

(A Unit of KOS Healthcare)



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

NAME : Mr. JASBIR SINGH

**AGE/ GENDER** : 48 YRS/MALE **PATIENT ID** : 1689193

COLLECTED BY: SURJESH REG. NO./LAB NO. : 012412030033

 REFERRED BY
 : 03/Dec/2024 10:46 AM

 BARCODE NO.
 : 01521904
 COLLECTION DATE
 : 04/Dec/2024 12:14PM

 CLIENT CODE.
 : KOS DIAGNOSTIC LAB
 REPORTING DATE
 : 03/Dec/2024 11:22AM

**CLIENT ADDRESS**: 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name Value Unit Biological Reference interval

### SWASTHYA WELLNESS PANEL: 1.5 COMPLETE BLOOD COUNT (CBC)

#### **RED BLOOD CELLS (RBCS) COUNT AND INDICES**

HAEMOGLOBIN (HB) by CALORIMETRIC	10.4 <sup>L</sup>	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	3.62	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	32 <sup>L</sup>	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV) by calculated by automated hematology analyzer	88.6	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by calculated by automated hematology analyzer	28.7	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	32.3	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	15.8	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	52.3	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	24.48	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	38.63	RATIO	BETA THALASSEMIA TRAIT:<= 65.0 IRON DEFICIENCY ANEMIA: > 65.0
WHITE BLOOD CELLS (WBCS)			
TOTAL LEUCOCYTE COUNT (TLC) by flow cytometry by Sf cube & microscopy	5600	/cmm	4000 - 11000
NUCLEATED RED BLOOD CELLS (nRBCS) by automated 6 part hematology analyzer	NIL		0.00 - 20.00
NUCLEATED RED BLOOD CELLS (nRBCS) %	NIL	%	< 10 %



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by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER



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Test Name	Value	Unit	Biological Reference interval
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	47 <sup>L</sup>	%	50 - 70
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	35	%	20 - 40
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	10 <sup>H</sup>	%	1 - 6
MONOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	8	%	2 - 12
BASOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	%	0 - 1
ABSOLUTE LEUKOCYTES (WBC) COUNT			
ABSOLUTE NEUTROPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2632	/cmm	2000 - 7500
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	1960	/cmm	800 - 4900
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	560 <sup>H</sup>	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	448	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110
PLATELETS AND OTHER PLATELET PREDICTIVE	MARKERS.		
PLATELET COUNT (PLT) by hydro dynamic focusing, electrical impedence	99000 <sup>L</sup>	/cmm	150000 - 450000
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	0.1	%	0.10 - 0.36
MEAN PLATELET VOLUME (MPV) by hydro dynamic focusing, electrical impedence	11	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	29000 <sup>L</sup>	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR) by hydro dynamic focusing, electrical impedence	32.4	%	11.0 - 45.0
PLATELET DISTRIBUTION WIDTH (PDW) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	16.3	%	15.0 - 17.0
ADVICE	KINDLY CORRE	LATE CLINICALLY	



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NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD

RECHECKED.



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**Value** Unit **Biological Reference interval Test Name** 

REPORTING DATE

#### **GLYCOSYLATED HAEMOGLOBIN (HBA1C)**

GLYCOSYLATED HAEMOGLOBIN (HbA1c): 6.3 4.0 - 6.4

WHOLE BLOOD

CLIENT CODE.

by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)

ESTIMATED AVERAGE PLASMA GLUCOSE 134.11 mg/dL 60.00 - 140.00

by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)

#### INTERPRETATION:

AS PER AMERICAN D	ABETES ASSOCIATION (ADA):	
REFERENCE GROUP	GLYCOSYLATED HEMOGL	OGIB (HBAIC) in %
Non diabetic Adults >= 18 years	<5.7	
At Risk (Prediabetes)	5.7 – 6.	4
Diagnosing Diabetes	>= 6.5	
	Age > 19 Y	ears
	Goals of Therapy:	< 7.0
Therapeutic goals for glycemic control	Actions Suggested:	>8.0
	Age < 19 Y	ears
	Goal of therapy:	<7.5

#### COMMENTS:

1. Glycosylated hemoglobin (HbA1c) test is three monthly monitoring done to assess compliace with therapeutic regimen in diabetic patients.

2. Since Hb1c reflects long term fluctuations in blood glucose concentration, a diabetic patient who has recently under good control may still have high concentration of HbAlc. Converse is true for a diabetic previously under good control but now poorly controlled.

