



PATIENT NAME : N PRABHAKAR

REF. DOCTOR : SELF

CODE/NAME & ADDRESS : C000138482

RELEX HEALTHCARE SERVICES INDIA PVT LTD
PLOT 63/A, GROUND FLOOR, RAGHAVENDRA
NILAYAM, 7TH PHASE, KPHB COLONY, HYDERABAD
HYDERABAD 500072
08047109222

ACCESSION NO : 0042WD000493

PATIENT ID : NPRAM04045342

CLIENT PATIENT ID:

ABHA NO :

AGE/SEX : 70 Years Male
DRAWN : 04/04/2023 00:00:00
RECEIVED : 04/04/2023 12:21:57
REPORTED : 04/04/2023 14:51:43

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HAEMATOLOGY - CBC

HEALTH SCREEN - 3

BLOOD COUNTS, EDTA WHOLE BLOOD

HEMOGLOBIN (HB)	12.6 Low	13.0 - 17.0	g/dL
METHOD : CYANMETHEMOGLOBIN METHOD			
RED BLOOD CELL (RBC) COUNT	4.53	4.5 - 5.5	mil/ μ L
METHOD : ELECTRICAL IMPEDANCE			
WHITE BLOOD CELL (WBC) COUNT	4.90	4.0 - 10.0	thou/ μ L
METHOD : ELECTRICAL IMPEDANCE			
PLATELET COUNT	164	150 - 410	thou/ μ L
METHOD : ELECTRICAL IMPEDANCE			

RBC AND PLATELET INDICES

HEMATOCRIT (PCV)	39.3 Low	40 - 50	%
METHOD : CALCULATED PARAMETER			
MEAN CORPUSCULAR VOLUME (MCV)	87.0	83 - 101	fL
METHOD : CALCULATED PARAMETER			
MEAN CORPUSCULAR HEMOGLOBIN (MCH)	27.8	27.0 - 32.0	pg
METHOD : CALCULATED PARAMETER			
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC)	32.0	31.5 - 34.5	g/dL
METHOD : CALCULATED PARAMETER			
RED CELL DISTRIBUTION WIDTH (RDW)	13.7	11.6 - 14.0	%
METHOD : CALCULATED PARAMETER			
MENTZER INDEX	19.2		
MEAN PLATELET VOLUME (MPV)	9.0	6.8 - 10.9	fL
METHOD : CALCULATED PARAMETER			

WBC DIFFERENTIAL COUNT

NEUTROPHILS	50	40 - 80	%
METHOD : ACV TECHNOLOGY			
LYMPHOCYTES	42 High	20 - 40	%
METHOD : ACV TECHNOLOGY			
MONOCYTES	5	2 - 10	%
METHOD : ACV TECHNOLOGY			
EOSINOPHILS	3	1 - 6	%
METHOD : ACV TECHNOLOGY			

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BASOPHILS

0

0 - 2

%

METHOD : ACV TECHNOLOGY

ABSOLUTE NEUTROPHIL COUNT

2.45

2.0 - 7.0

thou/ μ L

METHOD : CALCULATED PARAMETER

ABSOLUTE LYMPHOCYTE COUNT

2.06

1.0 - 3.0

thou/ μ L

METHOD : CALCULATED PARAMETER

ABSOLUTE MONOCYTE COUNT

0.25

0.2 - 1.0

thou/ μ L

METHOD : CALCULATED PARAMETER

ABSOLUTE EOSINOPHIL COUNT

0.15

0.02 - 0.50

thou/ μ L

METHOD : CALCULATED PARAMETER

ABSOLUTE BASOPHIL COUNT

0 Low

0.02 - 0.10

thou/ μ L

METHOD : CALCULATED PARAMETER

NEUTROPHIL LYMPHOCYTE RATIO (NLR)

1.2

METHOD : CALCULATED

MORPHOLOGY

RBC

NORMOCYTIC NORMOCHROMIC.

METHOD : MICROSCOPIC EXAMINATION

WBC

RELATIVE LYMPHOCYTOSIS.

METHOD : MICROSCOPIC EXAMINATION

PLATELETS

ADEQUATE ON SMEAR.

