





CLIENT CODE: C000021846 CLIENT'S NAME AND ADDRESS: SUSHIL SINGAL

SRL LIMITED 74, PASHCHIMI MARG, VASANT VIHAR NEW DELHI, 110057 NEW DELHI, INDIA Tel: 011-42295301,011-42295302, Fax:

CIN - U74899PB1995PLC045956 Email: customercare.palammarg@srl.in

PATIENT NAME: SUSHIL SINGAL PATIENTID: SUSHM968804740

ACCESSION NO: 0062SL001365 AGE: 63 Years sex: Male DATE OF BIRTH:

RECEIVED: 28/12/2019 11:38 DRAWN: 28/12/2019 11:36 REPORTED: 28/12/2019 16:47

REFERRING DOCTOR: SELF CLIENT PATIENT ID :

| Test Report Status <u>Final</u> | Results | | Biological Referenc | e Interval Units |
|--|---------|------|---------------------|------------------|
| COMPLETE CARE TOTAL | | | | |
| BLOOD COUNTS | | | | |
| HEMOGLOBIN | 15.6 | | 13.0 - 17.0 | a /dl |
| METHOD: CYANMETHEMOGLOBIN METHOD | 15.0 | | 13.0 - 17.0 | g/dL |
| RED BLOOD CELL COUNT | 5.43 | | 4.5 - 5.5 | mil/µL |
| METHOD : IMPEDANCE | 5.45 | | 4.0 - 0.0 | 1111/μ |
| WHITE BLOOD CELL COUNT | 6.50 | | 4.0 - 10.0 | thou/µL |
| METHOD: IMPEDANCE | 0.00 | | 1.0 10.0 | 11047 p.E |
| PLATELET COUNT | 154 | | 150 - 410 | thou/µL |
| METHOD: IMPEDANCE | | | | 0.10 d7 p.2 |
| RBC AND PLATELET INDICES | | | | |
| HEMATOCRIT | 45.9 | | 40 - 50 | % |
| METHOD : CALCULATED PARAMETER | 10. 7 | | 10 00 | 70 |
| MEAN CORPUSCULAR VOL | 85.0 | | 83 - 101 | fL |
| METHOD: CALCULATED PARAMETER | | | | |
| MEAN CORPUSCULAR HGB. | 28.8 | | 27.0 - 32.0 | pg |
| MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION METHOD: CALCULATED PARAMETER | 34.1 | | 31.5 - 34.5 | g/dL |
| RED CELL DISTRIBUTION WIDTH | 14.4 | High | 11.6 - 14.0 | % |
| METHOD: CALCULATED PARAMETER | | | | |
| MEAN PLATELET VOLUME | 9.9 | | 6.8 - 10.9 | fL |
| METHOD: CALCULATED PARAMETER | | | | |
| WBC DIFFERENTIAL COUNT | | | | |
| SEGMENTED NEUTROPHILS | 52 | | 40 - 80 | % |
| METHOD : IMPEDENCE / MICROSCOPY | | | | |
| ABSOLUTE NEUTROPHIL COUNT | 3.38 | | 2.0 - 7.0 | thou/µL |
| METHOD: CALCULATED PARAMETER | | | | · |
| EOSINOPHILS | 04 | | 1 - 6 | % |
| METHOD: IMPEDENCE / MICROSCOPY | | | | |
| ABSOLUTE EOSINOPHIL COUNT | 0.26 | | 0.02 - 0.50 | thou/µL |
| METHOD: CALCULATED PARAMETER | | | | |
| LYMPHOCYTES | 39 | | 20 - 40 | % |
| METHOD: IMPEDENCE / MICROSCOPY | | | | |
| ABSOLUTE LYMPHOCYTE COUNT | 2.54 | | 1.0 - 3.0 | thou/µL |
| METHOD: CALCULATED PARAMETER | | | | |
| MONOCYTES | 05 | | 2 - 10 | % |





