

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



	Dr. Vinay Chopra MD (Pathology & Micr Chairman & Consultar	obiology)		(Pathology)
NAME	: Mr. SUNVEER			
AGE/ GENDER	: 37 YRS/MALE		PATIENT ID	: 1682447
COLLECTED BY	: SURJESH		REG. NO./LAB NO.	: 012411260023
REFERRED BY	:		REGISTRATION DATE	: 26/Nov/2024 10:39 AM
BARCODE NO.	: 01521472		COLLECTION DATE	: 26/Nov/2024 11:01AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB		REPORTING DATE	: 26/Nov/2024 11:31AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMB.	ALA CANT'I		
Test Name		Value	Unit	Biological Reference interval
	COMP		LLNESS PANEL: 1. OOD COUNT (CBC)	5
Ked blood cells HAEMOGLOBIN (HH	(RBCS) COUNT AND INDICES	12.8	gm/dL	12.0 - 17.0
by CALORIMETRIC	5)	12.0	giii/ uL	12.0 - 17.0
RED BLOOD CELL (H	RBC) COUNT	5.02 ^H	Millions	/cmm 3.50 - 5.00
ACKED CELL VOLU	IME (PCV)	41.8	%	40.0 - 54.0
by CALCULATED BY AU	UTOMATED HEMATOLOGY ANALYZER	83.2	fL	80.0 - 100.0
by CALCULATED BY AU	JTOMATED HEMATOLOGY ANALYZER			
IEAN CORPUSCULA by CALCULATED BY AU	AR HAEMOGLOBIN (MCH) JTOMATED HEMATOLOGY ANALYZER	25.5 ^L	pg	27.0 - 34.0
AEAN CORPUSCUL	AR HEMOGLOBIN CONC. (MCHC)	30.7 ^L	g/dL	32.0 - 36.0
	JTION WIDTH (RDW-CV)	14.5	%	11.00 - 16.00
	JTOMATED HEMATOLOGY ANALYZER JTION WIDTH (RDW-SD)	45	fL	35.0 - 56.0
	JTOMATED HEMATOLOGY ANALYZER	45	IL	
MENTZERS INDEX		16.57	RATIO	BETA THALASSEMIA TRAIT: < 13.0
.,				IS.0 IRON DEFICIENCY ANEMIA:
	DV.	04.00	DATIO	>13.0
GREEN & KING IND by calculated	EX	24.03	RATIO	BETA THALASSEMIA TRAIT:<< 65.0
				IRON DEFICIENCY ANEMIA: >
	LS (WBCS)			65.0
WHITE BLOOD CFI		7000	/cmm	4000 - 11000
	COUNT (TLC)	7330		
OTAL LEUCOCYTE	BY SF CUBE & MICROSCOPY			0.00 20.00
NUCLEATED RED B		7330 NIL NIL		0.00 - 20.00 < 10 %





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: Ilnd Floor, Parry Hotel, Staff Road, Opp. GPO, Ambala Cantt -133 001, Haryana 0171-2643898, +91 99910 43898 | care@koshealthcare.com | www.koshealthcare.com







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
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS	50	%	50 - 70
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	40 ^H	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	4	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	6	%	2 - 12
BASOPHILS by flow cytometry by sf cube & microscopy ABSOLUTE LEUKOCYTES (WBC) COUNT	0	%	0 - 1
ABSOLUTE NEUTROPHIL COUNT by flow cytometry by sf cube & microscopy	3665	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2932	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	293	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	440	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110
ABSOLUTE IMMATURE GRANULOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0.0 - 999.0
PLATELETS AND OTHER PLATELET PREDICTIVE	MARKERS.		
PLATELET COUNT (PLT) by hydro dynamic focusing, electrical impedence	157000	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.25	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by hydro dynamic focusing, electrical impedence	16 ^H	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	106000 ^H	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	67.6 ^H	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	15.7	%	15.0 - 17.0





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Test Name	Val	lue Unit	Biological Reference interval

NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD



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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, A	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
GLYCOSYLATED HA WHOLE BLOOD	EMOGLOBIN (HbA1c):	5.8	AEMOGLOBIN (HBA1) %	4.0 - 6.4
	RMANCE LIQUID CHROMATOGRAPHY)			
ESTIMATED AVERA	RMANCE LIQUID CHROMATOGRAPHY) GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY)	119.76	mg/dL	60.00 - 140.00
ESTIMATED AVERA	GE PLASMA GLUCOSE			60.00 - 140.00
ESTIMATED AVERA by HPLC (HIGH PERFO INTERPRETATION:	GE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN I REFERENCE GROUP	DIABETES ASSOCI	IATION (ADA): LYCOSYLATED HEMOGLOGIB	
ESTIMATED AVERA by HPLC (HIGH PERFO INTERPRETATION: NON dia	AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN I REFERENCE GROUP abetic Adults >= 18 years	DIABETES ASSOCI	IATION (ADA):	
ESTIMATED AVERA by HPLC (HIGH PERFO NTERPRETATION: Non dia A	AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN I REFERENCE GROUP abetic Adults >= 18 years t Risk (Prediabetes)	DIABETES ASSOCI	ATION (ADA): LYCOSYLATED HEMOGLOGIB <5.7 5.7 - 6.4	
ESTIMATED AVERA by HPLC (HIGH PERFO INTERPRETATION: NON dia A	AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN I REFERENCE GROUP abetic Adults >= 18 years	DIABETES ASSOCI	ATION (ADA): LYCOSYLATED HEMOGLOGIB <5.7 5.7 - 6.4 >= 6.5	
ESTIMATED AVERA by HPLC (HIGH PERFO INTERPRETATION: NON dia A	AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN I REFERENCE GROUP abetic Adults >= 18 years t Risk (Prediabetes)	DIABETES ASSOCI	ATION (ADA): LYCOSYLATED HEMOGLOGIB <5.7 5.7 - 6.4 >= 6.5 Age > 19 Years	(HBAIC) in %
ESTIMATED AVERA by HPLC (HIGH PERFO INTERPRETATION: Non dia A D	AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN I REFERENCE GROUP abetic Adults >= 18 years t Risk (Prediabetes) iagnosing Diabetes	DIABETES ASSOCI	ATION (ADA): LYCOSYLATED HEMOGLOGIB <5.7 5.7 - 6.4 >= 6.5 Age > 19 Years s of Therapy:	(HBAIC) in %
ESTIMATED AVERA by HPLC (HIGH PERFO INTERPRETATION: Non dia A D	AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN I REFERENCE GROUP abetic Adults >= 18 years t Risk (Prediabetes)	DIABETES ASSOCI	ATION (ADA): LYCOSYLATED HEMOGLOGIB <5.7 5.7 - 6.4 >= 6.5 Age > 19 Years s of Therapy: ns Suggested:	(HBAIC) in %
ESTIMATED AVERA by HPLC (HIGH PERFO INTERPRETATION: Non dia A D	AGE PLASMA GLUCOSE RMANCE LIQUID CHROMATOGRAPHY) AS PER AMERICAN I REFERENCE GROUP abetic Adults >= 18 years t Risk (Prediabetes) iagnosing Diabetes	DIABETES ASSOCI	ATION (ADA): LYCOSYLATED HEMOGLOGIB <5.7 5.7 - 6.4 >= 6.5 Age > 19 Years s of Therapy:	(HBAIC) in %

COMMENTS:

TEST PERFORMED AT KOS DIAGNOSTIC LAB. AMBALA CANTT

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 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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		Chopra y & Microbiology) consultant Pathologist	Dr. Yugan MD CEO & Consultant	(Pathology)
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IENT ADDRESS	: 6349/1, NICHOLSON ROA	D, AMBALA CANTT		
est Name		Value	Unit	Biological Reference interval
stemic lupus eryth	ematosus W ESR			
DNDITION WITH LO low ESR can be see olycythaemia), sig s sickle cells in sick OTE: ESR and C - reactiv Generally, ESR dog	n with conditions that inhibit	l count (leucocytosis) , ar e ESR. kers of inflammation. es CRP, either at the start	nd some protein abno of inflammation or as	uch as a high red blood cell count rmalities. Some changes in red cell shape (suc s it resolves.





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		hopra & Microbiology) onsultant Pathologist	Dr. Yugan MD CEO & Consultant	(Pathology)
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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD), AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	CLINI	CAL CHEMISTI GLUCOSE FA	RY/BIOCHEMIST ASTING (F)	'RY
GLUCOSE FASTING by GLUCOSE OXIDAS	(F): PLASMA e - peroxidase (god-pod)	99.94	mg/dL	NORMAL: < 100.0 PREDIABETIC: 100.0 - 125.0 DIABETIC: > 0R = 126.0

KOS Diagnostic Lab (A Unit of KOS Healthcare)

IN ACCRDANCE WITH AMERICAN DIABETES ASSOCIATION GUIDELINES: 1. A fasting plasma glucose level below 100 mg/dl is considered normal. 2. A fasting plasma glucose level between 100 - 125 mg/dl is considered as glucose intolerant or prediabetic. A fasting and post-prandial blood

test (after consumption of 75 gms of glucose) is recommended for all such patients. 3. A fasting plasma glucose level of above 125 mg/dl is highly suggestive of diabetic state. A repeat post-prandial is strongly recommended for all such patients. A fasting plasma glucose level in excess of 125 mg/dl on both occasions is confirmatory for diabetic state.



