DIAGNOSTIC REPORT

Patient Ref. No. 9000012120056

CLIENT CODE : C000097418

CLIENT'S NAME AND ADDRESS :
FPSC LAKSHAYA DIAGNOSTIC
SHOP NO. 2, OPP. UNIWORLD GARDEN, SUBHASH CHOWK,
SOHNA ROAD
GURGAON 122018
HARYANA INDIA
1242217031 9953304011

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ELECTRONIC CITY,SECTOR-18,
GURGAON, 122015
HARYANA, INDIA
Tel : 9111591115, Fax : CIN - U74899PB1995PLC045956

PATIENT NAME : AJAY MALIK PATIENT ID : **AJAYM482092370**

ACCESSION NO : **0009VI014043** AGE : 28 Years SEX : Male

DRAWN : 08/09/2022 07:00 RECEIVED : 08/09/2022 14:05 REPORTED : 08/09/2022 18:11

REFERRING DOCTOR : SELF CLIENT PATIENT ID :

Test Report Status **Final**	Results		Biological Reference Interval	Units
COMPLETE CARE TOTAL WITH SMART REPORT				
BLOOD COUNTS,EDTA WHOLE BLOOD				
HEMOGLOBIN METHOD : SPECTROPHOTOMETRY	14.3		13.0 - 17.0	g/dL
RED BLOOD CELL COUNT METHOD : IMPEDANCE	4.74		4.5 - 5.5	mil/µL
WHITE BLOOD CELL COUNT METHOD : IMPEDANCE	6.54		4.0 - 10.0	thou/µL
PLATELET COUNT METHOD : IMPEDANCE	262		150 - 410	thou/µL
RBC AND PLATELET INDICES				
HEMATOCRIT METHOD : CALCULATED	42.5		40 - 50	%
MEAN CORPUSCULAR VOL METHOD : DERIVED FROM IMPEDANCE MEASURE	89.6		83 - 101	fL
MEAN CORPUSCULAR HGB. METHOD : CALCULATED PARAMETER	30.1		27.0 - 32.0	pg
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION METHOD : CALCULATED PARAMETER	33.6		31.5 - 34.5	g/dL
MENTZER INDEX	18.9			
RED CELL DISTRIBUTION WIDTH METHOD : DERIVED FROM IMPEDANCE MEASURE	13.7		11.6 - 14.0	%
MEAN PLATELET VOLUME METHOD : DERIVED FROM IMPEDANCE MEASURE	10.1		6.8 - 10.9	fL
WBC DIFFERENTIAL COUNT - NLR				
SEGMENTED NEUTROPHILS METHOD : DHSS FLOWCYTOMETRY	46		40 - 80	%
ABSOLUTE NEUTROPHIL COUNT METHOD : DHSS FLOWCYTOMETRY, CALCULATED	3.02		2.0 - 7.0	thou/µL
LYMPHOCYTES METHOD : DHSS FLOWCYTOMETRY	34		20 - 40	%
ABSOLUTE LYMPHOCYTE COUNT METHOD : DHSS FLOWCYTOMETRY, CALCULATED	2.19		1 - 3	thou/µL
NEUTROPHIL LYMPHOCYTE RATIO (NLR) METHOD : CALCULATED	1.4			
EOSINOPHILS	**10**	**High**	1 - 6	%

