

PATIENT NAME : MATHEW JOSEPH

REF. DOCTOR : DR. KAMINI S MEHTA

KHARGHAR
9870331316

ACCESSION NO : 0040WF002390
PATIENT ID : MATHM16030540
CLIENT PATIENT ID:
ABHA NO :

AGE/SEX : 18 Years Male
DRAWN : 10/06/2023 09:13:02
RECEIVED : 10/06/2023 09:16:04
REPORTED : 10/06/2023 13:27:14

Test Report Status	Final	Results	Biological Reference Interval	Units
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HAEMATOLOGY - CBC
AGILUS TOTAL 2**BLOOD COUNTS, EDTA WHOLE BLOOD**

HEMOGLOBIN (HB)	14.5	13.0 - 17.0	g/dL
METHOD : OXIDASE-PEROXIDASE REACTION			
RED BLOOD CELL (RBC) COUNT	4.91	4.5 - 5.5	mil/ μ L
METHOD : ELECTRONIC IMPEDIMENT VARIATION			
WHITE BLOOD CELL (WBC) COUNT	10.1 High	4.0 - 10.0	thou/ μ L
METHOD : ELECTRONIC IMPEDIMENT VARIATION			
PLATELET COUNT	225	150 - 410	thou/ μ L
METHOD : ELECTRONIC IMPEDIMENT VARIATION			

RBC AND PLATELET INDICES

HEMATOCRIT (PCV)	41.8	40 - 50	%
METHOD : ELECTRONIC IMPEDIMENT/CALCULATION			
MEAN CORPUSCULAR VOLUME (MCV)	85.0	83 - 101	fL
METHOD : CALCULATED PARAMETER			
MEAN CORPUSCULAR HEMOGLOBIN (MCH)	29.6	27.0 - 32.0	pg
METHOD : CALCULATED PARAMETER			
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION(MCHC)	34.7 High	31.5 - 34.5	g/dL
METHOD : CALCULATED PARAMETER			
RED CELL DISTRIBUTION WIDTH (RDW)	12.8	11.6 - 14.0	%
METHOD : CALCULATED PARAMETER			
MENTZER INDEX	17.3		
MEAN PLATELET VOLUME (MPV)	8.4	6.8 - 10.9	fL
METHOD : CALCULATED PARAMETER			

WBC DIFFERENTIAL COUNT

NEUTROPHILS	58	40 - 80	%
METHOD : IMPEDIMENT VARIATION			
LYMPHOCYTES	32	20 - 40	%
METHOD : IMPEDIMENT VARIATION			
MONOCYTES	07	2 - 10	%
METHOD : IMPEDIMENT VARIATION			
EOSINOPHILS	03	1 - 6	%
METHOD : IMPEDIMENT VARIATION			

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Consultant Pathologist

Page 1 Of 18



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Navi Mumbai, 410210
Maharashtra, India
Tel : 9111591115, Fax :
CIN - U74899PB1995PLC045956



Patient Ref. No. 77500003510696

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BASOPHILS METHOD : IMPEDANCE VARIATION	00	0 - 2		%
ABSOLUTE NEUTROPHIL COUNT METHOD : IMPEDANCE VARIATION	5.86	2.0 - 7.0		thou/ μ L
ABSOLUTE LYMPHOCYTE COUNT METHOD : IMPEDANCE VARIATION	3.23 High	1.0 - 3.0		thou/ μ L
ABSOLUTE MONOCYTE COUNT METHOD : IMPEDANCE VARIATION	0.71	0.2 - 1.0		thou/ μ L
ABSOLUTE EOSINOPHIL COUNT METHOD : IMPEDANCE VARIATION	0.30	0.02 - 0.50		thou/ μ L
NEUTROPHIL LYMPHOCYTE RATIO (NLR) METHOD : CALCULATED	1.8			
BAND (STAB) CELLS METHOD : IMPEDANCE VARIATION	00	0 - 5		%
MORPHOLOGY				
RBC	PREDOMINANTLY NORMOCYTIC NORMOCHROMIC			
WBC	NORMAL MORPHOLOGY			
PLATELETS	ADEQUATE			

Interpretation(s)

RBC AND PLATELET INDICES-Mentzer index (MCV/RBC) is an automated cell-counter based calculated screen tool to differentiate cases of Iron deficiency anaemia(>13) from Beta thalassaemia trait

(<13) in patients with microcytic anaemia. This needs to be interpreted in line with clinical correlation and suspicion. Estimation of HbA2 remains the gold standard for diagnosing a case of beta thalassaemia trait.

WBC DIFFERENTIAL COUNT-The optimal threshold of 3.3 for NLR showed a prognostic possibility of clinical symptoms to change from mild to severe in COVID positive patients. When age = 49.5 years old and NLR = 3.3, 46.1% COVID-19 patients with mild disease might become severe. By contrast, when age < 49.5 years old and NLR < 3.3, COVID-19 patients tend to show mild disease.

(Reference to - The diagnostic and predictive role of NLR, d-NLR and PLR in COVID-19 patients ; A.-P. Yang, et al.; International Immunopharmacology 84 (2020) 106504
This ratio element is a calculated parameter and out of NABL scope.

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Page 2 Of 18



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BIOCHEMISTRY**AGILUS TOTAL 2****GLUCOSE FASTING, FLUORIDE PLASMA**

FBS (FASTING BLOOD SUGAR) 93 Normal <100 mg/dL
 Impaired fasting glucose: 100 to 125
 Diabetes mellitus: > = 126 (on more than 1 occasion)
 (ADA guidelines 2021)

METHOD : HEXOKINASE

GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD

HBA1C 5.2 Non-diabetic: < 5.7 %
 Pre-diabetics: 5.7 - 6.4
 Diabetics: > or = 6.5
 Therapeutic goals: < 7.0
 Action suggested : > 8.0
 (ADA Guideline 2021)

METHOD : HIGH PERFORMANCE LIQUID CHROMATOGRAPHY (HPLC)