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CLIENT CODE.	: KOS DIAGNOSTIC LAB		PORTING DATE	: 26/Nov/2024 12:25PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROA	AD, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
		LIPID PROFI	F . BASIC	
CHOLECTEDAL TA				
CHOLESTEROL TOT by CHOLESTEROL OX		219.77 ^H	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: S by GLYCEROL PHOSP	ERUM HATE OXIDASE (ENZYMATIC)	142.2	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROI by SELECTIVE INHIBIT	L (DIRECT): SERUM	38.75	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROI by CALCULATED, SPE		152.58 ^H	mg/dL	OPTIMAL: < 100.0 ABOVE OPTIMAL: 100.0 - 129. BORDERLINE HIGH: 130.0 - 159.0 HIGH: 160.0 - 189.0 VERY HIGH: > OR = 190.0
NON HDL CHOLEST by CALCULATED, SPE		181.02 ^H	mg/dL	OPTIMAL: < 130.0 ABOVE OPTIMAL: 130.0 - 159. BORDERLINE HIGH: 160.0 - 189.0 HIGH: 190.0 - 219.0 VERY HIGH: > OR = 220.0
VLDL CHOLESTERC		28.44	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SER by CALCULATED, SPE	UM	581.74	mg/dL	350.00 - 700.00
CHOLESTEROL/HD by CALCULATED, SPE	L RATIO: SERUM	5.67 ^H	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0

KOS Diagnostic Lab (A Unit of KOS Healthcare)





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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		3.94 ^H	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/H by CALCULATED, SPE		3.67	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. Vinay Chopra Dr. Yugam Chopra MD (Pathology & Microbiology) MD (Pathology) Chairman & Consultant Pathologist **CEO & Consultant Pathologist** NAME : Mr. SUNVEER AGE/ GENDER : 37 YRS/MALE **PATIENT ID** :1682447 **COLLECTED BY** : SURJESH REG. NO./LAB NO. :012411260023 **REFERRED BY** : **REGISTRATION DATE** : 26/Nov/2024 10:39 AM **BARCODE NO.** :01521472 **COLLECTION DATE** : 26/Nov/2024 11:01AM CLIENT CODE. : KOS DIAGNOSTIC LAB **REPORTING DATE** : 26/Nov/2024 12:25PM **CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT Test Name Value Unit

Test Name	Value	Unit	Biological Reference interval
LIVER	FUNCTION TE	ST (COMPLETE)	
BILIRUBIN TOTAL: SERUM by diazotization, spectrophotometry	1.06	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.19	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.87	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	18.35	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	19.84	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	0.92	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL	90.69	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	16.34	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	6.97	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	4.42	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by calculated, spectrophotometry	2.55	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.73	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)
HEPATOCELLULAR CARCINOMIA & CHRONIC HEPATTIS	> 1.3 (Slightly Increased)





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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).

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) 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 Cho MD (Pathology & N Chairman & Consu	1icrobiology)	Dr. Yugam MD (f CEO & Consultant F	Pathology)
NAME	: Mr. SUNVEER			
AGE/ GENDER	: 37 YRS/MALE	РА	TIENT ID	: 1682447
COLLECTED BY	: SURJESH	RE	G. NO./LAB NO.	: 012411260023
REFERRED BY	:	RE	GISTRATION DATE	: 26/Nov/2024 10:39 AM
BARCODE NO.	:01521472	CO	LLECTION DATE	: 26/Nov/2024 11:01AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	RE	PORTING DATE	: 26/Nov/2024 12:25PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
	KIDNE	EY FUNCTION 2	TEST (COMPLETE)	
UREA: SERUM by UREASE - GLUTAN	IATE DEHYDROGENASE (GLDH)	17.73	mg/dL	10.00 - 50.00
CREATININE: SERI		1.02	mg/dL	0.40 - 1.40
	ROGEN (BUN): SERUM	8.29	mg/dL	7.0 - 25.0
RATIO: SERUM	ROGEN (BUN)/CREATININE	8.13 ^L	RATIO	10.0 - 20.0
UREA/CREATININ		17.38	RATIO	
URIC ACID: SERUM		6.29	mg/dL	3.60 - 7.70
CALCIUM: SERUM by ARSENAZO III, SPE	ECTROPHOTOMETRY	10.58	mg/dL	8.50 - 10.60
PHOSPHOROUS: SE	ERUM DATE, SPECTROPHOTOMETRY	2.95	mg/dL	2.30 - 4.70
ELECTROLYTES				
SODIUM: SERUM by ISE (ION SELECTIV	/E ELECTRODE)	140.5	mmol/L	135.0 - 150.0
POTASSIUM: SERU		3.94	mmol/L	3.50 - 5.00
CHLORIDE: SERUN by ISE (ION SELECTIV		105.38	mmol/L	90.0 - 110.0
ESTIMATED GLOM	IERULAR FILTERATION RATE			
ESTIMATED GLOM	IERULAR FILTERATION RATE	97.1		

