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	Dr. Vinay Cho MD (Pathology & Chairman & Const	Microbiology)	Dr. Yugam ( MD (P CEO & Consultant Pa	athology)
NAME	: Mr. R.P GUPTA			
AGE/ GENDER	: 88 YRS/MALE	Р	ATIENT ID	: 1629365
COLLECTED BY	: SURJESH	R	EG. NO./LAB NO.	: 012409300026
REFERRED BY	:	R	EGISTRATION DATE	: 30/Sep/2024 10:06 AM
BARCODE NO.	: 01517999	C	OLLECTION DATE	: 30/Sep/2024 10:25AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	R	EPORTING DATE	: 30/Sep/2024 10:41AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	SW	ASTHYA WELI	LNESS PANEL: 1.5	
	c	OMPLETE BLOG	OD COUNT (CBC)	
RED BLOOD CELLS (R	BCS) COUNT AND INDICES			
HAEMOGLOBIN (HB)		11.9 <sup>L</sup>	gm/dL	12.0 - 17.0
RED BLOOD CELL (RB	C) COUNT OCUSING, ELECTRICAL IMPEDENCE	4.25	Millions/cm	m 3.50 - 5.00
PACKED CELL VOLUN		36.7 <sup>L</sup>	%	40.0 - 54.0
MEAN CORPUSCULA		86.3	fL	80.0 - 100.0
	R HAEMOGLOBIN (MCH) UTOMATED HEMATOLOGY ANALYZE	28 R	pg	27.0 - 34.0
MEAN CORPUSCULA	R HEMOGLOBIN CONC. (MCHC)	32.5	g/dL	32.0 - 36.0
RED CELL DISTRIBUT	ION WIDTH (RDW-CV)	14.9	%	11.00 - 16.00
RED CELL DISTRIBUT	ION WIDTH (RDW-SD) UTOMATED HEMATOLOGY ANALYZE	48.2	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED		20.31	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDE. by calculated	X	30.26	RATIO	BETA THALASSEMIA TRAIT:<= 65. IRON DEFICIENCY ANEMIA: > 65.0
NHITE BLOOD CELLS	<u>(WBCS)</u>			
TOTAL LEUCOCYTE C	OUNT (TLC) ' by sf cube & microscopy	6920	/cmm	4000 - 11000
NUCLEATED RED BLC		NIL		0.00 - 20.00
NUCLEATED RED BLC	OOD CELLS (nRBCS) % UTOMATED HEMATOLOGY ANALYZE	NIL R	%	< 10 %
NEUTROPHILS by flow cytometry	BY SF CUBE & MICROSCOPY	52	%	50 - 70



**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





MD (Pathology & Microbiology) Chairman & Consultant Pathologist

Dr. Vinay Chopra



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

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Test Name	Value	Unit	Biological Reference interval
LYMPHOCYTES	31	%	20 - 40
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY EOSINOPHILS	5	%	1 - 6
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	5	70	1-0
MONOCYTES	12 <sup>H</sup>	%	2 - 12
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	%	0 - 1
BASOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	%	0 - 1
ABSOLUTE LEUKOCYTES (WBC) COUNT			
ABSOLUTE NEUTROPHIL COUNT	3598	/cmm	2000 - 7500
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	/		
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2145	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT	346	/cmm	40 - 440
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	830	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT	0	/cmm	0 - 110
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY		, on the	0 110
PLATELETS AND OTHER PLATELET PREDICTIVE MARKE	<u>RS.</u>		
PLATELET COUNT (PLT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	347000	/cmm	150000 - 450000
PLATELETCRIT (PCT)	0.31	%	0.10 - 0.36
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
	9	fL	6.50 - 12.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET LARGE CELL COUNT (P-LCC)	66000	/cmm	30000 - 90000
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELET LARGE CELL RATIO (P-LCR)	18.9	%	11.0 - 45.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE PLATELET DISTRIBUTION WIDTH (PDW)	16	%	15.0 - 17.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	10	70	13.0 17.0
NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD			



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

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 KOS Molecular Lab: IInd Floor, Parry Hotel, Staff Road, Opp. GPO, Ambala Cantt -133 001, Haryana