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METHOD : HEXOKINASE

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| METHOD : IMPEDENCE / MI | CDOCCODY | | | | |
| | | 0.22 | | 0.2 - 1.0 | #la a / l |
| ABSOLUTE MONOCYTE | | 0.32 | | 0.2 - 1.0 | thou/µL |
| METHOD : CALCULATED PAR | | EDTA CMEAD | | | |
| DIFFERENTIAL COUNT | | EDTA SMEAR | | | |
| METHOD : AUTOMATED ANA | | | | | |
| ERYTHRO SEDIMENT | E WHITE CELL COUNTS ARE OUTSIDE ATION RATE, BLOOD | THE NABL ACCREDITED SCOP | PE OF THE | LABORATORY. | |
| SEDIMENTATION RATE | (ESR) | 20 | High | 0 - 14 | mm at 1 hr |
| METHOD : MODIFIED WESTE | ERGREN | | | | |
| PERIPHERAL SMEAR | EXAM, EDTA WHOLE BLO | O D | | | |
| RBC | | PREDOMINANTLY NORMOCYTIC NORMOCHROMIC | | | |
| METHOD: STAINING/MICRO | OSCOPY | | | | |
| WBC | | NORMAL IN NUMBE | R, MOR | PHOLOGY AND DISTRIBUTION | |
| METHOD: STAINING/MICRO | OSCOPY | | | | |
| PLATELETS | | NORMAL IN NUMBER AND MORPHOLOGY. | | | |
| METHOD: STAINING/MICRO | OSCOPY | | | | |
| TOTAL IRON BINDIN | G CAPACITY, SERUM | | | | |
| IRON | | 90 | | 65 - 175 | μg/dL |
| METHOD : FERENE | | | | | |
| TOTAL IRON BINDING | CAPACITY | 446 | | 250 - 450 | μg/dL |
| % SATURATION | | 20 | | 13 - 45 | % |
| METHOD : CALCULATED PAR | RAMETER | | | | |
| GLUCOSE, FASTING, | PLASMA | | | | |
| GLUCOSE, FASTING, PL | LASMA | 173 | High | 82 - 99 | mg/dL |
| | | | - | | 5 |





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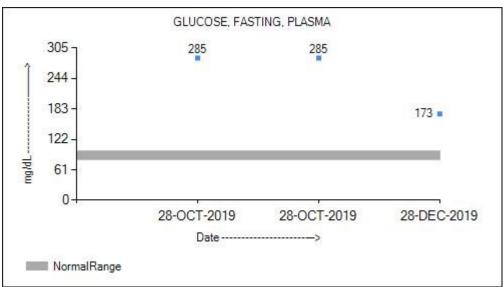
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GLYCOSYLATED HEMOGLOBIN, EDTA WHOLE BLOOD

GLYCOSYLATED HEMOGLOBIN (HBA1C) 7.8 High Non-diabetic: < 5.7 %

Pre-diabetics: 5.7 - 6.4 Diabetics: > or = 6.5 ADA Target: 7.0 Action suggested: > 8.0

