Patient Ref. No. 775000001633777

CLIENT CODE : C000138394

CLIENT'S NAME AND ADDRESS :
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MAHARASHTRA, INDIA
Tel : 9111591115, Fax : CIN - U74899PB1995PLC045956
Email : customercare.thane@srl.in

PATIENT NAME : BAIT RAGHUNATH RATNU PATIENT ID : BAITM051166181

ACCESSION NO : 0181VI000339 AGE : 55 Years SEX : Male

DRAWN : RECEIVED : 10/09/2022 10:42 REPORTED : 13/09/2022 15:19

REFERRING DOCTOR : SELF CLIENT PATIENT ID :

CLINICAL INFORMATION :

STOOL CANCEL

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

MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 40 MALE

PHYSICAL EXAMINATION, URINE			
COLOR	PALE YELLOW		
METHOD : VISUAL INSPECTION			
APPEARANCE	CLEAR		
METHOD : VISUAL INSPECTION			
SPECIFIC GRAVITY	1.005	1.003 - 1.035	
METHOD : IONIC CONCENTRATION METHOD			
BLOOD COUNTS,EDTA WHOLE BLOOD			
HEMOGLOBIN	14.8	13.0 - 17.0	g/dL
METHOD : SLS- HEMOGLOBIN DETECTION METHOD			
RED BLOOD CELL COUNT	5.03	4.5 - 5.5	mil/µL
METHOD : HYDRODYNAMIC FOCUSING BY DC DETECTION			
WHITE BLOOD CELL COUNT	8.32	4.0 - 10.0	thou/µL
METHOD : FLUORESCENCE FLOW CYTOMETRY			
PLATELET COUNT	297	150 - 410	thou/µL
METHOD : HYDRODYNAMIC FOCUSING BY DC DETECTION			
RBC AND PLATELET INDICES			
HEMATOCRIT	45.1	40.0 - 50.0	%
METHOD : CUMULATIVE PULSE HEIGHT DETECTION METHOD			
MEAN CORPUSCULAR VOL	89.7	83.0 - 101.0	fL
METHOD : CALCULATED FROM RBC & HCT			
MEAN CORPUSCULAR HGB.	29.4	27.0 - 32.0	pg
METHOD : CALCULATED FROM THE RBC & HGB			
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION	32.8	31.5 - 34.5	g/dL
METHOD : CALCULATED FROM THE HGB & HCT			
MENTZER INDEX	17.8		
RED CELL DISTRIBUTION WIDTH	13.1	11.6 - 14.0	%
METHOD : CALCULATED FROM RBC SIZE DISTRIBUTION CURVE			
MEAN PLATELET VOLUME	9.7	6.8 - 10.9	fL
METHOD : CALCULATED FROM PLATELET COUNT & PLATELET HEMATOCRIT			
CHEMICAL EXAMINATION, URINE			
PH	6.0	4.7 - 7.5	
METHOD : DOUBLE INDICATOR PRINCIPLE			

