



	Dr. Vinay Chopr MD (Pathology & Micr Chairman & Consultar	obiology)		(Pathology)
NAME	: Mr. MANOJ GUPTA			
AGE/ GENDER	: 54 YRS/MALE		PATIENT ID	: 1674266
COLLECTED BY	:		REG. NO./LAB NO.	:012411170001
REFERRED BY	: CENTRAL PHOENIX CLUB (AMBA)	LA CANTT)	REGISTRATION DATE	: 17/Nov/2024 06:31 AM
BARCODE NO.	: 01520944		COLLECTION DATE	: 17/Nov/2024 06:36AM
CLIENT CODE. CLIENT ADDRESS	: KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON ROAD, AMB.		REPORTING DATE	: 17/Nov/2024 09:03AM
CLIENT ADDRESS	. 0349/1, NICHOLSON KOAD, AMD	ALA CANTI		
Test Name		Value	Unit	Biological Reference interval
RED BLOOD CELL			ELLNESS PANEL: 1.4 .00D COUNT (CBC)	4
HAEMOGLOBIN (H		15.4	gm/dL	12.0 - 17.0
by CALORIMETRIC			Ŭ	
RED BLOOD CELL (by HYDRO DYNAMIC I	(RBC) COUNT FOCUSING, ELECTRICAL IMPEDENCE	5.36 ^H	Millions	/cmm 3.50 - 5.00
PACKED CELL VOL	UME (PCV) automated hematology analyzer	47.9	%	40.0 - 54.0
MEAN CORPUSCUL	ACTOMATED HEMATOLOGY ANALYZER AR VOLUME (MCV) AUTOMATED HEMATOLOGY ANALYZER	89.4	fL	80.0 - 100.0
MEAN CORPUSCUL	LAR HAEMOGLOBIN (MCH) AUTOMATED HEMATOLOGY ANALYZER	28.8	pg	27.0 - 34.0
MEAN CORPUSCUI	AR HEMOGLOBIN CONC. (MCHC)	32.2	g/dL	32.0 - 36.0
RED CELL DISTRIB	BUTION WIDTH (RDW-CV)	14.5	%	11.00 - 16.00
RED CELL DISTRIB	SUTION WIDTH (RDW-SD) AUTOMATED HEMATOLOGY ANALYZER	48.6	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED		16.68	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INI by CALCULATED	DEX	24.24	RATIO	BETA THALASSEMIA TRAIT:<= 65.0
WHITE BLOOR OF				IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CE		6000		4000 11000
	Y BY SF CUBE & MICROSCOPY	6990	/cmm	4000 - 11000
	BLOOD CELLS (nRBCS) rt hematology analyzer	NIL		0.00 - 20.00
NUCLEATED RED H	BLOOD CELLS (nRBCS) % AUTOMATED HEMATOLOGY ANALYZER	NIL	%	< 10 %





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Test Name		Value	Unit	Biological Reference interval
DIFFERENTIAL LE	UCOCYTE COUNT (DLC)			
NEUTROPHILS	Y BY SF CUBE & MICROSCOPY	45 ^L	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY	Y BY SF CUBE & MICROSCOPY	49 ^H	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY	Y BY SF CUBE & MICROSCOPY	2	%	1 - 6
MONOCYTES	/ BY SF CUBE & MICROSCOPY	4	%	2 - 12
BASOPHILS	/ BY SF CUBE & MICROSCOPY	0	%	0 - 1
	CYTES (WBC) COUNT			
ABSOLUTE NEUTR	OPHIL COUNT / by sf cube & microscopy	3146	/cmm	2000 - 7500
ABSOLUTE LYMPH	OCYTE COUNT / by sf cube & microscopy	3425	/cmm	800 - 4900
ABSOLUTE EOSINC	PHIL COUNT / by sf cube & microscopy	140	/cmm	40 - 440
ABSOLUTE MONOC	YTE COUNT / by sf cube & microscopy	280	/cmm	80 - 880
ABSOLUTE BASOPI		0	/cmm	0 - 110
PLATELETS AND O	THER PLATELET PREDICTIVE	MARKERS.		
PLATELET COUNT by HYDRO DYNAMIC F	(PLT) OCUSING, ELECTRICAL IMPEDENCE	255000	/cmm	150000 - 450000
PLATELETCRIT (PC		0.28	%	0.10 - 0.36
MEAN PLATELET V		11	fL	6.50 - 12.0
PLATELET LARGE	CELL COUNT (P-LCC)	82000	/cmm	30000 - 90000
PLATELET LARGE	CELL RATIO (P-LCR)	32.2	%	11.0 - 45.0
PLATELET DISTRIE	BUTION WIDTH (PDW)	16.5	%	15.0 - 17.0
ADVICE		KINDLY CO	RRELATE CLINICALL	Y



