Patient Ref. No. 181000000297084

CLIENT CODE : C000138394

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

PATIENT NAME : K RAJU NAIK PATIENT ID : **KRAJM010192181**

ACCESSION NO : **0181VC001229** AGE : 30 Years SEX : Male

DRAWN : RECEIVED : 28/03/2022 09:40 REPORTED : 30/03/2022 16:20

REFERRING DOCTOR : SELF CLIENT PATIENT ID :

Test Report Status **Final**	Results		Biological Reference Interval	Units
MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE				
BLOOD COUNTS,EDTA WHOLE BLOOD				
HEMOGLOBIN	15.0		13.0 - 17.0	g/dL
METHOD : SLS- HEMOGLOBIN DETECTION METHOD				
RED BLOOD CELL COUNT	**5.57**	**High**	4.5 - 5.5	mil/µL
METHOD : HYDRODYNAMIC FOCUSING BY DC DETECTION				
WHITE BLOOD CELL COUNT	8.88		4.0 - 10.0	thou/µL
METHOD : FLUORESCENCE FLOW CYTOMETRY				
PLATELET COUNT	**413**	**High**	150 - 410	thou/µL
METHOD : HYDRODYNAMIC FOCUSING BY DC DETECTION				
RBC AND PLATELET INDICES				
HEMATOCRIT	47.3		40.0 - 50.0	%
METHOD : CUMULATIVE PULSE HEIGHT DETECTION METHOD				
MEAN CORPUSCULAR VOL	84.9		83.0 - 101.0	fL
METHOD : CALCULATED FROM RBC & HCT				
MEAN CORPUSCULAR HGB.	**26.9**	**Low**	27.0 - 32.0	pg
METHOD : CALCULATED FROM THE RBC & HGB				
MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION	31.7		31.5 - 34.5	g/dL
METHOD : CALCULATED FROM THE HGB & HCT				
MENTZER INDEX	15.2			
RED CELL DISTRIBUTION WIDTH	13.5		11.6 - 14.0	%
METHOD : CALCULATED FROM RBC SIZE DISTRIBUTION CURVE				
MEAN PLATELET VOLUME	9.5		6.8 - 10.9	fL
METHOD : CALCULATED FROM PLATELET COUNT & PLATELET HEMATOCRIT				
WBC DIFFERENTIAL COUNT - NLR				
SEGMENTED NEUTROPHILS	62		40 - 80	%
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
ABSOLUTE NEUTROPHIL COUNT	5.49		2.0 - 7.0	thou/µL
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
LYMPHOCYTES	33		20 - 40	%
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
ABSOLUTE LYMPHOCYTE COUNT	2.94		1.0 - 3.0	thou/µL
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
NEUTROPHIL LYMPHOCYTE RATIO (NLR)	1.9			
EOSINOPHILS	1		1 - 6	%
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				

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ABSOLUTE EOSINOPHIL COUNT	0.08		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	0.32		0.2 - 1.0	thou/µL
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
DIFFERENTIAL COUNT PERFORMED ON:	EDTA SMEAR			
MORPHOLOGY				
RBC	NORMOCYTIC NORMOCHROMIC			
WBC	NORMAL MORPHOLOGY			
METHOD : MICROSCOPIC EXAMINATION				
PLATELETS	ADEQUATE			
ERYTHRO SEDIMENTATION RATE, BLOOD				
SEDIMENTATION RATE (ESR)	09		0 - 14	mm at 1 hr
METHOD : WESTERGREN METHOD				
GLUCOSE, FASTING, PLASMA				
GLUCOSE, FASTING, PLASMA	**116**	**High**	74.0 - 106.0	mg/dL
METHOD : GLUCOSE OXIDASE				
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, POST-PRANDIAL, PLASMA				
GLUCOSE, POST-PRANDIAL, PLASMA	98		74 - 140	mg/dL
METHOD : GLUCOSE OXIDASE				
CORONARY RISK PROFILE (LIPID PROFILE), SERUM.				
CHOLESTEROL	136		< 200 Desirable 200 - 239 Borderline High >/= 240 High	mg/dL
METHOD : CHOLESTEROL OXIDASE				
TRIGLYCERIDES	96		Normal: <150 Borderline high: 150 - 199 High: 200 - 499 Very high: > or = 500	mg/dL
METHOD : ENZYMATIC ASSAY				

