



MC-3003

PATIENT NAME : FARKHUNDA JABEEN**REF. DOCTOR :**

CODE/NAME & ADDRESS : C000138482
RELEX HEALTHCARE SERVICES INDIA PVT LTD
PLOT 63/A, GROUND FLOOR, RAGHAVENDRA
NILAYAM, 7TH PHASE,KPHB COLONY,
HYDERABAD 500072
08047109222

ACCESSION NO : **0042WB003997**
PATIENT ID : FARKF24028142
CLIENT PATIENT ID:
ABHA NO :

AGE/SEX : 42 Years Female
DRAWN : 24/02/2023 00:00:00
RECEIVED : 24/02/2023 11:15:24
REPORTED : 24/02/2023 15:24:24

Test Report Status	Final	Results	Biological Reference Interval	Units
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HAEMATOLOGY - CBC**HEALTH SCREEN - 3****BLOOD COUNTS,EDTA WHOLE BLOOD**

HEMOGLOBIN (HB) METHOD : CYANMETHEMOGLOBIN METHOD	11.8 Low	12.0 - 15.0	g/dL
RED BLOOD CELL (RBC) COUNT METHOD : ELECTRICAL IMPEDANCE	4.68	3.8 - 4.8	mil/ μ L
WHITE BLOOD CELL (WBC) COUNT METHOD : ELECTRICAL IMPEDANCE	8.20	4.0 - 10.0	thou/ μ L
PLATELET COUNT METHOD : ELECTRICAL IMPEDANCE	329	150 - 410	thou/ μ L

RBC AND PLATELET INDICES

HEMATOCRIT (PCV) METHOD : CALCULATED PARAMETER	38.3	36 - 46	%
MEAN CORPUSCULAR VOLUME (MCV) METHOD : CALCULATED PARAMETER	82.0 Low	83 - 101	fL
MEAN CORPUSCULAR HEMOGLOBIN (MCH) METHOD : CALCULATED PARAMETER	25.1 Low	27.0 - 32.0	pg
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC) METHOD : CALCULATED PARAMETER	30.7 Low	31.5 - 34.5	g/dL

RED CELL DISTRIBUTION WIDTH (RDW) METHOD : CALCULATED PARAMETER	14.3 High	11.6 - 14.0	%
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MENTZER INDEX	17.5
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MEAN PLATELET VOLUME (MPV) METHOD : CALCULATED PARAMETER	9.6	6.8 - 10.9	fL
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WBC DIFFERENTIAL COUNT

NEUTROPHILS METHOD : ACV TECHNOLOGY	59	40 - 80	%
LYMPHOCYTES METHOD : ACV TECHNOLOGY	37	20 - 40	%
MONOCYTES METHOD : ACV TECHNOLOGY	3	2 - 10	%
EOSINOPHILS METHOD : ACV TECHNOLOGY	1	1 - 6	%

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BASOPHILS		0	0 - 2	%
METHOD : ACV TECHNOLOGY				
ABSOLUTE NEUTROPHIL COUNT		4.84	2.0 - 7.0	thou/ μ L
METHOD : CALCULATED PARAMETER				
ABSOLUTE LYMPHOCYTE COUNT		3.03 High	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		0.08	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)		1.6		
METHOD : CALCULATED				
MORPHOLOGY				
RBC			NORMOCYTIC NORMOCHROMIC WITH FEW MICROCYTES.	
METHOD : MICROSCOPIC EXAMINATION				
WBC			WITHIN NORMAL LIMITS.	
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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FBS (FASTING BLOOD SUGAR) **206 High** 74 - 99 mg/dL

METHOD : SPECTROPHOTOMETRY HEXOKINASE

GLYCOSYLATED HEMOGLOBIN(HBA1C), EDTA WHOLE BLOOD

HBA1C **11.1 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)