METHOD : MICROSCOPIC EXAMINATION

Interpretation(s)

BLOOD COUNTS, EDTA WHOLE BLOOD-The cell morphology is well preserved for 24hrs. However after 24-48 hrs a progressive increase in MCV and HCT is observed leading to a decrease in MCHC. A direct smear is recommended for an accurate differential count and for examination of RBC morphology.

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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BIOCHEMISTRY

HEALTH SCREEN - 3

GLUCOSE FASTING, FLUORIDE PLASMA

FBS (FASTING BLOOD SUGAR)	91	82 - 99	mg/dL
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METHOD : SPECTROPHOTOMETRY HEXOKINASE

GLYCOSYLATED HEMOGLOBIN (HBA1C), EDTA WHOLE BLOOD

HBA1C	6.5 High	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)	%
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METHOD : ION- EXCHANGE HPLC

ESTIMATED AVERAGE GLUCOSE (EAG)	139.9 High	< 116.0	mg/dL
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METHOD : ION- EXCHANGE HPLC

BLOOD UREA NITROGEN (BUN), SERUM

BLOOD UREA NITROGEN	9	8 - 23	mg/dL
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METHOD : SPECTROPHOTOMETRY, UREASE UV

CREATININE, SERUM

CREATININE	1.45 High	0.80 - 1.30	mg/dL
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METHOD : SPECTROPHOTOMETRY, ALKALINE PICRATE KINETIC JAFFE'S

URIC ACID, SERUM

URIC ACID	5.9	3.5 - 7.2	mg/dL
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METHOD : SPECTROPHOTOMETRY, URICASE

TOTAL PROTEIN, SERUM

TOTAL PROTEIN	7.3	6.4 - 8.2	g/dL
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METHOD : SPECTROPHOTOMETRY, MODIFIED BIURET

ELECTROLYTES (NA/K/CL), SERUM

SODIUM, SERUM	143	136 - 145	mmol/L
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METHOD : INTEGRATED MULTISENSOR TECHNOLOGY-INDIRECT

POTASSIUM, SERUM	4.58	3.50 - 5.10	mmol/L
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METHOD : INTEGRATED MULTISENSOR TECHNOLOGY-INDIRECT

CHLORIDE, SERUM	101	98 - 107	mmol/L
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METHOD : INTEGRATED MULTISENSOR TECHNOLOGY-INDIRECT

Interpretation(s)

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Sodium 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.	Potassium 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.	Chloride 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, hyperkalemic 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, overtreatment 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)

LIVER FUNCTION PROFILE, SERUM

BILIRUBIN, TOTAL	0.47	0.2 - 1.0	mg/dL
METHOD : SPECTROPHOTOMETRY, JENDRASSIK & GROFF			
BILIRUBIN, DIRECT	0.09	0.0 - 0.2	mg/dL
METHOD : SPECTROPHOTOMETRY, JENDRASSIK & GROFF			
BILIRUBIN, INDIRECT	0.38	0.1 - 1.0	mg/dL
METHOD : SPECTROPHOTOMETRY, CALCULATED			
TOTAL PROTEIN	7.3	6.4 - 8.2	g/dL
METHOD : SPECTROPHOTOMETRY, MODIFIED BIURET			
ALBUMIN	3.2 Low	3.4 - 5.0	g/dL
METHOD : SPECTROPHOTOMETRY, BCP - DYE BINDING			
GLOBULIN	4.1	2.0 - 4.1	g/dL
METHOD : SPECTROPHOTOMETRY, CALCULATED			
ALBUMIN/GLOBULIN RATIO	0.8 Low	1.0 - 2.1	RATIO
METHOD : SPECTROPHOTOMETRY, CALCULATED			
ASPARTATE AMINOTRANSFERASE (AST/SGOT)	20	15 - 37	U/L



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METHOD : SPECTROPHOTOMETRY, UV WITH PYRIDOXAL -5-PHOSPHATE

ALANINE AMINOTRANSFERASE (ALT/SGPT) 26

< 45.0

U/L

METHOD : SPECTROPHOTOMETRY, UV WITH PYRIDOXAL -5-PHOSPHATE

ALKALINE PHOSPHATASE 57

30 - 120

U/L

METHOD : SPECTROPHOTOMETRY, P-NPP (AMP BUFFER)

