

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

NAME : Mrs. SWATI NIGAM

AGE/ GENDER : 37 YRS/FEMALE **PATIENT ID** : 1652074

COLLECTED BY REG. NO./LAB NO. : 012410240033

REFERRED BY **REGISTRATION DATE** : 24/Oct/2024 12:08 PM BARCODE NO. :01519482 **COLLECTION DATE** : 24/Oct/2024 12:19PM CLIENT CODE. : KOS DIAGNOSTIC LAB REPORTING DATE : 24/Oct/2024 12:36PM

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

Test Name Value Unit **Biological Reference interval**

SWASTHYA WELLNESS PANEL: 15.0 COMPLETE BLOOD COUNT (CBC)

RED BLOOD CELLS (RBCS) COUNT AND INDICES

HAEMOGLOBIN (HB) by CALORIMETRIC	10.1 ^L	gm/dL	12.0 - 16.0
RED BLOOD CELL (RBC) COUNT by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	4.94	Millions/cmm	3.50 - 5.00
PACKED CELL VOLUME (PCV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	35.8 ^L	%	37.0 - 50.0
MEAN CORPUSCULAR VOLUME (MCV) by Calculated by automated hematology analyzer	72.4 ^L	fL	80.0 - 100.0
MEAN CORPUSCULAR HAEMOGLOBIN (MCH) by Calculated by automated hematology analyzer	20.5 ^L	pg	27.0 - 34.0
MEAN CORPUSCULAR HEMOGLOBIN CONC. (MCHC) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	28.3 ^L	g/dL	32.0 - 36.0
RED CELL DISTRIBUTION WIDTH (RDW-CV) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	21.3 ^H	%	11.00 - 16.00
RED CELL DISTRIBUTION WIDTH (RDW-SD) by CALCULATED BY AUTOMATED HEMATOLOGY ANALYZER	55.8	fL	35.0 - 56.0
MENTZERS INDEX by CALCULATED	14.66	RATIO	BETA THALASSEMIA TRAIT: < 13.0 IRON DEFICIENCY ANEMIA: >13.0
GREEN & KING INDEX by CALCULATED	31.3	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	5950	/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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Test Name	Value	Unit	Biological Reference interval
DIFFERENTIAL LEUCOCYTE COUNT (DLC)			
NEUTROPHILS	81 ^H	%	50 - 70
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY		0.4	00.40
LYMPHOCYTES by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	11 ^L	%	20 - 40
EOSINOPHILS	$\mathbf{0^L}$	%	1 - 6
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
MONOCYTES	8	%	2 - 12
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY BASOPHILS	0	%	0 - 1
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	U	70	0 - 1
ABSOLUTE LEUKOCYTES (WBC) COUNT			
ABSOLUTE NEUTROPHIL COUNT	4820	/cmm	2000 - 7500
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
ABSOLUTE LYMPHOCYTE COUNT	654 ^L	/cmm	800 - 4900
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY		,	10 110
ABSOLUTE EOSINOPHIL COUNT by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	$\mathbf{0^L}$	/cmm	40 - 440
ABSOLUTE MONOCYTE COUNT	476	/cmm	80 - 880
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY	470	/ CIIIII	00 - 000
ABSOLUTE BASOPHIL COUNT	0	/cmm	0 - 110
by FLOW CYTOMETRY BY SF CUBE & MICROSCOPY			
PLATELETS AND OTHER PLATELET PREDICTIVE	MARKERS.		
PLATELET COUNT (PLT)	236000	/cmm	150000 - 450000
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELETCRIT (PCT)	0.33	%	0.10 - 0.36
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	11	.cr	0.50 19.0
MEAN PLATELET VOLUME (MPV) by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	14 ^H	fL	6.50 - 12.0
PLATELET LARGE CELL COUNT (P-LCC)	135000 ^H	/cmm	30000 - 90000
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE	133000	/ CIIIII	00000 00000
PLATELET LARGE CELL RATIO (P-LCR)	57 ^H	%	11.0 - 45.0
by HYDRO DYNAMIC FOCUSING, ELECTRICAL IMPEDENCE			
PLATELET DISTRIBUTION WIDTH (PDW)	15.9	%	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**



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Value Unit **Biological Reference interval Test Name**

CLINICAL CHEMISTRY/BIOCHEMISTRY GLUCOSE FASTING (F)

GLUCOSE FASTING (F): PLASMA NORMAL: < 100.0 110.68^H 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	160.83	mg/dL	OPTIMAL: < 200.0 BORDERLINE HIGH: 200.0 - 239.0 HIGH CHOLESTEROL: > OR = 240.0
TRIGLYCERIDES: SERUM by GLYCEROL PHOSPHATE OXIDASE (ENZYMATIC)	247.86 ^H	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	48.12	mg/dL	LOW HDL: < 30.0 BORDERLINE HIGH HDL: 30.0 - 60.0 HIGH HDL: > OR = 60.0
LDL CHOLESTEROL: SERUM by CALCULATED, SPECTROPHOTOMETRY	63.14	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	112.71	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	49.57 ^H	mg/dL	0.00 - 45.00
TOTAL LIPIDS: SERUM by CALCULATED, SPECTROPHOTOMETRY	569.52	mg/dL	350.00 - 700.00
CHOLESTEROL/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	3.34	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	1.31	RATIO	LOW RISK: 0.50 - 3.0 MODERATE RISK: 3.10 - 6.0 HIGH RISK: > 6.0
TRIGLYCERIDES/HDL RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	5.15 ^H	RATIO	3.00 - 5.00

