DIAGNOSTIC REPORT

SRL Diagnostics

CLIENT CODE : C000000614

CLIENT'S NAME AND ADDRESS :

INDIA

PATIENT NAME : PATIENT ID : RADHF3012422

ACCESSION NO : 0002QL072847 AGE : 75 Years SEX : Female DATE OF BIRTH :

DRAWN : 30-12-2017 14:25 RECEIVED : 30-12-2017 14:27 REPORTED : 10-02-2018 20:00

REFERRING DOCTOR : SELF CLIENT PATIENT ID : 0044

Test Report Status Final	Results	Biological Reference Interval	Units
COMPLETE CARE TOTAL			
COMPLETE BLOOD COUNT			
RED BLOOD CELL COUNT	3.89	3.8 - 4.8	mil/µL
METHOD : COULTER PRINCIPLE			
HEMOGLOBIN	12.4	12.0 - 15.0	g/dL
METHOD : PHOTOMETRIC MEASUREMENT, CYANMETHEMOGLOBIN METHOD			
HEMATOCRIT	36.0	36 - 46	%
METHOD : CALCULATED PARAMETER			
MEAN CORPUSCULAR VOL	92.4	83.0 - 101.0	fL
METHOD : DERIVED PARAMETER FROM RBC HISTOGRAM			
MEAN CORPUSCULAR HGB.	31.9	27.0 - 32.0	pg
METHOD : CALCULATED PARAMETER			
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION	34.5	31.5 - 34.5	g/dL
METHOD : CALCULATED PARAMETER			
RED CELL DISTRIBUTION WIDTH	13.6	11.6 - 14.0	%
METHOD : DERIVED PARAMETER FROM RBC HISTOGRAM			
PLATELET COUNT	246	150 - 410	thou/µL
METHOD : ELECTRONIC IMPEDENCE & MICROSCOPY			
MEAN PLATELET VOLUME	8.6	6.8 - 10.9	fL
METHOD : DERIVED PARAMETER FROM PLATELET HISTOGRAM			
WHITE BLOOD CELL COUNT	8.4	4.0 - 10.0	thou/µL
METHOD : COULTER PRINCIPLE			
WBC DIFFERENTIAL COUNT			
SEGMENTED NEUTROPHILS	58	40 - 80	%
METHOD : VCS TECHNOLOGY/ MICROSCOPY			
EOSINOPHILS	4	1 - 6	%
METHOD : VCS TECHNOLOGY/ MICROSCOPY			
LYMPHOCYTES	31	20 - 40	%
METHOD : VCS TECHNOLOGY/ MICROSCOPY			
MONOCYTES	7	2 - 10	%
METHOD : VCS TECHNOLOGY/ MICROSCOPY			
BASOPHILS	0	< 1 - 2	%
METHOD : VCS TECHNOLOGY/ MICROSCOPY			
ERYTHRO SEDIMENTATION RATE, BLOOD			
SEDIMENTATION RATE (ESR)	33	0 - 35	mm at 1 hr
METHOD : AUTOMATED (PHOTOMETRICAL CAPILLARY STOPPED FLOW KINETIC ANALYSIS)			
PERIPHERAL SMEAR EXAM, EDTA WHOLE BLOOD			
RBC	Normocytic normochromic		