3. Target goals of < 7.0 % may be beneficial in patients with short duration of diabetes, long life expectancy and no significant cardiovascular disease. In patients with significant complications of diabetes, limited life expectancy or extensive co-morbid conditions, targetting a goal of < 7.0% may not be

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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### **KOS Diagnostic Lab**

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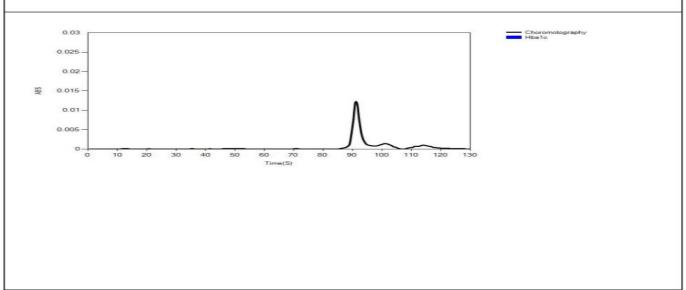
**Test Name Value** Unit **Biological Reference interval** 

REPORTING DATE

#### LIFOTRONIC Graph Report

Name :	Case:	Patient Type :	Test Date: 03/12/2024 14:12:05
Age:	Department:	Sample Type: Whole Blood EDTA	Sample ld: 01521904
Gender:			Total Area: 403

Peak Name	Retention Time(s)	Absorbance	Area	Result (Area %)
HbA0	67	123	400	96.1
HbA1c	00	0	0	0.0
La1c	25	1	1	0.2
HbF	00	0	0	0.0
Hba1b	15	1	1	0.2
Hba1a	09	1	1	0.3





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**Value** Unit **Biological Reference interval Test Name** 

#### **ERYTHROCYTE SEDIMENTATION RATE (ESR)**

ERYTHROCYTE SEDIMENTATION RATE (ESR)

mm/1st hr 56<sup>H</sup>

by RED CELL AGGREGATION BY CAPILLARY PHOTOMETRY

#### INTERPRETATION:

- 1. ESR is a non-specific test because an elevated result often indicates the presence of inflammation associated with infection, cancer and auto-immune disease, but does not tell the health practitioner exactly where the inflammation is in the body or what is causing it.

  2. An ESR can be affected by other conditions besides inflammation. For this reason, the ESR is typically used in conjunction with other test such
- as C-reactive protein
- 3. This test may also be used to monitor disease activity and response to therapy in both of the above diseases as well as some others, such as systemic lupus erythematosus
  CONDITION WITH LOW ESR

A low ESR can be seen with conditions that inhibit the normal sedimentation of red blood cells, such as a high red blood cell count (polycythaemia), significantly high white blood cell count (leucocytosis), and some protein abnormalities. Some changes in red cell shape (such as sickle cells in sickle cell anaemia) also lower the ESR. 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.
   Progs such as doubtern mathyldona, oral contracentives, popicillamino procesingmide, the only viling, and vitality in the orange of the contracentives.

- 6. Drugs such as dextran, methyldopa, oral contraceptives, penicillamine procainamide, theophylline, and vitamin A can increase ESR, while aspirin, cortisone, and quinine may decrease it



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**Value** Unit **Biological Reference interval Test Name** 

### **CLINICAL CHEMISTRY/BIOCHEMISTRY GLUCOSE FASTING (F)**

GLUCOSE FASTING (F): PLASMA NORMAL: < 100.0 106.18<sup>H</sup> mg/dL

by GLUCOSE OXIDASE - PEROXIDASE (GOD-POD) PREDIABETIC: 100.0 - 125.0

DIABETIC: > 0R = 126.0

INTERPRETATION
IN ACCORDANCE 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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Test Name	Value	Unit	Biological Reference interval
	LIPID PROFILE	: BASIC	
CHOLESTEROL TOTAL: SERUM by CHOLESTEROL OXIDASE PAP	215.18 <sup>H</sup>	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)	172.45 <sup>H</sup>	mg/dL	OPTIMAL: < 150.0 BORDERLINE HIGH: 150.0 - 199.0 HIGH: 200.0 - 499.0 VERY HIGH: > OR = 500.0
HDL CHOLESTEROL (DIRECT): SERUM by SELECTIVE INHIBITION	29.35 <sup>L</sup>	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMETRY	151.34 <sup>H</sup>	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 CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMETRY	185.83 <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: SERUM by CALCULATED, SPECTROPHOTOMETRY	34.49	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM by CALCULATED, SPECTROPHOTOMETRY	602.81	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	7.33 <sup>H</sup>	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: SERUM by CALCULATED, SPECTROPHOTOMETRY	5.16 <sup>H</sup>	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/HDL RATIO: SERUM by CALCULATED. SPECTROPHOTOMETRY	5.88 <sup>H</sup>	RATIO	3.00 - 5.00

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#### **INTERPRETATION:**

CLIENT CODE.