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METHOD : DHSS FLOWCYTOMETRY				
ABSOLUTE EOSINOPHIL COUNT	**0.64**	**High**	0.02 - 0.50	thou/µL
METHOD : DHSS FLOWCYTOMETRY, CALCULATED				
MONOCYTES	09		2 - 10	%
METHOD : DHSS FLOWCYTOMETRY				
ABSOLUTE MONOCYTE COUNT	0.59		0.20 - 1.00	thou/µL
METHOD : DHSS FLOWCYTOMETRY, CALCULATED				
BASOPHILS	1		0 - 2	%
METHOD : IMPEDANCE				
ABSOLUTE BASOPHIL COUNT	0.04		0.02 - 0.10	thou/µL
METHOD : DHSS FLOWCYTOMETRY, CALCULATED				
ERYTHRO SEDIMENTATION RATE, BLOOD				
SEDIMENTATION RATE (ESR)	4		0 - 14	mm at 1 hr
METHOD : AUTOMATED (PHOTOMETRICAL CAPILLARY STOPPED FLOW KINETIC ANALYSIS)				
PERIPHERAL SMEAR EXAM, EDTA WHOLE BLOOD				
RBC	PREDOMINANTLY NORMOCYTIC NORMOCHROMIC			
WBC	MILD EOSINOPHILIA NOTED.			
PLATELETS	NORMAL IN NUMBER AND MORPHOLOGY.			
TOTAL IRON BINDING CAPACITY, SERUM				
IRON	83		59 - 158	µg/dL
METHOD : SPECTROPHOTOMETRY,FERROZINE				
TOTAL IRON BINDING CAPACITY	358		250 - 400	µg/dL
METHOD : CALCULATED PARAMETER				
% SATURATION	23.1		20 - 50	%
METHOD : CALCULATED PARAMETER				
GLUCOSE, FASTING, PLASMA				
GLUCOSE, FASTING, PLASMA	**69**	**Low**	Normal 75 - 99 Pre-diabetics: 100 – 125 Diabetic: > or = 126	mg/dL
METHOD : SPECTROPHOTOMETRY 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 : CAPILLARY ELECTROPHORESIS				
MEAN PLASMA GLUCOSE	108.3		< 116	mg/dL
METHOD : CALCULATED PARAMETER				

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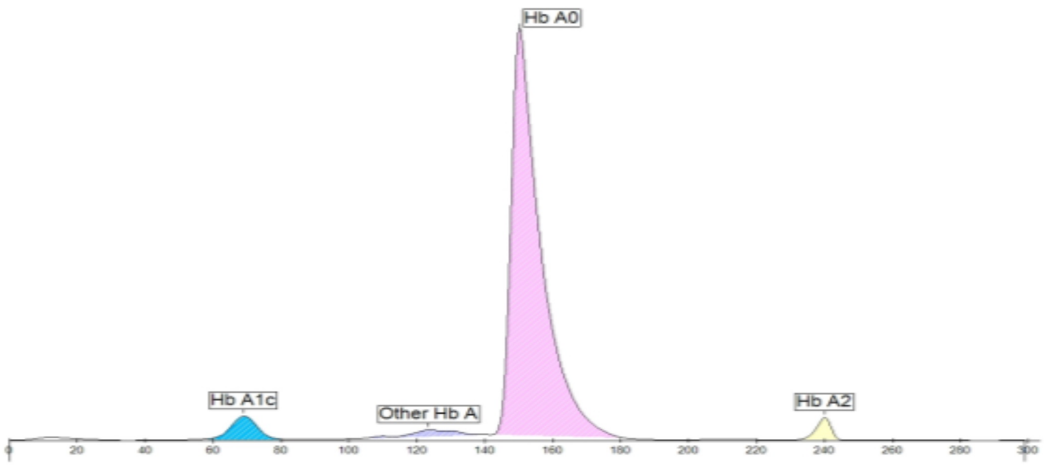
Test Report Status **Final**	Results	Biological Reference Interval	Units

PLOT NO.31,ELECTRONIC CITY,SECTOR 18, GURUGRAM

ID : 914289295
Name :

Sample Date: 9/8/2022
Sample num.: 140



A1c Haemoglobin Electrophoresis

Fractions	%	mmol/mol	Cal. %
Hb A1c	-	**35**	5.4
Other Hb A	2.3		
Hb A0	90.4		
Hb A2	2.6		

HbA1c % cal :**5.4 %**

Comments :