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METHOD : MICROSCOPIC EXAMINATION				
WBC	Normal morphology.			
METHOD : MICROSCOPIC EXAMINATION				
PLATELETS	Adequate in smear.			
METHOD : ELECTRONIC IMPEDENCE & MICROSCOPY				
TOTAL IRON BINDING CAPACITY, SERUM				
IRON	78		50 - 170	µg/dL
METHOD : SPECTROPHOTOMETRY – FERENE				
TOTAL IRON BINDING CAPACITY	391		250 - 450	µg/dL
METHOD : SPECTROPHOTOMETRY – FERENE				
% SATURATION	20		13 - 45	%
METHOD : CALCULATED PARAMETER				
GLUCOSE, FASTING, PLASMA				
GLUCOSE, FASTING, PLASMA	224	High	82 - 99	mg/dL
METHOD : SPECTROPHOTOMETRY HEXOKINASE				
GLYCOSYLATED HEMOGLOBIN, EDTA WHOLE BLOOD				
GLYCOSYLATED HEMOGLOBIN (HBA1C)	8.2	High	Non-diabetic: < 5.7 Pre-diabetics: 5.7 - 6.4 Diabetics: > or = 6.5 ADA Target: 7.0 Action suggested: > 8.0	%
METHOD : ION- EXCHANGE HPLC				
MEAN PLASMA GLUCOSE	188.6	High	< 116.0	mg/dL
LIVER FUNCTION PROFILE, SERUM				
BILIRUBIN, TOTAL	0.31		0.2 - 1.0	mg/dL
METHOD : SPECTROPHOTOMETRY, MODIFIED JENDRASSIK & GROFF				
BILIRUBIN, DIRECT	0.08		0.0 - 0.2	mg/dL
TOTAL PROTEIN	7.9		6.4 - 8.2	g/dL
METHOD : SPECTROPHOTOMETRY				
ALBUMIN	3.9		3.4 - 5.0	g/dL
METHOD : SPECTROPHOTOMETRY, BCP - DYE BINDING				
GLOBULIN	4.0		2.0 - 4.1	G/DL
METHOD : CALCULATED PARAMETER				
ALBUMIN/GLOBULIN RATIO	1.0		1.0 - 2.1	RATIO
METHOD : CALCULATED PARAMETER				
ASPARTATE AMINOTRANSFERASE (AST/SGOT)	30		15 - 37	U/L
METHOD : SPECTROPHOTOMETRY, UV WITH PYRIDOXAL -5-PHOSPHATE				
ALANINE AMINOTRANSFERASE (ALT/SGPT)	38	High	< 34.0	U/L

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METHOD : SPECTROPHOTOMETRY, UV WITH PYRIDOXAL -5-PHOSPHATE				
ALKALINE PHOSPHATASE	86		30 - 120	U/L
METHOD : SPECTROPHOTOMETRY, P-NPP (AMP BUFFER)				
GAMMA GLUTAMYL TRANSFERASE (GGT)	53		5 - 55	U/L
METHOD : SPECTROPHOTOMETRY				
LACTATE DEHYDROGENASE	154		110 - 210	U/L
METHOD : SPECTROPHOTOMETRY				
25 - HYDROXYVITAMIN D, SERUM				
25 - HYDROXYVITAMIN D	10.83	Low	Deficiency: < 20.0 Insufficiency: 20.0 - < 30.0 Sufficiency: 30.0 -100.0 Toxicity > 100.0	ng/mL
METHOD : CHEMILUMINESCENCE,COMPETITIVE IMMUNOASSAY				
CALCIUM, SERUM				
CALCIUM	8.9		8.5 - 10.1	mg/dL
METHOD : SPECTROPHOTOMETRY, O-CRESOLPHTHALEIN COMPLEXONE				
VITAMIN B12 LEVEL, SERUM				
VITAMIN B12	942.0	High	211.0 - 911.0	pg/mL
METHOD : CHEMILUMINESCENCE,COMPETITIVE IMMUNOASSAY				
THYROID PANEL, SERUM				
T3	92.87		60.0 - 181.0	ng/dl
METHOD : CHEMILUMINESCENCE, COMPETETIVE IMMUNOASSAY				
T4	6.50		4.5 - 10.9	µg/dl
METHOD : CHEMILUMINESCENCE, COMPETETIVE IMMUNOASSAY				
TSH 3RD GENERATION	5.897	High	0.55 - 4.78	µIU/mL
METHOD : CHEMILUMINESCENCE,TWO STEP SANDWICH IMMUNOASSAY				
CORONARY RISK PROFILE (LIPID PROFILE), SERUM				
CHOLESTEROL	227	High	< 200 Desirable 200 - 239 Borderline High >/= 240 High	mg/dL
METHOD : SPECTROPHOTOMETRY,CHOLESTEROL OXIDASE ESTERASE PEROXIDASE				
TRIGLYCERIDES	163	High	< 150 Normal 150 - 199 Borderline High 200 - 499 High >/=500 Very High	mg/dL
METHOD : SPECTROPHOTOMETRY, ENZYMATIC ENDPOINT				
HDL CHOLESTEROL	59		< 40 Low >/=60 High	mg/dL
METHOD : SPECTROPHOTOMETRY				