Action sugg

MEAN PLASMA GLUCOSE 177.2 High < 116.0 mg/dL

METHOD: CALCULATED PARAMETER





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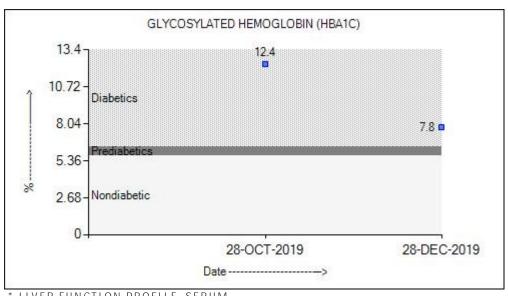
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| Ø | | |
|---------------------------------------|------|-------------|
| * LIVER FUNCTION PROFILE, SERUM | | |
| BILIRUBIN, TOTAL | 0.69 | 0.2 - 1.0 |
| METHOD: JENDRASSIK AND GROFF | | |
| BILIRUBIN, DIRECT | 0.18 | 0.0 - 0.2 |
| METHOD: DIAZOTIZATION | | |
| BILIRUBIN, INDIRECT | 0.51 | 0.1 - 1.0 |
| METHOD: CALCULATED PARAMETER | | |
| TOTAL PROTEIN | 8.1 | 6.4 - 8.2 |
| METHOD: SPECTROPHOTOMETRY | | |
| ALBUMIN | 4.1 | 3.4 - 5.0 |
| METHOD: SPECTROPHOTOMETRY | | |
| GLOBULIN | 4.0 | 2.0 - 4.1 |
| METHOD: CALCULATED PARAMETER | | |
| ALBUMIN/GLOBULIN RATIO | 1.0 | 1.0 - 2.1 |
| METHOD: CALCULATED PARAMETER | | |
| ASPARTATE AMINOTRANSFERASE (AST/SGOT) | 26 | 15 - 37 |
| METHOD: SPECTROPHOTOMETRY | | |
| ALANINE AMINOTRANSFERASE (ALT/SGPT) | 60 | High < 45.0 |
| METHOD: SPECTROPHOTOMETRY | | |
| ALKALINE PHOSPHATASE | 76 | 30 - 120 |
| METHOD: SPECTROPHOTOMETRY | | |





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| 0 | | 5.0 | | 45 05 | |
| GAMMA GLUTAMYL TRA METHOD: SPECTROPHOTOMI | | 58 | | 15 - 85 | U/L |
| LACTATE DEHYDROGEN | ASE | 175 | | 110 - 210 | U/L |
| METHOD : SPECTROPHOTOMI | | | | | |
| * 25 - HYDROXYVITA | | | | | |
| 25 - HYDROXYVITAMIN METHOD : ELECTROCHEMILU | | 32.45 | | Deficiency: < 20.0 Insufficiency: 20.0 - < 30.0 Sufficiency: 30.0 -100.0 Toxicity > 100.0 | ng/mL |
| CALCIUM, SERUM | WINESCENCE | | | | |
| CALCIUM | | 10.4 | High | 8.5 - 10.1 | mg/dL |
| METHOD : O-CRESOLPHTHAL | EIN COMPLEXONE | | | | Ü |
| * VITAMIN B12 LEVE | L, SERUM | | | | |
| VITAMIN B12 | | 320.5 | | 197 - 771 | pg/mL |
| METHOD : ELECTROCHEMILU | | | | | |
| * THYROID PANEL, SI | ERUM | | | | |
| Т3 | | 148.90 | | 80.00 - 200.00 | ng/dL |
| METHOD : ELECTROCHEMILU | MINESCENCE | 0.17 | | E 40 4440 | 7.11 |
| T4 METHOD: ELECTROCHEMILU | AMANECOENIOE | 9.16 | | 5.10 - 14.10 | μg/dl |
| TSH 3RD GENERATION | MINESCENCE | 2.730 | | 0.27 - 4.20 | μIU/mL |
| |)FILE (LIPID PROFILE) | | | 0.27 - 4.20 | μιστιπε |
| CHOLESTEROL | THE (EITHD I KOTTEE) | 150 | | < 200 Desirable | mg/dL |
| | | 150 | | 200 - 239 Borderline High >/= 240 High | mg/uL |
| | IDASE, ESTERASE, PEROXIDASE | 440 | | 450 N | |
| TRIGLYCERIDES | | 119 | | < 150 Normal 150 - 199 Borderline High 200 - 499 High >/= 500 Very High | mg/dL |
| METHOD : ENZYMATIC ASSAY | (| | | | |
| HDL CHOLESTEROL | | 38 | Low | < 40 Low >/= 60 High | mg/dL |
| METHOD : DIRECT MEASURE | - PEG | | | | |
| DIRECT LDL CHOLESTE | ROL | 85 | | < 100 Optimal 100 - 129 Near or above optimus - 159 Borderline High 160 - 189 High >/= 190 Very High | mg/dL mal |