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Test Name	Value	Unit	Biological Reference interval

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD



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	. 0010/1, 10101010001100110,			
Test Name		Value	Unit	Biological Reference interva
restrume			ome	Diological Meter ence inter va
	GLY	COSYLATED H	AEMOGLOBIN (HBA1C)	C C
	GLY MOGLOBIN (HbA1c):			C C
GLYCOSYLATED HAE WHOLE BLOOD	MOGLOBIN (HbA1c):	COSYLATED HA	AEMOGLOBIN (HBA1C)	U
GLYCOSYLATED HAE WHOLE BLOOD by HPLC (HIGH PERFORM	MOGLOBIN (HbA1c):	7.7 ^H	AEMOGLOBIN (HBA1C) %	4.0 - 6.4
GLYCOSYLATED HAE WHOLE BLOOD by HPLC (HIGH PERFORM ESTIMATED AVERAG by HPLC (HIGH PERFORM	MOGLOBIN (HbA1c):		AEMOGLOBIN (HBA1C)	U
GLYCOSYLATED HAE WHOLE BLOOD by HPLC (HIGH PERFORM ESTIMATED AVERAG by HPLC (HIGH PERFORM	MOGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) E PLASMA GLUCOSE	7.7 ^H	AEMOGLOBIN (HBA1C) %	4.0 - 6.4
GLYCOSYLATED HAE WHOLE BLOOD by HPLC (HIGH PERFORM ESTIMATED AVERAG by HPLC (HIGH PERFORM	MOGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) E PLASMA GLUCOSE	7.7 ^H 174.29 ^H	AEMOGLOBIN (HBA1C) % mg/dL	4.0 - 6.4
GLYCOSYLATED HAE WHOLE BLOOD by HPLC (HIGH PERFORM ESTIMATED AVERAG by HPLC (HIGH PERFORM INTERPRETATION:	MOGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) E PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY)	7.7 ^H 174.29 ^H BETES ASSOCIATION	AEMOGLOBIN (HBA1C) % mg/dL	4.0 - 6.4 60.00 - 140.00
GLYCOSYLATED HAE WHOLE BLOOD by HPLC (HIGH PERFORM ESTIMATED AVERAG by HPLC (HIGH PERFORM INTERPRETATION: NOT diab	MOGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) E PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN DIAE FERENCE GROUP Detic Adults >= 18 years	7.7 ^H 174.29 ^H BETES ASSOCIATION	AEMOGLOBIN (HBA1C) % mg/dL (ADA): /LATED HEMOGLOGIB (HBAIC) i <5.7	4.0 - 6.4 60.00 - 140.00
GLYCOSYLATED HAE WHOLE BLOOD by HPLC (HIGH PERFORM ESTIMATED AVERAG by HPLC (HIGH PERFORM INTERPRETATION: RE Non diab At f	MOGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) E PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN DIAE FERENCE GROUP Detic Adults >= 18 years Risk (Prediabetes)	7.7 ^H 174.29 ^H BETES ASSOCIATION	AEMOGLOBIN (HBA1C) % mg/dL (ADA): (LATED HEMOGLOGIB (HBAIC) i <5.7 5.7 - 6.4	4.0 - 6.4 60.00 - 140.00
GLYCOSYLATED HAE WHOLE BLOOD by HPLC (HIGH PERFORM ESTIMATED AVERAG by HPLC (HIGH PERFORM INTERPRETATION: RE Non diab At f	MOGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) E PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN DIAE FERENCE GROUP Detic Adults >= 18 years	7.7 ^H 174.29 ^H BETES ASSOCIATION	AEMOGLOBIN (HBA1C) % mg/dL (ADA): (LATED HEMOGLOGIB (HBAIC) i <5.7 5.7 - 6.4 >= 6.5	4.0 - 6.4 60.00 - 140.00
GLYCOSYLATED HAE WHOLE BLOOD by HPLC (HIGH PERFORM ESTIMATED AVERAG by HPLC (HIGH PERFORM INTERPRETATION: RE Non diab At f	MOGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) E PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN DIAE FERENCE GROUP Detic Adults >= 18 years Risk (Prediabetes)	7.7 ^H 174.29 ^H BETES ASSOCIATION GLYCOSY	AEMOGLOBIN (HBA1C) % mg/dL (ADA): 'LATED HEMOGLOGIB (HBAIC) i <5.7 5.7 - 6.4 >= 6.5 Age > 19 Years	4.0 - 6.4 60.00 - 140.00
GLYCOSYLATED HAE WHOLE BLOOD by HPLC (HIGH PERFORM ESTIMATED AVERAG by HPLC (HIGH PERFORM INTERPRETATION: RE Non diab At I Dia	MOGLOBIN (HbA1c): MANCE LIQUID CHROMATOGRAPHY) E PLASMA GLUCOSE MANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN DIAE FERENCE GROUP Detic Adults >= 18 years Risk (Prediabetes)	7.7 ^H 174.29 ^H BETES ASSOCIATION	AEMOGLOBIN (HBA1C) % mg/dL (ADA): 'LATED HEMOGLOGIB (HBAIC) i <5.7 5.7 - 6.4 >= 6.5 Age > 19 Years erapy: <7.0	4.0 - 6.4 60.00 - 140.00

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.

