DIAGNOSTIC REPORT

Cert. No. MC-2015

CLIENT CODE : C000097359

CLIENT'S NAME AND ADDRESS :
FPSC HK PATHOLOGY
SHOP NO.3, DDA MARKET, C-8, VASANT KUNJ

NEW DELHI 110070
DELHI INDIA
9818105133

SRL LIMITED
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 NAME : GAGANMEET SINGH PATIENT ID : GAGAM183186200

ACCESSION NO : 0009TJ067147 AGE : 34 Years SEX : Male DATE OF BIRTH :

DRAWN : 25/10/2020 12:49 RECEIVED : 25/10/2020 16:00 REPORTED : 25/10/2020 19:48

REFERRING DOCTOR : SELF CLIENT PATIENT ID :

Test Report Status Preliminary	Results	Biological Reference Interval	Units
COMPLETE CARE PREMIUM WITH SMART REPORT			
BLOOD COUNTS			
HEMOGLOBIN	14.0	13.0 - 17.0	g/dL
METHOD : PHOTOMETRIC MEASUREMENT			
RED BLOOD CELL COUNT	4.91	4.5 - 5.5	mil/µL
METHOD : COULTER IMPEDENCE PRINCIPLE			
WHITE BLOOD CELL COUNT	5.20	4.0 - 10.0	thou/µL
METHOD : COULTER IMPEDENCE PRINCIPLE			
PLATELET COUNT	267	150 - 410	thou/µL
METHOD : IMPEDENCE / PLATELET HISTOGRAM			
RBC AND PLATELET INDICES			
HEMATOCRIT	43.0	40 - 50	%
METHOD : CALCULATED PARAMETER			
MEAN CORPUSCULAR VOL	87.5	83.0 - 101.0	fL
METHOD : DERIVED PARAMETER			
MEAN CORPUSCULAR HGB.	28.4	27.0 - 32.0	pg
METHOD : CALCULATED PARAMETER			
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION	32.5	31.5 - 34.5	g/dL
METHOD : CALCULATED PARAMETER			
RED CELL DISTRIBUTION WIDTH	13.6	11.6 - 14.0	%
METHOD : DERIVED PARAMETER			
MEAN PLATELET VOLUME	8.2	6.8 - 10.9	fL
METHOD : DERIVED PARAMETER			
WBC DIFFERENTIAL COUNT			
SEGMENTED NEUTROPHILS	61	40 - 80	%
METHOD : VCS TECHNOLOGY/ MICROSCOPY			
ABSOLUTE NEUTROPHIL COUNT	3.17	2.0 - 7.0	thou/µL
METHOD : CALCULATED PARAMETER			
EOSINOPHILS	1	1 - 6	%
METHOD : VCS TECHNOLOGY/ MICROSCOPY			
ABSOLUTE EOSINOPHIL COUNT	0.05	0.02 - 0.50	thou/µL
METHOD : CALCULATED PARAMETER			
LYMPHOCYTES	31	20 - 40	%
METHOD : VCS TECHNOLOGY/ MICROSCOPY			
ABSOLUTE LYMPHOCYTE COUNT	1.61	1.0 - 3.0	thou/µL