ESTIMATED AVERAGE GLUCOSE(EAG) 102.5 < 116.0 mg/dL

METHOD : CALCULATED PARAMETER

LIVER FUNCTION PROFILE, SERUM

BILIRUBIN, TOTAL 0.56 < 1.1 mg/dL

METHOD : COLORIMETRIC DIAZO METHOD

BILIRUBIN, DIRECT 0.23 < or = 0.30 mg/dL

METHOD : DIAZO METHOD

BILIRUBIN, INDIRECT 0.33 0.00 - 0.60 mg/dL

TOTAL PROTEIN 7.3 6.0 - 8.0 g/dL

METHOD : BIURATE

ALBUMIN 4.3 g/dL

METHOD : BROMOCRESOL GREEN

GLOBULIN 3.0 2.0 - 3.5 g/dL

ALBUMIN/GLOBULIN RATIO 1.4 1.00 - 2.00 RATIO

ASPARTATE AMINOTRANSFERASE(AST/SGOT) 22 < 40 U/L

METHOD : UV WITH PYRIDOXAL-5 PHOSPHATE

ALANINE AMINOTRANSFERASE (ALT/SGPT) 17 < 41 U/L

METHOD : UV WITH PYRIDOXAL-5 PHOSPHATE

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Page 3 Of 18



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ALKALINE PHOSPHATASE	80	40 - 129		U/L
GAMMA GLUTAMYL TRANSFERASE (GGT)	12	8 - 61		U/L
METHOD : G-GLUTAMYL-CARBOXY-NITROANILIDE-IFCC				
LACTATE DEHYDROGENASE	149	135 - 225		U/L
METHOD : LACTATE -PYRUVATE				
BLOOD UREA NITROGEN (BUN), SERUM				
BLOOD UREA NITROGEN	10	6 - 20		mg/dL
METHOD : UREASE - UV				
CREATININE, SERUM				
CREATININE	1.16	0.70 - 1.20		mg/dL
METHOD : JAFEE METHOD				
BUN/CREAT RATIO				
BUN/CREAT RATIO	8.62	5.00 - 15.00		
URIC ACID, SERUM				
URIC ACID	7.3 High	3.4 - 7.0		mg/dL
METHOD : URICASE, COLORIMETRIC				
TOTAL PROTEIN, SERUM				
TOTAL PROTEIN	7.3	6 - 8		g/dL
METHOD : BIURATE				
ALBUMIN, SERUM				
ALBUMIN	4.3			g/dL
METHOD : BROMOCRESOL GREEN				
GLOBULIN				
GLOBULIN	3.0	2.0 - 3.5		g/dL
ELECTROLYTES (NA/K/CL), SERUM				
SODIUM, SERUM	140	136 - 145		mmol/L
METHOD : ISE INDIRECT				
POTASSIUM, SERUM	3.93	3.5 - 5.1		mmol/L
METHOD : ISE INDIRECT				
CHLORIDE, SERUM	104	98 - 107		mmol/L
METHOD : ISE INDIRECT				

Page 4 Of 18

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Interpretation(s)

Sodium	Potassium	Chloride
Decreased in: CCF, cirrhosis, vomiting, diarrhea, excessive sweating, salt-losing nephropathy, adrenal insufficiency, nephrotic syndrome, water intoxication, SIADH. Drugs: thiazides, diuretics, ACE inhibitors, chlorpropamide, carbamazepine, anti-depressants (SSRI), antipsychotics.	Decreased in: Low potassium intake, prolonged vomiting or diarrhea, RTA types I and II, hyperaldosteronism, Cushing's syndrome, osmotic diuresis (e.g., hyperglycemia), alkalosis, familial periodic paralysis, trauma (transient). Drugs: Adrenergic agents, diuretics.	Decreased in: Vomiting, diarrhea, renal failure combined with salt deprivation, over-treatment with diuretics, chronic respiratory acidosis, diabetic ketoacidosis, excessive sweating, SIADH, salt-losing nephropathy, porphyria, expansion of extracellular fluid volume, adrenal insufficiency, hyperaldosteronism, metabolic alkalosis. Drugs: chronic laxative, corticosteroids, diuretics.
Increased in: Dehydration (excessive sweating, severe vomiting or diarrhea), diabetes mellitus, diabetes insipidus, hyperaldosteronism, inadequate water intake. Drugs: steroids, licorice, oral contraceptives.	Increased in: Massive hemolysis, severe tissue damage, rhabdomyolysis, acidosis, dehydration, renal failure, Addison's disease, RTA type IV, hypokalemic familial periodic paralysis. Drugs: potassium salts, potassium-sparing diuretics, NSAIDs, beta-blockers, ACE inhibitors, high-dose trimethoprim-sulfamethoxazole.	Increased in: Renal failure, nephrotic syndrome, RTA, dehydration, over-treatment with saline, hyperparathyroidism, diabetes insipidus, metabolic acidosis from diarrhea (Loss of HCO ₃ ⁻), respiratory alkalosis, hyperadrenocorticism. Drugs: acetazolamide, androgens, hydrochlorothiazide, salicylates.
Interferences: Severe lipemia or hyperproteinemia; if sodium analysis involves a dilution step can cause spurious results. The serum sodium falls about 1.6 mEq/L for each 100 mg/dL increase in blood glucose.	Interferences: Hemolysis of sample, delayed separation of serum, prolonged fist clenching during blood drawing, and prolonged tourniquet placement. Very high WBC/PLT counts may cause spurious. Plasma potassium levels are normal.	Interferences: Test is helpful in assessing normal and increased anion gap metabolic acidosis and in distinguishing hypercalcemia due to hyperparathyroidism (high serum chloride) from that due to malignancy (Normal serum chloride)

MAGNESIUM, SERUM

MAGNESIUM, SERUM 2.0 1.7 - 2.2 mg/dL

METHOD : XYLIDYL BLUE

CALCIUM, SERUM

CALCIUM 9.3 8.6 - 10 mg/dL

METHOD : NM-BAPTA

PHOSPHORUS, SERUM

PHOSPHORUS 3.1 2.7 - 4.9 mg/dL

METHOD : UV PHOSPHO MOLYBDATE

MICROALBUMIN, URINE

SPOT URINE MICROALBUMIN 19.7 < 20 mg/L

METHOD : IMMUNOTURBIDIMETRIC ASSAY

CREATININE, URINE 53.4 Undefined mg/dL

METHOD : JAFFEE METHOD

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Page 5 Of 18



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MICROALBUMIN/ CREATININE RATIO	36.89 High	Normal : < 30 Microalbuminuria : 30 - 299 Clinical Albuminuria : > or = 300	mg/g creat
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Comments

NOTE: ABOVE REPORTED VALUES OF URINE MICROALBUMIN AND CREATININE ARE OBTAINED FROM SPOT URINE SPECIMEN RECEIVED.