LIVER FUNCTION PROFILE, SERUM

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BILIRUBIN, TOTAL	0.5		Upto 1.2	mg/dL
METHOD : COLORIMETRIC DIAZO METHOD				
BILIRUBIN, DIRECT	0.2		< 0.30	mg/dL
METHOD : COLORIMETRIC DIAZO METHOD				
BILIRUBIN, INDIRECT	0.30		0.1 - 1.0	mg/dL
METHOD : CALCULATED PARAMETER				
TOTAL PROTEIN	7.6		6.0 - 8.0	g/dL
METHOD : SPECTROPHOTOMETRY, BIURET				
ALBUMIN	4.9		3.97 - 4.94	g/dL
METHOD : SPECTROPHOTOMETRY, BROMOCRESOL GREEN(BCG) - DYE BINDING				
GLOBULIN	2.7		2.0 - 3.5	g/dL
METHOD : CALCULATED PARAMETER				
ALBUMIN/GLOBULIN RATIO	1.8		1.0 - 2.1	RATIO
METHOD : CALCULATED PARAMETER				
ASPARTATE AMINOTRANSFERASE (AST/SGOT)	39		< OR = 50	U/L
METHOD : SPECTROPHOTOMETRY, WITH PYRIDOXAL PHOSPHATE ACTIVATION-IFCC				
ALANINE AMINOTRANSFERASE (ALT/SGPT)	**54**	**High**	< OR = 50	U/L
METHOD : SPECTROPHOTOMETRY, WITH PYRIDOXAL PHOSPHATE ACTIVATION-IFCC				
ALKALINE PHOSPHATASE	100		40 - 129	U/L
METHOD : SPECTROPHOTOMETRY, PNPP, AMP BUFFER - IFCC				
GAMMA GLUTAMYL TRANSFERASE (GGT)	36		0 - 60	U/L
METHOD : ENZYMATIC COLORIMETRIC ASSAY STANDARDIZED AGAINST IFCC / SZASZ				
LACTATE DEHYDROGENASE	209		125 - 220	U/L
METHOD : SPECTROPHOTOMETRY, LACTATE TO PYRUVATE - UV-IFCC				
25 - HYDROXYVITAMIN D, SERUM				
25 - HYDROXYVITAMIN D	33.2		Deficiency: < 20.0 Insufficiency: 20.0 - < 30.0 Sufficiency: 30.0 -100.0 Toxicity > 100.0	ng/mL
METHOD : ELECTROCHEMILUMINESCENCE IMMUNO ASSAY				
CALCIUM, SERUM				
CALCIUM	**10.2**	**High**	8.6 - 10.0	mg/dL
METHOD : SPECTROPHOTOMETRY, NM - BAPTA				
VITAMIN B12 LEVEL, SERUM				
VITAMIN B12	415		197 - 771	pg/mL
METHOD : ELECTROCHEMILUMINESCENCE IMMUNO ASSAY				
THYROID PANEL, SERUM				
T3	104.0		80 - 200	ng/dL
METHOD : ELECTROCHEMILUMINESCENCE IMMUNO ASSAY				

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T4	7.30		5.1 - 14.1	µg/dL
METHOD : ELECTROCHEMILUMINESCENCE IMMUNO ASSAY				
TSH 3RD GENERATION	2.460		0.27 - 4.2	µIU/mL
METHOD : ELECTROCHEMILUMINESCENCE IMMUNO ASSAY				
CORONARY RISK PROFILE (LIPID PROFILE), SERUM				
CHOLESTEROL	**236**	**High**	Desirable cholesterol level < 200 Borderline high cholesterol 200 - 239 High cholesterol > / = 240	mg/dL
METHOD : ENZYMATIC COLORIMETRIC ASSAY				
TRIGLYCERIDES	**192**	**High**	Normal: < 150 Borderline high: 150 - 199 High: 200 - 499 Very High: >/= 500	mg/dL
METHOD : ENZYMATIC COLORIMETRIC ASSAY				
HDL CHOLESTEROL	47		Low HDL Cholesterol <40 High HDL Cholesterol >/= 60	mg/dL
METHOD : HOMOGENEOUS ENZYMATIC COLORIMETRIC ASSAY				
DIRECT LDL CHOLESTEROL	**163.00**	**High**	Optimal : < 100 Near optimal/above optimal : 100 - 129 Borderline high : 130 - 159 High : 160 - 189 Very high : > / = 190	mg/dL
METHOD : HOMOGENEOUS ENZYMATIC COLORIMETRIC ASSAY				
NON HDL CHOLESTEROL	**189**	**High**	Desirable : < 130 Above Desirable : 130 -159 Borderline High : 160 - 189 High : 190 - 219 Very high : > / = 220	mg/dL
METHOD : CALCULATED PARAMETER				
CHOL/HDL RATIO	**5.0**	**High**	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	**3.4**	**High**	Desirable/Low Risk - 0.5-3 Borderline/Moderate Risk- 3.1-6 High Risk- >6.0	
METHOD : CALCULATED PARAMETER				
VERY LOW DENSITY LIPOPROTEIN	**38.4**	**High**	</= 30.0	mg/dL
METHOD : CALCULATED PARAMETER				