<7.5

Goal of therapy:

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 appropriate. 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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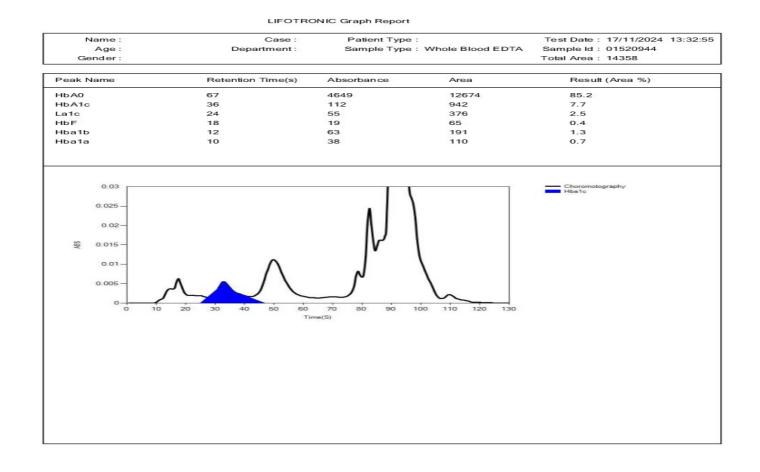


TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT





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Test Name	Value	Unit	Biological Reference interval





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by RED CELL AGGREC NTERPRETATION: . ESR is a non-specif mmune disease, but	DIMENTATION RATE (ESR) GATION BY CAPILLARY PHOTOMETI ic test because an elevated resu does not tell the health practitio	It often indicates the pr oner exactly where the i	mm/1st esence of inflammat nflammation is in the	hr 0 - 20





DR.YUGAM CHOPRA

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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	IBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	CLINICA		(/BIOCHEMIST	RY
GLUCOSE FASTING		GLUCOSE FAS	mg/dL	NORMAL: < 100.0

KOS Diagnostic Lab (A Unit of KOS Healthcare)

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





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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	GL	UCOSE POS	T PRANDIAL (PP)	
	ANDIAL (PP): PLASMA E - PEROXIDASE (GOD-POD)	147.89 ^H	mg/dL	NORMAL: < 140.00 PREDIABETIC: 140.0 - 200.0 DIABETIC: > 0R = 200.0

KOS Diagnostic Lab (A Unit of KOS Healthcare)

INTERPRETATION IN ACCORDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES: 1. A post-prandial plasma glucose level below 140 mg/dl is considered normal. 2. A post-prandial glucose level between 140 - 200 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 post-prandial plasma glucose level of above 200 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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BARCODE NO. : 01520944 CLIENT CODE. : KOS DIAGNO		раті		
CHOLESTEROL TOTAL: SERUM by CHOLESTEROL OXIDASE PAP TRIGLYCERIDES: SERUM by GLYCEROL PHOSPHATE OXIDASE (E HDL CHOLESTEROL (DIRECT): SE by SELECTIVE INHIBITION LDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMET	PHOENIX CLUB (AMBALA (NOSTIC LAB CHOLSON ROAD, AMBALA	REG. CANTT) REGE Coll Repo	ENT ID NO./LAB NO. STRATION DATE ECTION DATE DRTING DATE	: 1674266 : 012411170001 : 17/Nov/2024 06:31 AM : 17/Nov/2024 06:36AM : 17/Nov/2024 11:27AM
by CHOLESTEROL OXIDASE PAP TRIGLYCERIDES: SERUM by GLYCEROL PHOSPHATE OXIDASE (E HDL CHOLESTEROL (DIRECT): SE by SELECTIVE INHIBITION LDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMET NON HDL CHOLESTEROL: SERUM	Va	alue	Unit	Biological Reference interval
by CHOLESTEROL OXIDASE PAP TRIGLYCERIDES: SERUM by GLYCEROL PHOSPHATE OXIDASE (E HDL CHOLESTEROL (DIRECT): SE by SELECTIVE INHIBITION LDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMET NON HDL CHOLESTEROL: SERUM		ID PROFILI		OPTIMAL: < 200.0
by GLYCEROL PHOSPHATE OXIDASE (E HDL CHOLESTEROL (DIRECT): SE by SELECTIVE INHIBITION LDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMET	21	18.31 ^H	mg/dL	BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
by SELECTIVE INHIBITION LDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMET NON HDL CHOLESTEROL: SERUM		36.32	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
by CALCULATED, SPECTROPHOTOMET	ERUM 53	3.34	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
	TRY 1	37.71 ^H	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
		64.97 ^H	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159.0 BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMET		7.26	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM by CALCULATED, SPECTROPHOTOMET		72.94	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERU by CALCULATED, SPECTROPHOTOMET	RUM 4.	.09	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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Test Name		Value	Unit	Biological Reference interval
LDL/HDL RATIO: S by CALCULATED, SPE		2.58	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	2.56 ^L	RATIO	3.00 - 5.00

