CLIENT CODE.



# **KOS Diagnostic Lab**

(A Unit of KOS Healthcare)



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

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

: 23/Dec/2024 10:37AM

**NAME** : Mr. ROHAN GOEL

**AGE/ GENDER** : 25 YRS/MALE **PATIENT ID** : 1706294

**COLLECTED BY** : SURJESH REG. NO./LAB NO. :012412230016

REFERRED BY : CENTRAL PHOENIX CLUB (AMBALA CANTT) **REGISTRATION DATE** : 23/Dec/2024 09:54 AM BARCODE NO. :01522882 **COLLECTION DATE** : 23/Dec/2024 10:00AM

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

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

REPORTING DATE

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

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

HAEMOGLOBIN (HB)	15	gm/dL	12.0 - 17.0
RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	5.2 <sup>H</sup>	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	48.2	%	40.0 - 54.0
MEAN CORPUSCULAR VOLUME (MCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	92.6	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	28.9	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	31.2 <sup>L</sup>	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	13.7	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	47.4	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	17.81	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	24.44	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	8260	/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 %



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



by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER



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DIFFERENTIAL LEUCOCYTE COUNT (DLC)				
NEUTROPHILS	45 <sup>L</sup>	%	50 - 70	
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY LYMPHOCYTES	45 <sup>H</sup>	%	20 - 40	
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY				
EOSINOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	2	%	1 - 6	
MONOCYTES	8	%	2 - 12	
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY				
BASOPHILS by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	%	0 - 1	
ABSOLUTE LEUKOCYTES (WBC) COUNT				
ABSOLUTE NEUTROPHIL COUNT	3717	/cmm	2000 - 7500	
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY				
ABSOLUTE LYMPHOCYTE COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	3717	/cmm	800 - 4900	
ABSOLUTE EOSINOPHIL COUNT	165	/cmm	40 - 440	
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY ABSOLUTE MONOCYTE COUNT	661	/cmm	80 - 880	
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	001	/ CIIIII	60 - 66U	
ABSOLUTE BASOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0	/cmm	0 - 110	
PLATELETS AND OTHER PLATELET PREDICTIVE	MARKERS.			
PLATELET COUNT (PLT)	225000	/cmm	150000 - 450000	
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE				
PLATELETCRIT (PCT) by HYDRO DYNAMIC FOCUSING. ELECTRICAL IMPEDENCE	0.27	%	0.10 - 0.36	
MEAN PLATELET VOLUME (MPV)	12 <sup>H</sup>	fL	6.50 - 12.0	
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			20000 00000	
PLATELET LARGE CELL COUNT (P-LCC) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	92000 <sup>H</sup>	/cmm	30000 - 90000	
PLATELET LARGE CELL RATIO (P-LCR) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	40.9	%	11.0 - 45.0	
PLATELET DISTRIBUTION WIDTH (PDW)	16.4	%	15.0 - 17.0	
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE				
NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD				



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Test Name Value Unit Biological Reference interval

### GLYCOSYLATED HAEMOGLOBIN (HBA1C)

GLYCOSYLATED HAEMOGLOBIN (HbA1c): 5.1 % 4.0 - 6.4

WHOLE BLOOD

by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)

ESTIMATED AVERAGE PLASMA GLUCOSE 99.67 mg/dL 60.00 - 140.00

by HPLC (HIGH PERFORMANCE LIQUID CHROMATOGRAPHY)

### **INTERPRETATION:**

AS PER AMERICAN DIABETES ASSOCIATION (ADA):			
REFERENCE GROUP GLYCOSYLATED HEMOGLOGIB (HBAIC) in %			
Non diabetic Adults >= 18 years	<5.7		
At Risk (Prediabetes)	5.7 – 6.4		
Diagnosing Diabetes	>= 6.5		
	Age > 19 Years		
	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 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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# **ERYTHROCYTE SEDIMENTATION RATE (ESR)**

ERYTHROCYTE SEDIMENTATION RATE (ESR)

mm/1st hr

by RED CELL AGGREGATION BY CAPILLARY PHOTOMETRY

### INTERPRETATION:

CLIENT CODE.