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STOOL CANCEL

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PROTEIN	NOT DETECTED		NOT DETECTED	
METHOD : TETRA BROMOPHENOL BLUE/SULFOSALICYLIC ACID				
GLUCOSE	NOT DETECTED		NOT DETECTED	
METHOD : GLUCOSE OXIDASE PEROXIDASE				
KETONES	NOT DETECTED		NOT DETECTED	
METHOD : NITROPRUSSIDE REACTION				
BLOOD	NOT DETECTED		NOT DETECTED	
METHOD : PEROXIDASE				
UROBILINOGEN	NORMAL		NORMAL	
METHOD : MODIFIED EHRLICH REACTION				
NITRITE	NOT DETECTED		NOT DETECTED	
METHOD : 1,2,3,4-TETRAHYDROBENZO(H)QUINOLIN-3-OL				
LEUKOCYTE ESTERASE	NOT DETECTED		NOT DETECTED	
WBC DIFFERENTIAL COUNT - NLR				
SEGMENTED NEUTROPHILS	44		40 - 80	%
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
ABSOLUTE NEUTROPHIL COUNT	3.63		2.0 - 7.0	thou/µL
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
LYMPHOCYTES	**45**	**High**	20 - 40	%
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
ABSOLUTE LYMPHOCYTE COUNT	**3.74**	**High**	1.0 - 3.0	thou/µL
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	1.0			
EOSINOPHILS	**7**	**High**	1 - 6	%
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
ABSOLUTE EOSINOPHIL COUNT	**0.54**	**High**	0.02 - 0.50	thou/µL
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
MONOCYTES	4		2 - 10	%
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
ABSOLUTE MONOCYTE COUNT	0.37		0.2 - 1.0	thou/µL
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
DIFFERENTIAL COUNT PERFORMED ON:	EDTA SMEAR			
MICROSCOPIC EXAMINATION, URINE				
PUS CELL (WBC'S)	1-2		0-5	/HPF
METHOD : MICROSCOPIC EXAMINATION				

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STOOL CANCEL

Test Report Status **Final**	Results		Biological Reference Interval	Units
EPITHELIAL CELLS	0-1		0-5	/HPF
METHOD : MICROSCOPIC EXAMINATION				
ERYTHROCYTES (RBC'S)	NOT DETECTED		NOT DETECTED	/HPF
METHOD : MICROSCOPIC EXAMINATION				
CASTS	NOT DETECTED			
METHOD : MICROSCOPIC EXAMINATION				
CRYSTALS	NOT DETECTED			
METHOD : MICROSCOPIC EXAMINATION				
BACTERIA	NOT DETECTED		NOT DETECTED	
METHOD : MICROSCOPIC EXAMINATION				
YEAST	NOT DETECTED		NOT DETECTED	
MORPHOLOGY				
RBC	NORMOCYTIC NORMOCHROMIC			
WBC	EOSINOPHILIA PRESENT			
METHOD : MICROSCOPIC EXAMINATION				
PLATELETS	ADEQUATE			
ERYTHRO SEDIMENTATION RATE, BLOOD				
SEDIMENTATION RATE (ESR)	07		0 - 14	mm at 1 hr
METHOD : WESTERGREN METHOD				
GLYCOSYLATED HEMOGLOBIN, EDTA WHOLE BLOOD				
GLYCOSYLATED HEMOGLOBIN (HBA1C)	**6.0**	**High**	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	**125.5**	**High**	< 116.0	mg/dL
METHOD : CALCULATED PARAMETER				
GLUCOSE, FASTING, PLASMA				
GLUCOSE, FASTING, PLASMA	94		Normal 75 - 99 Pre-diabetics: 100 – 125 Diabetic: > or = 126	mg/dL
METHOD : ENZYMATIC REFERENCE METHOD WITH HEXOKINASE				
GLUCOSE, POST-PRANDIAL, PLASMA				
GLUCOSE, POST-PRANDIAL, PLASMA	**154**	**High**	70 - 139	mg/dL
METHOD : ENZYMATIC REFERENCE METHOD WITH HEXOKINASE				
CORONARY RISK PROFILE, SERUM				

