DIAGNOSTIC REPORT

Cert. No. MC-2947

CLIENT CODE : C000096053

CLIENT'S NAME AND ADDRESS :
JAY SHREE PATIENT CARE CENTRE
SHOP NO.24, SHAMINA MARKET, OPPOSITE K.G.M.C., NEW OPD BUILDING CHOWK,

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UTTAR PRADESH, INDIA
Tel : 9111591115, Fax : 0522 - 406 2980
CIN - U74899PB1995PLC045956
Email : customercare.lucknow@srl.in

PATIENT NAME : JUNIOR ANAND GUPTA

PATIENT ID : JUNIM967758620

ACCESSION NO : 0024UG008438 AGE : 47 Years SEX : Male DATE OF BIRTH :

DRAWN : 19/07/2021 12:16 RECEIVED : 19/07/2021 13:14 REPORTED : 19/07/2021 17:08

REFERRING DOCTOR : SELF CLIENT PATIENT ID :

Test Report Status Final	Results		Biological Reference Interval	Units
COMPLETE CARE PREMIUM WITH SMART REPORT				
BLOOD COUNTS				
HEMOGLOBIN	13.7		13.0 - 17.0	g/dL
METHOD : SPECTROPHOTOMETRY				
RED BLOOD CELL COUNT	4.83		4.5 - 5.5	mil/µL
METHOD : ELECTRONIC IMPEDANCE				
WHITE BLOOD CELL COUNT	6.00		4.0 - 10.0	thou/µL
METHOD : ELECTRONIC IMPEDANCE				
PLATELET COUNT	197		150 - 410	thou/µL
RBC AND PLATELET INDICES				
HEMATOCRIT	39.3	Low	40 - 50	%
METHOD : ELECTRONIC IMPEDANCE/CALCULATION				
MEAN CORPUSCULAR VOL	81.0	Low	83 - 101	fL
METHOD : CALCULATED PARAMETER				
MEAN CORPUSCULAR HGB.	28.3		27.0 - 32.0	pg
METHOD : CALCULATED PARAMETER				
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION	34.8	High	31.5 - 34.5	g/dL
METHOD : CALCULATED PARAMETER				
RED CELL DISTRIBUTION WIDTH	14.2	High	11.6 - 14.0	%
MEAN PLATELET VOLUME	7.8		6.8 - 10.9	fL
WBC DIFFERENTIAL COUNT - NLR				
SEGMENTED NEUTROPHILS	48		40 - 80	%
METHOD : MICROSCOPIC EXAMINATION				
ABSOLUTE NEUTROPHIL COUNT	2.88		2.0 - 7.0	thou/µL
METHOD : CALCULATED PARAMETER				
LYMPHOCYTES	48	High	20 - 40	%
ABSOLUTE LYMPHOCYTE COUNT	2.88		1.0 - 3.0	thou/µL
METHOD : CALCULATED PARAMETER				
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	1			
EOSINOPHILS	02		1 - 6	%
ABSOLUTE EOSINOPHIL COUNT	0.12		0.02 - 0.50	thou/µL
METHOD : CALCULATED PARAMETER				
MONOCYTES	02		2 - 10	%
ABSOLUTE MONOCYTE COUNT	0.12	Low	0.2 - 1.0	thou/µL
METHOD : CALCULATED PARAMETER				