Interpretation(s)**GLUCOSE FASTING,FLUORIDE PLASMA-TEST DESCRIPTION**

Normally, the glucose concentration in extracellular fluid is closely regulated so that a source of energy is readily available to tissues and so that no glucose is excreted in the urine.

Increased in: Diabetes mellitus, Cushing's syndrome (10 – 15%), chronic pancreatitis (30%). Drugs:corticosteroids,phenytoin, estrogen, thiazides.**Decreased in :**Pancreatic islet cell disease with increased insulin,insulinoma,adrenocortical insufficiency,hypopituitarism,diffuse liver disease, malignancy(adrenocortical,stomach,fibrosarcoma),infant of a diabetic mother,enzyme deficiency diseases(e.g.galactosemia),Drugs-insulin,ethanol,propranolol;sulfonlyureas,tolbutamide, and other oral hypoglycemic agents.**NOTE:** While random serum glucose levels correlate with home glucose monitoring results (weekly mean capillary glucose values), there is wide fluctuation within individuals.Thus, glycosylated hemoglobin(HbA1c) levels are favored to monitor glycemic control.

High fasting glucose level in comparison to post prandial glucose level may be seen due to effect of Oral Hypoglycaemics & Insulin treatment,Renal Glycosuria,Glycaemic index & response to food consumed,Alimentary Hypoglycemia,Increased insulin response & sensitivity etc.

GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD-Used For:

1. Evaluating the long-term control of blood glucose concentrations in diabetic patients.
 2. Diagnosing diabetes.
 3. Identifying patients at increased risk for diabetes (prediabetes).
- The ADA recommends measurement of HbA1c (typically 3-4 times per year for type 1 and poorly controlled type 2 diabetic patients, and 2 times per year for well-controlled type 2 diabetic patients) to determine whether a patient's metabolic control has remained continuously within the target range.
1. eAG (Estimated average glucose) converts percentage HbA1c to mg/dL, to compare blood glucose levels.
 2. eAG gives an evaluation of blood glucose levels for the last couple of months.
 3. eAG is calculated as eAG (mg/dL) = 28.7 * HbA1c - 46.7

HbA1c Estimation can get affected due to :

1. Shortened Erythrocyte survival : Any condition that shortens erythrocyte survival or decreases mean erythrocyte age (e.g. recovery from acute blood loss,hemolytic anemia) will falsely lower HbA1c test results.Fructosamine is recommended in these patients which indicates diabetes control over 15 days.

2.Vitamin C & E are reported to falsely lower test results.(possibly by inhibiting glycation of hemoglobin.

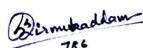
3. Iron deficiency anemia is reported to increase test results. Hypertriglyceridemia,uremia, hyperbilirubinemia, chronic alcoholism,chronic ingestion of salicylates & opiates addiction are reported to interfere with some assay methods,falsely increasing results.

4. Interference of hemoglobinopathies in HbA1c estimation is seen in

a) Homozygous hemoglobinopathy. Fructosamine is recommended for testing of HbA1c.

b) Heterozygous state detected (D10 is corrected for HbS & HbC trait.)

c) HbF > 25% on alternate platform (Boronate affinity chromatography) is recommended for testing of HbA1c.Abnormal Hemoglobin electrophoresis (HPLC method) is recommended for detecting a hemoglobinopathy

LIVER FUNCTION PROFILE, SERUM-**Bilirubin** is a yellowish pigment found in bile and is a breakdown product of normal heme catabolism. Bilirubin is excreted in bile and urine, and elevated levels may give yellow discoloration in jaundice.**Elevated levels** results from increased bilirubin production (eg, hemolysis and ineffective erythropoiesis), decreased bilirubin excretion (eg, obstruction and hepatitis), and abnormal bilirubin metabolism (eg, hereditary and neonatal jaundice). Conjugated (direct) bilirubin is elevated more than unconjugated (indirect) bilirubin in Viral hepatitis, Drug reactions, Alcoholic liver disease Conjugated (direct) bilirubin is also elevated more than unconjugated (indirect) bilirubin when there is some kind of blockage of the bile ducts like in Gallstones getting into the bile ducts, tumors & Scarring of the bile ducts. Increased unconjugated (indirect) bilirubin may be a result of Hemolytic or pernicious anemia, Transfusion reaction & a common metabolic condition termed Gilbert syndrome, due to low levels of the enzyme that attaches sugar molecules to bilirubin.**AST** is an enzyme found in various parts of the body. AST is found in the liver, heart, skeletal muscle, kidneys, brain, and red blood cells, and it is commonly measured

Page 6 Of 18

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clinically as a marker for liver health. AST levels increase during chronic viral hepatitis, blockage of the bile duct, cirrhosis of the liver, liver cancer, kidney failure, hemolytic anemia, pancreatitis, hemochromatosis. AST levels may also increase after a heart attack or strenuous activity. ALT test measures the amount of this enzyme in the blood. ALT is found mainly in the liver, but also in smaller amounts in the kidneys, heart, muscles, and pancreas. It is commonly measured as a part of a diagnostic evaluation of hepatocellular injury, to determine liver health. AST levels increase during acute hepatitis, sometimes due to a viral infection, ischemia to the liver, chronic hepatitis, obstruction of bile ducts, cirrhosis.

ALP is a protein found in almost all body tissues. Tissues with higher amounts of ALP include the liver, bile ducts and bone. Elevated ALP levels are seen in Biliary obstruction, Osteoblastic bone tumors, osteomalacia, hepatitis, Hyperparathyroidism, Leukemia, Lymphoma, Pagets disease, Rickets, Sarcoidosis etc. Lower-than-normal ALP levels seen in Hypophosphatasia, Malnutrition, Protein deficiency, Wilsons disease.

GGT is an enzyme found in cell membranes of many tissues mainly in the liver, kidney and pancreas. It is also found in other tissues including intestine, spleen, heart, brain and seminal vesicles. The highest concentration is in the kidney, but the liver is considered the source of normal enzyme activity. Serum GGT has been widely used as an index of liver dysfunction. Elevated serum GGT activity can be found in diseases of the liver, biliary system and pancreas. Conditions that increase serum GGT are obstructive liver disease, high alcohol consumption and use of enzyme-inducing drugs etc.