METHOD : ION- EXCHANGE HPLC

ESTIMATED AVERAGE GLUCOSE(EAG) **271.9 High** < 116.0 mg/dL

METHOD : ION- EXCHANGE HPLC

BLOOD UREA NITROGEN (BUN), SERUM

BLOOD UREA NITROGEN **10** 6 - 20 mg/dL

METHOD : SPECTROPHOTOMETRY, UREASE UV

CREATININE, SERUM

CREATININE **0.79** 0.60 - 1.10 mg/dL

METHOD : SPECTROPHOTOMETRY, ALKALINE PICRATE KINETIC JAFFE'S

URIC ACID, SERUM

URIC ACID **3.7** 2.6 - 6.0 mg/dL

METHOD : SPECTROPHOTOMETRY, URICASE

TOTAL PROTEIN, SERUM

TOTAL PROTEIN **7.6** 6.4 - 8.2 g/dL

METHOD : SPECTROPHOTOMETRY, MODIFIED BIURET

ELECTROLYTES (NA/K/CL), SERUM

SODIUM, SERUM **144** 136 - 145 mmol/L

METHOD : INTEGRATED MULTISENSOR TECHNOLOGY-INDIRECT

POTASSIUM, SERUM **5.06** 3.50 - 5.10 mmol/L

METHOD : INTEGRATED MULTISENSOR TECHNOLOGY-INDIRECT

CHLORIDE, SERUM **100** 98 - 107 mmol/L

METHOD : INTEGRATED MULTISENSOR TECHNOLOGY-INDIRECT

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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, 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 HCO3-), 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	0.34	0.2 - 1.0	mg/dL
METHOD : SPECTROPHOTOMETRY, JENDRASSIK & GROFF			
BILIRUBIN, DIRECT	0.05	0.0 - 0.2	mg/dL
METHOD : SPECTROPHOTOMETRY, JENDRASSIK & GROFF			
BILIRUBIN, INDIRECT	0.29	0.1 - 1.0	mg/dL
METHOD : SPECTROPHOTOMETRY, CALCULATED			
TOTAL PROTEIN	7.6	6.4 - 8.2	g/dL
METHOD : SPECTROPHOTOMETRY, MODIFIED BIURET			
ALBUMIN	4.2	3.4 - 5.0	g/dL
METHOD : SPECTROPHOTOMETRY, BCP - DYE BINDING			
GLOBULIN	3.4	2.0 - 4.1	g/dL
METHOD : SPECTROPHOTOMETRY, CALCULATED			
ALBUMIN/GLOBULIN RATIO	1.2	1.0 - 2.1	RATIO
METHOD : SPECTROPHOTOMETRY, CALCULATED			



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ASPARTATE AMINOTRANSFERASE (AST/SGOT)		18	15 - 37	U/L
		METHOD : SPECTROPHOTOMETRY, UV WITH PYRIDOXAL -5-PHOSPHATE		
ALANINE AMINOTRANSFERASE (ALT/SGPT)	33		< 34.0	U/L
		METHOD : SPECTROPHOTOMETRY, UV WITH PYRIDOXAL -5-PHOSPHATE		
ALKALINE PHOSPHATASE	97		30 - 120	U/L
		METHOD : SPECTROPHOTOMETRY, P-NPP (AMP BUFFER)		
GAMMA GLUTAMYL TRANSFERASE (GGT)	36		5 - 55	U/L
		METHOD : SPECTROPHOTOMETRY, G-GLUTAMYL-CARBOXY-NITRONILIDE		
LACTATE DEHYDROGENASE	150		100 - 190	U/L
		METHOD : SPECTROPHOTOMETRY, MODIFIED ENZYMATIC LACTATE - PYRUVATE		
ALBUMIN, SERUM				
ALBUMIN	4.2		3.4 - 5.0	g/dL
		METHOD : SPECTROPHOTOMETRY, BCP - DYE BINDING		
GLUCOSE, POST-PRANDIAL, PLASMA				
PPBS(POST PRANDIAL BLOOD SUGAR)	300 High		70 - 139	mg/dL
		METHOD : SPECTROPHOTOMETRY HEXOKINASE		



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HEALTH SCREEN - 3

BUN/CREAT RATIO

BUN/CREAT RATIO 12.66 5.00 - 15.00

METHOD : SPECTROPHOTOMETRY,CALCULATED

GLOBULIN

GLOBULIN 3.4 2.0 - 4.1 g/dL

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 :

I.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.

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

III.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.

IV.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.

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)

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- 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
- Muscular dystrophy

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-Serum 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, Waldenstrom'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.

LIVER FUNCTION PROFILE, SERUM-LIVER FUNCTION PROFILE

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.Serum 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,Waldenstrom'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.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

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.