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

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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#### **LIVER FUNCTION TEST (COMPLETE)**

BILIRUBIN TOTAL: SERUM by DIAZOTIZATION, SPECTROPHOTOMETRY	0.47	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.15	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.32	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	56.5 <sup>H</sup>	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	38.8	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.46	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL	105.22	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	74.31 <sup>H</sup>	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	4.5 <sup>L</sup>	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	2.23 <sup>L</sup>	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.27 <sup>L</sup>	gm/dL	2.30 - 3.50
A: GRATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	0.98 <sup>L</sup>	RATIO	1.00 - 2.00

#### INTERPRETATION

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

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

#### INCREASED:

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



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**CLIENT ADDRESS**: 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name Value Unit Biological Reference interval

#### **DECREASED:**

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

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

#### PROGNOSTIC SIGNIFICANCE:

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



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**RATIO** 

mg/dL

mg/dL

mg/dL

**NAME** : Mr. JASBIR SINGH

**AGE/ GENDER** : 48 YRS/MALE **PATIENT ID** : 1689193

**COLLECTED BY** : SURJESH :012412030033 REG. NO./LAB NO.

REFERRED BY **REGISTRATION DATE** : 03/Dec/2024 10:46 AM BARCODE NO. :01521904 **COLLECTION DATE** :04/Dec/2024 12:14PM CLIENT CODE. : KOS DIAGNOSTIC LAB REPORTING DATE : 03/Dec/2024 01:17PM

**CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name	Value	Unit	Biological Reference interval
KIDNE	Y FUNCTION TE	EST (COMPLETE)	
UREA: SERUM by urease - glutamate dehydrogenase (gldh)	57.88 <sup>H</sup>	mg/dL	10.00 - 50.00
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETERY	2.51 <sup>H</sup>	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY	27.05 <sup>H</sup>	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE	10.78	RATIO	10.0 - 20.0

by CALCULATED, SPECTROPHOTOMETRY	
UREA/CREATININE RATIO: SERUM	23.06
by CALCULATED, SPECTROPHOTOMETRY	
URIC ACID: SERUM	5.71

CALCIUM: SERUM 9.12 by ARSENAZO III, SPECTROPHOTOMETRY PHOSPHOROUS: SERUM 3.19

by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY

by URICASE - OXIDASE PEROXIDASE

**ELECTROLYTES** 

RATIO: SERUM

SODIUM: SERUM 142.2 mmol/L 135.0 - 150.0 by ISE (ION SELECTIVE ELECTRODE) POTASSIUM: SERUM 4.33 mmol/L 3.50 - 5.00by ISE (ION SELECTIVE ELECTRODE) 106.65 CHLORIDE: SERUM 90.0 - 110.0 mmol/L by ISE (ION SELECTIVE ELECTRODE)

**ESTIMATED GLOMERULAR FILTERATION RATE** 

ESTIMATED GLOMERULAR FILTERATION RATE 30.8

(eGFR): SERUM by CALCULATED

NOTE 2 RESULT RECHECKED TWICE **ADVICE** KINDLY CORRELATE CLINICALLY

**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



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3.60 - 7.70

8.50 - 10.60

2.30 - 4.70



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**Test Name Value** Unit **Biological Reference interval** 

glomerular filtration rate.

- 2. Catabolic states with increased tissue breakdown.
- 3. GI haemorrhage.
- 4. High protein intake.
- 5. Impaired renal function plus
- 6. Excess protein intake or production or tissue breakdown (e.g. infection, GI bleeding, thyrotoxicosis, Cushing's syndrome, high protein diet, burns, surgery, cachexia, high fever).
- 7. Urine reabsorption (e.g. ureter colostomy)
- 8. Reduced muscle mass (subnormal creatinine production)
- 9. Certain drugs (e.g. tetracycline, glucocorticoids)

#### INCREASED RATIO (>20:1) WITH ELEVATED CREATININE LEVELS:

- 1. Postrenal azotemia (BUN rises disproportionately more than creatinine) (e.g. obstructive uropathy).
- 2. Prerenal azotemia superimposed on renal disease.

#### DECREASED RATIO (<10:1) WITH DECREASED BUN:

- 1. Acute tubular necrosis.
- 2. Low protein diet and starvation.
- 3. Severe liver disease.
- 4. Other causes of decreased urea synthesis.
- 5. Repeated dialysis (urea rather than creatinine diffuses out of extracellular fluid).
- 6. Inherited hyperammonemias (urea is virtually absent in blood).
- 7. SIADH (syndrome of inappropiate antidiuretic harmone) due to tubular secretion of urea.
- 8. Pregnancy.