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DIRECT LDL CHOLESTEROL	139	High	< 100 Optimal 100 - 129 Near or above optimal 130 - 159 Borderline High 160 - 189 High >/= 190 Very High	mg/dL
METHOD : SPECTROPHOTOMETERY, DIRECT MEASURE-PEG/ CHOD				
CHOL/HDL RATIO	3.9		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	2.4		0.5 - 3.0 Desirable/Low Risk 3.1 - 6.0 Borderline/Moderate Risk >6.0 High Risk	
METHOD : CALCULATED PARAMETER				
VERY LOW DENSITY LIPOPROTEIN	32.6	High	</= 30.0	mg/dL
METHOD : CALCULATED PARAMETER				
SERUM BLOOD UREA NITROGEN				
BLOOD UREA NITROGEN	17		8 - 23	mg/dL
METHOD : SPECTROPHOTOMETRY, UREASE UV				
CREATININE, SERUM				
CREATININE	0.92		0.60 - 1.20	mg/dL
METHOD : SPECTROPHOTOMETRY, ALKALINE PICRATE KINETIC JAFFE'S				
BUN/CREAT RATIO				
BUN/CREAT RATIO	18.48	High	5.00 - 15.00	
METHOD : CALCULATED PARAMETER				
URIC ACID, SERUM				
URIC ACID	4.4		2.6 - 6.0	mg/dL
METHOD : SPECTROPHOTOMETRY, URICASE				
TOTAL PROTEIN, SERUM				
TOTAL PROTEIN	7.9		6.4 - 8.2	g/dL
METHOD : SPECTROPHOTOMETRY, BIURET				
ALBUMIN, SERUM				
ALBUMIN	3.9		3.4 - 5.0	g/dL
METHOD : SPECTROPHOTOMETRY, BCP - DYE BINDING				
GLOBULIN				
GLOBULIN	4.0		2.0 - 4.1	g/dL
METHOD : CALCULATED PARAMETER				
ELECTROLYTES (NA/K/CL), SERUM				
SODIUM	139		136 - 145	mmol/L
METHOD : IMT (ISE INDIRECT)				
POTASSIUM	4.82		3.50 - 5.10	mmol/L