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METHOD : CALCULATED PARAMETER			
MONOCYTES	7	2 - 10	%
METHOD : VCS TECHNOLOGY/ MICROSCOPY			
ABSOLUTE MONOCYTE COUNT	0.36	0.2 - 1.0	thou/µL
METHOD : CALCULATED PARAMETER			
BASOPHILS	0	< 1 - 2	%
METHOD : VCS TECHNOLOGY/ MICROSCOPY			
ABSOLUTE BASOPHIL COUNT	0.05	0.02 - 0.10	thou/µL
METHOD : CALCULATED PARAMETER			
DIFFERENTIAL COUNT PERFORMED ON:	EDTA SMEAR		
METHOD : AUTOMATED ANALYZER / MICROSCOPY			
DISCLAIMER: THE ABSOLUTE WHITE CELL COUNTS ARE OUTSIDE THE NABL ACCREDITED SCOPE OF THE LABORATORY.			
ERYTHRO SEDIMENTATION RATE, BLOOD			
SEDIMENTATION RATE (ESR)	5	0 - 14	mm at 1 hr
METHOD : AUTOMATED (PHOTOMETRICAL CAPILLARY STOPPED FLOW KINETIC ANALYSIS)			
PERIPHERAL SMEAR EXAM, EDTA WHOLE BLOOD			
RBC	PREDOMINANTLY NORMOCYTIC NORMOCHROMIC		
METHOD : MICROSCOPIC EXAMINATION			
WBC	NORMAL IN NUMBER, MORPHOLOGY AND DISTRIBUTION		
METHOD : MICROSCOPIC EXAMINATION			
PLATELETS	NORMAL IN NUMBER AND MORPHOLOGY.		
METHOD : MICROSCOPIC EXAMINATION			
GLUCOSE, FASTING, PLASMA			
GLUCOSE, FASTING, PLASMA	85	74 - 99	mg/dL
METHOD : SPECTROPHOTOMETRY, HEXOKINASE			
GLYCOSYLATED HEMOGLOBIN, EDTA WHOLE BLOOD			
GLYCOSYLATED HEMOGLOBIN (HBA1C)	5.3	Non-diabetic: < 5.7 Pre-diabetics: 5.7 - 6.4 Diabetics: > or = 6.5 ADA Target: 7.0 Action suggested: > 8.0	%
METHOD : HIGH PERFORMANCE LIQUID CHROMATOGRAPHY (HPLC)			
MEAN PLASMA GLUCOSE	105.4	< 116.0	mg/dL
HIGH SENSITIVITY C-REACTIVE PROTEIN, SERUM			
HIGH SENSITIVITY CRP	0.5	Low risk for CAD: < 1.00 Average risk for CAD: 1.00 - 3.00 High risk for CAD: > 3.00	mg/L

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Test Report Status Preliminary	Results	Biological Reference Interval	Units
METHOD : NEPHELOMETRY			
CORTISOL, SERUM			
CORTISOL	15.97	Morning (7 – 9 a.m.): 5.27 – 22.45 Afternoon (3 – 5 p.m.): 3.44 – 16.76	ug/dL
METHOD : CHEMILUMINESCENCE			
LIVER FUNCTION PROFILE, SERUM			
BILIRUBIN, TOTAL	0.9	0.2 - 1.2	mg/dL
METHOD : SPECTROPHOTOMETRY, VANADATE OXIDATION			
BILIRUBIN, DIRECT	0.3	0.01 - 0.30	mg/dL
METHOD : SPECTROPHOTOMETRY, VANADATE OXIDATION			
BILIRUBIN, INDIRECT	0.60	0.1 - 1.0	mg/dL
METHOD : CALCULATED PARAMETER			
TOTAL PROTEIN	7.0	5.7 - 8.2	g/dL
METHOD : SPECTROPHOTOMETRY, BIURET			
ALBUMIN	4.5	3.2 - 4.8	g/dL
METHOD : SPECTROPHOTOMETRY, BROMOCRESOL GREEN(BCG) - DYE BINDING			
GLOBULIN	2.5	2.0 - 4.1	g/dL
METHOD : CALCULATED PARAMETER			
ALBUMIN/GLOBULIN RATIO	1.8	1.0 - 2.1	RATIO
METHOD : CALCULATED PARAMETER			
ASPARTATE AMINOTRANSFERASE (AST/SGOT)	26	< 34.0	U/L
METHOD : SPECTROPHOTOMETRY,MODIFIED IFCC			
ALANINE AMINOTRANSFERASE (ALT/SGPT)	40	10 - 49	U/L
METHOD : SPECTROPHOTOMETRY,MODIFIED IFCC			
ALKALINE PHOSPHATASE	84	30 - 120	U/L
METHOD : SPECTROPHOTOMETRY, IFCC STANDARDIZATION			
GAMMA GLUTAMYL TRANSFERASE (GGT)	10	< 73.0	U/L
METHOD : SPECTROPHOTOMETRY,MODIFIED IFCC			
LACTATE DEHYDROGENASE	183	120 - 446	U/L
METHOD : SPECTROPHOTOMETRY, LACTATE TO PYRUVATE /NICOTINAMIDE ADENINE DINUCLEOTIDE (NAD).			
TOTAL IRON BINDING CAPACITY, SERUM			
IRON	85	65 - 175	µg/dL
METHOD : SPECTROPHOTOMETRY,FERROZINE			
TOTAL IRON BINDING CAPACITY	261	250 - 425	µg/dL
METHOD : SPECTROPHOTOMETRY,SEQUENTIAL RELEASE AND UPTAKE OF IRON			
% SATURATION	32.6	13 - 45	%
METHOD : CALCULATED PARAMETER			