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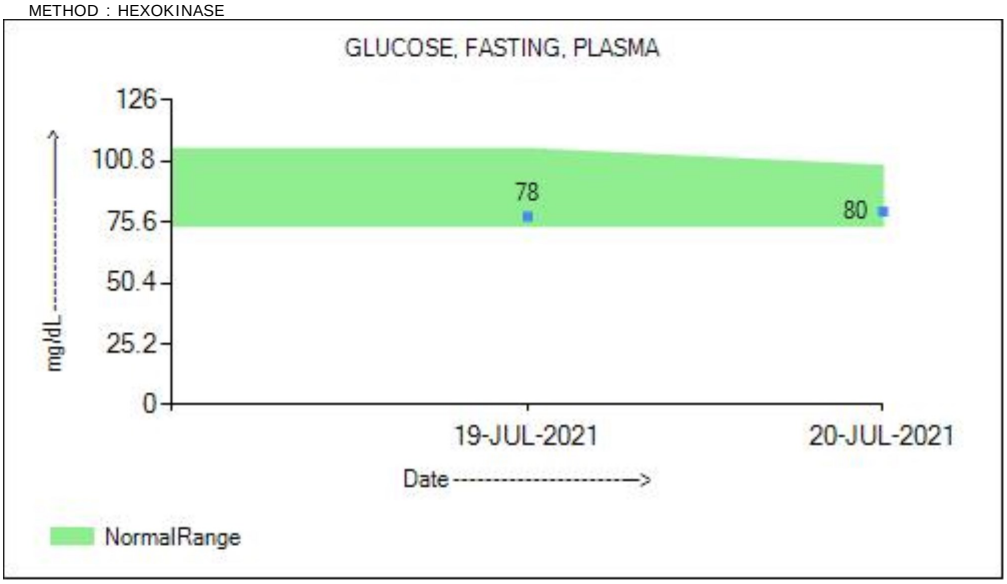
BASOPHILS	00	0 - 2	%
BAND (STAB) CELLS	00	0 - 5	%
DIFFERENTIAL COUNT PERFORMED ON:	EDTA SMEAR		
ERYTHRO SEDIMENTATION RATE, BLOOD			
SEDIMENTATION RATE (ESR)	06	0 - 14	mm at 1 hr
METHOD : WESTERGREN METHOD			
PERIPHERAL SMEAR EXAM, EDTA WHOLE BLOOD			
RBC	PREDOMINANTLY NORMOCYTIC NORMOCHROMIC		
WBC	WBC ARE NORMAL IN NUMBER WITH INCREASED LYMPHOCYTIC PERCENTAGE.		
PLATELETS	PLATELETS ARE ADEQUATE IN NUMBER		
IMPRESSION	NORMOCYTIC NORMOCHROMIC BLOOD PICTURE		
* GLUCOSE, FASTING, PLASMA			
GLUCOSE, FASTING, PLASMA	78	74 - 106	mg/dL
METHOD : HEXOKINASE			



* GLYCOSYLATED HEMOGLOBIN, EDTA WHOLE BLOOD			
GLYCOSYLATED HEMOGLOBIN (HBA1C)	5.4	Non-diabetic: < 5.7 Pre-diabetics: 5.7 - 6.4 Diabetics: > or = 6.5 ADA Target: 7.0 Action suggested: > 8.0	%
METHOD : HPLC			
MEAN PLASMA GLUCOSE	108.3	< 116.0	mg/dL

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METHOD : CALCULATED PARAMETER				
* HIGH SENSITIVITY C-REACTIVE PROTEIN, SERUM				
HIGH SENSITIVITY CRP	4.92	High	Relative risk for CVD: < 1.0 Low Risk 1.0 - 3.0 Average Risk > 3.0 High Risk	mg/L
HEPATITIS B SURFACE ANTIGEN, SERUM				
HEPATITIS B SURFACE ANTIGEN	NON REACTIVE		NON REACTIVE	
PATIENT VALUE	0.33		< 0.90 (Non Reactive) > or = 1.00 (Reactive)	S/CO
* LIVER FUNCTION PROFILE, SERUM				
BILIRUBIN, TOTAL	0.30		UPTO 1.2	mg/dL
METHOD : DIAZONIUM ION, BLANKED (ROCHE)				
BILIRUBIN, DIRECT	0.13		0.00 - 0.30	mg/dL
METHOD : DIAZOTIZATION				
BILIRUBIN, INDIRECT	0.17		0.00 - 0.60	mg/dL
METHOD : CALCULATED PARAMETER				
TOTAL PROTEIN	6.5		6.4 - 8.3	g/dL
METHOD : BIURET,SERUM BLANK,ENDPOINT				
ALBUMIN	4.2		3.97 - 4.94	g/dL
METHOD : BROMOCRESOL GREEN				
GLOBULIN	2.3		2.0 - 4.0 Neonates - Pre Mature: 0.29 - 1.04	g/dL
METHOD : CALCULATED PARAMETER				
ALBUMIN/GLOBULIN RATIO	1.8		1.0 - 2.0	RATIO
METHOD : CALCULATED PARAMETER				
ASPARTATE AMINOTRANSFERASE (AST/SGOT)	18		0 - 40	U/L
METHOD : UV WITHOUT P5P				
ALANINE AMINOTRANSFERASE (ALT/SGPT)	17		0 - 41	U/L
METHOD : UV WITHOUT P5P				
ALKALINE PHOSPHATASE	70		40 - 129	U/L
METHOD : PNPP, AMP BUFFER-IFCC				
GAMMA GLUTAMYL TRANSFERASE (GGT)	17		8 - 61	U/L
METHOD : G-GLUTAMYL-CARBOXY-NITROANILIDE-IFCC				
LACTATE DEHYDROGENASE	183		135 - 225	U/L
METHOD : L TO P, IFCC				
* TOTAL IRON BINDING CAPACITY, SERUM				
IRON	105		59 - 158	µg/dL