INTERPRETATION:

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

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

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

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





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	Dr. Vinay Chop MD (Pathology & Mi Chairman & Consult	crobiology)	Dr. Yugam MD (st CEO & Consultant	(Pathology)
NAME	: Mr. MANOJ GUPTA			
AGE/ GENDER	: 54 YRS/MALE		PATIENT ID	: 1674266
COLLECTED BY	:		REG. NO./LAB NO.	: 012411170001
REFERRED BY	: CENTRAL PHOENIX CLUB (AMB	ALA CANTT)	REGISTRATION DATE	: 17/Nov/2024 06:31 AM
BARCODE NO.	: 01520944		COLLECTION DATE	: 17/Nov/2024 06:36AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 17/Nov/2024 11:27AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	BALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	LIVER	FUNCTIO	N TEST (COMPLETE)	
BILIRUBIN TOTAL	: SERUM PECTROPHOTOMETRY	0.56	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
	C (CONJUGATED): SERUM	0.14	mg/dL	0.00 - 0.40
BILIRUBIN INDIRE	CT (UNCONJUGATED): SERUM	0.42	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PY	RIDOXAL PHOSPHATE	29	U/L	7.00 - 45.00
SGPT/ALT: SERUM	RIDOXAL PHOSPHATE	31.3	U/L	0.00 - 49.00
AST/ALT RATIO: S by CALCULATED, SPE	ERUM	0.93	RATIO	0.00 - 46.00
ALKALINE PHOSPI		91.68	U/L	40.0 - 130.0
GAMMA GLUTAMY by SZASZ, SPECTROF	L TRANSFERASE (GGT): SERUM PHTOMETRY	17.64	U/L	0.00 - 55.0
TOTAL PROTEINS: by BIURET, SPECTRO		7.63	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL G	REEN	4.32	gm/dL	3.50 - 5.50
GLOBULIN: SERUN by CALCULATED, SPE	1	3.31	gm/dL	2.30 - 3.50
A : G RATIO: SERU		1.31	RATIO	1.00 - 2.00

A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY

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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INTERPRETATION





	Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologis		(Pathology)
NAME	: Mr. MANOJ GUPTA		
AGE/ GENDER	: 54 YRS/MALE	PATIENT ID	: 1674266
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Test Name	Value	Unit	Biological Reference interval

DECREASED:

1. Acute Hepatitis due to virus, drugs, toxins (with AST increased 3 to 10 times upper limit of normal)

2. Extra Hepatic cholestatis: 0.8 (normal or slightly decreased).

PROGNOSTIC SIGNIFICANCE:

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



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NAME	: Mr. MANOJ GUPTA			
AGE/ GENDER	: 54 YRS/MALE		PATIENT ID	: 1674266
COLLECTED BY	:		REG. NO./LAB NO.	:012411170001
REFERRED BY	: CENTRAL PHOENIX CLUB (AM	BALA CANTT)	REGISTRATION DATE	: 17/Nov/2024 06:31 AM
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, Al	MBALA CANTT	,	
Test Name		Value	Unit	Biological Reference interval
	KIDNI	THE STATE OF THE S	N TEST (COMPLETE	
UREA: SERUM	IATE DEHYDROGENASE (GLDH)	36.32	mg/dL	10.00 - 50.00
CREATININE: SERU	UM	1.25	mg/dL	0.40 - 1.40
BLOOD UREA NITE	COGEN (BUN): SERUM	16.97	mg/dL	7.0 - 25.0
BLOOD UREA NITE RATIO: SERUM by CALCULATED, SPE	ROGEN (BUN)/CREATININE	13.58	RATIO	10.0 - 20.0
UREA/CREATININ by CALCULATED, SPE	E RATIO: SERUM	29.06	RATIO	
URIC ACID: SERUM	1	4.86	mg/dL	3.60 - 7.70
CALCIUM: SERUM by ARSENAZO III, SPE		10.21	mg/dL	8.50 - 10.60
PHOSPHOROUS: SE by PHOSPHOMOLYBE	ERUM DATE, SPECTROPHOTOMETRY	3.33	mg/dL	2.30 - 4.70
<u>ELECTROLYTES</u>				
SODIUM: SERUM by ISE (ION SELECTIV	(E ELECTRODE)	142.6	mmol/L	135.0 - 150.0
POTASSIUM: SERU by ISE (ION SELECTIV	M	4.72	mmol/L	3.50 - 5.00
CHLORIDE: SERUM by ISE (ION SELECTIV		106.95	mmol/L	90.0 - 110.0
ESTIMATED GLON	IERULAR FILTERATION RATE			
(eGFR): SERUM by CALCULATED	ERULAR FILTERATION RATE	68.4		
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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AME	: Mr. MANOJ GUPTA			
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LIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT		
'est Name		Value	Unit E	Biological Reference interval
ICREASED RATIO (>2 Postrenal azotemia Prerenal azotemia	superimposed on renal disease.	E LEVELS: nore than creatinine) (e.g. obstruc	tive uropathy).	
VCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia VECREASED RATIO (< Acute tubular necr Low protein diet ar Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. VECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients VAPPROPIATE RATIO Diabetic ketoacido hould produce an in Cephalosporin ther STIMATED GLOMERU CKD STAGE	tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE (BUN rises disproportionately m superimposed on renal disease. 10:1) WITH DECREASED BUN : osis. ad starvation. b. creased urea synthesis. urea rather than creatinine diffu monemias (urea is virtually absect in appropriate antidiuretic harm 10:1) WITH INCREASED CREATININ py (accelerates conversion of creatine). who develop renal failure. 1: sis (acetoacetate causes false in- creased BUN/creatinine ratio). apy (interferes with creatinine m JLAR FILTERATION RATE: DESCRIPTION	E LEVELS: nore than creatinine) (e.g. obstruct uses out of extracellular fluid). ent in blood). none) due to tubular secretion of the NE: eatine to creatinine). crease in creatinine with certain neasurement).	urea. methodologies,resulting	
JCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia ECREASED RATIO (< Acute tubular necr Low protein diet an Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. ECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients JAPPROPIATE RATIO Diabetic ketoacido nould produce an in Cephalosporin ther STIMATED GLOMERI CKD STAGE G1	tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE (BUN rises disproportionately m superimposed on renal disease. 10:1) WITH DECREASED BUN : osis. d starvation. 2. creased urea synthesis. urea rather than creatinine diffu- monemias (urea is virtually absect in appropriate antidiuretic harman 10:1) WITH INCREASED CREATININ py (accelerates conversion of crea- eleases muscle creatinine). who develop renal failure. : sis (acetoacetate causes false in- creased BUN/creatinine ratio). apy (interferes with creatinine m JLAR FILTERATION RATE: <u>DESCRIPTION</u> Normal kidney functioned 10:1)	E LEVELS: nore than creatinine) (e.g. obstruct uses out of extracellular fluid). ent in blood). none) due to tubular secretion of the NE: eatine to creatinine). Increase in creatinine with certain neasurement). GFR (mL/min/1.73m2 tion >90	urea. methodologies,resulting ASSOCIATED FIN No proteinu	DINGS ria
CREASED RATIO (>2 Postrenal azotemia Prerenal azotemia CREASED RATIO (< Acute tubular necr Low protein diet an Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. CREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients APPROPIATE RATIO Diabetic ketoacido ould produce an in Cephalosporin ther TIMATED GLOMERU CKD STAGE	tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE (BUN rises disproportionately m superimposed on renal disease. 10:1) WITH DECREASED BUN : osis. ad starvation. b. creased urea synthesis. urea rather than creatinine diffu monemias (urea is virtually absect in appropriate antidiuretic harm 10:1) WITH INCREASED CREATININ py (accelerates conversion of creatine). who develop renal failure. 1: sis (acetoacetate causes false in- creased BUN/creatinine ratio). apy (interferes with creatinine m JLAR FILTERATION RATE: DESCRIPTION Normal kidney functioned Kidney damage with	E LEVELS: nore than creatinine) (e.g. obstruct uses out of extracellular fluid). ent in blood). none) due to tubular secretion of the NE: eatine to creatinine). Crease in creatinine with certain neasurement). GFR (mL/min/1.73m2 tion >90 th >90	urea. methodologies,resulting	DINGS ria otein ,
CREASED RATIO (>2 Postrenal azotemia Prerenal azotemia CREASED RATIO (< Acute tubular necr Low protein diet ar Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. CREASED RATIO (< Phenacimide thera Rhabdomyolysis (r Muscular patients APPROPIATE RATIO Diabetic ketoacido ould produce an in Cephalosporin ther TIMATED GLOMERI CKD STAGE G1	tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE (BUN rises disproportionately m superimposed on renal disease. 10:1) WITH DECREASED BUN : osis. ad starvation. 2. creased urea synthesis. urea rather than creatinine diffu monemias (urea is virtually absect of inappropiate antidiuretic harm 10:1) WITH INCREASED CREATININ py (accelerates conversion of creatine). who develop renal failure. 1: sis (acetoacetate causes false in- creased BUN/creatinine ratio). apy (interferes with creatinine m ILAR FILTERATION RATE: DESCRIPTION Normal kidney function Kidney damage wit- normal or high GF Mild decrease in G	E LEVELS: nore than creatinine) (e.g. obstruct uses out of extracellular fluid). ent in blood). none) due to tubular secretion of the NE: eatine to creatinine). NE: eatine to creatinine with certain neasurement). GFR (mL/min/1.73m2 tion >90 th >90 FR 60 -89	methodologies,resulting ASSOCIATED FIN No proteinu Presence of Pro	DINGS ria otein ,
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 G3a G3a G3b	tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE (BUN rises disproportionately m superimposed on renal disease. 10:1) WITH DECREASED BUN : osis. ad starvation. 2. creased urea synthesis. urea rather than creatinine diffu monemias (urea is virtually absect of inappropiate antidiuretic harm 10:1) WITH INCREASED CREATININ py (accelerates conversion of creatine). who develop renal failure. 1: sis (acetoacetate causes false in- creased BUN/creatinine ratio). apy (interferes with creatinine m ILAR FILTERATION RATE: DESCRIPTION Normal kidney function Kidney damage withor and or high GF Mild decrease in G Moderate decrease in G	E LEVELS: nore than creatinine) (e.g. obstruct uses out of extracellular fluid). ent in blood). none) due to tubular secretion of the NE: eatine to creatinine). NE: eatine to creatinine with certain neasurement). GFR (mL/min/1.73m2 tion >90 th >90 th >90 FR 60 -89 n GFR 30-59	methodologies,resulting ASSOCIATED FIN No proteinu Presence of Pro	DINGS ria otein ,
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 the STIMATED GLOMERI G1 G2 G3a	tetracycline, glucocorticoids) 0:1) WITH ELEVATED CREATININE (BUN rises disproportionately m superimposed on renal disease. 10:1) WITH DECREASED BUN : osis. ad starvation. 2. creased urea synthesis. urea rather than creatinine diffu monemias (urea is virtually absect of inappropiate antidiuretic harm 10:1) WITH INCREASED CREATININ py (accelerates conversion of creatine). who develop renal failure. 1: sis (acetoacetate causes false in- creased BUN/creatinine ratio). apy (interferes with creatinine m ILAR FILTERATION RATE: DESCRIPTION Normal kidney function Kidney damage wit- normal or high GF Mild decrease in G	E LEVELS: nore than creatinine) (e.g. obstruct uses out of extracellular fluid). ent in blood). none) due to tubular secretion of the NE: eatine to creatinine). NE: eatine to creatinine with certain neasurement). GFR (mL/min/1.73m2 tion >90 th >90 th >90 FR 60 -89 n GFR 30-59	methodologies,resulting ASSOCIATED FIN No proteinu Presence of Pro	DINGS ria otein ,