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METHOD : IMT (ISE INDIRECT)			
CHLORIDE	103	98 - 107	mmol/L
METHOD : IMT (ISE INDIRECT)			
URINALYSIS			
COLOR	PALE YELLOW		
METHOD : REFLECTANCE SPECTROPHOTOMETRY			
APPEARANCE	SLIGHTLY HAZY		
METHOD : REFLECTANCE SPECTROPHOTOMETRY			
PH	6.0	4.7 - 7.5	
METHOD : REFLECTANCE SPECTROPHOTOMETRY- DOUBLE INDICATOR METHOD			
SPECIFIC GRAVITY	1.010	1.003 - 1.035	
METHOD : REFLECTANCE SPECTROPHOTOMETRY- PKA CHANGE OF AN IONIC POLYELECTROLYTE			
GLUCOSE	NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY, DOUBLE SEQUENTIAL ENZYME REACTION-GOD/POD			
PROTEIN	NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY - PROTEIN-ERROR-OF-INDICATOR PRINCIPLE			
KETONES	NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY, ROTHERA'S PRINCIPLE			
BLOOD	NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY, PEROXIDASE LIKE ACTIVITY OF HAEMOGLOBIN			
BILIRUBIN	NOT DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY, DIAZOTIZATION- COUPLING OF BILIRUBIN WITH DIAZOTIZED SALT			
UROBILINOGEN	NORMAL	NORMAL	
METHOD : REFLECTANCE SPECTROPHOTOMETRY - EHRLICH REACTION			
NITRITE	DETECTED	NOT DETECTED	
METHOD : REFLECTANCE SPECTROPHOTOMETRY, CONVERSION OF NITRATE TO NITRITE			
WBC	8-10	0-5	/HPF
METHOD : MICROSCOPIC EXAMINATION			
EPITHELIAL CELLS	1-2	0-5	/HPF
METHOD : MICROSCOPIC EXAMINATION			
RED BLOOD CELLS	NOT DETECTED	NOT DETECTED	/HPF
METHOD : MICROSCOPIC EXAMINATION			
CASTS	NOT DETECTED	NOT DETECTED	
METHOD : MICROSCOPIC EXAMINATION			
CRYSTALS	NOT DETECTED	NOT DETECTED	
METHOD : MICROSCOPIC EXAMINATION			
BACTERIA	DETECTED (+)	NOT DETECTED	
METHOD : MICROSCOPIC EXAMINATION			

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Comments

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

NOTE : FASTING URINE SPECIMEN RECEIVED ON 09/02/2018.

MAGNESIUM, SERUM

MAGNESIUM	1.6	Low	1.8 - 2.4	mg/dL

METHOD : SPECTROPHOTOMETRY, METHYLTHYMOL BLUE

Comments

NOTE : RECHECKED FOR SERUM MAGNESIUM.

Interpretation(s)
COMPLETE BLOOD COUNT-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.
ERYTHRO SEDIMENTATION RATE, BLOOD-Erythrocyte sedimentation rate (ESR) is a non - specific phenomena and is clinically useful in the diagnosis and monitoring of disorders associated with an increased production of acute phase reactants. The ESR is increased in pregnancy from about the 3rd month and returns to normal by the 4th week post partum. ESR is influenced by age, sex, menstrual cycle and drugs (eg. corticosteroids, contraceptives). It is especially low (0 -1mm) in polycythaemia, hypofibrinogenemia or congestive cardiac failure and when there are abnormalities of the red cells such as poikilocytosis, spherocytosis or sickle cells.

Reference :
1. Nathan and Oski's Haematology of Infancy and Childhood, 5th edition
2. Paediatric reference intervals. AACC Press, 7th edition. Edited by S. Soldin
3. The reference for the adult reference range is "Practical Haematology by Dacie and Lewis, 10th Edition"
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 they 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 x 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.
GLUCOSE, FASTING, PLASMA-ADA 2012 guidelines for adults as follows:
Pre-diabetics: 100 - 125 mg/dL
Diabetic: > or = 126 mg/dL

(Ref: Tietz 4th Edition & ADA 2012 Guidelines)
GLYCOSYLATED HEMOGLOBIN, EDTA WHOLE BLOOD-
Glycosylated hemoglobin (GHb) has been firmly established as an index of long-term blood glucose concentrations and as a measure of the risk for the development of complications in patients with diabetes mellitus. Formation of GHb is essentially irreversible, and the concentration in the blood depends on both the life span of the red blood cell (average 120 days) and the blood glucose concentration. Because the rate of formation of GHb is directly proportional to the concentration of glucose in the blood, the GHb concentration represents the integrated values for glucose over the preceding 6-8 weeks.
Any condition that alters the life span of the red blood cells has the potential to alter the GHb level. Samples from patients with hemolytic anemias will exhibit decreased glycated hemoglobin values due to the shortened life span of the red cells. This effect will depend upon the severity of the anemia. Samples from patients with polycythemia or post-splenectomy may exhibit increased glycated hemoglobin values due to a somewhat longer life span of the red cells.
Glycosylated hemoglobins results from patients with HbSS, HbCC, and HbSC and HbD must be interpreted with caution, given the pathological processes, including anemia, increased red cell turnover, transfusion requirements, that adversely impact HbA1c as a marker of long-term glycemic control. In these conditions, alternative forms of

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testing such as glycated serum protein (fructosamine) should be considered.