 0171-2643898, +91 99910 43898
 care@koshealthcare.com

 www.koshealthcare.com
 www.koshealthcare.com







	Dr. Vinay ChopraDr. Yugam ChopraMD (Pathology & Microbiology)MD (Pathology)Chairman & Consultant PathologistCEO & Consultant Pathologist				
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BARCODE NO.	:01517999	COLLEG	TION DATE	: 30/Sep/2024 02:12PM	
CLIENT CODE.	: KOS DIAGNOSTIC LAB		TING DATE	: 30/Sep/2024 02:38PM	
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A				
Test Name		Value	Unit	Biological Reference in	terval
	GLY	COSYLATED HAEMOG	LOBIN (HBA1C)		
GLYCOSYLATED HAEM		6.3	%	4.0 - 6.4	
WHOLE BLOOD		0.0	70	1.0 0.1	
by HPLC (HIGH PERFORI ESTIMATED AVERAGE	MANCE LIQUID CHROMATOGRAPHY)	134.11	mg/dL	60.00 - 140.00	
	MANCE LIQUID CHROMATOGRAPHY)	134.11	Thy/uL	00.00 - 140.00	
INTERPRETATION:					
	AS PER AMERICAN	DIABETES ASSOCIATION (A	DA):		
R	EFERENCE GROUP		ATED HEMOGLOGIB	(HBAIC) in %	
	petic Adults >= 18 years	/	<5.7		
	At Risk (Prediabetes) 5.7 – 6.4				
Non diat At		>= 6.5			
Non diat At	ignosing Diabetes				
Non diat At			Age > 19 Years		
Non diat At Dia		Goals of Thera Actions Sugges	Age > 19 Years	< 7.0 >8.0	

### 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.

Goal of therapy:

<7.5

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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 0171-2643898, +91 99910 43898
 care@koshealthcare.com
 www.koshealthcare.com



TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT





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CLIENT CODE.	: KOS DIAGNOSTIC LAB	<b>REPORTING DATE</b>	: 30/Sep/2024 10:59AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT	
Test Name		Value Unit	Biological Reference interval
	ERYTH	ROCYTE SEDIMENTATION RATE (ES	SR)
	MENTATION RATE (ESR)	39 <sup>H</sup> mm/1st	hr 0 - 20
1. ESR is a non-specif immune disease, but 2. An ESR can be affe as C-reactive protein	does not tell the health practition ected by other conditions besides in be used to monitor disease activit ematosus	er exactly where the inflammation is in th nflammation. For this reason, the ESR is ty	tion associated with infection, cancer and auto ne body or what is causing it. ypically used in conjunction with other test such above diseases as well as some others, such as
A low ESR can be see (polycythaemia), sign	en with conditions that inhibit the	normal sedimentation of red blood cells, s int (leucocytosis) , and some protein abno R.	such as a high red blood cell count ormalities. Some changes in red cell shape (suc

#### NOTE:

 ESR and C - reactive protein (C-RP) are both markers of inflammation.
 Generally, ESR does not change as rapidly as does CRP, either at the start of inflammation or as it resolves.
 **CRP is not affected by as many other factors as is ESR, making it a better marker of inflammation.** If the ESR is elevated, it is typically a result of two types of proteins, globulins or fibrinogen.
 Women tend to have a higher ESR, and menstruation and pregnancy can cause temporary elevations.
 Drugs such as dextran, methyldopa, oral contraceptives, penicillamine procainamide, theophylline, and vitamin A can increase ESR, while exprise contrace and quiping may decrease it. aspirin, cortisone, and quinine may decrease it



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	MD (Pathology & Chairman & Cor	& Microbiology) nsultant Pathologist	MD CEO & Consultant	(Pathology) Pathologist
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CLIENT CODE.	: KOS DIAGNOSTIC LAB : 6349/1, NICHOLSON ROAD,		ORTING DATE	: 30/Sep/2024 11:35AM
CLIENT CODE. CLIENT ADDRESS Test Name			DRTING DATE	: 30/Sep/2024 11:35AM Biological Reference interval
CLIENT CODE. CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT	Unit	Biological Reference interval
CLIENT CODE. CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD,	AMBALA CANTT Value	Unit /BIOCHEMISTR	Biological Reference interval