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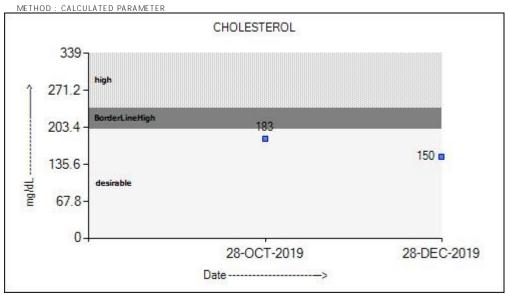
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| | | | | |
| METHOD: DIRECT MEASURE | | | | |
| NON HDL CHOLESTEROL | 112 | Desirable: Less than 130 mg/dL Above Desirable: 130 - 159 Borderline High: 160 - 189 High: 190 - 219 Very high: > or = 220 | | |
| METHOD: CALCULATED PARAMETER | | | | |
| CHOL/HDL RATIO | 4. O | 3.3 - 4.4 Low Risk 4.5 - 7.0 Average Risk 7.1 - 11.0 Moderate Risk > 11.0 High Risk | | |
| METHOD: CALCULATED PARAMETER | | . J | | |
| LDL/HDL RATIO | 2.2 | 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 | 23.8 | = 30.0</math mg/dL | | |







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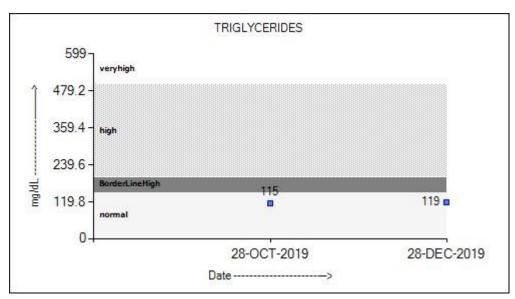
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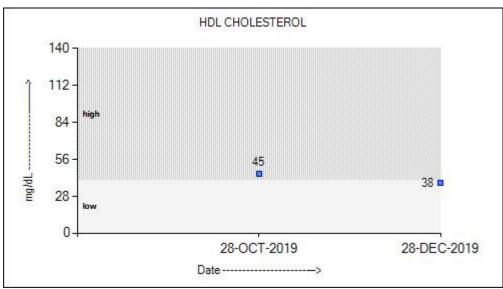
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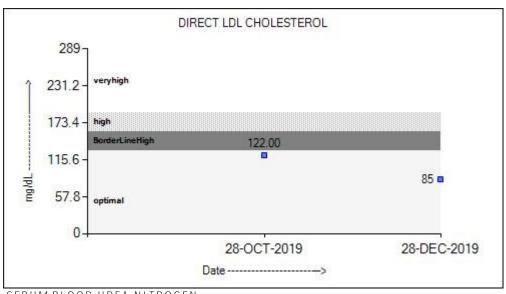
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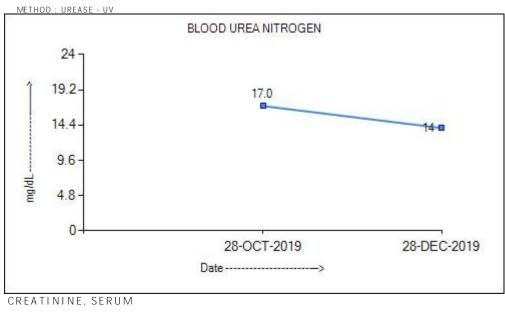
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SERUM BLOOD UREA NITROGEN