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SERUM BLOOD UREA NITROGEN				
BLOOD UREA NITROGEN	9.3		6 - 20	mg/dL
METHOD : SPECTROPHOTOMETRY, KINETIC TEST WITH UREASE AND GLUTAMATE DEHYDROGENASE				
CREATININE, SERUM				
CREATININE	1.03		0.7 - 1.2	mg/dL
METHOD : SPECTROPHOTOMETRIC, JAFFE'S KINETICS				
BUN/CREAT RATIO				
BUN/CREAT RATIO	9.00		8.0 - 15.0	
METHOD : CALCULATED PARAMETER				
URIC ACID, SERUM				
URIC ACID	**8.4**	**High**	3.4 - 7.0	mg/dL
METHOD : SPECTROPHOTOMETRY, URICASE				
TOTAL PROTEIN, SERUM				
TOTAL PROTEIN	7.6		6.0 - 8.0	g/dL
METHOD : SPECTROPHOTOMETRY, BIURET				
ALBUMIN, SERUM				
ALBUMIN	4.9		3.97 - 4.94	g/dL
METHOD : SPECTROPHOTOMETRY, BROMOCRESOL GREEN(BCG) - DYE BINDING				
GLOBULIN				
GLOBULIN	2.7		2.0 - 3.5	g/dL
METHOD : CALCULATED PARAMETER				
ELECTROLYTES (NA/K/CL), SERUM				
SODIUM	143		136 - 145	mmol/L
METHOD : ISE INDIRECT				
POTASSIUM	**5.7**	**High**	3.5 - 5.1	mmol/L
METHOD : ISE INDIRECT				
CHLORIDE	107		98 - 107	mmol/L
METHOD : ISE INDIRECT				
PHYSICAL EXAMINATION, URINE				
COLOR	Paleyellow			
APPEARANCE	CLEAR			
SPECIFIC GRAVITY	1.025		1.010 - 1.030	

Comments

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

CHEMICAL EXAMINATION, URINE

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PH	6.0	5.00 - 7.50	
PROTEIN	NOT DETECTED	NOT DETECTED	
GLUCOSE	NOT DETECTED	NOT DETECTED	
KETONES	NOT DETECTED	NOT DETECTED	
BLOOD	NOT DETECTED	NOT DETECTED	
BILIRUBIN	NOT DETECTED	NOT DETECTED	
UROBILINOGEN	NOT DETECTED	NORMAL	
NITRITE	NOT DETECTED	NOT DETECTED	
LEUKOCYTE ESTERASE	NOT DETECTED	NOT DETECTED	
MICROSCOPIC EXAMINATION, URINE			
PUS CELL (WBC'S)	1-2	0-5	/HPF
EPITHELIAL CELLS	0-1	0-5	/HPF
ERYTHROCYTES (RBC'S)	NOT DETECTED	NOT DETECTED	/HPF
CASTS	NOT DETECTED		
CRYSTALS	NOT DETECTED		
BACTERIA	NOT DETECTED	NOT DETECTED	
YEAST	NOT DTECTED	NOT DETECTED	
MAGNESIUM, SERUM			
MAGNESIUM, SERUM	2.2	1.6 - 2.6	mg/dL

METHOD : COLORIMETRIC ENDPOINT METHOD

Interpretation(s)
BLOOD COUNTS,EDTA WHOLE BLOOD-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-Mentzer index (MCV/RBC) is an automated cell-counter based calculated screen tool to differentiate cases of Iron deficiency anaemia(>13) from Beta thalassaemia trait
(<13) in patients with microcytic anaemia. This needs to be interpreted in line with clinical correlation and suspicion. Estimation of HbA2 remains the gold standard for diagnosing a case of beta thalassaemia trait.
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"

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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 2021 guidelines for adults, after 8 hrs fasting is as follows:
Pre-diabetics: 100 - 125 mg/dL
Diabetic: > or = 126 mg/dL
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.''