#### **DECREASED RATIO (<10:1) WITH INCREASED CREATININE:**

- 1. Phenacimide therapy (accelerates conversion of creatine to creatinine).
- 2. Rhabdomyolysis (releases muscle creatinine).
- 3. Muscular patients who develop renal failure.

#### **INAPPROPIATE RATIO:**

1. Diabetic ketoacidosis (acetoacetate causes false increase in creatinine with certain methodologies, resulting in normal ratio when dehydration should produce an increased BUN/creatinine ratio).

2. Cephalosporin therapy (interferes with creatinine measurement) ESTIMATED GLOMERULAR FILTERATION RATE:

STIMATED GEOMERGEAR TIETERATION RATE.					
CKD STAGE	DESCRIPTION	GFR ( mL/min/1.73m2 )	ASSOCIATED FINDINGS		
G1	Normal kidney function	>90	No proteinuria		
G2	Kidney damage with	>90	Presence of Protein,		
	normal or high GFR		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			



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**Test Name** Value Unit **Biological Reference interval** 

#### COMMENTS:

1. 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.

2. eGFR calculated using the 2009 CKD-EPI creatinine equation and GFR category reported as per KDIGO guideline 2012

3. In patients, with eGFR creating between 45-59 ml/min/1.73 m2 (G3) and without any marker of Kidney damage, It is recommended to measure

4. eGFR category G1 OR G2 does not fullfill 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).

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 PROI	FILE	
IRON: SERUM by FERROZINE, SPECTROPHOTOMETRY	53.12 <sup>L</sup>	μg/dL	59.0 - 158.0
UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM by FERROZINE, SPECTROPHOTOMETERY	59.65 <sup>L</sup>	μg/dL	150.0 - 336.0
TOTAL IRON BINDING CAPACITY (TIBC) :SERUM by SPECTROPHOTOMETERY	192.14 <sup>L</sup>	μg/dL	230 - 430
%TRANSFERRIN SATURATION: SERUM by CALCULATED, SPECTROPHOTOMETERY (FERENE)	47.1	%	15.0 - 50.0
TRANSFERRIN: SERUM by SPECTROPHOTOMETERY (FERENE)	80.07 <sup>L</sup>	mg/dL	200.0 - 350.0

INTERIALION.			
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

#### 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.

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 meaning 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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**Value** Unit **Biological Reference interval Test Name** 

#### **ENDOCRINOLOGY**

#### THYROID FUNCTION TEST: TOTAL

TRIIODOTHYRONINE (T3): SERUM 1.282 ng/mL 0.35 - 1.93

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

THYROXINE (T4): SERUM μgm/dL 6.3 4.87 - 12.60

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

THYROID STIMULATING HORMONE (TSH): SERUM  $5.999^{H}$ μIU/mL 0.35 - 5.50by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

3rd GENERATION, ULTRASENSITIVE

#### INTERPRETATION:

TSH levels are subject to circadian variation, reaching peak levels between 2-4 a.m and at a minimum between 6-10 pm. The variation is of the order of 50%. Hence time of the day has influence on the measured serum TSH concentrations. TSH stimulates the production and secretion of the metabolically active hormones, thyroxine (T4) and triiodothyronine (T3). Failure at any level of regulation of the hypothalamic-pituitary-thyroid axis will result in either underproduction (hypothyroidism) or overproduction(hyperthyroidism) of T4 and/or T3.

CLINICAL CONDITION	Т3	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
- 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.

TRIIODOTHYRONINE (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



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COLLECTED BY : SURJESH REG. NO./LAB NO. : 012412030033

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 : 03/Dec/2024 10:46 AM

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 CLIENT CODE.
 : KOS DIAGNOSTIC LAB
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 : 03/Dec/2024 12:01PM

**CLIENT ADDRESS**: 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name			Value	Uni	t	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	IMENDATIONS OF TSH L	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, 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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 REPORTING DATE
 : 03/Dec/2024 01:48PM

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Test Name Value Unit Biological Reference interval

#### INTACT PARATHYROID HORMONE (PTH)

INTACT PARATHROID HORMONE (PTH): SERUM 124.9<sup>H</sup> pg/mL 9.5 - 75.0 by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

#### Intrepretation:-

Parathyroid hormone (PTH) is produced and secreted by the parathyroid glands, which are located along the posterior aspect of the thyroid gland. The serum calcium level regulates PTH secretion via negative feedback through the parathyroid calcium sensing receptor (CASR). Decreased calcium levels stimulate PTH release. Secreted PTH interacts with its specific type II G-protein receptor, causing rapid increases in renal tubular reabsorption of calcium and decreased phosphorus reabsorption. It also participates in long-term calciostatic functions by enhancing mobilization of calcium from bone and increasing renal synthesis of 1,25-dihydroxy vitamin D, which, in turn, increases intestinal calcium absorption.