DIAGNOSTIC REPORT

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COLLEGE of AMERICAN PATHOLOGISTS

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SRL Diagnostics

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Test Report Status Preliminary	Results	Biological Reference Interval	Units
FERRITIN, SERUM			
FERRITIN	234.8	22 - 322	ng/mL
METHOD : CHEMILUMINESCENCE			
MICROALBUMIN, URINE			
SPOT URINE MICROALBUMIN	<3.0	< 30	mg/L
METHOD : PEG-ENHANCED IMMUNOTURBIDIMETRIC			
CREATININE, URINE	21	UNDEFINED	mg/dL
METHOD : JAFFE, ALKALINE PICRATE, KINETIC WITH BLANK RATE CORRECTION			
MICROALBUMIN/ CREATININE RATIO	NOT CALCULATED	NORMAL <30.0 MICROALBUMINURIA 30.0-299.0 CLINICAL ALBUMINURIA =or>300.0	mg/g creat
METHOD : CALCULATED PARAMETER			

Comments

NOTE: URINE MICROALBUMIN VALUE RECHECKED & IS REPORTED AS < 3.0mg/L.URINE MICROALBUMIN/CREATININE RATIO IS A CALCULATED PARAMETER AND HENCE REPORTED AS NOT CALCULATED.

25 - HYDROXYVITAMIN D, SERUM			
25 - HYDROXYVITAMIN D	52.26	Deficieny < 20.0 Insufficiency 20.0 - < 30.0 Sufficiency 30.0 - 100.0 Toxicity >100.0	ng/mL
METHOD : CHEMILUMINESCENCE			
CALCIUM, SERUM			
CALCIUM	9.0	8.3 - 10.6	mg/dL
METHOD : SPECTROPHOTOMETRY, CALORIMETRIC METHOD			
VITAMIN B12 LEVEL, SERUM			
VITAMIN B12	419.0	211 - 911	pg/mL
METHOD : CHEMILUMINESCENCE			
CORONARY RISK PROFILE (LIPID PROFILE), SERUM			
CHOLESTEROL	158	Desirable cholesterol level : < 200 Borderline high cholesterol : 200 - 239 High cholesterol : > or = 240	mg/dL
TRIGLYCERIDES	90	Normal: < 150 Borderline high : 150 - 199 High: 200 - 499 Very High : > /= 500	mg/dL
METHOD : SPECTROPHOTOMETRY,GPO-POD METHOD			

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Test Report Status Preliminary	Results		Biological Reference Interval	Units
HDL CHOLESTEROL	49		Low HDL cholesterol < 40 High HDL cholesterol > or = 60	mg/dL
METHOD : SPECTROPHOTOMETRY, HOMOGENEOUS DIRECT ENZYMATIC COLORIMETRIC				
DIRECT LDL CHOLESTEROL	104.00	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	109		Desirable : < 130 Above Desirable : 130 -159 Borderline High : 160 - 189 High : 190 - 219 Very high : > or = 220	mg/dL
METHOD : CALCULATED PARAMETER				
CHOL/HDL RATIO	3.2	Low	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				
LDL/HDL RATIO	2.1		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	18.0		< or = 30	mg/dL
METHOD : CALCULATED PARAMETER				
SERUM BLOOD UREA NITROGEN				
BLOOD UREA NITROGEN	5.5	Low	6 - 20	mg/dL
METHOD : SPECTROPHOTOMETRY, UREASE WITH GLDH				
CREATININE, SERUM				
CREATININE	0.83	Low	0.90 - 1.30	mg/dL
METHOD : JAFFE, ALKALINE PICRATE, KINETIC WITH BLANK RATE CORRECTION				
BUN/CREAT RATIO				
BUN/CREAT RATIO	6.63	Low	10 - 20	
METHOD : CALCULATED PARAMETER				
URIC ACID, SERUM				
URIC ACID	6.6		3.7 - 9.2	mg/dL

