





CLIENT CODE: CO00067889

CLIENT'S NAME AND ADDRESS: AAROGYA WELLNESS CENTER

196, HUDA PLOT, NEAR DEVINDER VIHAR, SECTOR-56,

GURGAON 122011 HARYANA INDIA 9891150190 9654240312

SRL, REFERENCE LAB, GP-26, MARUTI INDUSTRIAL ESTATE, UDYOG

VIHAR, SECTOR-18, GURGAON, 122015 HARYANA, INDIA

Tel: 1800-222-000, 1800-102-8282, Fax: CIN -

U74899PB1995PLC045956 Email: connect@srl.in

PATIENT ID : PATIENT NAME: SHRUTI KAUL

ACCESSION NO: 0009TH046439 AGE: 39 Years SEX: Female DATE OF BIRTH:

DRAWN: 20/08/2020 08:00 RECEIVED: 20/08/2020 12: 21 21/08/2020 21:23 REPORTED:

REFERRING DOCTOR: SELF CLIENT PATIENT ID .

Test Report Status Results Biological Reference Interval Units <u>Final</u>

<u>VITAMIN PLUS</u>

25-HYDROXY VITAMIN D, SERUM

25-HYDROXY VITAMIN D 37.1 Deficieny < 20.0 ng/ml

Insufficiency 20.0 - < 30.0Sufficiency 30.0 - 100.0 Toxicity > 100.0

METHOD: CHEMILUMINESCENCE

VITAMIN B12 LEVEL, SERUM

VITAMIN B12 1020 Low 211 - 911 pg/mL

METHOD: CHEMILUMINESCENCE

Interpretation(s)

25-HYDROXY VITAMIN D, SERUM-

Vitamin D is a fat soluble steroid hormone precursor that is mainly produced in the skin by exposure to sunlight. Vitamin D is biologically inert and must undergo two successive hydroxylations in the liver and kidney to become the biologically active 1,25-dihydroxyvitamin D

Vitamin D3and D2 are bound to the vitamin D binding protein and transported to the liver where both are hydroxylated to form vitamin D (25-hydroxyvitamin D).

25-hydroxyvitamin D determines the overall vitamin D status as it is the major storage form of vitamin D in the human body. This primary circulating form of vitamin D is biologically inactive with levels approximately 1000 fold greater than the circulating 1,25-dihydroxyvitamin D. The half life of circulating vitamin D (25-hydroxyvitamin D) is 2-3 weeks.

Clinical significance: Vitamin D is essential for bone health. In children severe deficiency leads to rickets. Milder degrees of insufficiency are believed to cause reduced efficiency in the utilization of dietary calcium. Vitamin D deficiency causes muscle weakness in elderly, the risk of falling has been attributed to the effect of vitamin D on muscle function. Vitamin D deficiency is a common cause of secondary hyperparathyroidism. Elevated PTH levels, especially in elderly vitamin D deficient adults can result in osteomalacia, increased bone turnover, reduced bone mass and risk of bone fractures. Low vitamin D concentrations are also associated with lower bone mineral density. In conjunction with other clinical data, the results may be used as an aid in the assessment of bone metabolism.

Insufficiency has also been linked to diabetes, different forms of cancer, cardiovascular disease, autoimmune diseases and innate immunity

Reference:

1. Holick M. Vitamin D: the underappreciated D-lightful hormone that is important for skeletal and cellular health. Curr Opin Endocrinol Diabetes 2002, 9,87-98
2. Holick MF. Vitamin D deficiency. N Engl J Med 2007, 357, 266-281
3. Tietz Textbook of Clinical Chemistry and Molecular Diagnostics, edited by Carl A Burtis, Edward R.Ashwood, David E Bruns, 4th Edition, Elsevier publication, 2006, 1920-1926

4. Wallach's Interpretation of Diagnostic tests, 9th Edition, Ed Mary A Williamson and L Michael Snyder. Pub Lippincott Williams and Wilkins, 2011, 386-388