The assay is useful for:

- Differential diagnosis of hypercalcemia
- Diagnosis of primary, secondary, and tertiary hyperparathyroidism
- Diagnosis of hypoparathyroidism
- Monitoring end-stage renal failure patients for possible renal osteodystrophy

#### Interpretation of results:

- An (appropriately) low PTH level and high phosphorus level in a hypercalcemic patient suggests that the hypercalcemia is not caused by PTH or PTH-like substances.
- An (appropriately) low PTH level with a low phosphorus level in a hypercalcemic patient suggests the diagnosis of paraneoplastic hypercalcemia.
- A low or normal PTH in a patient with hypocalcemia suggests hypoparathyroidism.

Low serum calcium and high PTH levels in a patient with normal renal function suggest resistance to PTH action (pseudohypoparathyroidism type 1a, 1b, 1c, or 2) or, very rarely, bio-ineffective PTH.

Elevated PTH value with a normal serum calcium in many cases in India is due to secondary hyperparathyroidism, primary cause being Vitamin D deficiency.



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 CLIENT CODE.
 : KOS DIAGNOSTIC LAB
 REPORTING DATE
 : 05/Dec/2024 10:03AM

CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name Value Unit Biological Reference interval

# IMMUNOPATHOLOGY/SEROLOGY ANTI NUCLEAR ANTIBODY/FACTOR (ANA/ANF)

ANTI NUCLEUR ANTIBODIES (ANA): SERUM 0.84 INDEX VALUE NEGATIVE: < 1.0

by ELISA (ENZYME LINKED IMMUNOASSAY)

BORDERLINE: 1.0 - 1.20

POSITIVE: > 1.20

#### **INTERPRETATION:-**

1. For diagnostic purposes, ANA value should be used as an adjuvant to other clinical and laboratory data available.

2.Measurement of antinuclear antibodies (ANAs) in serum is the most commonly performed screening test for patients suspected of having a systemic rheumatic disease, also referred to as connective tissue disease.

3.ANAs occur in patients with a variety of autoimmune diseases, both systemic and organ-specific. They are particularly common in the systemic rheumatic diseases, which include lupus erythematosus (LE), discoid LE, drug-induced LE, mixed connective tissue disease, Sjogren syndrome scleroderma (systemic sclerosis), CREST (calcinosis, Raynaud's phenomenon, esophageal dysmotility, sclerodactyly, telangiectasia) syndrome, polymyositis/dermatomyositis, and rheumatoid arthritis.

NOTE:

1. The diagnosis of a systemic rheumatic disease is based primarily on the presence of compatible clinical signs and symptoms.

The results of tests for autoantibodies including ANA and specific autoantibodies are ancillary. Additional diagnostic criteria include consistent histopathology or specific radiographic findings. Although individual systemic rheumatic diseases are relatively uncommon, a great many patients present with clinical findings that are compatible with a systemic rheumatic disease ANA screening may be useful for ruling out the disease.

2.Secondary, disease specific auto antibodies maybe ordered for patients who are screen positive as ancillary aids for the diagnosis of specific auto-immune disorders.



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**CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT

Value Unit **Biological Reference interval Test Name** 

#### **VITAMINS**

#### VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM ng/mL DEFICIENCY: < 20.0  $10.469^{L}$ 

by CLIA (CHEMILUMINESCENCE IMMUNOASSAY) INSUFFICIENCY: 20.0 - 30.0 SUFFICIENCY: 30.0 - 100.0

**TOXICITY:** > 100.0

**INTERPRETATION:** 

DEFICIENT:	< 20	ng/mL
INSUFFICIENT:	21 - 29	ng/mL
PREFFERED RANGE:	30 - 100	ng/mL
INTOXICATION:	> 100	ng/mL

- 1. Vitamin D compounds are derived from dietary ergocalciferol (from plants, Vitamin D2), or cholecalciferol (from animals, Vitamin D3), or by conversion of 7- dihydrocholecalciferol to Vitamin D3 in the skin upon Ultraviolet exposure.

  2.25-OH--Vitamin D represents the main body resevoir and transport form of Vitamin D and transport form of Vitamin D, being stored in adipose
- tissue and tightly bound by a transport protein while in circulation.
- 3. Vitamin D plays a primary role in the maintenance of calcium homeostatis. It promotes calcium absorption, renal calcium absorption and phosphate reabsorption, skeletal calcium deposition, calcium mobilization, mainly regulated by parathyroid harmone (PTH).