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HDL CHOLESTEROL	41		< 40 Low >/=60 High	mg/dL
METHOD : DIRECT- NON IMMUNOLOGICAL				
DIRECT LDL CHOLESTEROL	93		< 100 Optimal 100 - 129 Near or above optimal 130 - 159 Borderline High 160 - 189 High >/= 190 Very High	mg/dL
METHOD : ENZYMATIC ASSAY				
NON HDL CHOLESTEROL	95			mg/dL
METHOD : CALCULATED PARAMETER				
CHOL/HDL RATIO	3.3		3.3- 4.4 Low Risk 4.5 -7.0 Average Risk 7.1 -11.0 Moderate Risk > 11.0 High Risk	
METHOD : CALCULATED PARAMETER				
LDL/HDL RATIO	2.3		0.5 - 3.0 Desirable/Low Risk 3.1 - 6.0 Borderline/Moderate Risk >6.0 High Risk	
METHOD : CALCULATED PARAMETER				
VERY LOW DENSITY LIPOPROTEIN	19.0		10 - 35	mg/dL
METHOD : CALCULATED PARAMETER				
LIVER FUNCTION PROFILE, SERUM				
BILIRUBIN, TOTAL	**1.63**	**High**	0.2 - 1.3	mg/dL
METHOD : DIPHYLLINE DIAZONIUM SALTS				
BILIRUBIN, DIRECT	0.10		0.0 - 0.3	mg/dL
METHOD : DIPHYLLINE DIAZONIUM SALTS				
BILIRUBIN, INDIRECT	**1.53**	**High**	0.0 - 1.1	mg/dL
METHOD : DIPHYLLINE DIAZONIUM SALTS				
TOTAL PROTEIN	7.8		6.3 - 8.3	g/dL
ALBUMIN	4.5		3.5 - 5.0	g/dL
GLOBULIN	3.4		2.0 - 3.5	g/dL
ALBUMIN/GLOBULIN RATIO	1.4		1.0 - 2.0	RATIO
ASPARTATE AMINOTRANSFERASE (AST/SGOT)	24		17 - 59	U/L
ALANINE AMINOTRANSFERASE (ALT/SGPT)	28		< 50.0	U/L
ALKALINE PHOSPHATASE	**140**	**High**	38 - 126	U/L
GAMMA GLUTAMYL TRANSFERASE (GGT)	35		15 - 73	U/L
LACTATE DEHYDROGENASE	154		120 - 246	U/L
SERUM BLOOD UREA NITROGEN				
BLOOD UREA NITROGEN	12		9.0 - 20.0	mg/dL

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METHOD : UREASE WITH INDICATOR DYE				
CREATININE, SERUM				
CREATININE	0.89		0.66 - 1.25	mg/dL
METHOD : ENZYMETIC IDMS				
BUN/CREAT RATIO				
BUN/CREAT RATIO	13.48			
URIC ACID, SERUM				
URIC ACID	7.0		3.5 - 8.5	mg/dL
METHOD : URICASE UV				
TOTAL PROTEIN, SERUM				
TOTAL PROTEIN	7.8		6.3 - 8.30	g/dL
METHOD : BIURET, END POINT				
ALBUMIN, SERUM				
ALBUMIN	4.5		3.5 - 5.0	g/dL
METHOD : BCG DYE BINDING METHOD				
GLOBULIN				
GLOBULIN	3.4		2.0 - 3.5	g/dL
METHOD : CALCULATED PARAMETER				
ELECTROLYTES (NA/K/CL), SERUM				
SODIUM	**147**	**High**	137 - 145	mmol/L
METHOD : ION SELECTIVE ELECTRODE TECHNOLOGY				
POTASSIUM	5.0		3.50 - 5.10	mmol/L
CHLORIDE	103		98 - 107	mmol/L
URINALYSIS				
COLOR	PALE YELLOW			
METHOD : VISUAL INSPECTION				
APPEARANCE	CLEAR			
METHOD : VISUAL INSPECTION				
PH	5.5		4.7 - 7.5	
METHOD : DOUBLE INDICATOR PRINCIPLE				
SPECIFIC GRAVITY	1.025		1.003 - 1.035	
METHOD : IONIC CONCENTRATION METHOD				
GLUCOSE	NOT DETECTED		NOT DETECTED	
METHOD : GLUCOSE OXIDASE PEROXIDASE				
PROTEIN	NOT DETECTED		NOT DETECTED	
METHOD : TETRA BROMOPHENOL BLUE/SULFOSALICYLIC ACID				
KETONES	NOT DETECTED		NOT DETECTED	