BIO CHEMISTRY

CORONARY RISK PROFILE (LIPID PROFILE) **SERUM**

CHOLESTEROL 165 Desirable cholesterol mq/dL

> level: < 200 Borderline high

cholesterol: 200 - 239 High cholesterol : > or = 240

mg/dLTRIGLYCERIDES 142 Normal: < 150

Borderline high: 150 - 199

High: 200 - 499 Very High: > /= 500







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|--|-------------------|------|---|---------|--|
| Test Report Status <u>Final</u> | Results | | Biological Reference Interval | Units | |
| METHOD CONCORDINATION COO DOD METHOD | | | | | |
| METHOD: SPECTROPHOTOMETRY, GPO-POD METHOD | 4.4 | | Low IIDL shalastaral | m a /dl | |
| HDL CHOLESTEROL | 46 | | Low HDL cholesterol < 40 | mg/dL | |
| | | | High HDL cholesterol > or = 60 | | |
| METHOD: SPECTROPHOTOMETRY, HOMOGENEOUS DIRECT ENZYMA | ATIC COLORIMETRIC | | | | |
| DIRECT LDL CHOLESTEROL | 111.00 | High | Adult Optimal: < 100 Near Optimal: 100 - 129 Borderline High: 130 - 159 High: 160 - 189 Very High: > or = 190 | mg/dL | |
| METHOD: SPECTROPHOTOMETRY, ELIMINATION / CATALASE | | | | | |
| NON HDL CHOLESTEROL | 119 | | Desirable: < 130 Above Desirable: 130-159 Borderline High: 160-189 High: 190-219 Very high: > or = 220 | mg/dL | |
| METHOD: CALCULATED PARAMETER | | | 3 0 | | |
| CHOL/HDL RATIO | 3.6 | | Low Risk: 3.3 - 4.4 Average Risk: 4.5 - 7.0 Moderate Risk: 7.1 - 11.0 High Risk: > 11.0 | | |
| METHOD: CALCULATED PARAMETER | 0.4 | | Desirable // and Dist | | |
| LDL/HDL RATIO | 2.4 | | Desirable/Low Risk: 0.5 - 3.0 Borderline/Moderate Risk: 3.1 - 6.0 High Risk: > 6.0 | | |
| METHOD: CALCULATED PARAMETER | | | | | |
| VERY LOW DENSITY LIPOPROTEIN | 28.4 | | < or = 30 | mg/dL | |
| METHOD: CALCULATED PARAMETER | | | | | |
| GLYCOSYLATED HEMOGLOBIN, BLOOD | | | | | |
| GLYCOSYLATED HEMOGLOBIN (HBA1C) METHOD: HIGH PERFORMANCE LIQUID CHROMATOGRAPHY (HPLC) | 5.5 | | Non-diabetic: < 5.7 Pre-diabetics: 5.7 - 6.4 Diabetics: > or = 6.5 ADA Target: 7.0 Action suggested: > 8.0 | % | |
| MEAN PLASMA GLUCOSE | 111 | | < 116.0 | m a /dl | |
| | 1 1 1 | | \ 110.U | mg/dL | |
| LIVER FUNCTION PROFILE. SERUM | | | | | |
| BILIRUBIN, TOTAL | 0.4 | | 0.2 - 1.2 | mg/dL | |
| | | | | | |