  4. Severe deficiency may lead to failure to mineralize newly formed osteoid in bone, resulting in rickets in children and osteomalacia in adults.
- DECREASED:
- 1.Lack of sunshine exposure.
- 2.Inadequate intake, malabsorption (celiac disease)
- 3. Depressed Hepatic Vitamin D 25- hydroxylase activity
- 4. Secondary to advanced Liver disease
- 5. Osteoporosis and Secondary Hyperparathroidism (Mild to Moderate deficiency)
- 6.Enzyme Inducing drugs: anti-epileptic drugs like phenytoin, phenobarbital and carbamazepine, that increases Vitamin D metabolism. INCREASED:
- 1. Hypervitaminosis D is Rare, and is seen only after prolonged exposure to extremely high doses of Vitamin D. When it occurs, it can result in severe hypercalcemia and hyperphophatemia.

CAUTION: Replacement therapy in deficient individuals must be monitored by periodic assessment of Vitamin D levels in order to prevent hypervitaminosis D

NOTE:-Dark coloured individuals as compare to whites, is at higher risk of developing Vitamin D deficiency due to excess of melanin pigment which interefere with Vitamin D absorption.



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



(A Unit of KOS Healthcare)



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MD (Pathology & Microbiology)
Chairman & Consultant Pathologist

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

NAME : Mr. JASBIR SINGH

**AGE/ GENDER** : 48 YRS/MALE **PATIENT ID** : 1689193

COLLECTED BY : SURJESH REG. NO./LAB NO. : 012412030033

 REFERRED BY
 : 03/Dec/2024 10:46 AM

 BARCODE NO.
 : 01521904
 COLLECTION DATE
 : 04/Dec/2024 12:14PM

 CLIENT CODE.
 : KOS DIAGNOSTIC LAB
 REPORTING DATE
 : 03/Dec/2024 12:33PM

CLIENT ADDRESS : 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name Value Unit Biological Reference interval

#### VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM **1516.8**<sup>H</sup> pg/mL 190.0 - 830

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

#### **INTERPRETATION:-**

INCREASED VITAMIN B12	DECREASED VITAMIN B12
1.Ingestion of Vitamin C	1.Pregnancy
2.Ingestion of Estrogen	2.DRUGS:Aspirin, Anti-convulsants, Colchicine
3.Ingestion of Vitamin A	3.Ethanol Igestion
4.Hepatocellular injury	4. Contraceptive Harmones
5.Myeloproliferative disorder	5.Haemodialysis
6.Uremia	6. Multiple Myeloma

- 1. Vitamin B12 (cobalamin) is necessary for hematopoiesis and normal neuronal function.
- 2.In humans, it is obtained only from animal proteins and requires intrinsic factor (IF) for absorption.
- 3. The body uses its vitamin B12 stores very economically, reabsorbing vitamin B12 from the ileum and returning it to the liver; very little is excreted.
- 4.Vitamin B12 deficiency may be due to lack of IF secretion by gastric mucosa (eg. gastrectomy, gastric atrophy) or intestinal malabsorption (eg, ileal resection, small intestinal diseases).
- 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.



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REFERRED BY **REGISTRATION DATE** : 03/Dec/2024 10:50 AM BARCODE NO. :01521904 **COLLECTION DATE** :04/Dec/2024 12:14PM CLIENT CODE. : KOS DIAGNOSTIC LAB REPORTING DATE : 03/Dec/2024 11:50AM

**CLIENT ADDRESS** : 6349/1, NICHOLSON ROAD, AMBALA CANTT

**Value** Unit **Biological Reference interval Test Name** 

#### VITAMIN B9/FOLIC ACID/FOLATE

VITAMIN B9/FOLIC ACID/FOLATE: SERUM by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

10.2 ng/mL DEFICIENT: < 3.37

**INTERMEDIATE: 3.37 - 5.38** 

NORMAL: > 5.38

**INTERPRETATION** 

RESULT IN ng/mL	REMARKS
0.35 – 3.37	DEFICIENT
3.38 – 5.38	INTERMEDIATE
5.39 - 100.00	NORMAL

#### NOTE:

- 1. Drugs like Methotrexate & Leucovorin interfere with folate measurement 2. To differentiate vitamin B12 & folate deficiency, measurement of Methyl malonic acid in urine & serum Homocysteine level is suggested
- Risk of toxicity from folic acid is low as it is a water soluble vitamin regularly excreted in urine

- 1. Folate plays an important role in the synthesis of purine & pyrimidines in the body and is important for the maturation of erythrocytes.

  2. It is widely available from plants and to a lesser extent organ meats, but more than half the folate content of food is lost during cooking.

  3. Folate deficiency is commonly prevalent in alcoholic liver disease, pregnancy and the elderly. It may result from poor intestinal absorption, nutrition deficiency, excessive demand as in pregnancy or in malignancy and in response to certain drugs like Methotres & anticonvulsants.