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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		hopra & Microbiology) nsultant Pathologist	Dr. Yugam MD CEO & Consultant	(Pathology)
NAME	: Mr. R.P GUPTA			
AGE/ GENDER	: 88 YRS/MALE		ENT ID	: 1629365
COLLECTED BY	: SURJESH		NO./LAB NO.	:012409300026
REFERRED BY	: : 01517999		STRATION DATE	: 30/Sep/2024 10:06 AM
BARCODE NO. CLIENT CODE.	: KOS DIAGNOSTIC LAB		ECTION DATE DRTING DATE	: 30/Sep/2024 10:25AM : 30/Sep/2024 11:35AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD		DATE	. 50/ Sep/ 2024 11.55AM
Test Name		Value	Unit	Biological Reference interval
		LIPID PROFILE	: BASIC	
CHOLESTEROL TOTA by CHOLESTEROL OX		172.35	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.
TRIGLYCERIDES: SER by GLYCEROL PHOSP	UM HATE OXIDASE (ENZYMATIC)	91.53	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROL ( by SELECTIVE INHIBITI		41.33	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: S by CALCULATED, SPE		112.71	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129.0 BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0
NON HDL CHOLESTE by calculated, spe		131.02 <sup>H</sup>	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:		18.31	mg/dL	0.00 - 45.00
by CALCULATED, SPE TOTAL LIPIDS: SERUN by CALCULATED, SPE	N	436.23	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL F by CALCULATED, SPE		4.17	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0
LDL/HDL RATIO: SER by CALCULATED, SPE		2.73	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0

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

57

2.56

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

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Test Name		Value	Unit	Biological Reference interval
TRIGLYCERIDES/HD	L RATIO: SERUM ECTROPHOTOMETRY	2.21 <sup>L</sup>	RATIO	3.00 - 5.00

### **INTERPRETATION:**

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

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

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

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





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Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Pathologist TTA E BATTIENT ID 100000

Unit

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Value

			3	
LIV	ER FUNCTION TE	ST (COMPLETE)		
BILIRUBIN TOTAL: SERUM by diazotization, spectrophotometry	0.42	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20	
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.12	mg/dL	0.00 - 0.40	
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.3	mg/dL	0.10 - 1.00	
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	15.3	U/L	7.00 - 45.00	
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	10.7	U/L	0.00 - 49.00	
AST/ALT RATIO: SERUM by calculated, spectrophotometry	1.43	RATIO	0.00 - 46.00	
ALKALINE PHOSPHATASE: SERUM by Para NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL	66.15	U/L	40.0 - 130.0	
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by szasz, spectrophtometry	22.12	U/L	0.00 - 55.0	
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	6.31	gm/dL	6.20 - 8.00	
ALBUMIN: SERUM by bromocresol green	3.46 <sup>L</sup>	gm/dL	3.50 - 5.50	
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.85	gm/dL	2.30 - 3.50	
A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.21	RATIO	1.00 - 2.00	

## **INTERPRETATION**

NOTE: To be correlated in individuals having SGOT and SGPT values higher than Normal Referance Range.

USE:- Differential diagnosis of diseases of hepatobiliary system and pancreas.

# INCREASED:

DRUG HEPATOTOXICITY	> 2
ALCOHOLIC HEPATITIS	> 2 (Highly Suggestive)
CIRRHOSIS	1.4 - 2.0
INTRAHEPATIC CHOLESTATIS	> 1.5
HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS	> 1.3 (Slightly Increased)





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



**Biological Reference interval** 

Test Name

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#### DECREASED:

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

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

### PROGNOSTIC SIGNIFICANCE:

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



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<b>CLIENT ADDRESS</b> : 6349/1, N	ICHOLSON ROAD, AMBALA CANT	T	
Test Name	Value	Unit	Biological Reference interval
UREA: SERUM by UREASE - GLUTAMATE DEHYDRO CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOME BLOOD UREA NITROGEN (BUN): S by CALCULATED, SPECTROPHOTOM	31.59 GENASE (GLDH) 0.95 TERY SERUM 14.76	TION TEST (COMPLETE) mg/dL mg/dL mg/dL	10.00 - 50.00 0.40 - 1.40 7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/C RATIO: SERUM by calculated, spectrophotom	CREATININE 15.54	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUN by CALCULATED, SPECTROPHOTOM		RATIO	
URIC ACID: SERUM by URICASE - OXIDASE PEROXIDAS	е 7.77 <sup>Н</sup>	mg/dL	3.60 - 7.70
CALCIUM: SERUM by ARSENAZO III, SPECTROPHOTOM	8.74 ETRY	mg/dL	8.50 - 10.60
PHOSPHOROUS: SERUM by PHOSPHOMOLYBDATE, SPECTRO ELECTROLYTES	3.26	mg/dL	2.30 - 4.70
SODIUM: SERUM by ise (ion selective electrode)	137.8	mmol/L	135.0 - 150.0
POTASSIUM: SERUM	4.67	mmol/L	3.50 - 5.00

103.35

1. Prerenal azotemia (BUN rises without increase in creatinine) e.g. heart failure, salt depletion, dehydration, blood loss) due to decreased

77

glomerular filtration rate.