References
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, 879-884.
2. Forsham PH. Diabetes Mellitus:A rational plan for management. Postgrad Med 1982, 71,139-154.
3. Mayer TK, Freedman ZR: Protein glycosylation in Diabetes Mellitus: A review of laboratory measurements and their clinical utility. Clin Chim Acta 1983, 127, 147-184.

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

25 - HYDROXYVITAMIN D, SERUM-

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

CALCIUM, SERUM-Commom 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 absorbtion (vitamin d intoxication), increased skeletal reasorption (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 torniquet application during sampling. Thus, this along with fist clenching should be avoided before phlebotomy.

THYROID PANEL, SERUM-Triiodothyronine T3 , is a thyroid hormone. It affects 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.

Thyroxine T4, Thyroxine's principal function is to stimulate the metabolism of all cells and tissues in the body. Excessive secretion of thyroxine in the body is hyperthyroidism, and deficient secretion is called 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.

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

Below mentioned are the guidelines for Pregnancy related reference ranges for Total T4, TSH & Total T3

Levels in Pregnancy	TOTAL T4 (µg/dL)	TSH3G (µIU/mL)	TOTAL T3 (ng/dL)
First Trimester	6.6 - 12.4	0.1 - 2.5	81 - 190
2nd Trimester	6.6 - 15.5	0.2 - 3.0	100 - 260
3rd Trimester	6.6 - 15.5	0.3 - 3.0	100 - 260

Below mentioned are the guidelines for age related reference ranges for T3, T4 and TSH.

T3 (ng/dL)	T4 (µg/dL)	TSH3G (µIU/mL)
Cord Blood: 30 - 70	1-3 day: 8.2 - 19.9	< 2 years – Not Established
New Born: 75 - 260	1 Week: 6.0 - 15.9	
1-5 Years: 100 - 260	1-12 Months: 6.1 - 14.9	
5 - 10 Years: 90 - 240	1 - 3 Years: 6.8 - 13.5	
10 - 15 Years: 80 - 210	3 - 10 Years: 5.5 - 12.8	

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Reference:
1. Burtis C.A., Ashwood E. R. Bruns D.E. Teitz textbook of Clinical Chemistry and Molecular Diagnostics, 4th Edition.
2. Gowenlock A.H. Varley''''s Practical Clinical Biochemistry, 6th Edition.
3. Behrman R.E. Kilegman R.M., Jenson H. B. Nelson Text Book of Pediatrics, 17th Edition
CORONARY RISK PROFILE (LIPID PROFILE), SERUM-Serum cholesterol is a blood test that can provide valuable information for the risk of coronary artery disease This test can help determine your risk of the build up of plaques in your arteries that can lead to narrowed or blocked arteries throughout your body (atherosclerosis). High cholesterol levels usually don''t cause any signs or symptoms, so a cholesterol test is an important tool. High cholesterol levels often are a significant risk factor for heart disease and important for diagnosis of hyperlipoproteinemia, atherosclerosis, hepatic and thyroid diseases.