Total Protein also known as total protein, is a biochemical test for measuring the total amount of protein in serum. Protein in the plasma is made up of albumin and globulin. Higher-than-normal levels may be due to: Chronic inflammation or infection, including HIV and hepatitis B or C, Multiple myeloma, Waldenstroms disease. Lower-than-normal levels may be due to: Agammaglobulinemia, Bleeding (hemorrhage), Burns, Glomerulonephritis, Liver disease, Malabsorption, Malnutrition, Nephrotic syndrome, Protein-losing enteropathy etc.

Albumin is the most abundant protein in human blood plasma. It is produced in the liver. Albumin constitutes about half of the blood serum protein. Low blood albumin levels (hypoalbuminemia) can be caused by: Liver disease like cirrhosis of the liver, nephrotic syndrome, protein-losing enteropathy, Burns, hemodilution, increased vascular permeability or decreased lymphatic clearance, malnutrition and wasting etc.

BLOOD UREA NITROGEN (BUN), SERUM-**Causes of Increased levels** include Pre renal (High protein diet, Increased protein catabolism, GI hemorrhage, Cortisol, Dehydration, CHT Renal), Renal Failure, Post Renal (Malignancy, Nephrolithiasis, Prostatism)

Causes of decreased level include Liver disease, SIADH.

CREATININE, SERUM-Higher than normal level may be due to:

- Blockage in the urinary tract, Kidney problems, such as kidney damage or failure, infection, or reduced blood flow, Loss of body fluid (dehydration), Muscle problems, such as breakdown of muscle fibers, Problems during pregnancy, such as seizures (eclampsia), or high blood pressure caused by pregnancy (preeclampsia)

Lower than normal level may be due to: Myasthenia Gravis, Muscuophy

URIC ACID, SERUM-Causes of Increased levels:-Dietary(High Protein Intake,Prolonged Fasting,Rapid weight loss),Gout,Lesch nyhan syndrome,Type 2 DM, Metabolic syndrome **Causes of decreased levels-**Low Zinc intake,OCP, Multiple Sclerosis

TOTAL PROTEIN, SERUM-is a biochemical test for measuring the total amount of protein in serum. Protein in the plasma is made up of albumin and globulin.

Higher-than-normal levels may be due to: Chronic inflammation or infection, including HIV and hepatitis B or C, Multiple myeloma, Waldenstroms disease.

Lower-than-normal levels may be due to: Agammaglobulinemia, Bleeding (hemorrhage), Burns, Glomerulonephritis, Liver disease, Malabsorption, Malnutrition, Nephrotic syndrome, Protein-losing enteropathy etc.

ALBUMIN, SERUM-

Human serum albumin is the most abundant protein in human blood plasma. It is produced in the liver. Albumin constitutes about half of the blood serum protein. **Low blood albumin levels (hypoalbuminemia) can be caused by:** Liver disease like cirrhosis of the liver, nephrotic syndrome, protein-losing enteropathy, Burns, hemodilution, increased vascular permeability or decreased lymphatic clearance, malnutrition and wasting etc.

MAGNESIUM, SERUM-Description- Magnesium is primarily an intracellular ion associated with GI absorption and renal excretion. Second most abundant ion in bone. It functions as co-factor in numerous enzymes e.g. ATPase . 65-70% of Mg is in ionized state and nearly 35% is protein bound.

Interpretation-

Increased in- Dehydration, Tissue trauma, Renal failure, Hypothyroidism, excessive intake of antacid.

Decrease in- Chronic diarrhea, Enteric fistula, Starvation, Chronic alcoholism, Total parenteral Nutrition, Diuretics.

Note- Hypomagnesemia is associated with weakness, tetany, disorientation and somnolence

Limitation-

- Hemolysis yields elevated levels of Mg being an intracellular ion.
- Serum magnesium levels may remain normal even when total body stores of magnesium are depleted upto 20%

CALCIUM, SERUM-Common causes of decreased value of calcium (hypocalcemia) are chronic renal failure, hypomagnesemia and hypoalbuminemia.

Hypercalcemia (increased value of calcium) can be caused by increased intestinal absorption (vitamin D intoxication), increased skeletal reabsorption (immobilization), or a combination of mechanisms (primary hyperparathyroidism). Primary hyperparathyroidism and malignancy accounts for 90-95% of all cases of hypercalcemia. Values of total calcium is affected by serum proteins, particularly albumin thus, latter's value should be taken into account when interpreting serum calcium levels. The following regression equation may be helpful.

Corrected total calcium (mg/dl)= total calcium (mg/dl) + 0.8 (4- albumin [g/dl])*

because regression equations vary among group of patients in different physiological and pathological conditions, mathematical corrections are only approximations. The possible mathematical corrections should be replaced by direct determination of free calcium by ISE (available with srl) a common and important source of preanalytical error in the measurement of calcium is prolonged tourniquet application during sampling. Thus, this along with fist clenching should be avoided before phlebotomy.

MICROALBUMIN, URINE-Microalbuminuria is defined as an increase in urinary excretion of albumin above the reference interval for healthy nondiabetic subjects but at a concentration that is generally detectable by crude clinical tests such as dipsticks designed to measure total protein, the diagnosis of microalbuminuria requires demonstration of increased albumin secretion in at least two out of three urine samples collected in the absence of infection or an acute metabolic crisis. It is now considered a clinically important indicator of deteriorating renal function in diabetic subjects in diabetic.. patients Regular screening of urinary albumin secretion is valuable in monitoring both type 1 and type 2 diabetes.

Screening should commence 5 years after diagnosis in patients with type 1 diabetes and at diagnosis in patients with type 2 diabetes without proteinuria.

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Page 7 Of 18



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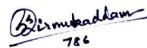
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9870331316

ACCESSION NO : 0040WF002390	AGE/SEX : 18 Years Male
PATIENT ID : MATHM16030540	DRAWN : 10/06/2023 09:13:02
CLIENT PATIENT ID:	RECEIVED : 10/06/2023 09:16:04
ABHA NO :	REPORTED : 10/06/2023 13:27:14

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Screening is not indicated in patients with established proteinuria. All the patients with diabetes mellitus should be screened on annual basis up to the age of 75 years. It is important to consider causes of increased albumin excretion, specially in cases of type 1 diabetes present for less than 5 years. These can include nondiabetic renal disease, menstrual contamination, vaginal discharge, uncontrolled hypertension , urinary tract infection, heart failure, and strenuous exercise.