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METHOD : FEROZINE - NO DEPROTEINIZATION				
TOTAL IRON BINDING CAPACITY	179	Low	250 - 450	µg/dL
METHOD : CALCULATED PARAMETER				
% SATURATION	59	High	13 - 45	%
* FERRITIN, SERUM				
FERRITIN	112.3		30 - 400	ng/mL
25 - HYDROXYVITAMIN D, SERUM				
25 - HYDROXYVITAMIN D	28.82	Low	Deficiency: < 20.0 Insufficiency: 20.0 - < 30.0 Sufficiency: 30.0 -100.0 Toxicity > 100.0	ng/mL
* CALCIUM, SERUM				
CALCIUM	8.4	Low	8.6 - 10.0	mg/dL
METHOD : BAPTA				
* VITAMIN B12 LEVEL, SERUM				
VITAMIN B12	371.8		Normal adults: 197-771	pg/mL
CORONARY RISK PROFILE (LIPID PROFILE), SERUM				
CHOLESTEROL	198		< 200 Desirable 200 - 239 Borderline High >/= 240 High	mg/dL
METHOD : CHOLESTEROL OXIDASE, ESTERASE,PEROXIDASE				
TRIGLYCERIDES	185	High	< 150 Normal 150 - 199 Borderline High 200 - 499 High >/= 500 Very High	mg/dL
METHOD : ENZYMATIC, END POINT				
HDL CHOLESTEROL	44		< 40 Low >/=60 High	mg/dL
METHOD : DIRECT MEASURE POLYMER-POLYANION				
DIRECT LDL CHOLESTEROL	127	High	< 100 Optimal 100 - 129 Near or above optimal 130 - 160 Borderline High 161 - 189 High >/= 190 Very High	mg/dL
METHOD : DIRECT MEASURE				
NON HDL CHOLESTEROL	154	High	Desirable: Less than 130 Above Desirable: 130 - 159 Borderline High: 160 - 189 High: 190 - 219 Very high: > or = 220	mg/dL
METHOD : CALCULATED PARAMETER				

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CHOL/HDL RATIO	4.5	High	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				
LDL/HDL RATIO	2.9		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	37.0	High	Desirable value : 10 - 35	mg/dL
METHOD : CALCULATED PARAMETER				
* SERUM BLOOD UREA NITROGEN				
BLOOD UREA NITROGEN	10		6 - 20	mg/dL
METHOD : UREASE - UV				
* CREATININE, SERUM				
CREATININE	0.90		0.70 - 1.20	mg/dL
METHOD : ALKALINE PICRATE-KINETIC				
BUN/CREAT RATIO				
BUN/CREAT RATIO	11.11		5.00 - 15.00	
METHOD : CALCULATED PARAMETER				
* URIC ACID, SERUM				
URIC ACID	4.0		3.4 - 7.0	mg/dL
METHOD : URICASE, COLORIMETRIC				
* TOTAL PROTEIN, SERUM				
TOTAL PROTEIN	6.5	Low	6.6 - 8.7	g/dL
METHOD : BIURET,SERUM BLANK,ENDPOINT				
* ALBUMIN, SERUM				
ALBUMIN	4.2		3.97 - 4.94	g/dL
METHOD : BROMOCRESOL GREEN				
* GLOBULIN				
GLOBULIN	2.3		2.0 - 4.0 Neonates - Pre Mature: 0.29 - 1.04	g/dL
METHOD : CALCULATED PARAMETER				
ELECTROLYTES (NA/K/CL), SERUM				
SODIUM	140		136 - 145	mmol/L
METHOD : ISE INDIRECT				
POTASSIUM	4.29		3.5 - 5.1	mmol/L