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

GLUCOSE FASTING (F): PLASMA 89.77 NORMAL: < 100.0 mg/dL

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

DIABETIC: > 0R = 126.0

: 23/Dec/2024 11:04AM

CLIENT CODE.

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	E: BASIC	
CHOLESTEROL TOTAL: SERUM by CHOLESTEROL OXIDASE PAP	140.19	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)	94.38	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	31.42	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMETRY	89.89	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	108.77	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	18.88	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM by CALCULATED, SPECTROPHOTOMETRY	374.76	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	4.46 <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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LDL/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.86	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	3	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

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	0.69	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.16	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.53	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	16.7	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	31.7	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by Calculated, spectrophotometry	0.53	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by Para nitrophenyl phosphatase by amino methyl propanol	129.48	U/L	40.0 - 130.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	29.4	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	6.99	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	4.25	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.74	gm/dL	2.30 - 3.50
A: GRATIO: SERUM by calculated, spectrophotometry	1.55	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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### **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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Test Name	Value	Unit	Biological Reference interval
KIDNI	EY FUNCTION TE	EST (COMPLETE)	
UREA: SERUM by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)	21.58	mg/dL	10.00 - 50.00
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETERY	0.9	mg/dL	0.40 - 1.40
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY	10.08	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	11.2	RATIO	10.0 - 20.0
UREA/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	23.98	RATIO	
URIC ACID: SERUM by URICASE - OXIDASE PEROXIDASE	6.61	mg/dL	3.60 - 7.70
CALCIUM: SERUM by ARSENAZO III, SPECTROPHOTOMETRY	10.37	mg/dL	8.50 - 10.60
PHOSPHOROUS: SERUM by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY	3.44	mg/dL	2.30 - 4.70
<u>ELECTROLYTES</u>			
SODIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)	142.2	mmol/L	135.0 - 150.0
POTASSIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)	4.05	mmol/L	3.50 - 5.00
CHLORIDE: SERUM by ISE (ION SELECTIVE ELECTRODE)	106.65	mmol/L	90.0 - 110.0
ESTIMATED GLOMERULAR FILTERATION RATE			

ESTIMATED GLOMERULAR FILTERATION RATE 121.6

(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.



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**NAME** : Mr. ROHAN GOEL

AGE/ GENDER : 25 YRS/MALE **PATIENT ID** : 1706294

**COLLECTED BY** : SURJESH REG. NO./LAB NO. :012412230016

REFERRED BY : CENTRAL PHOENIX CLUB (AMBALA CANTT) **REGISTRATION DATE** : 23/Dec/2024 09:54 AM BARCODE NO. :01522882 **COLLECTION DATE** : 23/Dec/2024 10:00AM

CLIENT CODE. : KOS DIAGNOSTIC LAB REPORTING DATE :23/Dec/2024 12:14PM

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

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**:

STATE OF SECULE OF THE FERTITION OF THE			
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	



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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

eGFR with Cystatin C for confirmation of CKD

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 PROF	TLE	
IRON: SERUM by FERROZINE, SPECTROPHOTOMETRY	120.01	μg/dL	59.0 - 158.0
UNSATURATED IRON BINDING CAPACITY (UIBC) :SERUM by FERROZINE, SPECTROPHOTOMETERY	226.79	μg/dL	150.0 - 336.0
TOTAL IRON BINDING CAPACITY (TIBC) :SERUM by SPECTROPHOTOMETERY	346.8	μg/dL	230 - 430
%TRANSFERRIN SATURATION: SERUM by CALCULATED, SPECTROPHOTOMETERY (FERENE)	34.6	%	15.0 - 50.0
TRANSFERRIN: SERUM by SPECTROPHOTOMETERY (FERENE)	246.23	mg/dL	200.0 - 350.0

# INTERPRETATION:

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 measure of protein transferrin which transports iron from the gut to storage sites in the bone marrow.