To differentiate between pre- and post renal azotemia. INCREASED RATIO (>20:1) WITH NORMAL CREATININE:

2. Catabolic states with increased tissue breakdown.

by ISE (ION SELECTIVE ELECTRODE)

by ISE (ION SELECTIVE ELECTRODE)

ESTIMATED GLOMERULAR FILTERATION RATE ESTIMATED GLOMERULAR FILTERATION RATE

CHLORIDE: SERUM

(eGFR): SERUM by CALCULATED INTERPRETATION:



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

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

mmol/L

90.0 - 110.0

 KOS Central Lab: 6349/1, Nicholson Road, Ambala Cantt - 133 001, Haryana

 KOS Molecular Lab: IInd Floor, Parry Hotel, Staff Road, Opp. GPO, Ambala Cantt - 133 001, Haryana

 0171-2643898, +91 99910 43898
 care@koshealthcare.com

 www.koshealthcare.com



TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT





		y <b>Chopra</b> ogy & Microbiology) & Consultant Pathologist	Dr. Yugam MD CEO & Consultant	(Pathology)
NAME	: Mr. R.P GUPTA			
AGE/ GENDER	: 88 YRS/MALE	PATIE	NT ID	: 1629365
COLLECTED BY	: SURJESH	REG. N	O./LAB NO.	: 012409300026
REFERRED BY			<b>FRATION DATE</b>	: 30/Sep/2024 10:06 AM
BARCODE NO.	: 01517999		CTION DATE	: 30/Sep/2024 10:05 MM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		TING DATE	: 30/Sep/2024 11:48AM
CLIENT ADDRESS	: 6349/1, NICHOLSON RC	JAD, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		tely more than creatinine) (e.g	obstructive urona	thy).
<ol> <li>Prerenal azotemia</li> <li>DECREASED RATIO (&lt;</li> <li>Acute tubular necr</li> <li>Low protein diet al</li> <li>Severe liver diseas</li> </ol>	10:1) WITH DECREASED BUN osis. nd starvation.			
DECREASED RATIO (< Acute tubular necr Low protein diet and Severe liver diseas Other causes of de Repeated dialysis	10:1) WITH DECREASED BUN osis. nd starvation. e. creased urea synthesis. (urea rather than creatinine	: e diffuses out of extracellular f		
DECREASED RATIO (< Acute tubular necr Low protein diet al Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy.	<b>10:1) WITH DECREASED BUN</b> osis. nd starvation. e. creased urea synthesis. (urea rather than creatinine monemias (urea is virtually of inappropiate antidiuretic	: e diffuses out of extracellular f absent in blood). harmone) due to tubular secre	luid).	,,,,.
DECREASED RATIO (< Acute tubular necr Low protein diet al Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (<	10:1) WITH DECREASED BUN osis. nd starvation. e. creased urea synthesis. (urea rather than creatinine monemias (urea is virtually of inappropiate antidiuretic 10:1) WITH INCREASED CREA	: e diffuses out of extracellular f absent in blood). harmone) due to tubular secre TININE:	luid).	
DECREASED RATIO (< Acute tubular necr Low protein diet al Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (< Phenacimide thera	10:1) WITH DECREASED BUN osis. ad starvation. e. creased urea synthesis. (urea rather than creatinine monemias (urea is virtually of inappropiate antidiuretic 10:1) WITH INCREASED CREA py (accelerates conversion	: e diffuses out of extracellular f absent in blood). harmone) due to tubular secre <b>TININE:</b> of creatine to creatinine).	luid).	
DECREASED RATIO (< Acute tubular necr Low protein diet al Severe liver diseas Other causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (< Phenacimide thera Rhabdomyolysis (r	10:1) WITH DECREASED BUN osis. nd starvation. e. creased urea synthesis. (urea rather than creatinine monemias (urea is virtually of inappropiate antidiuretic 10:1) WITH INCREASED CREA	: e diffuses out of extracellular f absent in blood). harmone) due to tubular secre <b>TININE:</b> of creatine to creatinine).	luid).	
DECREASED RATIO (< Acute tubular necr Low protein diet al Severe liver diseas Coher causes of de Repeated dialysis Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (< Rhabdomyolysis (r Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO	10:1) WITH DECREASED BUN osis. and starvation. e. (urea rather than creatinine monemias (urea is virtually of inappropiate antidiuretic 10:1) WITH INCREASED CREA py (accelerates conversion eleases muscle creatinine). who develop renal failure.	: e diffuses out of extracellular f absent in blood). harmone) due to tubular secre <b>TININE:</b> of creatine to creatinine).	luid). etion of urea.	gies,resulting in normal ratio when dehydrati