BLOOD UREA NITROGEN 8 - 23 mg/dL



CREATININE 0.91 0.80 - 1.30

mg/dL





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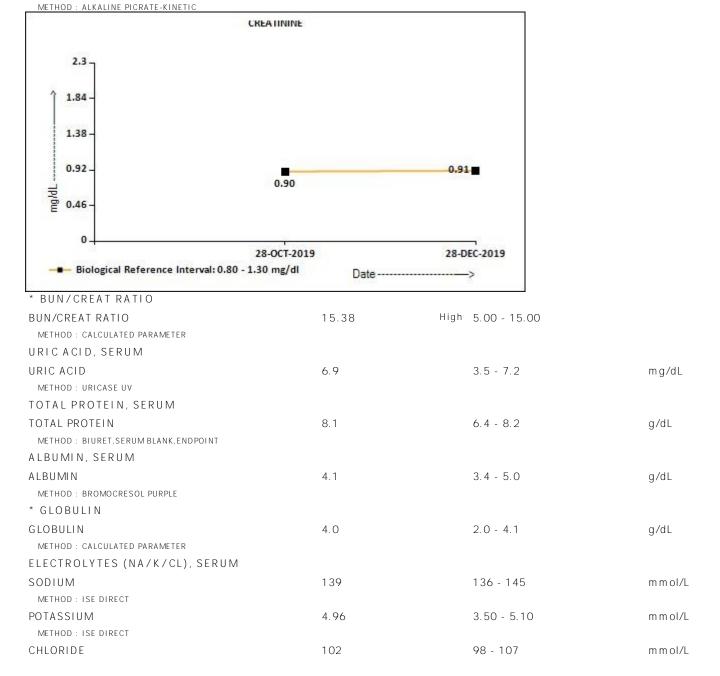
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METHOD: ISE DIRECT

COLOR PALE YELLOW

METHOD : MACROSCOPY

APPEARANCE Clear

METHOD: VISUAL EXAMINATION

PH 7.0 4.7 - 7.5

METHOD: PHINDICATOR AND REFLECTANCE, SPECTROPHOTOMETRY

SPECIFIC GRAVITY 1.015 1.003 - 1.035

METHOD: PKA CHANGE WITH REFLECTANCE, SPECTROPHOTOMETRY

GLUCOSE NOT DETECTED NOT DETECTED

 ${\tt METHOD}: {\tt GLUCOSE} \ {\tt OXIDASE} \ {\tt WITH} \ {\tt REFLECTANCE}, \ {\tt SPECTROPHOTOMETRY}$

PROTEIN NOT DETECTED NOT DETECTED

METHOD: PROTEIN ERROR OF INDICATORS WITH REFLECTANCE, SPECTROPHOTOMETRY

KETONES NOT DETECTED NOT DETECTED

METHOD: ROTHERA'S WITH REFLECTANCE, SPECTROPHOTOMETRY

BLOOD NOT DETECTED NOT DETECTED

METHOD: PEROXIDASE METHOD WITH REFLECTANCE, SPECTROPHOTOMETRY

BILIRUBIN NOT DETECTED NOT DETECTED

METHOD: DIAZOTIZED WITH REFLECTANCE, SPECTROPHOTOMETRY

UROBILINOGEN NORMAL NORMAL

METHOD: EHRLICH REACTION WITH REFLECTANCE, SPECTROPHOTOMETRY

NITRITE NOT DETECTED NOT DETECTED

 ${\tt METHOD:DIAZONIUM\ COMPOUND\ WITH\ REFLECTANCE,\ SPECTROPHOTOMETRY}$

WBC 1-2 0-5 /HPF EPITHELIAL CELLS 1-2 0-5 /HPF RED BLOOD CELLS NOT DETECTED NOT DETECTED /HPF

CASTS NOT DETECTED

CRYSTALS NOT DETECTED

BACTERIA NOT DETECTED NOT DETECTED

Comments

NOTE: - MICROSCOPIC EXAMINATION OF URINE IS PERFORMED BY CENTRIFUGED URINARI SEDIMENT.