## % TRANSFERRIN SATURATION:

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



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

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

REPORTING DATE

### **FERRITIN**

FERRITIN: SERUM 77.41 ng/mL 21.81 - 274.66

by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

CLIENT CODE.

Serum ferritin appears to be in equilibrium with tissue ferritin and is a good indicator of storage iron in normal subjects and in most disorders. In patients with some hepatocellular diseases, malignancies and inflammatory diseases, serum ferritin is a disproportionately high estimate of storage iron because serum ferritin is an acute phase reactant. In such disorders iron deficiency anemia may exist with a normal serum ferritin concentration. In the presence of inflammation, persons with low serum ferritin are likely to respond to iron therapy.

### **DECREASED:**

- 1. Iron depletion appears to be the only condition associated with reduced serum ferritin concentrations.
- Hypothyroidism.
   Vitamin-C deficiency

# INCREASED FERRITIN DUE TO IRON OVERLOAD (PRIMARY):

- 1. Hemochromatosis or hemosiderosis.
- Wilson Disease

# INCREASED FERRITIN DUE TO IRON OVERLOAD (SECONDARY):

- 1. Transfusion overload
- Excess dietary Iron
   Porphyria Cutanea tada

# 4. Ineffective erythropolesis. INCREASED FERRITIN WITHOUT IRON OVERLOAD:

- 1. Liver disorders (NASH) or viral hepatitis (B/C)
- 2. Inflammatory conditions (Ferritin is a acute phase reactant) both acute and chronic.
- 3. Leukaemia, hodgkin's disease.
- 4. Alcohol excess.
- 5. Other malignancies in which increases probably reflect the escape of ferritin from damaged liver cells, impaired clearance from the plasma, synthesis of ferritin by tumour cells.
- Ferritin levels below 10 ng/ml have been reported as indicative of iron deficiency anemia.

### NOTE:

1. As Ferritin is an acute phase reactant, it is often raised in both acute and chronic inflammatory condition of the body such as infections leading to false positive results. It can thererfore mask a diagnostically low result. In such Cases serum ferritin levels should always be correlated with C-Reactive proteins to rule out any inflammatory conditions.

2. Patients with iron deficiency anaemia may occasionally have elevated or normal ferritin levels. This is usually seen in patients already receiving iron therapy or in patients with concomitant hepatocellular injury.



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

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

REPORTING DATE

# APOLIPOPROTEIN A-1 (APO-A1)

APOLIPOPROTEIN A-1: SERUM 111.05 mg/dL 101.00 - 223.00

by NEPHLOMETRY

### **INTERPRETATION:**

CLIENT CODE.

1. Apolipoprotein A1 (ApoA1) is the primary protein associated with high-density lipoprotein (HDL) particles, and plays a central role in reverse cholesterol transport.(1) HDL cholesterol (HDL-C) and ApoA1 concentrations are inversely related to the risk for coronary artery disease (CAD). 2. There are a variable number of ApoA1 proteins per HDL particle. Therefore, ApoA1 is not a 1:1 surrogate marker for HDL particles. Similarly, the number of ApoA1 proteins and the amount of cholesterol contained in HDL particles is highly variable. This heterogeneity has led to unique clinical findings related to ApoA1 compared with HDL-C.

3.Increased ApoA1 concentrations are more strongly associated with a reduction in risk of a first myocardial infarction than HDL-C concentrations.(

4.Low concentrations of ApoA1, but not HDL-C, are predictive of preclinical atherosclerosis as assed by computed tomography estimated coronary artery calcium (CAC) scoring.

5.Increased ApoA1, but not HDL-C concentrations, are associated with reduced cardiovascular events among statin-treated patients, even when LDL-C <50 mg/dL.(5) In statin-treated patients, patients whose ApoA1 increased while on treatment were at lower risk than those whose ApoA1 did not increase.

6.Low levels of apolipoprotein A1 (ApoA1) confer increased risk of atherosclerotic cardiovascular disease.