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|--|---------|-------------------------------|---------|--|--|
| | | | | | |
| METHOD: SPECTROPHOTOMETRY, VANADATE OXIDATION | | | | | |
| BILIRUBIN, DIRECT | 0.20 | 0.01 - 0.30 | mg/dL | | |
| METHOD: SPECTROPHOTOMETRY, VANADATE OXIDATION | | | | | |
| BILIRUBIN, INDIRECT | 0.2 | 0.1 - 1.0 | mg/dL | | |
| METHOD: CALCULATED PARAMETER | | | | | |
| TOTAL PROTEIN | 6.6 | 5.7 - 8.2 | g/dL | | |
| METHOD: SPECTROPHOTOMETRY, BIURET | | | | | |
| ALBUMIN | 4.4 | 3.2 - 4.8 | g/dL | | |
| METHOD: SPECTROPHOTOMETRY, BROMOCRESOL GREEN(BCG) - DYE BINDING | | | | | |
| GLOBULIN | 2.2 | 2.0 - 4.1 | g/dL | | |
| METHOD: CALCULATED PARAMETER | | | | | |
| ALBUMIN/GLOBULIN RATIO | 2.0 | 1.0 - 2.1 | RATIO | | |
| METHOD: CALCULATED PARAMETER | | | | | |
| ASPARTATE AMINOTRANSFERASE (AST/SGOT) | 13 | < 34.0 | U/L | | |
| METHOD: SPECTROPHOTOMETRY, MODIFIED IFCC | | | | | |
| ALANINE AMINOTRANSFERASE (ALT/SGPT) | 16 | 10 - 49 | U/L | | |
| METHOD: SPECTROPHOTOMETRY, MODIFIED IFCC | | | | | |
| ALKALINE PHOSPHATASE | 86 | 30 - 120 | U/L | | |
| METHOD: SPECTROPHOTOMETRY, IFCC STANDARDIZATION | | | | | |
| GAMMA GLUTAMYL TRANSFERASE (GGT) | 16 | < 38.0 | U/L | | |
| METHOD: SPECTROPHOTOMETRY, MODIFIED IFCC | | | | | |
| LACTATE DEHYDROGENASE | 148 | 120 - 246 | U/L | | |
| METHOD: SPECTROPHOTOMETRY, LACTATE TO PYRUVATE /NICOTINAMIDE ADENINE DINUCLEOTIDE (NAD). | | | | | |
| HOMEOSTATIC MODEL ASSESSMENT (HOMA) | • | | | | |
| 2. SERUM | | | | | |
| *% BETA CELL FUNCTION | 115.7 | Not Established | % | | |
| *% INSULIN SENSITIVITY | 87.1 | Not Established | % | | |
| *HOMA IR (INSULIN RESISTANCE) | 1.15 | < 1.8 | • = | | |
| GLUCOSE, FASTING, PLASMA | | 74 - 99 | m a /dl | | |
| METHOD: SPECTROPHOTOMETRY, HEXOKINASE | 86 | 14 - 77 | mg/dL | | |
| INSULIN | 8.97 | Fasting: | m U /L | | |
| INSULIN | 0.71 | Fasting: | IIIU/L | | |

METHOD: CHEMILUMINESCENCE

Interpretation(s)
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.

3.0 - 25.0

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







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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

GLYCOSYLATED HEMOGLOBIN, BLOOD-GLYCOSYLATED HEMOGLOBIN. BLOOD

Glycation is nonenzymatic addition of sugar residue to amino groups of proteins. HbA1C is formed by the condensation of glucose with n-terminal valine residue of each beta chain of hb a to form an unstable schif base. It is the major fraction, constituting approximately 80% of HbA1. Formation of glycated hemoglobin (GHb) is essentially irreversible and the concentration in the blood depends on both the lifespan of the red blood cells (RBC) (120 days) and the blood glucose concentration. The GHB concentration represents the integrated values for glucose aver the period of 6 to 8 weeks. GHb values are free of day to day glucose fluctuations and are unaffected by recent exercise or food ingestion. Concentration of plasma glucose concentration in GHb depends on the time interval, with more recent values providing a larger contribution than earlier values.

The interpretation of GHb depends on RBC having a normal life span. Patients with hemolytic disease or other conditions with shortened RBC survival exhibit a substantial reduction of GHb. High GHb have been reported in iron deficiency anemia.

