



Patient Ref. No. 775000001580647

CLIENT CODE : C000138394

CLIENT'S NAME AND ADDRESS :
ACROFEMI HEALTHCARE LTD (MEDI WHEEL)
F-703, F-703, LADO SARAI, MEHRAULI
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DELHI INDIA
8800465156

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THANE, 400602
MAHARASHTRA, INDIA
Tel : 9111591115, Fax : CIN - U74899PB1995PLC045956
Email : customercare.thane@srl.in

PATIENT NAME : THARANIPRAKASH M K

PATIENT ID : THARM120587181

ACCESSION NO : 0181VH000536 AGE : 35 Years SEX : Male

DRAWN : RECEIVED : 13/08/2022 08:52 REPORTED : 18/08/2022 14:35

REFERRING DOCTOR : SELF

CLIENT PATIENT ID :

Test Report Status	Final	Results	Biological Reference Interval	Units
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MEDI WHEEL FULL BODY HEALTH CHECK UP BELOW 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 15.8 13.0 - 17.0 g/dL
METHOD : SLS- HEMOGLOBIN DETECTION METHOD

RED BLOOD CELL COUNT 5.05 4.5 - 5.5 ml/ μ L
METHOD : HYDRODYNAMIC FOCUSING BY DC DETECTION

WHITE BLOOD CELL COUNT 8.37 4.0 - 10.0 thou/ μ L
METHOD : FLUORESCENCE FLOW CYTOMETRY

PLATELET COUNT 310 150 - 410 thou/ μ L
METHOD : HYDRODYNAMIC FOCUSING BY DC DETECTION

RBC AND PLATELET INDICES

HEMATOCRIT 45.7 40.0 - 50.0 %
METHOD : CUMULATIVE PULSE HEIGHT DETECTION METHOD

MEAN CORPUSCULAR VOL 90.5 83.0 - 101.0 fL
METHOD : CALCULATED FROM RBC & HCT

MEAN CORPUSCULAR HGB 31.3 27.0 - 32.0 pg
METHOD : CALCULATED FROM THE RBC & HGB

MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION 34.6 High 31.5 - 34.5 g/dL
METHOD : CALCULATED FROM THE HGB & HCT

MENTZER INDEX 17.9

RED CELL DISTRIBUTION WIDTH 12.4 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

CHEMICAL EXAMINATION, URINE

PH 6.0 4.7 - 7.5
METHOD : DOUBLE INDICATOR PRINCIPLE

PROTEIN NOT DETECTED NOT DETECTED
METHOD : TETRA BROMOPHENOL BLUE/SULFOSALICYLIC ACID



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GLUCOSE		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		54	40 - 80	%
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
ABSOLUTE NEUTROPHIL COUNT		4.50	2.0 - 7.0	thou/ μ L
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
LYMPHOCYTES		37	20 - 40	%
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
ABSOLUTE LYMPHOCYTE COUNT		3.09	High 1.0 - 3.0	thou/ μ L
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
NEUTROPHIL LYMPHOCYTE RATIC (NLR)		1.5		
EOSINOPHILS		6	1 - 6	%
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
ABSOLUTE EOSINOPHIL COUNT		0.49	0.02 - 0.50	thou/ μ L
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
MONOCYTES		3	2 - 10	%
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
ABSOLUTE MONOCYTE COUNT		0.26	0.2 - 1.0	thou/ μ L
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
DIFFERENTIAL COUNT PERFORMED ON:				
MICROSCOPIC EXAMINATION, URINE				
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		



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METHOD : MICROSCOPIC EXAMINATION

CRYSTALS NOT DETECTED

METHOD : MICROSCOPIC EXAMINATION

BACTERIA NOT DETECTED NOT DETECTED

METHOD : MICROSCOPIC EXAMINATION

YEAST NOT DETECTED NOT DETECTED

REMARKS PRESENCE OF URINARY GLUCOSE RECHECKED BY MANUAL METHOD.