7. ApoA1 < 25 mg/dL may aid in the detection of a genetic disorder such as Tangier disease



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BARCODE NO. : 01522882 COLLECTION DATE : 23/Dec/2024 10:00AM

CLIENT CODE. : KOS DIAGNOSTIC LAB REPORTING DATE : 23/Dec/2024 03:06PM

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

Test Name Value Unit Biological Reference interval

# **APOLIPOPROTEIN B (APO-B)**

APOLIPOPROTEIN B: SERUM 121.5 mg/dL 53.0 - 182.0

by NEPHLOMETRY

## **INTERPRETATION:**

- 1.Apolipoprotein B (ApoB) is the primary protein component of low-density lipoprotein (LDL). LDL contains a variable amount of cholesterol, but each LDL contains exactly 1 ApoB protein. Therefore, ApoB is a superior indicator of circulating LDL compared to LDL cholesterol (LDL-C).
- 2. ApoB has been demonstrated to perform equally with LDL particles measured by nuclear magnetic resonance spectroscopy.
- 3.ApoB is strongly associated with increased risk of developing cardiovascular disease (CVD) and often outperforms LDL-C at predicting risk of coronary heart disease.
- 4. Patients with acceptable non-HDL-C (or LDL-C) but elevated ApoB remain at higher risk of developing CVD; conversely, patients with acceptably low ApoB but moderate non-HDL-C or LDL-C elevations are at a reduced risk for CVD.

### **SIGNIFICANCE**

Elevated ApoB confers increased risk of coronary artery disease ApoB can be used as a therapeutic target analogous to non-HDL-C and LDL-C.

RISK CATEGORY	THERAPEUTIC TARGET		
	APO B NON HDL-C LDL-C		LDL-C
MODERATE TO HIGH	< 90 mg/dL	< 130 mg/dL	< 100 mg/dL
VERY HIGH	< 80 mg/dL	< 100 mg/dL	< 70 mg/dL

Extremely low values of ApoB (<48 mg/dL) are related to malabsorption of food lipids and can lead to polyneuropathy.



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Test Name Value Unit Biological Reference interval

# **ENDOCRINOLOGY**

## THYROID FUNCTION TEST: TOTAL

TRIIODOTHYRONINE (T3): SERUM 1.37 ng/mL 0.35 - 1.93

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

THYROXINE (T4): SERUM 8.46  $\mu$ gm/dL 4.87 - 12.60

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

THYROID STIMULATING HORMONE (TSH): SERUM 2.519 μIU/mL 0.35 - 5.50

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

 ${\bf 3rd}\;{\bf GENERATION}, {\bf ULTRASENSITIVE}$ 

## **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, anabolic steroids and glucocorticoids may falsely affect the T3 and T4 levels and may cause false thyroid values for thyroid function tests.
- 2. Normal levels of T4 can also be seen in Hyperthyroid patients with :T3 Thyrotoxicosis, Decreased binding capacity due to hypoproteinemia or ingestion of certain drugs (e.g.: phenytoin, salicylates)
- 3. Serum T4 levels in neonates and infants are higher than values in the normal adult, due to the increased concentration of TBG in neonate serum.
- 4. TSH may be normal in central hypothyroidism, recent rapid correction of hyperthyroidism or hypothyroidism, pregnancy, phenytoin therapy.

TRIIODOTH	RONINE (T3) THYROXIN		NE (T4) THYROID STIMULATING HORMONE		LATING 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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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	
	RECON	MENDATIONS 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, 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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**Value** Unit **Biological Reference interval Test Name** 

## **TESTOSTERONE: TOTAL**

TESTOSTERONE - TOTAL: SERUM 4.09 ng/mL 0.47 - 9.80

by CMIA (CHEMILUMINESCENT MICROPARTICLE IMMUNOASSAY)

1. Testosterone is secreted in females by the ovary and formed indirectly from androstenedione in adrenal glands.
2. In males it is secreted by the testes. It circulates in blood bound largely to sex hormone binding globulin (SHBG). Less than 1% of the total testosterone is in the free form.

