



Patient Ref. No. 775000001663499

CLIENT CODE : C000138394

CLIENT'S NAME AND ADDRESS :  
ACROFEMI HEALTHCARE LTD ( MEDI WHEEL )  
F-703, F-703, LADO SARAI, MEHRAULI  
SOUTH WEST DELHI  
NEW DELHI 110030  
DELHI INDIA  
8800465156

SRL Ltd  
S.K. Tower, Hari Niwas, LBS Marg  
THANE, 400602  
MAHARASHTRA, INDIA  
Tel : 9111591115, Fax : CIN - U74899PB1995PLC045956  
Email : customercare.thane@srl.in

PATIENT NAME : RAJASHRI GOSAVI

PATIENT ID : RAJAM310184181

ACCESSION NO : 0181VI000893 AGE : 38 Years SEX : Male

DRAWN : RECEIVED : 24/09/2022 10:04 REPORTED : 27/09/2022 15:56

REFERRING DOCTOR : SELF

CLIENT PATIENT ID :

Test Report Status	Final	Results	Biological Reference Interval	Units
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**MEDI WHEEL FULL BODY HEALTH CHECKUP BELOW 40FFEMALE**

**PHYSICAL EXAMINATION, URINE**

COLOR PALE YELLOW

METHOD : VISUAL INSPECTION

APPEARANCE CLEAR

METHOD : VISUAL INSPECTION

SPECIFIC GRAVITY 1.010 1.003 - 1.035

METHOD : IONIC CONCENTRATION METHOD

**BLOOD COUNTS, EDTA WHOLE BLOOD**

HEMOGLOBIN 13.6 13.0 - 17.0 g/dL

METHOD : SLS- HEMOGLOBIN DETECTION METHOD

RED BLOOD CELL COUNT 4.63 4.5 - 5.5 mil/ $\mu$ L

METHOD : HYDRODYNAMIC FOCUSING BY DC DETECTION

WHITE BLOOD CELL COUNT 8.55 4.0 - 10.0 thou/ $\mu$ L

METHOD : FLUORESCENCE FLOW CYTOMETRY

PLATELET COUNT 261 150 - 410 thou/ $\mu$ L

METHOD : HYDRODYNAMIC FOCUSING BY DC DETECTION

**RBC AND PLATELET INDICES**

HEMATOCRIT 42.0 40.0 - 50.0 %

METHOD : CUMULATIVE PULSE HEIGHT DETECTION METHOD

MEAN CORPUSCULAR VOL 90.7 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 32.4 31.5 - 34.5 g/dL

METHOD : CALCULATED FROM THE HGB & HCT

MENTZER INDEX 19.6

RED CELL DISTRIBUTION WIDTH 12.3 11.6 - 14.0 %

METHOD : CALCULATED FROM RBC SIZE DISTRIBUTION CURVE

MEAN PLATELET VOLUME 10.9 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		NOT DETECTED	NOT DETECTED	
METHOD : GLUCOSE OXIDASE PEROXIDASE				
KETONES		NOT DETECTED	NOT DETECTED	
METHOD : NITROPRUSSIDE REACTION				
BLOOD		NOT DETECTED	NOT DETECTED	
METHOD : PEROXIDASE				
BILIRUBIN		NOT DETECTED	NOT DETECTED	
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	
<b>WBC DIFFERENTIAL COUNT - NLR</b>				
SEGMENTED NEUTROPHILS		65	40 - 80	%
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
ABSOLUTE NEUTROPHIL COUNT		5.53	2.0 - 7.0	thou/ $\mu$ L
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
LYMPHOCYTES		26	20 - 40	%
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
ABSOLUTE LYMPHOCYTE COUNT		2.21	1.0 - 3.0	thou/ $\mu$ L
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
NEUTROPHIL LYMPHOCYTE RATIC (NLR)		2.5		
EOSINOPHILS		4	1 - 6	%
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
ABSOLUTE EOSINOPHIL COUNT		0.33	0.02 - 0.50	thou/ $\mu$ L
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
MONOCYTES		5	2 - 10	%
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
ABSOLUTE MONOCYTE COUNT		0.41	0.2 - 1.0	thou/ $\mu$ L
METHOD : FLOW CYTOMETRY WITH LIGHT SCATTERING				
DIFFERENTIAL COUNT PERFORMED ON:		EDTA SMEAR		
<b>MICROSCOPIC EXAMINATION, URINE</b>				
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				