GAMMA GLUTAMYL TRANSFERASE (GGT) 36

15 - 85

U/L

METHOD : SPECTROPHOTOMETRY, G-GLUTAMYL-CARBOXY-NITRONILIDE

LACTATE DEHYDROGENASE 198

110 - 210

U/L

METHOD : SPECTROPHOTOMETRY, MODIFIED ENZYMATIC LACTATE - PYRUVATE

ALBUMIN, SERUM

ALBUMIN 3.2 Low

3.4 - 5.0

g/dL

METHOD : SPECTROPHOTOMETRY, BCP - DYE BINDING

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HEALTH SCREEN - 3**BUN/CREAT RATIO**

BUN/CREAT RATIO	6.21	5.00 - 15.00	
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METHOD : SPECTROPHOTOMETRY,CALCULATED

GLOBULIN

GLOBULIN	4.1	2.0 - 4.1	g/dL
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METHOD : SPECTROPHOTOMETRY,CALCULATED

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, sulfonylureas, 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 addition 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

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

Causes of decreased level include Liver disease, SIADH.

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.



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

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 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, Paget's disease, Rickets, Sarcoidosis etc. Lower-than-normal ALP levels seen in Hypophosphatasia, Malnutrition, Protein deficiency, Wilson's 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, Waldenström's 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.

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.



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Patient Ref. No. 775000002805460



PATIENT NAME : N PRABHAKAR

REF. DOCTOR : SELF

CODE/NAME & ADDRESS : C000138482

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HYDERABAD 500072
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ACCESSION NO : 0042WD000493

PATIENT ID : NPRAM04045342

CLIENT PATIENT ID:

ABHA NO :

AGE/SEX : 70 Years Male
DRAWN : 04/04/2023 00:00:00
RECEIVED : 04/04/2023 12:21:57
REPORTED : 04/04/2023 14:51:43

Test Report Status	Preliminary	Results	Biological Reference Interval	Units
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BIOCHEMISTRY - LIPID

HEALTH SCREEN - 3

LIPID PROFILE, SERUM

CHOLESTEROL, TOTAL	170	< 200 Desirable 200 - 239 Borderline High ≥ 240 High	mg/dL
--------------------	-----	--	-------

METHOD : SPECTROPHOTOMETRY, CHOLESTEROL OXIDASE ESTERASE PEROXIDASE

TRIGLYCERIDES	145	< 150 Normal 150 - 199 Borderline High 200 - 499 High ≥ 500 Very High	mg/dL
---------------	-----	--	-------

METHOD : SPECTROPHOTOMETRY, LIPASE

HDL CHOLESTEROL	58	< 40 Low ≥ 60 High	mg/dL
-----------------	----	-----------------------	-------

METHOD : SPECTROPHOTOMETRY, POLYANIONIC DETERGENT/CHOD

LDL CHOLESTEROL, DIRECT	75	< 100 Optimal 100 - 129 Near or above optimal 130 - 159 Borderline High 160 - 189 High ≥ 190 Very High	mg/dL
-------------------------	----	--	-------

METHOD : SPECTROPHOTOMETRY, ELIMINATION METHOD WITHOUT SAMPLE PRETREATMENT

NON HDL CHOLESTEROL	112	Desirable: Less than 130 Above Desirable: 130 - 159 Borderline High: 160 - 189 High: 190 - 219 Very high: > or = 220	mg/dL
---------------------	-----	--	-------

VERY LOW DENSITY LIPOPROTEIN	29.0	< 30.0	mg/dL
------------------------------	------	--------	-------

METHOD : SPECTROPHOTOMETRY, CALCULATED

CHOL/HDL RATIO	2.9 Low	3.3 - 4.4 Low Risk 4.5 - 7.0 Average Risk 7.1 - 11.0 Moderate Risk > 11.0 High Risk
LDL/HDL RATIO	1.3	0.5 - 3.0 Desirable/Low Risk 3.1 - 6.0 Borderline/Moderate Risk > 6.0 High Risk

METHOD : SPECTROPHOTOMETRY, CALCULATED

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Interpretation(s)

1) Cholesterol levels help assess the patient risk status and to follow the progress of patient under treatment to lower serum cholesterol concentrations.

