 Years rapy:	4.0 - 6.4 60.00 - 140.00 (HBAIC) in % < 7.0
WHOLE BLOOD by HPLC (HIGH PERFO. ESTIMATED AVERA by HPLC (HIGH PERFO. INTERPRETATION:	AEMOGLOBIN (HbA1c): RMANCE LIQUID CHROMATOGRAPHY) AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN REFERENCE GROUP abetic Adults >= 18 years t Risk (Prediabetes)	8.4 <sup>H</sup> 194.38 <sup>H</sup> DIABETES ASSOCIATION GLYCOSY	% mg/dL (ADA): LATED HEMOGLOGIB <5.7 5.7 - 6.4 >= 6.5 Age > 19 Years rapy:	4.0 - 6.4 60.00 - 140.00 (HBAIC) in %

**KOS Diagnostic Lab** 

(A Unit of KOS Healthcare)

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 falsely lower HbA1c results.

6.HbA1c results from patients with HbSS,HbSC and HbD must be interpreted with caution, given the pathological processes including anemia, increased red cell turnover, and transfusion requirement that adversely impact HbA1c as a marker of long-term gycemic control.

7.Specimens from patients with polycythemia or post-splenctomy may exhibit increse in HbA1c values due to a somewhat longer life span of the red cells.



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



TEST PERFORMED AT KOS DIAGNOSTIC LAB. AMBALA CANTT



TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



<b>Dr. Vinay Chopra</b> MD (Pathology & Microbiology Chairman & Consultant Patholo			(Pathology)	
NAME	: Mrs. MEENAKSHI	GUPTA		
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BARCODE NO.	:01523022		<b>COLLECTION DATE</b>	: 26/Dec/2024 11:23AM
CLIENT CODE.	: KOS DIAGNOSTIC	LAB	REPORTING DATE	: 26/Dec/2024 12:03PM
CLIENT ADDRESS	: 6349/1, NICHOLS	ON ROAD, AMBALA CANT	ГТ	
Test Name		Value	Unit	<b>Biological Reference interval</b>
	1	ERYTHROCYTE SEI	DIMENTATION RATE (	(ESR)
2. An ESR can be affe as C-reactive protein 3. This test may also systemic lupus eryth <b>CONDITION WITH LO</b> A low ESR can be see (polycythaemia), sig as sickle cells in sick <b>NOTE:</b> 1. ESR and C - reactiv 2. Generally, ESR dog	ected by other condition be used to monitor dis ematosus <b>W ESR</b> en with conditions that nificantly high white bl le cell anaemia) also lo re protein (C-RP) are bo es not change as rapidl	ns besides inflammation. sease activity and respons inhibit the normal sedim lood cell count (leucocyto ower the ESR. oth markers of inflammati ly as does CRP, either at ti	se to therapy in both of the a mentation of red blood cells, s osis) , and some protein abno on. he start of inflammation or a	picallý used in conjunction with other test such above diseases as well as some others, such as such as a high red blood cell count ormalities. Some changes in red cell shape (such as it resolves.
4. If the ESR is elevat 5. Women tend to ha 6. Drugs such as dex	ted, it is typically a resu ave a higher ESR, and m	ult of two types of protein nenstruation and pregnan contraceptives, penicilla	better marker of inflammations, globulins or fibrinogen. cy can cause temporary eleva mine procainamide, theophy	





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MBBS, MD (PATHOLOGY)







		ogy & Microbiology) Consultant Pathologist	Dr. Yugam ( MD (P CEO & Consultant Pa	athology)
NAME	: Mrs. MEENAKSHI GUP	ТА		
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BARCODE NO.	:01523022	COI	LECTION DATE	: 26/Dec/2024 11:23AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REF	ORTING DATE	: 26/Dec/2024 01:13PM
CLIENT ADDRESS	: 6349/1, NICHOLSON RO	DAD, AMBALA CANTT		
Test Name		Value	Unit	<b>Biological Reference interval</b>
	CLI	INICAL CHEMISTR	Y/BIOCHEMISTR	Y
		GLUCOSE FAS	STING (F)	
	G (F): PLASMA	106.52 <sup>H</sup>	mg/dL	NORMAL: < 100.0