(eGFR): SERUM by CALCULATED

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.



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

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

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



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TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT





		Dr. Vinay Chopi MD (Pathology & Mic Chairman & Consulta	robiology)		fugam Cl MD (Pat nsultant Path	nology)			
NAME	: Mr. SUNVE	ER							
AGE/ GENDER	: 37 YRS/MAL	Æ		PATIENT ID	:	1682447			
COLLECTED BY	: SURJESH			REG. NO./LAB NO.		0124112600	92		
REFERRED BY				REGISTRATION D		26/Nov/2024			
BARCODE NO.	:01521472			COLLECTION DAT		26/Nov/2024			
CLIENT CODE.	: KOS DIAGNO	OSTIC LAB		REPORTING DATE	E :	26/Nov/2024	12:25PM		
CLIENT ADDRESS	: 6349/1, NIC	HOLSON ROAD, AMI	BALA CANTT						
Test Name			Value	Un	it	Biolog	gical Refer	ence inter	rval
 Reduced muscle m Certain drugs (e.g. INCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (Acute tubular necr 	ass (subnormal tetracycline, glu 10:1) WITH ELEV 1 (BUN rises disp superimposed (10:1) WITH DECR osis.	ucocorticoids) ATED CREATININE LEV proportionately more on renal disease.	ELS:	ne) (e.g. obstructive	e uropathy)				
 Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido Should produce an in Cephalosporin ther ESTIMATED GLOMERL CKD STAGE 	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 sy furea rather tha monemias (urea finappropiate of to:1) WITH INCR py (accelerates eleases muscle who develop re sis (acetoacetal creased BUN/cr apy (interferes JLAR FILTERATIO	creatinine productic ucocorticoids) ATED CREATININE LEV proportionately more on renal disease. EASED BUN : a trually absent in antidiuretic harmone EASED CREATININE: conversion of creatir creatinine). nal failure. te causes false increated eatinine ratio). with creatinine meas in RATE: DESCRIPTION	YELS: than creatinin out of extract blood).) due to tubul ne to creatinin se in creatinin urement).	ellular fluid). lar secretion of urea ne). ne with certain met	n. hodologies ASSOCI	resulting in no		when dehy	drati
 Certain drugs (e.g., NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia Prerenal azotemia CECREASED RATIO (<' Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome of Pregnancy. PCEREASED RATIO (Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido Should produce an in Cephalosporin ther ESTIMATED GLOMERL G1 	ass (subnormal tetracycline, glu tetracycline, glu tetracycline, glu tetracycline, glu to:1) WITH ELEVA (BUN rises disp superimposed of to:1) WITH DECR osis. The starvation. tetra rather tha monemias (urea finappropiate of the starvation. the starvation.	creatinine productic ucocorticoids) ATED CREATININE LEV proportionately more on renal disease. EASED BUN : a trually absent in antidiuretic harmone EASED CREATININE: conversion of creatir creatinine). nal failure. te causes false increated eatinine ratio). with creatinine meas in RATE: DESCRIPTION rmal kidney function	YELS: than creatinin out of extract blood).) due to tubul ne to creatinin se in creatinin urement).	ellular fluid). lar secretion of urea ne). ne with certain met nL/min/1.73m2) >90	n. hodologies ASSOCI	resulting in no ATED FINDINGS proteinuria		when dehy	drati
 Certain drugs (e.g. NCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (Rhabdomyolysis (r Muscular patients NAPPROPIATE RATIO Diabetic ketoacido Should produce an in Cephalosporin ther ESTIMATED GLOMERL CKD STAGE 	ass (subnormal tetracycline, glu tetracycline, glu tetracycline, glu tetracycline, glu tetracycline, glu tetracycline, glu superimposed of superimposed of tetracycline, glu tetracycline, glu t	creatinine productic ucocorticoids) ATED CREATININE LEV proportionately more on renal disease. EASED BUN : In thesis. In creatinine diffuses a is virtually absent in antidiuretic harmone EASED CREATININE: conversion of creatir creatinine). nal failure. In a failure. I	YELS: than creatinin out of extract blood).) due to tubul ne to creatinin se in creatinin urement).	ellular fluid). lar secretion of urea ne). ne with certain met	n. hodologies ASSOCI No Preser	resulting in no ATED FINDINGS proteinuria	6	when dehy	drati