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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			
PUS CELL (WBC'S)	1-2	0-5	/HPF
METHOD : MICROSCOPIC EXAMINATION			
EPITHELIAL CELLS	1-2	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			
THYROID PANEL, SERUM			
T3	102.2	58 - 159	ng/dL
METHOD : CHEMILUMINESCENT MICROPARTICLE IMMUNO ASSAY			
T4	8.27	4.87 - 11.71	µg/dL
METHOD : CHEMILUMINESCENT MICROPARTICLE IMMUNO ASSAY			
TSH 3RD GENERATION	1.158	0.350 - 4.940	µIU/mL
METHOD : CHEMILUMINESCENT MICROPARTICLE IMMUNO ASSAY			
STOOL: OVA & PARASITE			
COLOUR	BROWN		
METHOD : VISUAL			
CONSISTENCY	WELL FORMED		
METHOD : VISUAL			
ODOUR	FAECAL		
METHOD : PHYSICAL			
MUCUS	NOT DETECTED	NOT DETECTED	
METHOD : VISUAL			
VISIBLE BLOOD	ABSENT	ABSENT	
METHOD : VISUAL			
POLYMORPHONUCLEAR LEUKOCYTES	1-2	0 - 5	/HPF

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METHOD : MICROSCOPIC EXAMINATION			
RED BLOOD CELLS	NOT DETECTED	NOT DETECTED	/HPF
METHOD : MICROSCOPIC EXAMINATION			
TROPHOZOITES	NOT DETECTED	NOT DETECTED	
METHOD : MICROSCOPIC EXAMINATION			
CYSTS	NOT DETECTED	NOT DETECTED	
METHOD : MICROSCOPIC EXAMINATION			
OVA	NOT DETECTED		
METHOD : MICROSCOPIC EXAMINATION			
LARVAE	NOT DETECTED	NOT DETECTED	
METHOD : MICROSCOPIC EXAMINATION			
OCCULT BLOOD	NOT DETECTED	NOT DETECTED	
METHOD : HEMOSPOT			
REMARK	NO OVA CYST SEEN AFTER PERFORMING CONCENTRATION TECHNIQUE FOR STOOL SAMPLE.		
ABO GROUP & RH TYPE, EDTA WHOLE BLOOD			
ABO GROUP	TYPE O		
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	TMT : NEGATIVE		
ECG			
ECG	WITHIN NORMAL LIMITS		
MEDICAL HISTORY			
RELEVANT PRESENT HISTORY	NOT SIGNIFICANT		
RELEVANT PAST HISTORY	OPERATED FOR FRACTURELEFT HUMERUS 15 MONTHS BACK. PLATE IN SITU.COVID IN DEC 2021.HOME QUARANITNED.		
RELEVANT PERSONAL HISTORY	UNMARRIED / MIXED DIET / NO ALLERGIES / NO SMOKING / OCC ALCOHOL.		
RELEVANT FAMILY HISTORY	DIABETES : FATHER.		
HISTORY OF MEDICATIONS	NOT SIGNIFICANT		
ANTHROPOMETRIC DATA & BMI			
HEIGHT IN METERS	1.72		mts
WEIGHT IN KGS.	81		Kgs

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BMI	27	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		
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	78/MIN.REGULAR, ALL PERIPHERAL PULSES WELL FELT, NO CAROTID BRUIT		
RESPIRATORY RATE	NORMAL		
CARDIOVASCULAR SYSTEM			
BP	120/80 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		