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Test Name	Value	Unit	Biological Reference interval
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANT	Г	
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 17/Nov/2024 11:27AM
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NAME	: Mr. MANOJ GUPTA		
	Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologi		(Pathology)

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 Name		Value	Unit	Biological Reference interval
		IRON P	PROFILE	
IRON: SERUM by FERROZINE, SPEC	TROPHOTOMETRY	105.1	μg/dL	59.0 - 158.0
SERUM	ON BINDING CAPACITY (UIBC) 214.79	μg/dL	150.0 - 336.0
by FERROZINE, SPEC TOTAL IRON BIND :SERUM by SPECTROPHOTOM	ING CAPACITY (TIBC)	319.89	µg/dL	230 - 430
%TRANSFERRIN S	ATURATION: SERUM	32.86	%	15.0 - 50.0
TRANSFERRIN: SE by SPECTROPHOTOM	RUM	227.12	mg/dL	200.0 - 350.0
INTERPRETATION:-				
VARIAB	BLES ANEMIA OF CH	RONIC DISEASE	IRON DEFICIENCY ANEMIA	A THALASSEMIA α/β TRAIT

	IRON DEFICIENCY ANEMIA	THALASSEMIA α/β TRAIT
Normal to Reduced	Reduced	Normal
Decreased	Increased	Normal
Decreased	Decreased < 12-15 %	Normal
Normal to Increased	Decreased	Normal or Increased
	Decreased Decreased	DecreasedIncreasedDecreasedDecreased < 12-15 %

IRON:

1.Serum iron studies is recommended for differential diagnosis of microcytic hypochromic anemia.i.e iron deficiency anemia, zinc deficiency anemia, anemia of chronic disease and thalassemia syndromes.