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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	
<b>MORPHOLOGY</b>				
RBC		NORMOCYTIC NORMOCHROMIC		
WBC		NORMAL MORPHOLOGY		
METHOD : MICROSCOPIC EXAMINATION				
PLATELETS		ADEQUATE		
<b>ERYTHRO SEDIMENTATION RATE, BLOOD</b>				
SEDIMENTATION RATE (ESR)	21	High	0 - 14	mm at 1 hr
METHOD : WESTERGREN METHOD				
<b>GLUCOSE, FASTING, PLASMA</b>				
GLUCOSE, FASTING, PLASMA	92		Normal 75 - 99 Pre-diabetics: 100 - 125 Diabetic: > or = 126	mg/dL
METHOD : ENZYMATIC REFERENCE METHOD WITH HEXOKINASE				
<b>GLYCOSYLATED HEMOGLOBIN, EDTA WHOLE BLOOD</b>				
GLYCOSYLATED HEMOGLOBIN (HBA1C)	5.3		Non-diabetic: < 5.7 Pre-diabetics: 5.7 - 6.4 Diabetics: > or = 6.5 ADA Target: 7.0 Action suggested: > 8.0	%
METHOD : HPLC				
MEAN PLASMA GLUCOSE	105.4		< 116.0	mg/dL
METHOD : CALCULATED PARAMETER				
<b>GLUCOSE, POST-PRANDIAL, PLASMA</b>				
GLUCOSE, POST-PRANDIAL, PLASMA	96		70 - 139	mg/dL
METHOD : ENZYMATIC REFERENCE METHOD WITH HEXOKINASE				
<b>CORONARY RISK PROFILE, SERUM</b>				
CHOLESTEROL	180		Desirable cholesterol level < 200 Borderline high cholesterol 200 - 239 High cholesterol > / = 240	mg/dL
METHOD : ENZYMATIC COLORIMETRIC ASSAY				



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TRIGLYCERIDES		169	High Normal: < 150 Borderline high: 150 - 199 High: 200 - 499 Very High: >/= 500	mg/dL
METHOD : ENZYMATIC COLORIMETRIC ASSAY				
HDL CHOLESTEROL		35	Low Low HDL Cholesterol <40 High HDL Cholesterol >/= 60	mg/dL
METHOD : ENZYMATIC, COLORIMETRIC				
CHOLESTEROL LDL		111	High Adult levels: Optimal < 100 Near optimal/above optimal: 100-129 Borderline high : 130-159 High : 160-189 Very high : = 190	mg/dL
METHOD : ENZYMATIC COLORIMETRIC ASSAY				
NON HDL CHOLESTEROL		145	High Desirable : < 130 Above Desirable : 130 -159 Borderline High : 160 - 189 High : 190 - 219 Very high : > / = 220	mg/dL
CHOL/HDL RATIO		5.1	High Low Risk : 3.3 - 4.4 Average Risk : 4.5 - 7.0 Moderate Risk : 7.1 - 11.0 High Risk : > 11.0	
LDL/HDL RATIO		3.2	High 0.5 - 3.0 Desirable/Low Risk 3.1 - 6.0 Borderline/Moderate Risk >6.0 High Risk	
VERY LOW DENSITY LIPOPROTEIN		33.8	High < OR = 30.0	mg/dL
<b>LIVER FUNCTION PROFILE, SERUM</b>				
BILIRUBIN, TOTAL		0.49	Upto 1.2	mg/dL
METHOD : COLORIMETRIC DIAZO				
BILIRUBIN, DIRECT		0.22	< 0.30	mg/dL
BILIRUBIN, INDIRECT		0.27	0.1 - 1.0	mg/dL
TOTAL PROTEIN		7.4	6.0 - 8.0	g/dL
METHOD : COLORIMETRIC				
ALBUMIN		4.6	3.97 - 4.94	g/dL
METHOD : COLORIMETRIC				
GLOBULIN		2.8	2.0 - 3.5	g/dL
ALBUMIN/GLOBULIN RATIO		1.6	1.0 - 2.1	RATIO
ASPARTATE AMINOTRANSFERASE (AST/SGOT)		20	< OR = 50	U/L
METHOD : UV ABSORBANCE				