 A. Certain drugs (e.g., NCREASED RATIO (>2) I. Postrenal azotemia DECREASED RATIO (<2) I. Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Severe liver disease Other causes of de Repeated dialysis (SIADH (syndrome of the second s	ass (subnormal tetracycline, glu tetracycline, glu tetracycline, glu tetracycline, glu to:1) WITH ELEVA (BUN rises disp superimposed of to:1) WITH DECR osis. The starvation. tetra rather tha monemias (urea finappropiate of trapp (accelerates eleases muscle who develop re terased BUN/cr apy (interferes JLAR FILTERATIO Nor Nor	creatinine productic ucocorticoids) ATED CREATININE LEV proportionately more on renal disease. EASED BUN : In creatinine diffuses a is virtually absent in antidiuretic harmone EASED CREATININE: conversion of creatir creatinine). nal failure. In creatinine meas in creatinine meas in RATE: DESCRIPTION mal kidney function idney damage with ormal or high GFR	YELS: than creatinin out of extract blood).) due to tubul ne to creatinin se in creatinin urement).	ellular fluid). lar secretion of urea ne). ne with certain met nL/min/1.73m2) >90 >90	n. hodologies ASSOCI No Preser	resulting in no ATED FINDINGS proteinuria	6	when dehy	drati
 P. Certain drugs (e.g., INCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (< Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome of Pregnancy. DECREASED RATIO (Rhabdomyolysis (r Muscular patients MAPPROPIATE RATIO Diabetic ketoacido Should produce an in Cephalosporin ther ESTIMATED GLOMERI G1 	ass (subnormal tetracycline, glu tetracycline, glu tetracycline, glu tetracycline, glu to:1) WITH ELEVA (BUN rises disp superimposed of to:1) WITH DECR osis. the starvation. te. creased urea sy urea rather tha monemias (urea finappropiate of the sy urea rather tha monemias (urea finappropiate of the sy urea rather tha monemias (urea of inappropiate of the sy urea rather tha monemias (urea finappropiate of the sy urea rather tha monemias (urea finappropiate of the sy urea rather tha monemias (urea fina	creatinine productic ucocorticoids) ATED CREATININE LEV proportionately more on renal disease. EASED BUN : In thesis. In creatinine diffuses a is virtually absent in antidiuretic harmone EASED CREATININE: conversion of creatir creatinine). nal failure. In a failure. I	YELS: than creatinin out of extract blood).) due to tubul ne to creatinin se in creatinin urement). GFR (m	ellular fluid). lar secretion of urea ne). ne with certain met nL/min/1.73m2) >90 >90 60 -89	n. hodologies ASSOCI No Preser	resulting in no ATED FINDINGS proteinuria	6	when dehy	drati
 P. Certain drugs (e.g. INCREASED RATIO (>2 Postrenal azotemia Prerenal azotemia DECREASED RATIO (< 1. Acute tubular necr Low protein diet ar Severe liver disease Other causes of de Repeated dialysis (Inherited hyperam SIADH (syndrome c Pregnancy. DECREASED RATIO (Rhabdomyolysis (r Muscular patients INAPPROPIATE RATIO Cephalosporin ther ESTIMATED GLOMERL G1 G2 	ass (subnormal tetracycline, glu tetracycline, glu tetracycline, glu tetracycline, glu tetracycline, glu to:1) WITH ELEVA of BUN rises disp superimposed of to:1) WITH DECR osis. the starvation. te. creased urea sy furea rather tha monemias (urea finappropiate a finappropiate a finappro	creatinine productic ucocorticoids) ATED CREATININE LEV proportionately more on renal disease. EASED BUN : In creatinine diffuses a is virtually absent in antidiuretic harmone EASED CREATININE: conversion of creatir creatinine). nal failure. In creatinine meas in RATE: DESCRIPTION mal kidney function idney damage with ormal or high GFR_ ild decrease in GFR	YELS: than creatinin out of extract blood).) due to tubul ne to creatinin se in creatinin urement). GFR (m	ellular fluid). lar secretion of urea ne). ne with certain met nL/min/1.73m2) >90 >90	n. hodologies ASSOCI No Preser	resulting in no ATED FINDINGS proteinuria	6	when dehy	drati