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. The absolute risk of retinopathy and nephropathy are directly proportional to the mean of HbA1C. 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 erythropolesis), 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 reactive for the polytic reactive for the polytic reactive for the polytic reactive of the polytic reactive of the polytic reactive for the polytic for 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 system and particleas. Conditions that inclease serum GGT are obstructive liver disease, fighr alcohol consumption and use of enzymer-inducing drugs etc. Serum GGT are obstructive liver disease, fighr alcohol consumption and use of enzymer-inducing drugs etc. Serum GGT are obstructive liver disease, fighr alcohol consumption and use of enzymer-inducing drugs etc. Serum GGT are obstructive liver disease, 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 HOMEOSTATIC MODEL ASSESSMENT (HOMA) 2, SERUM-Homeostatic model assessment (HOMA) 2 - IR

Homeostatic model assessment (HOMA) is a method for assessing beta-cell function and insulin resistance (IR) from fasting glucose and insulin levels. The relationship between glucose and insulin in the basal state reflects the balance between hepatic glucose output and insulin secretion, which is maintained by a feedback loop between the liver and beta-cells. The computer model can be used to determine insulin sensitivity and beta-cell function from paired fasting plasma glucose and specific insulin concentrations across a range of 2.9-43.8 mU/L for insulin and 54.1-450.5 mg/dl for glucose. HOMA2 model accounts of variations in hepatic and peripheral glucose resistance, increases in the insulin secretion curve for plasma glucose concentrations above 10 mmol/L (180 mg/dL)







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and the contribution of circulating proinsulin

HOMA model correlates well with estimates using the euglycemic clamp method. It can be used to track changes in insulin sensitivity and beta-cell function longitudinally in individuals. The model can also be used to indicate whether reduced insulin sensitivity or beta-cell failure predominates. Few research studies have indicated that mortality rate correlates with the HOMA2-IR index in patients with acute myocardial infarction. Moreover, patients with elevated IR have a higher incidence of previous metabolic and cardiovascular events. Therefore, IR may play a short-term prognostic role in patients with AMI. HOMA-IR is a useful predictor of treatment response in type 2 diabetic patients

Homa 2 index = 1.8 is considered as insulin resistance

Limitations

Either HOMA % S or HOMA % B values should not be referred in isolation

The insulin-glucose HOMA model cannot be used to assess beta-cell function in those taking exogenous insulin.

NEPHELOMETRY

HIGH SENSITIVITY C-REACTIVE PROTEIN. SERUM

HIGH SENSITIVITY CRP 5 6 High Low risk for CAD: mg/L

< 1.00

Average risk for CAD: 1.00 - 3.00 High risk for CAD:

> 3.00

METHOD: NEPHELOMETRY

Interpretation(s)
HIGH SENSITIVITY C-REACTIVE PROTEIN, SERUM-

High sensitivity CRP measurements may be used as an independent risk marker for the identification of individuals at risk for future cardiovascular disease. Measurement of hs CRP, when used in conjunction with traditional clinical laboratory evaluation of acute coronary syndromes, may be useful as an independent marker of prognosis for recurrent events, in patients with stable coronary disease or acute coronary syndromes.

When using this assay for risk assessment, patients with persistently unexplained, marked elevation of hs- CRP (> 10mg/l) after repeated testing should be evaluated for non cardiovascular etiologies. In Rheumatic and other inflammatory diseases, value of CRP less than 10 mg/l is considered satisfactory. More than 10 mg/l suggests disease activity. Patients with evidence of active infection, systemic inflammatory processes or trauma should not be tested for cardiovascular disease risk assessment until these conditions have abated

Hs- CRP levels should not be substituted for assessment of traditional cardiovascular risk factors.

Turbidity and particles in the sample may interfere with the determination. Patient samples which contain heterophilic antibodies could react in immunoassays to give a falsely elevated or depressed result.

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

References

1. Teitz textbook of clinical chemistry and Molecular diagnostics, edited by Carl A Burtis, Edward R. Ashrwood, David E Bruns, 4th edition, Elseiver publication, 2006, 962-966

2. Parson TA, Mensah GA, et al. Marker of inflammation and cardiovascular disease: application to clinical and public health practice. Circulation 2003,107,499-511

3. Rheumatoid arthritis disease activity measures: American College of Rheumatology recommendations for use in clinical practice: Jacyln Anderson, Liron Caplin et al, Wiley online, 2012

* * End Of Report* *

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

Dr. Aarti Khanna Nagpal, DNB Head-Haematology

Dr Shakti Aggarwal, MD Head - Clinical Chemistry Dr. Anurag Bansal LAB DIRECTOR







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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

- 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.

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