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STOOL CANCEL

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CHOLESTEROL	**205**	**High**	Desirable cholesterol level < 200 Borderline high cholesterol 200 - 239 High cholesterol > / = 240	mg/dL
METHOD : ENZYMATIC COLORIMETRIC ASSAY				
TRIGLYCERIDES	**289**	**High**	Normal: < 150 Borderline high: 150 - 199 High: 200 - 499 Very High: >/= 500	mg/dL
METHOD : ENZYMATIC COLORIMETRIC ASSAY				
HDL CHOLESTEROL	25.1			mg/dL
CHOLESTEROL LDL	122			mg/dL
NON HDL CHOLESTEROL	180			mg/dL
CHOL/HDL RATIO	8.2			
LDL/HDL RATIO	4.9			
VERY LOW DENSITY LIPOPROTEIN	**57.8**	**High**	< OR = 30.0	mg/dL
LIVER FUNCTION PROFILE, SERUM				
BILIRUBIN, TOTAL	0.47		Upto 1.2	mg/dL
METHOD : COLORIMETRIC DIAZO				
BILIRUBIN, DIRECT	0.18		< 0.30	mg/dL
BILIRUBIN, INDIRECT	0.29		0.1 - 1.0	mg/dL
TOTAL PROTEIN	7.9		6.0 - 8.0	g/dL
METHOD : COLORIMETRIC				
ALBUMIN	4.8		3.97 - 4.94	g/dL
METHOD : COLORIMETRIC				
GLOBULIN	3.1		2.0 - 3.5	g/dL
ALBUMIN/GLOBULIN RATIO	1.6		1.0 - 2.1	RATIO
ASPARTATE AMINOTRANSFERASE (AST/SGOT)	36		< OR = 50	U/L
METHOD : UV ABSORBANCE				
ALANINE AMINOTRANSFERASE (ALT/SGPT)	37		< OR = 50	U/L
METHOD : UV ABSORBANCE				
ALKALINE PHOSPHATASE	115		40 - 129	U/L
METHOD : COLORIMETRIC				
GAMMA GLUTAMYL TRANSFERASE (GGT)	40		0 - 60	U/L

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STOOL CANCEL

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METHOD : ENZYMATIC, COLORIMETRIC				
LACTATE DEHYDROGENASE	154		125 - 220	U/L
METHOD : UV ABSORBANCE				
SERUM BLOOD UREA NITROGEN				
BLOOD UREA NITROGEN	16		6 - 20	mg/dL
METHOD : ENZYMATIC ASSAY				
CREATININE, SERUM				
CREATININE	1.01		0.7 - 1.2	mg/dL
METHOD : COLORIMETRIC				
BUN/CREAT RATIO				
BUN/CREAT RATIO	**15.84**	**High**	8.0 - 15.0	
URIC ACID, SERUM				
URIC ACID	5.0		3.4 - 7.0	mg/dL
METHOD : ENZYMATIC COLORIMETRIC ASSAY				
TOTAL PROTEIN, SERUM				
TOTAL PROTEIN	7.9		6.0 - 8.0	g/dL
METHOD : COLORIMETRIC				
ALBUMIN, SERUM				
ALBUMIN	4.8		3.97 - 4.94	g/dL
METHOD : COLORIMETRIC				
GLOBULIN				
GLOBULIN	3.1		2.0 - 3.5	g/dL
ELECTROLYTES (NA/K/CL), SERUM				
SODIUM	138		136 - 145	mmol/L
POTASSIUM	4.93		3.5 - 5.1	mmol/L
CHLORIDE	100		98 - 107	mmol/L
THYROID PANEL, SERUM				
T3	110.0		80 - 200	ng/dL
METHOD : ELECTROCHEMILUMINESCENCE				
T4	8.20		5.1 - 14.1	µg/dL
METHOD : ELECTROCHEMILUMINESCENCE				
TSH 3RD GENERATION	2.800		0.27 - 4.2	µIU/mL
METHOD : ELECTROCHEMILUMINESCENCE				
ABO GROUP & RH TYPE, EDTA WHOLE BLOOD				