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

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









	Dr. Vinay Chopra MD (Pathology & Microbiology) Chairman & Consultant Patholo		(Pathology)
NAME	: Mr. SUNVEER		
AGE/ GENDER	: 37 YRS/MALE	PATIENT ID	: 1682447
COLLECTED BY	: SURJESH	REG. NO./LAB NO.	: 012411260023
REFERRED BY	:	REGISTRATION DATE	: 26/Nov/2024 10:39 AM
BARCODE NO.	: 01521472	COLLECTION DATE	: 26/Nov/2024 11:01AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPORTING DATE	: 26/Nov/2024 12:25PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AMBALA CAN	TT	
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





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

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NAME	: Mr. SUNVEER			
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Test Name		Value	Unit	Biological Reference interval
		IRON F	PROFILE	

	IRON P	ROFILE	
IRON: SERUM by FERROZINE, SPECTROPHOTOMETRY	68.5	μg/dL	59.0 - 158.0
UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM by FERROZINE, SPECTROPHOTOMETERY	250.87	µg/dL	150.0 - 336.0
TOTAL IRON BINDING CAPACITY (TIBC) :SERUM by SPECTROPHOTOMETERY	319.37	µg/dL	230 - 430
%TRANSFERRIN SATURATION: SERUM by CALCULATED, SPECTROPHOTOMETERY (FERENE)	21.45	%	15.0 - 50.0
TRANSFERRIN: SERUM by SPECTROPHOTOMETERY (FERENE)	226.75	mg/dL	200.0 - 350.0
INTERPRETATION:-			
VARIABLES ANEMIA OF CHRON	VIC DISEASE	IRON DEFICIENCY ANEMIA TH	IALASSEMIA α/β TRAIT

VARIABLES	ANEMIA OF CHRONIC DISEASE	IRON DEFICIENCY ANEMIA	THALASSEMIA α/β 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

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.



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

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

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CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	IBALA CANTT			
Test Name		Value	Unit	Biological Refere	ence interval
		ENDOCRIN	OLOGY		
	THY	ROID FUNCTIO	N TEST: TOTAL		
TRIIODOTHYRONII	NE (T3): SERUM iescent microparticle immunoass,	0.528 AY)	ng/mL	0.35 - 1.93	
THYROXINE (T4): S by CMIA (CHEMILUMIN	ERUM ESCENT MICROPARTICLE IMMUNOASS	9.4 AY)	µgm/dL	4.87 - 12.60	
	TING HORMONE (TSH): SERUM		µIU/mL	0.35 - 5.50	
BY CMIA (CHEMILUMIN 3rd GENERATION, ULT	ESCENT MICROPARTICLE IMMUNOASS, RASENSITIVE	4 <i>Y)</i>			
INTERPRETATION:					
day has influence on the i triiodothyronine (T3).Fai	circadian variation, reaching peak levels be measured serum TSH concentrations. TSH lure at any level of regulation of the hypo roidism) of T4 and/or T3.	stimulates the production	n and secretion of the m	etabolically active hormones, thyrox	ine (T4)and
	T3	Т	4	TSH	
Primary Hypothyroidis	n: Reduced	Re	duced I	ncreased (Significantly)	
Subclinical Hypothyroi	dism: Normal or Low No	rmal Norma	l or Low Normal	High	

LIMI	TAT	IONS	÷

Primary Hyperthyroidism:

Subclinical Hyperthyroidism:

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.