  4. Decreased Levels Megaloblastic anemia, Infantile hyperthyroidism, Alcoholism, Malnutrition, Scurvy, Liver disease, B12 deficiency, dietary
- amino acid excess, adult Celiac disease, Tropical Sprue, Crohn's disease, Hemolytic anemias, Carcinomas, Myelofibrosis, vitamin Bó deficiency, pregnancy, Whipple's disease, extensive intestinal resection and severe exfoliative dermatitis



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**Value** Unit **Biological Reference interval Test Name** 

### **CLINICAL PATHOLOGY URINE ROUTINE & MICROSCOPIC EXAMINATION**

#### PHYSICAL EXAMINATION

QUANTITY RECIEVED 10 ml by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

PALE YELLOW AMBER YELLOW COLOUR

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

TRANSPARANCY **HAZY CLEAR** 

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

SPECIFIC GRAVITY 1.01 1.002 - 1.030

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

**CHEMICAL EXAMINATION** 

**ACIDIC** REACTION by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

3+ **NEGATIVE (-ve)** by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

**SUGAR** Negative NEGATIVE (-ve)

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

рН 6.5 5.0 - 7.5by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

BILIRUBIN Negative NEGATIVE (-ve)

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

NEGATIVE (-ve) **NITRITE** Negative

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY. **UROBILINOGEN** Normal EU/dL 0.2 - 1.0

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

KETONE BODIES Negative NEGATIVE (-ve) by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

**NEGATIVE (-ve)** 

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY ASCORBIC ACID NEGATIVE (-ve) NEGATIVE (-ve)

5-6

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

**MICROSCOPIC EXAMINATION** 

RED BLOOD CELLS (RBCs)

by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT



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

/HPF

0 - 3





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Test Name	Value	Unit	Biological Reference interval
PUS CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	3-4	/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) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	ABSENT		ABSENT



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# KOS Diagnostic Lab (A Unit of KOS Healthcare)



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 COLLECTION DATE
 : 04/Dec/2024 12:14PM

 CLIENT CODE.
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 REPORTING DATE
 : 04/Dec/2024 06:13PM

**CLIENT ADDRESS**: 6349/1, NICHOLSON ROAD, AMBALA CANTT

Test Name Value Unit Biological Reference interval

#### **PROTEINS: 24 HOURS URINE**

URINE VOLUME: 24 HOUR 1550 ml

by SPECTROPHOTOMETRY

PROTEINS: 24 HOURS URINE by BIURET, SPECTROPHOTOMETRY 4307.92<sup>H</sup> mg/ 24 HOURS 25 -160

**INTERPRETATION:** 

TYPES OF PROTEINURIA	TOTAL PROTEINS IN mg/24 HOURS	CONDITIONS
MINIMAL PROTEINURIA:	150 - 500 mg/24 hours	Chronic pyelonephritis, Chronic Interstial Nephritis, Renal Tubular disease, Postural
MODERATE PROTEINURIA:	500 - 1000 mg/24 hours	Nephrosclerosis, Multiple Myeloma, Toxic Nephropathy, Renal Calculi
HEAVY PROTEINURIA:	1000 - 3000 mg/24 hours	Nephrotic Syndrome, Acute Rapidly Progressive & Chronic Glomerulonephritis, Diabetes mellitus, Lupus erythematosus, Druga like Pencillamine, Heavy metals like Gold & Mercury.

#### NOTE:

1.Excreation of total protein in individuals is highly variable with or without kidney disease.

2. Conditions affecting protein excreation other than kidney didease are urinary tract infection, diet, mensturation & physical activity.

#### COMMENT

1. Diagnosis of kidney disease and response to therapy is usually obtained by quatitattively analyzing the amount of protein excreated in urine over a 24 hour period.



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**Value** Unit **Test Name Biological Reference interval** 

#### SPECIAL INVESTIGATIONS

#### PROTEIN ELECTROPHORESIS: SERUM

TOTAL PROTEINS: SERUM by MIGRATION GEL ELECTROPHORESIS	5.68 <sup>L</sup>	gm/dL	6.20 - 8.00
ALBUMIN: SERUM  by MIGRATION GEL ELECTROPHORESIS	$2.16^{L}$	gm/dL	3.50 - 5.50
A: G RATIO: SERUM by MIGRATION GEL ELECTROPHORESIS	0.61 <sup>L</sup>	RATIO	1.00 - 2.00
ALPHA 1 GLOBULIN by MIGRATION GEL ELECTROPHORESIS	0.24	gm/dL	0.11 - 0.40
ALPHA 2 GLOBULIN by MIGRATION GEL ELECTROPHORESIS	0.81	gm/dL	0.43 - 1.03
BETA 1 GLOBULIN by MIGRATION GEL ELECTROPHORESIS	0.32	gm/dL	0.30 - 0.59
BETA 2 GLOBULIN by MIGRATION GEL ELECTROPHORESIS	0.49	gm/dL	0.20 - 0.53
GAMMA GLOBULIN by MIGRATION GEL ELECTROPHORESIS	1.65	gm/dL	0.75 - 1.80
MYELOMA (M) BAND/SPIKE by MIGRATION GEL ELECTROPHORESIS	NO MONOCLONAL BAND SEEN	gm/dL	
INTERPRETATION	Hypergammaglobulir		