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ABO GROUP	TYPE B		
METHOD : GEL COLUMN AGGLUTINATION METHOD.			
RH TYPE	POSITIVE		
METHOD : GEL COLUMN AGGLUTINATION METHOD.			
XRAY-CHEST			
IMPRESSION	NO ABNORMALITY DETECTED		
TMT OR ECHO			
TMT OR ECHO	2D ECHO :- Aortic valve sclerosis Concentric LVH. No regional wall motion abnormality. Good Left Ventricular systolic function. LVEF 60 % Normal LV Diastolic function. No e/o pulmonary hypertension.		
ECG			
ECG	LEFT ANTERIOR FASCICULAR BLOCK.		
MEDICAL HISTORY			
RELEVANT PRESENT HISTORY	HYPERTENSIV SINCE 10 YEARS.		
RELEVANT PAST HISTORY	PAST H/O RENAL CALCULUS TREATED CONSERVATIVELY. ADMITTED FOR PALPITATIONS IN 2011.NO DETAILS. MALARIA IN THE PAST.		
RELEVANT PERSONAL HISTORY	MARRIED / 3 CHILD / MIXED DIET / NO ALLERGIES / NO SMOKING / OCC ALCOHOL.		
RELEVANT FAMILY HISTORY	ASTHMA : FATHER,BROTHER.		
HISTORY OF MEDICATIONS	NOT SIGNIFICANT		
ANTHROPOMETRIC DATA & BMI			
HEIGHT IN METERS	1.65		mts
WEIGHT IN KGS.	76		Kgs
BMI	28	BMI & Weight Status as follows: kg/sqmts Below 18.5: Underweight 18.5 - 24.9: Normal 25.0 - 29.9: Overweight 30.0 and Above: Obese	
GENERAL EXAMINATION			
MENTAL / EMOTIONAL STATE	NORMAL		
PHYSICAL ATTITUDE	NORMAL		
GENERAL APPEARANCE / NUTRITIONAL STATUS	OVERWEIGHT		
BUILT / SKELETAL FRAMEWORK	AVERAGE		

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ACCESSION NO : **0181VI000339** AGE : 55 Years SEX : Male

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FACIAL APPEARANCE	NORMAL		
SKIN	NORMAL		
UPPER LIMB	NORMAL		
LOWER LIMB	NORMAL		
NECK	NORMAL		
NECK LYMPHATICS / SALIVARY GLANDS	NOT ENLARGED OR TENDER		
THYROID GLAND	NOT ENLARGED		
CAROTID PULSATION	NORMAL		
TEMPERATURE	NORMAL		
PULSE	76/MIN.REGULAR, ALL PERIPHERAL PULSES WELL FELT, NO CAROTID BRUIT		
RESPIRATORY RATE	NORMAL		
CARDIOVASCULAR SYSTEM			
BP	160/90 MM HG (SUPINE)		mm/Hg
PERICARDIUM	NORMAL		
APEX BEAT	NORMAL		
HEART SOUNDS	NORMAL		
MURMURS	ABSENT		
RESPIRATORY SYSTEM			
SIZE AND SHAPE OF CHEST	NORMAL		
MOVEMENTS OF CHEST	SYMMETRICAL		
BREATH SOUNDS INTENSITY	NORMAL		
BREATH SOUNDS QUALITY	VESICULAR (NORMAL)		
ADDED SOUNDS	ABSENT		
PER ABDOMEN			
APPEARANCE	NORMAL		
VENOUS PROMINENCE	ABSENT		
LIVER	NOT PALPABLE		
SPLEEN	NOT PALPABLE		
HERNIA	ABSENT		
CENTRAL NERVOUS SYSTEM			
HIGHER FUNCTIONS	NORMAL		