Increased

Normal or High Normal

Reduced (at times undetectable)

Reduced

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 (1	
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

Increased

Normal or High Normal





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 & Microbio Chairman & Consultant Pat	G, /	(Pathology)
NAME	: Mr. SUNVEER		
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Test Name		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	
	RECOM	MENDATIONS OF TSH LE	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





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KOS Diagnostic Lab (A Unit of KOS Healthcare)

	MD (P	'inay Chopra athology & Microbiology) nan & Consultant Pathologi		(Pathology)
IAME IGE/ GENDER	: Mr. SUNVEER : 37 YRS/MALE		PATIENT ID	: 1682447
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CLIENT ADDRESS	: 6349/1, NICHOLSO	ON ROAD, AMBALA CANTT	Г	
Fest Name		Value	Unit	Biological Reference interval
		VI	TAMINS	
			YDROXY VITAMIN D	3
	DROXY VITAMIN D3 ESCENCE IMMUNOASSAY		ng/mL	DEFICIENCY: < 20.0 INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0 TOXICITY: > 100.0
NTERPRETATION:		20		
	CIENT: ICIENT:	< 20 21 - 29		g/mLg/mL
PREFFERED RANGE:		30 - 100 > 100	r	g/mL g/mL
2.25-OHVitamin D re issue and tightly bou 3. Vitamin D plays a p bhosphate reabsorpti 4. Severe deficiency m DECREASED: 1. Lack of sunshine exi 2. Inadeguate intake, 3. Depressed Hepatic	epresents the main bo ind by a transport pro- rimary role in the mai on, skeletal calcium d aay lead to failure to n posure. malabsorption (celiac Vitamin D 25- hydroxy ced Liver disease econdary Hyperparath	tein while in circulation. ntenance of calcium home eposition, calcium mobiliz nineralize newly formed os disease) dase activity proidism (Mild to Moderate	form of Vitamin D and trans costatis. It promotes calcius ation, mainly regulated by steoid in bone, resulting in e deficiency)	sport form of Vitamin D, being stored in adipose m absorption, renal calcium absorption and parathvroid harmone (PTH). rickets in children and osteomalacia in adults. that increases Vitamin D metabolism.

DR.YUGAM CHOPRA

CONSULTANT PATHOLOGIST

MBBS, MD (PATHOLOGY)

MBBS, MD (PATHOLOGY & MICROBIOLOGY)

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

DR.VINAY CHOPRA CONSULTANT PATHOLOGIST

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

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Dr. Vinay Cho MD (Pathology & Chairman & Cons				
NAME	: Mr. SUNVEER			
AGE/ GENDER	: 37 YRS/MALE	PATI	ENT ID	: 1682447
COLLECTED BY	: SURJESH	REG. 1	NO./LAB NO.	: 012411260023
REFERRED BY	:	REGIS	TRATION DATE	: 26/Nov/2024 10:39 AM
BARCODE NO.	:01521472	COLL	ECTION DATE	: 26/Nov/2024 11:01AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	REPO	RTING DATE	: 26/Nov/2024 12:58PM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAI			
Test Name		Value	Unit	Biological Reference interval
		VITAMIN B12/CO	BALAMIN	
by CMIA (CHEMILUMIN INTERPRETATION:-	IESCENT MICROPARTICLE IMMUNC	168.72 ^L	pg/mL	190.0 - 830
by CMIA (CHEMILUMIN <u>NTERPRETATION:-</u> INCREAS	IESCENT MICROPARTICLE IMMUNC	168.72 ^L		
by CMIA (CHEMILUMIN <u>NTERPRETATION:-</u> INCREA: 1.Ingestion of Vitan	IESCENT MICROPARTICLE IMMUNC SED VITAMIN B12 nin C	DASSAY) 168.72^L	pg/mL DECREASED VITAMIN	NB12
by CMIA (CHEMILUMIN INTERPRETATION:- INCREAS 1.Ingestion of Vitan 2.Ingestion of Estro	IESCENT MICROPARTICLE IMMUNO SED VITAMIN B12 nin C gen	DASSAY) 168.72 ^L 1.Pregnancy 2.DRUGS:Aspir	pg/mL DECREASED VITAMIN	NB12
by CMIA (CHEMILUMIN <u>INTERPRETATION:-</u> <u>INCREAS</u> 1.Ingestion of Vitan 2.Ingestion of Estro 3.Ingestion of Vitan	IESCENT MICROPARTICLE IMMUNO SED VITAMIN B12 nin C gen nin A	DASSAY) 168.72 ^L 1.Pregnancy 2.DRUGS:Aspir 3.Ethanol Igest	pg/mL DECREASED VITAMIN in, Anti-convulsants ion	NB12
by CMIA (CHEMILUMIN INTERPRETATION:- INCREAS 1.Ingestion of Vitan 2.Ingestion of Vitan 3.Ingestion of Vitan 4.Hepatocellular in	IESCENT MICROPARTICLE IMMUNO SED VITAMIN B12 nin C gen nin A jury	DASSAY) 168.72 ^L 1.Pregnancy 2.DRUGS:Aspir 3.Ethanol Igest 4. Contraceptiv	pg/mL DECREASED VITAMIN in, Anti-convulsants ion e Harmones	NB12
INTERPRETATION:- INCREAS 1.Ingestion of Vitan 2.Ingestion of Vitan 3.Ingestion of Vitan 4.Hepatocellular in 5.Myeloproliferativ 6.Uremia	IESCENT MICROPARTICLE IMMUNO SED VITAMIN B12 nin C gen nin A jury	168.72 ^L 1.Pregnancy 2.DRUGS:Aspir 3.Ethanol Igest 4. Contraceptiv 5.Haemodialys 6. Multiple My	pg/mL	NB12