Dr. Swapnil Sirmukaddam
Consultant Pathologist

Page 8 Of 18

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 Maharashtra, India
 Tel : 9111591115, Fax :
 CIN - U74899PB1995PLC045956

**Patient Ref. No. 77500003510696**

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REF. DOCTOR : DR. KAMINI S MEHTA

KHARGHAR
9870331316
 ACCESSION NO : 0040WF002390
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BIOCHEMISTRY - LIPID**AGILUS TOTAL 2****LIPID PROFILE, SERUM**

CHOLESTEROL, TOTAL	121	Desirable cholesterol level mg/dL < 200 Borderline high cholesterol 200 - 239 High cholesterol > / = 240
METHOD : CHOLESTEROL OXIDASE, ESTERASE, PEROXIDASE		
TRIGLYCERIDES	52	Normal: < 150 mg/dL Borderline high: 150 - 199 High: 200 - 499 Very High: >/= 500
METHOD : GLUCOSE OXIDASE / PEROXIDASE (GOD - POD) METHOD		
HDL CHOLESTEROL	38 Low	Low HDL cholesterol mg/dL < 40 High HDL cholesterol > / = 60
METHOD : CHOLESTEROL OXIDASE, ESTERASE, PEROXIDASE		
LDL CHOLESTEROL, DIRECT	73	Adult Optimal : < 100 mg/dL Near optimal : 100 - 129 Borderline high : 130 - 159 High : 160 - 189 Very high : > or = 190
METHOD : CHOLESTEROL OXIDASE, ESTERASE, PEROXIDASE		
NON HDL CHOLESTEROL	83	Desirable: Less than 130 mg/dL Above Desirable: 130-159 Borderline High: 160-189 High: 190 -219 Very High: >or = 220
VERY LOW DENSITY LIPOPROTEIN	10.4	Desirable value: 10 - 35 mg/dL
CHOL/HDL RATIO	3.2 Low	3.3 - 4.4 Low Risk 4.5-7.0 Average Risk 7.1 - 11.0 Moderate Risk > 11.0 High Risk

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 Consultant Pathologist

Page 9 Of 18



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--------------------	-------	---------	-------------------------------	-------

LDL/HDL RATIO	1.9	0.5 - 3.0 Desirable/ Low Risk 3.1-6.0 Borderline /Moderate Risk > 6.0 High Risk
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Interpretation(s)

Serum lipid profile is measured for cardiovascular risk prediction. Lipid Association of India recommends LDL-C as primary target and Non HDL-C as co-primary treatment target.

Risk Stratification for ASCVD (Atherosclerotic cardiovascular disease) by Lipid Association of India

Risk Category	
Extreme risk group	A. CAD with > 1 feature of high risk group
	B. CAD with > 1 feature of Very high risk group or recurrent ACS (within 1 year) despite LDL-C < or = 50 mg/dl or polyvascular disease
Very High Risk	1. Established ASCVD 2. Diabetes with 2 major risk factors or evidence of end organ damage 3. Familial Homozygous Hypercholesterolemia
High Risk	1. Three major ASCVD risk factors. 2. Diabetes with 1 major risk factor or no evidence of end organ damage. 3. CKD stage 3B or 4. 4. LDL >190 mg/dl 5. Extreme of a single risk factor. 6. Coronary Artery Calcium - CAC >300 AU. 7. Lipoprotein a >/= 50mg/dl 8. Non stenotic carotid plaque
Moderate Risk	2 major ASCVD risk factors
Low Risk	0-1 major ASCVD risk factors

Major ASCVD (Atherosclerotic cardiovascular disease) Risk Factors

1. Age > or = 45 years in males and > or = 55 years in females	3. Current Cigarette smoking or tobacco use
2. Family history of premature ASCVD	4. High blood pressure
5. Low HDL	

Newer treatment goals and statin initiation thresholds based on the risk categories proposed by LAI in 2020.

Risk Group	Treatment Goals		Consider Drug Therapy	
	LDL-C (mg/dl)	Non-HDL (mg/dl)	LDL-C (mg/dl)	Non-HDL (mg/dl)
Extreme Risk Group Category A	<50 (Optional goal < OR = 30)	< 80 (Optional goal <OR = 60)	>OR = 50	>OR = 80
Extreme Risk Group Category B	<OR = 30	<OR = 60	> 30	>60
Very High Risk	<50	<80	>OR= 50	>OR= 80
High Risk	<70	<100	>OR= 70	>OR= 100
Moderate Risk	<100	<130	>OR= 100	>OR= 130
Low Risk	<100	<130	>OR= 130*	>OR= 160

*After an adequate non-pharmacological intervention for at least 3 months.

References: Management of Dyslipidaemia for the Prevention of Stroke: Clinical Practice Recommendations from the Lipid Association of India. Current Vascular Pharmacology, 2022, 20, 134-155.

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Page 10 Of 18



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SPECIALISED CHEMISTRY - ANEMIA**AGILUS TOTAL 2****TOTAL IRON BINDING CAPACITY, SERUM**

IRON	73	59 - 158	µg/dL
METHOD : FEROZINE - NO DEPROTEINIZATION			
TOTAL IRON BINDING CAPACITY	274	228 - 428	µg/dL
METHOD : CALCULATED PARAMETER			
% SATURATION	27	20 - 50	%

Interpretation(s)

TOTAL IRON BINDING CAPACITY, SERUM-Total iron binding capacity (TIBC) measures the blood's capacity to bind iron with transferrin and thus is an indirect way of assessing transferrin level.

Taken together with serum iron and percent transferrin saturation this test is performed when there is a concern about anemia, iron deficiency or iron deficiency anemia. However, because the liver produces transferrin, alterations in liver function (such as cirrhosis, hepatitis, or liver failure) must be considered when performing this test.

Increased in:

- iron deficiency
- acute and chronic blood loss
- acute liver damage
- progesterone birth control pills

Decreased in:

- hemochromatosis
- cirrhosis of the liver
- thalassemia
- anemias of infection and chronic diseases
- nephrosis
- hyperthyroidism

The percent Transferrin saturation = Serum Iron/TIBC × 100

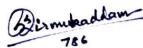
Unsaturated Binding Capacity (UIBC)=TIBC - Serum Iron.