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METHOD : ISE INDIRECT				
CHLORIDE	106		98 - 107	mmol/L
METHOD : ISE INDIRECT				
* FOLIC ACID, SERUM				
FOLIC ACID	13.70		3.89 - 26.80	ng/mL
TOTAL IGE, SERUM				
TOTAL IGE	105.6	High	< 100.0	IU/mL
* FREE TRIIODOTHYRONINE (FT3), SERUM				
FREE TRIIODOTHYRONINE (FT3)	2.83		2.0 - 4.4	pg/mL
* FREE THYROXINE (FT4), SERUM				
FREE THYROXINE (FT4)	0.92	Low	0.93 - 1.70	ng/dL
* TSH 3RD GENERATION ULTRA(TSH3 - UL), SERUM				
TSH 3RD GENERATION	1.883		0.55 - 4.78	µIU/mL
METHOD : CHEMILUMINESCENCE				

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.
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.
WBC DIFFERENTIAL COUNT - NLR-
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.
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"
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.
"Targets should be individualized More or less stringent glycemic goals may be appropriate for individual patients. Goals should be individualized based on duration of diabetes, age/life expectancy, comorbid conditions, known CVD or advanced microvascular complications, hypoglycemia unawareness, and individual patient considerations.''

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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.

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.
HEPATITIS B SURFACE ANTIGEN, SERUM-Hepatitis B is caused by infection with HBV, a enveloped DNA agent that is classified as hepadnavirus.This test detects the presence of viral surface antigen (HbsAg) in serum sample and is indicative of an active HBV infection, either acute or chronic.

Test Utility:
HbsAg is the first serologic marker appearing in the serum 6-16 weeks following hepatitis B viral infection. In typical HBV infection, HBsAg will be detected 2-4 weeks before the liver enzyme levels (ALT) become abnormal and 3-5 weeks before patient develops jaundice.In acute cases HbsAg usually disappears 1-2 months after the onset of symptoms.Persistence of HbsAg for more than 6 months indicates development of either a chronic carrier state or chronic liver disease.The presence of HbsAg is frequently associated with infectivity. HbsAg when accompanied by Hepatitis Be antigen and/or hepatitis B viral DNA almost always indicates infectivity.

Limitations:
- For diagnostic purposes, results should be used in conjunction with patient history and other hepatitis markers for diagnosis of acute or chronic infection. If the antibody results are inconsistent with clinical evidence, additional testing is suggested to confirm the result.
- HBsAg detection will only indicate the presence of surface antigens in the serum and should not be used as the sole criteria for diagnosis, staging or monitoring of HBV infection This test may be negative during ""window period"" i.e. after disappearance of anti-HBs.
- The current assay being a highly sensitive test , may yield a small percentage of false positive reports. Hence all HbsAg positive specimens should be confirmed with an assay based upon Neutralisation of Human anti Hepatitis B Surface antibody.
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
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.

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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.
FERRITIN, SERUM-Ferritin is a high-molecular-weight protein that contains approximately 20% iron. It occurs normally in almost all tissues of the body but especially in hepatocytes and reticuloendothelial cells, where it serves as an iron reserve. When needed, the iron molecules are released from the apoferritin shell and bind to transferrin, the circulating plasma protein that transports iron to the erythropoietic cells.

A low serum ferritin value is thought to be the best laboratory indicator of iron depletion. Virtually all patients with low serum iron and low ferritin have iron deficiency. Serum Ferritin concentration, when considered with other factors such as serum iron, iron-binding capacity and tissue iron stores is valuable in the diagnosis of iron deficiency anemia, anemia of chronic infection and conditions such as thalassemia and hemochromatosis that are associated with iron overload. It is particularly useful in distinguishing between iron-deficiency anemia (serum ferritin levels diminished) and "anemia of chronic disease" (serum ferritin levels usually normal or elevated).

Ferritin is an acute phase reactant. It can be found to be elevated in the following conditions and do not reflect actual body iron stores: 1.Inflammation 2.Significant tissue destruction 3.Liver diseases 4.Malignancies such as acute leukemia and Hodgkin''''''''''''''''s disease 5.Therapy with iron supplements.