CKD STAGE	DESCRIPTION	GFR ( mL/min/1.73m2 )	ASSOCIATED FINDINGS
G1	Normal kidney function	>90	No proteinuria
G2	Kidney damage with normal or high GFR	>90	Presence of Protein , Albumin or cast in urine
G3a	Mild decrease in GFR	60 -89	
G3b	Moderate decrease in GFR	30-59	
G4	Severe decrease in GFR	15-29	
G5	Kidney failure	<15	





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

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

KOS Central Lab: 6349/1, Nicholson Road, Ambala Cantt -133 001, Haryana KOS Molecular Lab: IInd Floor, Parry Hotel, Staff Road, Opp. GPO, Ambala Cantt -133 001, Haryana 0171-2643898, +91 99910 43898 | care@koshealthcare.com | www.koshealthcare.com







	Dr. Vinay Chopi MD (Pathology & Mio Chairman & Consulta	crobiology) M	m Chopra D (Pathology) nt Pathologist
NAME	: Mr. R.P GUPTA		
AGE/ GENDER	: 88 YRS/MALE	PATIENT ID	: 1629365
COLLECTED BY	: SURJESH	<b>REG. NO./LAB NO.</b>	: 012409300026
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 30/Sep/2024 10:06 AM
BARCODE NO.	:01517999	COLLECTION DATE	: 30/Sep/2024 10:25AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	<b>REPORTING DATE</b>	: 30/Sep/2024 11:48AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMI	BALA CANTT	
Test Name		Value Unit	Biological Reference interval

COMMENTS:

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

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

ADVICE:

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





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

KOS Central Lab: 6349/1, Nicholson Road, Ambala Cantt -133 001, Haryana KOS Molecular Lab: IInd Floor, Parry Hotel, Staff Road, Opp. GPO, Ambala Cantt - 133 001, Haryana 0171-2643898, +91 99910 43898 care@koshealthcare.com www.koshealthcare.com







	Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Patholog		(Pathology)
NAME	: Mr. R.P GUPTA		
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BARCODE NO.	: 01517999	<b>COLLECTION DATE</b>	: 30/Sep/2024 02:12PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 30/Sep/2024 04:05PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANT	Т	
			/
Test Name	Value	Unit	Biological Reference interval

	IRON	I PROFILE	
IRON: SERUM by ferrozine, spectrophotometry	65.2	μg/dL	59.0 - 158.0
UNSATURATED IRON BINDING CAPA SERUM by FERROZINE, SPECTROPHOTOMETER		μg/dL	150.0 - 336.0
TOTAL IRON BINDING CAPACITY (TIB SERUM by Spectrophotometery	C) 224.94 <sup>L</sup>	μg/dL	230 - 430
%TRANSFERRIN SATURATION: SERUN by calculated, spectrophotometer		%	15.0 - 50.0
TRANSFERRIN: SERUM by SPECTROPHOTOMETERY (FERENE) INTERPRETATION:-	159.71 <sup>L</sup>	mg/dL	200.0 - 350.0
VARIABLES	ANEMIA OF CHRONIC DISEASE	IRON DEFICIENCY ANEMIA	THALASSEMIA α/β TRAIT
	Normal to Deduced	Deduced	Nermeel

VARIABLES	ANEMIA OF CHRONIC DISEASE	IRON DEFICIENCY ANEMIA	THALASSEMIA $\alpha/\beta$ TRAIT
SERUM IRON:	Normal to Reduced Reduced		Normal
TOTAL IRON BINDING CAPACITY:	Decreased	Increased	Normal
% TRANSFERRIN SATURATION:	Decreased	Decreased < 12-15 %	Normal
SERUM FERRITIN:	Normal to Increased	Decreased	Normal or Increased
IDON			

IRON:

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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.

2. 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.