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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CLIENT ADDRESS	: 6349/1, NICHOLSON ROA	D, AMBALA CANTT		
Test Name		Value	Unit	<b>Biological Reference interval</b>
		LIPID PROF	ILE : BASIC	
CHOLESTEROL TO by CHOLESTEROL O		79.6	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR =
TRIGLYCERIDES: S by GLYCEROL PHOSE	ERUM PHATE OXIDASE (ENZYMATIC)	66.16	mg/dL	240.0 OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0
HDL CHOLESTERO by SELECTIVE INHIBIT	L (DIRECT): SERUM TION	25.04 <sup>L</sup>	mg/dL	VERY HIGH: > OR = 500.0 LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0
DL CHOLESTERO		41.33	mg/dL	OPTIMAL: > OK = 60.0 OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129. BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0
NON HDL CHOLES' by CALCULATED, SPE	TEROL: SERUM ECTROPHOTOMETRY	54.56	mg/dL	VERY HIGH: > OR = 190.0 OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159. BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0
LDL CHOLESTER		13.23	mg/dL	VERY HIGH: > OR = 220.0 0.00 - 45.00
TOTAL LIPIDS: SEF	ectrophotometry RUM ectrophotometry	225.36 <sup>L</sup>	mg/dL	350.00 - 700.00
CHOLESTEROL/HI		3.18	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 by CALCULATED, SPE		1.65	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	2.64 <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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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	0.36	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.13	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.23	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	21.2	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	24.6	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	0.86	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL	58.02	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	13.65	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	7.1	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	4.97	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.13 <sup>L</sup>	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.33 <sup>H</sup>	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)





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

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

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Page 9 of 13

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT





	<b>Dr. Vinay Chopra</b> MD (Pathology & Micro Chairman & Consultan	obiology) MI	m <b>Chopra</b> D (Pathology) ht Pathologist
NAME	: Mrs. MEENAKSHI GUPTA		
AGE/ GENDER	: 36 YRS/FEMALE	PATIENT ID	: 1709121
COLLECTED BY	:	REG. NO./LAB NO.	: 012412260015
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 26/Dec/2024 10:37 AM
BARCODE NO.	:01523022	COLLECTION DATE	: 26/Dec/2024 11:23AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	<b>REPORTING DATE</b>	: 26/Dec/2024 01:13PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBA	LA CANTT	
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



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

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	Dr. Vinay Cho MD (Pathology & N Chairman & Consu		Dr. Yugam MD (F CEO & Consultant P	Pathology)	
NAME	: Mrs. MEENAKSHI GUPTA				
AGE/ GENDER	: 36 YRS/FEMALE	PA	TIENT ID	: 1709121	
<b>COLLECTED BY</b>	:	RE	G. NO./LAB NO.	: 012412260015	
<b>REFERRED BY</b>	:	RE	GISTRATION DATE	: 26/Dec/2024 10:37 AM	
BARCODE NO.	: 01523022	CO	LLECTION DATE	: 26/Dec/2024 11:23AM	
CLIENT CODE.	: KOS DIAGNOSTIC LAB	RE	PORTING DATE	: 26/Dec/2024 01:13PM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT			
Test Name		Value	Unit	<b>Biological Reference interval</b>	
	KIDNI	EY FUNCTION '	FEST (COMPLETE)		
UREA: SERUM		29.22	mg/dL	10.00 - 50.00	
	NATE DEHYDROGENASE (GLDH)	~0.~~	Ũ	10.00 00.00	
CREATININE: SER		0.74	mg/dL	0.40 - 1.20	
	ROGEN (BUN): SERUM	13.65	mg/dL	7.0 - 25.0	
by CALCULATED, SPE	ECTROPHOTOMETRY	10.15			
BLOOD UREA NITE RATIO: SERUM	ROGEN (BUN)/CREATININE	18.45	RATIO	10.0 - 20.0	
	ECTROPHOTOMETRY				
UREA/CREATININ	E RATIO: SERUM ECTROPHOTOMETRY	39.49	RATIO		
URIC ACID: SERUM		2.5	mg/dL	2.50 - 6.80	
by URICASE - OXIDAS			ů.		
CALCIUM: SERUM by ARSENAZO III, SPE	ECTROPHOTOMETRY	10.18	mg/dL	8.50 - 10.60	
PHOSPHOROUS: SH	ERUM	3.42	mg/dL	2.30 - 4.70	
	DATE, SPECTROPHOTOMETRY				
ELECTROLYTES		140		105.0 150.0	
SODIUM: SERUM by ISE (ION SELECTIV	/E ELECTRODE)	142	mmol/L	135.0 - 150.0	
POTASSIUM: SERU		4.06	mmol/L	3.50 - 5.00	
by ISE (ION SELECTIV CHLORIDE: SERUM		106.5	mmol/L	90.0 - 110.0	
by ISE (ION SELECTIV	/E ELECTRODE)			00.0 110.0	
	MERULAR FILTERATION RATE				
	IERULAR FILTERATION RATE	107.5			
(eGFR): SERUM by CALCULATED					
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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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT



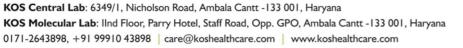


		<b>Dr. Vinay Chopra</b> MD (Pathology & Microbiology) Chairman & Consultant Pathologist		Dr. Yugam Chopra MD (Pathology) t CEO & Consultant Pathologist				
AME	: Mrs. MEENA	KSHI GUPTA						
GE/ GENDER	: 36 YRS/FEM	ALE	PA	TIENT ID	: 17091	21		
OLLECTED BY	:		RE	G. NO./LAB NO.	:0124	12260015		
EFERRED BY			RF	GISTRATION DA	<b>TE</b> · 26/De	ec/2024 10:3	87 AM	
ARCODE NO.	: 01523022			LLECTION DATE		$ec/2024\ 11:2$		
LIENT CODE.	: KOS DIAGNO			PORTING DATE		ec/202401:1		
				FORING DATE	. 20/ De	c/ 2024 01.1		
LIENT ADDRESS	: 6349/1, NIC	HOLSON ROAD, AMB	ALA CANTI					
Cest Name			Value	Unit	:	Biologica	al Reference	interval
. Reduced muscle m . Certain drugs (e.g. NCREASED RATIO (>2 . Postrenal azotemia	tetracycline, glu 0:1) WITH ELEVA a (BUN rises disp	creatinine production cocorticoids) <b>TED CREATININE LEV</b> roportionately more	ELS:	(e.g. obstructive	uropathy).			
Reduced muscle m     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.     Pregnancy.     Phenacimide thera     Rhabdomyolysis (r     Muscular patients     NAPPROPIATE RATIO     Diabetic ketoacido hould produce an in	ass (subnormal tetracycline, glu <b>io:1) WITH ELEVA</b> a (BUN rises disp superimposed of <b>io:1) WITH DECR</b> osis. ad starvation. b. creased urea syn urea rather than monemias (urea of inappropiate a <b>io:1) WITH INCRI</b> py (accelerates of eleases muscle of who develop ref sis (acetoacetat creased BUN/cro rapy (interferes of	creatinine production cocorticoids) <b>TED CREATININE LEV</b> roportionately more n renal disease. <b>EASED BUN :</b> thesis. creatinine diffuses of is virtually absent in ntidiuretic harmone) <b>EASED CREATININE:</b> conversion of creatin creatinine). hal failure. e causes false increase eatinine ratio). with creatinine measu	ELS: than creatinine) but of extracellu blood). due to tubular e to creatinine). e in creatinine irement).	llar fluid). secretion of urea.			al ratio when	dehydratio
. Reduced muscle m . Certain drugs (e.g. . VCREASED RATIO (>2 . Postrenal azotemia . Prerenal 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 GLOMERU	ass (subnormal tetracycline, glu of 1) WITH ELEVA (BUN rises disp superimposed of or 1) WITH DECR osis. and starvation. e. creased urea syn urea rather than monemias (urea of inappropiate a signappropiate a loc1) WITH INCRE py (accelerates of eleases muscle of who develop ref sis (acetoacetat creased BUN/cro rapy (interferes of JLAR FILTERATIO	creatinine production cocorticoids) <b>TED CREATININE LEV</b> roportionately more n renal disease. <b>EASED BUN :</b> the creatinine diffuses of is virtually absent in ntidiuretic harmone) <b>EASED CREATININE:</b> conversion of creatin creatinine). hal failure. the causes false increase extinine ratio). with creatinine measu	ELS: than creatinine) but of extracellu blood). due to tubular e to creatinine). e in creatinine rement).	ular fluid). secretion of urea. with certain meth	odologies,resul	FINDINGS	al ratio when	dehydratio
. Reduced muscle m . Certain drugs (e.g. . VCREASED RATIO (>2 . Postrenal azotemia . Prerenal 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. . Phenacimide thera . Rhabdomyolysis (r . Muscular patients . NapPROPIATE RATIO . Diabetic ketoacido hould produce an in . Cephalosporin thei STIMATED GLOMERU . CKD STAGE	ass (subnormal tetracycline, glu tetracycline, glu to:1) WITH ELEVA (BUN rises disp superimposed of (0:1) WITH DECR osis. ad starvation. e. creased urea syn urea rather than monemias (urea of inappropiate a finappropiate a (urea rather than monemias (urea of inappropiate a sis (accelerates of eleases muscle of who develop ref sis (acetoacetat creased BUN/cro apy (interferes of DLAR FILTERATIO	creatinine production cocorticoids) <b>TED CREATININE LEV</b> roportionately more n renal disease. <b>EASED BUN :</b> The thesis. a creatinine diffuses of is virtually absent in ntidiuretic harmone) <b>EASED CREATININE:</b> conversion of creatin creatinine). hal failure. the causes false increase extinine ratio). vith creatinine measu <b>V RATE:</b> <b>DESCRIPTION</b> mal kidney function dney damage with	ELS: than creatinine) but of extracellu blood). due to tubular e to creatinine). e in creatinine rement).	ular fluid). secretion of urea. with certain meth min/1.73m2)	odologies,resul ASSOCIATED No prote Presence of	FINDINGS inuria Protein ,	al ratio when	dehydratio