Limitations: Estrogens and oral contraceptives increase TIBC and Asparaginase, chloramphenicol, corticotropin, cortisone and testosterone decrease the TIBC level.

Reference:

1. Tietz Textbook of Clinical Chemistry and Molecular Diagnostics, edited by Carl A Burtis, Edward R. Ashwood, David E Bruns, 4th Edition, Elsevier publication, 2006, 563, 1314-1315.

2. Wallach's Interpretation of Diagnostic tests, 9th Edition, Ed Mary A Williamson and L Michael Snyder. Pub Lippincott Williams and Wilkins, 2011, 234-235.



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Page 11 Of 18



View Details

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--------------------	-------	---------	-------------------------------	-------

CLINICAL PATH - URINALYSIS

AGILUS TOTAL 2**PHYSICAL EXAMINATION, URINE**

COLOR PALE YELLOW
METHOD : PHYSICAL

APPEARANCE Clear
METHOD : PHYSICAL

CHEMICAL EXAMINATION, URINE

PH 6.0 4.7 - 7.5
METHOD : DOUBLE INDICATOR PRINCIPLE

SPECIFIC GRAVITY <=1.005 1.003 - 1.035
METHOD : PKA CHANGE IN RELATION TO IONIC CONCENTRATION

PROTEIN NOT DETECTED NEGATIVE
METHOD : 0.3% TETRABROMPHENOL BLUE INDICATOR

GLUCOSE NOT DETECTED NEGATIVE
METHOD : GOD-POD METHOD

KETONES NOT DETECTED NOT DETECTED
METHOD : REACTION OF ACETOACETIC ACID WITH NITROPRUSSIDE

BLOOD NOT DETECTED NOT DETECTED
METHOD : REACTION OF DISOPROPYLBENZENE DIHYDROPEROXIDE AND 3,3',5,5' TETRAMETHYL BENZIDINE

BILIRUBIN NOT DETECTED NOT DETECTED
METHOD : COUPLING OF BILIRUBIN WITH DIAZOTIZED DICHLOROANALINE

UROBILINOGEN NORMAL NORMAL
METHOD : EHRlich REACTION REFLECTANCE

NITRITE NOT DETECTED NOT DETECTED
METHOD : REACTION WITH P-ARSANILIC ACID & COUPLING WITH TETRAHYDROBENZOQUINOLINOL

LEUKOCYTE ESTERASE NOT DETECTED NOT DETECTED

MICROSCOPIC EXAMINATION, URINE

RED BLOOD CELLS NOT DETECTED NOT DETECTED /HPF
METHOD : MICROSCOPIC EXAMINATION

PUS CELL (WBC'S) 2-3 0-5 /HPF
METHOD : MICROSCOPIC EXAMINATION

EPITHELIAL CELLS 1-2 0-5 /HPF
METHOD : MICROSCOPIC EXAMINATION

CASTS NOT DETECTED

Dr. Swapnil Sirmukaddam
Consultant Pathologist

Page 12 Of 18



View Details

View Report

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--------------------	--------------	---------	-------------------------------	-------

METHOD : MICROSCOPIC EXAMINATION

CRYSTALS

NOT DETECTED

METHOD : MICROSCOPIC EXAMINATION

BACTERIA

NOT DETECTED NOT DETECTED

METHOD : MICROSCOPIC EXAMINATION

YEAST

NOT DETECTED NOT DETECTED

REMARKS

URINE ANALYSIS : MICROSCOPIC EXAMINATION IS CARRIED OUT ON CENTRIFUGED URINARY SEDIMENT.

METHOD : MICROSCOPIC EXAMINATION


Dr. Swapnil Sirmukaddam
Consultant Pathologist

Page 13 Of 18

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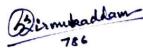
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--------------------	-------	---------	-------------------------------	-------

Interpretation(s)

The following table describes the probable conditions, in which the analytes are present in urine

Presence of	Conditions
Proteins	Inflammation or immune illnesses
Pus (White Blood Cells)	Urinary tract infection, urinary tract or kidney stone, tumors or any kind of kidney impairment
Glucose	Diabetes or kidney disease
Ketones	Diabetic ketoacidosis (DKA), starvation or thirst
Urobilinogen	Liver disease such as hepatitis or cirrhosis
Blood	Renal or genital disorders/trauma
Bilirubin	Liver disease
Erythrocytes	Urological diseases (e.g. kidney and bladder cancer, urolithiasis), urinary tract infection and glomerular diseases
Leukocytes	Urinary tract infection, glomerulonephritis, interstitial nephritis either acute or chronic, polycystic kidney disease, urolithiasis, contamination by genital secretions
Epithelial cells	Urolithiasis, bladder carcinoma or hydronephrosis, ureteric stents or bladder catheters for prolonged periods of time
Granular Casts	Low intratubular pH, high urine osmolality and sodium concentration, interaction with Bence-Jones protein
Hyaline casts	Physical stress, fever, dehydration, acute congestive heart failure, renal diseases
Calcium oxalate	Metabolic stone disease, primary or secondary hyperoxaluria, intravenous infusion of large doses of vitamin C, the use of vasodilator naftidrofuryl oxalate or the gastrointestinal lipase inhibitor orlistat, ingestion of ethylene glycol or of star fruit (Averrhoa carambola) or its juice
Uric acid	arthritis
Bacteria	Urinary infection when present in significant numbers & with pus cells.
Trichomonas vaginalis	Vaginitis, cervicitis or salpingitis


Dr. Swapnil Sirmukaddam
 Consultant Pathologist

Page 14 Of 18



View Details

View Report

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--------------------	-------	---------	-------------------------------	-------

SPECIALISED CHEMISTRY - HORMONE**AGILUS TOTAL 2****FREE THYROXINE (FT4), SERUM**

FREE THYROXINE (FT4) 1.21 0.98 – 1.63 ng/dL
METHOD : ELECTROCHEMILUMINESCENCE, COMPETITIVE IMMUNOASSAY

FREE TRIIODOTHYRONINE (FT3), SERUM

FREE TRIIODOTHYRONINE (FT3) 3.06 2.56 – 5.01 pg/mL
METHOD : ELECTROCHEMILUMINESCENCE, COMPETITIVE IMMUNOASSAY

TSH 3RD GENERATION ULTRASENSITIVE, SERUM

TSH (ULTRASENSITIVE) 1.100 0.510 - 4.300 µIU/mL
METHOD : ELECTROCHEMILUMINESCENCE, COMPETITIVE IMMUNOASSAY

Interpretation(s)

FREE THYROXINE (FT4), SERUM-Thyroxine(T4)circulates in the blood as an equilibrium mixture of free and serum protein bound hormone. Less than 0.03% is present in the circulation as unbound, free T4. This small percentage of the total T4 represents the physiologically available hormone which is biologically active.