2) Serum Triglyceride (TG) are a type of fat and a major source of energy for the body. Both quantity and composition of the diet impact on plasma triglyceride concentrations. Elevations in TG levels are the result of overproduction and impaired clearance. High TG are associated with increased risk for CAD (Coronary artery disease) in patients with other risk factors, such as low HDL-C, some patient groups with elevated apolipoprotein B concentrations, and patients with forms of LDL that may be particularly atherogenic.

3) HDL-C plays a crucial role in the initial step of reverse cholesterol transport, this considered to be the primary atheroprotective function of HDL

4) LDL -C plays a key role in causing and influencing the progression of atherosclerosis and, in particular, coronary sclerosis. The majority of cholesterol stored in atherosclerotic plaques originates from LDL, thus LDL-C value is the most powerful clinical predictor.

5) Non HDL cholesterol: Non-HDL-C measures the cholesterol content of all atherogenic lipoproteins, including LDL hence it is a better marker of risk in both primary and secondary prevention studies. Non-HDL-C also covers, to some extent, the excess ASCVD risk imparted by the sdLDL, which is significantly more atherogenic than the normal large buoyant particles, an elevated non-HDL-C indirectly suggests greater proportion of the small, dense variety of LDL particles

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
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	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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Test Report Status **Preliminary**

Results

Biological Reference Interval Units

MICRO BIOLOGY

CULTURE , SPUTUM SUSCEPTIBILITY

RESULT PENDING



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MC-3003

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CLINICAL PATH - URINALYSIS**HEALTH SCREEN - 3****PHYSICAL EXAMINATION, URINE**

COLOR	PALE YELLOW
APPEARANCE	CLEAR

CHEMICAL EXAMINATION, URINE

PH	6.0	4.7 - 7.5
SPECIFIC GRAVITY	1.015	1.003 - 1.035
PROTEIN	NOT DETECTED	NOT DETECTED
GLUCOSE	NOT DETECTED	NOT DETECTED
KETONES	NOT DETECTED	NOT DETECTED
BLOOD	NOT DETECTED	NOT DETECTED
BILIRUBIN	NOT DETECTED	NOT DETECTED
UROBILINOGEN	NORMAL	NORMAL
NITRITE	NOT DETECTED	NOT DETECTED
LEUKOCYTE ESTERASE	NOT DETECTED	NOT DETECTED

MICROSCOPIC EXAMINATION, URINE

RED BLOOD CELLS	NOT DETECTED	NOT DETECTED	/HPF
PUS CELL (WBC'S)	0-1	0-5	/HPF
EPITHELIAL CELLS	0-1	0-5	/HPF
CASTS	NOT DETECTED		
CRYSTALS	NOT DETECTED		
BACTERIA	NOT DETECTED	NOT DETECTED	
YEAST	NOT DETECTED	NOT DETECTED	

Comments

NOTE : URINE MICROSCOPIC EXAMINATION IS CARRIED OUT ON CENTRIFUGED URINE SEDIMENT.

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SPECIALISED CHEMISTRY - HORMONE

HEALTH SCREEN - 3

THYROID PANEL, SERUM

T3	81.10	80.0 - 200.0	ng/dL
T4	5.30	5.10 - 14.10	µg/dL
TSH (ULTRASENSITIVE)	4.000	0.270 - 4.200	µIU/mL

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SPECIALISED CHEMISTRY - VITAMIN

HEALTH SCREEN - 3

VITAMIN B12(CYANOCOBALAMINE), SERUM

VITAMIN B12	1112.0 High	197 - 771	pg/mL
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25 - HYDROXYVITAMIN D(VITAMIN D TOTAL), SERUM

25 - HYDROXYVITAMIN D	19.81 Low	Deficiency: < 20.0 Insufficiency: 20.0 - < 30.0 Sufficiency: 30.0 - 100.0 Toxicity > 100.0	ng/mL
-----------------------	-----------	--	-------

Interpretation(s)

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.Importance 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 at least 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 MMA > 0.30µmol/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.

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 D25(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 at least 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.

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

End Of Report

Please visit www.srlworld.com for related Test Information for this accession

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2. All tests are performed and reported as per the turnaround time stated in the SRL 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. SRL 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.

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