MORPHOLOGY

RBC NORMOCYTIC NORMOCHROMIC

WBC NORMAL MORPHOLOGY

METHOD : MICROSCOPIC EXAMINATION

PLATELETS ADEQUATE

ERYTHRO SEDIMENTATION RATE, BLOOD

SEDIMENTATION RATE (ESR) 10 0 - 14 mm at 1 hr

METHOD : WESTERNGREN METHOD

GLUCOSE, FASTING, PLASMA

GLUCOSE, FASTING, PLASMA 139 High Normal 75 - 99 mg/dL
Pre-diabetics: 100 - 125
Diabetic: > or = 126

METHOD : ENZYMATIC REFERENCE METHOD WITH HEXOKINASE

GLYCOSYLATED HEMOGLOBIN, EDTA WHOLE BLOOD

GLYCOSYLATED HEMOGLOBIN (HBA1C) 7.2 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 159.9 High < 116.0 mg/dL

METHOD : CALCULATED PARAMETER

GLUCOSE, POST-PRANDIAL, PLASMA

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

METHOD : ENZYMATIC REFERENCE METHOD WITH HEXOKINASE

LIVER FUNCTION PROFILE, SERUM

BILIRUBIN, TOTAL 0.84 Upto 1.2 mg/dL

METHOD : COLORIMETRIC DIAZO

BILIRUBIN, DIRECT 0.30 < 0.30 mg/dL

BILIRUBIN, INDIRECT 0.54 0.1 - 1.0 mg/dL

TOTAL PROTEIN 7.2 6.0 - 8.0 g/dL

METHOD : COLORIMETRIC



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ALBUMIN		4.6	3.97 - 4.94	g/dL
METHOD : COLORIMETRIC				
GLOBULIN		2.6	2.0 - 3.5	g/dL
ALBUMIN/GLOBULIN RATIO		1.8	1.0 - 2.1	RATIO
ASPARTATE AMINOTRANSFERASE (AST/SGOT)		26	< OR = 50	U/L
METHOD : UV ABSORBANCE				
ALANINE AMINOTRANSFERASE (ALT/SGPT)		36	< OR = 50	U/L
METHOD : UV ABSORBANCE				
ALKALINE PHOSPHATASE		70	40 - 129	U/L
METHOD : COLORIMETRIC				
GAMMA GLUTAMYL TRANSFERASE (GGT)		159	High 0 - 60	U/L
METHOD : ENZYMATIC, COLORIMETRIC				
LACTATE DEHYDROGENASE		134	125 - 220	U/L
METHOD : UV ABSORBANCE				
SERUM BLOOD UREA NITROGEN				
BLOOD UREA NITROGEN		12	6 - 20	mg/dL
METHOD : ENZYMATIC ASSAY				
CREATININE, SERUM				
CREATININE		0.78	0.7 - 1.2	mg/dL
METHOD : COLORIMETRIC				
BUN/CREAT RATIO				
BUN/CREAT RATIO		15.38	High 8.0 - 15.0	
URIC ACID, SERUM				
URIC ACID		6.3	3.4 - 7.0	mg/dL
METHOD : ENZYMATIC COLORIMETRIC ASSAY				
TOTAL PROTEIN, SERUM				
TOTAL PROTEIN		7.2	6.0 - 8.0	g/dL
METHOD : COLORIMETRIC				
ALBUMIN, SERUM				
ALBUMIN		4.6	3.97 - 4.94	g/dL
METHOD : COLORIMETRIC				
GLOBULIN				
GLOBULIN		2.6	2.0 - 3.5	g/dL
ELECTROLYTES (NA/K/CL), SERUM				
SODIUM		139	136 - 145	mmol/L
POTASSIUM		4.41	3.5 - 4.5	mmol/L
CHLORIDE		104	98 - 107	mmol/L



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THYROID PANEL, SERUM

T3	133.0	80 - 200	ng/dL
METHOD : ELECTROCHEMILUMINESCENCE			
T4	8.27	5.1 - 14.1	µg/dL
METHOD : ELECTROCHEMILUMINESCENCE			
TSH 3RD GENERATION	1.230	0.27 - 4.2	µIU/mL
METHOD : ELECTROCHEMILUMINESCENCE			

STOOL: OVA & PARASITE

COLOUR	BROWN		
METHOD : VISUAL			
CONSISTENCY	SEMI FORMED		
METHOD : VISUAL			
ODOUR	FAECAL		
METHOD : PHYSICAL			
MUCUS	NOT DETECTED	NOT DETECTED	
METHOD : VISUAL			
VISIBLE BLOOD	ABSENT	ABSENT	
METHOD : VISUAL			
POLYMPHONUCLEAR LEUKOCYTES	2 - 3	0 - 5	/HPF
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



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METHOD : GEL COLUMN AGGLUTINATION METHOD.