3.The bioavailable fraction includes the free form and that "weakly bound" to albumin (40% of the total in men and 20% of the total in women) and bound to cortisol binding globulin (CBG). It is the most potent circulating androgenic hormone.

4.The total testosterone bound to SHBG fluctuates since SHBG levels are affected by medication, disease, sex steroids and insulin.

### **CLINIC USE:**

- 1.Assesment of testicular functions in males
   2.Management of hirsutism and virilization in females
   INCREASED LEVELS:

- 1. Precocious puberty (Males)
- 2. Androgen resistance
- 3.Testoxicosis
- 4.Congenital Adrenal Hyperplasia 5.Polycystic ovarian disease
- 7. Ovárián tumors

### **DECREASED LEVELS:**

- 1.Delayed puberty (Males)
- 2. Gonádotropin deficiency
- 3. Testicular defects
- 4. Systemic diseases



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



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



(A Unit of KOS Healthcare)



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

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

**NAME** : Mr. ROHAN GOEL

**AGE/ GENDER** : 25 YRS/MALE **PATIENT ID** : 1706294

**COLLECTED BY** : SURJESH :012412230016 REG. NO./LAB NO.

REFERRED BY : CENTRAL PHOENIX CLUB (AMBALA CANTT) **REGISTRATION DATE** : 23/Dec/2024 09:54 AM BARCODE NO. :01522882 **COLLECTION DATE** : 23/Dec/2024 10:00AM

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

Value Unit **Biological Reference interval Test Name** 

REPORTING DATE

# **VITAMINS**

# VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM ng/mL DEFICIENCY: < 20.0  $20^{L}$ 

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

**TOXICITY:** > 100.0

:23/Dec/2024 12:14PM

**INTERPRETATION:** 

CLIENT CODE.

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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Test Name Value Unit Biological Reference interval

### **VITAMIN B12/COBALAMIN**

VITAMIN B12/COBALAMIN: SERUM **151<sup>L</sup>** pg/mL 190.0 - 890.0

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

# VITAMIN B9/FOLIC ACID/FOLATE

REPORTING DATE

VITAMIN B9/FOLIC ACID/FOLATE: SERUM 3.96 ng/mL DEFICIENT: < 3.37

by CLIA (CHEMILUMINESCENCE IMMUNOASSAY) **INTERMEDIATE: 3.37 - 5.38** 

NORMAL: > 5.38

: 23/Dec/2024 01:22PM

INTERPRETATION

CLIENT CODE.

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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Test Name Value Unit Biological Reference interval

# CLINICAL PATHOLOGY URINE ROUTINE & MICROSCOPIC EXAMINATION

### **PHYSICAL EXAMINATION**

QUANTITY RECIEVED 10 ml

COLOUR PALE YELLOW PALE YELLOW

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

TRANSPARANCY CLEAR CLEAR by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

SPECIFIC GRAVITY >=1.030 1.002 - 1.030

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

CHEMICAL EXAMINATION

REACTION ACIDIC by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

PROTEIN Negative NEGATIVE (-ve)

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

SUGAR Negative NEGATIVE (-ve)

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

pH 6 5.0 - 7.5

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

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

NITRITE Negative NEGATIVE (-ve)

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY.

UROBILINOGEN Normal EU/dL 0.2 - 1.0

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

KETONE BODIES

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

NEGATIVE (-ve)

BLOOD Negative NEGATIVE (-ve)

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

ASCORBIC ACID

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

NEGATIVE (-ve)

NEGATIVE (-ve)

MICROSCOPIC EXAMINATION

RED BLOOD CELLS (RBCs)

NEGATIVE (-ve) /HPF 0 - 3



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Test Name	Value	Unit	Biological Reference interval
by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT			
PUS CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	1-3	/HPF	0 - 5
EPITHELIAL CELLS by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	1-2	/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

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



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