

(A Unit of KOS Healthcare)



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

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

NAME : Mrs. PUSHPA

**AGE/ GENDER** : 55 YRS/FEMALE **PATIENT ID** : 1646729

COLLECTED BY: SURJESH REG. NO./LAB NO. : 012410180016

 REFERRED BY
 : 18/Oct/2024 10:27 AM

 BARCODE NO.
 : 01519109
 COLLECTION DATE
 : 18/Oct/2024 10:42AM

 CLIENT CODE.
 : KOS DIAGNOSTIC LAB
 REPORTING DATE
 : 18/Oct/2024 11:00AM

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

Test Name Value Unit Biological Reference interval

# SWASTHYA WELLNESS PANEL: 1.0 COMPLETE BLOOD COUNT (CBC)

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

HAEMOGLOBIN (HB) by CALORIMETRIC	13.6	gm/dL	12.0 - 16.0
RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	5.43 <sup>H</sup>	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	43.8	%	37.0 - 50.0
MEAN CORPUSCULAR VOLUME (MCV) by calculated by automated hematology analyzer	80.6	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by calculated by automated hematology analyzer	25 <sup>L</sup>	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by calculated by automated hematology analyzer	31.1 <sup>L</sup>	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	14.5	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	44.7	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	14.84	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	21.48	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	6930	/cmm	4000 - 11000
NUCLEATED RED BLOOD CELLS (nRBCS) by automated 6 part hematology analyzer	NIL		0.00 - 20.00
NUCLEATED RED BLOOD CELLS (nRBCS) % by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	NIL	%	< 10 %
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS by Flow cytometry by SF cube & microscopy	67	%	50 - 70



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Tt NI	M-I	11	District Defenses in the
Test Name	Value	Unit	Biological Reference interval
LYMPHOCYTES	26	%	20 - 40
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
EOSINOPHILS	2	%	1 - 6
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	_	0/	2 12
MONOCYTES  by Flow cytometry by SF cube & microscopy	5	%	2 - 12
BASOPHILS	0	%	0 - 1
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	U	/0	0-1
ABSOLUTE LEUKOCYTES (WBC) COUNT			
ABSOLUTE NEUTROPHIL COUNT	4643	/cmm	2000 - 7500
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE LYMPHOCYTE COUNT	1802	/cmm	800 - 4900
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE EOSINOPHIL COUNT	139	/cmm	40 - 440
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	0.47	,	00.000
ABSOLUTE MONOCYTE COUNT  by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	346	/cmm	80 - 880
ABSOLUTE BASOPHIL COUNT	0	/cmm	0 - 110
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	U	/CITIII	0-110
PLATELETS AND OTHER PLATELET PREDICTIVE MARKE	RS.		
PLATELET COUNT (PLT)	278000	/cmm	150000 - 450000
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	270000	7 0111111	100000 100000
PLATELETCRIT (PCT)	0.32	%	0.10 - 0.36
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
MEAN PLATELET VOLUME (MPV)	12	fL	6.50 - 12.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELET LARGE CELL COUNT (P-LCC)  by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	112000 <sup>H</sup>	/cmm	30000 - 90000
PLATELET LARGE CELL RATIO (P-LCR)	40.4	%	11.0 - 45.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	10.7	70	11.0 43.0
PLATELET DISTRIBUTION WIDTH (PDW)	16.7	%	15.0 - 17.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
NOTE: TEST CONDUCTED ON EDTA WHOLE BLOOD			



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Value Unit **Biological Reference interval** Test Name

#### ERYTHROCYTE SEDIMENTATION RATE (ESR)

**ERYTHROCYTE SEDIMENTATION RATE (ESR)** 

23H

mm/1st hr

0 - 20

: 18/Oct/2024 11:33AM

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 autoimmune 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:

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



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

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

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	251.17 <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)	114.98	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	80.07 <sup>H</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	148.1 <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	171.1 <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	23	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM  by CALCULATED, SPECTROPHOTOMETRY	617.32	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	3.14	RATIO	LOW RISK: 3.30 - 4.40 AVERAGE RISK: 4.50 - 7.0 MODERATE RISK: 7.10 - 11.0 HIGH RISK: > 11.0
LDL/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.85	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0



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TRIGLYCERIDES/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY

1.44<sup>L</sup>

**RATIO** 

3.00 - 5.00

**INTERPRETATION:** 

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

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

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	0.65	mg/dL	INFANT: 0.20 - 8.00 ADULT: 0.00 - 1.20
BILIRUBIN DIRECT (CONJUGATED): SERUM by DIAZO MODIFIED, SPECTROPHOTOMETRY	0.27	mg/dL	0.00 - 0.40
BILIRUBIN INDIRECT (UNCONJUGATED): SERUM by CALCULATED, SPECTROPHOTOMETRY	0.38	mg/dL	0.10 - 1.00
SGOT/AST: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	32.28	U/L	7.00 - 45.00
SGPT/ALT: SERUM by IFCC, WITHOUT PYRIDOXAL PHOSPHATE	44.28	U/L	0.00 - 49.00
AST/ALT RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	0.73	RATIO	0.00 - 46.00
ALKALINE PHOSPHATASE: SERUM by PARA NITROPHENYL PHOSPHATASE BY AMINO METI PROPANOL	91.2 HYL	U/L	40.0 - 150.0
GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM by SZASZ, SPECTROPHTOMETRY	33.4	U/L	0.00 - 55.0
TOTAL PROTEINS: SERUM by BIURET, SPECTROPHOTOMETRY	7.42	gm/dL	6.20 - 8.00
ALBUMIN: SERUM by BROMOCRESOL GREEN	4.71	gm/dL	3.50 - 5.50
GLOBULIN: SERUM by CALCULATED, SPECTROPHOTOMETRY	2.71	gm/dL	2.30 - 3.50
A : G RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	1.74	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		



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HEPATOCELLULAR CARCINOMA & CHRONIC HEPATITIS > 1.3 (Slightly Increased)

CLIENT CODE.

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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mmol/L

90.0 - 110.0

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Test Name	Value	Unit	Biological Reference interval
	KIDNEY FUNCTION TI	EST (COMPLETE)	
UREA: SERUM by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)	32.99	mg/dL	10.00 - 50.00
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETERY	0.51	mg/dL	0.40 - 1.20
RI OOD LIDEN NITDOGENI (RIINI): SEDLIM	15.42	ma/dl	7.0. 25.0

BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY	15.42	mg/dL	7.0 - 25.0
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM	30.24 <sup>H</sup>	RATIO	10.0 - 20.0
by CALCULATED, SPECTROPHOTOMETRY			
UREA/CREATININE RATIO: SERUM	64.69	RATIO	
by CALCULATED, SPECTROPHOTOMETRY		/ 11	0.50 / 00
URIC ACID: SERUM by URICASE - OXIDASE PEROXIDASE	4.4	mg/dL	2.50 - 6.80
CALCIUM: SERUM	8.86	mg/dL	8.50 - 10.60
by ARSENAZO III, SPECTROPHOTOMETRY		g	
PHOSPHOROUS: SERUM	3.9	mg/dL	2.30 - 4.70
by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY			
<u>ELECTROLYTES</u>			
SODIUM: SERUM	136.9	mmol/L	135.0 - 150.0
by ISE (ION SELECTIVE ELECTRODE)			
POTASSIUM: SERUM	3.8	mmol/L	3.50 - 5.00

102.68

#### CHLORIDE: SERUM by ISE (ION SELECTIVE ELECTRODE) **ESTIMATED GLOMERULAR FILTERATION RATE**

by ISE (ION SELECTIVE ELECTRODE)

**ESTIMATED GLOMERULAR FILTERATION RATE** 110.2

(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.



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Test Name Value Unit **Biological Reference interval** 

- 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:

CKD STAGE	DESCRIPTION	GFR ( mL/min/1.73m2 )	ASSOCIATED FINDINGS
CKD STAGE	DESCRIPTION	GFK ( IIIL/ IIIIII/ 1./ 3IIIZ )	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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Dr. Yugam Chopra MD (Pathology) CEO & Consultant Pathologist

**NAME** : Mrs. PUSHPA

AGE/ GENDER : 55 YRS/FEMALE **PATIENT ID** : 1646729

COLLECTED BY : SURJESH REG. NO./LAB NO. :012410180016

REFERRED BY **REGISTRATION DATE** : 18/Oct/2024 10:27 AM BARCODE NO. **COLLECTION DATE** : 18/Oct/2024 10:42AM :01519109

CLIENT CODE. : KOS DIAGNOSTIC LAB REPORTING DATE : 18/Oct/2024 12:08PM

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

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 creatinine 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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#### **VITAMINS**

#### VITAMIN D/25 HYDROXY VITAMIN D3

VITAMIN D (25-HYDROXY VITAMIN D3): SERUM by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

17.9<sup>L</sup>

ng/mL

**DEFICIENCY:** < 20.0

**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.
- 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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#### **VITAMIN B12/COBALAMIN**

VITAMIN B12/COBALAMIN: SERUM 948<sup>H</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, 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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#### CLINICAL PATHOLOGY

#### **URINE ROUTINE & MICROSCOPIC EXAMINATION**

#### PHYSICAL EXAMINATION

QUANTITY RECIEVED 10 ml by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

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

Negative

NEGATIVE (-ve)

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

BLOOD Negative NEGATIVE (-ve)

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY

ASCORBIC ACID

NEGATIVE (-ve)

NEGATIVE (-ve)

by DIP STICK/REFLECTANCE SPECTROPHOTOMETRY MICROSCOPIC EXAMINATION



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RED BLOOD CELLS (RBCs) by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	NEGATIVE (-ve)	/HPF	0 - 3
PUS CELLS  by MICROSCOPY ON CENTRIFUGED URINARY SEDIMENT	2-4	/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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