REPORTING DATE

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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Value Unit **Test Name Biological Reference interval** LIVER FUNCTION TEST (COMPLETE) BILIRUBIN TOTAL: SERUM 0.59 mg/dL INFANT: 0.20 - 8.00 by DIAZOTIZATION, SPECTROPHOTOMETRY ADULT: 0.00 - 1.20 0.00 - 0.40BILIRUBIN DIRECT (CONJUGATED): SERUM 0.14 mg/dL by DIAZO MODIFIED, SPECTROPHOTOMETRY BILIRUBIN INDIRECT (UNCONJUGATED): SERUM 0.45 mg/dL 0.10 - 1.00

by IFCC, WITHOUT PYRIDOXAL PHOSPHATE SGPT/ALT: SERUM 21.8 U/L 0.00 - 49.00by IFCC, WITHOUT PYRIDOXAL PHOSPHATE AST/ALT RATIO: SERUM 0.92 **RATIO** 0.00 - 46.00by CALCULATED, SPECTROPHOTOMETRY ALKALINE PHOSPHATASE: SERUM 66.08 U/L 40.0 - 130.0 by PARA NITROPHENYL PHOSPHATASE BY AMINO METHYL PROPANOL GAMMA GLUTAMYL TRANSFERASE (GGT): SERUM 23.64 U/L 0.00 - 55.0

20.1

by SZASZ, SPECTROPHTOMETRY 6.39 TOTAL PROTEINS: SERUM gm/dL 6.20 - 8.00by BIURET, SPECTROPHOTOMETRY ALBUMIN: SERUM 3.75 gm/dL 3.50 - 5.50by BROMOCRESOL GREEN 2.64 2.30 - 3.50 **GLOBULIN: SERUM** gm/dL by CALCULATED, SPECTROPHOTOMETRY

1.42

by CALCULATED, SPECTROPHOTOMETRY INTERPRETATION

by CALCULATED, SPECTROPHOTOMETRY

SGOT/AST: SERUM

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:

A: G RATIO: SERUM

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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7.00 - 45.00

1.00 - 2.00

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Test Name Value Unit Biological Reference interval

DECREASED:

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

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

PROGNOSTIC SIGNIFICANCE:

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



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KIDNI	EY FUNCTION T	EST (COMPLETE)		
UREA: SERUM by UREASE - GLUTAMATE DEHYDROGENASE (GLDH)	10.39	mg/dL	10.00 - 50.00	
CREATININE: SERUM by ENZYMATIC, SPECTROPHOTOMETERY	0.82	mg/dL	0.40 - 1.20	
BLOOD UREA NITROGEN (BUN): SERUM by CALCULATED, SPECTROPHOTOMETRY	4.86 ^L	mg/dL	7.0 - 25.0	
BLOOD UREA NITROGEN (BUN)/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	5.93 ^L	RATIO	10.0 - 20.0	
UREA/CREATININE RATIO: SERUM by CALCULATED, SPECTROPHOTOMETRY	12.67	RATIO		
URIC ACID: SERUM by URICASE - OXIDASE PEROXIDASE	4.99	mg/dL	2.50 - 6.80	
CALCIUM: SERUM by ARSENAZO III, SPECTROPHOTOMETRY	9.4	mg/dL	8.50 - 10.60	
PHOSPHOROUS: SERUM by PHOSPHOMOLYBDATE, SPECTROPHOTOMETRY	3.68	mg/dL	2.30 - 4.70	
ELECTROLYTES				
SODIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)	146.2	mmol/L	135.0 - 150.0	
POTASSIUM: SERUM by ISE (ION SELECTIVE ELECTRODE)	4.01	mmol/L	3.50 - 5.00	
CHLORIDE: SERUM by ISE (ION SELECTIVE ELECTRODE)	109.65	mmol/L	90.0 - 110.0	
ESTIMATED GLOMERULAR FILTERATION RATE				

ESTIMATED GLOMERULAR FILTERATION RATE 94.4

(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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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**:

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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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



CONSULTANT PATHOLOGIST MBBS, MD (PATHOLOGY & MICROBIOLOGY)





(A Unit of KOS Healthcare)



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

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

NAME : Mrs. SWATI NIGAM

AGE/ GENDER : 37 YRS/FEMALE **PATIENT ID** : 1652074

COLLECTED BY REG. NO./LAB NO. : 012410240033

REFERRED BY **REGISTRATION DATE** : 24/Oct/2024 12:08 PM BARCODE NO. :01519482 **COLLECTION DATE** : 24/Oct/2024 12:19PM CLIENT CODE. : KOS DIAGNOSTIC LAB REPORTING DATE : 24/Oct/2024 01:24PM

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

Value Unit **Biological Reference interval Test Name**

FERRITIN

FERRITIN: SERUM 42.88 ng/mL 4.63 - 204.0

by CLIA (CHEMILUMINESCENCE IMMUNOASSAY)

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.
- 2. Hypothyroidism.3. 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.
- 6. 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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 : 24/Oct/2024 01:48PM

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

Test Name Value Unit Biological Reference interval

VITAMINS VITAMIN B12/COBALAMIN

VITAMIN B12/COBALAMIN: SERUM 659 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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NAME : Mrs. SWATI NIGAM

AGE/ GENDER : 37 YRS/FEMALE **PATIENT ID** : 1652074

COLLECTED BY REG. NO./LAB NO. :012410240033

REFERRED BY **REGISTRATION DATE** : 24/Oct/2024 12:08 PM BARCODE NO. :01519482 **COLLECTION DATE** : 24/Oct/2024 12:19PM CLIENT CODE. : KOS DIAGNOSTIC LAB REPORTING DATE : 24/Oct/2024 03:07PM

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)

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

Rechecked

** End Of Report



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