Free T3 and Free T4 values therefore, provide the best indication of thyroid dysfunction, instead of Total T3 or Total T4, since these are not affected by changes in the serum binding proteins. Free T3 is typically elevated to a greater degree than free thyroxine (T4) in Graves' disease.

Occasionally, free T3 alone is elevated (T3 thyrotoxicosis) in about 5% of the hyperthyroid population. In contrast, levels of free T4 are elevated to a greater degree than free T3 in toxic multinodular goiter and excessive T4 therapy. Serum free T3 is useful in distinguishing these forms of hyperthyroidism.

Free T3 may also be important in monitoring patients on anti-thyroid therapy where treatment is focused on reducing the T3 production and the T4 conversion to T3. Serum free T3 may also be useful in assessing the severity of the thyrotoxic state.

FREE TRIIODOTHYRONINE (FT3), SERUM-FREE T3 :T3 is bound to thyroxine binding globulin(TBG),prealbumin, and albumin. Only 0.2-0.4% of the total T3 is present in solution as unbound or free T3.This free fraction represents the physiologically active thyroid hormone.

Free T3 and Free T4 values therefore, provide the best indication of thyroid dysfunction, instead of Total T3 or Total T4, since these are not affected by changes in the serum binding proteins. Free T3 is typically elevated to a greater degree than free thyroxine (T4) in Graves' disease.

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TSH 3RD GENERATION ULTRASENSITIVE, SERUM-TSH stands for thyroid stimulating hormone.This hormone stimulates the Thyroid gland to make thyroid hormones that regulate the way our body uses energy. These also play an important role in regulating weight, temperature, muscle strength, and even your mood. TSH is made in a gland in the brain called the pituitary. When thyroid levels in our body are low, the pituitary gland makes more TSH. When thyroid levels are high, the pituitary gland makes less TSH. TSH levels that are too high or too low can indicate that thyroid is not working correctly.

There is a circadian rhythm of TSH secretion, with peak values at the onset of sleep and nadir concentrations during the afternoon hours. Peak and nadir concentrations differ by approximately +/- 50%. The effect on circulating T4 and T3 concentrations is not significant because of the large size of the extrathyroidal T4 pool.

In healthy subjects there is no significant impact of body weight, physical training, body habitus, posture, immobilization, mild to moderate exercise, or ambulatory status on thyroid function, and no significant geographic environmental variation. Nutrition also has a minimal impact except for variation in iodine intake. Subthreshold concentrations of iodine intake are associated with increased TSH secretion, goiter, increased thyroid iodine uptake, decreased T4 production, an increased T3/T4 secretion ratio, and an increased ratio of circulating T3/T4 concentrations. Excessive iodine intake can block thyroid hormone biosynthesis by inhibiting the enzymes involved in the biosynthetic process, resulting in reduced T4 secretion, increased TSH concentrations, goiter, and hypothyroidism if the iodine excess is chronic.

High TSH levels can mean your thyroid is not making enough thyroid hormones, a condition called hypothyroidism.**Low TSH levels** can mean your thyroid is making too much of the hormones, a condition called hyperthyroidism. A TSH test does not explain why TSH levels are too high or too low.

In cases of Subclinical hypothyroidism, a single test can be misleading, so a second test is usually done 2 or 3 months later. In both tests, the blood is taken at the same time of day because TSH levels can fluctuate over the course of 24 hours. Subclinical hypothyroidism is diagnosed when both TSH readings are high but the thyroid hormone thyroxine is still within the normal range.

Being severely overweight and certain medications can also increase TSH. TSH levels are likely to fluctuate more during pregnancy.

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Page 15 Of 18



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TSH values may be transiently altered because of Non thyroidal Illness like severe infections, liver disease, renal failure, heart failure, severe burns, trauma, surgery etc. TSH levels that are slightly or only moderately elevated do not necessarily need to be treated. Some people who have high TSH levels never even develop symptoms. It is also very common for TSH levels to return to normal in children and teenagers.

REF: 1. TIETZ Fundamentals of Clinical chemistry 2Guidlines of the American Thyroid association during pregnancy and Postpartum,2011.



Page 16 Of 18

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SPECIALISED CHEMISTRY - VITAMIN**AGILUS TOTAL 2****25 - HYDROXYVITAMIN D(VITAMIN D TOTAL), SERUM**

25 - HYDROXYVITAMIN D **11.65 Low** SEVERE DEFICIENCY < 10 ng/mL
 INSUFFICIENCY 10 - 30
 SUFFICIENCY > 30
 TOXICITY > 100

METHOD : ELECTROCHEMILUMINESCENCE, COMPETITIVE IMMUNOASSAY

VITAMIN B12(CYANOCOBALAMINE), SERUM

VITAMIN B12 343.9 197 - 771 pg/mL

METHOD : ELECTROCHEMILUMINESCENCE, COMPETITIVE IMMUNOASSAY

Interpretation(s)**25 - HYDROXYVITAMIN D(VITAMIN D TOTAL), SERUM-Test description**

Vitamin D has anti-inflammatory and immune-modulating properties and it works towards the bones, teeth, intestines, immune system, pancreas, muscles and brain. It helps to maintain normal calcium and phosphate levels. Vitamin D is a fat-soluble vitamin. Also called as "Sunshine Vitamin". Two main forms as Cholecalciferol (vitamin D3) which is synthesized in skin from 7-dehydrocholesterol in response to sunlight (Type B UV) exposure & Ergocalciferol (vitamin D2) present mainly in dietary sources.