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VENOUS PROMINENCE	ABSENT		
LIVER	NOT PALPABLE		
SPLEEN	NOT PALPABLE		
HERNIA	ABSENT		
CENTRAL NERVOUS SYSTEM			
HIGHER FUNCTIONS	NORMAL		
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 6/24		
DISTANT VISION LEFT EYE WITHOUT GLASSES	REDUCED VISUAL ACUITY 6/24		
DISTANT VISION RIGHT EYE WITH GLASSES	GLASSES NOT BROUGHT		
DISTANT VISION LEFT EYE WITH GLASSES	GLASSES NOT BROUGHT		
NEAR VISION RIGHT EYE WITHOUT GLASSES	WITHIN NORMAL LIMIT		
NEAR VISION LEFT EYE WITHOUT GLASSES	WITHIN NORMAL LIMIT		
NEAR VISION RIGHT EYE WITH GLASSES	WITHIN NORMAL LIMIT		
NEAR VISION LEFT EYE WITH GLASSES	WITHIN NORMAL LIMIT		
COLOUR VISION	NORMAL		
SUMMARY			
RELEVANT HISTORY	NOT SIGNIFICANT		
RELEVANT GP EXAMINATION FINDINGS	OVERWEIGHT : BMI 27		

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RELEVANT LAB INVESTIGATIONS	RED BLOOD CELL COUNT : 5.57 PLATELET COUNT : 413 MEAN CORPUSCULAR HGB. : 26.9 F.B.SUGAR : 116 HBA1C : 6.0 MEAN PLASMA GLUCOSE : 125.5 BILIRUBIN, TOTAL : 1.63 BILIRUBIN, INDIRECT : 1.53 ALKALINE PHOSPHATASE : 140 SODIUM : 147		
RELEVANT NON PATHOLOGY DIAGNOSTICS	ECG : NORMAL X-RAY : NORMAL TMT : NEGATIVE. USG : GRADE I FATTY LIVER		
REMARKS / RECOMMENDATIONS	1) LOW CALORIE, HIGH FIBRE DIET, REGULAR EXERCISE. 2) REPEAT B.SUGAR,LIVER PROFILE AFTER 3 MONTHS OF DIET AND EXERCISE. 3) OPHTHALMOLOGY CONSULT FOR REDUCED VISUAL ACUITY.		

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"
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

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PATIENT NAME : K RAJU NAIK PATIENT ID : **KRAJM010192181**

ACCESSION NO : **0181VC001229** AGE : 30 Years SEX : Male

DRAWN : RECEIVED : 28/03/2022 09:40 REPORTED : 30/03/2022 16:20

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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.
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.
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.

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.
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,

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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.
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-

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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,
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
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 - 2.0	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
STOOL: OVA & PARASITE-
Acute infective diarrhoea and gastroenteritis (diarrhoea with vomiting) are major causes of ill health and premature death in developing countries. Loss of water and electrolytes from the body can lead to severe dehydration which if untreated, can be rapidly fatal in young children, especially that are malnourished, hypoglycaemic, and generally in poor health.

Laboratory diagnosis of parasitic infection is mainly based on microscopic examination and the gross examination of the stool specimen. Depending on the nature of the parasite, the microscopic observations include the identification of cysts, ova, trophozoites, larvae or portions of adult structure. The two classes of parasites that cause human infection are the Protozoa and Helminths. The protozoan infections include amoebiasis mainly caused by Entamoeba histolytica and giardiasis caused by Giardia lamblia. The common helminthic parasites are Trichuris trichiura, Ascaris lumbricoides, Strongyloides stercoralis, Taenia sp. etc
ABO GROUP & RH TYPE, EDTA WHOLE BLOOD-
Blood 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 plasma. To determine blood 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."

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The test is performed by both forward as well as reverse grouping methods.
MEDICAL
HISTORY-**********
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MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 40 MALE

ULTRASOUND ABDOMEN

ULTRASOUND ABDOMEN

GRADE I FATTY LIVER.

****End Of Report****
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Consultant Microbiologist	**Consultant Pathologist**	**Consultant Pathologist**	.

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