5.Vitamin B12 deficiency frequently causes macrocytic anemia, glossitis, peripheral neuropathy, weakness, hyperreflexia, ataxia, loss of 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.





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



Page 18 of 2

TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT





		Chopra y & Microbiology) Consultant Pathologist	Dr. Yugam MD CEO & Consultant	(Pathology)
NAME	: Mr. SUNVEER			
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CLIENT CODE.	: KOS DIAGNOSTIC LAB	RE	PORTING DATE	: 26/Nov/2024 11:48AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROA	D, AMBALA CANTT		
Test Name		Value	Unit	Biological Reference interva
		CLINICAL PA	THOLOCY	
	UDINE	ROUTINE & MICRO		TION
PHYSICAL EXAMIN		NOUTINE & MICKU	SCOI IC EAAMIN	ATION
QUANTITY RECIEV		10	ml	
	TANCE SPECTROPHOTOMETRY	10	1111	
COLOUR		AMBER YELI	LOW	PALE YELLOW
by DIP STICK/REFLEC TRANSPARANCY	TANCE SPECTROPHOTOMETRY	CLEAR		CLEAR
	TANCE SPECTROPHOTOMETRY	ULLAR		CLEAR
SPECIFIC GRAVITY		1.01		1.002 - 1.030
CHEMICAL EXAMI	TANCE SPECTROPHOTOMETRY			
REACTION	MATION	ACIDIC		
	TANCE SPECTROPHOTOMETRY	ACIDIC		
PROTEIN		Negative		NEGATIVE (-ve)
SUGAR	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
	TANCE SPECTROPHOTOMETRY			
pH	TANCE SPECTROPHOTOMETRY	5.5		5.0 - 7.5
BILIRUBIN	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	C .		
NITRITE	TANCE SPECTROPHOTOMETRY.	Negative		NEGATIVE (-ve)
UROBILINOGEN		Normal	EU/dL	0.2 - 1.0
-	TANCE SPECTROPHOTOMETRY			
KETONE BODIES by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	Negative		NEGATIVE (-ve)
BLOOD		Negative		NEGATIVE (-ve)
•	TANCE SPECTROPHOTOMETRY	NECATIVE (vo)	
ASCORBIC ACID by DIP STICK/REFLEC	TANCE SPECTROPHOTOMETRY	NEGATIVE (-	ve)	NEGATIVE (-ve)
MICROSCOPIC EXA				
RED BLOOD CELLS	(RBCs)	NEGATIVE (-	ve) /HPF	0 - 3

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

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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

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



TEST PERFORMED AT KOS DIAGNOSTIC LAB, AMBALA CANTT.



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



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

NAME	: Mr. SUNVEER			
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BARCODE NO.	: 01521472	C	OLLECTION DATE	: 26/Nov/2024 11:01AM
CLIENT CODE.	: KOS DIAGNOSTIC LAB	R	EPORTING DATE	: 26/Nov/2024 11:48AM
CLIENT ADDRESS	: 6349/1, NICHOLSON ROAD, AM	MBALA CANTT		
Test Name		Value	Unit	Biological Reference interval
by MICROSCOPY ON C	CENTRIFUGED URINARY SEDIMENT			
PUS CELLS by MICROSCOPY ON C	CENTRIFUGED URINARY SEDIMENT	2-4	/HPF	0 - 5
EDITUELIAL CELLS		0.2	/LIDE	ADCENT

/IIII ADDENI	
(-ve) NEGATIVE (-ve)	
ABSENT	
	(-ve)NEGATIVE (-ve)(-ve)NEGATIVE (-ve)(-ve)NEGATIVE (-ve)

*** End Of Report ***



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

V 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