Vit D_{2/3}(OH)D deficiency is seen due to poor or inadequate sunlight exposure, Nutritional or dietary deficiency or fat malabsorption, Severe Hepatocellular disease, Secondary hyperparathyroidism, Hypocalcemia tetany which can cause involuntary contraction of muscles, leading to cramps and spasms, Rickets in children, Osteomalacia in adults- due to vitamin D deficiency mainly, Older adults- osteoporosis. (Increased risk of bone fractures) due to long-term effect of calcium and/or vitamin D deficiency, Other conditions that are precipitated by Vit D deficiency included increased cardiovascular risk, low immunity & chronic renal failure.

Elevated levels may be seen in patients taking supplements(hence recommended to repeat after 3 months for estimation of accurate levels), Vitamin D intoxication, sarcoidosis and malignancies containing non regulated 1-alpha hydroxylase in the lesion.

Recommendations

1. To prevent biotin interference the patient should be atleast 8 hours fasting before submitting the sample. 2. 25(OH)D is the analyte of choice for determination of the Vitamin D status as it is the major storage & active form of Vitamin D and has longer half-life. 3. Kidney Disease Outcomes Quality Initiatives (KDOQI) and Kidney Disease Improving Global Outcomes (KDIGO) recommend activated vitamin D testing for CKD patients.

Note-Our Vitamin D assays is standardized to be in alignment with the ID-LC/MS/MS 25(OH)vitamin D Reference Method Procedure (RMP), the reference procedure for the Vitamin D Standardization Program (VDSP). The VDSP, a collaboration of the National Institutes of Health Office of Dietary Supplements, National Institute of Technology and Standards, Centers for Disease Control and Ghent University, is an initiative to standardize 25(OH)vitamin D measurement across methods.

Reference:

1. Wallach Interpretation of diagnostic test, 10th edition.

VITAMIN B12(CYANOCOBALAMINE), SERUM-Test description

1. Measures the amount of Vitamin B12/ Cyanocobalamin or Methyl cobalamin in blood.2. Done in Anemic conditions like Megaloblastic anemia, pernicious anemia, dietary folate deficiencies,3. Workup of neuropathies especially due to diabetes.4. Nerve health and it is monitored in treatment of nerve damage.5. Important vitamin for women of childbearing age and for older people.

1. Part of water-soluble B complex of vitamins. 2. It is essential in DNA synthesis, hematopoiesis & CNS integrity.3. Source for B12 is dietary foods like milk, yoghurt, eggs, meat, fortified cereals, bread. 4. Absorption depends on the HCl secreted by the stomach and occurs in intestines. 5. It is part of enterohepatic circulation, hence excreted in feces(approx. 0.1% per day)

Test interpretation

Higher than normal levels are in patients on Vitamin supplements or patients with COPD, CRF, Diabetes, Liver cell damage, Obesity, Polycythemia.

Decreased levels seen in

Inflammatory bowel disease, Pernicious anemia - genetic deficiency of intrinsic factor - necessary for Vit B12 absorption. Strict vegetarians lead to sub-clinical B12 deficiency- high among elderly patients, Malabsorption due to gastrectomy, smoking, pregnancy, multiple myeloma & hemodialysis, Alcohol & drugs like amino salicylic acid, anticonvulsants, cholestyramine, cimetidine, Hyperthyroidism (High levels of thyroid), Seen in mothers of children with (NTD) Neural tube defects- hence fortification and supplements are advised in expecting mothers

Recommendations-1. To prevent biotin interference the patient should be atleast 8 hours fasting before submitting the sample. 2. Vit B12 and Folic acid evaluated together in macrocytic anemias to avoid methyl folate trap. Carmel's composite criteria for inadequate Vit B12 status: Serum vitamin B12 < 148 pmol/L, or 148-258 pmol/L and

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Consultant Pathologist

Page 17 Of 18



View Details

View Report

PERFORMED AT :

Agilus Diagnostics Ltd (Formerly SRL Ltd)
 Bhoomi Tower, 1st Floor, Hall No.1, Plot No.28 Sector 4, Kharghar
 Navi Mumbai, 410210
 Maharashtra, India
 Tel : 9111591115, Fax :
 CIN - U74899PB1995PLC045956



Patient Ref. No. 77500003510696

PATIENT NAME : MATHEW JOSEPH

REF. DOCTOR : DR. KAMINI S MEHTA

KHARGHAR

9870331316

ACCESSION NO : 0040WF002390

PATIENT ID : MATHM16030540

CLIENT PATIENT ID:

ABHA NO :

AGE/SEX : 18 Years Male

DRAWN : 10/06/2023 09:13:02

RECEIVED : 10/06/2023 09:16:04

REPORTED : 10/06/2023 13:27:14

Test Report Status **Final**

Results

Biological Reference Interval

Units

MMA > 0.30 μ mol/L, or tHcy > 13 nmol/L (females) and >15 nmol/L (males).**Associated Test-Holo-Tc:** Marker of vitamin B12 status -specificity and sensitivity better than serum vitamin B12, hence recommended in borderline and deficient cases for confirmation.

References-O'Leary F, Samman S. Vitamin B12 in health and disease, Nutrients, 2010 Mar;2(3):299-316.

End Of Report

Please visit www.srlworld.com for related Test Information for this accession**CONDITIONS OF LABORATORY TESTING & REPORTING**

1. It is presumed that the test sample belongs to the patient named or identified in the test requisition form.
2. All tests are performed and reported as per the turnaround time stated in the AGILUS Directory of Services.
3. Result delays could occur due to unforeseen circumstances such as non-availability of kits / equipment breakdown / natural calamities / technical downtime or any other unforeseen event.
4. A requested test might not be performed if:
 - i. Specimen received is insufficient or inappropriate
 - ii. Specimen quality is unsatisfactory
 - iii. Incorrect specimen type
 - iv. Discrepancy between identification on specimen container label and test requisition form
5. AGILUS Diagnostics confirms that all tests have been performed or assayed with highest quality standards, clinical safety & technical integrity.
6. Laboratory results should not be interpreted in isolation; it must be correlated with clinical information and be interpreted by registered medical practitioners only to determine final diagnosis.
7. Test results may vary based on time of collection, physiological condition of the patient, current medication or nutritional and dietary changes. Please consult your doctor or call us for any clarification.
8. Test results cannot be used for Medico legal purposes.
9. In case of queries please call customer care (91115 91115) within 48 hours of the report.

Agilus Diagnostics LimitedFortis Hospital, Sector 62, Phase VIII,
Mohali 160062

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Page 18 Of 18



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