GLUCOSE, POST-PRANDIAL, PLASMA-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.Additional test HbA1c

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Patient Ref. No. 775000002431311



MC-3003

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Test Report Status Final**Results****Biological Reference Interval****Units**

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- 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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Test Report Status	Final	Results	Biological Reference Interval	Units
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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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COLOR PALE YELLOW
 METHOD : MANUAL

APPEARANCE SLIGHTLY HAZY
 METHOD : MANUAL

CHEMICAL EXAMINATION, URINE

PH 5.5 4.7 - 7.5
 METHOD : REFLECTANCE SPECTROPHOTOMETRY

SPECIFIC GRAVITY 1.030 1.003 - 1.035
 METHOD : REFLECTANCE SPECTROPHOTOMETRY

PROTEIN NOT DETECTED NOT DETECTED
 METHOD : REFLECTANCE SPECTROPHOTOMETRY

GLUCOSE NOT DETECTED NOT DETECTED
 METHOD : REFLECTANCE SPECTROPHOTOMETRY

KETONES NOT DETECTED NOT DETECTED
 METHOD : REFLECTANCE SPECTROPHOTOMETRY

BLOOD NOT DETECTED NOT DETECTED
 METHOD : REFLECTANCE SPECTROPHOTOMETRY

BILIRUBIN NOT DETECTED NOT DETECTED
 METHOD : REFLECTANCE SPECTROPHOTOMETRY

UROBILINOGEN NORMAL NORMAL
 METHOD : REFLECTANCE SPECTROPHOTOMETRY

NITRITE NOT DETECTED NOT DETECTED
 METHOD : REFLECTANCE SPECTROPHOTOMETRY

LEUKOCYTE ESTERASE NOT DETECTED NOT DETECTED
 METHOD : MICROSCOPIC EXAMINATION

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

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

EPITHELIAL CELLS 2-3 0-5 /HPF
 METHOD : MICROSCOPIC EXAMINATION

CASTS NOT DETECTED

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Test Report Status **Final**

Results

Biological Reference Interval **Units**

METHOD : MICROSCOPIC EXAMINATION

CRYSTALS

NOT DETECTED

METHOD : MICROSCOPIC EXAMINATION

BACTERIA

NOT DETECTED

NOT DETECTED

METHOD : MICROSCOPIC EXAMINATION

YEAST

NOT DETECTED

NOT DETECTED

Comments

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

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Test Report Status**Final****Results****Biological Reference Interval****Units****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 (<i>Averrhoa carambola</i>) or its juice
Uric acid	arthritis
Bacteria	Urinary infection when present in significant numbers & with pus cells.
Trichomonas vaginalis	Vaginitis, cervicitis or salpingitis

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Test Report Status Final**Results****Biological Reference Interval****Units****SPECIALISED CHEMISTRY - HORMONE****HEALTH SCREEN - 3****THYROID PANEL, SERUM**

T3	79.86 Low	Non-Pregnant Women	ng/dL
		80.0 - 200.0	
		Pregnant Women	
		1st Trimester: 105.0 - 230.0	
		2nd Trimester: 129.0 - 262.0	
		3rd Trimester: 135.0 - 262.0	
METHOD : ECLIA			
T4	5.89	Non-Pregnant Women	µg/dL
		5.10 - 14.10	
		Pregnant Women	
		1st Trimester: 7.33 - 14.80	
		2nd Trimester: 7.93 - 16.10	
		3rd Trimester: 6.95 - 15.70	
METHOD : ECLIA			
TSH (ULTRASENSITIVE)	2.400	Non Pregnant Women	µIU/mL
		0.27 - 4.20	
		Pregnant Women	
		1st Trimester: 0.33 - 4.59	
		2nd Trimester: 0.35 - 4.10	
		3rd Trimester: 0.21 - 3.15	
METHOD : ECLIA			

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Test Report Status **Final**

Results

Biological Reference Interval

Units

Interpretation(s)

Triiodothyronine T3 , Thyroxine T4, and Thyroid Stimulating Hormone TSH are thyroid hormones which affect almost every physiological process in the body, including growth, development, metabolism, body temperature, and heart rate.

Production of T3 and its prohormone thyroxine (T4) is activated by thyroid-stimulating hormone (TSH), which is released from the pituitary gland. Elevated concentrations of T3, and T4 in the blood inhibit the production of TSH.

Excessive secretion of thyroxine in the body is hyperthyroidism, and deficient secretion is called hypothyroidism.