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

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







	Dr. Vinay Che MD (Pathology & Chairman & Cons		Dr. Yugam MD CEO & Consultant	(Pathology)
NAME	: Mr. R.P GUPTA			
AGE/ GENDER	: 88 YRS/MALE	PAT	IENT ID	: 1629365
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BARCODE NO.	: 01517999	COL	LECTION DATE	: 30/Sep/2024 02:12PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REP	ORTING DATE	: 30/Sep/2024 03:03PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		ENDOCRIN	OLOGY	
	т	HYROID FUNCTION	N TEST: TOTAL	
	E (T3): SERUM	0.745	ng/mL	0.35 - 1.93
THYROXINE (T4): SE	E (T3): SERUM NESCENT MICROPARTICLE IMMUNOAS	6.68	ng/mL µgm/dL	0.35 - 1.93 4.87 - 12.60

overproduction(hyperthyroidism) of T4 and/or T3. CLINICAL CONDITION T3 T4 TSH Primary Hypothyroidism: Reduced Reduced Increased (Significantly) Subclinical Hypothyroidism: Normal or Low Normal Normal or Low Normal High Reduced (at times undetectable) Primary Hyperthyroidism: Increased Increased 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 (eg: phenytoin , salicylates).

3. Serum T4 levles 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 hypothroidism, pregnancy, phenytoin therapy.

TRIIODOTH	(RONINE (T3)	THYROXINE (T4)		THYROID STIMUL	ATING 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





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

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KOS Central Lab: 6349/1, Nicholson Road, Ambala Cantt -133 001, Haryana KOS Molecular Lab: IInd Floor, Parry Hotel, Staff Road, Opp. GPO, Ambala Cantt -133 001, Haryana 0171-2643898, +91 99910 43898 | care@koshealthcare.com | www.koshealthcare.com



TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT







	<b>Dr. Vinay Chopra</b> MD (Pathology & Microbiology) Chairman & Consultant Pathologis		Pathology)
NAME	: Mr. R.P GUPTA		
AGE/ GENDER	: 88 YRS/MALE	PATIENT ID	: 1629365
COLLECTED BY	: SURJESH	REG. NO./LAB NO.	: 012409300026
<b>REFERRED BY</b>	:	<b>REGISTRATION DATE</b>	: 30/Sep/2024 10:06 AM
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT		

Test Name			Value	Unit	t	Biological Reference interval
6 - 12 Months	0.74 - 2.40	6 - 12 Months	7.10 - 16.16	6 – 12 Months	0.70 - 7.00	
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	
	RECO	MMENDATIONS OF TSH LI	EVELS 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, idonie 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 goitre & Thyroiditis.

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

3. Autonomously functioning Thyroid adenoma

4. Secondary pituatary or hypothalmic 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 Chopra MD (Pathology & Micro Chairman & Consultant		Dr. Yugam ( MD (F EO & Consultant P	Pathology)
NAME : Mr. R.P G	UPTA			
AGE/ GENDER : 88 YRS/M	ALE	PATIEN	TID	: 1629365
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			ATION DATE	
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	NOSTIC LAB	REPORT	ING DATE	: 30/Sep/2024 03:03PM
CLIENT ADDRESS : 6349/1, N	NICHOLSON ROAD, AMBA	LA CANTT		
Test Name		Value	Unit	Biological Reference interval
		VITAMINS		
	VITAMIN	D/25 HYDROXY	VITAMIN D3	
VITAMIN D (25-HYDROXY VITAM		25.144 <sup>L</sup>	ng/mL	DEFICIENCY: < 20.0
by CLIA (CHEMILUMINESCENCE IMM		23.144	<u></u>	INSUFFICIENCY: 20.0 - 30.0
				SUFFICIENCY: 30.0 - 100.0
				TOXICITY: > 100.0
INTERPRETATION:				
DEFICIENT:		< 20	ng/i	
INSUFFICIENT:		- 29 - 100	ng/i	
PREFFERED RANGE: INTOXICATION:		100	ng/i	
tissue and tightly bound by a tran 3.Vitamin D plays a primary role in phosphate reabsorption, skeletal in 4.Severe deficiency may lead to fae <b>DECREASED:</b> 1. Lack of sunshine exposure. 2. Inadequate intake, malabsorption 3. Depressed Hepatic Vitamin D 25 4. Secondary to advanced Liver dis 5. Osteoporosis and Secondary Hype 6. Enzyme Inducing drugs: anti-epient <b>INCREASED:</b> 1. Hypervitaminosis D is Rare, and severe hypercalcemia and hyperpient <b>CAUTION:</b> Replacement therapy in hypervitaminosis D	e main body resevoir and t sport protein while in circ n the maintenance of calci calcium deposition, calciu illure to mineralize newly t on (celiac disease) - hydroxylase activity ease berparathroidism (Mild to leptic drugs like phenytoir is seen only after prolong nophatemia. deficient individuals must compare to whites, is at hi	ransport form of Vita ulation. ium homeostatis. It i m mobilization, mair formed osteoid in bo Moderate deficienc , phenobarbital and ed exposure to extre t be monitored by pe	amin D and transpo promotes calcium a nly regulated by pa ne, resulting in ric () carbamazepine, th mely high doses of riodic assessment o	ort form of Vitamin D, being stored in adipose absorption, renal calcium absorption and rathyroid harmone (PTH). kets in children and osteomalacia in adults. at increases Vitamin D metabolism. <sup>6</sup> Vitamin D. When it occurs, it can result in of Vitamin D levels in order to prevent <i>hcy due to excess of melanin pigment which</i>