Interferences:
Heterophilic antibodies in human serum can react with reagent immunoglobulins, interfering with in vitro immunoassays. Patients routinely exposed to animals or to animal serum products can be prone to this interference and anomalous values may be observed.
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.
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,

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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
• 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,

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salt-losing nephritis, metabolic alkalosis, congestive heart failure, Addisonian crisis, certain types of metabolic acidosis, persistent gastric secretion and prolonged vomiting,
FOLIC ACID, SERUM-Folates are compounds of pteroylglutamic acid (PGA) that function as coenzymes in metabolic reactions involving the transfer of single-carbon units from a donor to a recipient compound. Folate, with vitamin B12, is essential for DNA synthesis, which is required for normal red blood cell maturation. Human obtain folate from dietary sources including fruits, green and leafy vegetables, yeast, and organ meats. Folate is absorbed through the small intestine and stored in the liver.

Low folate intake, malabsorption as result of gastrointestinal diseases, pregnancy, and drugs such as phenytoin are causes of folate deficiency. Folate deficiency is also associated with chronic alcoholism. Folate and vitamin B12 deficiency impair DNA synthesis, causing macrocytic anemias. These anemias are characterized by abnormal maturation of red blood cell precursors in the bone marrow, the presence of megaloblasts, and decreased red blood cell survival.

Since both folate and vitamin B12 deficiency can cause macrocytic anemia, appropriate treatment depends on the differential diagnosis of the deficiency. Serum folate measurement provides an early index of folate status. However, folate is much more concentrated in red blood cells than in serum so the red blood cell folate measurement more closely reflects tissue stores. Red blood cell folate concentration is considered the most reliable indicator of folate status.

Methotrexate and Leucovorin interfere with folate measurement because these drugs cross-react with folate binding proteins.
TOTAL IGE, SERUM-Introduction: The ImmunoCAP total IgE measures the total quantity of circulating IgE in human serum samples.

Test Utility:
• For allergy testing : IgE antibodies appear as a result of sensitization to allergens, and the measurement of circulating total IgE assists the clinical diagnosis of IgE-mediated allergic disorders. Elevated levels of circulating total IgE are usually seen in atopic eczema ,60% of patients with extrinsic asthma, and about 30% cases of hay fever.
• Parasitic diseases: Values may be elevated in ascariasis, visceral larva migrans, hookworm disease, schistosomiasis, echinococcus infestation).
• Diagnosis of monoclonal IgE myeloma.
• Diagnosis of bronchopulmonary aspergillosis.
• Total IgE may be decreased in hereditary deficiencies, acquired immunodeficiency, ataxia telangiaectasia & non-IgE myeloma.
Limitation: A normal level of IgE does not eliminate the possibility of allergy, hence test is not recommended as a stand-alone screen. Value is influenced by type of allergen, duration of stimulation, presence of symptoms, hyposensitization treatment.
FREE TRIIODOTHYRONINE (FT3), SERUM-The guidelines for age related reference ranges for FT3.

Cord Blood	1.5 - 3.9 pg/mL
Children	2.1 - 4.4 pg/mL
Pregnancy	2.0 - 3.8 pg/mL

FREE THYROXINE (FT4), SERUM-The guidlines for age related reference ranges for FT4.

New Born (1-4 days)	2.2 - 5.3 ng/dL
Children	0.8 - 2.7 ng/dL
Pregnancy	
1st Trimester	0.7 - 2.0 ng/dL
2nd & 3rd Trimester	0.5 - 1.6 ng/dL

TSH 3RD GENERATION ULTRA(TSH3 - UL), SERUM-Comment: The Biological Reference Interval of TSH-3rd Generation Ultra [TSH3-UL] is not established for age less than 2 years.

Below mentioned are the guidelines for Pregnancy related reference ranges for TSH.

Levels in Pregnancy	TSH (µIU/mL)
First Trimester	0.1 - 2.5
2nd Trimester	0.2 – 3.0
3rd Trimester	0.3 – 3.0

BIO CHEMISTRY

* MAGNESIUM, SERUM

MAGNESIUM	1.8	1.6 - 2.6	mg/dL

METHOD : XYLIDYL BLUE

Interpretation(s)

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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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Dr. Nidhi Awasthi, MD
Pathologist

Dr Abhishant Pandey, MD
Pathologist
LAB HEAD

Dr. Richa Tripathi
Consultant Microbiologist

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