MAGNESIUM, SERUM

MAGNESIUM 1.5 Low 1.8 - 2.4 mg/dL

METHOD: METHYLTHYMOL BLUE

Interpretation(s)

BLOOD COUNTS-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.





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RBC AND PLATELET INDICES-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 polkilocytosis, spherocytosis or sickle cells.

Reference:

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

- 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 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





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PATIENT ID : PATIENT NAME: SUSHIL SINGAL SUSHM968804740

ACCESSION NO: 0062SL001365 AGE: 63 Years SEX: Male DATE OF BIRTH:

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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

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.

levels. The following regression equation may be neighbor.

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 first clenching should be avoided before phlebotomy. THYROID PANEL, SERUM-

Trillodo Hyronine 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 (T5H), 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 TOTAL T4 TSH3G TOTAL T3

(µIU/mL) 0.1 - 2.5 0.2 - 3.0 0.3 - 3.0 (ng/dL) 81 - 190 100 - 260 100 - 260 Pregnancy First Trimester (µg/dL) 6.6 - 12.4 2nd Trimester 6.6 - 15.5 6.6 - 15.5 3rd Trimester Below mentioned are the guidelines for age related reference ranges for T3 and T4. T3

(µg/dL) (ng/dL) New Born: 75 - 260 1-3 day: 8.2 - 19.9 1 Week: 6.0 - 15.9

NOTE: TSH concentrations in apparently normal euthyroid subjects are known to be highly skewed, with a strong tailed distribution towards higher TSH values. This is well documented in the pediatric population including the infant age group.

Kindly note: Method specific reference ranges are appearing on the report under biological reference range.

- Reference:
 1. Burtis C.A., Ashwood E. R. Bruns D.E. Teitz textbook of Clinical Chemistry and Molecular Diagnostics, 4th Edition.

1. Burits C.A., Ashwood E. R. Bruns D.E. Teltz 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 (attherosclerosis). High cholestrolly dependent of the place of the page as in page to the page of the page as in page to the page of th

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.





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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 diseaseSTADH.

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
- Problem's during pregnancy, such as seizures (eclampsia)), or high blood pressure caused by pregnancy (preeclampsia)

Lower than normal level may be due to

- Myasthenia GravisMuscular dystrophy

URIC ACID, SERUM-Causes of Increased levels

Dietary

- · High Protein Intake
- Prolonged FastingRapid weight loss.

Gout

Lesch nyhan syndrome.

Гуре 2 DM.

Metabolic syndrome.

Causes of decreased levels • Low Zinc Intake

- OCP's
- · Multiple Sclerosis

Nutritional tips to manage increased Uric acid levels \bullet Drink plenty of fluids

- · Limit animal proteins
- High Fibre foodsVit 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

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





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SEX: Male

Sodium levels are Increased in dehydration, cushing"""'s syndrome, aldosteronism & decreased in Addison"""'s disease, hypopituitarism, liver disease. Hypokalemia (low K) Addison"""'s disease, metabolic acidosis, acute starvation, dehydration, and with rapid K infusion.Chloride is increased in dehydration, 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.

Billirubin: In certain liver diseases such as billary obstruction or hepatitis, billirubin 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:

 $mg/dl = meq/l \times 1.22 = mmol/l \times 2.43$

NEPHELOMETRY

* HIGH SENSITIVITY C-REACTIVE PROTEIN. **SERUM**

HIGH SENSITIVITY CRP 3.71 High 0.0 - 3.0 $m\,g/L$

METHOD: SPECTROPHOTOMETRY

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.





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* * End Of Report* *

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

tempra

Dr. Anu Kundra Senior Consultant Pathologist

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 $% \left(1\right) =\left(1\right) \left(1\right) +\left(1\right) \left(1\right) \left(1\right) +\left(1\right) \left(1\right) \left($
- 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.

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