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ALANINE AMINOTRANSFERASE (ALT/SGPT)		16	< OR = 50	U/L
METHOD : UV ABSORBANCE				
ALKALINE PHOSPHATASE		81	40 - 129	U/L
METHOD : COLORIMETRIC				
GAMMA GLUTAMYL TRANSFERASE (GGT)		20	0 - 60	U/L
METHOD : ENZYMATIC, COLORIMETRIC				
LACTATE DEHYDROGENASE		188	125 - 220	U/L
METHOD : UV ABSORBANCE				
<b>SERUM BLOOD UREA NITROGEN</b>				
BLOOD UREA NITROGEN		8	6 - 20	mg/dL
METHOD : ENZYMATIC ASSAY				
<b>CREATININE, SERUM</b>				
CREATININE		0.82	0.7 - 1.2	mg/dL
METHOD : COLORIMETRIC				
<b>BUN/CREAT RATIO</b>				
BUN/CREAT RATIO		9.76	8.0 - 15.0	
<b>URIC ACID, SERUM</b>				
URIC ACID		4.9	3.4 - 7.0	mg/dL
METHOD : ENZYMATIC COLORIMETRIC ASSAY				
<b>TOTAL PROTEIN, SERUM</b>				
TOTAL PROTEIN		7.4	6.0 - 8.0	g/dL
METHOD : COLORIMETRIC				
<b>ALBUMIN, SERUM</b>				
ALBUMIN		4.6	3.97 - 4.94	g/dL
METHOD : COLORIMETRIC				
<b>GLOBULIN</b>				
GLOBULIN		2.8	2.0 - 3.5	g/dL
<b>ELECTROLYTES (NA/K/CL), SERUM</b>				
SODIUM		133	<b>Low</b> 136 - 145	mmol/L
POTASSIUM		4.40	3.5 - 5.1	mmol/L
CHLORIDE		101	98 - 107	mmol/L
<b>THYROID PANEL, SERUM</b>				
T3		109.0	80 - 200	ng/dL
METHOD : ELECTROCHEMILUMINESCENCE				
T4		8.96	5.1 - 14.1	µg/dL
METHOD : ELECTROCHEMILUMINESCENCE				
TSH 3RD GENERATION		1.390	0.27 - 4.2	µIU/mL



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METHOD : ELECTROCHEMILUMINESCENCE

**PAPANICOLAOU SMEAR**

TEST METHOD

CONVENTIONAL GYNEC CYTOLOGY

METHOD : MICROSCOPIC EXAMINATION

SPECIMEN TYPE

P- 1101/22

TWO UNSTAINED CERVICAL SMEARS RECEIVED

METHOD : MICROSCOPIC EXAMINATION

REPORTING SYSTEM

2014 BETHESDA SYSTEM FOR REPORTING CERVICAL CYTOLOGY

SPECIMEN ADEQUACY

SATISFACTORY

METHOD : PAP STAIN & MICROSCOPIC EXAMINATION

MICROSCOPY

DRYING ARTEFAETS NOTED. THE SMEARS SHOW MAINLY SUPERFICIAL SQUAMOUS CELLS , FEW INTERMEDIATE SQUAMOUS CELLS, MANY CLUSTERS OF ENDOCERVICAL CELLS IN THE BACKGROUND OF MODERATE POLYMORPHS & RBC'S.

METHOD : PAP STAIN

INTERPRETATION / RESULT

NEGATIVE FOR INTRAEPITHELIAL LESION OR MALIGNANCY WITH MODERATE INFLAMMATION.

METHOD : PAP STAIN & MICROSCOPIC EXAMINATION

**Comments**

PLEASE NOTE PAPANICOLAOU SMEAR STUDY IS A SCREENING PROCEDURE FOR CERVICAL CANCER WITH INHERENT FALSE NEGATIVE RESULTS HENCE SHOULD BE INTERPRETED WITH CAUTION. NO CYTOLOGICAL EVIDENCE OF HPV INFECTION IN THE SMEARS STUDIED. SMEARS WILL BE PRESERVE FOR 5 YEARS ONLY.

**ABO GROUP & RH TYPE, EDTA WHOLE BLOOD**

ABO GROUP

TYPE O

METHOD : GEL COLUMN AGGLUTINATION METHOD.

RH TYPE

NEGATIVE

METHOD : GEL COLUMN AGGLUTINATION METHOD.

**XRAY-CHEST**

IMPRESSION

NO ABNORMALITY DETECTED

**TMT OR ECHO**

TMT OR ECHO

NEGATIVE

**ECG**

ECG

WITHIN NORMAL LIMITS

**MEDICAL HISTORY**

RELEVANT PRESENT HISTORY

NOT SIGNIFICANT

RELEVANT PAST HISTORY

cholecystectomy in 2018.



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

MENSTRUAL HISTORY (FOR FEMALES) regular 27-28/3 day

LMP (FOR FEMALES) 01/09/2022

OBSTETRIC HISTORY (FOR FEMALES) 1 lscs,a0,i1

LCB (FOR FEMALES) 7 YEARS BACK.