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METHOD : SPECTROPHOTOMETRY, URICASE/PEROXIDASE				
TOTAL PROTEIN, SERUM				
TOTAL PROTEIN	7.0		5.7 - 8.2	g/dL
METHOD : SPECTROPHOTOMETRY, BIURET				
ALBUMIN, SERUM				
ALBUMIN	4.5		3.2 - 4.8	g/dL
METHOD : SPECTROPHOTOMETRY, BROMOCRESOL GREEN(BCG) - DYE BINDING				
GLOBULIN				
GLOBULIN	2.5		2.0 - 4.1	g/dL
METHOD : CALCULATED PARAMETER				
ELECTROLYTES (NA/K/CL), SERUM				
SODIUM	137		136 - 145	mmol/L
METHOD : INDIRECT INTEGRATED MULTISENSOR TECHNOLOGY (IMT).				
POTASSIUM	4.4		3.5 - 5.1	mmol/L
METHOD : INDIRECT INTEGRATED MULTISENSOR TECHNOLOGY (IMT).				
CHLORIDE	109	High	98 - 107	mmol/L
URINALYSIS				
COLOR	PALE YELLOW			
APPEARANCE	CLEAR			
PH	7.0		4.7 - 7.5	
SPECIFIC GRAVITY	<=1.005		1.003 - 1.035	
GLUCOSE	NOT DETECTED		NOT DETECTED	
PROTEIN	NOT DETECTED		NOT DETECTED	
KETONES	NOT DETECTED		NOT DETECTED	
BLOOD	NOT DETECTED		NOT DETECTED	
BILIRUBIN	NOT DETECTED		NOT DETECTED	
UROBILINOGEN	NORMAL		NORMAL	
NITRITE	NOT DETECTED		NOT DETECTED	
WBC	1-2		0-5	/HPF
EPITHELIAL CELLS	0-1		0-5	/HPF
RED BLOOD CELLS	NOT DETECTED		NOT DETECTED	/HPF
CASTS	NOT DETECTED			
CRYSTALS	NOT DETECTED			
BACTERIA	NOT DETECTED		NOT DETECTED	
METHOD : DIP STICK/MICRO SCOPY/REFLECTANCE SPECTROPHOTOMETRY				

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NOTE :MICROSCOPIC EXAMINATION OF URINE IS PERFORMED ON CENTRIFUGED URINARY SEDIMENT.
IN NORMAL URINE SAMPLES CAST AND CRYSTALS ARE NOT DETECTED.

APOLIPOPROTEIN - B, SERUM			
APOLIPOPROTEIN - B	0.64	0.53 - 1.73	g/L
METHOD : NEPHELOMETRY			
FOLIC ACID, SERUM			
FOLIC ACID	6.62	S: > 5.38 RBC: 280 – 791	ng/mL
METHOD : CHEMILUMINESCENCE			
TOTAL IGE, SERUM	RESULT PENDING		
FREE TRIIODOTHYRONINE (FT3), SERUM			
FREE TRIIODOTHYRONINE (FT3)	3.30	2.3 - 4.2	pg/mL
FREE THYROXINE (FT4), SERUM			
FREE THYROXINE (FT4)	1.36	0.89 - 1.76	ng/dL
TSH 3RD GENERATION ULTRA(TSH3 - UL), SERUM			
TSH 3RD GENERATION	1.931	0.55 - 4.78	µIU/mL