#### **ADVICE**

#### KINDLY CORRELATE CLINICALLY

- 1. Serum protein electrophoresis is commonly used to identify patients with multiple myeloma and disorders of serum proteins.
- 2. Electrophoresis is a method of separating proteins based on their physical properties, the pattern of serum protein electrophoresis results depends on the frations of 2 types of protein: albumin and globulin (alpha 1 alpha2, beta and gamma.)
- 3.A homogeneous spike-like peak in a focal region of the gamma-globulin zone indicates a monoclonal gammopathy.
- 4. Monoclonal gammopathies are associated with a clonal process that is malignant or potentially malignant, including multiple myeloma, Waldenstrom macroglobulinemia, solitary plasmacytoma, smoldering multiple myeloma, monoclonal gammopathy of undetermined significance, plasma cell leukemia, heavy chain disease, and amyloidosis.
- 5.M-protein (in the gamma region) level greater than 3 g/dL should be interpreted along with other radiologic and haematological findings to arrive at a diagnosis of Multiple myeloma and must not be considered in isolation.
- 6.Occasionally M protein may appear as a narrow spike in the beta or alpha2 regions also.
- 7. Up to one fifth of patients with Myeloma may have an M-protein spike of less than 1 g /dL.
- 8. Hypogammaglobulinemia on serum protein electrophoresis occurs in about 10% of patients with multiple myeloma who do not have a serum



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M-protein spike

9.Most of these patients have a large amount of Bence Jones protein (monoclonal free kappa or lambda chain) in their urine, wherein urine protein electrophoresis should be performed. Monoclonal gammopathy is present in up to 8 percent of healthy geriatric patients.

NOTE:

The following conditions require serum immunofixation to confirm monoclonality or to differentiate monoclonal and polyclonal disoders.

1.A well defined "M" band.

2.Faint band

3. Chronic inflammatory pattern (decreased albumin, increased alpha, increased gamma fractions)

4. Isolated increase in any region with an otherwise normal pattern.

5. Shouldering of albumin peak along anodal or cathodal side may be seen with lipoproteins, drugs, bilirubin or radiological contrast.

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



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#### Mr. JASBIR SINGH

PID NO: P33724536814517 Age: 48 Year(s) Sex: Male



#### Reference: DR.VINAY CHOPRA

Sample Collected At:
DR VINAY KUMAR CHOPRA
DR VINAY KUMAR CHOPRA KOS
Diagnostic Lab 6349/I Nicholson Road
Ambala Cantt HRY 133001. 06-HR 13
Sample Processed At: Metropolis
Healthcare Ltd E-21, B1 Mohan Co-op Ind
Estate New Delhi-110044

#### VID: 240111108713274

Registered On: 06/12/2024 12:22 PM Collected On: 05/12/2024 12:20PM Reported On: 07/12/2024 03:13 PM

**Investigation** 

**Observed Value** 

Unit

**Biological Reference Interval** 

### PROTEIN ELECTROPHORESIS

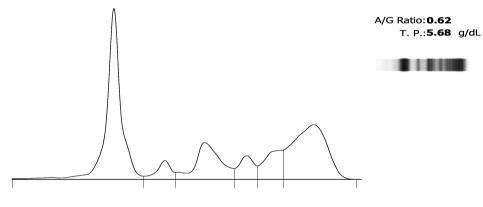
Name: JASBIR SINGH

Sample : **9** Sex :

м

Date: **12/7/2024**ID: **0527897386** 

Age :48



Fractions	%		Ref. %	Conc.	Ref. Conc.
Albumin	38.1	<	55.8 - 66.1	2.16	3.57 - 5.42
Alpha 1	4.2		2.9 - 4.9	0.24	0.19 - 0.40
Alpha 2	14.3	>	7.1 - 11.8	0.81	0.45 - 0.96
Beta 1	5.6		4.7 - 7.2	0.32	0.30 - 0.59
Beta 2	8.7	>	3.2 - 6.5	0.49	0.20 - 0.53
Gamma	29.1	>	11.1 - 18.8	1.65	0.71 - 1.54

Signature

-- End of Report --



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Page 3 of 3

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