It is essential to isolate iron deficiency anemia from Beta thalassemia syndromes because during iron replacement which is therapeutic for iron deficiency anemia, is severely contra-indicated in Thalassemia.
 TOTAL IRON BINDING CAPACITY (TIBC):

1.It is a direct measure of protein transferrin which transports iron from the gut to storage sites in the bone marrow.

% TRANSFERRIN SATURATION:

1. Occurs in idiopathic hemochromatosis and transfusional hemosiderosis where no unsaturated iron binding capacity is available for iron mobilization. Similar condition is seen in congenital deficiency of transferrin.



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	Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologis		(Pathology)
NAME	: Mr. MANOJ GUPTA		
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT	2	
Test Name	Value	Unit	Biological Reference interval
	ENDOC	RINOLOGY	
	THYROID FUNC	CTION TEST: TOTAL	
TRIIODOTHYRONIN	NE (T3): SERUM 0.932 ESCENT MICROPARTICLE IMMUNOASSAY)	ng/mL	0.35 - 1.93
by CMIA (CHEMILUMIN		ugm /dI	4.87 - 12.60
THYROXINE (T4): S	ERUM 8.77 ESCENT MICROPARTICLE IMMUNOASSAY)	µgm/dL	4.87 - 12.00
THYROXINE (T4): S by CMIA (CHEMILUMINE THYROID STIMULA		μgin/dL μIU/mL	0.35 - 5.50
THYROXINE (T4): S by CMIA (CHEMILUMINE THYROID STIMULA	ESCENT MICROPARTICLE IMMUNOASSAY) TING HORMONE (TSH): SERUM ESCENT MICROPARTICLE IMMUNOASSAY)		
THYROXINE (T4): S by CMIA (CHEMILUMINI THYROID STIMULA by CMIA (CHEMILUMINI 3rd GENERATION, ULTE INTERPRETATION: TSH levels are subject to c day has influence on the n	ESCENT MICROPARTICLE IMMUNOASSAY) TING HORMONE (TSH): SERUM 2.862 ESCENT MICROPARTICLE IMMUNOASSAY) RASENSITIVE ircadian variation, reaching peak levels between 2-4 a.m ar neasured serum TSH concentrations. TSH stimulates the pri- ure at any level of regulation of the hypothalamic-pituitar	µIU/mL and at a minimum between 6-10 p roduction and secretion of the m	0.35 - 5.50 m. The variation is of the order of 50%.Hence time of the tabolically active hormones, thyroxine (T4)and

CLINICAL CONDITION	T3	T4	TSH
Primary Hypothyroidism:	Reduced	Reduced	Increased (Significantly)
Subclinical Hypothyroidism:	Normal or Low Normal	Normal or Low Normal	High
Primary Hyperthyroidism:	Increased	Increased	Reduced (at times undetectable)
Subclinical Hyperthyroidism:	Normal or High Normal	Normal or High Normal	Reduced

LIMITATIONS:-

1. T3 and T4 circulates in reversibly bound form with Thyroid binding globulins (TBG), and to a lesser extent albumin and Thyroid binding Pre Albumin so conditions in which TBG and protein levels alter such as pregnancy, excess estrogens, androgens, anabolic steroids and glucocorticoids may falsely affect the T3 and T4 levels and may cause false thyroid values for thyroid function tests.

2. Normal levels of T4 can also be seen in Hyperthyroid patients with :T3 Thyrotoxicosis, Decreased binding capacity due to hypoproteinemia or ingestion of certain drugs (e.g.: phenytoin , salicylates).

3. Serum T4 levels in neonates and infants are higher than values in the normal adult , due to the increased concentration of TBG in neonate serum.

4. TSH may be normal in central hypothyroidism , recent rapid correction of hyperthyroidism or hypothyroidism , pregnancy , phenytoin therapy.