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.
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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References
1. Tietz Textbook of Clinical Chemistry and Molecular Diagnostics, edited by Carl A Burtis, Edward R.Ashwood, David E Bruns, 4th Edition, Elsevier publication, 2006, 879-884.
2. Forsham PH. Diabetes Mellitus:A rational plan for management. Postgrad Med 1982, 71,139-154.
3. Mayer TK, Freedman ZR: Protein glycosylation in Diabetes Mellitus: A review of laboratory measurements and their clinical utility. Clin Chim Acta 1983, 127, 147-184.
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.
CORTISOL, SERUM-Cortisol is the primary glucocorticoid hormone synthesized and secreted by the adrenal cortex. It is essential for life because it regulates carbohydrate, protein, and lipid metabolism, maintains normal blood pressure, and inhibits allergic and inflammatory reactions. Cortisol is synthesized and secreted by the cortex of the adrenal gland under the direction of adrenocorticotropic hormone. Increased ACTH levels stimulate cortisol secretion. The increased cortisol levels inhibit CRH secretion, which subsequently inhibits ACTH secretion. This negative feedback mechanism results in decreased cortisol levels.
Circulating cortisol levels follow a diurnal pattern in healthy individuals. Levels are highest in the morning after waking and lowest in the evening. Disorders of the hypothalamic-pituitaryadrenal axis override this diurnal pattern. Decreased cortisol levels are induced by either primary or secondary adrenal insufficiency. Addison's disease is caused by primary adrenal insufficiency due to metabolic errors or destruction of the adrenal cortex. Secondary adrenal insufficiency is caused by pituitary destruction or failure, resulting in loss of ACTH stimulation of the adrenal gland. Cushing's syndrome is caused by increased levels of cortisol due to either primary or secondary adrenal hyperfunction. Causes of primary adrenal hyperfunction are adrenal tumors and nodular adrenal hyperplasia. Secondary adrenal hyperfunction is caused by pituitary overproduction of ACTH or ectopic production of ACTH by a tumor. Increased cortisol levels are induced by pregnancy and by stress due to depression, trauma, surgery, hypoglycemia, alcoholism, uncontrolled diabetes, and starvation.
A 24-hour urinary cortisol measurement is the method of choice in the initial screening for Cushing's syndrome because it provides the best assessment of cortisol production. Urinary cortisol is not subject to the diurnal pattern of secretion and accurately differentiates healthy persons from patients with Cushing's syndrome.
Limitations
Circulating cortisol results from patients receiving Prednisolone or Prednisone therapy may be falsely elevated.
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.

Reference:
1. Pudek MR. Adrenal hormones. In: Kaplan LA, Pesce AJ, editors. Clinical chemistry: therapy, analysis, and correlation. St. Louis: CV Mosby, 1989. p.672–81.
2. Whitley RJ, Meikle AW, Watts NB. Endocrinology, part VI: adrenocortical steroids. In: Burtis CA, Ashwood ER, editors. Textbook of clinical chemistry, 2nd ed. Philadelphia: WB Saunders, 1994. p.1808–21.
3. Chodosh LA, Daniels GH. Addison's disease. Endocrinologist 1993 3(3):166–81.
4. Miller J, Crapo L. The biochemical diagnosis of hypercortisolism. Endocrinologist 1994 4(1):7–16.
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. 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.
MICROALBUMIN, URINE-
Microalbuminuria is defined as an increase in urinary excretion of albumin above the reference interval for healthy nondiabetic subjects but at a concentration that is generally detectable by crude clinical tests such as dipstics designed to measure total protein.the diagnosis of microalbuminuria requires demonstartion of increased albumin secretion in atleasy two out of three urine samples collected in the absence of infection or an acute metabolic crisis.

It is now considered a clinically important indicator of detiriorating renal function in diabetic subjects..in .diabetic..patients. Regular screening of urinary albumin secretion is valuable in monitoring both type 1 and type 2 diabetes.

Screening should comence 5 years after diagnosis in patients with type 1 diabetes and at diagnosis in patients with type 2 diabetes without proteinuria.