RELEVANT FAMILY HISTORY MOTHER:-HIGH BLOOD PRESSURE / HYPOTHYROIDISM

HISTORY OF MEDICATIONS NOT SIGNIFICANT

**ANTHROPOMETRIC DATA & BMI**

HEIGHT IN METERS 1.54 mts

WEIGHT IN KGS. 61 Kgs

BMI 26 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

BREAST (FOR FEMALES) NORMAL

TEMPERATURE NORMAL

PULSE 86/MIN.REGULAR, ALL PERIPHERAL PULSES WELL FELT, NO CAROTID BRUIT

RESPIRATORY RATE NORMAL

**CARDIOVASCULAR SYSTEM**

BP 110/70 MM HG mm/Hg  
(SUPINE)



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PERICARDIUM	NORMAL
APEX BEAT	NORMAL
HEART SOUNDS	NORMAL
MURMURS	ABSENT
<b>RESPIRATORY SYSTEM</b>	
SIZE AND SHAPE OF CHEST	NORMAL
MOVEMENTS OF CHEST	SYMMETRICAL
BREATH SOUNDS INTENSITY	NORMAL
BREATH SOUNDS QUALITY	VESICULAR (NORMAL)
ADDED SOUNDS	ABSENT
<b>PER ABDOMEN</b>	
APPEARANCE	NORMAL
VENOUS PROMINENCE	ABSENT
LIVER	NOT PALPABLE
SPLEEN	NOT PALPABLE
HERNIA	ABSENT
<b>CENTRAL NERVOUS SYSTEM</b>	
HIGHER FUNCTIONS	NORMAL
CRANIAL NERVES	NORMAL
CEREBELLAR FUNCTIONS	NORMAL
SENSORY SYSTEM	NORMAL
MOTOR SYSTEM	NORMAL
REFLEXES	NORMAL
<b>MUSCULOSKELETAL SYSTEM</b>	
SPINE	NORMAL
JOINTS	NORMAL
<b>BASIC EYE EXAMINATION</b>	
CONJUNCTIVA	NORMAL
EYELIDS	NORMAL
EYE MOVEMENTS	NORMAL
CORNEA	NORMAL
DISTANT VISION RIGHT EYE WITHOUT GLASSES	REDUCED VISUAL ACUITY 6/9
DISTANT VISION LEFT EYE WITHOUT GLASSES	WITHIN NORMAL LIMIT
NEAR VISION RIGHT EYE WITHOUT GLASSES	WITHIN NORMAL LIMIT



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

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

ADVICE:-
1)LOW FAT,LOW CALORIE, LOW CARBOHYDRATE, HIGH FIBRE DIET.
2)REGULAR EXERCISE.REGULAR WALK FOR 30-40 MIN DAILY.
3)REPEAT LIPID PROFILE AFTER 3 MONTHS OF DIET AND EXERCISE.

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
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



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PATIENT NAME : RAJASHRI GOSAVI

PATIENT ID : RAJAM310184181

ACCESSION NO : 0181VI000893 AGE : 38 Years SEX : Male

DRAWN : RECEIVED : 24/09/2022 10:04 REPORTED : 27/09/2022 15:56

REFERRING DOCTOR : SELF

CLIENT PATIENT ID :

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

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 glycosylated 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 glycosylated 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 Burts, 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, Freedm an 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.

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 and 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 hemorrhage, 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)



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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-

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 (hyperchloremic 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 Pregnancy, TOTAL T4 (µg/dL), TSH3G (µIU/mL), TOTAL T3 (ng/dL). Rows for First Trimester, 2nd Trimester, 3rd Trimester.

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

Table with 2 columns: T3 (ng/dL), T4 (µg/dL). Rows for New Born, 1-3 day, 1 Week.

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.



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Kindly note: Method specific reference ranges are appearing on the report under biological reference range.

Reference:

1. Burts C.A., Ashwood E. R. Bruns D.E. Tetz textbook of Clinical Chemistry and 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. Nelson Text Book of Pediatrics, 17th Edition

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 CHECKUP BELOW 40FFEMALE

ULTRASOUND ABDOMEN  
ULTRASOUND ABDOMEN  
GRADE I FATTY LIVER

**\*\*End Of Report\*\***

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Dr. Priyal Chinchkhede  
Consultant Pathologist

Dr. Ushma Wartikar  
Consultant Pathologist

Dr. (Mrs) Neelu K Bhojani  
Lab Head



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