XRAY-CHEST

IMPRESSION NO ABNORMALITY DETECTED

TMT OR ECHO

TMT OR ECHO NEGATIVE

ECG

ECG SINUS TACHYCARDIA.

MEDICAL HISTORY

RELEVANT PRESENT HISTORY DIABETES SINCE 4 YEARS.

RELEVANT PAST HISTORY H/O URI SINCE 1 WEEK.

RELEVANT PERSONAL HISTORY MARRIED / 1 CHILD / VEG DIET / NO ALLERGIES / NO SMOKING / NC ALCOHOL.

RELEVANT FAMILY HISTORY DIABETS : BOTH PARENTS.

HISTORY OF MEDICATIONS GLYCOMET GP : 1-0-1.
VOGLIBOSE : 0-1-0.

ANTHROPOMETRIC DATA & BMI

HEIGHT IN METERS 1.72 mts

WEIGHT IN KGS. 70 Kgs

BMI 24
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 HEALTHY

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



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PULSE	96/MIN.REGULAR, ALL PERIPHERAL PULSES WELL FELT, NO CAROTID BRUIT			
RESPIRATORY RATE	NORMAL			
CARDIOVASCULAR SYSTEM				
BP	130/80 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			
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			



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EYE MOVEMENTS		NORMAL		
CORNEA		NORMAL		
DISTANT VISION RIGHT EYE WITHOUT GLASSES		WITHIN NORMAL LIMIT		
DISTANT VISION LEFT EYE WITHOUT GLASSES		WITHIN NORMAL LIMIT		
NEAR VISION RIGHT EYE WITHOUT GLASSES		WITHIN NORMAL LIMIT		
NEAR VISION LEFT EYE WITHOUT GLASSES		WITHIN NORMAL LIMIT		
COLOUR VISION		NORMAL		

SUMMARY

RELEVANT HISTORY NOT SIGNIFICANT

RELEVANT GP EXAMINATION FINDINGS NOT SIGNIFICANT

REMARKS / RECOMMENDATIONS
1) FOLLOW UP WITH PHYSICIAN FOR B.SUGAR CONTROL.
2) LOW FAT, LOW CARBOHYDRATE, HIGH FIBRE DIET.
3) REGULAR EXERCISE, REGULAR WALK FOR 30-40 MIN DAILY.
4) REPEAT B.SUGAR AFTER 3 MONTHS OF DIET AND EXERCISE.
5) TO DO LIPID PROFILE.

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.

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.)

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.

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



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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 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 HbS5, 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 result 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 Hypophosphatemia, 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-



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PATIENT NAME : THARANIPRAKASH M K

PATIENT ID : THARM120587181

ACCESSION NO : 0181VH000536 AGE : 35 Years SEX : Male

DRAWN : RECEIVED : 13/08/2022 08:52 REPORTED : 18/08/2022 14:35

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CLIENT PATIENT ID :

Table with 4 columns: Test Report Status (Final), Results, Biological Reference Interval, Units

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, hemodialysis, 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 in Addison's disease, hypopituitarism, liver disease. Hypokalemia (low K) is common in vomiting, diarrhea, alcoholism, tolic 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 hyperfunction, 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.

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

Table with 4 columns: Levels in, TOTAL T4 (µg/dL), TSH3G (µIU/mL), TOTAL T3 (ng/dL). Rows for Pregnancy, First Trimester, 2nd Trimester, 3rd Trimester.

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

T3 T4



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Test Report Status	Final	Results	Biological Reference Interval	Units
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(ng/dL)	(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:
- Burbs C.A., Ashwood E. R. Bruns D.E. Tetz textbook of Clinical Chemistry and Molecular Diagnostics, 4th Edition.
 - Gowenlock A.H. Varley's Practical Clinical Biochemistry, 6th Edition.
 - Behrman R.E. Kliegman 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."

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 INVOLABLE 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 BELOW 40 MALE

ULTRASOUND ABDOMEN

ULTRASOUND ABDOMEN

MILD HEPATOMEGALY WITH GRADE I FATTY LIVER.

****End Of Report****

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

Dr. Sheetal Sawant
Consultant Microbiologist

Dr. Ushma Wartikar
Consultant Pathologist

Dr. (Mrs) Neelu K Bhojani
Lab Head



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