In primary hypothyroidism, TSH levels are significantly elevated, while in secondary and tertiary hyperthyroidism, TSH levels are low.

Below mentioned are the guidelines for Pregnancy related reference ranges for Total T4, TSH & Total T3. Measurement of the serum TT3 level is a more sensitive test for the diagnosis of hyperthyroidism, and measurement of TT4 is more useful in the diagnosis of hypothyroidism. Most of the thyroid hormone in blood is bound to transport proteins. Only a very small fraction of the circulating hormone is free and biologically active. It is advisable to detect Free T3, FreeT4 along with TSH, instead of testing for albumin bound Total T3, Total T4.

Sr. No.	TSH	Total T4	FT4	Total T3	Possible Conditions
1	High	Low	Low	Low	(1) Primary Hypothyroidism (2) Chronic autoimmune Thyroiditis (3) Post Thyroidectomy (4) Post Radio-Iodine treatment
2	High	Normal	Normal	Normal	(1) Subclinical Hypothyroidism (2) Patient with insufficient thyroid hormone replacement therapy (3) In cases of Autoimmune/Hashimoto thyroiditis (4) Isolated increase in TSH levels can be due to Subclinical inflammation, drugs like amphetamines, Iodine containing drug and dopamine antagonist e.g. domperidone and other physiological reasons.
3	Normal/Low	Low	Low	Low	(1) Secondary and Tertiary Hypothyroidism
4	Low	High	High	High	(1) Primary Hyperthyroidism (Graves Disease) (2) Multinodular Goitre (3) Toxic Nodular Goitre (4) Thyroiditis (5) Over treatment of thyroid hormone (6) Drug effect e.g. Glucocorticoids, dopamine, T4 replacement therapy (7) First trimester of Pregnancy
5	Low	Normal	Normal	Normal	(1) Subclinical Hyperthyroidism
6	High	High	High	High	(1) TSH secreting pituitary adenoma (2) TRH secreting tumor
7	Low	Low	Low	Low	(1) Central Hypothyroidism (2) Euthyroid sick syndrome (3) Recent treatment for Hyperthyroidism
8	Normal/Low	Normal	Normal	High	(1) T3 thyrotoxicosis (2) Non-Thyroidal illness
9	Low	High	High	Normal	(1) T4 Ingestion (2) Thyroiditis (3) Interfering Anti TPO antibodies

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

NOTE: It is advisable to detect Free T3,FreeT4 along with TSH, instead of testing for albumin bound Total T3, Total T4. TSH is not affected by variation in thyroid - binding protein. TSH has a diurnal rhythm, with peaks at 2:00 - 4:00 a.m. And troughs at 5:00 - 6:00 p.m. With ultradian variations.



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Test Report Status**Final****Results****Biological Reference Interval****Units****SPECIALISED CHEMISTRY - VITAMIN****HEALTH SCREEN - 3****VITAMIN B12(CYANOCOBALAMINE), SERUM**

VITAMIN B12

818.8 High

197 - 771

pg/mL

METHOD : ECLIA

25 - HYDROXYVITAMIN D(VITAMIN D TOTAL), SERUM

25 - HYDROXYVITAMIN D

21.29 Low

Deficiency:

< 20.0

Insufficiency:

20.0 - < 30.0

Sufficiency:

30.0 -100.0

Toxicity > 100.0

ng/mL

METHOD : ECLIA

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.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 MMA > 30pmol/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

Dr. Ravi Teja J
Consultant Pathologist

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Email : customercare.hyderabad@srl.in

**Patient Ref. No. 775000002431311**



MC-3003

PATIENT NAME : FARKHUNDA JABEEN

REF. DOCTOR :

CODE/NAME & ADDRESS : C000138482 RELEX HEALTHCARE SERVICES INDIA PVT LTD PLOT 63/A, GROUND FLOOR, RAGHAVENDRA NILAYAM, 7TH PHASE,KPHB COLONY, HYDERABAD 500072 08047109222	ACCESSION NO : 0042WB003997 PATIENT ID : FARKF24028142 CLIENT PATIENT ID: ABHA NO :	AGE/SEX : 42 Years Female DRAWN : 24/02/2023 00:00:00 RECEIVED : 24/02/2023 11:15:24 REPORTED : 24/02/2023 15:24:24
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Test Report Status	Final	Results	Biological Reference Interval	Units
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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''''''''s 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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