References
1. Tietz Textbook of Clinical Chemistry and Molecular Diagnostics, edited by Carl A Burtis, Edward R.Ashwood, David E Bruns, 4th Edition, Elsevier publication, 2006, 879-884.
2. Forsham PH. Diabetes Mellitus:A rational plan for management. Postgrad Med 1982, 71,139-154.
3. Mayer TK, Freedman ZR: Protein glycosylation in Diabetes Mellitus: A review of laboratory measurements and their clinical utility. Clin Chim Acta 1983, 127, 147-184.
LIVER FUNCTION PROFILE, SERUM-LIVER FUNCTION PROFILE
Bilirubin is a yellowish pigment found in bile and is a breakdown product of normal heme catabolism. Bilirubin is excreted in bile and urine, and elevated levels may give yellow discoloration in jaundice.Elevated levels results from increased bilirubin production (eg, hemolysis and ineffective erythropoiesis), decreased bilirubin excretion (eg, obstruction and hepatitis), and abnormal bilirubin metabolism (eg, hereditary and neonatal jaundice). Conjugated (direct) bilirubin is elevated more than unconjugated (indirect) bilirubin in Viral hepatitis, Drug reactions, Alcoholic liver disease Conjugated (direct) bilirubin is also elevated more than unconjugated (indirect) bilirubin when there is some kind of blockage of the bile ducts like in Gallstones getting into the bile ducts, tumors &Scarring of the bile ducts. Increased unconjugated (indirect) bilirubin may be a result of Hemolytic or pernicious anemia, Transfusion reaction & a common metabolic condition termed Gilbert syndrome, due to low levels of the enzyme that attaches sugar molecules to bilirubin.
AST is an enzyme found in various parts of the body. AST is found in the liver, heart, skeletal muscle, kidneys, brain, and red blood cells, and it is commonly measured clinically as a marker for liver health. AST levels increase during chronic viral hepatitis, blockage of the bile duct, cirrhosis of the liver,liver cancer,kidney failure,hemolytic anemia,pancreatitis,hemochromatosis. AST levels may also increase after a heart attack or strenuous activity.ALT test measures the amount of this enzyme in the blood.ALT is found mainly in the liver, but also in smaller amounts in the kidneys,heart,muscles, and pancreas.It is commonly measured as a part of a diagnostic evaluation of hepatocellular injury, to determine liver health.AST levels increase during acute hepatitis,sometimes due to a viral infection,ischemia to the liver,chronic hepatitis,obstruction of bile ducts,cirrhosis.
ALP is a protein found in almost all body tissues.Tissues with higher amounts of ALP include the liver,bile ducts and bone.Elevated ALP levels are seen in Biliary obstruction, Osteoblastic bone tumors, osteomalacia, hepatitis, Hyperparathyroidism, Leukemia, Lymphoma, Paget''s disease,Rickets,Sarcoidosis etc. Lower-than-normal ALP levels seen in Hypophosphatasia,Malnutrition,Protein deficiency,Wilson''s disease.GGT is an enzyme found in cell membranes of many tissues mainly in the liver,kidney and pancreas.It is also found in other tissues including intestine,spleen,heart, brain and seminal vesicles.The highest concentration is in the kidney,but the liver is considered the source of normal enzyme activity.Serum GGT has been widely used as an index of liver dysfunction.Elevated serum GGT activity can be found in diseases of the liver,biliary system

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Tel : 9111591115, Fax : CIN - U74899PB1995PLC045956