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 www.koshealthcare.com



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BARCODE NO.	: 01517999		ECTION DATE	: 30/Sep/2024 02:12PM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		DRTING DATE	: 30/Sep/2024 03:22PM
CLIENT ADDRESS			DRIING DATE	. 50/ Sep/ 2024 03.221 M
LIENI ADDRESS	: 6349/1, NICHOLSON ROAD	, AMBALA CANTI		
Test Name		Value	Unit	Biological Reference interval
			DBALAMIN	
by CMIA (CHEMILUMIN NTERPRETATION:-	ESCENT MICROPARTICLE IMMUNO	220.25	pg/mL	190.0 - 830
by CMIA (CHEMILUMIN INTERPRETATION:- INCREAS	ESCENT MICROPARTICLE IMMUNO	220.25 ASSAY)		
by CMIA (CHEMILUMIN INTERPRETATION:- INCREAS 1.Ingestion of Vitan	IESCENT MICROPARTICLE IMMUNO SED VITAMIN B12 nin C	220.25 ASSAY)	pg/mL	NB12
by CMIA (CHEMILUMIN INTERPRETATION:- INCREAS 1.Ingestion of Vitan 2.Ingestion of Estro	IESCENT MICROPARTICLE IMMUNO. SED VITAMIN B12 hin C gen	220.25 ASSAY) 1.Pregnancy 2.DRUGS:Aspi	pg/mL DECREASED VITAMIN rin, Anti-convulsants	NB12
INTERPRETATION:- INCREAS 1.Ingestion of Vitan	IESCENT MICROPARTICLE IMMUNO SED VITAMIN B12 hin C gen hin A	220.25 ASSAY)	pg/mL DECREASED VITAMIN rin, Anti-convulsants tion	NB12
by CMIA (CHEMILUMIN INTERPRETATION:- INCREAS 1.Ingestion of Vitan 2.Ingestion of Estro 3.Ingestion of Vitan	IESCENT MICROPARTICLE IMMUNO SED VITAMIN B12 hin C gen hin A jury	220.25 ASSAY) 1.Pregnancy 2.DRUGS:Aspi 3.Ethanol Iges	pg/mL DECREASED VITAMIN rin, Anti-convulsants tion ve Harmones	NB12
by CMIA (CHEMILUMIN <u>INTERPRETATION:-</u> <u>INCREAS</u> 1.Ingestion of Vitan 2.Ingestion of Vitan 4.Hepatocellular in 5.Myeloproliferativ 6.Uremia	IESCENT MICROPARTICLE IMMUNO. SED VITAMIN B12 hin C gen hin A jury e disorder	220.25 ASSAY)  220.25  1.Pregnancy 2.DRUGS:Aspi 3.Ethanol Iges 4. Contracepti 5.Haemodialy 6. Multiple M	pg/mL DECREASED VITAMIN rin, Anti-convulsants tion ve Harmones rsis yeloma	NB12
by CMIA (CHEMILUMIN INTERPRETATION:- INCREAS 1.Ingestion of Vitan 2.Ingestion of Vitan 4.Hepatocellular in 5.Myeloproliferativ 6.Uremia 1.Vitamin B12 (cobal	IESCENT MICROPARTICLE IMMUNO SED VITAMIN B12 hin C gen hin A jury	220.25 ASSAY)	pg/mL DECREASED VITAMIN rin, Anti-convulsants tion ve Harmones rsis yeloma onal function.	<b>J B12</b>

proprioception, poor coordination, and affective behavioral changes. These manifestations may occur in any combination; many patients have the neurologic defects without macrocytic anemia.

6.Serum methylmalonic acid and homocysteine levels are also elevated in vitamin B12 deficiency states.