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CRANIAL NERVES	NORMAL		
CEREBELLAR FUNCTIONS	NORMAL		
SENSORY SYSTEM	NORMAL		
MOTOR SYSTEM	NORMAL		
REFLEXES	NORMAL		
MUSCULOSKELETAL SYSTEM			
SPINE	NORMAL		
JOINTS	NORMAL		
BASIC EYE EXAMINATION			
CONJUNCTIVA	NORMAL		
EYELIDS	NORMAL		
EYE MOVEMENTS	NORMAL		
CORNEA	NORMAL		
DISTANT VISION RIGHT EYE WITHOUT GLASSES	REDUCED VISUAL ACUITY 6/18		
DISTANT VISION LEFT EYE WITHOUT GLASSES	REDUCED VISUAL ACUITY 6/18		
DISTANT VISION RIGHT EYE WITH GLASSES	GLASSES NOT BROUGHT.		
DISTANT VISION LEFT EYE WITH GLASSES	GLASSES NOT BROUGHT.		
NEAR VISION RIGHT EYE WITHOUT GLASSES	REDUCED VISUAL ACUITY N/36		
NEAR VISION LEFT EYE WITHOUT GLASSES	REDUCED VISUAL ACUITY N/36		
NEAR VISION RIGHT EYE WITH GLASSES	GLASSES NOT BROUGHT.		
NEAR VISION LEFT EYE WITH GLASSES	GLASSES NOT BROUGHT.		
COLOUR VISION	NORMAL		
SUMMARY			
RELEVANT HISTORY	NOT SIGNIFICANT		
RELEVANT GP EXAMINATION FINDINGS	OVERWEIGHT : BMI 28 REDUCED ACUITY FOR DISTANT AND NEAR VISION.		
REMARKS / RECOMMENDATIONS	1) TAKEN ANTIHYPERTENSION MEDICATIONS REGULARLY. 2) REGULAR FOLLOW UP WITH PHYSICIAN FOR BP CONTROL & DYSLIPIDEMIA & IN VIEW OF ECG FINFINGS. 3) UROLOGY CONSULT FOR RENAL CALCULUS. 4) STRICT LOW FAT,LOW CALORIE, LOW CARBOHYDRATE, HIGH FIBRE DIET, 5) REGULAR EXERCISE.REGULAR WALK FOR 30-40 MIN DAILY. 6) ADD YOGA, PRANAYAM MEDITATION TO DAILY ROUTINE. 7) REPEAT LIPID PROFILE AFTER 3 MONTHS OF DIET AND EXERCISE.		

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Interpretation(s)
BLOOD COUNTS,EDTA WHOLE BLOOD-
The cell morphology is well preservec for 24hrs. However after 24-48 hrs a progressive increase ir MCV and HCT is observed leading to a decrease in MCHC. A direct smear is recommendec for an accurate differential count anc for examination of RBC morphology.
WBC DIFFERENTIAL COUNT - NLR-
The optimal threshold of 3.3 for NLR showec a prognostic possibility of clinical symptoms to change from milc to severe in COVID positive patients. When age = 49.5 years old anc 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 anc predictive role of NLR, d-NLR anc PLR in COVID-19 patients ; A.-P. Yang, et al.; International Immunopharmacology 84 (2020) 106504
This ratio element is a calculatec parameter anc out of NABL scope.
MICROSCOPIC EXAMINATION, URINE-
Routine urine analysis assists in screening and diagnosis of various metabolic, urological, kidney and liver disorders
Protein: Elevated proteins car be an early sign of kidney disease. Urinary protein excretion can also be temporarily elevated by strenuous exercise, orthostatic proteinuria, dehydration, urinary tract infections anc 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 car 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 acic 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 concentratec the urine is. Increasec 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 anc cirrhosis and in cases of hemolytic anemia
ERYTHRO SEDIMENTATION RATE, BLOOD-
Erythrocyte sedimentation rate (ESR) is a non - specific phenomena anc is clinically useful in the diagnosis anc monitoring of disorders associated with an increasec production of acute phase reactants. The ESR is increasec 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 anc drugs (eg. corticosteroids, contraceptives). It is especially low (0-1mm) in polycythaemia, hypofibrinogenemia or congestive cardiac failure anc 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"
GLYCOSYLATED HEMOGLOBIN, EDTA WHOLE BLOOD-
Glycosylatec hemoglobin (GHb) has been firmly established as an index of long-term blood glucose concentrations anc as a measure of the risk for the development of complications in patients with diabetes mellitus. Formation of GHb is essentially irreversible, anc the concentration in the blood depends on both the life span of the rec blood cell (average 120 days) anc 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 increasec glycated hemoglobin values due to a somewhat longer life span of the rec cells.
Glycosylatec hemoglobins results from patients with HbSS, HbCC, and HbSC and HbD must be interpreted with caution, given the pathological processes, including anemia, increased rec cell turnover, transfusion requirements, that adversely impact HbA1c as a marker of long-term glycemic control. Ir 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.