PATIENT NAME : AJAY MALIK PATIENT ID : **AJAYM482092370**

ACCESSION NO : **0009VI014043** AGE : 28 Years SEX : Male

DRAWN : 08/09/2022 07:00 RECEIVED : 08/09/2022 14:05 REPORTED : 08/09/2022 18:11

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and pancreas.Conditions that increase serum GGT are obstructive liver disease,high alcohol consumption and use of enzyme-inducing drugs etc.Serum total protein,also known as total protein,is a biochemical test for measuring the total amount of protein in serum.Protein in the plasma is made up of albumin and globulin.Higher-than-normal levels may be due to:Chronic inflammation or infection,including HIV and hepatitis B or C,Multiple myeloma,Waldenstrom''s disease.Lower-than-normal levels may be due to: Agammaglobulinemia,Bleeding (hemorrhage),Burns,Glomerulonephritis,Liver disease, Malabsorption,Malnutrition,Nephrotic syndrome,Protein-losing enteropathy etc.Human serum albumin is the most abundant protein in human blood plasma.It is produced in the liver.Albumin constitutes about half of the blood serum protein.Low blood albumin levels (hypoalbuminemia) can be caused by:Liver disease like cirrhosis of the liver, nephrotic syndrome,protein-losing enteropathy,Burns,hemodilution,increased vascular permeability or decreased lymphatic clearance,malnutrition and wasting etc
25 - HYDROXYVITAMIN D, SERUM-Note: Our Vitamin D assays is standardized to be in alignment with the ID-LC/MS/MS 25(OH)vitamin D Reference Method Procedure (RMP), the reference procedure for the Vitamin D Standardization Program (VDSP). The VDSP, a collaboration of the National Institutes of Health Office of Dietary Supplements, National Institute of Technology and Standards, Centers for Disease Control and Ghent University, is an initiative to standardize 25(OH)vitamin D measurement across methods
CALCIUM, SERUM-Commom causes of decreased value of calcium (hypocalcemia) are chronic renal failure, hypomagnesemia and hypoalbuminemia.

Hypercalcemia (increased value of calcium) can be caused by increased intestinal absorbtion (vitamin d intoxication), increased skeletal reasorption (immobilization), or a combination of mechanisms (primary hyperparathyroidism). Primary hyperparathyroidism and malignancy accounts for 90-95% of all cases of hypercalcemia.

Values of total calcium is affected by serum proteins, particularly albumin thus, latter's value should be taken into account when interpreting serum calcium levels. The following regression equation may be helpful.
Corrected total calcium (mg/dl)= total calcium (mg/dl) + 0.8 (4- albumin [g/dl])*
because regression equations vary among group of patients in different physiological and pathological conditions, mathematical corrections are only approximations. The possible mathematical corrections should be replaced by direct determination of free calcium by ISE (available with srl) a common and important source of preanalytical error in the measurement of calcium is prolonged torniquet application during sampling. Thus, this along with fist clenching should be avoided before phlebotomy.
THYROID PANEL, SERUM-Triiodothyronine T3 , is a thyroid hormone. It affects almost every physiological process in the body, including growth, development, metabolism, body temperature, and heart rate. Production of T3 and its prohormone thyroxine (T4) is activated by thyroid-stimulating hormone (TSH), which is released from the pituitary gland. Elevated concentrations of T3, and T4 in the blood inhibit the production of TSH.
Thyroxine T4, Thyroxine's principal function is to stimulate the metabolism of all cells and tissues in the body. Excessive secretion of thyroxine in the body is hyperthyroidism, and deficient secretion is called hypothyroidism. Most of the thyroid hormone in blood is bound to transport proteins. Only a very small fraction of the circulating hormone is free and biologically active.
In primary hypothyroidism, TSH levels are significantly elevated, while in secondary and tertiary hypothyroidism, TSH levels are low.
Below mentioned are the guidelines for Pregnancy related reference ranges for Total T4, TSH & Total T3

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

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

	T3 (ng/dL)	T4 (µg/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.
2. Gowenlock A.H. Varley''s Practical Clinical Biochemistry, 6th Edition.
3. Behrman R.E. Kilegman R.M., Jenson H. B. Nelson Text Book of Pediatrics, 17th Edition
CORONARY RISK PROFILE (LIPID PROFILE), SERUM-Serum cholesterol is a blood test that can provide valuable information for the risk of coronary artery disease This test can help determine your risk of the build up of plaques in your arteries that can lead to narrowed or blocked arteries throughout your body (atherosclerosis). High cholesterol levels usually don''''t cause any signs or symptoms, so a cholesterol test is an important tool. High cholesterol levels often are a significant risk factor for heart disease and important for diagnosis of hyperlipoproteinemia, atherosclerosis, hepatic and thyroid diseases.

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

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

SERUM LDL The small dense LDL test can be used to determine cardiovascular risk in individuals with metabolic syndrome or established/progressing coronary artery

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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.