TRIIODOTH	YRONINE (T3)	THYROXINE (T4)		THYROID STIMULATING HORMONE (TSH)		
Age	Refferance Range (ng/mL)	Age	Refferance Range (µg/dL)	Age	Reference Range (μIU/mL)	
0 - 7 Days	0.20 - 2.65	0 - 7 Days	5.90 - 18.58	0 - 7 Days	2.43 - 24.3	
7 Days - 3 Months	0.36 - 2.59	7 Days - 3 Months	6.39 - 17.66	7 Days - 3 Months	0.58 - 11.00	
3 - 6 Months	0.51 - 2.52	3 - 6 Months	6.75 - 17.04	3 Days – 6 Months	0.70 - 8.40	
6 - 12 Months	0.74 - 2.40	6 - 12 Months	7.10 - 16.16	6 – 12 Months	0.70 - 7.00	





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

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





TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT





	Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologis		(Pathology)
NAME	: Mr. MANOJ GUPTA		
AGE/ GENDER	: 54 YRS/MALE	PATIENT ID	: 1674266
COLLECTED BY	:	REG. NO./LAB NO.	: 012411170001
REFERRED BY	: CENTRAL PHOENIX CLUB (AMBALA CANTT)	REGISTRATION DATE	: 17/Nov/2024 06:31 AM
BARCODE NO.	: 01520944	COLLECTION DATE	: 17/Nov/2024 06:36AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 17/Nov/2024 11:27AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name		Value	Value Unit		Biological Reference interval	
1 - 10 Years	0.92 - 2.28	1 - 10 Years	6.00 - 13.80	1 – 10 Years	0.60 - 5.50	
11- 19 Years	0.35 - 1.93	11 - 19 Years	4.87-13.20	11 – 19 Years	0.50 - 5.50	
> 20 years (Adults)	0.35 - 1.93	> 20 Years (Adults)	4.87 - 12.60	> 20 Years (Adults)	0.35-5.50	
	RECON	IMENDATIONS OF TSH LI	VELS DURING PRE	GNANCY (µIU/mL)		
	1st Trimester			0.10 - 2.50		
	2nd Trimester			0.20 - 3.00		
	3rd Trimester			0.30 - 4.10		

INCREASED TSH LEVELS:

1. Primary or untreated hypothyroidism may vary from 3 times to more than 100 times normal depending upon degree of hypofunction.

2. Hypothyroid patients receiving insufficient thyroid replacement therapy.

3. Hashimotos thyroiditis

4.DRUGS: Amphetamines, iodine containing agents & dopamine antagonist.

5.Neonatal period, increase in 1st 2-3 days of life due to post-natal surge

DECREASED TSH LEVELS:

1. Toxic multi-nodular goiter & Thyroiditis.

2. Over replacement of thyroid hormone in treatment of hypothyroidism.

3. Autonomously functioning Thyroid adenoma

4. Secondary pituitary or hypothalamic hypothyroidism

5. Acute psychiatric illness

6.Severe dehydration.

7.DRUGS: Glucocorticoids, Dopamine, Levodopa, T4 replacement therapy, Anti-thyroid drugs for thyrotoxicosis.

8.Pregnancy: 1st and 2nd Trimester





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







	Dr. Vinay Cho MD (Pathology & N Chairman & Consu	1icrobiology)	Dr. Yugam Chopra MD (Pathology) CEO & Consultant Pathologist				
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BARCODE NO.	:01520944						
CLIENT CODE.	: KOS DIAGNOSTIC LAB REPORTING DAT			: 17/Nov/2024 01:19PM			
CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT							
Test Name		Value	Unit	Biological Reference interval			
CLINICAL PATHOLOGY URINE ROUTINE & MICROSCOPIC EXAMINATION							
PHYSICAL EXAMIN	NATION						
QUANTITY RECIEV		10	ml				
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY COLOUR		PALE YELLOW		PALE YELLOW			
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY						
TRANSPARANCY by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY		HAZY		CLEAR			
SPECIFIC GRAVITY		1.02		1.002 - 1.030			
	TANCE SPECTROPHOTOMETRY						
CHEMICAL EXAMI REACTION	NATION	ACIDIC					
	TANCE SPECTROPHOTOMETRY	ACIDIC					
PROTEIN		Negative		NEGATIVE (-ve)			
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY SUGAR		1+		NEGATIVE (-ve)			
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY							
pH by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	<=5.0		5.0 - 7.5			
BILIRUBIN		Negative		NEGATIVE (-ve)			
NITRITE	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)			
UROBILINOGEN	TANCE SPECTROPHOTOMETRY.	Normal	EU/dL	0.2 - 1.0			
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)			
	TANCE SPECTROPHOTOMETRY						
BLOOD	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)			
ASCORBIC ACID	TABLE OF LOT NOF HOT OWEIRT	NEGATIVE (-ve)		NEGATIVE (-ve)			
by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY							
MICROSCOPIC EXA			/HDF	0.2			
RED BLOOD CELLS by MICROSCOPY ON C	(RBCs) CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)	/HPF	0 - 3			





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

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

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Test Name	Value	Unit	Biological Reference interval			
DUCCELLS	2 /	/UDF	0.5			

PUS CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	2-4	/HPF	0 - 5
EPITHELIAL CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	1-3	/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



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

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