Serum Triglyceride are a type of fat in the blood. When you eat, your body converts any calories it doesn''t need into triglycerides, which are stored in fat cells. High triglyceride levels are associated with several factors, including being overweight, eating too many sweets or drinking too much alcohol, smoking, being sedentary, or having diabetes with elevated blood sugar levels. Analysis has proven useful in the diagnosis and treatment of patients with diabetes mellitus, nephrosis, liver obstruction, other diseases involving lipid metabolism, and various endocrine disorders. In conjunction with high density lipoprotein and total serum cholesterol, a triglyceride determination provides valuable information for the assessment of coronary heart disease risk.It is done in fasting state.

High-density lipoprotein (HDL) cholesterol. This is sometimes called the ""good"" cholesterol because it helps carry away LDL cholesterol, thus keeping arteries open and blood flowing more freely.HDL cholesterol is inversely related to the risk for cardiovascular disease. It increases following regular exercise, moderate alcohol consumption and with oral estrogen therapy. Decreased levels are associated with obesity, stress, cigarette smoking and diabetes mellitus.

SERUM LDL The small dense LDL test can be used to determine cardiovascular risk in individuals with metabolic syndrome or established/progressing coronary artery disease, individuals with triglyceride levels between 70 and 140 mg/dL, as well as individuals with a diet high in trans-fat or carbohydrates. Elevated sdLDL levels are associated with metabolic syndrome and an 'atherogenic lipoprotein profile', and are a strong, independent predictor of cardiovascular disease.
Elevated levels of LDL arise from multiple sources. A major factor is sedentary lifestyle with a diet high in saturated fat. Insulin-resistance and pre-diabetes have also been implicated, as has genetic predisposition. Measurement of sdLDL allows the clinician to get a more comprehensive picture of lipid risk factors and tailor treatment accordingly. Reducing LDL levels will reduce the risk of CVD and MI.

Recommendations:
Results of Lipids should always be interpreted in conjunction with the patient's medical history, clinical presentation and other findings.

NON FASTING LIPID PROFILE includes Total Cholesterol, HDL Cholesterol and calculated non-HDL Cholesterol. It does not include triglycerides and may be best used in patients for whom fasting is difficult.
SERUM BLOOD UREA NITROGEN-Causes of Increased levels
Pre renal
• High protein diet, Increased protein catabolism, GI haemorrhage, Cortisol, Dehydration, CHF Renal
• Renal Failure
Post Renal
• Malignancy, Nephrolithiasis, Prostatism

Causes of decreased levels
• 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
• 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's
• Multiple Sclerosis

Nutritional tips to manage increased Uric acid levels
• Drink plenty of fluids
• Limit animal proteins

DIAGNOSTIC REPORT

SRL Diagnostics

CLIENT CODE : C000000614

CLIENT'S NAME AND ADDRESS :
-

INDIA

PATIENT NAME : - PATIENT ID : RADHF3012422

ACCESSION NO : 0002QL072847 AGE : 75 Years SEX : Female DATE OF BIRTH :

DRAWN : 30-12-2017 14:25 RECEIVED : 30-12-2017 14:27 REPORTED : 10-02-2018 20:00

REFERRING DOCTOR : SELF CLIENT PATIENT ID : 0044

Test Report Status Final	Results	Biological Reference Interval	Units

• High Fibre foods
• Vit C Intake
• Antioxidant rich foods

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.
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.
ELECTROLYTES (NA/K/CL), SERUM-ELECTROLYTES (NA/K/CL), SERUM