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Patient Ref. No. 775000001633777

CLIENT CODE : C000138394

CLIENT'S NAME AND ADDRESS :
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PATIENT NAME : BAIT RAGHUNATH RATNU PATIENT ID : **BAITM051166181**

ACCESSION NO : **0181VI000339** AGE : 55 Years SEX : Male

DRAWN : RECEIVED : 10/09/2022 10:42 REPORTED : 13/09/2022 15:19

REFERRING DOCTOR : SELF CLIENT PATIENT ID :

CLINICAL INFORMATION :

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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
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.
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
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.

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Type 2 DM.
Metabolic syndrome.

Causes of decreasec levels
• Low Zinc Intake
• OCP's
• Multiple Sclerosis

Nutritional tips to manage increased Uric acic 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 blooc plasma. It is produced in the liver. Albumin constitutes about half of the blooc serum protein. Low blood albumin levels (hypoalbuminemia) can be causec 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-
Sodium levels are Increased in dehydration, cushing's syndrome, aldosteronism & decreased ir 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 associatec 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 prolongec vomiting,

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 anc its prohormone thyroxine (T4) is activated by thyroid-stimulating hormone (TSH), which is releasec 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 ir the body. Excessive secretion of thyroxine in the body is hyperthyroidism, and deficient secretion is callec hypothyroidism. Most of the thyroid hormone in blooc is bound to transport proteins. Only a very small fraction of the circulating hormone is free anc biologically active.
In primary hypothyroidism, TSH levels are significantly elevated, while in secondary and tertiary hypothyroidism, TSH levels are low.
Below mentionec 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
3rc Trimester	6.6 - 15.5	0.3 - 3.0	100 - 260

Below mentionec are the guidelines for age related reference ranges for T3 anc 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 documentec in the pediatric population including the infant age group.
Kindly note: Methoc specific reference ranges are appearing or the report under biological reference range.

Reference:
1. Burtis C.A., Ashwooc E. R. Bruns D.E. Teitz textbook of Clinical Chemistry anc Molecular Diagnostics, 4th Edition.
2. Gowenlock A.H. Varley's Practical Clinical Biochemistry, 6th Edition.
3. Behrman R.E. Kliegman R.M., Jenson H. B. Nelsor Text Book of Pediatrics, 17th Edition

ABO GROUP & RH TYPE, EDTA WHOLE BLOOD-
Blooc group is identified by antigens and antibodies present in the blood. Antigens are protein molecules found on the surface of red blood cells. Antibodies are found in

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plasma. To determine blooc group, red cells are mixed with different antibody solutions to give A,B,O or AB.

Disclaimer: "Please note, as the results of previous ABO and Rh group (Blood Group) for pregnant women are not available, please check with the patient records for availability of the same."

The test is performed by both forward as well as reverse grouping methods.
MEDICAL
HISTORY-**********
THIS REPORT CARRIES THE SIGNATURE OF OUR LABORATORY DIRECTOR. THIS IS AN INVIOLABLE FEATURE OF OUR LAB MANAGEMENT SOFTWARE. HOWEVER, ALL EXAMINATIONS AND INVESTIGATIONS HAVE BEEN CONDUCTED BY OUR PANEL OF DOCTORS.

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MEDI WHEEL FULL BODY HEALTH CHECK UP ABOVE 40 MALE

ULTRASOUND ABDOMEN

ULTRASOUND ABDOMEN
GRADE I FATTY LIVER.
BILATERAL BRIGHT KIDNEYS SERUM CREATININE CORRELATION.
RIGHT RENAL NON OBSTRUCTING CALCULUS.

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

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Consultant Pathologist

Dr. Ushma Wartikar
Consultant Pathologist

Dr.(Mrs)Neelu K Bhojani
Lab Head

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