7.Follow-up testing for antibodies to intrinsic factor (IF) is recommended to identify this potential cause of vitamin B12 malabsorption. **NOTE:**A normal serum concentration of vitamin B12 does not rule out tissue deficiency of vitamin B12. The most sensitive test for vitamin B12 deficiency at the cellular level is the assay for MMA. If clinical symptoms suggest deficiency, measurement of MMA and homocysteine should be considered, even if serum vitamin B12 concentrations are normal.





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

 KOS Central Lab: 6349/1, Nicholson Road, Ambala Cantt -133 001, Haryana

 KOS Molecular Lab: IInd Floor, Parry Hotel, Staff Road, Opp. GPO, Ambala Cantt -133 001, Haryana

 0171-2643898, +91 99910 43898
 care@koshealthcare.com
 www.koshealthcare.com







	<b>Dr. Vinay Cho</b> MD (Pathology & Chairman & Cons	Microbiology)	Dr. Yugam MD CEO & Consultant	(Pathology)
AGE/ GENDER:COLLECTED BY:REFERRED BY:BARCODE NO.:CLIENT CODE.:	<b>Mr. R.P GUPTA</b> 88 YRS/MALE SURJESH 01517999 KOS DIAGNOSTIC LAB 6349/1, NICHOLSON ROAD, <i>F</i>	REGIST COLLEC REPORT	T ID D./LAB NO. RATION DATE TION DATE FING DATE	: 1629365 <b>: 012409300026</b> : 30/Sep/2024 10:06 AM : 30/Sep/2024 10:25AM : 01/Oct/2024 03:19PM
Test Name		Value	Unit	Biological Reference interval
PHYSICAL EXAMINATIO		CLINICAL PATHO		ION
COLOUR by DIP STICK/REFLECTAN TRANSPARANCY by DIP STICK/REFLECTAN SPECIFIC GRAVITY	ICE SPECTROPHOTOMETRY ICE SPECTROPHOTOMETRY ICE SPECTROPHOTOMETRY ICE SPECTROPHOTOMETRY DN	10 PALE YELLOW HAZY 1.02	ml	PALE YELLOW CLEAR 1.002 - 1.030
PROTEIN by DIP STICK/REFLECTAN SUGAR by DIP STICK/REFLECTAN PH by DIP STICK/REFLECTAN BILIRUBIN	ICE SPECTROPHOTOMETRY ICE SPECTROPHOTOMETRY ICE SPECTROPHOTOMETRY ICE SPECTROPHOTOMETRY	ACIDIC Negative Negative <=5.0 Negative Negative		NEGATIVE (-ve) NEGATIVE (-ve) 5.0 - 7.5 NEGATIVE (-ve) NEGATIVE (-ve)
by DIP STICK/REFLECTAN UROBILINOGEN by DIP STICK/REFLECTAN KETONE BODIES by DIP STICK/REFLECTAN BLOOD	ICE SPECTROPHOTOMETRY. ICE SPECTROPHOTOMETRY ICE SPECTROPHOTOMETRY ICE SPECTROPHOTOMETRY	Normal Negative Negative Negative NEGATIVE (-ve)	EU/dL	NEGATIVE (-ve) 0.2 - 1.0 NEGATIVE (-ve) NEGATIVE (-ve) NEGATIVE (-ve)

MICROSCOPIC EXAMINATION

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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.





MD (Pathology & Microbiology) Chairman & Consultant Pathologist

Dr. Vinay Chopra



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

NAME	: Mr. R.P GUPTA					
GE/ GENDER : 88 YRS/MALE		PATIENT ID		: 1629365		
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BARCODE NO.	: 01517999					
CLIENT CODE.	: KOS DIAGNOSTIC LAB			: 01/Oct/2024 03:19PM		
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CANTT					
Test Name		Value	Unit	Biological Reference interval		
RED BLOOD CELLS (RBCS) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT PUS CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT EPITHELIAL CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT CRYSTALS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT CASTS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT		NEGATIVE (-ve)	/HPF	0 - 3		
		5-7	/HPF	0 - 5		
		1-3	/HPF	ABSENT		
		NEGATIVE (-ve) NEGATIVE (-ve)		NEGATIVE (-ve) NEGATIVE (-ve)		
					BACTERIA	

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT OTHERS

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT TRICHOMONAS VAGINALIS (PROTOZOA)

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT

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

NEGATIVE (-ve)

ABSENT





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NEGATIVE (-ve)

ABSENT