Screening is not indicated in patients with established proteinuria. All the patients with diabetes mellitus should be screened on annual basis upto the age of 75 years.

It is important to consider causes of increased albumin excretion, specially in cases of type 1 diabetes present for less than 5 years. These can include nondiabetic renal disease, menstural contamination, vaginal discharge, uncontrolled hypertension , urinary tract infection, heart failure, and strenous exercise.
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

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PATIENT NAME : GAGANMEET SINGH PATIENT ID : GAGAM183186200

ACCESSION NO : 0009TJ067147 AGE : 34 Years SEX : Male DATE OF BIRTH :

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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, 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.

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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, salt-losing nephritis, metabolic alkalosis, congestive heart failure, Addisonian crisis, certain types of metabolic acidosis, persistent gastric secretion and prolonged vomiting,
URINALYSIS-Routine urine analysis assists in screening and diagnosis of various metabolic, urological, kidney and liver disorders
Protein: Elevated proteins can be an early sign of kidney disease. Urinary protein excretion can also be temporarily elevated by strenuous exercise, orthostatic proteinuria, dehydration, urinary tract infections and acute illness with fever
Glucose: Uncontrolled diabetes mellitus can lead to presence of glucose in urine. Other causes include pregnancy, hormonal disturbances, liver disease and certain medications.
Ketones: Uncontrolled diabetes mellitus can lead to presence of ketones in urine. Ketones can also be seen in starvation, frequent vomiting, pregnancy and strenuous exercise.
Blood: Occult blood can occur in urine as intact erythrocytes or haemoglobin, which can occur in various urological, nephrological and bleeding disorders.
Leukocytes: An increase in leukocytes is an indication of inflammation in urinary tract or kidneys. Most common cause is bacterial urinary tract infection.
Nitrite: Many bacteria give positive results when their number is high. Nitrite concentration during infection increases with length of time the urine specimen is retained in bladder prior to collection.
pH: The kidneys play an important role in maintaining acid base balance of the body. Conditions of the body producing acidosis/ alkalosis or ingestion of certain type of food can affect the pH of urine.
Specific gravity: Specific gravity gives an indication of how concentrated the urine is. Increased specific gravity is seen in conditions like dehydration, glycosuria and proteinuria while decreased specific gravity is seen in excessive fluid intake, renal failure and diabetes insipidus.
Bilirubin: In certain liver diseases such as biliary obstruction or hepatitis, bilirubin gets excreted in urine.
Urobilinogen: Positive results are seen in liver diseases like hepatitis and cirrhosis and in cases of hemolytic anemia
APOLIPOPROTEIN - B, SERUM-Apolipoproteins are carrier proteins that combine with lipids to form lipoprotein particles, which have hydrophobic lipids at the core and hydrophilic side chains made of amino acids. There are several classes of lipoproteins ranging in density, from VLDL, or very low density lipoproteins, to VHDL, or very high density lipoproteins. There are nine different apolipoproteins that are found in human body, and they can act as signals, that cause lipoproteins to act on certain tissues or that activate enzymes that act on those lipoproteins

Apolipoprotein B (Apo B) is a major protein component of low-density lipoprotein (LDL) comprising >90% of the LDL proteins and constituting 20% to 25% of the total weight of LDL. Increased plasma concentration of Apo B-containing lipoproteins is associated with an increased risk of developing atherosclerotic disease.

Abetalipoproteinemia and severe hypobetalipoproteinemia can cause malabsorption of food lipids and polyneuropathy. In patients with hyperapobetalipoproteinemia (HALB), a disorder associated with increased risk of developing CHD and with an estimated prevalence of 30% in patients with premature CHD, Apo B is increased disproportionately in relation to LDL cholesterol. Apo B quantitation is required to identify these patients and is necessary in distinguishing HALB from another common lipoprotein abnormality, familial combined hyperlipidemia . Elevated levels of apolipoprotein B are more powerful indicators of disease than cholesterol or LDL in angiographic coronary artery disease.
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.

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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.
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

End Of Report

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

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CONDITIONS OF LABORATORY TESTING & REPORTING

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

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