Sodium levels are Increased in dehydration, cushing''s syndrome, aldosteronism & decreased in Addison''s disease, hypopituitarism,liver disease. Hypokalemia (low K) is common in vomiting, diarrhea, alcoholism, folic acid deficiency and primary aldosteronism. Hyperkalemia may be seen in end-stage renal failure, hemolysis, trauma, Addison''s disease, metabolic acidosis, acute starvation, dehydration, and with rapid K infusion.Chloride is increased in dehydration, renal tubular acidosis (hyperchloremia metabolic acidosis), acute renal failure, metabolic acidosis associated with prolonged diarrhea and loss of sodium bicarbonate, diabetes insipidus, adrenocortical hyperfuction, salicylate intoxication and with excessive infusion of isotonic saline or extremely high dietary intake of salt.Chloride is decreased in overhydration, chronic respiratory acidosis, salt-losing nephritis, metabolic alkalosis, congestive heart failure, Addisonian crisis, certain types of metabolic acidosis, persistent gastric secretion and prolonged vomiting,
URINALYSIS-Routine urine analysis assists in screening and diagnosis of various metabolic, urological, kidney and liver disorders
Protein: Elevated proteins can be an early sign of kidney disease. Urinary protein excretion can also be temporarily elevated by strenuous exercise, orthostatic proteinuria, dehydration, urinary tract infections and acute illness with fever
Glucose: Uncontrolled diabetes mellitus can lead to presence of glucose in urine. Other causes include pregnancy, hormonal disturbances, liver disease and certain medications.
Ketones: Uncontrolled diabetes mellitus can lead to presence of ketones in urine. Ketones can also be seen in starvation, frequent vomiting, pregnancy and strenuous exercise.
Blood: Occult blood can occur in urine as intact erythrocytes or haemoglobin, which can occur in various urological, nephrological and bleeding disorders.
Leukocytes: An increase in leukocytes is an indication of inflammation in urinary tract or kidneys. Most common cause is bacterial urinary tract infection.
Nitrite: Many bacteria give positive results when their number is high. Nitrite concentration during infection increases with length of time the urine specimen is retained in bladder prior to collection.
pH: The kidneys play an important role in maintaining acid base balance of the body. Conditions of the body producing acidosis/ alkalosis or ingestion of certain type of food can affect the pH of urine.
Specific gravity: Specific gravity gives an indication of how concentrated the urine is. Increased specific gravity is seen in conditions like dehydration, glycosuria and proteinuria while decreased specific gravity is seen in excessive fluid intake, renal failure and diabetes insipidus.
Bilirubin: In certain liver diseases such as biliary obstruction or hepatitis, bilirubin gets excreted in urine.
Urobilinogen: Positive results are seen in liver diseases like hepatitis and cirrhosis and in cases of hemolytic anemia
MAGNESIUM, SERUM-Moderate or severe magnesium deficiency is usually due to losses of magnesium from gastrointestinal tract or kidneys as in vomiting and diarrhoea in former and alcohol, diabetes mellitus (osmotic diuresis), loop diuretics (furosemide) and aminoglycoside antibiotics in latter.

Symptomatic hypermagnesemia is almost always caused by excessive intake with concomitant renal failure, thereby decreasing the ability of the kidneys to excrete excess magnesium.

Magnesium concentration in erythrocytes are approximately three times those of serum. Conversion factors for the units used to express magnesium concentration are:
mg/dl= meq/l x 1.22 = mmol/l x 2.43

End Of Report

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

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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 SRL Directory of services (DOS).
3. SRL confirms that all tests have been performed or assayed with highest quality standards, clinical safety & technical integrity.
4. A requested test might not be performed if:
   a. Specimen received is insufficient or inappropriate specimen quality is unsatisfactory
   b. Incorrect specimen type
   c. Request for testing is withdrawn by the ordering doctor or patient
   d. There is a discrepancy between the label on the specimen container and the name on the test requisition form
5. The results of a laboratory test are dependent on the quality of the sample as well as the assay technology.
6. Result delays could be because of uncontrolled circumstances. e.g. assay run failure.
7. Tests parameters marked by asterisks are excluded from the “scope" of NABL accredited tests. (If laboratory is accredited).
8. Laboratory results should be correlated with clinical information to determine Final diagnosis.
9. Test results are not valid for Medico- legal purposes.
10. In case of queries or unexpected test results please call at SRL customer care (Toll free: 1800-222-000). Post proper investigation repeat analysis may be carried out.

SRL Limited

Fortis Hospital, Sector 62, Phase VIII,
Mohali 160062