Non HDL Cholesterol - Adult treatment panel ATP III suggested the addition of Non-HDL Cholesterol as an indicator of all atherogenic lipoproteins (mainly LDL and VLDL). NICE guidelines recommend Non-HDL Cholesterol measurement before initiating lipid lowering therapy. It has also been shown to be a better marker of risk in both primary and secondary prevention studies.

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

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DIAGNOSTIC REPORT

Patient Ref. No. 9000012120056

CLIENT CODE : C000097418

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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,
MICROSCOPIC EXAMINATION, URINE-
Routine urine analysis assists in screening and diagnosis of various metabolic, urological, kidney and liver disorders
Protein: Elevated proteins can be an early sign of kidney disease. Urinary protein excretion can also be temporarily elevated by strenuous exercise, orthostatic proteinuria, dehydration, urinary tract infections and acute illness with fever
Glucose: Uncontrolled diabetes mellitus can lead to presence of glucose in urine. Other causes include pregnancy, hormonal disturbances, liver disease and certain medications.
Ketones: Uncontrolled diabetes mellitus can lead to presence of ketones in urine. Ketones can also be seen in starvation, frequent vomiting, pregnancy and strenuous exercise.
Blood: Occult blood can occur in urine as intact erythrocytes or haemoglobin, which can occur in various urological, nephrological and bleeding disorders.
Leukocytes: An increase in leukocytes is an indication of inflammation in urinary tract or kidneys. Most common cause is bacterial urinary tract infection.
Nitrite: Many bacteria give positive results when their number is high. Nitrite concentration during infection increases with length of time the urine specimen is retained in bladder prior to collection.
pH: The kidneys play an important role in maintaining acid base balance of the body. Conditions of the body producing acidosis/ alkalosis or ingestion of certain type of food can affect the pH of urine.
Specific gravity: Specific gravity gives an indication of how concentrated the urine is. Increased specific gravity is seen in conditions like dehydration, glycosuria and proteinuria while decreased specific gravity is seen in excessive fluid intake, renal failure and diabetes insipidus.
Bilirubin: In certain liver diseases such as biliary obstruction or hepatitis, bilirubin gets excreted in urine.
Urobilinogen: Positive results are seen in liver diseases like hepatitis and cirrhosis and in cases of hemolytic anemia
MAGNESIUM, SERUM-Moderate or severe magnesium deficiency is usually due to losses of magnesium from gastrointestinal tract or kidneys as in vomiting and diarrhoea in former and alcohol, diabetes mellitus (osmotic diuresis), loop diuretics (furosemide) and aminoglycoside antibiotics in latter.

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

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

BIO CHEMISTRY

GLUCOSE, POST-PRANDIAL, PLASMA

GLUCOSE, POST-PRANDIAL, PLASMA	80	70 - 139	mg/dL

METHOD : SPECTROPHOTOMETRY, HEXOKINASE

Interpretation(s)
GLUCOSE, POST-PRANDIAL, PLASMA-ADA Guidelines for 2hr post prandial glucose levels is only after ingestion of 75grams of glucose in 300 ml water,over a period of 5 minutes.

****End Of Report****
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Dr. Arpita Roy, MD
Section Head Flowcytometry & Head QA

Dr. Anurag Bansal
LAB DIRECTOR

Dr.Nishtha Wadhwa
Clinical Biochemist

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.
3. Result delays could occur due to unforeseen circumstances such as non-availability of kits / equipment breakdown / natural calamities / technical downtime or any other unforeseen event.
4. A requested test might not be performed if:
   i. Specimen received is insufficient or inappropriate
   ii. Specimen quality is unsatisfactory
   iii. Incorrect specimen type
   iv. Discrepancy between identification on specimen container label and test requisition form
5. SRL confirms that all tests have been performed or assayed with highest quality standards, clinical safety & technical integrity.
6. Laboratory results should not be interpreted in isolation; it must be correlated with clinical information and be interpreted by registered medical practitioners only to determine final diagnosis.
7. Test results may vary based on time of collection, physiological condition of the patient, current medication or nutritional and dietary changes. Please consult your doctor or call us for any clarification.
8. Test results cannot be used for Medico legal purposes.
9. In case of queries please call customer care (91115 91115) within 48 hours of the report.

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