Reduced muscle m Certain drugs (e.g. ICREASED 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 IAPPROPIATE RATIO Diabetic ketoacido nould produce an in Cephalosporin ther STIMATED GLOMERI G1 G2	ass (subnormal tetracycline, glu tetracycline, glu to:1) WITH ELEVA (BUN rises disp superimposed of to:1) WITH DECR osis. and starvation. e. creased urea syn urea rather than monemias (urea of inappropiate a to:1) WITH INCRE py (accelerates of eleases muscle of who develop ref sis (acetoacetat creased BUN/cro apy (interferes v JLAR FILTERATIO	creatinine production cocorticoids) <b>TED CREATININE LEV</b> roportionately more n renal disease. <b>EASED BUN :</b> Athesis. a creatinine diffuses of is virtually absent in ntidiuretic harmone) <b>EASED CREATININE:</b> conversion of creatin creatinine). hal failure. Cases false increase extinine ratio). vith creatinine measu <b>VATE:</b> <b>DESCRIPTION</b> mal kidney function dney damage with prmal or high GFR	ELS: than creatinine) but of extracellu blood). due to tubular e to creatinine). e in creatinine rement). GFR ( mL/i	ular fluid). secretion of urea. with certain meth <u>min/1.73m2 )</u> >90	odologies,resul ASSOCIATED	FINDINGS inuria Protein ,	al ratio when	dehydratio
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Reduced muscle m Certain drugs (e.g. VCREASED 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 VAPPROPIATE RATIO Diabetic ketoacido nould produce an in Cephalosporin ther STIMATED GLOMERI G1 G2	ass (subnormal tetracycline, glu o:1) WITH ELEVA a (BUN rises disp superimposed o io:1) WITH DECR osis. ad starvation. e. creased urea syl urea rather that monemias (urea of inappropiate a finappropiate a io:1) WITH INCRE py (accelerates o eleases muscle o who develop rei sis (acetoacetat creased BUN/cro apy (interferes v JLAR FILTERATIO	creatinine production cocorticoids) <b>TED CREATININE LEV</b> roportionately more n renal disease. <b>EASED BUN :</b> Athesis. a creatinine diffuses of is virtually absent in ntidiuretic harmone) <b>EASED CREATININE:</b> conversion of creatin creatinine). hal failure. Cases false increase extinine ratio). vith creatinine measu <b>VATE:</b> <b>DESCRIPTION</b> mal kidney function dney damage with prmal or high GFR	ELS: than creatinine) but of extracellu blood). due to tubular e to creatinine). e in creatinine rement). GFR ( mL/i 60 3	ular fluid). secretion of urea. with certain meth <u>min/1.73m2 )</u> >90	odologies,resul ASSOCIATED No prote Presence of	FINDINGS inuria Protein ,	al ratio when	dehydratio



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









	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbiology Chairman & Consultant Pathol		(Pathology)
NAME	: Mrs. MEENAKSHI GUPTA		
AGE/ GENDER	: 36 YRS/FEMALE	PATIENT ID	: 1709121
<b>COLLECTED BY</b>	:	<b>REG. NO./LAB NO.</b>	: 012412260015
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<b>CLIENT ADDRESS</b>	: 6349/1, NICHOLSON ROAD, AMBALA CAN	ITT	
Test Name	Value	Unit	<b>Biological Reference interval</b>

COMMENTS:

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

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

ADVICE:

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

End Of Report \*\*\*





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